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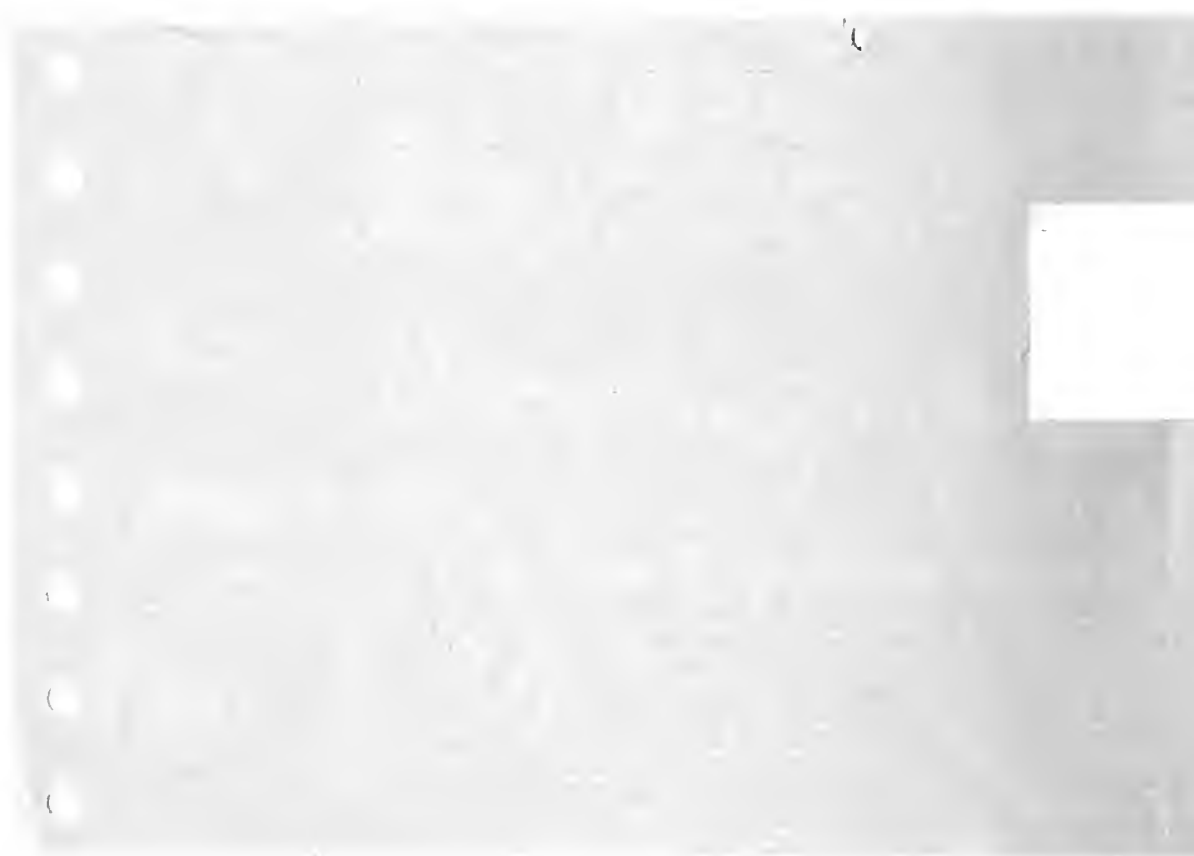

University of Illinois

PORK INDUSTRY CONFERENCE

December 2-3, 1982
College of Agriculture
Department of Animal Science
Cooperative Extension Service
Agricultural Experiment Station
University of Illinois at Urbana-Champaign

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WELCOME TO THE PORK INDUSTRY CONFERENCE! It is a pleasure to have you attend.

Commercial swine production has become industrialized. It is now a business in which experts in animal production, more than ever, must be concerned about finance, personnel management, labor efficiency, quality control, industry image, government regulation, etc. In other words, it is big business which requires constant awareness of advancements in technology, changes in organizational management and government policies and changes in the factors of production. Only those who make an effort to be informed in all facets of the business will be likely to operate profitably.

This conference is designed to assist swine producers to stay abreast of developments vital to their business. We hope that it will be recognized as and become a part of a continuing education program for professional, large-scale swine producers.

You are here because you need and value information to apply to your business. We hope to provide it.

We will be interested in your suggestions to make future conferences of maximum benefit to you.

D. E. Becker, Head
Department of Animal Science

The Department of Animal Science at the University of Illinois

THE DEPARTMENT OF ANIMAL SCIENCE IS CONCERNED WITH THE MULTIDISCIPLINARY ACTIVITIES associated with the production, care, and utilization of animals useful to mankind. It includes primary fields of study in behavior, genetics, environmental physiology and management, meat science and muscle biology, nutrition, and reproductive physiology. Beef cattle, horses, poultry, sheep, swine, and various companion and laboratory animals are studied to assist animal producers and owners to obtain more efficient performance, more economical production and other such improvements which ultimately benefit the general public.

Our staff includes about 140 people of whom 50 are academic staff members; 40 are nonacademic staff members in the offices, research farms, and laboratories; and about 50 are part-time teaching or research assistants who are pursuing graduate study. Several of the senior academic staff members have received national and international recognition for their accomplishments.

The work of the Department is divided into extension, research, and teaching. Extension or off-campus teaching is handled by eight full-time staff members. Each extension specialist conducts seminars, clinics, field days, etc., for livestock or poultry producers. Their primary work is to apply new research findings to the business of animal production or product processing.

Over the years the Department has had very active and productive research programs. Some of the more notable research accomplishments include the discovery of the value of antibiotics in livestock feeding, the elucidation of the amino acid needs of swine and poultry, the utilization of inorganic nitrogen by ruminants, the development of simplified corn-soybean meal rations for swine feeding, and the development of confinement production technics, particularly the use of slatted floors. Current research studies pertain to such topics as recycling of animal wastes, decreasing prenatal mortality in gestating sows, the inheritance of blood groups in swine, exercise physiology in horses, alcohol treatment of soybean meal, factors affecting amino acid needs of domestic animals, and animal behavior.

With the recent increase in student numbers, the teaching load in Animal Science increased significantly. At present our Department provides instruction for about 2,800 students a year in the classroom, and staff members advise 400 undergraduate (mostly juniors and seniors) and 90 graduate majors. Our graduates have many opportunities for employment.

The future of the Department of Animal Science appears bright. Meat consumption and the use of animals for companionship and in recreational activities are high and seem likely to increase further. Such an expanding animal industry will require an educational program to produce well-trained animal scientists and research programs geared to produce new and improved technology.

University of Illinois
PORK INDUSTRY CONFERENCE
December 2-3, 1982
Ramada Inn, Champaign

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Contents

Use of High-Oil Corn, Heated Soybeans, and Sunflower Seeds in Diets for Growing-Finishing Swine

K.L. ADAMS AND A.H. JENSEN

Some of the suggested advantages from adding fat to swine rations include: increases caloric density, improves acceptability, decreases carotene loss, minimizes dust, improves physical appearance, decreases wear on mixing and handling equipment, and may reduce feed wastage. Recently, several reports have indicated that the addition of fat to diets during late gestation and lactation resulted in modified milk composition and production, and in improved piglet survival when pigs from control sows had less than 80% survival and when piglets weighed less the 1.1 kg at birth (Seerley et al.,1974; Moser and Lewis, 1980; Boyd et al.,1982). Also, for weaned pigs from 3 to 8 weeks of age, increasing the caloric density of the diet has increased weight gains (Campbell et al.,1975; Aherne et al.,1980). Maximum diet energy intake occurred at a density of 3640 kcal of metabolizable energy per kg of diet (Campbell et al.,1975). Densities of this magnitude are usually not obtained in practical swine diets, thus fat, since it contains 2.25 times as much metabolizable energy as equal weights of carbohydrate or protein, is an attractive source of supplemental energy. However, fat (tallow, grease, lard, etc.) is usually relatively expensive, difficult to uniformly mix with other dietary ingredients, has limited storage time and the flow characteristics (especially in self-feeders) may be undesirable. The use of high-oil seeds, on the other hand, as possible alternative energy sources, could largely eliminate the mixing and handling problems.

The studies reported here were conducted to evaluate the utilization of the oil in high-oil corn, in heated soybeans and in sunflower seeds, by growing and finishing pigs. Typical composition values for these three high-oil seeds are shown in Table 1.

EVALUATION OF HIGH-OIL CORN

Watson and Freeman (1975), Creech and Alexander (1978) and Weber (1978) have described the development and characteristics of high-oil (HOC). The increase in oil content is largely due to an increase in the germ portion of the kernel. There is also, on the average, an increase of .38% protein with each 1% increase in oil. Selection for fatty acid composition of, and even for the position of the fatty acids in, the triglyceride molecule seems to be possible. Further, according to Creech and Alexander (1978), bushel-yield per acre of HOC with oil contents of

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from 6 to 8.5% were similar to high yielding commercial hybrid corn varieties.

Relatively little published information is available concerning the utilization of HOC by pigs. Terrill et al. (1951) used 46-kg pigs and Nordstrom et al. (1972) and Lynch et al. (1972) used 20-kg pigs in growth trials. Terrill et al. (1951) fed the corn (whole kernels) and a protein supplement free-choice. Nordstrom et al. (1972) fed diets that were isonitrogenous but not isocaloric. Lynch et al. (1972) used corns differing only 1.9% in oil content and obtained no significant difference in feeding value. Terrill et al. (1951) did see a reduction in the protein supplement consumed, but this was thought to be due to the increased protein content of the high-oil corn. Both Terrill et al. (1951) and Nordstrom et al. (1972) noticed a slight tendency for "soft" carcasses from pigs fed the high-oil corns, but this was not considered significant. Adams and Jensen (unpublished data) found no significant effect of HOC diets on carcass backfat thickness, carcass length or loin-eye area. Nor was the sliceability of the bacon affected.

It is essential that an appropriate dietary calorie:lysine ratio be maintained to ensure most efficient use of the added energy provided by corns containing 7 to 8% oil. Data in Table 2 represent typical results from the use of HOC in diets for weaned, four-week-old pigs, and they emphasize the necessity of knowing the lysine content of the corn. For example, Diet 2 was formulated, using HOC, to have the same protein level as Diet 1 (regular corn-soybean meal diet). But this resulted in a lower dietary lysine level in Diet 2 than that in Diet 1, with consequent wide calorie:lysine ratio. And gain/feed value was appreciably lower with Diet 2 (.497 vs .473). When diets with HOC were formulated to be the same calorie:lysine ratio (added synthetic lysine in Diet 3; higher protein level in Diet 4) gain/feed values were higher with the HOC diets (.516, .510) than with the regular corn diet (.497). This would indicate that in properly formulated diets the oil in HOC was efficiently utilized.

EVALUATION OF SOYBEANS

Heated soybeans (SB) have been compared to soybean meal as a source of amino acids. Frequently, daily gain and gain:feed values were higher with the heated soybeans diet (Hanke et al.,1972; Wahlstrom et al.,1971; McConnel et al.,1975). This probably reflected, at least in part, the higher energy (oil level) in the soybeans, but oil utilization per se was not measured and the added oil levels ranged only from 3 to 5% (Jimenez et al.,1963; Seerley et al.,1974a; Noland et al., 1976; Zamora and Veum, 1979).

As with HOC, diet formulation is critical in evaluation of the use of the dietary energy provided by SB (Table 3). We found that roasted soybeans (bean temperature of 115 to 120°C) as a source of protein and of supplemental energy could result in improvement of gain/feed values. Extrusion of the soybeans frequently resulted in better gain/feed values than those obtained with roasted soybeans (Table 4). Other early reports varied in this respect and interpretation is subject to such factors as roasting temperature, fineness of grind before mixing in the diet and calorie:lysine ratio.

EVALUATION OF SUNFLOWER SEEDS

Sunflower seeds (SFS) are another potential source of supplemental energy in swine diets. They are the second most popular oil-seed crop in the world, with

about 4 million acres in the United States in 1980 and world acreage was about 26.5 million in 1977 (Beard, 1981). Average tabular values for contents of protein and oil are 20 and 40%, respectively.

Laudert and Allee (1974) observed reduced feed intake by growing-finishing pigs as the dietary level of SFS increased. Baird (1980), however, reported that similar gains and improved feed efficiency by finishing pigs were obtained when 25 or 50% of the dietary corn was replaced by SFS. With young pigs, we found that feed intake was reduced (Table 5), possibly reflecting the high fiber content of the diets with the higher levels of SFS. Gargallo and Zimmerman (1980) reported that SFS hulls had no energetic value for the finishing pig. However, in our data (Table 5) average gain/feed value for the four SFS diets (.514) was higher than that for the control diet (.502).

OIL IN THE SEED VS RESPECTIVE EXTRACTED OIL

The use of high-oil corn (HOC), heated soybeans (SB) and sunflower seeds (SFS) to provide supplemental energy in swine diets can minimize the problems of mixing, handling and storing usually associated with use of animal fats and extracted oils. However, the relative digestibilities of the oils in the seeds compared to the respective extracted oils have not been specifically determined. Also, digestibilities of oil seeds that have been reported (Laudert and Allee, 1974; Kepler et al., 1980; Kepler et al.,1981) were apparently determined without taking into account the fecal fats present as soaps, which are not removed in the standard ether extraction procedure, or correcting for endogenous fecal fat excretion.

Thus, a study was initiated to compare the digestibilities of the oils in HOC, SB's and SFS with those of extracted corn oil, soybean oil and sunflower seed oil.

MATERIALS AND METHODS

Six diets were used in a 2 x 3 factorial (form of oil, intact (I) vs extracted (E); source of oil, HOC, SB, SFS) arrangement to evaluate the effect of form and source of oil on the digestibility of the oil by the young pig (Table 6). The diets in each oil-source-set (i.e., HOC vs regular corn plus corn oil; SB vs soybean meal plus soybean oil; SFS vs sunflower seed meal plus sunflower seed oil) were formulated to be isonitrogenous, isocaloric, isolysine or isomethionine (depending on which amino acid was first limiting) and to contain similar amounts of oil.

Pigs averaging 6.0 kg were used in a balance trial. They were confined to individual metabolism cages in an environmentally regulated room, and assigned to dietary treatment on the basis of litter and weight. After a seven-day adaptation period, each pig was randomly assigned an intact-seed-oil or the respective extracted-oil diet. During the subsequent five days total collection of feces and urine were made. This sequence was then repeated using the alternate oil source. Diets were equal-fed at the level of the pig eating the least during the adaptation period.

RESULTS

Digestibility values are shown in Table 7. The values for dietary fat include a correction for estimated endogenous fat excretion. This estimate was obtained by the regression of total grams of fat consumed on the total grams of fat excreted. The corrected fat digestibilities for the intact seed oil diets were lower ($P<.001$) than for the extracted-oil diets, but there were no significant differences in digestibilities due to source of oil. Based on single degree-of-freedom comparisons there were differences in corrected fat digestibilities between the intact seed oil and the extracted oils in the soybean ($P<.001$) and SFS ($P<.005$) diets, but not in the HOC diets. This would explain the interaction ($P<.02$) observed between source and form of oil.

Overall, dry matter ($P<.001$), energy ($P<.001$) and protein ($P<.01$) digestibilities in the diets with intact seed oil were less than in the diets with extracted oil. Oil source affected ($P<.001$) digestibilities of dry matter and energy but not of protein. Single degree-of-freedom comparisons between intact seed oil and extracted-oil diets showed differences in digestibilities of dry matter ($P<.001$), energy ($P<.001$) and protein ($P<.005$) in the soybean diets and in dry matter ($P<.005$) and energy ($P<.005$) in the SFS diets. There were no significant differences in HOC diets. Thus, there was a significant interaction between source and form of oil in the diets. There were no significant differences in the percent retained nitrogen due to dietary treatment (Table 8). The metabolizable energy in the intact oil diets was lower than that in the extracted oil diet ($P<.001$). There was a significant difference due to source ($P<.005$) and a significant source by form interaction ($P<.005$) in metabolizable energy with no difference between the intact- and extracted-HOC diets but a significant difference between the intact- and extracted-soybean and SFS diets ($P<.001$).

EFFECT OF PROCESSING ON UTILIZATION OF THE OILS IN SOYBEANS AND SUNFLOWER SEEDS

There is little published information relative to the effects of certain processing methods on the utilization of SB and SFS by young pigs. Heating (roasting or by extrusion) of SB destroys the trypsin-inhibitor making them a good source of amino acids for the pig. But the effects of processing on the utilization of the oil have not been clearly identified. Thus we carried out a study to evaluate the effects of roasting and extruding of SB, coarse and fine grinding of roasted SB, roasting of SFS and dehulling of SFS on the utilization by the young pig.

The results obtained indicated that the energy values were higher for extruded than for roasted soybeans, but grinding to different degrees of fineness did not significantly affect feeding value of roasted SB. Heating SFS did not improve their feeding value. The oil in SFS was slightly more digestible than the oil in the roasted SB. Dehulled SFS had significantly higher digestible energy value than did regular SFS.

SUMMARY

High-oil corn, heated soybeans and sunflower seeds can be effectively used in swine diets. Diet formulation to ensure appropriate energy:lysine ratio is essential for most efficient use of the oil energy in these high-oil seeds.

The oil in the seed was less digestible than was the oil after removal from

the seed.

Roasted and extruded soybeans are good sources of supplemental protein and energy in swine diets. Extruded beans were used more efficiently than were roasted beans.

Heating appeared to reduce digestibility of sunflower seeds. The oil in sunflower seeds was slightly more digestible than the oil in roasted soybeans, but total dietary energy was lower with the sunflower seed diets due to the higher fiber content. Dehulled SFS had significantly higher digestible energy value than did regular SFS.

The data reported here suggest that, even though the extracted oils were more digestible than the respective intact-seed oils, high-oil corn, heated soybeans and sunflower seeds are good sources of supplemental dietary energy and their use posed no problems in diet mixing, handling and storing.

Table 1. Composition of High-Oil Corn, Heated Soybeans and Sunflower Seeds

	Percent				
Seed	Crude protein[a]	Lysine	Crude fiber	Oil	Gross energy[b]
High-oil corn	10.8	.26	2.9	7.5	4154
Heated soybeans[c]	40.0	2.5	5.0	18.0	5404
Sunflower seeds	21.0	.5	29.0	36.0	6091

[a] Nitrogen x 6.25.

[b] Determined in an Adiabatic Bomb Calorimeter, kcal/kg.

[c] Heated in an infra-red roaster, temperature of beans 115 to 120°C as discharged from the roaster.

Table 2. Regular and High-Oil Corns in Diets for Young Swine

Diet Number	1	2	3	4
Corn	Regular		High-oil	
Added Lysine	-	-	+	-
Corn[a]	68.30	72.30	72.10	67.00
Soybean meal	29.00	25.00	25.00	30.30
Premix	2.70	2.70	2.70	2.70
Lysine (98% L-lysine-HCl)	-	-	0.20	-
Total	100.00	100.00	100.00	100.00
Crude protein, %	20.0	20.0	20.0	22.00
Lysine	1.03	.93	1.09	1.09
Kcal/% lysine	1705	2000	1705	1705
Results (28-days)				
Average daily gain, kg[b]	1.34	1.35	1.30	1.35
Average daily feed, kg	2.71	2.87	2.52	2.70
Average gain/feed	.497	.473	.516	.510

(Footnotes on next page)

Table 2 footnotes.

[a]Assay values:

	C.P., %	Lysine, %	Gross energy, kcal/kg
Regular corn	8.8	.24	3870
High-oil corn	10.8	.26	4154

[b]Average initial weight, 10 kg.

Table 3. Use of Roasted Soybeans As Source of Supplemental Protein and Energy In Diets For Finishing Pigs[a]

Diet	Corn:SBM[b]	Corn:roasted soybeans	
Dietary protein, %	16(14)[c]	16(14)[c]	17(15)[c]
kcal/% protein	113	120	113
Average daily gain, kg[d]	.73	.70	.77
Average daily feed, kg	2.50	2.27	2.34
Average gain/feed	.292	.308	.329

[a]UI, 1970.

[b]Solvent-extracted soybean meal (48.5% C.P.).

[c]Dietary protein level reduced two percentage points when pigs averaged 50 kg.

[d]Each value represents an average for two groups of five pigs each. Average initial weight was about 25 kg.

Table 4. Comparison of Roasted and Extruded Soybeans as Sources of Supplemental Protein and Energy in Diets for Young Swine[a]

Supplement	Soybean meal[b]	Roasted soybeans	Extruded soybeans
Dietary protein, %	16	17	17
kcal/% protein	110	112	112
Average daily gain, kg[c]	.77	.82	.76
Average daily feed, kg	1.83	1.83	1.49
Average gain/feed	.420	.449	.491

[a]UI, 1973.

[b]Solvent- extracted soybean meal (48.5%).

[c]Each value is an average for two groups of five pigs each, average initial weight of 8.0 kg. Trial lasted 42 days.

Table 5. Effects of Dietary Level of Sunflower Seeds on Performance of Young Pigs

Dietary SFS, %	0	6.5	13.0	26.0	26.0
Added lysine, %[a]	-	-	-	-	0.14
kcal/% lysine	1645	1690	1760	1850	1645
Average daily gain, kg[b,c]	.63[1]	.65[1]	.61[1,2]	.58[2]	.57[2]
Average daily feed, kg[c]	1.25[1]	1.24[1]	1.21[1,2]	1.12[2,3]	1.09[3]
Average gain/feed[d]	.502[1]	.525[2]	.503[1]	.519[2]	.525[2]

[a]Respective dietary lysine levels were 1.06, 1.07, 1.07, 1.09 and 1.23%.

(Footnotes continued on next page)

[b]Each value is an average of four pens of seven pigs, fed for 21 days (UI,1982).

[c]Values with different superscripts differ significantly (P<.05).

[d]Values with different superscripts differ significantly (P<.01).

Table 6. Composition of Diets With Intact or Extracted Oils

Oil source	Corn		Soybean		Sunflower	
Oil form	I	E	I	E	I	E
Source	76.50	-	51.00	-	-	-
Regular corn	-	69.70	-	-	-	-
Soybean meal	20.70	24.80	-	39.54	23.40	23.40
Sunflower seeds	-	-	-	-	56.00	-
Sunflower seed meal	-	-	-	-	-	39.12
Cornstarch	-	-	46.00	46.00	17.50	17.50
Corn oil	-	2.81	-	-	-	-
Soybean oil (crude)	-	-	-	12.08	-	-
Sunflower seed oil	-	-	-	-	-	15.21
Lysine-HCl[a]	.14	.07	-	-	.22	.22
Methionine	-	-	.08	.08	-	-
Premix[a]	2.66	2.62	2.92	2.30	2.88	4.55
	100.00	100.00	100.00	100.00	100.00	100.00
Calculated composition						
Protein, %	18	18	20	20	23	23
Lysine, %	1.0	1.0	1.28	1.28	1.25	1.25
Methionine-Cystine, %	.60	.60	.64	.64	.72	.72
Fat, %	6.5	6.5	11.40	12.10	22.40	22.70
Gross energy, kcal/kg	4058	4022	4360	4360	5060	5060
Analyzed composition						
Protein, %	19.2	19.0	21.5	19.1	22.8	27.3
Fat, %	7.4	7.9	12.3	14.3	23.5	22.9
Gross energy, kcal/kg	4124	4128	4369	4491	5077	5057

[a]Contained 78% lysine activity.

[b]Mineral, vitamin and antibiotic mix.

Table 7. Effect of Dietary Treatment on Percent Fat, Dry Matter, Energy and Protein Digestibilities

Oil form	Source: Corn	Soybean	Sunflower	Average
Fat				
Intact-seed	77.6	72.1	75.0[b]	74.9
Extracted	84.7	96.9[a]	88.9[b]	90.2[c]
Average	81.1	84.5	81.9	
Dry Matter				
Intact-seed	82.0	83.0	74.3[b]	79.8
Extracted	85.1	92.8[a]	88.9[b]	90.2[c]
Average[c]	83.5	87.9	77.6	
Energy				
Intact-seed	80.3	78.2	74.6[b]	77.7
Extracted	83.6	83.0[a]	82.1[b]	86.2[c]
Average[c]	82.0	85.6	78.3	
Protein				
Intact-seed	75.0	73.2	76.5	74.9
Extracted	74.8	87.0[b]	82.4	81.4[c]
Average	74.9	80.1	79.4	

[a]Extracted soybean oil values greater ($P<.001$ for fat, dry matter, energy; $P<.005$ for protein) than for intact-soybean oil.

[b]Extracted SFS oil values greater ($P<.001$ for dry matter; $P<.005$ for fat and energy) than for intact-SFS oil.

[c]Average value for extracted oil significantly ($P<.01$) higher than for intact-seed oils.

Table 8. Effect of Dietary Treatment on Percent Retained Protein and Percent Metabolizable Energy

Oil form	Source: Corn	Soybean	Sunflower	Average
Retained nitrogen				
Intact-seed	58.5	52.7	50.5	53.9
Extracted	56.1	56.9	60.3	57.8
Average	57.3	54.8	55.4	
Metabolizable energy				
Intact-seed	78.1	75.1	70.7	74.6
Extracted	78.8	90.1[a]	80.2[b]	83.0[c]
Average[d]	78.5	82.6	75.5	

[a]Extracted soybean oil value greater ($P<.001$) than for intact-soybean seed oil.

[b]Extracted SFS oil value greater ($P<.001$) than for intact-SFS oil.

(Footnotes continued on next page)

Table 8 footnotes (continued)

[c]Average value for extracted oils greater ($P<.001$) than for intact-seed oils.

[d]Average ME values differ among sources ($P<.005$).

LITERATURE CITED

Aherne, F. X., V. Danielsen and H. W. Nielsen. 1980. Fat utilization by three-week weaned pigs. University of Alberta 59th Annual Feeders Day Report. p. 83.

Baird, D. M. 1980. Sunflower meal and sunflower seed with supplemental lysine for finishing pigs. J. Anim. Sci. 51 (Suppl. 2):185 (Abstr.).

Beard, B. H. 1981. The sunflower crop. Sci. Am. 244:150.

Boyd, R. D., B. D. Moser, E. R. Peo, Jr., A. J. Lewis and R. K. Johnson. 1982. Effect of tallow and choline chloride addition to the diet of sows on milk yield and preweaning pig performance. J. Anim. Sci. 54:1.

Campbell, R. D., M. R. Taverner and P. D. Mullancy. 1975. The effect of dietary concentrations of digestable energy on the performance and carcass characteristics of early weaned pigs. Anim. Prod. 21:285.

Creech, R. G. and D. E. Alexander. 1978. Breeding for industrial and nutritional quality in maize. Chapt. 16 in Maize Breeding and Genetics. Edited by D. B. Walden. Publ. by John Wiley & Sons, Inc.

Gargallo, J. and D. Zimmerman. 1980. Effects of sunflower hulls on large intestine functionality in finishing swine. J. Anim. Sci. 51(Suppl.2):178 (Abstr.)

Hanke, H. E., J. W. Rust, R. J. Meade and L. E. Hanson. 1972. Influence of source of soybean protein, and pelleting, on rate of gain and gain/feed of growing swine. J. Anim. Sci. 35:958.

Jimenez, A. A., T. W. Perry, R. A. Picket and W. M. Beeson. 1963. Raw and heat-treated soybeans for growing-finishing swine, and their effect on fat firmness. J. Anim. Sci. 22:471.

Kepler, M. A., G. W. Libal and R. C. Wahlstrom. 1980. Effect of feeding ground sunflower seeds to gestating and lactating sows. South Dakota State Univ. Swine Day Report 80-8.

Kepler, A. M., G. W. Libal and R. C. Wahlstrom. 1981. Digestibility of sunflower seeds in swine diets. South Dakota State Univ. Swine Day Report 81-6.

Laudert, S. and G. L. Allee. 1974. Nutritive value of sunflower seed for swine. Proc. 1974. Swine Industry Day Report of Progress 221, Kansas Agric. Exp. Stn. p. 21.

Lynch, P. B., D. H. Baker, B. G. Harmon and A. H. Jensen. 1972. Feeding value for growing-finishing swine of corns of different oil contents. J. Anim. Sci. 32:1108 (Abstr.).

McConnel, J. C., G. C. Skelley, D. L. Handlin and W. E. Johnston. 1975. Corn, wheat, milo and barley with soybean meal or roasted soybeans and their effect on feedlot performance, carcass traits and pork acceptability. J. Anim. Sci. 41:1021.

Moser, B. D. and A. J. Lewis. 1980. Adding fat to sow diets - an update. Feedstuffs. March 3:36.

Noland, P. R., D. R. Campbell, R. K. Gage, Jr., R. N. Sharp and Z. B. Johnson. 1976. Evaluation of processed soybeans and grains in diets for pigs. J. Anim. Sci. 43:763.

Nordstrom, J. W., B. R. Behrends, R. J. Meade and E. H. Thompson. 1972. Effect of feeding high oil corns to growing-finishing swine. J. Anim. Sci. 35:357.

Seerley, R. W., J. W. Emberson, H. C. McCampbell, D. Burdick and L. W. Grimes. 1974a. Cooked soybeans in swine and rat diets. J. Anim. Sci. 39:1082.

Seerley, R. W., T. A. Pace, C. W. Foley and R. D. Scarth. 1974b. Effect of energy intake prior to parturition on milk lipids and survival rate, thermostability and carcass composition of piglets. J. Anim. Sci. 38:64.

Terrill, S. W., J. C. Moffitt, J. L. Krider, E. B. Earley and S. Bull. 1951. Value of low- and high-fat corns for fattening hogs. J. Anim. Sci. 10:902.

Wahlstrom, R. C., G. W. Libal and R. J. Berns. 1971. Effect of cooked soybeans on performance, fatty acid composition and pork carcass characteristics. J. Anim. Sci. 32:891.

Watson, S. A. and J. E. Freeman. 1975. Breeding corn for increased oil content. Proc. 13th Corn & Sorghum Res. Conf. p. 251.

Weber, E. J. 1978. Corn lipids. Cereal Chem. 55:572.

Zamora, R. G. and T. L. Veum. 1979. Whole soybeans fermented with aspergillus oryzae and rhizopus oligosporus for growing pigs. J. Anim. Sci. 48:67.

Digestive Development in the Pig and Nutritional Implications

R.A. EASTER AND R.P. CHAPPLE

The study of digestive mechanisms in the pig has been largely left to those specializing in gastroenterology, biochemistry, enzymology and pathology. Nutritionists have focused principally on metabolic and whole animal responses to feeding programs, largely ignoring the intermediate process of digestion. Digestive capacity significantly affects feedstuff utilization and should be a consideration in the selection of all feeding programs.

The pig is a simple-stomached omnivore with digestive features remarkably similar to the human. Major anatomical structures of the porcine digestive system are shown in figure 1. Each section of the alimentary tract contributes to the system's basic role: the degradation of large dietary molecules to simple constituent molecules that can be readily absorbed. Digestion and the control of digestion are complex processes; herein a simple overview will be presented as a basis of describing key nutritional implications of digestive development.

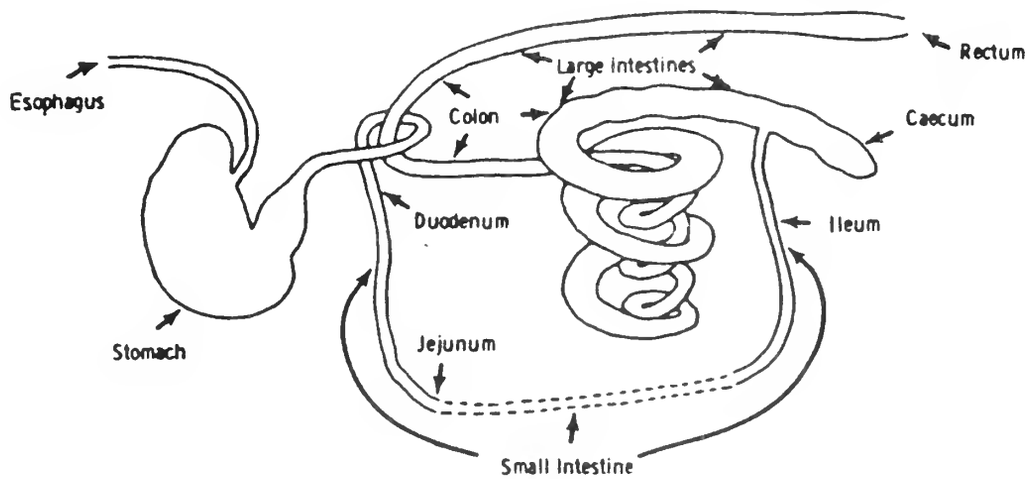

FIGURE 1. Schematic Representation of the Porcine Digestive Tract

The digestive process begins with the mastication of consumed food. Chewing facilitates digestion in several ways: particle size is reduced enhancing the exposure of large molecules to digestive fluids, salivary secretions are mixed with the food and limited enzymatic digestion is initiated. Porcine saliva contains significant quantities of only one enzyme, alpha amylase, none-the-less starch (amylose) digestion begins in the mouth and continues during passage through the esophagus and the stomach until halted by the acidic pH there.

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Chewing to reduce particle size is apparently inadequate as mechanical grinding of a diet prior to feeding will enhance performance (table 1). Young pigs chew feed more thoroughly than older pigs, thus the older pig generally benefits more from grinding of feed than does the young pig. Increased fineness of grind is associated with an increased incidence and severity of esophago-gastric ulcers.

Table 1. Effect of Fineness of Grind on Growth Rate, Feed Efficiency and Gastric Ulcer Incidence of Growing/Finishing Pigs[a]

Item	Fineness of Grind		
	1/16 in screen Fine	1/2 in screen Medium	1 in screen Coarse
Average daily gain, kg	.65	.63	.63
Feed/kg gain, kg	3.19	3.56	3.67
Mean ulcer index[b]	6.29	8.54	8.80

[a]Pickett et al. (1969).

[b]The smaller the value, the more severe the condition.

Feed entering the stomach is subjected to cycles of vigerous mixing for about three hours following a meal. Movement of the digesta from the stomach into the proximal small intestine occurs over a relatively long period of time and is regulated initially by stomach fill and subsequently by neural and hormonal effects. Clemens et al.(1975) have suggested that the half-time for particulate emptying from the stomach in the adult, concentrate-fed pig is about 10 hours. It should be apparent that the modulation of digesta passage from the stomach into the small intestine serves to provide a steady flow of nutrients to sites of intestinal absorption over relatively extended time periods.

Pathologies that interfer with stomach emptying may adversely affect digestion. Diet may also affect passage; dietary fat (Hunt and Know, 1968) as well as nutrient density have been shown to inhibit passage of digesta from the stomach. Digesta release in the suckling pig is also regulated by the intra-gastric precipitation of milk casein to form a semisolid curd. It has been suggested (Pettigrew, 1974) that "curd formation" or the lack thereof may contribute to the difficulty of obtaining maximum gains when very young pigs are fed non-milk diets.

The stomach is characterized by secretory surfaces. Some alkaline secretions are produced near the point of entry at the esophagus, however, the predominant gastric secretion is hydrochloric acid. In growing pigs the stomach pH is quite low, however, the suckling piglet has limited secretory capacity and elevated pH values are common. This is of practical concern as bacterial growth in the gastrointestinal tract is greatly increased at higher pH levels. The control of gastric pH is thought by some (Kidder and Manner, 1978) to be a major factor in diarrhea in early-weaned and artificially reared piglets.

The proteolytic enzyme pepsin is also found in the stomach where it initiates the degradation of proteins to peptides and ultimately free amino acids. The

activation of pepsin requires a pH less than 4 (Ryle, 1960) again pointing to the importance of correct stomach pH, particularly in the young pig.

The small intestine is the primary digestive and absorptive site for the pig. Anatomically it is divided into three sections: the duodenum, jejunum and ileum. Each section accounts for about one-third of the length of the small intestine. Digestion in the small intestine is promoted by enzymes of the intestinal mucosa (the inner lining of the intestine) and the pancreas and by bile acids produced by the liver and delivered via the bile duct. The relationship of the liver and pancreas to the small intestine is shown in figure 2.

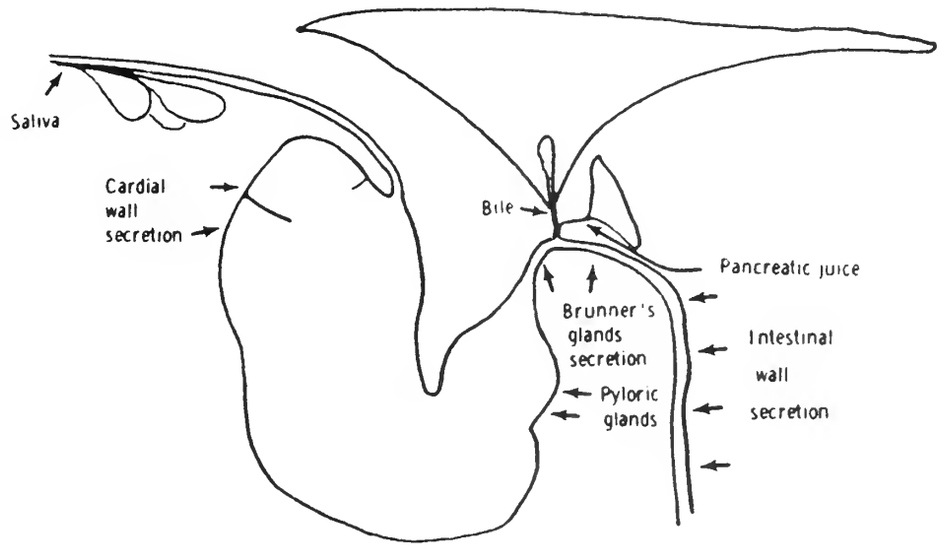

FIGURE 2. Relationship of the Liver and Pancreas to the Small Intestine

The mucosa is extremely active tissue that produces some digestive enzymes and provides absorptive surface for nutrient uptake. Structures referred to as villi add greatly to the total surface area. Denudation or loss of villi, an event which occurs in pathological conditions such as TGE (transmissible gastroenteritis) infection, greatly reduces absorptive capactiy and contributes to death in severe cases.

Digestion in the small intestine is accomplished through the enzymatic degradation of complex starch, protein and fat molecules. The major classes of digestive enzymes, their source and the compounds degraded are shown in table 2.

Protein digestion will be considered first. Dietary proteins are initially acted upon by pepsin in the lower region of the stomach to begin the process of reducing the complex protein molecules to large peptides. These peptides are attacked in the small intestine by the protolytic enzymes of pancreatic origin. The resulting small peptides are hydrolyzed to the individual amino acids by amino peptidases on the mucosal surface of the intestine. The free amino acids are absorbed by active transport into the mucosal cells and are subsequently passed to circulating blood for movement throughout the body. The phenomenon of active transport simply stated means that there are functional systems in the mucosal cell membranes that move the amino acids into the cell by the expenditure of energy. The transport sites are thought to be specific for categories of amino acids and there is evidence that competition for transport may inhibit the uptake of specific amino acids. The nutritional implication of this competition is not clear, however, it may be a factor in the performance reduction observed with severely imbalanced diets.

Table 2. Classes of Digestive Enzymes Active in the Small Intestine[a]

Enzyme Class		Origin	Substrate
Protease	pepsin	stomach	proteins
	trypsin, chymotrypsin, carboxypeptidase	pancreas	proteins and peptides
	aminopeptidase	mucosa	small peptides
Carbohydrase			
	amylase	pancreas	starch (amylose)
	lactase	mucosa	lactose (milk sugar)
	maltases	mucosa	maltose
	isomaltase		isomaltose, dextrins
	sucrase		sucrose (table sugar)
	glucoamylase		maltodextrins, starch limit dextrins
Lipase		pancreas	fat
Nuclease		pancreas	nucleic acids

[a]From Kidder and Manners (1978).

Dietary factors which inhibit the function of specific proteolytic enzymes will depress animal performance unless inactivated. The classical example is the Kunitz trypsin inhibitor found in raw soybeans. The data (table 3) show that trypsin activity is greatly reduced in the presence of the inhibitor found in raw soybeans while the level is quite high when pigs are fed soybean meal that has been heat-treated to destroy the inhibitor. Growth response is easily predicted from the level of enzyme activity in the intestine.

Table 3. Effects of Feeding Raw Soybeans (Harosoy Variety) on Trypsin Activity and Piglet Weight Gain[a]

Item	Heat treated Soybean meal	Raw soybeans
Trypsin activity[b]	287	29
Daily gain, kg	0.16	-0.05

[a]From Yen et al. (1977).

[b]Units of trypsin activity per kg body weight.

Protein digestion is time dependent with amino acid concentrations in plasma reaching maxima several hours after the ingestion of a meal. Crystalline amino acids, as they don't have to be released from proteins by digestive enzymes, are absorbed rather quickly following a meal. There is evidence that this differential absorption impacts on the utilization of crystalline amino acids provided

as supplements to intact proteins for meal fed animals such as the gestating sow. The explanation is simple. The rapidly absorbed amino acid from a crystalline source reaches the cellular site of protein synthesis before the other required amino acids and degradation of the early-arriving amino acid begins. Subsequently, other required amino acids, arising from intact protein digestion, arrive but cannot be completely utilyzed because a portion of the crystalline amino acid has been degraded.

Table 4. Effect of Meal Frequency on Performance of Pigs Fed Diets Supplemented with Crystalline Lysine [a]

Frequency of Feeding Meals/day	Lysine added, %	Average daily gain, kg	Feed efficiency gain/feed
1	.00	.435	.32
1	.20	.491	.36
6	.00	.446	.33
6	.20	.530	.38

[a]From Batterham and O'Neill (1978).

The data in table 4 support this concept. The addition of crystalline lysine improved both rate and efficiency of gain regardless of meal frequency. Note, however, that the improvement was greater when feeding was most frequent.

Rarely is protein digestion complete and undigested proteins pass from the small intestine into the lower tract and, practically speaking, are of no further value to the pig. This digestive failure involves many physical and chemical limitations that act to prevent complete hydrolysis of feed proteins by proteolytic enzymes. Much attention has been given to the problem, particularly to the matter of estimating amino acid availability. Some progress has been made but serious limitations continue to prevent full exploitation of availability concepts in diet formulation.

Fat digestion also occurs in the small intestine. Bile, a product of the liver secreted in response to hormone stimulation, is rich in sterol derivatives which aid in the emulsification of fats. Pancreatic lipase hydrolyzes dietary triglycerides (fats) to free fatty acids and monoglycerides. These compounds then aggregate in micelles or clusters of a few dozen molecules. Micelle formation is aided by the presence of bile salts. The micelles are relatively small and pass between the microvilli where monoglycerides and free fatty acids are taken up from the micellar solution by the mucosal cells.

Once absorbed the long-chain monoglycerides are rejoined to long chain fatty acids to form diglycerides and then triglycerides. The triglycerides aggregate in droplets called chylomicrons and pass into the lymphatic circulation and eventually to the systemic blood. Short chain farry acids enter the blood by a slightly different pathway.

There is little doubt that the older pig can efficiently digest and absorb dietary fat. Early reports (Eusebio et al., 1965; Frobish et al., 1970) indicated that the young pig could not efficiently utilyze fat (table 5). Most found these

results surprising since sow's milk contains from 30 to 40 percent fat on a dry matter basis. Milk fat does differ in character from typical feed-grade fats such as animal fat and vegetable oils. Milk triglycerides are rich in short-chain fatty acids (8:0, 10:0 and 12:0) and there is some evidence that these are more rapidly digested by the very young animal (Aw and Grigor, 1980) due to the action of a lipase (fat digesting enzyme) produced in the stomach.

Table 5. Effect of Fat Addition on Gain and Efficiency of Feed and Energy Utilization [a]

Item	Fat Source				
	Control	Butter	Coconut oil	Lard	Vegetable oil
Total gain, kg	7.62	6.68	6.20	5.84	6.23
Feed/gain	1.71	1.62	1.70	1.75	1.72
ME/gain, kcal/kg	5004	5733	6002	6209	6075

[a]Frobish et al. (1970). Values are averages for three experiments conducted for 28-day periods, average initial weight was 4.7 kg.

Two factors appear to have contributed to early errors in underestimating fat utilization by the young pig. First, simple digestion studies failed to appropriately correct for endogenous fecal fat; a source of error which has been recently reviewed by Adams (1982). Secondly, both Illinois (Allee et al., 1971) and North Carolina (Cline et al., 1977) reports demonstrated that improper diet formulation can cause depressed performance, not poor fat utilization *per se*.

Daily feed intake of pigs fed free-choice is dictated by the quantity of feed required to meet energy needs. Normally, less of a high energy diet will be consumed, consequently the concentration of all nutrients must be increased inorder that requirements can be met at lower intakes. As the results in table 6 indicate, rate of gain is not reduced when fat is added to a properly adjusted diet. It is generally accepted that dietary fat can be utilized by the young pig though additional information is needed particularly in regard to the effect of physical form of the fat on digestion.

Table 6. Performance of Three-week-old Pigs Fed Diets Formulated to Equal Nutrient/Calorie Ratios [a]

Item	Percent Fat in Diet		
	12	25	43
Dry matter consumed/gain	1.90	1.69	1.57
Average daily gain, g	375	410	403

[a]Cline et al. (1977)

Carbohydrate digestion is initiated in the mouth and stomach and proceeds in the small intestine to produce mono and disaccharides which are then absorbed. Most absorption is of the monosaccharides such as glucose, galactose and fructose. In the growing and adult pig fed standard diets, starch is the predominant carbohydrate consumed while lactose or milk sugar dominates in the diet of the suckling pig. Both must be broken down to simple constituents before absorption can occur.

The enzyme responsible for starch digestion, alpha amylase, is produced for the small intestine by the pancreas. Once amylase has converted starch to maltose maltotriose, alpha-limit dextrins and some glucose, further digestion must be accomplished by the enzymes of the intestinal mucosa. These enzymes are lactase, trehalase and four maltases (isomaltase, sucrase and two forms of glucoamylase). Trehalose has little significance ot pigs consuming standard diets. Lactase hydrolyzes the lactose found in milk to its constituent molecules, galactose and glucose; sucrase is essential for the utilization of sucrose and the maltases for the digestion of the various intermediates in starch degradation.

The classical study by Becker et al. (1954) illustrated the inability of the day-old pig to utilize sucrose, a sugar that is easily digested in later life. In that study six of seven pigs fed a sucrose-based diet died of diarrhea within nine days of assignment to the diet. Subsequent research confirmned that the neonatal pig is virtually unable to digest sucrose.

The age related changes in mucosal and pancreatic carbohydrase production have been extensively evaluated since 1954. The pattern depicted in figure 3 is typical of that observed regardless of breed of dam or treatment of dam prior to farrowing. At birth the pig is unable to digest either starch or sucrose, but is well prepared to hydrolyze lactose. With advancing age the ability to hydrolyze both maltose and sucrose increases dramatically while lactase production declines.

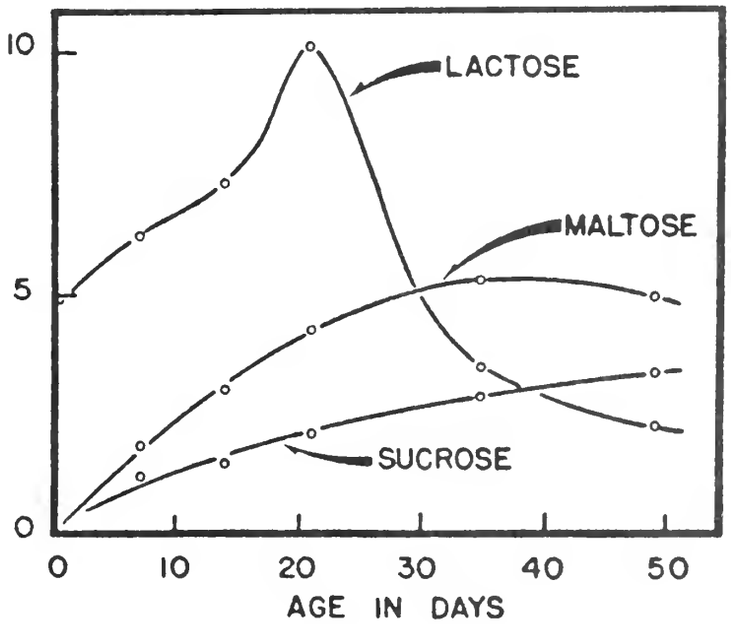

FIGURE 3. Effect of Piglet Age on Carbohydrase Production

The validity of the early results are confirmed by more recent data shown in table 7. Maltase activity in the fetal pig (day-105) is very low while sucrase is completely absent. Lactase activity is also quite low. Lactase is greatly elevated by the time of birth while sucrase and maltase increase only gradually with the greatest increase occurring between the fourth and eighth weeks of life.

Table 7 Relationship of Age to Total Disaccharidase Activity in the Intestine [a]

Enzyme [b]	Fetus Day-105	Age, weeks						
		Birth	1	2	3	4	6	8
Lactase	111	1,222	2,963	2,102	2,778	3,240	3,240	2,940
Maltase	74	106	588	1,361	4,166	4,583	7,916	15,324
Sucrase	0	0	393	2,242	2,578	3,046	3,102	5,648

[a]Aumaitre and Corring (1978)

[b]Values expressed as micromoles of substrate hydrolyzed per minute/whole intestine.

Likewise, the pancreatic production of amylase is low at birth and does not increase significantly until after the fourth week of life (table 8).

Table 8. Relationship of Age to Specific Activity of Amylase in Pancreatic Tissue [a]

Item	Birth	Age, weeks					
		1	2	3	4	6	8
Amylase [b]	70	78	134	150	527	1,529	1,506

[a] Corring et al. (1978).

[b] Specific activity of amylase per milligram of protein.

Numerous other reports (Kitts et al., 1956; Hartman et al., 1961; Manners et al., 1976; and Shields et al., 1980) confirmn the developmental sequence described above. From this it should be apparent that the neonatal pig is best prepared to utilize lactose; while the capacity to digest starch and several of the simple disaccharides is not well developed until the fourth to eighth week of life.

Feeding programs that conform to the digestive capacity of the pig have the greatest probability of success during this period. Recent evidence (table 9) that the growth of weanling pigs can be improved by the dietary inclusion of up to 25% dried whey (a good source of lactose) is not surprising.

Milk products are typically an expensive carbohydrate source in comparison to cereal grains, therefore it appeared desirable to attempt to cause the pig to secrete adequate quantities of amylase and the pertinent disaccharidases earlier in life. Fortunately the mechanisms controlling digestive development have been evaluated in other species (Moog, 1979).

Table 9. Effect of Dried Whey or Dried Skim Milk Addition to the Diet of Pigs Weaned at 14-Days of Age[a]

Item	Corn-soybean meal	25% Whey	15% Dried Skim Milk
Daily gain, kg	.120	.161	.128
Daily feed, kg	.264	.314	.226
Gain to feed ratio	.475	.529	.589

[a]Graham et al. (1981), values are from 14 to 42 days-of-age.

The induction of intestinal sucrase and maltase activities in adult men (Rosenweig et al., 1971) and rats by consumption of sucrose or fructose is well established. Lactose will influence intestinal lactase only after prolonged periods. The specific pancreatic amylase and lipase activity in growing pigs can also be induced, following a two to three day lag phase, by increasing the starch or fat content of the diet, respectively (Corring, 1980).

Although diet does influence the development of the small intestine and pancreas the triggering mechanism(s) for maturation are not yet completely understood. It appears that the hormone thyroxine plays a permissive role in certain aspects of intestinal development and a primary role in others. It is likely that both thyroxine and glucocorticoids are involved in the regulation of small intestine maturation.

It has been repeatedly shown that the administration of either glucocorticoids or ACTH to suckling rats will precociously induce pancreatic amylase (Sasaki et al., 1976), jejunal sucrase (Doell and Kretchmer, 1964) and maltase (Galand and Forstner, 1974). We decided to determine if similar, early induction of the enzyme systems could be effected in suckling piglets.

A series of 11 experiments have been completed in the effort to prematurely induce carbohydrate digesting enzymes in suckling pigs. Two hormones, adrenocorticotrophic hormone (ACTH) and hydrocortisone (HYD) have been employed. It appears that the young pig responds to hydrocortisone injections (table 10) by increasing the production of amylase, sucrase and maltase while lactase production is supressed. This is consistent with the previously mentioned experience with laboratory rats (Moog, 1975). Also, the weight of the small intestine, liver and pancreas are increased by the hydrocortisone administration. The hydrocortisone causes an apparent regression in the adrenal glands.

Experiments to measure the effect of hormone treatment on piglet growth have been limited in scope and the results are largely inconclusive. The data presented in table 11 suggest that both survival and weight gain of piglets receiving hydrocortisone before a 14-day weaning are improved over the untreated control pigs. The response to ACTH is confounded by the death loss experienced with pigs receiving that treatment.

In principle it now appears possible to enhance the suckling pig's capacity to digest starch and other carbohydrates early in life by administration of hormones. The specific dose, time of treatment and best compound are still being investigated.

Table 10. Carbohydrase Activities and Organ Weights of Piglets Injected with Graded Levels of Hydrocortisone from Day-14 to 26 of Lactation[a]

Item	Injection dosage, mg			
	0	8	16	24
	Activity per kg body weight[b]			
Pancreatic amylase	809	819	966	1,318
Intestinal amylase	144	187	274	407
sucrase	1.70	1.83	2.52	1.54
maltase	8.41	8.45	12.59	9.00
lactase	1.45	1.12	1.91	.75
	Organ weight per kg body weight[c]			
Small intestine	43.8	47.8	60.9	54.0
Liver	26.6	29.2	32.2	31.3
Pancreas	1.32	1.90	2.52	2.25
Adrenal glands	137	150	116	123

[a]Chapple, R. P.; Ph.D., Thesis, University of Illinois, in preparation

[b]Total enzyme activities are reported as g of each substrate hydrolyzed · h ·kg^{-1} body weight.

[c]Expressed as g of tissue per kg body weight.

Table 11. Effect of Either Adrenocorticotrophic Hormone (ACTH) or Hydrocortisone (HYD) Injections on Performance of Pigs Weaned at 14 Days-of-Age[a]

Item	Treatment[b]		
	Control	ACTH	HYD
Pigs weaned	22	22	24
Weaning weight, kg	3.91	4.07	4.25
Pigs surviving to 8 weeks	16	9	21
56 day weight, kg	11.04	13.76	14.13

[a]Chapple, R. P.; Ph.D. Thesis, University of Illinois, in preparation.

[b]Treated pigs were injected with either 15 IU/kg body weight of ACTH or 25 mg/kg body weight hydrocortisone-21 acetate 4 and 2 days before weaning.

The role of the lower bowel (cecum and large intestine) in porcine digestion has been reviewed elsewhere (Mason, 1980) and will be only briefly discussed here. Evidence is strong that amino acid absorption in the lower tract is nil. Carbohydrates are acted upon by the microbial enzymes to produce acetic, propionic, butyric and lactic acids which can be absorbed and contribute significantly to the energy status of the animal. The fermentation under certain conditions results

in osmotic imbalances that contribute to excess water loss in the form of diarrhea.

The lower bowel does play a role in both mineral and water reabsorption and excessive diarrhea can lead to both dehydration and electolyte deficiencies.

GENERAL REFERENCES

Kidder, D. E. and M. J. Manners. 1978. Digestion in the Pig. Kingston Press, Melcombe Road, Bath, England, U.K.

Low, A. G., and I. G. Partridge. 1980. Current Concepts of Digestion and Absorption in Pigs. Technical Bulletin 3. The National Institute for Research in Dairying, Reading, England, U.K.

LITERATURE CITED

Adams, K. L., The utilization of intact-seed oil by the young pig. Ph.D. Thesis. University of Illinois, Urbana.

Allee, G. L., D. H. Baker and G. A. Leveille. 1971. Fat utilization and lipogenesis in the young pig. J. Nutr. 101:1415.

Aumaitre, A. and T. Corring. 1978. Development of digestive enzymes in the piglet from birth to 8 weeks II. Intestine and intestinal disaccharidases. Nutr. Metab. 22:244.

Aw, T. Y. and M. R. Grigor. 1980. Digestion and absorption of milk triglycerides in 14-day-old suckling rats. J. Nutr. 110:2133.

Baily, C. B., W. D. Kitts and A. J. Wood. 1956. The development of the digestive enzyme system of the pig during its pre-weaning phase of growth. B: Intestinal lactase, sucrase and maltase. Can J. Agr. Sci. 36:51.

Batterham, E. J., and G. H. O'Neill. 1978. The effect of frequency of feeding on the response by growing pigs to supplements of free lysine. Br. J. Nutr. 39:265.

Becker, D. E., D. E. Ullrey, S. W. Terrill and R. A. Notzold. 1954. Failure of the newborn pig to utilize dietary sucrose. Science. 120:345.

Clemens, E. T., C. E. Stevens and M. Southworth. 1975. Sites of organic acid production and pattern of digesta movement in the gastrointestinal tract of swine. J. Nutr. 105:759.

Cline, T. R., J. A. Coalson, J. G. Lecce and E. E. Jones. 1977. Utilization of fat by baby pigs. J. Anim. Sci. 44:72.

Corring, T., A. Aumaitre and G. Durand. 1978. Development of digestive enzymes in the piglet from birth to 8 weeks I. Pancreas and pancreatic enzymes. Nutr. Metab. 22:231.

Corring, T., 1980. The adaptation of digestive enzymes to the diet: Its physiological significance. Reprod. Nutr. Develop. 2:1217.

Doell, R. G. and N. Kretchmer. 1964. Intestinal invertase: precocious development of activity after injection of hydrocortisone. Science 143:42.

Eusebio, J. A., V. W. Hays, V. C. Speer and J. T. McCall. 1965. Utilization of fat by young pigs. J. Anim. Sci. 24:1001.

Frobish, L. T., V. W. Hays, V. C. Speer and R. C. Ewan. 1970. Effect of fat source and level on utilization of fat by young pigs. J. Anim. Sci. 30:197.

Galand, G. and G. G. Forstner. 1974. Soluble neutral and acid maltases in the suckling-rat intestine. Biochem. J. 144:281.

Graham., P. L., D. C. Mahan and R. G. Shields, Jr. 1981. Effect of starter diet and length of feeding regimen on performance and digestive enzyme activity of 2-week old weaned pigs. J. Anim. Sci. 53:299.

Hartman, P. A., V. W. Hays, R. O. Baker, L. H. Neagle and D. V. Catron. 1961. Digestive enzyme development in the young pig. J. Anim. Sci. 20:114.

Hunt, J. M. and M. T. Know. 1968. Regulation of gastric emptying in Handbood of Physiology. Sect 6., Vol 4, pp 1917-1935. American Physiological Society.

Kitts, W. D., C. B. Bailey and A. J. Wood. 1956. The development of the digestive enzyme systems of the pig during its pre-weaning phase of growth. A. Pancreatic amylase and lipase. Can. J. Agr. Sci. 36:45.

Manners, M. J. 1976. The development of digestive function in the pig. Proc. Nutr. Soc. 35:49.

Mason, V. C. 1980. Role of the large intestine in the process of digestion and absorption in the pig. In A. G. Low and I. G. Partridge. Current Concepts of Digestion and Absorption in Pigs. Technical Bulletin 3. The National Institute for Research in Dairying, Reading, England, U. K.

Moog, F. 1979. Endocrine influences on the functional differentiation of the small intestine. J. Anim. Sci. 49:239.

Pettigrew, J. E. 1974. Milk proteins for artificially reared piglets. Ph.D. Thesis. University of Illinois, Urbana.

Pickett, R. A., W. H. Fugate, R. B. Harrington, T. W. Perry and T. M. Curtin. 1969. Influence of feed preparation and number of pigs per pen on performance and occurrence of esophagogastric ulcers in swine. J. Anim. Sci. 28:837.

Rosensweig, N.S., R. H. Hermand and F. B. Stifel. 1971. Dietary regulation of small intestinal enzyme activity in man. Amer. J. Clin. Nutr. 24:65.

Ryle, A. P. 1960. Parapepsinogen II, the Zymogen of parapepsin II. Biochem. J. 75:145.

Sasaki, R., M. Mura, T. Takeuchi, C. Furihata, T. Matsushimo and T. Sugimura. 1976. Premature induction of amylase in pancreas and parotid gland of growing rats by dexamethasone. Biochem. Biophys. Acta. 428:619.

Shields, R. G., Jr., K. E. Ekstrom and D. C. Mahan. 1980. Effect of weaning age and feeding method on digestive enzyme development in swine from birth to ten weeks. J. Anim. Sci. 50:257.

Yen, J. T., A. H. Jensen and J. Simon. 1977. Effect of dietary raw soybean and soybean trypsin inhibitor on trypsin and chymotrypsin activities in the pancreas and in small intestinal juice of growing swine. J. Nutr. 107:156.

Selection and Management of Gilts

F.X. AHERNE

The production efficiency and profitability of a farrow-to-finish operation is determined to a large extent by the average number of pigs weaned per sow per year. The potential weak links in maximizing sow reproductive efficiency are: heat detection, service management and care of baby pig at birth and weaning. These practices are what influence litter size at birth, number of litters per year and piglet mortality and thus influence number of pigs weaned per sow per year.

CULLING

Most survey data shows that the average sow produces four to five litters before being culled (Table 1). Poor conception rates, repeated production of small litters, disease and feet and leg problems are the most common reasons for culling sows in most swine herds. There are several advantages for commercial producers to keep sows for five or six litters or as long as they are performing satisfactorily. Most sows are reaching maximum litter size at farrowing and weaning at about their sixth litter. Therefore keeping the number of gilt replacements down will tend to improve production efficiency since sows will farrow and wean an average of 1 to 2 pigs more per litter than gilts. Some other advantages of keeping sows are better immunity to disease, higher conception rates, lower feed requirement, and ease of synchronizing estrus through weaning. Regardless of the advantages of keeping sows in the herd as long as possible, it is obvious that most commercial producers will replace 30 to 40% of their herd each year. Therefore, the selection and management of gilts is an important consideration in maximizing reproductive efficiency.

SELECTION AND MANAGEMENT OF GILT REPLACEMENTS

The selection and pre-service management of gilts is one area in which we are now beginning to see major changes. Traditionally gilts were selected at market weight and then kept until they reached 7 or 8 months and a weight of 110 to 130 kg, before breeding. At this age and weight it was expected that the gilts had reached their second or third heat. The basis of this practice was that gilts served at the third estrus produce larger litters than if served at first heat (puberty). However, there is now a considerable amount of evidence that gilts can

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be made to reach their second or third estrus by the time they reach market weight and that if served at that time will give as large a litter as gilts served at 130 kg or 7 to 8 months of age (Table 2).

There is some evidence that lifetime productivity of gilts served at second estrus may be as great or greater than that of gilts served at the third estrus (Table 3). Therefore, it may be advisable to serve gilts at the second heat rather than waiting for the third heat. However, it has been suggested that weight at service is an important factor in determining gilt performance. The data in Table 4 suggest that for best performance gilts should be served at 130 to 140 kg. However, this data is confounded since the gilts at 130 to 140 kg are probably at third estrus, whereas those of 90 kg were probably at first estrus. As shown previously, it is sexual maturity that influences reproductive performance and not body weight. Some data from Europe also suggest that subsequent culling rate is higher in gilts served at younger age (Table 5). This has not been our experience. It is possible that the gilts in this survey were fed restricted levels of feed during the finishing period which resulted in subsequent reproductive failure and/or leg weakness.

PUBERTY

A review of the literature suggests that gilts reach puberty at an average age of approximately 200 days. Puberty, in this paper, will be defined simply as the first estrus period at which the gilt ovulates. A great variation exists in age (120-235 days) and weight (70-140 kg) at which gilts reach puberty and many factors influence the onset of puberty. Some of these factors will be discussed in this paper.

BREED

There is evidence of some pronounced breed differences in age at puberty (Table 6). In this experiment a lower percentage of York gilts had reached puberty by 9 months of age than any of the other breeds. The average age of puberty was considerably lower for the Landrace breed than for the other three breeds with York and Duroc being the latest maturing. It has also been observed that crossbred gilts reach puberty earlier than either of the parent breeds (Table 7). It has also been reported that gilts sired by one boar may reach puberty significantly earlier than those sired by another boar (Table 8). This sire effect also indicates genetic differences in potential to respond to external factors associated with the stimulation of puberty.

We have observed that the fastest growing gilts are slowest to reach puberty (Table 9). It is possible therefore, that selection for early maturity would reduce growth rate. In a recent experiment, we exposed fast growing gilts to two mature boars at 55, 75 and 85 kg, but did not stimulate onset of heat in any of the 40 gilts. These gilts were marketed at an average age of 126 days at 97.5 kg. It is possible that such fast growing gilts would need an injection of estrogen before they could respond to boar stimulation. A recent report has shown that injections of estradiol benzoate to gilts at 140 days of age stimulated 60% of the gilts to reach puberty within five days of injection (Table 10).

SEASON

It has been observed that market weight gilts going to slaughter in the late summer show less sexual development than those going to slaughter at any other time of year. It has also been reported that the percentage of gilts showing normal estrus cycles at 9 months of age is less for gilts reaching breeding age during the summer than for gilts reaching breeding age during winter (Table 11). Similar results have also been observed by other researchers (Table 12). In this experiment gilts born in fall reached puberty at a significantly younger age than those born in spring. The well established affect of a boar in stimulating the onset of puberty was also observed in this esperiment.

LIGHT

Age at puberty may be influenced by length of daylight and rearing gilts in confinement under complete darkness has been shown to increase the age at puberty (Table 13). In fact reducing the hours of light to which gilts were exposed to six hours per day significantly increased age at puberty (Table 14).

HOUSING

Recently, several studies have been conducted to evaluate the effect of stocking density and confinement on sexual development of gilts. England and Spurr (1969) observed that gilts penned individually before breeding herd had lower conception rate, exhibited a higher percentage of irregular heats and subsequent litter size was smaller (Table 15). Other workers have also shown that individually penned gilts take longer to reach puberty (Table 16). In this study group size of up to 30 gilts per pen did not adversely affect the onset of puberty. In another study it was shown that individually penned gilts take longer to reach puberty and do not respond to boar stimulation (Table 17). Christenson *et al.* (1980) have shown in several studies that non-confinement or raising gilts on pasture does have a positive effect in reducing age at puberty and the percentage of gilts in dirt lots or on pasture that were cycling by nine months of age was significantly higher (Tables 18, 19). However, Ford and Teague (1978) have reported that crowding gilts does not apparently influence either the number of gilts reaching puberty or the age of the gilts at puberty (Table 20).

NUTRITION

In general, level of feed intake has little effect on age at puberty. A summary of results by Anderson and Melampy (1972) showed delayed puberty due to restricted feeding in 9 of 14 experiments whereas in five experiment restricted feeding hastened the onset of puberty (Table 21). In several recent experiments Friend *et al.* (1981) have also shown that ad libitum feeding reduces age at puberty (Table 22). However, severe restriction of growth rate by feeding only 50% of ad libitum has significantly reduced age at puberty (Haines *et al.*, 1959).

Several other experiments have shown that severe amino acid deficiency will significantly increase age at puberty (Table 23). It is also suggested that restricted feeding of gilts during the finishing phase may reduce calcium and phosphorus intake below that required for maximum bone strength and may lead to increased feet and leg problems subsequently.

It is therefore recommended that gilts be fed ad libitum a 14% protein diet

from initial selection at 65-70 kg until they are served. It is important that the gilt be in good body condition at time of service. Data from the ROP home test results over the last nine years shows clearly that the gilts selected today are considerably leaner and younger at 90 kg than they were nine years ago (Table 24). Today at mating, gilts may contain only 16 kg of fat whereas originally they contained 25-30 kg fat (Table 25). The effects of level of feeding of pregnant and lactating sows on body fat reserves is shown in Table 26.

As can be seen even gilts fed at very high levels had lost a considerable amount of body fat at the end of their third parity. It is considered that 4 kg of subcutaneous fat in the body (approximately 6.3 mm of mid-back fat depth) is indicative of impending reproductive problems.

Recent evidence by Whittemore et al., 1980 suggests that gilts or sows may actually show a positive body weight gain while still suffering a considerable loss in body fat reserves. It may be that we should be considering some system of measuring backfat on our gilts and sows in order to forestall any reproductive problems due to insufficient essential body fat.

PUBERTY INDUCTION

It has been know for many years that gilts may be stimulated to come into heat by the stress of transportation or as a result of being introduced to a new environment (Table 27). However, introduction to a mature boar is the most potent stimulus to the onset of heat (Figure 1). In this experiment 11 or 12 gilts exposed to a boar at 165 days of age reached puberty within days of exposure. Most experiments have confirmed that boar exposure is most effective but that the response is more likely to be that 50% of the exposed gilts reach puberty within 10 days (Table 28). It was suggested that puberty may be delayed if boar exposure was initiated when gilts were too young. However, later experiments have shown that this may not be so (Table 29). However, gilts of approximately 75 kg and 140 days at first exposure tend to respond best to boar exposure. A recent experiment suggests that 30 minute exposure to the boar is as effective as continuous exposure (Table 30). Gilts may be raised with castrates, gilts or boars up to 165 days of age without any significant effect on their subsequent response to boar exposure (Table 31). It has also been observed that there are differences between boars in their ability to stimulate gilts (Table 32). In another trial it was shown that using a urinary pheromone (boar mate) or an immature boar had little effect in stimulating the onset of heat in gilts (Table 33). A recent report suggests that it is the androgen steroid in the froth from the submasillary gland of the mouth that is the potent stimulus that elicits sexual response from the gilt. Therefore, the minimum boar age for successful gilt stimultion would be in excess of 10 months of age (Table 34).

The recommendation, therefore, is to do your initial gilt slection when they weigh 60-65 kg. Select these gilts from sows that have had three or more litters. By selecting gilts from sows that have the best four or five litter average performance, we have a much better chance of choosing productive gilts. The best sows in the herd can be chosen on the basis of litter size and birth weight of pigs born. An example of a simple selection index for sows is presented in Table 35. This index is calculated as the average number of pigs born alive in four or more litters plus four times the average birth weight in kilograms for pigs born in those litters. In our herd, we buy in performance tested boars and select our gilts from sows which had four or more litter and whose index is 16 or more.

Using the idea of puberty induction one can develop a system of selection of replacement gilts (Figure 2). Select 100% more gilts at 65 kg than required for replacements which allows for later culling at market weight. At selection the gilts are mixed in groups of 10 to 12 per pen. After mixing the gilts, a mature boar is put into the pen with the gilts for 20 minutes each day. Within 10 days of exposure to the boar 50% of the gilts come into heat.

Check these gilts again 18 to 24 days later and record which gilts are cycling normally. At market weight, select replacements from the gilts that had several heats. Final selection at this stage should be based on feet and legs, number of sound teats and individual performance, such as growth rate and back fat probe. Little emphasis should be placed on traits that are not correlated to performance criteria. Recently, it has been suggested that gilts that are raised in small litter groups will produce larger litters than those raised in large litter groups (Table 36). The reason for this is not know. However, it should be clearly understood that this does not meant that we select gilts from small litters, simply that if gilts are raised in small litters they subsequently produce larger litters.

Having selected cycling gilts, they are then served at the first heat after they reach market weight, which is usually about 110 kg liveweight. Litter size at birth and weaning and piglet birth weight will equal that of gilts selected in a more conventional manner.

Table 1. Relationship between culling and sow ouput in Dutch herds

	Culling Rate		
	Low	Average	High
Litter/sow	6.56	4.55	3.42
Replacements/yr. (pct.)	31.3	43.4	55.4
Litter/sow/yr.	2.06	1.97	1.89
Weaners/sow/yr.	17.9	17.1	16.4

Source: Krose and Van Male, 1979.

Table 2. Effect of weight of gilts at time of service on litter size. All served at 2nd heat

Gilt wt. kg.	Litter size
90-99	9.3
100-109	9.1
110-119	9.1
120-129	9.2
130-139	9.1

Hillyer, 1978.

Table 3. The effect of mating gilts at first, second or third estrus

Estrus when first mated	1	2	3
Piglets born 1. parity	8.4	9.8	10.4
Piglets alive 1. parity	8.3	9.6	9.8
Number of piglets per sow 3. parities	32.4	33.2	32.9
Number of piglets born alive 3. parities	30.9	32.9	31.6

MacPherson *et al.*, 1977.

Table 4. Effect of gilt weight at mating on first litter performance

	Weight at mating, kg					
	90	100	110	120	130	140
Born/litter	8.2	9.4	8.8	9.6	10.2	10.2
Weaned	5.8	5.6	6.9	8.0	8.4	8.4

O'Grady, 1979.

Table 5. Effect of age of gilt at fertile mating on culling rate and litters produced

Age at fertile mating (months)	No. of gilts	Percentage Culled after litter No. 1	2	1-6 Inc.	Litters to culling
7.0 - 8.0	350	29.1	16.0	86.8	3.65
8.5 - 10.0	319	20.4	10.3	65.8	5.08

Source: Jovic *et al.*, 1975.

Table 6. Effects of breed on age at puberty

Breed	No. of gilts	Behavioral anestrous	Age at puberty days
Landrance	68	10	173
Hampshire	89	12	207
Duroc	44	1	224
Yorkshire	119	20	221

Christenson and Ford, 1979.

Table 7. Relationship between energy intake and puberty in crossbreed and purebred pigs

	At puberty			
	Age (days)		Weight (kg)	
Breed	Restricted diet	Full diet	Restricted diet	Full diet
Cross				
D x L x H	200	200	81	96
Pure				
D, H, PC	231	220	87	101

D = Duroc, L = Landrace, H = Hampshire, PC = Poland China.

Table 8. The effects of sire on age at puberty in gilts

	Boar			
	A	B	C	D
Age at puberty	161	181	181	185

Hughes and Cole, 1975.

Table 9. The effects of growth rate on age at puberty

	Gilts which did not reach puberty	Gilts which did reach puberty
Wt. at slaughter (kg)	108.9	109.6
Daily gain (kg)	.82	.75

Aherne and Price, 1979.

Table 10. The effect of estrogens on attainment of puberty

	Estrogen treated		Control	
Boar exposure	+	-	+	-
Wt. at start (kg)	66.5	67.1	66.0	66.4
Days to puberty	13.2	20.1	19.5	36.0
Age at puberty (days)	153.2	160.1	159.5	176.0

Hughes and Cole, 1978.

Table 11. Influence of season on estrus traits

Trait	October to April	April to October
Gilts at 9 months of age:		
No. of gilts	168	242
Reaching puberty (%)	86	81
Cyclic (%)	81	76
Age at puberty (days)	197	191

Christenson, 1981.

Table 12. Effects of season and presence of boar

Date of birth	Spring	Fall	Boar Present	Boar Absent
Age at puberty (days)	237	202*	208	222
Wt. at puberty (kg)	113.7	103.0	104	113

Movrogenis and Robison, 1976.

Table 13. Effects of different light treatment on age at puberty and number of corpora lutea in gilts

	Initial		Puberty		No. of
Group	Weight (kg)	Age (days)	Weight (kg)	Age (days)	corpora lutea*
Complete darkness	38	104	101	201	11.3
6h darkness/day	37	104	83	165	13.5
Natural daylight	37	104	91	175	12.6

*Slaughtered approximately 30 days after insemination and corpora lutea were counted.

Hacker *et al.*, 1979.

Table 14. Performance of gilts with 6 or 8 hrs light/day

	6 Hours	8 Hours
ADG lb to 200 lb	1.65	1.68
F/G	2.84	2.80
Age at puberty	232 days	190 days
No. corpora lutea	13.5	14.0
No. born	9.5	9.7

Hacker, 1979.

Table 15. Estrus behaviour of gilts under confinement

	Individual pens	Group pens
Conception rate	83	94
% exhibiting irregular heats	28	16
Number born	8.2	9.1

England and Spurr, 1969.

Table 16. Effects of group size on age at puberty

	Group size	
Age at puberty (days)	222	207
Wt. at puberty (kg)	108	108

Mavrogenis and Robison, 1976.

Table 17.

	Gilts mixed boar adjacent	Gilts mixed no. boar adjacent	Individually penned boar adjacent	Individually penned no. boar adjacent
Age at start (days)	140	140	140	140
Age at puberty (days)	197	222	220	226

Robinson, 1974.

Table 18. Pasture vs Confinement, % Estrus

Age	Confinement	Pasture
5	0	4.9
6	29.1	37.8
7	52.3	59.8
8	58.1	81.7
9	68.6	91.5

Christenson *et al.*, 1979.

Table 19. Influence of housing on estrus traits

Traits	Confinement	Non-confinement
Gilts at 9 months of age:		
No. of gilts	222	188
Reaching puberty (%)	77	91
Cyclic (%)	71	85
Age at puberty (days)	197	191
Wt. at puberty (kg)	99	95

Christenson, 1981.

Table 20. Gilts raised with various levels of floor restriction

% Floor restriction	0	25%	50%
Age at puberty (days)			
Trial 1	200	196	207
Trial 2	190	184	201

Ford and Teague, 1978.

Table 21. Effects of energy intake on onset of puberty

	At puberty			
	Age (days)		Weight	
No. of trials	Resticted	Ad libitum	Restricted	Ad libitum
9	217	201	74	91
5	201	212	74	94

Anderson and Melamphy, 1972.

Table 22. Effects of level of feed intake on age at puberty

	Expt. 1		Extp. 2	
	Ad libitum	Restricted	Ad libitum	Restricted
Age at puberty (days)	173	194	159	170
Wt. at puberty (kg)	101	92	97	92

Friend *et al.*, 1981.

Table 23. Effects of protein adequacy on age at puberty

Trait	14% protein	10% protein
ADG (kg)	.63	.54
Age at puberty	160	179

Cunningham *et al.*, 1974.

Table 24. Gilts Home Tested

Year	Backfat (mm)	Average age at 90 kg (days)
1971	21.8	187
1972	21.3	185
1973	19.6	191
1974	19.1	188
1975	18.5	179
1976	17.8	174
1977	17.4	172
1978	16.8	177
1979	16.0	177
1980	15.7	174

Table 25. Body composition of hogs

	Liveweight (kg)			
	23	68	91	114
Muscle (kg)	9.4	23.9	31.7	37.7
Fat (kg)	4.1	14.6	21.8	32.8

Richmond and Berg, 1971.

Table 26. Composition of carcasses of gilts at mating weight and sows after completion of three reproduction cycles

		Sows on treatment Energy intake			
Carcass composition	Gilt	Low	Medium	High	Very high
Carcass Wt. (kg)	96	95	103	112	132
Subcutaneous fat (kg)	24.5	5	6.5	12	20
Fat in carcass (%)	25.5	5.3	6.3	10.7	15.2
Lean in carcass (%)	53.1	58.9	60.2	54.5	53.0

O'Grady *et al.*, 1975.

Table 27. Influence of various stimuli on pubertal response

Treatment	Estrus	%
Mixed only (M)	2/26	7.7
M transported (T)	2/24	8.3
M, T & relocated (R)	7/25	28.0
M, T, R & boar exposed	20/23	86.9

Based on a 10-day response period following initiation of treatment.
All treatments initiated at approximately 165 days.

Table 28

Weight of gilts	% Estrus Within 7-10 days
57-62	49
84-89	68
(George and England, 1974)	
85	50
105	50
(Brooks and Cole, 1973)	

Table 29. Effects of gilt age at first boar contact on attainment of puberty

	Age at first boar contact (days)								
	125	132	139	146	153	160	167	174	181
Age at puberty (days)	179	174	166	168	181	179	179	199	197
Days to puberty	54	42	27	22	28	19	12	25	16
Wt. at puberty (days)	83.6	80.0	78.0	74.0	79.5	80.0	80.0	88.0	88.5
Ovulation rate	13.7	13.0	12.0	14.0	13.3	12.5	13.5	12.5	14.7

Kirkwood and Hughes, 1979.

Table 30. The effect of boar exposure of puberty attainment and ovulation rate in the gilt

	Age at puberty (days)	Interval to puberty (days)	Ovulation rate
30-min. boar exposure	198.7	33.7	15.2
Continuous boar exposure	194.6	29.5	15.0
Control	232.1	67.1	15.3

Kirkwood and Hughes, 1980.

Table 31. Age and Weight at Puberty

Housing to 160 days	With castrates		With boars	
Exposed to mature boars at 160 days	-	+	-	+
Age at puberty (days)	220	182	197	181
Weight at puberty (kg)	106	81	95	78

Paterson and Lindsay, 1980.

Table 32. Effect of individual boars on puberty attainment and ovulation rate in the gilt

	Age at Puberty (days)	Interval to Puberty (days)	Ovulation rate
Boar A	178.4	13.4	13.2
Boar B	208.8	43.8	16.4

Kirkwood and Hughes, 1980.

Table 33. Puberty Induction in Gilts

	Age at puberty (days)	Interval to puberty (days)
Control	226	63
Pen change daily	211	46
Pen change daily + urinary pheromone	220	54
Boar exposure (6 month old boar)	204	39

Boar mate aerosol.
Kirkwood and Hughes, 1980.

Table 34. Effect of age of boar on puberty induction

Boar	Age at puberty (days)	Wt. at puberty (kg)
2 year old	182.0	88.8
11 months	181.6	87.5
6.5 months	206.0	102.4
Control	203	98.3

Kirkwood and Hughes, 1981.

Table 35.

	Sow 1		Sow 2	
	Number born	Avg. birth weight (kg)	Number born	Avg. birth weight (kg)
Litter 1	9	1.13	12	1.10
Litter 2	11	1.23	12	1.13
Litter 3	10	1.28	11	1.23
Litter 4	9	1.32	6	1.30
Average	9.75	1.24	10.25	1.19
Index	9.75 + 4 x 1.24 = 14.7		10.25 + 4 x 1.19 = 15.0	

Table 36. Fraternity Size Effects

Category	Litter size which gilt raised	Subsequent litter size of gilts
Expt. 1		
Small	5.5	10.5
Control	9.1	11.1
Expt. 2		
Small	6.0	11.5
Control	9.7	10.4
Expt. 3		
Small	5.8	11.9
Control	8.6	10.9

Rutledge, 1980.

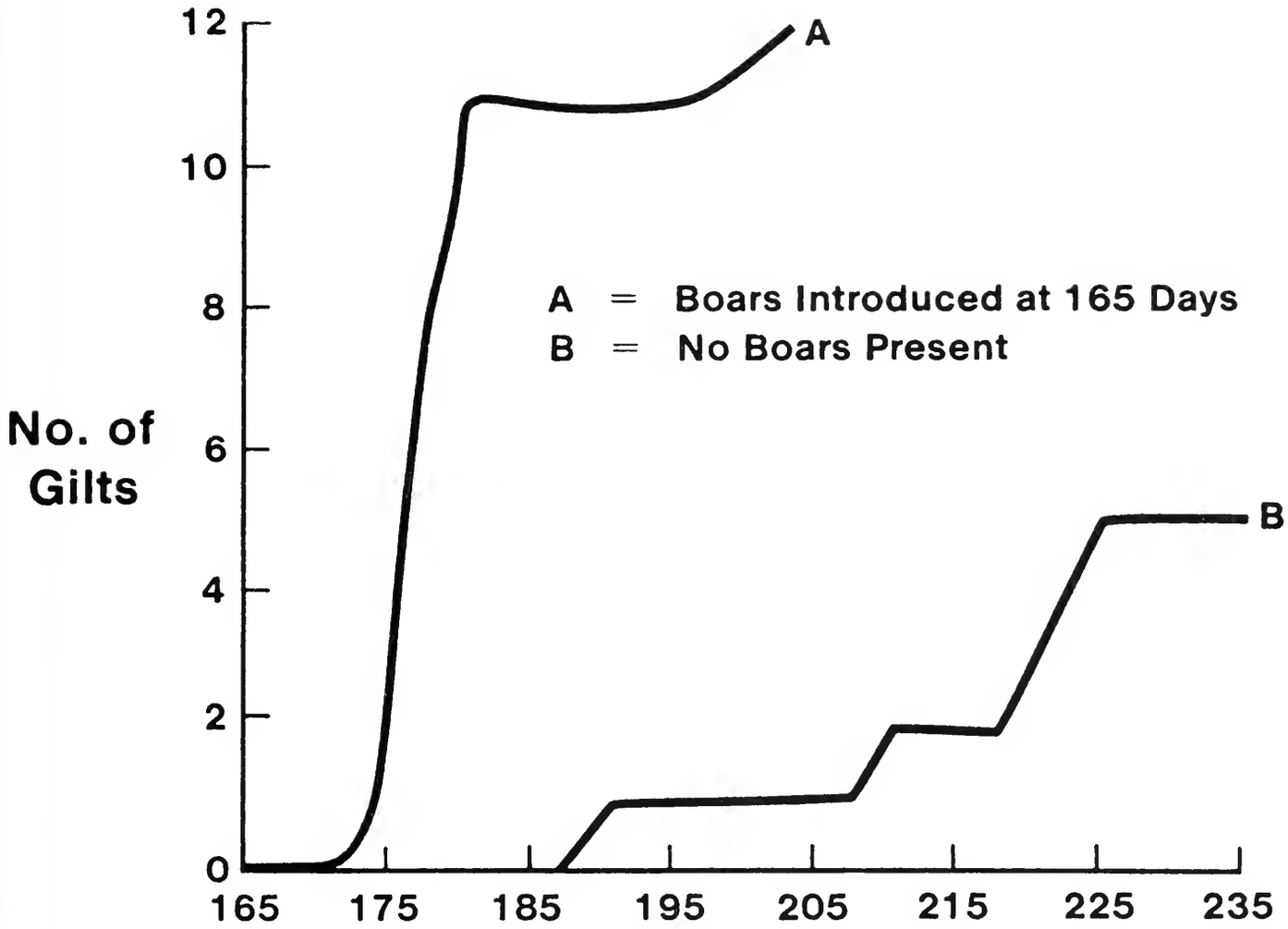

Figure 1. Effect of Boars on Estrus of Gilts. Brooks, 1976.

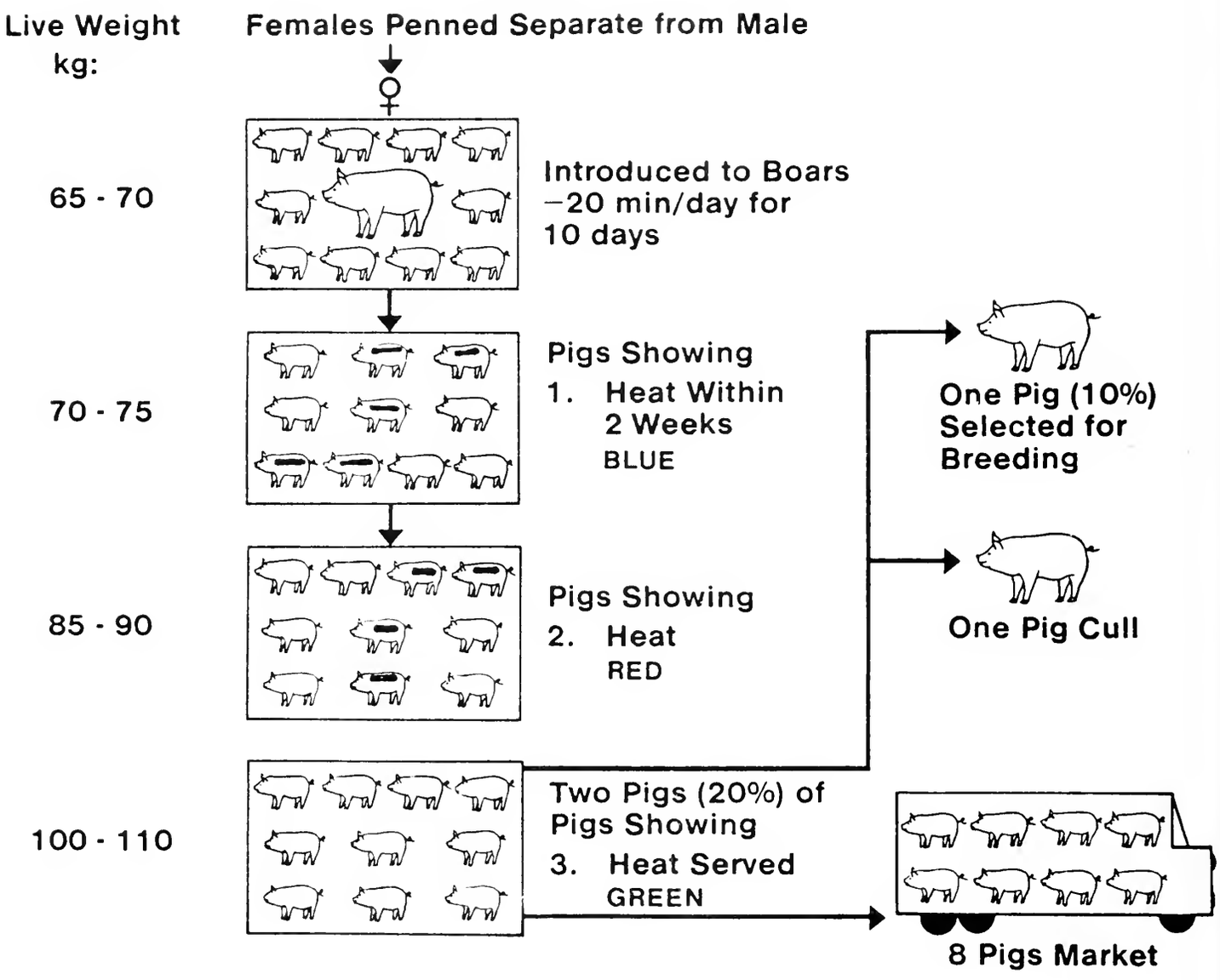

Figure 2. Selection of Female Pigs for Breeding.

Literature Cited

Aherne, F. X. and M. A. Price. 1979. Relationship between age at puberty (first ovulation) and body composition at market weight in gilts. European Association for Animal Production.

Anderson, L. L. and R. M. Melampy. 1972. Factors affecting ovulation rate in the pig. The Breeding Pig. 329.

Brooks, P.H. and D. J. A. Cole. 1973. Meat production from pigs which have farrowed. 1. Reproductive performance and food conversion efficiency. Anim. Prod. 17:305.

Christenson, P. K. 1981. Influence of Confinement and Season of the year on puberty and estrus activity in gilts. J. Anim. Sci. 52:821.

Christenson, P. K. and J. J. Ford. 1979. Puberty and estrus in confinement reared gilts. J. Anim. Sci. 49:743.

Cunningham, P. J., C. H. Naber, D. R. Zimmerman and E. R. Peo, Jr. 1974. Influence of nutritional regime on age at puberty in gilts. J. Anim. Sci. 39:63.

England, P. C. and D. T. Spurr. 1969. Litter size of swine confined during gestation. J. Anim. Sci. 22:220.

Ford, J. J. and N. S. Teague. 1978. Effect of floor space restriction on age at puberty in gilts and on performance of barrows and gilts. J. Anim. Sci. 47:828.

Friend, D. W., E. Laimond, M. S. Wolynetz and K. R. Price. 1981. Piglet pork production from gilts bred at puberty: chemical composition of the carcass and assessment of meat quality. J. Anim. Sci. 49:330.

George, P. B. and D. C. England. 1974. Estrus and early pregnancy of gilts in confinement. J. Anim. Sci. 38:1322.

Hacker, R. R., G. J. King, B. N. Ntunde and R. Narendran. 1979. Plasma Oestrogen, progesterone and other reproductive responses of gilts to photoperiods. J. Rept. Fert. 57:447.

Haines, C. E., A. C. Warnick and H. D. Wallace. 1959. The effect of two levels of energy intake on reproductive phenomena in duroc jersey gilts. J. Anim. Sci. 18:347.

Hillyer, G. M. 1978. Improving sow herd performance. Proc. Edinburgh Pig Conference.

Hughes, P. E., and D. J. A. Cole. 1978. Reproduction in the gilt. 3. The effect of exaginous oestrogen on the attainment of puberty and subsequent reproductive performance. Anim. Prod. 27:11.

Hughes, P. E. and D. J. A. Cole. 1975. Reproduction in the gilt. 1. The influence of age and weight at puberty on ovulation rate and embryo survival in the gilt. Anim. Prod. 21:183.

Kirkwood, R. N. and P. E. Hughes. 1980. A note on the influence of boar effect component stimuli on puberty attainment in the gilt. Anim. Prod. 31:209.

Kirkwood, R. N. and P. E. Hughes. 1980a. A note on the efficiacy of continous versus limited boar exposure on puberty attainment in the gilt. Anim. Prod. 31:205.

Kirkwood, R. N. and P. E. Hughes. 1979. The influence of age at first boar contact on puberty attainment in the gilt. Anim. Prod. 29:231.

Kroes, Y. and J. P. Van Male. 1979. Reproductive lifetime of sows in relation to economy of production. Livst. Prod. Sci. 6:179.

MacPherson, R. M., F. D. DeB. Hovell and A. S. Jones. 1977. Performance of sows first mated at puberty or second or third oestrus and carcass assessment of once bred gilts. Anim. Prod. 24:333.

Mavrogenis, A. P. and O. W. Robison. 1976. Factors affecting puberty in swine. J. Anim. Sci. 42:1251.

O'Grady, J. F., F. W. H. Elsley, R. M. MacPherson and I. McDonald. 1975. The response of lactating sows and their litters to different dietary energy allowances. 2. Weight changes and carcass composition of sows. Anim. Prod. 20:257.

Paterson, A. M. and D. R. Lindsay. 1980. Induction of puberty in gilts. 1. The effects of rearing conditions on reproductive performance and response to mature boars after early puberty. Anim. Prod. 31:291.

Rutledge, J. J. 1980. Fraternity size and swine reproduction. 1. Effect on fecundity of gilts. J. Anim. Sci. 51:868.

Whittemore, C. T., M. F. Franklin and B. S. Pearce. 1980. Fat changes in the breeding sow. Anim. Prod. 31:183.

Evaluation of Chinese Swine Germplasm in France: Preliminary Results

D. GIANOLA, C. LEGAULT, AND J.C. CARITEZ

INTRODUCTION

Prolificacy is of utmost importance in swine production. Little progress in increasing the number of pigs born per litter has been realized in the United States in the last forty years. Within breeds, most of the variation in litter size is non-genetic as heritability of litter size is of the order of 10% (Legault, 1970; Strang and King, 1970; Strang and Smith, 1979; Christensen, 1980). Therefore, it is not surprising that litter size is refractory to selection, particularly on an intra-herd basis where population sizes are low and random genetic change is important. Considering the important contribution of non-genetic factors to variability in prolificacy (90%), it is discouraging that extensive investment in research related to nutrition, physiology, disease control and housing systems has not yielded dividends in terms of litter size. Public expenditure on genetic improvement of prolificacy in swine has been negligible as suggested by the limited experimental evidence.

There have been only two well designed experiments aimed at improving litter size in swine, one in France (Ollivier, 1973; Ollivier and Bolet, 1981) and the other in Wisconsin (Rutledge, 1980). In the French experiment (120 females mated to 10 boars in each generation) genetic progress after five generations of selection was of .15 pigs/litter/year; after 11 generations of selection, progress was null. In the Wisconsin trial, selection proceeds either in standardized (S:6 pigs per litter) or unstandardized (N) litters. After three generations of selection, litter sizes in the S, N and control lines were 11.9, 10.6 and 10.9 pigs born per litter, respectively. Although these preliminary results are encouraging, caution is required as population sizes were small and the variance of the response is very likely large. Selection for components of litter size, i.e., ovulation rate and embryo survival, as indirect means of altering prolificacy have also been examined. While selection for ovulation rate was effective in increasing 3.7 ova over 9 generations compared to an unselected control (Cunningham et al., 1979), there was no response in litter size as embryonic mortality increased. The University of Nebraska is now undertaking an experiment to improve litter size by joint selection on ovulation rate and embryo survival. Data pertaining to the first cycle of selection in this experiment are now being analyzed (Johnson, 1982; personal communication).

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It may be possible to effect dramatic improvements in prolificacy by utilizing genes from highly prolific foreign populations. In the People's Republic of China there are breeds of exceptional prolificacy which might be of value in Western breeding programs. The Government of France authorized an importation of Chinese pigs, which took place in 1979. The objective of this paper is to discuss some preliminary results being obtained by the Centre National de Recherches Zootechniques (National Animal Production Research Center) of France and their possible implications for the U.S. swine industry.

SWINE BREEDING IN CHINA: A BRIEF INTRODUCTION

The national swine herd in the People's Republic of China is of about 300 million head, five times larger than that of the United States. However, the areas of China and of the United States are similar. There are between 100-150 breeds or strains of pigs, of which 40 may have economic importance (Epstein, 1969; Zheng, 1981). However, many of these "breeds" represent the same type of pig with the name varying according to the locality where they are raised. In addition, there was a tendency after 1949 of following the thinking of the Russian geneticist Mitchourin which led to widespread development of "new" breeds, thus contributing to the above number.

The history of scientific animal breeding in China following 1949 is dramatic. From 1949 to 1956, the teaching of Mendelian genetics and of statistics was forbidden, apparently on the grounds that these sciences provided wedges for the penetration of bourgeois ideology. In 1956, the Tsing Tao conference allowed the resumption of teaching of Mendelian genetics but, unfortunately, all work stopped during the "Great Cultural Revolution" between 1959 and 1966. Scientific reconstruction started after 1977 but, apparently, only one or two institutions recovered from the Lysenkoism blow. In the meantime, swine breeding suffered as the proliferation of "new" breeds led to the neglect of selective improvement of existing breeds and, perhaps, to the extinction of local sources of germplasm. At present, swine research programs are being streamlined, Chinese scholars have regained contact with the scientific community, and many Chinese postgraduate students are doing course work and research in European and American institutions, including the Department of Animal Science of the University of Illinois, where two Ph.D. students will start coursework in Animal Breeding and Genetics during 1983.

Breeds

Chinese pigs have been traditionally divided into two classes, the *North China* and the *South China* types (Phillips and Hsu, 1944). Zhang, Wu and Rempel (1982) distinguish a third type, *Tai Hu* found along the Yangtze river and the Tai Hu lake (provinces of Jiangsu, Shanghai and Zhejiang). In general, Chinese breeds of swine can be characterized as follows: a) exceptionally prolific (9 to 17 pigs born alive per litter); b) early maturing (puberty attained between 2 and 4 months of age); c) long-lived and docile; d) adapted to production systems where forages are routinely fed; e) low growth rate and mature size (e.g., 250 to 800 g of bodyweight gain per day); and f) fat carcasses (20 to 50 mm backfat) and poor conformation.

The two groups of main interest, from the viewpoint of prolificacy, are the *North China* and the *Tai Hu* pigs. Breeds that have been identified as exception-

ally prolific are the Da Min and Min Zhu in the *North China* group; and the Er Hua Lian, Meishan, Fen Jing and Jia Xing in the *Tai Hu* group. The Ting breed, found in the Hebei province by Epstein (1969) and by Gianola, Rundquist and Thompson (1980), seems to be the precursor of *North China* pigs (Zhang, Wu and Rempel, 1982) and is likely on the verge of extinction.

Summary statistics for *Tai-Hu* and *North China* pigs relative to sow productivity traits are presented in Table 1. The data for *Tai-Hu* pigs are weighted averages involving pigs from nine different farms, and the records for the Da Min breed come from another farm (Zhang, Wu and Rempel, 1982; Wu and Zhang, 1982). Data on Min Zhu pigs were obtained by Gianola, Rundquist and Thompson (1980). Averages for five breeds from a summary of studies conducted in experimental stations of the U.S.A. (Johnson, 1980) are presented for comparative purposes. Caution should be exercised in interpreting these data as differences between breeds are confounded with differences between countries, feeding regimes, herds and management systems. For example, the weaning traits on Chinese pigs are based on a 60-day weaning period while the American data pertain to animals weaned between 42 and 56 days of age. Using unweighted means, the general picture is the following: 1) Chinese females are considerably more prolific than their Western counterparts, irrespective of whether prolificacy is measured at birth or at weaning. The superiority of Chinese dams is, on the average, of 3.4 and 4.1 pigs at birth and at weaning, respectively. 2) Preweaning losses average 2.4 and 3.1 pigs in Chinese and Western breeds, respectively. 3) Chinese pigs are lighter at birth and at weaning.

Table 1. Birth and weaning statistics in Chinese and occidental breeds

	No. Pigs			Litterweight (kg)		Pig weight (kg)	
Breed[a]	Born alive	Weaned	No. nipples	Birth	Weaning	Birth	Weaning[b]
Er Hua Lian	12.6	10.8	--	9.8	114.4	.80	10.50
Meishan	14.0	12.2	16.8	12.6	192.8	.90	15.80
Fen Jing	13.9	11.9	17.4	10.8	141.3	.80	11.24
Jia Xing	13.0	10.9	16.4	10.8	99.4	.80	9.13
Da Min	13.9	10.8	--	13.7	131.5	1.00	12.23
Min Zhu	13.8	10.5	--	13.4	134.0	1.00	13.40
Yorkshire	10.8	7.4	--	12.5	96.2	1.16	13.00
Landrace	9.1	7.3	--	12.7	100.7	1.40	13.80
Hampshire	9.0	6.1	--	12.2	78.4	1.35	12.85
Duroc	10.1	6.4	--	14.0	84.4	1.39	13.19
Chester	11.6	8.0	--	12.6	103.0	1.09	12.87

[a]Data for Chinese breeds from Zhang, Wu and Rempel (1982). Min Zhu statistics from Gianola, Rundquist and Thompson (1980). Data on occidental breeds from Johnson (1980)

[b]Chinese pigs weaned at 60 days. The other breeds were weaned between 42 and 56 days of age

In *Tai Hu* pigs, growth is slow (Table 2), and the time to reach 100 kg is considerably longer than the 170-190 day period required for Western breeds growing at about .6-.7 kg/day (Johnson, 1980): However, nutrition may be largely

responsible for the differences. It is of interest, however, that Da Min and Min Zhu pigs grew faster than *Tai Hu* pigs, and at acceptable rates. The Da Min and Min Zhu were longer, thicker and taller than *Tai-Hu* animals (Table 3).

Table 2. Body weight, growth rate and estimated age at 100 kg[a] in Chinese breeds

Breed	Body weight (kg) 120 d	180 d	240 d	Growth rate (60-240 d) kg/day	Estimated age at 100 kg (d)
Er Hua Lian	--	33.2	54.2	.24	373
Meishan	32.5	52.0	77.5	.34	248
Fen Jing	41.7	60.5	78.9	.34	261
Jia Xing	22.5	38.1	56.5	.26	350
Da Min	37.8	66.9	106.3	.50	176
Min Zhu	--	65.1	84.8	.40	217

[a]Adapted from Zhang, Wu and Rempel (1982), and Wu and Zhang (1982)

Table 3. Body measurements in Chinese pigs at 240 days of age[a]

Breed	Body length (cm)	Heart girth (cm)	Body height (cm)
Er Hua Lian	95.2	84.9	49.2
Fen Jing	111.9	96.2	61.1
Jia Xing	115.6	97.8	61.7
Da Min	126.9	113.4	67.5
Min Zhu	121.7	104.8	61.3

[a]From Zhang, Wu and Rempel (1982), and Wu and Zhang (1982)

There is very limited data on carcass composition of Chinese breeds. Guerin (1982) reviewed the literature and some of his findings on slaughter variables for *Tai Hu* animals are presented in Table 4. Clearly, dressing percentage was much lower than the figures usually accepted for Western breeds (70-78%). More detailed carcass data are shown in Table 5. If purebred Landrace is regarded as a "control", the picture is the following: 1) while dressing percentage was lower in Meishans, crosses and backcrosses involving Da Min pigs were comparable to Landrace. 2) Carcass backfat and fat content were much higher in purebred and crossbred Da Min than in Landrace. 3) Lean content was definitely lower and carcass length and loin-eye area were smaller in animals carrying Da Min genes.

Chinese breeds attain puberty much earlier in their life cycle than Western breeds. Epstein (1969) reported that Fen Jing and Min pigs could be mated at 5 and 6 months of age, respectively. In a visit to China during 1976, Legault found Meishan gilts that were mated at 89 days of age at 26.8 kg body weight; they produced 7.6 and 6.6 pigs at farrowing and weaning, respectively. Fang et al. (1980a,b,c,d) conducted a number of studies concerning reproductive function in Er Hua Lian pigs. In males, mounting activity started at 14 days of age, and mounting of gilts in estrous occurred first at 43 days. Ejaculation and separation of the penis from the prepuce occurred at 50-60 days at 8-12 kg body weight. The semen, however, did not contain spermatozoa until the males were 60-75 days

of age. In the females, puberty occurred at an average age of 64 days (40-86), at a weight of 15 kg (8-20 kg). The duration of estrus averaged 7 and 4 days in gilts and sows, respectively. The estral cycle averaged 17 days (8-32) in gilts and the interval between weaning and first estrus was 4.5 days (2-11). Data on ovulatory output and litter size are presented in Table 6.

Table 4. Backfat, slaughter weight and yield in Tai Hu pigs[a]

Breed	Backfat (mm)	Slaughter weight	Yield (%)[b]
Meishan	22 at 72 kg	72 kg at 8 mo.	56
Fen Jing	32 at 68 kg	85 kg at 8 mo.	62
Jia Xing	50 at 75 kg	--	70

[a]Source: Guerin (1982)

[b]Excluding feet and head

Table 5. Carcass statistics in Meishan, Da Min, Landrace crosses and backcrosses, for pigs slaughtered at 90 kg

			Carcass					
Genotype	No. pigs	Yield	Length (cm)	Backfat (mm)	Bone %	Lean %	Fat %	Loin eye area $(cm)^2$
Meishan	9	66.8	--	24	12.4	44.0	28.1	--
Da Min (D)	12	71.5	90.0	32	9.2	45.4	35.6	23.2
Landrace (L)	12	72.3	97.6	26	9.3	56.4	28.1	31.1
L x D	14	72.4	93.2	36	8.7	46.3	27.2	23.6
D x (L x D)	12	73.2	95.4	36	8.4	47.4	39.4	26.7
L x (D x L)	9	73.7	96.4	37	7.8	47.6	39.5	26.7

Source: Zhang, Wu and Rempel (1982)

Table 6. Ovulatory rate and litter size in Er Hua Lian females

Age of gilt	Ovulation rate	Litter size Born	Litter size Alive	Age of gilt	Ovulation rate	Litter size Born	Litter size Alive
40-50d	5.67	-	-	100-110d	16.67	14.14	12.87
50-60d	6.33	-	-	110-120d	17.33	13.76	13.06
60-70d	9.75	-	-	150d	20.00	14.76	13.61
70-80d	9.33	-	-	180d	22.00	15.07	13.36
80-90d	8.67*	10.75	10.20	210d	23.70	15.30	13.21
90-100d	14.33	13.05	11.75	240d	26.00	15.15	12.82

Source: Fang et al. (1980)

*Data on ovulation rate and litter size are inconsistent due to an ambiguity in the summary of the paper

This study suggests that Er Hua Lian females could be mated as early as 3-4 months of age without compromising first farrowing performance. Perhaps these results also apply to other breeds in the Tai Hu group.

In China, swine production takes place in a system which attempts to minimize the utilization of grain as a nutritional input. In fact, only 7% of the national grain output is used in animal production (Guerin, 1982). Forages, aquatic plants (e.g., water hyacinth, *Eichkornia crassiper* or *speciosa* in Guang Zhou) and domestic and industrial waste substantially contribute to the feeding program of swine. Legault (1978) discussed feeding programs applied in the Shanghai area to Fen Jing and Mei Shan pigs; these are described in Table 7. Feeding programs based on forages and natural and artificial selection have very probably had an impact on the morphology of Chinese pigs as suggested by the development of the digestive tract (Table 8) and the "pot belly" phenotype encountered in most breeds.

Table 7. Feeding program (kg/day) applied to Fen Jing and Mei Shan animals in the Shanghai area

Category	Fen Jing		Mei Shan	
	Concentrates[a]	Forages[b]	Concentrates[a]	Forages[b]
1. Gestating sows				
a) First 2 months	1.5	7.5	1.0	5.0
b) Last 2 months	2.0	8.0	1.5	7.0
2. Lactating sows	2.5	7.3	2.8	7.0
3. Pigs, 0-60 d (total)	10.0	10.0	11.0	11.0
4. Pigs, 60-120 d	.8	2.3	.8	1.2
5. Pigs, 120-180 d	1.0	3.0	2.0	4.0
6. Pigs, 180-210 d	1.3	3.6	2.0	4.0

[a]Mixture of a "fine" ingredient (wheat, oats, soybean meal, rapeseed meal, rice) and a "hard" ingredient (rice bran, etc.)

[b]Aquatic plants, cabbage, silage, sweet potatoes

Source: Legault (1978)

Table 8. Development of the digestive tract in Xin Hua and Hua pigs compared to Yorkshire

Breed	Organ weight as a % of live weight		
	Stomach	Small intestine	Large intestine
Xinhua	1.5	3.3	5.3
Hua	1.5	2.2	4.7
Yorkshire	1.1	1.4	2.4

Source: Legault (1978)

THE FRENCH EXPERIMENTS

Background

The governments of France and of the People's Republic of China started negotiations in 1975. On November 24, 1979, a group of nine Chinese pigs arrived at the *Domaine Experimental du Magneraud*, a research farm located in Western France at about 35 km distance from the Atlantic Ocean. The group consisted of 3 animals (one male and two females) of each of three breeds: Mei Shan (MS), Jia Xing (JX) and Jin Hua (JH). As discussed before, the MS and JX breeds belong to the *Tai Hu* group. The JH comes from the Zhe Jiang province, and although less prolific and smaller than *Tai Hu* animals, is well known in China for the quality of its ham.

The first mating was conducted as follows:

Boars	No. Females				
	MS	JX	JH	LW[a]	FL[a]
MS	2	-	-	4	4
JX	-	2	-	4	4
JH	-	-	2	4	4

[a]LW: Large White; FL: French Landrace

The Large White and French Landrace females were culled after weaning the second litter and were progressively replaced by the six F_1 types. Also, MS females born at Magneraud were inseminated by LW boars to produce LW x MS gilts. The herd now consists of 84 females handled in 7 groups of 12 animals each. Pigs are weaned at 29-30 days of age, and after weaning sows stay for about 4 weeks in an open-front building in close proximity to the boars until pregnancy is confirmed. During pregnancy, females stay in a pasture of 2500 m^2 with access to shelters until the last week of gestation. During gestation, the females are fed 1.6-2.2 kg of concentrate (15.5% crude protein and 3100 Kcal digestible energy/day), and 3-4 kg of forage/day. Suckling pigs have free access to a commercial ration after 5 days of age.

Sow productivity traits. Data on age at puberty, number of nipples, litter size and weight, and feed consumption are presented in Table 8.

Table 9. Puberty and sow productivity statistics in Mei Shan (MS), Jia Xing (JX), Jin Hua (JH), Large White (LW), French Landrace (FL) and their crosses

Genotype	No. Nipples	Age at Puberty(d)	Litter size		Litter weight(kg)		Feed consumed (lactation,kg)
			Born alive	Weaned	Birth	21 days	
MS	16.3	81	13.7	13.1	15.3	55.7	113
JX	19.9	91	9.5	9.4	8.1	36.8	91
JH	16.5	109	10.1	8.8	6.6	27.8	92
LW	-	-	10.2	9.2	14.8	55.3	160
FL	-	-	8.8	8.6	13.9	54.1	160
MS x LW	14.7	87	13.2	11.4	19.5	61.3	123
MS x FL			12.1	11.0	16.7	60.0	125
JX x LW	16.7	93	14.0	13.3	14.0	62.1	132
JX x FL			13.5	12.1	14.6	58.4	127
JH x LW	15.8	96	9.8	9.0	10.1	40.2	118
JH x FL			11.4	10.2	12.1	46.7	114

Source: Legault and Caritez (1982)

Bearing in mind that results represent progeny of only one sire from each of the Chinese breeds, the general trends are the following: 1) Chinese breeds and crosses attained puberty much earlier than would be expected in Western breeds. Heterosis of the order of 30-40% is suggested. 2) The high prolificacy of Mei Shan, both at birth and at weaning was confirmed. This was not the case with Jia Xing. However, JX crosses were more prolific than their Mei Shan counterparts, suggesting that the JX foundation animals might have been related, i.e., crossing

could have led to recovery from inbreeding depression. Excluding the JH crosses, the numerical productivity of the remaining 4 crossbred genotypes is about 30% larger than that of European breeds. This is equivalent to 5-8 additional pigs weaned per sow per year. Preliminary estimates of heterosis involving MS x LW and LW x MS crosses are 12.1% for litter size at birth, 14.6% for number born alive, and 7.9% for number weaned. 3) Litter weight at 21 days of lactation was larger for MS, MS-crosses and JX-crosses, than for Large White and Landrace. Nevertheless, this comparison is biased in favor of the European breeds as LW and FL sows nursed crossbred pigs while purebred Mei Shan sows nursed purebred pigs. 4) LW and LF sows consumed 30-35 kg more of concentrates during lactation than Chinese breeds and crosses nursing equivalent litter weights at 21 days of age (MS, MS-crosses and JX crosses).

In summary, these results suggest that crossing MS and JX with LW and FL results in females that can reach reproductive status at least one month before LW or LF, consuming less concentrate feed (savings of 120-180 kg concentrate per sow per year) and weaning 5-8 extra pigs per sow per year. In addition, it appears that the subsequent prolificacy of the crossbred female is not compromised by being raised in a large fraternity. This would justify using a purebred Chinese as a maternal line because the cost of producing an F_1 female would be lower.

Ovulation rate and embryo survival. Rombauts, Mazzari and du Mesnil Du Buisson (1982) studied the estrus cycle, ovulation rate, embryo survival and ovarian and uterine characteristics in purebred Chinese and F_1 gilts. The F_1 females were flushed for the duration of the cycle before service. The matings were planned to produce a 75% Chinese conceptus, retaining 100% of the individual heterosis: 1) MS♂ to (JX x LW, JX x FL), 2) JX♂ to (MS x LW, MS x FL), and 3) MS♂ (JH x LW, JH x FL). Ovulation rate was studied by endoscopy in 5 and 4 successive cycles for the pure Chinese and F_1 animals, respectively. The females were slaughtered at 319-357 days of age at a body weight of 150-177 kg to check for embryo survival. It was observed that en route to or in the operation room, MS and JX had a calm behavior and required lower doses of pentothal and halothane than LW of the same weight. However, the F_1 females required higher doses than LW of equivalent weight. Presence of heat was easily detectable, in the absence of a boar, in the purebred Chinese and F_1 females. Three MS females were accidentally mated by their littermates in Magneraud and farrowed at 204, 201 and 206 days of age producing litters of 1, 4 and 5 pigs, respectively.

Data on duration of estrus cycle and ovulation rate are shown in Table 10. The duration of the estrus cycle in Chinese and F_1 gilts was not significantly different from that usually found in European breeds; the JX breed had a somewhat shorter cycle. The length of time that the female remained in estrus tended to be somewhat longer than that found in European breeds (about 53 hours), especially for Mei Shan and Mei Shan crosses. Ovulatory output was comparable to that found in LW (15.4), FL (13.7) and LW x FL (14.8) by Legault and Gruand (1981). However, the JX x FL crosses had a mean ovulation rate (20.6) substantially higher than that found in the other genetic groups. In LW, FL and LW x FL, embryo survival at 30 days of gestation has been found to be about 61%, 70% and 71%, respectively (Legault and Gruand, 1981). Embryo survival was markedly higher in JH x FL and MS x FL (90.9% and 89%, respectively), and comparable to LW and LF otherwise. Although the standard errors were very large to make meaningful inferences, it would appear that embryo survival is somewhat higher in Chinese breeds and crosses. Gueri (1982) reported data from the Shanghai Institute of Animal Husbandry and Veterinary Research, indicating that fetal survival in Mei Shan gilts was 97%, 89% and

Table 10. Estrus cycle variables, ovulation rate and embryo survival in Meishan (MS), Jia Xing (JX), and in crosses between MS, JX and Jin Hua (JH) with Large White (LW) or French Landrace (FL)

Genotype	Estrus cycle duration (d)	Estrus duration (hours)	Ovulation rate (No C.L.)	Embryo survival at 50 days (%)
MS	19.9	74.4	14.7	-
JX	19.0	59.1	15.9	-
MS x LW	19.8	69.8	13.8	68.7
MS x FL	20.5	72.7	13.9	89.0
JX x LW	19.3	69.0	16.8	48.6
JX x FL	20.0	57.7	20.6	59.3
JH x LW	21.3	60.0	14.8	77.8
JH x FL	20.8	65.8	14.2	90.9

Source: Rombauts, Mazzari and du Mesnil Du Bisson (1982)

84% in females of 4-5 months, 8-9 months, and 2 years of age, respectively.

Table 11. Body weight, growth rate and backfat in Meishan (MS), Jia Xing (JX) and Jin Hua (JH) females

Genotype	Body weight (kg)			Growth rate	End of test		
	Birth	21d	42d	0-150d, g/day	Age(d)	Wt.(kg)	Backfat(mm)
MS	.88	4.1	12.1	474	157	69.0	31.5
JX	.84	4.1	10.8	423	160	59.5	27.5
JH	.64	3.1	9.7	315	150	46.0	27.7

Source: Legault, Caritez, Gruand and Sellier (1982)

Growth. Variables related to growth in purebred Chinese gilts are presented in Table 11. Jin Hua animals were the lightest at birth, 21 and 42 days of age and had the lowest growth rate. At about five months of age, Mei Shan gilts were the heaviest with an average body weight of 69 kg at 157 days of age. Growing at 474 g/day, it would take 185-190 days for Meishan females to get to 100 kg of body weight.

Similar data were obtained for the F_1 crosses (Tables 12 and 13). Crossbred females and barrows were heavy at birth, grew rapidly (544-630 g/day) and reached slaughter weight (95-100 kg) at a mean age between 158 and 167 days, depending on the genotype of the animal. This performance was satisfactory and comparable with the data for U.S.A. experiment stations analyzed by Johnson (1980). JX-crossbreds were leaner than MS or JH crosses as indicated by backfat measurements in both gilts and barrows. Slaughter yield was somewhat low in the three groups (71-73%) but within the range encompassed by the variability among Western breeds. Carcass quality was judged mediocre by EEC standards because the proportion of lean cuts (ham and loin) was low relative to "non-lean" cuts. Water retention capacity and color were good, particularly in JH-crosses.

Growth and carcass quality in 1/4 Chinese-3/4 European pigs

A group of F_1 females were mated to Belgian Landrace (BL) boars to produce

Table 12. Growth statistics in Meishan (MS), Jia Xing (JX) and Jin Hua (JH) crossbred gilts

Variable	Genotype		
	MS-crosses	JX-crosses	JH-crosses
Birth weight (kg)	1.46	1.37	1.46
21-day weight (kg)	6.68	6.01	6.41
End of test age (d)	136	142	136
End of test wt. (kg)	84.2	84.9	75.6
Growth rate, birth-end of test (g/day)	610	586	544
Growth rate, 30-85 kg (g/day)	774	708	765
Backfat at 85 kg (mm)	26.3	24.5	27.7

Source: Legault, Caritez, Gruand, Sellier (1982)

Table 13. Growth and slaughter data in Meishan (MS), Jia Xing (JX) and Jin Hua (JH) crossbred barrows

Variable	Genotype		
	MS-crosses	JX-crosses	JH-crosses
Birth wt. (kg)	1.46	1.37	1.44
21-day wt. (kg)	6.22	6.19	6.05
42-day wt. (kg)	15.30	14.40	14.80
Slaughter age (d)	158	156	167
Slaughter wt. (kg)	100.00	99.60	95.30
Growth rate, birth-slaughter (g/day)	625	630	564
Yield without head (%)	71.3	71.4	73.0
Carcass backfat (mm)	38.1	34.7	41.2
% Ham and loin	46.9	46.9	46.4
Reflectance	423	431	377
Water retention (seconds)	144	136	167
pH_2	5.5	5.5	5.5

Source: Legault, Caritez, Gruand, Sellier (1982)

1/4 Chinese animals. These were compared to BLX(LWxFL) pigs used as controls. The Belgian Landrace is a heavy muscled, well conformed animal, widely used in France and Belgium as a terminal sire breed. Data related to this experiment are presented in Table 14. Compared to the controls, slaughter age was advanced in MS crosses but retarded in $\frac{1}{4}$ JX and $\frac{1}{4}$ JH animals. However, growth rate and feed efficiency were lower in $\frac{1}{4}$ Chinese animals. In general, differences in yield were not very marked, but ham and loin weight and ham and loin as a % of the carcass were lower in $\frac{1}{4}$ Chinese animals. Subcutaneous fat and kidney fat (not presented in the table) were higher in absolute value, and relative to carcass weight in $\frac{1}{4}$ Chinese pigs. Reflectance, water retention capacity and pH were better, in general, in $\frac{1}{4}$ Chinese carcasses When carcasses were graded according to the EEC commercial scale (I: best, IV: worst) the distribution (in % within genotype) was the following:

EEC Class	Control	¼ Mei Shan	¼ Jia Xing	¼ Jin Hua
I	22	-	-	-
II	30	26	25	27
III	48	49	41	35
IV	-	34	23	38

From a commercial viewpoint, ¼ Chinese pigs were at a disadvantage: finishing cost between 30-100 kg was higher and sale price at 100 kg was lower, primarily because of conformation.

Table 14. Growth and slaughter data in 25% Chinese (Mei Shan, Jia Xing or Jin Hua), 50% Belgian Landrace, and 25% French Landrace or Large White pigs

	Genotype			
Variable	Control[a]	¼ Mei Shan	¼ Jia Xing	¼ Jin Hua
Growth rate, 30-100 kg (g/d)	740	697	653	637
Feed/gain, 30-85 kg	3.04	3.30	3.35	3.32
Slaughter age (d)	183	174	194	200
Yield (%)	75.8	74.4	75.0	76.7
Carcass length (cm)	97.9	98.1	99.0	95.8
Ham wt. (kg)	9.2	8.7	8.6	8.7
Loin wt. (kg)	11.9	10.8	11.0	11.5
% Ham and loin	57.0	53.5	53.4	53.6
Reflectance	433	427	394	402
Water retention (seconds)	62	84	87	75
pH Ham	5.4	5.6	5.4	5.3

[a] ½ Belgian Landrace: ¼ French Landrace: ¼ Large White

Source: Legault, Caritez, Gruand and Sellier (1982)

IMPLICATIONS FOR THE U.S. SWINE INDUSTRY

The French results have to be interpreted cautiously due to their preliminary nature and, more fundamentally, because the limited size of the samples of Chinese breeds introduced precludes meaningful extrapolation. However, experimental data were in agreement with what was expected from the literature and from yield verifications (Epstein, 1969; Legault, 1978; Gianola, Rundquist and Thompson, 1980). Age at puberty both in purebred and in F_1 females was considerably lower than for American or European breeds (3-4 months vs. 6-7 months). In principle, this would permit mating at an earlier age, thus reducing generation interval by an important factor, and increasing the number of pigs produced per female per year of presence in the breeding herd. In addition, Mei Shan purebreds and crossbreds, and Jia Xing crossbreds were considerably more prolific than Large White and French Landrace females. This superiority is reflected in an increased estimated numerical productivity of the order of 5-8 extra pigs weaned per sow per year. Concomitantly, purebred or crossed Chinese sows nursing a litter weight equivalent to that nursed by LW or FL females, consumed 30-35 kg less of concentrate ration per litter nursed or 120-180 kg less on a per sow per year basis.

Numerical productivity, defined as the number of pigs weaned per sow per year

is a critical factor affecting physical and economic efficiency in a swine herd. At the national level, a smaller number of breeding females would be needed to produce a given pig output. For example (Nielsen, 1980), the number of sows needed to produce 1000 pigs decreased from 83 to 42 as numerical productivity increased from 12 to 24 pigs per sow per year. While variable costs may increase somewhat as numerical productivity increases, fixed costs would be spread over a larger number of animals. Numerical productivity of a sow per year of presence in the breeding herd can be expressed (Legault, 1978) as

$$P = \frac{N \times A \times (1-M) \times 365}{i_1 + (G+L+I)(N-1)+i_2} \qquad [1]$$

where

N = number of litters weaned per sow culled;
A = number of pigs born alive per litter;
M = mortality rate between birth and weaning;
G = gestation length;
L = duration of lactation period;
I = interval between weaning and conception;
i_1 = interval between 150 days of age and first farrowing, and
i_2 = interval between last farrowing and culling.

Consider the terms in the denominator of the above formula. In Legault's paper, i_1 was defined as the interval between 200 days of age and first farrowing. This does not apply to some Chinese breeds or crosses which could be, at least in principle, be bred at 150 days of age if not earlier. It is reasonable to regard G and L as constants at 115 and 30 days, respectively. The interval between weaning and conception, I, generally varies between 5 and 10 days, although it is technically feasible to modify this range to -8 to 10 days. For example, sows can be induced into estrus during lactation by limited nursing techniques (Thompson, Hanford and Jensen, 1981). Likewise, some Chinese breeds like the Jia Xing have a propensity to exhibit fertile estrus during lactation. The interval i_2 can be assumed to be 50 days. In the numerator, N can vary between 1 and 7 depending on the age structure of the herd. The variable A is strongly breed dependent, with herd averages ranging between 8 and 12 in American and European stocks, and between 9 and 20 in breeds found in the People's Republic of China. Pre-weaning mortality rate, M, varies between 5 and 30%.

Johnson (1980) summarized data from experimental stations cooperating in the Regional Research Project "Genetic Improvement of Efficiency in the Production of Quality Pork". The data come from well managed experimental herds and can be used to obtain an estimate, perhaps biased upwardly, of numerical productivity in the United States. Summary statistics are presented in Table 15. Taking first farrowing at 360 days of age, i_1 = 360-220 = 140, an interval weaning-conception of 7 days and i_2 = 50 days, with 3 litters produced per female, the following estimates of numerical productivity are obtained:

1. Purebred dams with purebred litters: $P_1 = 15.43$
2. Purebred dams with crossbred litters: $P_2 = 16.97$
3. Crossbred dams: $P_3 = 18.03$

However, if i_1 is defined as the interval between 150 days of age and first farrowing, numerical productivity changes to

1. Purebred dams with purebred litters — $P_1 = 13.51$
2. Purebred dams with crossbred litters — $P_2 = 14.87$
3. Crossbred dams — $P_3 = 15.79$

Table 15. Summary of litter size and preweaning mortality in U.S. experiment stations

System	Variable: No. live pigs at birth	Variable: Preweaning Mortality Rate[a]
1. Purebred dams with purebred litters	9.75	28.9%
2. Purebred dams with crossbred litters	9.85	22.5%
3. Crossbred dams	10.23	20.8%

[a]These are overestimates of mortality rate with a 30-day weaning period, as most experiments analyzed in the report dealt with weanings at 42 or 56 days.

Source: Johnson (1980)

The above are very likely underestimates of true sow productivity as Johnson's statistics came, in most part, from gilt populations. However, they provide a point of departure to analyze the effect of changing, simultaneously, factors affecting numerical productivity as defined in equation [1].

What is the scope for increasing numerical productivity, and which are the critical variables? The effects of varying litter size at birth from 10 to 13 pigs, mortality rate from 20 to 5%, the interval i_1 between 110 and 210 days, and the interval between weaning and conception from 10 to 2 days are illustrated in Table 16 for each of two "systems". These systems are 2 or 3 litters produced per female (N=2, N=3) prior to removal from the breeding herd. The results presented in Table 16 indicate the following: at about 20% mortality rate, numerical productivity can be increased from 14-15 to 24-25 pigs per sow per year of presence in the breeding herd, by increasing litter size at birth from 10 to 13 pigs and reducing age at first farrowing from 360 to 260 days of age. Likewise, at 5% preweaning mortality rate, numerical productivity can be increased from 16-18 to about 30 pigs per sow per year if litter size and age at farrowing change as indicated above. There is limited scope for increasing sow productivity through reducing the interval between weaning and conception from 10 to 2 days; at best, this would bring about an increase in numerical productivity of about 1 pig. A first farrowing at 260 days and a litter size of 13 pigs is possible in F_1 Chinese x Western females as suggested by the literature and by the experiments in France.

Animals with $\frac{1}{4}$ of Chinese breeding grew well and reached slaughter weight at an acceptable age. However, the economy of gain, dressing percentage, carcass composition and carcass grade were not competititve with $\frac{1}{2}$ Belgian Landrace:

Table 16. Effects of number of pigs born alive (A), mortality rate (M), interval between 150 days of age and first farrowing (i_1) and interval between weaning and conception (I) on number of pigs produced per sow per year of presence in the breeding herd in two production systems

	2 Litters per female (N=2)							
	M=.20				M=.05			
	i_1=210		i_1 =110		i_1=210		i_1=110	
A	I=10	I=2	I=10	I=2	I=10	I=2	I=10	I=2
10	14.1	14.3	18.5	19.0	16.7	17.0	22.0	22.6
11	15.5	15.8	20.4	20.9	18.4	18.7	24.2	24.8
12	16.9	17.2	22.2	22.8	20.1	20.4	26.4	27.1
13	18.3	18.7	24.1	24.7	21.7	22.2	28.6	29.4
	3 litters per female (N=3)							
	M=.20				M=.05			
	i_1=210		i_1=110		i_1=210		i_1=110	
A	I=10	I=2	I=10	I=2	I=10	I=2	I=10	I=2
10	15.4	15.8	18.6	19.3	18.3	18.8	22.1	22.9
11	16.9	17.4	20.5	21.2	20.1	20.7	24.3	25.2
12	18.4	18.9	22.3	23.1	21.9	22.5	26.6	27.5
13	20.0	20.6	24.2	25.1	23.7	24.4	28.8	29.8

¼ Large White:¼ French Landrace pigs. In fact, the reduction in the production cost of a pig weaned by an F_1 female was neutralized by the lower food conversion of the ¼ Chinese pig and by the penalty received by their carcasses in the EEC system (Legault, Caritez, Gruand and Sellier, 1982). Nevertheless, these results are encouraging. It would be possible to obtain better ¼ Chinese products by exerting intense selection of the F_1 gilts to enter the breeding herd or by utilizing elite LW or FL boars to produce the crossbred female. Further, it is of interest to consider the production of a 1/8 Chinese animal and this is currently being investigated in France. The economic interest of this alternative will depend on the productivity of the 25% Chinese females. An additional possibility would be the development of Chinese-Western "composite" breeds. For example, "composite animals" could be mated to Yorkshire females to produce an F_1. This, in turn, could be mated to a highly muscular breed (e.g., Pietrain) to increase the lean content and conformation of the progeny, all of which would be slaughtered.

In summary, Chinese genes offer great promise regarding the improvement of numerical productivity in swine. At the same time, they introduce problems in the areas of economy and composition of gain. Fortunately, selection for feed efficiency and lean content have been proven to be effective in swine so it would be possible, at least in theory, to move Chinese populations to satisfactory levels for these traits.

There have been additional introductions of Chinese swine into Europe following the French importation. A large Mei Shan herd has been established in Hungary and a French swine breeding company has also imported Mei Shan animals. The Animal Breeding Research Organization and the Meat and Livestock Commission of

the United Kingdom are considering an importation of embryos (King, Steane and Webb, 1982, personal communication).

The University of Illinois, supported in part by the Illinois Pork Producers Association, sent a team to China in 1980 to investigate sources of swine germplasm. The team from the Department of Animal Science recommended an importation of semen from the Mei Shan, Min Zhu, Fen Jing, Ning Xiang and Da Hua Bai breeds, as the introduction of live pigs was apparently infeasible. A request was sent to the Animal Plant and Health Inspection Service (APHIS) in 1980, which was denied. In 1981, the Office of International Cooperation and Development, U.S.D.A., decided to support the Illinois effort through coordination with the National Programs Staff of the U.S.D.A. In the meantime, APHIS has been developing regulations for the import of semen and/or swine from China into the United States. Apparently (Wong, 1982, personal communication) these regulations have been sent to the Chinese government for study and consideration. We hope to be able to show you Mei Shan and Min Zhu animals, or at least crossbreds, at our Swine Research Center in future issues of the University of Illinois Pork Industry Conference.

Literature Cited

Christensen, . . 1980. Cited by Vangen, O. 1981. Problems and possibilities for selection for fecundity in multiparous species. Pig News and Inf. 2:257-263.

Cunningham, P. J., England, M. E., Young, L. D. and Zimmerman, D. R. 1979. Selection for ovulation rate in swine: correlated response in litter size and weight. J. Anim. Sci. 48:409-516.

Epstein, H. 1969. Domestic Animals in China. Commonwealth Agricultural Bureaux, Farnham Royal, Slough, United Kingdom.

Fang, B. Q., Hu, J. F., Huang, D. X., Pei, D. M., Tan, J. L. and Zhang, P. S. 1980. Studies on reproductive traits in Er Hua Lian pigs. 1. The development of reproductive organs of boars and sows. Anim. Husb. and Vet. Med. (China) 12:1-6.

Fang, B. Q, Hu, J. F., Huang, D. X., Pei, D. M., Tan, J. L. and Zhang, P. S. 1980. Studies on reproductive traits in Er Hua Lian pigs. 2. The development of sexual behaviour and reproductive functions of boars. Anim. Husb. and Vet. Med. (China) 12:4-8.

Fang, B. Q, Hu, J. F., Huang, D. X., Pei, D. M., Tan, J. L. and Zhang, P. S. 1980. Studies on reproductive traits in Er Hua Lian pigs. 3. The development of sexual behaviour and reproductive functions of gilts. Anim. Husb. and Vet. Med. (China) 12:12-15.

Fang, B. Q, Hu, J. F., Huang, D. X., Pei, D. M., Tan, J. L. and Zhang, P. S. 1980. Studies on reproductive traits in Er Hua Lian pigs. 4. Preliminary analysis and observations on gestation period and parturition in sows. Animal Husb. and Vet. Med. (China) 13:9-12.

Gianola, D., Rundquist, J. F. and Thompson, L. H. 1980. Travel notes on swine production in the People's Republic of China. Dept. Anim. Sci., U. of Illinois, Urbana.

Guerin, C. 1982. Premieres experimentations sur le porc Chinois en France: resultats d'une experience de croisement Chinois x Pietrain. I.N.R.A., Station de Génétique Quantitative et Appliquée, Jouy-en-Josas, France.

Johnson, R. K. 1980. Heterosis and breed effects in swine. North Central Regional Publication No. 262. Agr. Exp. Station, U. of Nebraska, Lincoln.

Legault, C. 1970. Étude statistique et génétique des performances d'elevage des truies de race Large White. Ann. Genet. Sel. Anim. 2:209-227.

Legault, C. 1978. Analyse des composantes de la productivité numérique des truies. Ann. Zootech. 27:457-470.

Legault, C., Gruand, J. 1981. Effets additifs et non additifs des genes sur la précocité sexuelle, le taux d'ovulation et la mortalité embryonnaire chez la jeune truie. J. Rec. Porc. en France 13:247-254.

Legault, C. and Caritez, J. C. 1982. Premier bilan de l'experimentation sur le porc Chinois en France. 1. Performances de reproduction en race pure et en croisement. J. Rech. Porc. en France 14:127-136.

Legault, C. 1978. Particularités zootechniques des porcs élevés en Republique Populaire de Chine. Bull. Tech. Inf. 327:115-125.

Legault, C., Caritez, J. C., Gruand, J. and Sellier, P. 1982. Premier bilan l'experimentation sur le porc Chinois en France. 3. Croissance et composition corporelle en croisement a deux ou trois voies. J. Rech. Porc. en France 14:143-150.

Nielsen, H. E. 1980. Maximizing sow productivity. University of Illiois Pork Industry Conference. 1980: 11-17. Dept. Anim. Sci., U. of Illinois, Urbana.

Ollivier, L. 1973. Five generations of selection for increasing litter size in swine. Genetics 72:202 (Abstr.)

Ollivier, L. and Bolet, G. 1981. La sélection sur la prolificité chez le porc: Résultats d'une experience de sélection sur dix générations. J. Rech. Porc. en France 11:261-268.

Phillips, R. and Hsu, T. Y. 1944. Chinese swine and their performance compared with modern and crosses between Chinese and modern breeds. J. Hered. 35:365-379.

Rombauts, P., Mazzari, G. and du Mesnil Du Buisson. 1982. Premier bilan de l'experimentation sur le porc Chinois en France. 2. Estimation de composantes de la prolificite:taux d'ovulation et survie foetale. J. Rech. Porc. en France 14:137-142.

Rutledge, J. J. 1980. Fraternity size and swine reproduction. 1. Effect on fecundity of gilts. J. Anim. Sci. 51:868-870.

Strang, G. S. and King, J. W. B. 1970. Litter productivity in Large White pigs. 2. Heritability and repeatability estimates. Anim. Prod. 12:235-243.

Strang, G. S. and Smith, C. 1979. A note on the heritability of litter traits in pigs. Anim. Prod. 28:403-406.

Thompson, L. H., Hanford, K. J. and Jensen, A. H. 1981. Estrus and fertility in lactating sows and piglet performance as influenced by limited nursing. J. Anim. Sci. 53:1419-1423.

Wu, J. S. and Zhang, W. C. 1982. Genetic analysis of some Chinese breeds as a resource for world hog improvement. Proc. 2nd Cong. Genet. Appl. Liv. Prod. VIII:593-600. Ed. Garsi., Madrid.

Zhang, W. C., Wu, J. S. and Rempel, W. E. 1982. Performance characteristics of prolific breeds of pigs in China. Mimeo., Dept. of Animal Science, U. of Minnesota, St. Paul.

Zheng, Z. G. 1981. The pig resources of China: a perspective. Natural Resources (China) 2:65-71.

Social Interaction of the Herdsman and the Breeding Herd

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It is well known that some people are consistently better than others at handling animals. Good handling of animals appears to be a skill acquired by experience. For instance, it seems that a good handler has a wide experience of the behaviour of animals under various handling situations; has the ability to anticipate the reactions of the animal and thus, if necessary, take the appropriate action; makes minimum use of fearful stimuli (eg. shouting and waving); avoids punishing animals; uses slow and deliberate movements; and talks quietly and perhaps strokes the animals. However, part of good handling of animals may have an innate basis. It is often suggested that a "person who is good with animals" has an empathy or sympathetic attachment for animals and this would be very important if a person's intentions and emotions are communicated (perhaps via behavioural or olfactory (smell) cues) to the animal.

While some of this knowledge is commonsense, research is required to identify and clarify the vital components of good stockmanship to the inexperienced handler and to reinforce the importance of these components to the experienced handler. The objective of this paper is to demonstrate the importance of the development of a good relationship between the handler and his/her animals, achieved through sufficient positive handling, on the productivity and welfare status of the animals. In order to do so, three recent studies will be described.

Our interest in this area was stimulated by a study conducted in the Netherlands in 1979 (Hemsworth, Brand and Willems, 1981). The study was conducted on 12 one-man farms of similar design, genetic stock, locality, feeding and management advice. Thus, the farms had similar inputs apart from the stockman. The objective of this study was to examine the association between the man-animal relationship, indicated by the behavioural response of the pigs to the presence of humans, and the reproductive performance of the farm.

To quantify the behavioural response of pigs to the presence of humans at each farm, pregnant sows were observed in two behavioural tests: In the first test the withdrawal response of sows to the approaching experimenter was observed while in the second the approach behaviour of the sow to the stationary experimenter was observed. A significant association was found between the behavioural response of sows to the experimenter and the reproductive performance of the farm: Sows displayed an increased withdrawal response to the approaching experimenter and a

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decreased approach response to the stationary experimenter (both indicative of increased fear in the presence of the experimenter) at farms in which the average number of piglets born per sow per year was low. The results suggest that at farms where sows were fearful in the presence of humans, reproductive performance was lower than at farms where sows were confident in the presence of humans.

Therefore, identification of the factors which influence the relationship between the stockperson and his/her stock may have significant implication to the pig industry. Two factors which may influence this relationship are the quality and quantity of handling by the stockperson. A second study, conducted at the Animal Research Institute, Werribee, examined the influence of quality of handling by humans on behaviour, growth and plasma corticosteroids (as a measure of stress) of the young gilt (Hemsworth, Barnett and Hansen, 1981).

Two handling treatments, pleasant and unpleasant, were imposed three times per week for 2 minutes in duration from 11 to 22 weeks of age. Whenever the gilt approached the experimenter during each 2-minute period, the gilt was either gently stroked (pleasant treatment) or slapped or briefly shocked with a battery operated prodder (unpleasant treatment). The gilts were fed ad libitum from 11 to 22 weeks of age and while there was no difference between the two handling treatments in feed consumed, the gilts in the unpleasant handling treatment had a significantly slower growth rate during this period (669 g/day compared with 709 g/day for gilts in the pleasant handling treatment. Feed conversion rate (kg of feed per kg of gain) was 3.8 and 3.5, respectively.) In addition, at 24 weeks of age the gilts in the unpleasant handling treatment had elevated corticosteroid levels "at rest" in isolation from humans. Gilts in the unpleasant handling treatment displayed a marked withdrawal or avoidance of the experimenter at 25 weeks of age. Therefore, these results suggest that regular unpleasant handling will result in a chronic stress response in the pig and that a consequence of this stress response is a depression in growth rate.

We have recently conducted a third study to examine the effects of both quality and quantity of handling by humans on behaviour, reproduction and corticosteroid levels of the gilt and boar (Hemsworth, Barnett and Hansen, unpublished data). Unpleasant and pleasant handling treatments, similar to those in the previous study, were imposed three times per week for 5 minutes in duration from 11 weeks of age for the entire study. In addition, a third treatment, involving little contact with humans apart from the routine husbandry practices, was also examined.

At 95 and 123 days of age pigs in the unpleasant handling treatment and, to a lesser extent, those in the control treatment displayed increased withdrawal or avoidance of the handler compared with pigs in the pleasant handling treatment.

Boars in the pleasant handling treatment attained a fully coordinated mating response at an earlier age and had larger testicles (length by width) at 160 days of age than boars in the unpleasant treatment (161 and 192 days, respectively and 63.3 and 53.2 cm^2, respectively). This difference in testicle size was no longer apparent at 238 days of age. There was a tendency for gilts in the unpleasant and control handling treatments to have a delayed puberty (221 and 230 days respectively compared with 210 days for the pleasant handling treatment). Thus, it appears that the unpleasant handling treatment may adversely affect the rate of sexual development of the boar and gilt.

All gilts were mated by fertile boars at their second oestrus. While there

were no differences in the sexual behaviour of the gilts, the pregnancy rate at 50 to 60 days post-mating for gilts in the unpleasant, control and pleasant handling treatments was 33.3% (3/9), 55.6% (5/9) and 87.5% (7/8), respectively. Hormone data indicate that pigs in the unpleasant handling treatment had a chronic elevation of corticosteroids "at rest" in isolation of humans (chronic stress response) and those in both the unpleasant and control handling treatments had a short term elevation of corticosteroids following exposure to humans (acute stress response). It seems likely that these responses around the time of mating may impair reproductive efficiency in the female pig.

Therefore, these three studies show that adverse handling by the stockperson can lead to physiological responses indicative of poor welfare status (a chronic stress response) with consequent effects on production (growth and reproduction).

In this area of handling by humans there are a number of other aspects that require examination. Even with good stockpersons, there may be certain stress situations for the stockperson in which an animal is more likely to be adversely handled. Such situations may include personal stress; attempting to move or handle animals with poor facilities or with insufficient time or labour; a disorganized work program; or time-consuming and monotonous activities that are ancillary to animal production (eg. cleaning pens). These situations obviously need to be identified and a behavioural study of the stockperson in his/her daily routine may be useful in identifying these stress situations. Another aspect that requires investigation is consistency of handling. The effects of inconsistent handling, involving both pleasant and unpleasant handling at irregular intervals, on corticosteroid levels of the pig may be similar or even worse than those of regular unpleasant handling where the animal may be able to cope to some extent through an anticipatory mechanism. This aspect has implication to large units where there may be rotation of the workforce or where there is a high turn-over of staff.

In conclusion, the three simple studies described clearly demonstrate the importance of the development of a good man/animal relationship in the commercial piggery. The stockpersons should aim to regularly handle their stock in a pleasant manner, avoiding adverse handling. Stockpersons can gain some idea of the quality of the relationship between them and the stock by observing the behavioural response of the stock to their gradual approach. If the pigs consistently display a rapid and vocal withdrawal to the stockperson's approach, then the man-animal relationship at the farm must be questioned.

LITERATURE CITED

Hemsworth, P. H., Barnett, J. L. and Hansen, C. (1981). The influence of handling by humans on the behaviour, growth and corticosteroids in the juvenile female pig. Horm. Behav. 15, 396-403.

Hemsworth, P. H., Brand, A. and Willem, P. J. (1981). The behavioural response of sows to the presence of human beings and their productivity. Livestock Prod. Sci. 8, 67-74.

How Does Liver Function Affect Reproduction?

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The liver is one of the largest organs in the body. About 1/3 of the blood pumped from the heart goes through the liver. In addition to its relatively important size it also has many important metabolic functions. Because the venous blood from the digestive tract goes directly to and through the liver before it enters the general circulation, everything that enters and is absorbed by the stomach or intestine must first pass the liver before it reaches the body. The liver also detoxifies most toxic materials whether they are part of the diet, the environment, or produced by the animal itself.

The liver usually metabolizes potentially toxic materials in such a way that renders them non-toxic. This metabolized toxin is more readily excreted in the bile and then into the digesta and finally the feces or into the urine. One of the important systems for detoxification is the mixed function oxidase system. The mixed function oxidases are enzymes that develop in the liver, head, lungs, kidneys and, to a significant extent, in the intestinal mucosa. This enzyme system is not one enzyme but many. Each enzyme may metabolize several compounds even though a high level of the enzyme was induced by only one compound.

The induction of high levels of mixed function oxidase can result from many causes; high protein diet, high levels of certain amino acids, barbiturates, steroid hormones, chlorinated hydrocarbons, certain gases, organic solvents, charcoal-broiled meat, insecticides, herbicides, cigarette smoke, heavy metals, anesthetics, alcohol and many other materials the liver views as a toxin. Steroid hormones such as estrogen, progesterone, testosterone and adrenal corticosteroids have a direct and major effect on reproduction. These steroids are metabolized by the mixed function oxidase system and the rate of metabolism is influenced by the inducing materials listed.

When the mixed function oxidase enzyme system is impaired, steroid metabolism is reduced and reproduction is impaired. On the other hand, an overstimulated mixed function oxidase enzyme system may also create problems. Two examples follow. Eagles, ospreys and pelicans eat fish. Some of these fish contain DDT. DDT induces a high level of mixed function oxidase in the liver. This high level of enzyme also rapidly metabolizes estrogens needed for mobilization of calcium for egg shells. When the system is overstimulated by DDT, the birds lay soft-shelled eggs threatening them with extinction. Women who take barbiturates in

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the form of tranquilizers, anti-epileptic drugs and anti-tuberculosis drugs find that birth control pills are ineffective. The mixed function oxidase system stimulated by the tranquilizer also rapidly metabolizes the steroids in the birth control pill abolishing the contraceptive effect. Even more dramatic is the observation that alcoholics universally suffer from severe hormone imbalances and male alcoholics are nearly always impotent.

How does this fit in with reproduction in livestock? One of the classic demonstrations of the effect of plane of nutrition is an increase in the number of ovulations from the effect of flushing. More twins in sheep and higher ovulation rate in pigs often result when the plane of nutrition is increased as least ten days before ovulation. An increased level of mixed function oxidase is associated with an increased plane of nutrition and especially with increased protein in the diet. A high plane of nutrition in pregnant animals is associated with greater embyronal losses. A high level of protein in the diet is associated with reduced levels of progesterone in pregnant pigs.

The level of steroid hormones in the body may be influenced by high levels of orally administered antibiotics. There are bacteria in the lower gut that undo the work the liver has done and, in fact, help put some of the excreted compounds back into the body in their original active form. This is termed entero-hepatic circulation. A compound may be ingested or produced in the body, travel to the liver, be removed and excreted in the bile, go to the large intestine, be freed by bacteria and re-enter the circulation. Thus, merely withdrawing a material from the diet is no assurance that it will disappear from the blood. One striking example of the effect of interfering with normal entero-hepatic circulation has been noted in the pregnant woman. A three-day course of a high level of an oral antibiotic effectively destroyed the bacteria in the intestine. Because the form of estrogens produced by the liver could not be absorbed until bacteria acted on it, little of the estrogens were reabsorbed. These estrogens then were excreted in the feces. As a consequence, the level of estrogens in the blood fell to half of pretreatment level. A single ingestion or injection of a steroid may persist for several days courtesy of the bacteria in the lower gut who free the steroid so it may be reabsorbed and recirculated over and over. The rate of food passage and gut sterilizing doses of antibiotics may have an influence on the circulating level of steroid hormones and, thus, influence reproduction.

Our preliminary work has given us several clues that may help in understanding just how the liver function might influence reproduction. The flushing effect may possibly be mimicked by compounds such as barbiturates or insecticides. If this proves to be true with repeated tests, then ovulation rate may be influenced by a variety of compounds introduced in the diet or applied externally. The scheduling of such treatments must be done to coincide with the anticipated growth of ovarian follicles. This means that producers that expect optimum reproduction may have to plan in advance for usual management procedures. The possible implications here are far-reaching and may involve careful planning of parasite control and careful monitoring of diet and the environment.

Ingested estrogen, progesterone and testosterone are metabolized to a significant extent by a healthy intestinal mucosa and completely by a functioning liver. There steroid hormones have a profound and direct effect on reproduction. The mixed function oxidase system is quite efficient and thorough. It can metabolize a level of steroid many fold greater than is present normally.

What are the prospects for putting to use information that may come from research in this area? The question of the underlying basis for the effect of certain diets on reproduction may be resolved. Perhaps flushing, arsanilic acid, alfalfa, antibiotic feeding, growth stimulants and parasite treatment have a common link as they influence reproduction. Many dietary components may have primarily a non-nutrient effect even though they are associated with an outcome ascribed to nutrition. A greater understanding may lead to a variety of diets and management procedures tailored to the reproductive state. Some of the dietary components and treatments may be directed solely to influencing mixed function oxidase or similar enzyme systems.

REFERENCES

Adlercreutz, H. and F. Martin. 1980. Biliary excretion and intestinal metabolism of progesterone and estrogens in man. Journal of Steroid Biochemistry 13: 231-244.

Adlercreutz, H., F. Martin, P. Järvenpää and T. Fotsis. 1979. Steroid absorption and enterohepatic recycling. Contraception 20:201-223.

Anderson, K.E., A.H. Conney and A. Kappas. 1982. Nutritional influences on chemical biotransformations in humans. Nutrition Reviews 40:161-171.

Anderson, R.A. Jr., B.R. Willis, C. Oswald, J.M. Reddy, S.A. Beyler and L.J.D. Zaneveld. 1980. Hormonal imbalance and alterations in testicular morphology induced by chronic ingestion of ethanol. Biochemical Pharmacology 29:1409-1919.

Bolt, H.M., H. Kappas and M. Bolt. 1975. Effect of Rifampicin treatment on the metabolism of oestradiol and 17α ethinyloestradiol by human liver microsomes. European Journal of Clinical Pharmacology 8:301-307.

Dyck, G.W., W.M. Palmer and S. Simaraks. 1980. Progesterone and luteinizing hormone concentration in serum of pregnant gilts on different levels of feed consumption. Canadian Journal of Animal Science 60:877-884.

Hartiala, K. 1973. Metabolism of hormones, drugs and other substances by the gut. Physiological Reviews 53:496-534.

Jordan, E.R. and L.V. Swanson. 1979. Serum progesterone and luteinizing hormone in dairy cattle fed varying levels of crude protein. Journal of Animal Science 48:1154-1158.

Thurman, R.G. and F.K. Kauffman. 1980. Factors regulating drug metabolism in intact hepatocytes. Pharmacological Reviews 31:229-251.

A Primer on Genetic Engineering

WALTER L. HURLEY

INTRODUCTION

Genetic engineering, biotechnology, recombinant DNA--all these words are generating considerable excitement at present. To the biologist they offer exciting new tools and approaches to study living systems. To the veterinarian they mean the potential for new drugs in large, inexpensive quantities, or for cures to genetic diseases. To the agricultural producer, they may mean animals or crops that grow faster and more efficiently, and are healthier than those now available. Most of these images are futuristic, as the full potential of genetic engineering will not be realized for years to come.

Engineering is defined as a science by which properties of matter and sources of energy in nature are made useful to man in structures, machines, and products. Genetic engineering then is the use of information contained in genes existing in nature to manipulate life forms to do something we want them to do. We do not have the ability to create life as the popular press sometimes implies. We are dealing with a technology; that is, we have the techniques by which we can genetically alter existing life forms. As it turns out our innovative techniques are very similar to those used in nature as part of the evolutionary process. Mother Nature created them first and we are only able to borrow her ideas. There really is nothing new under the sun.

I believe that to fully appreciate genetic engineering and to be able to realistically assess what it can do, we must first have an understanding of the fundamentals of one of its most critical parts--recombinant DNA technology. First we will cover the basics of recombinant DNA technology and then expand on those basics with a specific example of genetic engineering of bacteria to synthesize a protein useful to livestock producers. Finally, we will look at some potential areas that genetically engineered bacteria may prove useful to animal producers. Both positive potential of genetic engineering and some problem areas will be discussed.

Review of DNA

Before beginning the discussion of recombinant DNA technology, a brief review of DNA and protein synthesis is advisable. This review is found in Figures 1, 2 and 3. DNA is a polymer composed of four types of units or bases: adenine (A),

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a. b.

...AGCTTGCCA...

...TCGAACGGT...

Figure 1. a) Bases of DNA are linked together in a strand. DNA is double-stranded due to base pairing of A's and T's, and of C's and G's. b) The double-stranded nature of DNA causes it to form the well known double helix.

guanine (G), thymine (T), and cytosine (C). Each base is attached to a sugar ring structure and these sugar ring structures are linked together in a strand to form the polymer. In nature, DNA is double-stranded due to pairing of bases on one strand with those on the other. The A bases only pair with T's and G's only with C's. This base pairing of the two DNA strands is shown in Figure 1a. The double-stranded nature of DNA is what gives it the famous double helix structure (1b). For the replication of DNA, the strands come apart and a new strand is synthesized on each old strand, using the old strands as templates to maintain the base-paired sequence (Figure 2).

Protein synthesis (Figure 3) is the result of an exquisite process where the code (sequence of bases on a DNA strand) of the gene's DNA (template) is used to synthesize (transcribe) an RNA molecule which is complementary to that gene sequence. The structure of RNA is similar to that of DNA, however RNA is not double-stranded. The coded sequence information of the RNA molecule is then translated to an amino acid sequence of a protein by ribosomes. The sequence of amino acids in a protein chain is what determines that protein's activity, whether it be an enzyme, a hormone or a structural protein.

Until recently we have assumed that DNA is a static molecule. That is, once a long sequence strand is made it does not change much. We now know, from using recombinant DNA technology, that DNA in nature is very dynamic. Pieces move around from one place on the DNA to another, recombinant events occur often in nature, pieces of DNA are dropped so that other pieces may come closer together. These are the same approaches we use in recombinant DNA techniques, and they have been happening all along in nature.

Recombinant DNA Technology

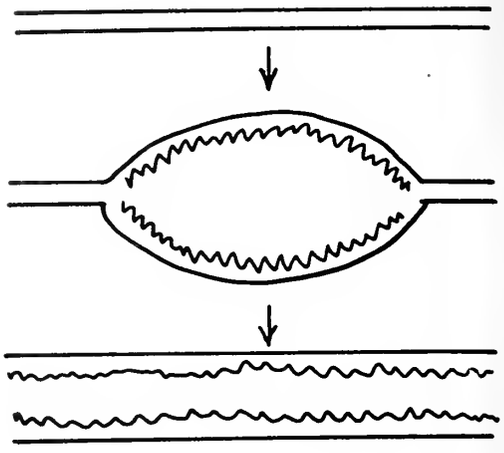
Recombinant DNA technology is exactly that--a technology. This technology (or methodology) can be defined as the techniques of joining DNA molecules together *in vitro* (outside of a living cell) and introducing them into living cells where they can replicate. The purpose is to separate a DNA molecule of particular interest from other DNA molecules, and to use the DNA replicating systems that nature has devised to produce enough of this DNA molecule to use and study. In this technology several things are needed to be successful:

1. A DNA vehicle (called a vector) which can replicate in living cells when it has a piece of foreign DNA inserted into it.
2. A DNA molecule to be replicated (the one we are interested in; called the insert).
3. A method of specifically joining insert and vector together to make the recombinant molecule.

Figure 2. DNA REPLICATION. The specificity of base pairing (only A to T and G to C) gives DNA some simple but unique properties for maintaining the nucleotide base sequence. When DNA is replicated (cell division) the strands come apart and a new strand is synthesized from both original template strands. From the original molecule there are now two identical double-stranded molecules, each with one of the original strands and one new strand.

Original DNA

Newly synthesized DNA



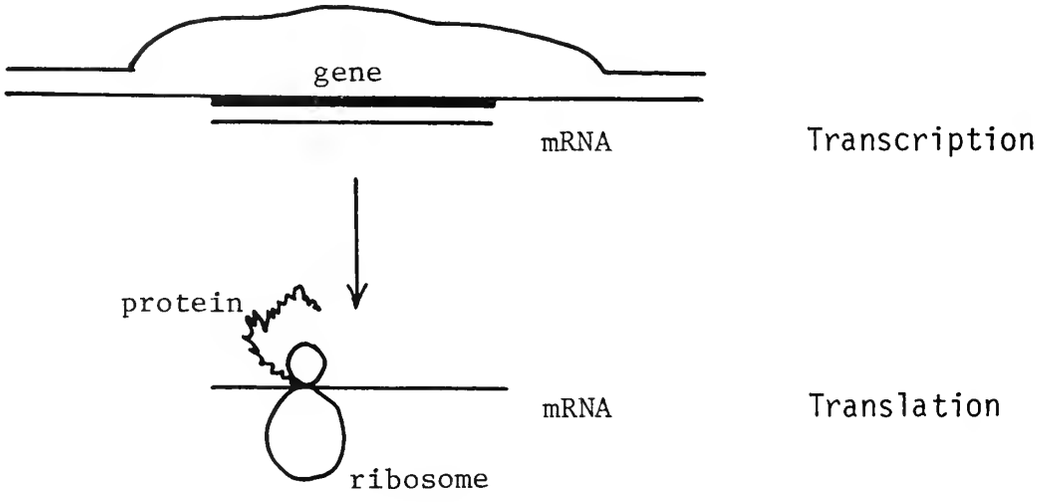


Figure 3. PROTEIN SYNTHESIS. Figure 3 is a very simplistic diagram of protein synthesis. The DNA strands of a gene come apart and a messenger RNA (chemically similar to DNA except the base U replaces T) is synthesized using one specific strand as a template (transcription) and is complementary to that strand. This mRNA contains the nucleotide sequence (code) necessary for synthesis of the protein. The mRNA then moves to structures called ribosomes (made of proteins and ribosomal RNA) which are the actual protein synthesizing machines. The mRNA is read through the ribosome and a protein is synthesized using the nucleotide sequence as a code to determine which amino acid goes where in the protein chain (translation). Each amino acid is coded for by three consecutive nucleotide bases called a codon. If the codon GCU is for alanine, AUG for methionine, and AAG for lysine, then the sequence GCUAUGAAG in a mRNA would translate into the peptide alanine-methionine-lysine. That RNA sequence is of course complementary to the gene (DNA) it was synthesized from -- CGATACTTC (remember T's are in DNA but U's are in RNA, so both T's and U's base pair with A). This is the genetic code and how it is first transcribed to a complementary messenger RNA sequence, which in turn is translated to an appropriate protein amino acid sequence.

4. A method of introducing this recombinant DNA into a host organism (bacteria, animal or plant cell) in which it can replicate (called DNA transformation).
5. A means of screening for those cells which contain the desired recombinant DNA molecule.

Each of these components is essential to the whole technology and each requires a number of methods or procedures. Generally, if a piece of insert DNA, such as the gene for a hormone, is put into a cell by itself, the DNA will likely be degraded. The DNA replicative machinery of a cell is geared toward taking care of its own DNA. Unless given specific instructions the cell does not know what to do with the foreign DNA. We do not know how to tell the cell what to do with the foreign DNA, so we rely on ways that nature has already devised. Viruses and plasmids are DNAs that can get into cells and be replicated without actually being part of the cell's chromosomal DNA. Viruses are mainly DNA (or RNA) containing some genetic code that, once inside the cell, can redirect the cellular machinery to do what the virus wants (primarily to be replicated), rather than what the cell wants. Plasmids are fairly small circular double-stranded DNAs that inhabit microorganisms. To continue existing, plasmids must bestow some particular selective advantage to that bacteria. Often that advantage comes from the existence in the plasmid DNA of one or more genes coding for proteins able to degrade antibiotics. For instance, plasmid pBR322 (commonly used in recombinant DNA work, Figure 4) has genes which give its host cell resistance to the antibiotics tetracycline and ampicillin. Plasmids are apparently the major cause behind increases in antibiotic resistant bacterial strains. The bacteria themselves have not changed, but those bacteria in the population which contain plasmids are selected while those without plasmid are killed by antibiotics. Plasmid DNA is replicated along with cellular DNA. Antibiotic resistance can be used as part of the screening process described below. Many viruses and plasmids, as well as host bacteria used in recombinant DNA research have been genetically altered to maintain properties of interest to us, but to minimize the potential environmental hazards.

The DNA molecule we are interested in is called an "insert" molecule. It may be the gene for a hormone, an enzyme or just some particular segment of DNA we have special interest in. Many times this insert DNA is not a single DNA sequence, but a heterogenous mixture of many DNAs. In that case, screening for the one particular sequence of interest is of great importance. An example of this is the initial identification of a DNA corresponding to a specific protein. Often messenger RNA (RNA coding for proteins) is extracted from a tissue and double-stranded DNAs complementary (cDNA) to the RNA are synthesized using purified enzymes with the RNA as a template. The goal then is to pick which particular cDNA is the one of interest out of all the thousands of other sequences represented in the original RNA by the cDNA mixture.

Once we have a suitable vector and the insert DNA (either a single sequence or a mixture of DNAs), we need a means of joining vector and insert together. Again we look to existing approaches in living cells. Many bacteria contain enzymes (restriction endonucleases) which will cut double-stranded DNA at points containing a specific nucleotide sequence. As shown in Figure 5, the enzyme Bam Hl recognizes and cuts DNA at the sequence GGATCC, to give what are called overhanging ends. The sequence of one overhanging end -GATC is exactly complementary to the sequence of the other overhanging end CTAG-. So, these two pieces of DNA can easily and specifically be put back together to re-form the Bam Hl site. Also, it does not matter what two pieces of DNA are put together as long

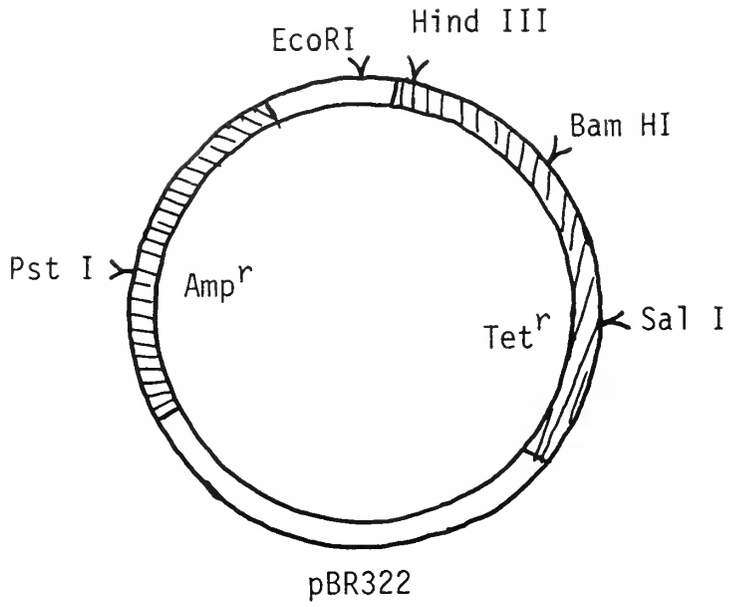


Figure 4. PLASMID VECTOR pBR322. A circular double-stranded DNA which inhabits bacterial cells. Plasmid pBR322 has genes coding for proteins which give its host bacterial cell resistance to the antibiotics tetracycline (Tet^r) and ampicillin (Amp^r). Restriction enzyme sites are shown (see Figure 5).

BAM HI

. . . GGATCC . . .

. . . CCTAGG . . .

. . . G GATCC . . .

. . . CCTAG G . . .

Figure 5. RESTRICTION ENDONUCLEASES. Enzymes which cleave double-stranded DNA at a site determined by a specific short sequence. Here the enzyme Bam HI recognizes a specific 6 base pair sequence, cutting the DNA to give gragments with the overhanging ends shown. See text for more discussion.

as they have this Bam Hl cut overhanging ends. In other words, if the desired vector has a Bam Hl site in an appropriate place, and the insert DNA has Bam Hl ends, then these molecules can be very specifically joined to give a recombinant molecule. Another enzyme, called DNA ligase is used to covalently link these "sticky" ends. There are many restriction endonucleases available, giving us a whole battery of tools to cut DNA specifically. It will be noted that by using this technique the final recombinant molecule has been designed such that we can still specifically cut the insert back out of the vector with the same restriction enzyme.

DNA transformation is the means by which we get the recombinant molecule into some living cell. This can be any type of cell--bacterial, plant or animal. In the case of nucleated cells (plants and animals) we must also worry about getting the DNA into the cell's nucleus where it can be replicated or transcribed. Generally, getting DNA into the cell literally involves mixing DNA with the cells. How cells take up DNA is not well understood, but only a small proportion of them do take it up. Again we must screen for those cells that took up DNA, and exclude those cells which did not.

Screening is one of the more tedious aspects of recombinant DNA technology (or genetic engineering). We must screen at several different levels depending on our particular goal. Firstly, cells which have taken-up DNA must be screened from those that did not. This can be done by antibiotic resistance of plasmids in bacteria, or presence of a particular necessary enzyme in other cells. Next, cells containing the particular DNA of interest must be identified. This requires use of some unique property of that DNA (such as its sequence, or the protein it codes for). Finally, if our goal is to genetically engineer a cell to produce a specific protein, we must determine which cells are actually expressing the new genetic information properly.

Now I will discuss some examples of the use of recombinant DNA technology, keeping in mind the things presented above. The first example is not really genetic engineering, but demonstrates many of the points of recombinant DNA technology. This is the construction of a genomic library. Say we want to clone a particular gene from an animal so that it may be studied or perhaps used to genetically engineer another animal. How do we get that gene? If we take total genomic DNA from a tissue of that animal and cut it with a restriction enzyme we would get a great mixture of fragments, one of which would contain the gene. We may be able to identify that gene with an appropriate probe, but purifying the gene in large enough quantities is unlikely. We use recombinant DNA techniques to develop a library of the genomic fragments. A mixture of cut DNA fragments is ligated specifically into some vector and used to transform bacterial cells. The cells are allowed to replicate themselves or the virus they contain. Now, although there is still a mixture of DNA fragments, each fragment exists in many copies and is in a vector which will allow it to be replicated at will. This is a genomic library--all fragments from the genome are in a vector and represented in the library. After identifying a cell or viral plaque which contains the gene of interest we can isolate that cell or virus away from all other types by cloning methods.

A specific example of such a genomic library is the one I have used in my own lab (developed by Dr. F. Rottman, Michigan State University). The procedure is diagrammed in Figure 6. Note that the DNA (double-stranded) is represented by a single line rather than a double line in Figures 6, 7 and 8. Bovine genomic DNA was isolated and cleaved with the enzyme Mbo I. This enzyme recognizes the

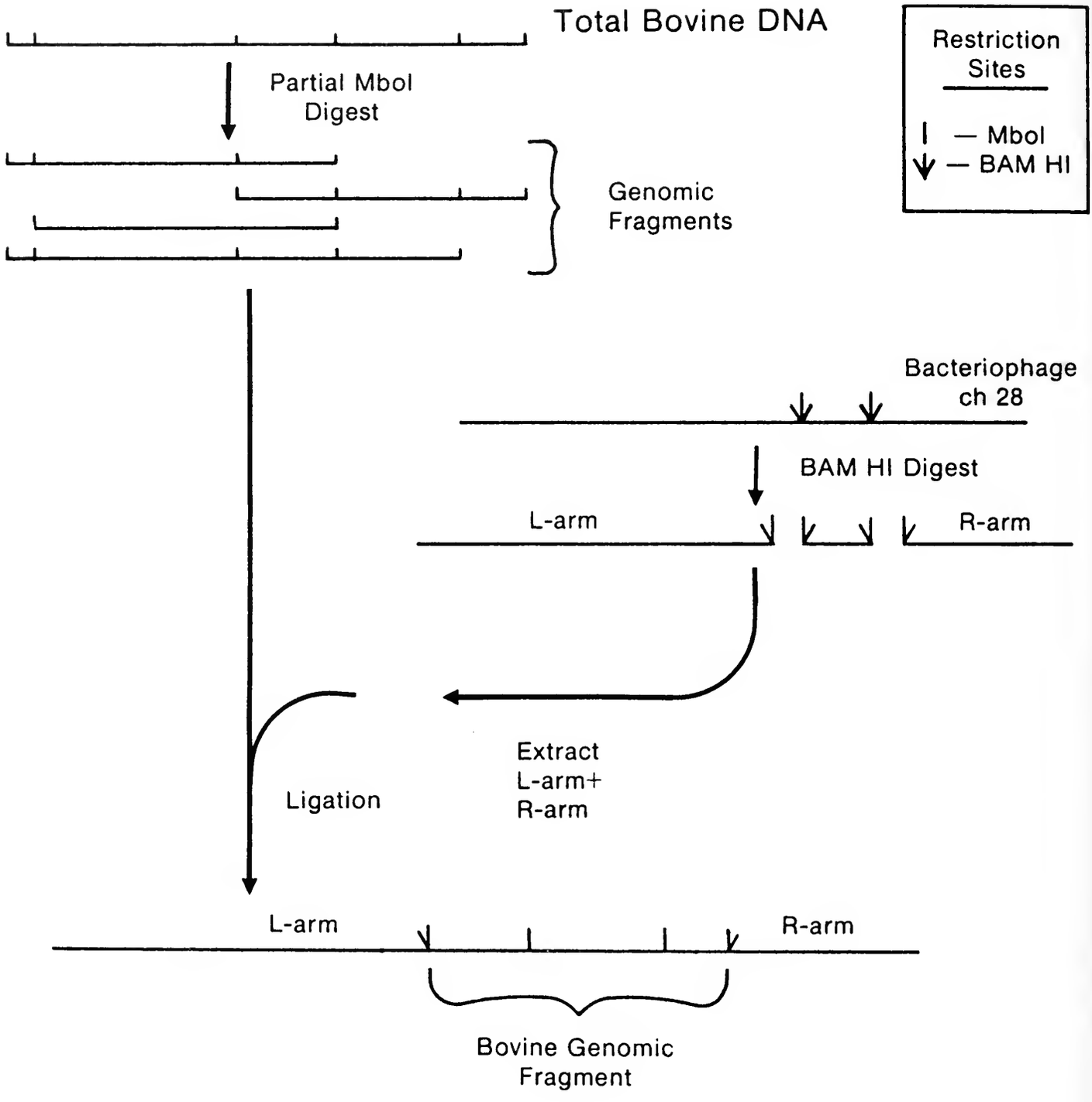


Figure 6. BOVINE GENOMIC LIBRARY. See text for discussion.

internal four base pairs of the Bam HI site giving the same overhang as digesting with Bam HI. However, because Mbo I recognizes four base pairs while Bam HI recognizes six, there will be many more Mbo I sites than Bam HI sites in the DNA. This DNA was only partially cleaved with Mbo I giving a random assortment of the fragments, some which are overlapping. Next, in a separate test tube, the vector is cut with Bam HI. This particular vector is a bacteriophage (virus which infects bacteria) which has two Bam HI sites. Cutting with Bam HI will give three DNA fragments. The right and left end fragments are purified and mixed with the Mbo I cut bovine genomic DNA fragments. The overhang of the Mbo I fragments and of the Bam HI fragments will base pair, and in the presence of DNA ligase will form covalently linked recombinant molecules. The right and left end of the phage DNA are necessary for virus infection and replication, the genomic insert is in the middle region which does not disrupt the viral activity. These recombinant virus molecules are then loaded into empty virus coat-protein caspids (outer virus protein shell). Viruses are amplified by infecting cells with the virus mixture and collecting multiplied new viruses when they lyse the bacterial cells. We now have many copies of each cow genomic fragment, and the library can be reused many times. If we were looking for the growth hormone gene we could use a DNA probe to identify which virus contains the cow growth hormone gene.

In the example above we were only interested in having the virus and bacteria synthesize more DNA. The next example is a case where we want the bacteria to produce a particular protein--genetic engineering. I have chosen this example because it will likely have direct impact on the livestock industry in the fairly near future.

Foot and mouth disease is common in some parts of the world and a constant threat to any livestock region. The disease is caused by an RNA virus about 8,000 bases long with a poly A tract on one end. This is similar to the general form of many messenger RNAs in plants and animals. It would be of great value to develop a means of vaccinating against this virus which is reliable, inexpensive and has no risk of side affects. How to do this? We know that the viral parts against which the animal produces antibodies are the coat (caspid) proteins. These proteins are coded for by the viral RNA. How could we make a DNA copy of that RNA code and use it to make bacteria produce this protein? One group has used the following approach (Kupper, *et al.*, Nature 289:555). Complementary DNA (cDNA) fragments were synthesized from the viral RNA (Figure 7), using purified enzymes and the viral RNA as a template. Coding sequence for the protein of interest, VPI, was known to be between the Bam HI and Hind III sites on one of the cDNA fragments (Figure 7). This cDNA was cloned into the Pst 1 site of pBR322 which disrupts the ampicillin resistance gene of the plasmid (Figure 4). Thus, any bacterial cell transformed with this recombinant plasmid will be resistant to tetracycline (still has intact tet^r gene), but will die in the presence of ampicillin. From Figure 7 we see that if we cut the cDNA with both Bam HI and Hind III we will get a fragment which has one end of the VPI coding region cut off (Bam HI site). As it turns out, this does not matter for our purpose here.

Another vector was developed as shown in Figure 8, which we will call the expression vector. This vector has only one Bam HI and one Hind III site in it, so if we ligate the Bam HI-Hind III insert (which was cut out of pBR322) into this expression vector it will only be inserted in one orientation. This is important so that the correct strand of the DNA is in the correct position to be transcribed into an mRNA that makes sense for translating to the VPI protein. The importance of the expression vector is two-fold. Firstly, DNA does not

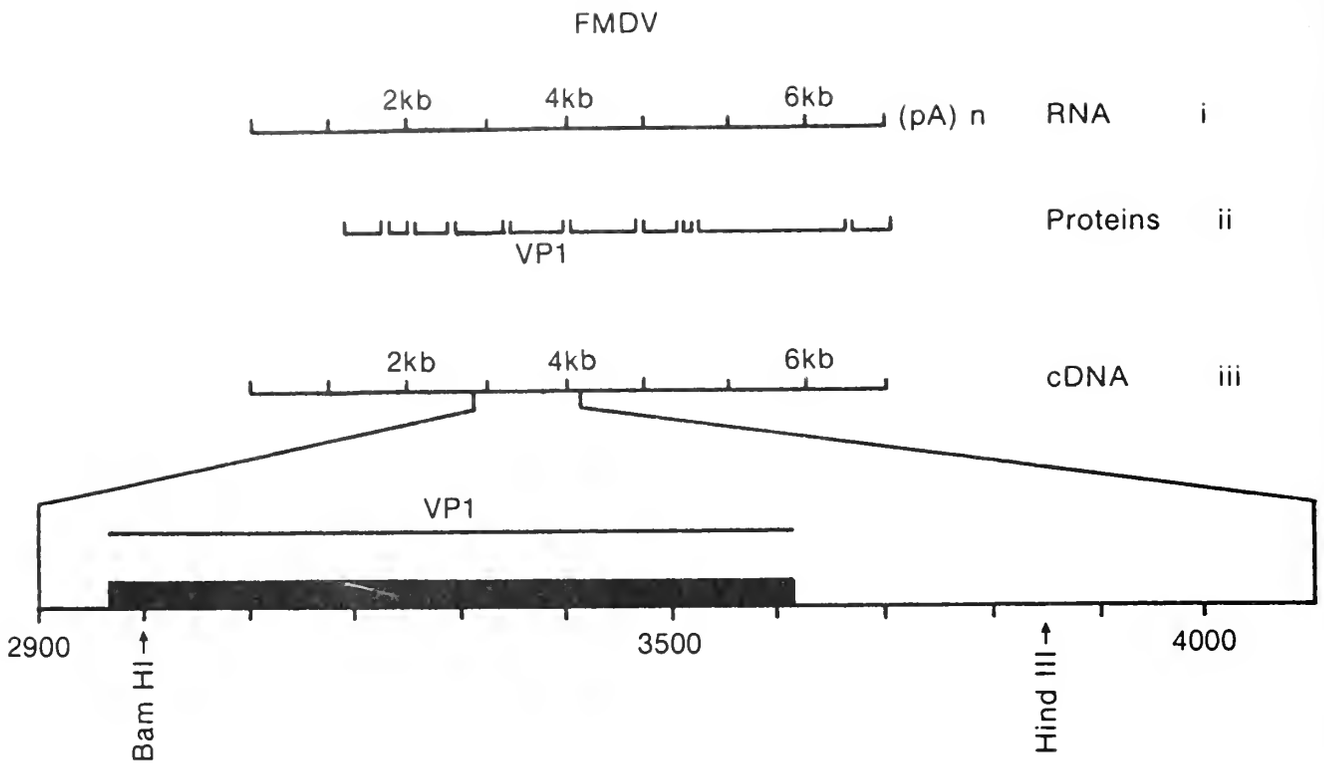


Figure 7. FOOT AND MOUTH DISEASE VIRUS. The FMD virus (i) is an RNA virus (single stranded), about 8,000 bases long (1,000 bases = 1 Kb) with a string of A's on one end (designated (pA)n). This is a very similar structure to messenger RNAs of cells. When this viral messenger RNA is translated (see Figure 3), a number of proteins are synthesized (ii). One such protein designated VPI is a major viral coat protein to which antibodies against FMDV are produced by an animal. A set of complementary or cDNAs were enzymatically synthesized in vitro from the viral messenger RNA (iii). The cDNA containing the sequence for VPI is represented on an expanded scale at the bottom of the figure. Note that the DNA segment between the Bam HI site and the Hind III site contains most of the DNA sequence coding for VPI. See text for further discussion.

necessarily start transcribing on its own. There must be sequence regions which "control" the transcription of a gene. The area marked PL is such a region. Not just any controller can be used. One which is compatible with the cellular transcribing machinery of that particular cell type must be used. This PL region is compatible with the bacterial system chosen by these investigators. A second important feature of the expression vector is the MS2 region. This actually is the beginning of a bacteriophage (MS2) gene and gives the plasmid a specifically placed transcription initiation site which can be recognized by the bacterial enzymes. We now have an appropriate starting place for transcription. Another feature of the expression vector is the Bam HI site. Amino acids in a protein chain are coded by a triplet of nucleotide bases called a codon (Figure 3). Unless the FMDV insert is placed into the expression vector properly the nucleotides will be out of phase--that is shifted over, so that when the transcribed mRNA is translated it will result in the wrong amino acid sequence even though the correct nucleotide sequence is present. So the FMDV insert must be put into the expression vector with a particular reference to the transcription initiation site of MS2. This is shown in Figure 9, where ligation of the Bam HI ends of

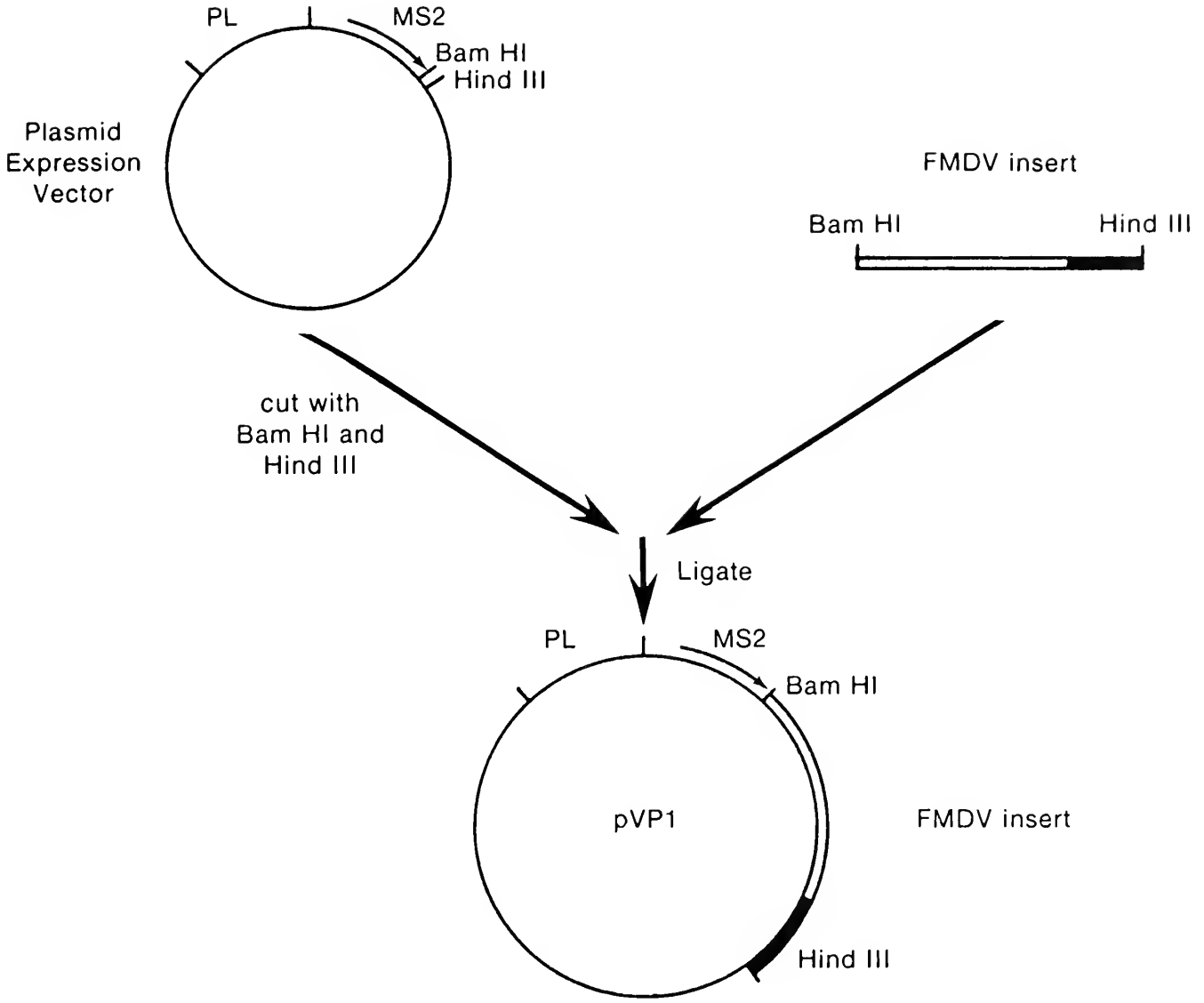


Figure 8. FINAL RECOMBINANT MOLECULE USED TO GENETICALLY ENGINEER BACTERIA. Expression vector contains a transcription control region (PL) and the specific transcription initiation site at the beginning of MS2. FMDV insert DNA is cut out of pBR322 with Bam HI and Hind III and this cDNA fragment containing the VPI gene is ligated into the plasmid expression vector which was also cut open with Bam HI and Hind III. The cDNA fragment will align in the expression vector in only one orientation because only the Bam HI ends of expression vector and fragment will go together, and likewise only the Hind III ends of each DNA will go together. The final recombinant plasmid called pVPI is the one which will be expressed in the bacteria to produce the hybrid VPI protein.

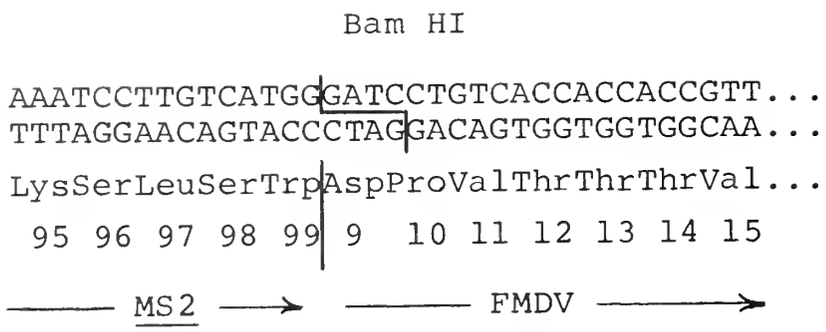


Figure 9. JUNCTION OF FMDV DNA AND EXPRESSION VECTOR. The sequence around the Bam HI site of pVPI is shown here. In order for the mRNA transcribed from this recombinant DNA molecule to be translated into a protein which makes sense with respect to the VPI amino acid sequence, the codons for VPI must be in phase with codons of MS2. By joining the pieces of DNA at the Bam HI site luckily the VPI codons are in phase. In other instances different approaches may be needed to keep the codons in phase. (from Kupper, et al., Nature 289:555, 1981).

insert and expression vector fortuitously results in aligning the insert DNA in phase with the MS2 DNA. Transcription then starts at the beginning of MS2 and proceeds down the DNA past the Bam HI site with all codons still in phase, and past the Hind III site into plasmid DNA sequence where a termination site exists about 13 more amino acids downstream (not shown). When this mRNA is translated it results in a hybrid protein, about three-quarters of which is VPI protein. Bacteria containing this recombinant plasmid and synthesizing the hybrid protein can be grown in large cultures. The purified protein can then be used to immunize livestock against the disease.

In this procedure all components of recombinant DNA technology were used--vectors, inserts, transformed bacteria, and methods to screen for DNA sequences or the hybrid protein. In addition, several other factors came into play. There had to be appropriate transcription control regions. There had to be a transcription initiation site that the bacterial transcribing enzymes could recognize. And, the protein DNA code had to be properly in phase with these other components to result in the protein we want. These same factors must be accounted for anytime one plans to genetically engineer any cell.

Bacteria, plant and animal cells all have different transcribing machinery. Every cell type and every insert DNA proposed for genetic engineering will require different vectors or different approaches. Much of the success of genetic engineering is up to the ingenuity of the investigator in designing the best recombinant molecule for the particular need. In the example above, the hybrid protein may be adequate for immunizing animals, but if a complete protein is needed without extra amino acids, then other approaches must be taken. In a case like genetically engineering bacteria to synthesize biologically active insulin another problem arises. The hormone insulin is actually two polypeptide chains linked together in a specific manner. We could fairly easily get bacteria to make the two chains, but how do we tell the bacteria how to link them together properly? These are just some of the complications with genetically engineering bacteria.

Prospectives of Genetically Engineering Bacteria

Bacteria could potentially be engineered to do a multitude of things. The foot and mouth disease vaccine and bovine growth hormone are two examples of proteins being synthesized by genetically engineered bacteria; both should be available for marketing within the next few years. Theoretically any protein could be made this way--hormones, hormone receptors, structural proteins and enzymes. These bacteria would make the desired protein easier to obtain in larger quantities than present expensive tissue extraction procedures. Bacteria could be altered to produce compounds they would not normally produce like vitamins, hormones and organic compounds for industrial use. Likewise, bacteria could produce enzymes which would permit them to degrade industrial pollutants or decompose animal waste faster. Gastrointestinal bacteria play a large part in animal nutrition. Such bacteria may be engineered to aid the animal in being even more efficient in feed utilization.

All is not blue sky and roses, however. Most existing microbial populations are mixed, with many types of bacteria, protozoa, yeast, and algae, interacting. For instance in the rumen we could take out one type of bacteria, and genetically engineer it to be a "super" bacteria for digesting fiber. If we now place it back in the rumen, it may disappear because it cannot properly compete with existing organisms. Obviously, the potential of genetically engineering microorganisms must await more knowledge about their normal environment and their interactions, whether it is in the cow's rumen, in a pig's intestine or in the manure tank. All organisms must be investigated as each microorganism is a different entity.

Genetic Engineering of Plants and Animals

Genetic engineering of plants and animals will be covered by the next two papers. Here I would like to make a few general statements. Much of the potential of genetically engineering microorganisms is also true with animals and plants. In the livestock industry we have the potential to make pigs grow faster and cows give more milk--both on less feed. We may be able to cure genetic diseases. Yet, there are many problems to be faced. Plant and animal genes and cells are much more complex than those of bacteria. It will be more difficult in multicellular organisms to incorporate recombinant DNA (containing the gene) into the chromosomes of a cell in a permanent fashion, so that all cells derived from it will carry the gene.

The complexity of a pig is a reflection of the number of its genes which are responsible for a particular characteristic. Growth is something on every livestock producers mind. But, how many genes control growth? Which one or ones are most important? What other genes are important in regulating those genes responsible for growth? Which set of these genes are essential for engineering faster growing pigs? For instance, we could clone the porcine growth hormone gene and put it into a pig embryo hoping to get faster growing pigs. But, that embryo genome already has at least one good growth hormone gene. Will the genetically engineered animal with two genes have twice as much growth hormone in its blood, or will the normal regulation of the growth hormone gene keep the blood level of hormone from two genes the same as from one gene in a non-engineered pig? If it does then we would have done a great deal of work for nothing, because it will not grow any faster. Rather, should we be looking beyond growth hormone, perhaps at the somatomedin hormones which apparently are directly involved in some of

growth hormone's actions? Or, should we be looking more at those genes which regulate growth hormone synthesis and secretion? Would it be better to take the growth hormone gene from another species and put it in the pig where it might be regulated in such a way as to give faster growing, more efficient pigs? These are the kinds of problems and questions that must be answered before the potential of genetic engineering can be fully realized.

SUMMARY

With genetic engineering we may be able to make bacteria, plants and animals do things they are not now genetically capable of doing. Recombinant DNA technology is at the heart of this potential, because it allows us to manipulate individual genes. But, recombinant DNA is by no means the total story. Before the full potential of genetic engineering is realized, we must know much more about the biology of those organisms in which we have an interest. We must know which are the appropriate genes to use and how they would best fit into the organism.

Genetic engineering unquestionably has immense potential and for many years you will continue to hear much about that potential. However, little of this will be tempered with a realistic assessment of this potential. I have tried to point out some of the problem areas, so that you may better make your own assessment of the true potential of genetic engineering.

Genetic and Embryonic Engineering in Mammals

CLEMENT L. MARKERT

Molecular genetics occupies center stage in experimental biology today, and rightly so. The tools for examining the structure of chromosomes and for altering them are well developed, and we can now aspire to write the chemical formula for large stretches of the genome--eventually for all of it. The sequence of the three billion nucleotides making up the mammalian genome will in some distant day be stored in a computer for ready recall, examination, and alteration. On a modest scale we can make some changes in the design of the genome even today. We can remove the total genetic material from cells, chop it into many pieces, and identify individual genes and associated regulatory DNA on the separated pieces of the chromosomes. We can also make copies of individual genes and clone these in bacterial hosts. We can also synthesize genes in their standard form or in specific mutant varieties. The genome even of mammals has proved to be far more labile than ever previously imagined. Genes fabricated in vitro and multiplied by cloning in bacteria can be inserted by viral or plasmid vectors into chromosomes within cells and the inserted genes can function. What does this increasing capacity and marvelous technology lead to? It makes us confident that we can one day realize the goal of genetic and embryonic engineering, that is to fabricate the genotypes of organisms to suit our own taste. However, many of the possibilities for exploiting genetic and embryonic engineering available to us today do not rely upon molecular genetics but alter the genome in quite different but still useful ways.

Perhaps we should first distinguish genetic from embryonic engineering. Briefly stated, genetic engineering requires the alteration of the genome. Embryonic engineering, on the other hand, involves altering embryonic development but without necessarily changing the genome. Embryonic engineering includes such topics as cloning, twinning, chimeras, separation of X and Y bearing sperm, fertilization by injected sperm, and cultivating embryos in vitro. The first and most important requirement for manipulating mammalian embryos in vitro was the formulation of media in which embryos could develop outside the female reproductive tract (Whitten, 1971). Such culture media were indispensible to the manipulations of the mouse embryo that will be described in this report. Ancillary to cultivation in vitro is the ability to superovulate mice through proper hormonal treatment, to remove the oocytes from the ampulla of the oviduct, to fertilize the ova in vitro, and to cultivate embryos to the blastocyst stage before insertion into the uterus of suitably prepared female recipients.

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The second major advance was the development of the tools and techniques for microsurgery on mammalian eggs. The development of these techniques was initiated by T. P. Lin (1971), who first showed that it was possible to insert a fine pipette into a mouse egg and inject a drop of oil without killing the egg. The further development and refinement of these microsurgical techniques (Markert and Petters, 1977) makes it possible to perform several different kinds of surgical intervention in mammalian eggs. These microsurgical interventions can alter the genome and lead to cloning and vegetative or parthenogenetic reproduction.

Among the developments made possible by the ability to culture embryos in vitro is the ability to produce identical twins by the separation of the blastomeres at the two-cell stage. This can easily be done by removing the zona pellucida with pronase and then by either shaking the blastomeres apart mechanically or by a mild digestion with trypsin. Such separated blastomeres will then continue to divide to produce small morulae which when implanted in the uterus will continue to develop to produce identical twins. Even quadruplets might be produced by the same method, using the blastomeres of the four-cell stage, but with mice this has proved to be exceedingly difficult even though the first four blastomeres are developmentally equivalent. The total mass of material in one blastomere of the four-cell stage appears to be inadequate to support the development of an entire embryo to the stage of implantation. However, identical twins are relatively easy to produce in mice.

Our ability to capacitate sperm and to fertilize eggs in vitro facilitates some of the engineering manipulations on the early embryo but fertilization is dispensable. Eggs can easily be activated to develop parthenogenetically. Such parthenotes may be either haploid or diploid but no mammalian parthenote has ever developed to term. The maximum development achieved by mouse parthenotes is about the twenty-five somite stage of development (Kaufman et al., 1977), which is very extensive, to be sure, but still far from normal term development. Parthenotes can be produced by a number of different egg-activating stimuli: ionophores, electric shock, a variety of chemicals, changes in temperature, and numerous other stimuli have been applied more or less successfully to provoke the egg to develop. In mice there is one strain, the LT/Sv strain, that produces oocytes that frequently begin development without having been fertilized. These parthenotes are nearly always diploid, presumably as a result of the suppression of the second polar body. Other parthenotes produced by chemical activation of the egg or by microsurgical removal of one pronucleus from the fertilized egg are initially haploid but very frequently diploidize spontaneously. Parthenotes pose very interesting biological problems, and if these can be solved, would open the possibility for creating economically valuable strains of mammals by embryonic engineering. Successful parthenogenetic development is found in a few representatives of every vertebrate class except mammals (cf Markert, 1982).

In mammals, parthenotes are easy to start, but they never develop satisfactorily. They die rapidly, and the fraction of parthenotes that continues to develop diminishes steadily until all are dead by about the twenty-five somite stage. Nevertheless, this developmental failure is not based upon the lethality of individual cells. The early parthenote, at about the four or 8-cell stage, can be rescued by combination with normal embryos to create chimeras which, when implanted in the uterus, do develop to term (Stevens, 1978). In these chimeras are found cellular representatives of the parthenotes in virtually every tissue, including gametes. Thus, the developmental failure of parthenotes represents a failure of the community of embryonic cells rather than a qualitative incapacity on the part of individual cells to differentiate normally. The nature of this

failure is quite unknown but is the subject of active investigation in a few laboratories.

Perhaps the block to successful parthenogenetic development in mammals is an evolutionary response to the dangers inherent in abandoning sexual reproduction in favor of parthenogenesis. Since mammalian eggs are so readily activated to start developing, if there were not some block to their further development, then parthenogenesis would probably be common among mammals. Parthenogenesis may present serious evolutionary dangers to a species, but for practices of animal husbandry there are no serious problems and many advantages. To overcome the failure of parthenotes probably will require better knowledge of meiosis and of the contributions of the sperm to the zygote. Perhaps sperm make three distinct contributions: activation of the egg, their haploid genome, and a third undefined contribution required for normal development. There is no positive evidence for this third putative contribution, but it is difficult to avoid the conclusion that the contribution is real when we compare parthenote and homozygous diploid development after microsurgical removal of the sperm pronucleus (Hoppe and Illmensee, 1977).

CHIMERA PRODUCTION

After Mintz (1962) and Tarkowski (1961) first reported the successful production of mouse chimeras, an extensive exploitation of this technique to investigate many fundamental and interesting problems in biology has occurred (Mintz, 1974; Markert and Petters, 1978; McLaren, 1975; Lu and Markert, 1980). As research tools, chimeras have enormous value even though they have no immediate economic value. One of the most important problems that can be addressed through chimeras is the mechanism of primary sex determination. When we make chimeras by adding two embryos together, we of course, half the time, add a male to a female embryo. Such composite sex chimeras might be expected to develop abnormally or to become hermaphrodites. Not so. They develop into perfectly normal individuals, usually males, but occasionally females. An understanding of these chimeras might be helpful in understanding freemartin cattle.

Chimeras do allow an examination of the ability of cells of different genetic makeup, even from different species, to collaborate in the development of functional tissues and organs. The genetic responsibility for the fine points of cell differentiation can be assessed in chimeras but often not at all in non-chimeric individuals. By placing cells of different genetic makeup in competition with one another during the development of given tissues and organs, it is possible to judge the relative value of different genotypes to bring about particular kinds of cell differentiation. The production of interspecific chimeras should allow us to examine the degree of species specificity in the language of communication between cells. So far, chimeras have been made between rats and mice (Gardner, 1973), between Mus musculus and Mus caroli (Rossant, 1980), and between different species of Peromyscus, the field mouse, common throughout most of the United States. The rat-mouse chimeras developed only for a short time before dying. The chimeras between the two different species of house mice developed to term and reached adulthood. The genus Peromyscus includes species of many different degress of relatedness from sibling species that can hybridize successfully to those that cannot cross at all. Chimeric embryos even between different species are easily made but the implantation of such chimeric embryos into recipient females means that half of the chimera finds itself in a foreign uterus. We have found that such chimeras do not implant successfully although

they develop readily in vitro to the blastocyst stage. There is an alternative way of making chimeras which would permit the cells of one species to remain segregated inside of the blastocyst cavity and thus isolated from directed contact with the uterus. In such chimeras the trophectoderm can be entirely from the host species. This technique has so far not been used successfully with Peromyscus. Nevertheless, the attempt to produce such inter-species chimeras forced us to examine the problem of the relative roles of the genotypes of the embryo and the maternal organism in permitting normal development. It is common knowledge that the embryo differs genetically from the maternal organism, and yet is not rejected even though it constitutes a foreign graft into the uterus. Cells of the same genetic composition grafted elsewhere in the female would certainly be rejected. Since the uterine environment permits development of embryos of quite different genotype from that of the maternal organism, it seems surprising that the female rejects embryos of a different species. Only one case of gestation to term in the uterus of a different species has been observed (Markert and Klein, 1983). Embryos of Peromyscus maniculatus were transplanted into the uteri of Peromyscus leucopus and developed to term. The reciprocal transfer, P. leucopus embryos into the uteri of P. maniculatus, has not proved successful. Whether the rejection of the foreign embryo is based upon immunochemical reactions or upon some physiological incompatibility is not known, but is a subject worthy of extensive investigation.

MICROSURGERY

Mammalian eggs on examination with a light microscope appear to be fragile structures not likely to withstand extensive manipulation. However, mammalian eggs are in fact resilient tough cells that can sustain extensive damage and still develop normally. This biological capacity has made possible many of the microsurgical procedures that will be reported here and that will surely be extensively used in the future. After T. P. Lin's work the first microsurgical intervention into the developing embryo was the removal of one pronucleus from the fertilized egg (Markert and Petters, 1977). The procedure we developed for enucleating mouse eggs is as follows: fertilized eggs are removed from the oviduct of a mouse or eggs may be fertilized in vitro. About twelve hours after fertilization, two pronuclei are clearly visible in the egg. The egg, surrounded by the zona pellucida, can be firmly held by suction with a holding pipette. An enucleation pipette, finely beveled on a diamond grinding wheel, can now be inserted into the egg and attached by suction to one of the pronuclei. The pipette is then slowly withdrawn from the egg, taking the nucleus with it. The nuclear membrane is very tough and elastic and the nucleus can be withdrawn from the egg without much difficulty. By the use of a somewhat larger pipette, it is possible to suck the entire pronucleus into the enucleation pipette before withdrawal from the egg. The plasma membrane is also elastic; it usually constricts and heals immediately after the enucleation pipette is withdrawn. If the egg is treated with cytochalasin B about an hour before these operations are undertaken, microfilament contraction is diminished, injury to the cytoarchitecture of the egg reduced, and a higher percentage of successful operations obtained. These procedures were first used to produce haploid embryos which developed to the blastocyst stage in vitro, but none of them ever developed to term. Incubation overnight in the presence of cytochalasin B permits the remaining pronucleus to replicate without a corresponding division of the cytoplasm. Thus, twelve hours or so after removing one pronucleus, a second pronucleus becomes apparent. Both pronuclei are of course genetically identical. When the cytochalasin B is washed away, the two pronuclei approach one another, the nuclear membranes disappear, a

spindle forms and the chromosomes from both pronuclei assemble on the spindle to produce a diploid completely homozygous zygote. Such zygotes readily develop in vitro to the late morula or blastocyst stage. The homozygous embryos can then be inserted in the uterus of a suitably prepared recipient female. In our laboratory, several hundred such embryos have been implanted but not a single one has ever developed all the way to term. Nonetheless, extensive development occurs, sometimes reaching more than half way through gestation. Hoppe and Illmensee (1977) reported that seven embryos prepared in this fashion continued to develop when transferred to recipient females. Of the seven mice born, two were androgenetic in origin and five were gynogenetic. All seven proved to be fertile and have reproduced. This is a remarkable achievement requiring great technical skill and perhaps quite a bit of luck. In view of the larger number of failures in at least two other laboratories (Markert, 1982; Modlinski, 1980), one cannot embark upon a program of such enucleation with any certainty of success.

The ability to microsurgically intervene in the egg has not only made possible the removal of a pronucleus but has also made possible nuclear transplantation. Illmensee and Hoppe in one extensive series of experiments did succeed in removing both pronuclei from fertilized eggs and then in implanting into the enucleated egg a nucleus derived from a cell of the inner cell mass. Three embryos were born as a result of such nuclear transplantation, representing the first case of cloning in mammals. When the same kind of experiment was carried out using nuclei from trophectoderm cells, none developed. These results are essentially what one would expect on the basis of our previous experience with amphibian nuclear transplantation (Briggs, 1977; DiBerardino, 1980; Gurdon et al., 1975).

Early embryonic nuclei appear to retain the capacity to replace a zygote nucleus, but in later stages of development the nuclei are no longer totipotent. There is no economic advantage to cloning embryos of valuable domestic mammals before one has the opportunity of assessing the phenotype. The ability to clone adults who have demonstrated their worth would clearly be valuable. This may eventually be possible, but only after the nucleus has been restored to developmental totipotency. One line of investigation that holds such promise is the transplantation of the nucleus of an oocyte where it is subject to the same cytoplasmic influences that exist during normal gamete formation. Such experiments have been performed with amphibians in which they are technically much easier than in mammals (Hoffner and DiBerardino, 1980). These investigators showed that nuclei exposed to oocyte cytoplasm acquired enhanced developmental capacity but not sufficient to equal the zygote nucleus. They were not developmentally totipotent. If the DNA of differentiated cells has not been altered by deletion or rearrangement, then we can reasonably expect to restore totipotency to nuclei taken from the differentiated cells of the adult. We do know of only a few instances in which DNA is deleted or rearranged during cell differentiation. Lymphocytes are the prime example but they appear now to be a rare exception. The DNA can also be altered during development by chemical modifications as by methylation of cytosine bases. Such methylation is reversible.

The X-chromosome presents the best example of persistent, yet reversible, inactivation during cell differentiation and cell replication (Gartler and Andina, 1976; Liskay and Evans, 1980). The inactive condition of the X-chromosome is maintained through many cell divisions in eutherian mammals, but this virtually permanent inactivation of the X-chromosome is reversed during oocyte differentiation, again offering the prospect that by exposing differentiated nuclei to the right cytoplasmic environments, it may be possible to restore developmental

totipotency (Venolia et al., 1982; Chapman et al., 1982). If and when that is achieved, true cloning of adult mammals will certainly be possible.

SPERM INJECTION

The implantation of a nucleus into an enucleated egg is technically difficult because of the size of the nucleus and the large pipette that must be used. It is much easier, however, to inject sperm because they are much smaller and more resistant to mechanical damage. We have injected the heads of sperm after the tails were knocked off by ultrasonication and found that these sperm are fully capable of fertilizing the egg and participating in normal development. In other words, the elaborate series of membrane interactions that occur when the sperm normally fertilizes an egg are unnecessary and can be circumvented by simple implantation of the sperm directly into the oocyte. We can also inject entire sperm without prior sonication and the same effect is achieved. Such motile sperm simply present a greater challenge in the capture of the sperm with a micropipette prior to injection into the egg (Thadani, 1980; Uehara and Yanagimachi, 1976, 1977). These techniques make possible the fertilization of an egg by a single selected sperm and eliminate the necessity of using large numbers of sperm to fertilize eggs in vitro. Sperm injection also enables a test of whether or not the phenotype of the sperm reflects its genotype. Our evidence is quite preliminary, but it does suggest that sperm, no matter how morphologically or phenotypically defective they may be, are all fully capable of participating in normal development if injected into the egg. Thus, valuable samples of sperm from prize bulls could be extended indefinitely by using the sperm one at a time by microinjection, rather than using millions of sperm in artificial insemination to fertilize single eggs. A further elaboration of these procedures, which we are now investigating, is based upon the identification of X and Y-bearing sperm by staining. Sperm carrying the Y chromosome in some species can be made highly visible by fluorescent dyes, and thus it would be possible to pick up either X or Y-bearing sperm individually and inject them into the egg in order to specify the sex of the resulting zygote. This method of sex selection will succeed only if the stain does not prevent normal sperm reactions to the cytoplasm.

CHROMOSOMAL AND GENE INJECTIONS

Since nuclei and sperm can be injected into mammalian eggs, it follows that it is even easier to inject individual chromosomes or genes. We have injected numerous chromosomes into eggs just as the pronucleus was being formed or as the spindle of the first mitotic division became apparent. However, we have not yet succeeded in producing an embryo showing replicating injected chromosomes. The problem is largely one of positioning the injected chromosome so that it will become attached to microtubules of the spindle. When chromosomes are injected successfully, the resulting aneuploid will be of no economic value but will offer the prospect of studying the individual chromosomal control of various aspects of cell differentiation. Such trisomies can now be produced by the use of breeding stocks carrying various chromosomes in Robertsonian fusion arrangements. These were initially derived from the tobacco mouse in which many such chromosomal fusions exist. However, the injection techniques should be superior because any particular chromosome could be selected for injection into the egg to produce the desired aneuploids directly. Although such aneuploids would not be expected to develop normally, they can be rescued by combination with normal embryos to make chimeras in which the trisomic cells can then be examined for their ability to

undergo various kinds of cellular differentiation.

Finally, individual genes or groups of genes can be injected directly into the nucleus of the egg at the pronuclear stage or later (Gordon and Ruddle, 1981). Such injected genes have been shown in several laboratories to become incorporated in the chromosomes and even, in a few instances, to be expressed in the production of protein products in the derived animals. The genes incorporated in the chromosomes are of course passed to succeeding generations through normal sexual reproduction.

Since we can now manufacture genes in the test tube and produce any sequence of nucleotides we wish, it is clearly possible to inject predetermined types of DNA into the nucleus of a mammalian egg and expect the injected DNA to become incorporated and possibly expressed during cell differentiation. There is one great problem, however, to be overcome in any experiment designed to alter genetic makeup by injection of DNA. That is the problem of regulating gene expression. We are completely ignorant of the mechanisms for regulating gene function during development. We know a great deal about the structure in individual genes, and even the structure of the flanking sequences of DNA that are involved in the function of the genes, but we know essentially nothing about what determines whether a particular gene will function in one cell type and not in another or at what level the gene will function. In short, until we understand the processes of gene regulation in normal development, we are not likely to be able to make much use of injected genes to alter in a useful fashion the function of mammalian genomes.

Since the karyotype is quite stable in species and differs from one species to the next, it seems likely that the topography of genes in the chromosomes is important in their proper regulation during development. If this is so, then it will be necessary to place injected genes in particular regions of the genome if they are to function in a useful fashion. We may develop such experimental capacity, but that capacity will not be very useful until we have identified the appropriate location.

PRACTICAL PROBLEMS IN APPLYING LABORATORY TECHNIQUES TO OTHER MAMMALS

What can be done with a mouse can surely be done eventually with pigs and cattle and sheep and even with human beings. However, every species is unique and unfortunately quite different with respect to some of the practical conditions required to manipulate the genome. We can, for example, cultivate the mouse egg from ovulation to the blastocyst stage in vitro, but we cannot do so for the rat egg. In fact, there are only a half a dozen species in which the culture conditions have been worked out so as to make possible the cultivation of the egg to the blastocyst stage. Even in the mouse, the culture media are not ideal. The oviduct does better.

The fabrication of suitable culture media presents perhaps the greatest initial practical problem in the extension of genetic and embryonic engineering techniques to other mammals. Certainly such media can be developed, but the work is tedious and uncertain. Once a satisfactory culture medium has been devised, then the problem of microsurgical manipulation of different eggs arises. Some eggs are virtually transparent, others are opaque; the pig egg, for example, is so filled with yolk that it is nearly impossible to observe nuclear structures within the egg. The eggs of some species are more tolerant of mechanical injury than

others. The rat egg, e.g., is tougher than the mouse egg. However, it seems likely that virtually all mammalian eggs will be able to sustain at least the minimum damage required to insert a pipette into the egg. In summary, the techniques already tried in the laboratory, or logical extrapolations from them, make possible the specific and selective alteration of the genomes of mammals and also make possible the vegetative reproduction of any particular genotype. Surely such remarkable developments in the ability to control the genetic makeup of mammals will have wide-ranging consequences for the creation and maintenance of new strains of commercially valuable mammals.

BIBLIOGRAPHY

Briggs, R. (1977). Genetics of cell type determination. In Cell Interactions in Differentiation. L. Saxen and L. Weiss, Eds. Academic Press, New York. pp. 23-43.

Chapman, V. M., P. G. Kratzer, L. D. Siracusa, B. A. Quarantillo, R. Evans, and R. M. Liskay. (1982). Evidence for DNA modification in the maintenance of X-chromosome inactivation of adult mouse tissues (Searle's translocation/DNA-mediated cell transformation/hypoxanthine phosphoribosyl-transferase). Proc. Natl. Acad. of Sciences USA 79: 17, pp. 5357-5361.

DiBerardino, M. A. (1980). Genetic stability and modulation of metazoan nuclei transplanted into eggs and oocytes. Differentiation 17: 17-30.

Gardner, R. L. and Johnson, M. H. (1973). Investigation of early mammalian development using interspecific chimaeras between rat and mouse. Nature New Biol. 246: 86-89.

Gartler, S. M., and Andina, R. J. (1976). Mammalian X-chromosome inactivation. Adv. Human Genet. 7:99-140.

Gordon, J. W. and Ruddle, F. H. (1981). Integration and stable germ line transmission of genes injected into mouse pronuclei. Science 214:1244-46.

Gurdon, J. B., Laskey, R. A., and Reeves, O. R. (1975). The developmental capacity of nuclei transplanted from keratinized cells of adult frogs. J. Embryol. Exp. Morphol. 34: 93-112.

Hoffner, N. J. and DiBerardino, M. A. (1980). Developmental potential of somatic nuclei transplanted into meiotic oocytes of Rana pipiens. Science 209: 517-519.

Hoppe, P. C. and Illmensee, K. (1977). Microsurgically produced homozygous-diploid uniparental mice. Proc. Natl. Acad. Sci. USA 74: 5657-5661.

Hoppe, P. C. and Illmensee, K. (1982). Full-term development after transplantation of parthenogenetic embryonic nuclei into fertilized mouse eggs. Proc. Natl. Acad. Sci. USA 79: 1912-1916.

Hoppe, P. C., and Pitts, S. (1973). Fertilization in vitro and development of mouse ova. Biol. Reprod., 8:420-426.

Illmensee, K. and Hoppe, P. C. (1981). Nuclear transplantation in Mus musculus: developmental potential of nuclei from preimplantation embryos. Cell 23:9-18.

Lin, T. P. (1971). Egg micromanipulation. In: Methods in Mammalian Embryology. J. C. Daniel, Jr., ed. W. H. Freeman, San Francisco, pp. 157-171.

Liskay, R. M. and Evans, R. J. (1980). Inactive X chromosome DNA does not

function in DNA-mediated cell transformation for the hypoxanthine phosphoribosyltransferase gene. PNAS 77:4895-98.

Lu, Te-Yu and Markert, Clement L. (1980). Manufacture of diploid/tetraploid chimeric mice. Proc. Natl. Acad. Sci. USA, Vol. 77, No. 10, pp. 6012-6016, Oct. 1980.

Markert, C. L. (1982). Parthenogenesis, Homozygosity, and Cloning in Mammals. J. Heredit.: Vol. 73.

Markert, C. L. and Klein, M. S. (1983). Heterospecific gestation of mammalian embryos: Production of Peromyscus maniculatus offspring by P. Leucopus mothers. (In Preparation)

Markert, C. L. and Petters, R. M. (1977). Homozygous mouse embryos produced by microsurgery. J. Exp. Zool. 201: 295-302. 1977.

Markert, C. L. and Petters, R. M. (1978). Manufactured Hexaparental Mice show that adults are derived from three embryonic cells. Science, Vol. 202, 6 Oct. 1978, pp. 56-58.

Markert, C. L. and Seidel, G. E., Jr. (1981). Parthenogenesis, identical twins, and cloning in mammals. In New Technologies in Animal Breeding. B. G. Brackett, G. E. Seidel, and S. M. Seidel, Eds. Academic Press, New York. pp. 181-200.

McLaren, A. (1972c). Late-labelling as an aid to chromosomal sexing of cultured mouse blood cells. Cytogenetics 11, 35-45.

McLaren, A. (1975a). Sex chimaerism and germ cell distribution in a series of chimaeric mice. J. Embryol. Exp. Morph. 33, 205-16.

McLaren, A. (1975b). The independence of germ-cell genotype from somatic influence in chimaeric mice. Genet. Res. 25, 83-7.

McLaren, A. and Biggers, J. D. (1958). Successful development and birth of mice cultivated in vitro as early embryos. Nature, Lond. 182, 877-8.

McLaren, A., Chandley, A.C. and Kofman-Alfaro, S. (1972). A study of meiotic germ cells in the gonads of foetal mouse chimaeras. J. Embryol. Exp. Morph. 27, 515-24.

Mintz, B. (1962a). Formation of genotypically mosaic mouse embryos. Amer. Zool. 2, 432 (Abstr. 310).

Mintz, B. (1974). Gene control of mammalian differentiation. Annual Review of Genetics 8: 411-470.

Modlinski, J. A. (1980). Preimplantation development of microsurgically obtained haploid and homozygous diploid mouse embryos and effects of pretreatment with Cytochalasin B on enucleated eggs. J. Embryol. Exp.Morphol. 60:153-161.

Rossant, J. and W. I. Frels. (1980). Interspecific chimeras in mammals: successful production of live chimeras between Mus. musculus and Mus. caroli. Science 208: 419-421.

Stevens, L. C.(1978). Totipotent cells of parthenogenetic origin in a chimaeric mouse. Nature 276: 266-267.

Stevens, L. C., Varnum, D.S., and Eicher, E. M. (1977). Viable chimaeras produced from normal and parthenogenetic mouse embryos. Nature 269: 515-517.

Surani, M.A.H., Barton, S.C. and Kaufman, M.H. (1977). Development to term of chimaeras between diploid parthenogenetic and fertilized embryos. Nature (London) 270, 601-03.

Tarkowski, A. K. (1961). Mouse chimaeras developed from fused eggs. Nature, Lond., 190, 857-60.

Thadani, V. M. (1980). A study of hetero-specific sperm-egg interactions in the rat, mouse, and deer mouse using in vitro fertilization and sperm injection. J. Exp. Zool. 212: 435-453.

Uehara, T. and Yanagimachi, R. (1976). Microsurgical injection of spermatozoa into hamster eggs with subsequent transformation of sperm nuclei into male pronuclei. Biol. Reprod. 15: 467-470.

Uehara, T. and Yanagimachi, R. (1977). Behavior of nuclei of testicular, caput and cauda epididymal spermatozoa injected into hamster eggs. Biol. Reprod. 16: 315-321.

Venolia, L., Gartler, S.M., Wassman, E.R., Yen, P., Mohandes, T., and Shapiro, L.J. (1982). Transformation with DNA from 5-azacytidine-reactivated X chromosomes. PNAS 79:2352-54.

Whitten, W. K. (1971). Nutrient requirements for the culture and reimplantation embryos in vitro. Advan. Biosci. 6, 129-139.

Genetic Engineering and Plant Breeding

D.E. ALEXANDER

"I have no doubt that in reality the future will be vastly more surprising than anyone can imagine. My suspicion is that the universe is not only queerer than we suppose, but queerer than we can suppose" J. B. S. Haldane

It is appropriate for a plant breeder to quote Haldane as a prologue to his observations on genetic engineering and about its ultimate usefulness in improving plants. Haldane suggests that "queerness" is an attribute of nature, and of course, that man is ultimately incapable of understanding it. I think we can agree on the latter, but I do not concede that partial understanding is without value. We certainly do not fully appreciate, let alone imagine, how the rapidly accumulating knowledge about molecular genetics can be used. But few were able to predict at the turn of the century how Mendelian genetic theory might be used. So we find ourselves at the threshold of great potential without even being able to imagine with any security at all what the outcome will be.

Perhaps it is appropriate to develop perspective about "genetic engineering". We define it as the manipulation of genetic material by "unconventional" means - whatever that means! But included are such techniques as transfer of genetic material among and within species by asexual means, the culturing of individual cell and/or tissue and regeneration of plants from them, the modification of the genetic stuff itself by chemical means, and so on. But conventional means of accomplishing some of the same objectives are excluded, for example, transfer of genetic material (genes, mitochondria, etc.) through crossing between or within species. I point this out merely to emphasize that some of the traditional approaches to plant improvement are now being attempted by other means.

But genetic engineering concepts are particularly appealing because they are based on models that stretch the imagination. For example, the transfer of nitrogen fixing genes (nif genes) from nitrogen-fixing bacteria to corn. Conceptually, the effort is impossible through conventional means, i.e., crossing of rhizobium with corn and recovery of the appropriate genes in segregating populations. But recent research uncovers mechanisms that possibly make the transfer possible. Without those discoveries nitrogen fixation by cereals would only be science fiction. With them fiction could become reality.

The influence of molecular genetics on biology is profound. The elucidation

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of the structure of DNA, the genetic stuff itself, has added a completely new dimension to biology and to the way biologists think about it. The new dimension is essentially a mechanistic one. DNA has a definite structure and that structure can be deduced by purely physical means. It follows that artificial DNA can be constructed or that copies of existing DNA can be synthesized without using ancestoral DNA as a pattern. This is "heady" stuff and with it has come more optimism, more confidence about how we might use the new knowledge, than we can fairly justify. We see this in the flurry of investment in genetic engineering research particularly in the private sector. We also see its manifestation in private funding to support University research.

Although we intuitively know genetic engineering will ultimately be useful in modifying plants, we have abundant evidence that classical plant breeding has already been profoundly successful. That perspective can be of value in defining the directions genetic engineering research should take and the manner in which the findings may be made useful. To contribute to this understanding we shall use the corn breeding experience as an example.

CORN BREEDING: PAST ACCOMPLISHMENTS

Corn was created by man. As we know it, it cannot exist in the wild without his intervention. But its ancestor or ancestors must have existed in the wild. It is most likely that teosinte, a wild plant common to Mexico and Central America, is "wild" corn. Man presumably selected variants within it that were palatable and easily harvested and perpetuated those strains. Teosinte was transformed from a weedy grass, to essentially what we know today, by people we might be inclined to view as primitive. The difference between teosinte and the corn varieties Europeans inherited is for greater than the difference between "Indian corns" and modern hybrids. I make this point because it emphasizes the fact that adaptation and modification of a species is essentially an evolutionary process, a process of selecting and discarding, a slow process dependent on an interaction of the genetic mechanism and environment. The resulting product has arisen as a compromise between hereditary forces and environmental opportunity. To illustrate: Had nitrogen been essentially absent from soil, survival of corn would not have been possible without innate nitrogen fixing capability.

Modern corn breeders were handed a magnificent species, one that had been subjected to thousands of generations of selection under diverse environments and with literally hundreds of relatively unrelated populations each replete with great genetic variability. They created new productive varieties, often by selecting within segregating progenies of crosses between unrelated stock produced by Indian breeders. Typical of these varieties are Reid Yellow Dent and Lancaster Sure Crop. They were widely grown in the U.S. until hybrids became available in the early 1940's. These improved varieties, as well as other we have not named, were sources of inbreds that served as parents of modern hybrid corn.

Grain Yield

Hybrids changed Corn Agriculture. Not only was there a quantum jump in yield with their adoption, but a continuing increase in yield has paralleled the creation of new hybrids. U.S. yields were stable from the 1870's until the early 1940's. The average U.S. yield never exceeded 31 bushels per acre until 1941. They increased rapidly with adoption of hybrids and the simultaneous improvement of cultural practices. They have continued to improve with time. For example, the

gain in yield since 1930 amounted to 1.5 bushels per acre year in Illinois. Of more significance is the fact that the rate of increase has accelerated since 1950. Mean yield increase since then amounted to 2 bushels per acre per year.

It is widely accepted that the quantum jump in yield in the late 1930's was a consequence of hybrid adoption. It also is conventional wisdom that yield improvement since that initial jump was a consequence of improved cultural practices, i.e., more timely planting and harvesting increased nitrogen use, better weed and insect control, etc. Unquestionably, they contributed greatly to the trend.

Russell (1) and Duvick (2) have shown that much of the yield increases are due to genetic improvement. Russell, through an exhaustive series of experiments carried out over three years has shown that no less than 60% and as much as 80% of the increase if hybrids over open-pollinated varieties can be assigned to it.

Hallauer (3) has shown that heritable variation for grain yield was undiminished in several breeding pools after several cycles of selection. Dudley (4) found heritable variation for oil and protein in the Illinois High Oil and Illinois High Protein strains after generations of selection even though oil and protein content had increased from 4.7% and 10.9% to 15.2% and 25.2% respectively. These experiences as well as others, clearly establish that further improvement in yield is not only possible, but unquestionably will happen, if classical breeding schemes continue.

Agronomic Quality

Also grain yield is of paramount importance, other definable qualities contribute to it. To illustrate: A hybrid may have high genetic capacity to produce grain and actually produce it, but lodge badly thus making it difficult to harvest. Not only must a hybrid meet a certain specification well, it also must meet others at acceptable levels. Among them are resistance to disease and insects, ability to withstand stress created by head or drouth or by high planting rates, and ability to mature and dry grain rapidly consistent with the length of growing season. All of these qualities are under polygenic control although some are influenced substantially by major genes. Classical breeding schemes have produced hybrids that will meet certain combinations of these qualities but rarely, if at all, does one meet all of them. In fact, it may be quite superfluous for a hybrid grown in the northern reaches of the Corn Belt to have resistance to Maize Dwarf Mosaic, a viral disease that does not overwinter there.

OPPORTUNITY FOR GENETIC ENGINEERING IN CORN IMPROVEMENT

We have emphasized that those attributes of prime value in corn are polygenicially inherited, i.e., may genes each with a small effect influence yield. If we confine our attention to a single genetic engineering technique, for example the modification of DNA, we find inherent difficulties with its use in improving these complex traits. First of all, literally nothing is known about these genes in a physical sense. We don't know how many exist, we have no techniques or even tentative approaches on how to identify them individually. We have little or no information on how they act upon yield, for example. It would seem completely unworkable now to attempt to modify "yield genes" or "stress genes" in a meaningful way. Furthermore, it seem likely that we shall not soon or easily learn enough about the genetic mechanisms controlling complex traits to make this sort of work

possible.

An illustration of the complexities that arise in this connection is that of transferring nif (nitrogen fixing) genes from nitrogen fixing bacteria to cereals. Although the genetic fine structures (The DNA sequences) of several of these genes has been deduced, and transfer mechanisms exist, none can effect the transfer to corn. If transfer agents (vectors) are found that are compatible, and I believe that they must exist and will be found, transfer of these genes should ultimately be accomplished. If we assume that the necessary transfer has been carried out, and those genes function in a manner compatible with the functioning of the hereditary stuff of corn itself, substantial problems remain. It is reasonable to assume that the energy required to drive the nitrogen-fixing process would compete with those processes affecting other metabolic activities, say, synthesis of starch or oil in grain, or even in the early growth of the plant. The net effect would be to reduce yield. This is to be expected because energy is required to fix nitrogen and the energy source, of course, is sugar produced through photosynthesis. This proposition may be a "straw man" and easily refuted once nitrogen fixing systems are functioning and in breeding pools subjected to testing for yield.

A particularly intriguing possibility is that of modifying the DNA responsible for the coding of storage proteins. Corn endosperm zein is of relatively low biological quality for non-ruminants, because it is deficient in lysine and tryptophan. Zein is a heterogeneous protein and therefore several genes are responsible for its structure. The DNA sequence of one or more of these genes has been decoded. Alteration of that sequence so that lysine and/or tryptophan is substituted for less valuable amino acids may be possible. Alternatively, man-made DNA, i.e. artificial genes, might be created and made a part of the cell so that they would produce proteins of specific amino acid content.

For the sake of diversion, let us consider even further extravagencies in genetic engineering. There is little *a priori* evidence that animal genes cannot function in plant cells. In fact, one would expect that they could. If a gene that codes for a particular growth hormone were identified and located (and these techniques now exist), that DNA sequence theoretically could be transferred into a cell of corn or alfalfa, for example. If it were able to function to produce the hormone, presumably that hormone would be a component of the plant. One can imagine that made-to-order feeds might be bred for special purposes, or that inexpensive recovery of these foreign proteins from plant debris might be possible.

Let us now return to a more realistic consideration of what genetic engineering might accomplish in the not-too-distant future. Culture of tissue in artificial media and the subsequent regeneration of plants from it has been successfully carried out in several species including corn. It is possible to select among millions of cells for the capacity to survive in the presence of toxic substances, for example toxins produced by pathogens or to weedicides. The regeneration of plantlets from surviving cells offers the possibility that they too will possess resistance.

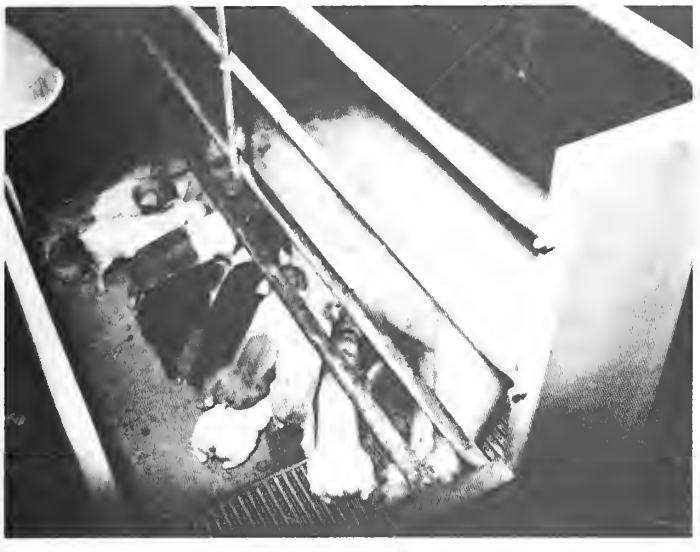
LITERATURE CITED

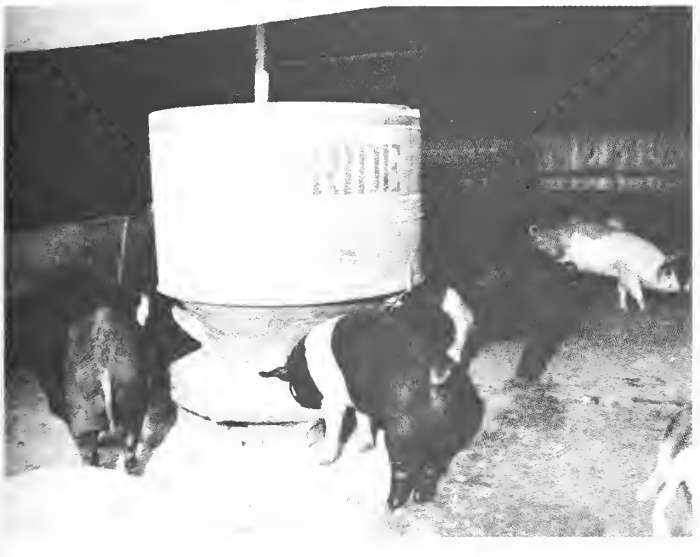
Russell, W. A. 1974. Comparative performance for maize hybrids representing different eras of maize breeding. Proc. 29th Annual Corn and Sorghum Research Conference. Am. Seed Trade Ass'n, Washington, D.C.

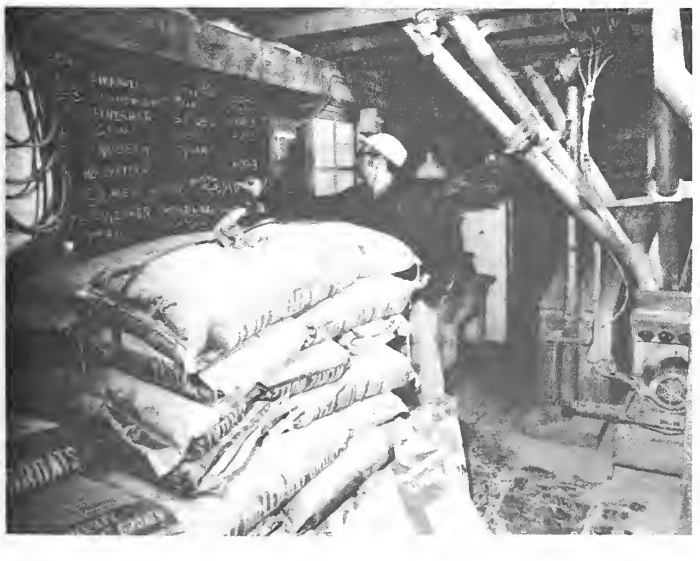
Duvick, D. N. 1977. Genetic rates of gain in hybrid maize yields during the past 40 years. Maydica XXII:187-196.

Hallauer, A. R. and J. B. Miranda. 1981. In Quantitative Genetic in Maize Breeding. Iowa State University Press.

Dudley, J. W. and R. J. Lambert. 1969. Genetic variability after 65 generations of selection in Illinois High Oil, Low Oil, High Protein and Low Protein strains of Zea mays L. Crop Science 9:179-181.







University of Illinois

PORK INDUSTRY CONFERENCE

December 1-2, 1983
College of Agriculture
Department of Animal Science
Cooperative Extension Service
Agricultural Experiment Station
University of Illinois at Urbana-Champaign

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WELCOME TO THE PORK INDUSTRY CONFERENCE! It is a pleasure to have you attend.

Commercial swine production has become industrialized. It is now a business in which experts in animal production, more than ever, must be concerned about finance, personnel management, labor efficiency, quality control, industry image, government regulation, etc. In other words, it is big business which requires constant awareness of advancements in technology, changes in organizational management and government policies and changes in the factors of production. Only those who manage to keep informed in all facets of the business will be likely to operate profitably.

This conference is designed to assist swine producers to stay abreast of developments vital to their business. We hope that it will be recognized as and become a part of a continuing education program for professional, large-scale swine producers.

You are here because you need and value information to apply to your business. We hope to provide it.

We will be interested in your suggestions to make future conferences of maximum benefit to you.

D. E. Becker, Head
Department of Animal Science

The Department of Animal Science at the University of Illinois

THE DEPARTMENT OF ANIMAL SCIENCE IS CONCERNED WITH THE MULTIDISCIPLINARY ACTIVITIES associated with the production, care, and utilization of animals useful to mankind. It includes primary fields of study in behavior, genetics, environmental physiology and management, meat science and muscle biology, nutrition, and reproductive physiology. Beef cattle, horses, poultry, sheep, swine, and various companion and laboratory animals are studied to assist animal producers and owners to obtain more efficient performance, more economical production and other such improvements which ultimately benefit the general public.

Our staff includes about 140 people of whom 50 are academic staff members; 40 are nonacademic staff members in the offices, research farms, and laboratories; and about 50 are part-time teaching or research assistants who are pursuing graduate study. Several of the senior academic staff members have received national and international recognition for their accomplishments.

The work of the Department is divided into extension, research, and teaching. Extension or off-campus teaching is handled by 10 staff members. Each extension specialist conducts seminars, clinics, field days, etc., for livestock or poultry producers. Their primary work is to apply new research findings to the business of animal production or product processing.

Over the years the Department has had very active and productive research programs. Some of the more notable research accomplishments include the discovery of the value of antibiotics in livestock feeding, the elucidation of the amino acid needs of swine and poultry, the utilization of inorganic nitrogen by ruminants, the development of simplified corn-soybean meal rations for swine feeding, and the development of confinement production technics, particularly the use of slatted floors. Current research studies pertain to such topics as recycling of animal wastes, decreasing prenatal mortality in gestating sows, the inheritance of blood groups in swine, exercise physiology in horses, alcohol treatment of soybean meal, factors affecting amino acid needs of domestic animals, and animal behavior.

With the recent increase in student numbers, the teaching load in Animal Science increased significantly. At present our Department provides instruction for about 2,800 students a year in the classroom, and staff members advise 400 undergraduate (mostly juniors and seniors) and 90 graduate majors. Our graduates have many opportunities for employment.

The future of the Department of Animal Science appears bright. Meat consumption and the use of animals for companionship and in recreational activities are high and will increase as the population increases in number. Such an expanding animal industry will require an educational program to produce well-trained animal scientists and research programs geared to produce new and improved technology.

University of Illinois
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Contents

Breeding for Larger Litters in Swine

CHRISTIAN LEGAULT

Among the numerous components of sow "numerical productivity" (number of piglets weaned per sow per year), prolificacy, defined as litter size at birth is generally considered to be the most important from an economic as well as from a genetic standpoint. However, in spite of the spectacular improvement of numerical productivity of sows observed in several countries during the last 10 years, litter size has remained constant during the last 20 years. Is this a failure of classical method of improvement and should we consider raising prolificacy a priority?

It is generally accepted that improving pig prolificacy through selection is a difficult task with little prospect of success because of low heritability. On the other hand, crossbreeding is known to be the most rapid way of improving litter size given a limited period of time. It is also well established that the improvement to be expected from crossbreeding cannot exceed an amount which depends on the heterosis effect.

A successful improvement of prolificacy of Sus scrofa has been realized over a long period of time. This is illustrated by recently published figures for average litter size: 4.5 for the French wild pig (Aumaitre et al., 1982) and 14.8 in a Chinese breed (Legault and Caritez, 1982). This difference of about 10 piglets gives an indication of the possibilities for genetic progress. The question of genetic improvement of litter size is receiving much attention in several countries and many reviews have recently been devoted to this subject (Johansson, 1981; Vangen, 1981; Hill, 1982; Ollivier, 1982; Bolet and Legault, 1982; Van der Steen, 1983). This will be the objective of this review with emphasis on some new approaches such as the development of "hyperprolific" strains and the use of some Chinese breeds.

WHY LITTER SIZE

General definitions

The number of animals weaned/year/dam or "numerical productivity" is regarded as one of the best estimates of reproductive performance of females in

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all domestic species (Ortavant and Thibault, 1971). This measure, proposed by Desvignes (1968) for measuring ewe productivity, has been adapted to swine (Legault et al., 1975).

The numerical productivity of a sow (P_n) can be expressed in its most general form as the following product:

$$P_n = O(1-e_m)(1-p_m)f$$

where:

O is the ovulation rate;
e_m is the rate of embryonic losses (from ovulation to farrowing);
p_m is the rate of piglet mortality (from birth to weaning);
f_m is the "apparent fertility rate" or reproductive rhythm represented by the number of farrowings/sow/year.

The "apparent fertility rate" or reproductive rhythm can be expressed either by year of reproductive life, or by year of presence of the sow in the herd. In the first case, the most classical one, reproductive rhythm is given by

$$f = \frac{365}{I} = \frac{365}{(G+L+I_{WF})} \quad , \quad \text{where :}$$

I is the interval between farrowings (days);
G is the length of gestation (days) ;
L is the length of lactation (days);
I_{WF} is the weaning - fertilization interval (days)

However, this definition which is satisfactory from a biological point of view, does not take into account two unproductive but expensive periods of the reproductive lifespan of the sow : the interval between 100 kg live weight and first farrowing (m_1) and the interval between last farrowing and actual culling of the sow (m_2). Numerical productivity can then be estimated as follows:

$$p_n' = \frac{N\,O(1-e_m)(1-p_m)}{m_1+(N-1)\,I+m_2} \times 365$$

where N represents the average number of litters weaned per culled sow. The difference between P_n and P_n' may be as large as 2-3 piglets/sow/year.

Given P_n, one can estimate the cost of the weaned piglet (p) as follows:

$$p = \frac{C}{P_n} + c$$

where: C represents the yearly cost of the sow (feed, labor, veterinary inputs, etc.), and c represents the cost of feed consumed by each piglet from birth to the beginning of the fattening period (25 kg). The effect of improving one

unit of P_n on cost of the weaned piglet is given by the derivative of p with respect to P_n. This is shown in Figure 1 for 5 values of C.

Among all the components of numerical productivity, the traits that deserve to be considered for breeding are litter size and its components (O, e_m and p_m), the weaning-fertilization interval (I_{WF}) and age at puberty because of its possible effect on m_1.

Relative importance of the components of sow numerical productivity

Average values of the main components of the number of piglets reared per sow per year of reproductive life (P_n), and per year of presence on the farm (P_n') obtained in 1977 in 3464 French commercial pig herds were analyzed (Legault, 1978) taking into account the size of the herd (4 classes defined in terms of the number of litters weaned during the reference period). Multiple linear regression equations showed that irrespective of the size of the herd, P_n' is determined by 4 significant variables in the following order: litter size at birth, rate of mortality from birth to weaning (p_m), farrowing-culling interval (m_2) and length of lactation (L). The first two variables accounted for 36 to 56% of the variance of: P_n'. This study suggested that efforts should be directed to increasing litter size at birth and to lowering mortality rate in suckling piglets.

Trends of components of numerical productivity

An analysis of the variation from 1972 to 1981 of 5 components of numerical productivity (number of piglets born, born alive and weaned per litter, lactation length, weaning-fertilization interval) was made on a total of 299, 464 litters born in 325 French commercial herds participating in the "National computerized program for analysis of on-the-farm sow records" (Noguera et al., 1983). Herds were classified into 4 regions to take into account climatic variation: Brittany, South, Center and North-East. Then, within each region, the data were subjected to an analysis of variance including 3 fixed effects, (parity, year-month of farrowing and year-herd effects) and 1 random effect (sow within herd) so as to obtain estimates of the year effects. The effects of the previous lactation length on current litter size at birth and weaning-fertilization interval were also studied.

In Brittany and in the South of France, litter size at birth decreased by about 0.4 pigs during the first 5 years but regained the initial level in the next 5 years (Figure 2). This variable was steady in the Center, while it decreased 0.5 units in the North-East during the same period. Litter size at weaning increased in all regions from 1975, with the exception of the North-East.

This variation may be partly explained by a decrease of about 9 days in lactation length from 1972 to 1976 in the four regions, followed by a decrease of about 3 days from 1977 to 1981. The number of piglets born per litter decreased on average 0.2 units with a 10-day reduction in the duration of the previous lactation. The weaning-fertilization interval decreased by about 3 days except in the North-East. These variations may partly be explained by that of lactation length.

Numerical sow productivity increased on average 0.23 piglets/sow/year from

1972 to 1981, with regions varying from 0.19 to 0.30 piglets/sow/year (table 1).

Table 1: Trends in numerical productivity of sows from 1972 to 1981 in four areas of France.

Region	Year										b
	72	73	74	75	76	77	78	79	80	81	
Brittany	18,55	19,15	19,45	19,41	19,46	19,86	20,32	20,54	20,79	21,02	0,25
South	18,12	18,79	19,14	19,22	19,59	19,81	20,41	10,76	20,84	20,79	0,30
Center	18,62	19,08	19,75	19,51	19,90	19,95	20,20	20,37	20,43	20,51	0,19
North East	18,96	18,90	18,67	18,56	19,22	19,38	19,63	19,69	20,85	20,12	0,19
Overall	18,55	18,97	19,25	19,16	19,52	19,73	20,13	20,32	20,72	20,61	0,23

b: Linear regression coefficient, P_n/year

This study showed that the increased sow productivity observed in the last 10 years was mainly due to new herd management systems leading to an accelerated rhythm of reproduction. As this situation is now becoming stabilized for biological and economic reasons, future advances should be obtained by increasing sow prolificacy and piglet survival rate.

The conclusions of this study are in agreement with those of a previous one based on selection herds of the Large White and French Landrace breeds. Numerical productivity increased by about 3 piglets/year in both breeds in the course of the 10 year reference period, while litter size at birth remained constant (fig. 3). In this specific study, it was estimated that 40% of this progress was due to earlier weaning, 35% due to a decrease of the weaning-fertilization interval, and 18% due to increased survival of piglets from birth to weaning.

THEORETICAL ASPECTS

Genetic parameters

We do not intend to make a complete review of the very numerous estimates of heritability of litter size available in the literature (e.g., Johansson, 1981; Vangen, 1981; Hill, 1982; Ollivier, 1982; Bolet and Legault, 1982). We observe a very wide range of estimates probably due to sampling or environmental reasons. A value of .10 is accepted; estimates are generally smaller when calculated from daughter-dam regression than from paternal half-sib correlation (Table 2). Repeatabiity of litter size is about .15 at birth and .13 at weaning. Animals born in large litters have a reduced share of maternal resources before and after birth, and this environmental "handicap" may tend to reduce their subsequent body and litter size. There is good evidence in mice of a negative environmental correlation between litter size of dam and of daughter, and experiments with pigs have indicated that gilts born in litters artificially standardized to a small number have large litters subsequently (see Robison, 1981, for a review). Robison has suggested that heritability estimates of litter size from dam-daughter correlations, are, therefore, biased too low. Selection for litter size might be more effective if environmental

Table 2. Average of estimates of heritability of litter size available in the literature

Method*	Average estimate**	Litter size	
		Born	Born alive
A	1	0.12	0.09
	2	0.08	0.08
B	1	0.23	0.18
	2	0.15	0.11

*A : daughter/dam regression (26 estimates)
B : Paternal half-sib correlation (12 estimates).
**1 : mean of estimates,
2 : mean of estimates weighted by the number of pairs or litters per estimate.

effects were reduced by standardizing litters at birth. In table 3, estimates of heritability of litter size are presented by parity.

Genetic correlations between sizes of litters farrowed at successive parities are not equal to 1 (TABLE 4). Recent studies (Vidovic, 1982; Bolet

Table 3. Estimates of heritability of litter size, by parity.

Author	Number of pairs	Variate	Heritability ± Standard error: Regression by parity		Regression of the litter i on the birth litter	
ALSING et al. (1979)	1001	Total born	1	32 ± 8	1	4 ± 6
VANGEN (1980)	821	Total born			1	9 ± 7
	517				1+2	44 ± 11
VENEV (1980)	402	Born alive	1	17 ± 14	1	-14 ± 12
	279		1+2	30 ± 14	1+2	15 ± 12
NITTER et al. (1981)	--	--	2	24 ± 16	1	34 ± 15
			1+2	35 ± 18	2	-9 ± 20
					1+2	21 ± 10
OLLIVIER and BOLET (1981)	572	Total born	1	9 ± 10	1	9 ± 10
	315		2	5 ± 12	2	-10 ± 12
			1+2	2 ± 15		

*From Bolet and Legault (1982)

and Legault, 1982; Johansson and Kennedy, 1982) seem to indicate that the genetic correlation between first and other litters is quite low, whereas

correlations involving other parities are rather high and close to 1.

There is abundant data reviewed by Sellier (1976) indicating substantial heterosis for reproductive traits such as litter size, embryonic and pre-weaning survival, weaning-fertilization interval and age at puberty. Heterosis for litter size in pigs is, on average, close to 11% relative to pure breeds. This results from an 8% maternal heterosis due to using crossbred sows, and a 3% direct heterosis due to using sires from a third breed. Corresponding figures are, respectively, 3 and 0% for ovulation rate, and 11 and 6% for litter size at weaning (Table 5).

Table 4. Genetic parameters of litter size, by parity

Parity	1	2	3	4	5
1	0.09	0.72	0.48	0.06	-
	-	0.99	1.00	0.32	0.84
	0.10	0.52	0.89	-	-
2	0.12	0.08	2.38	0.58	-
	0.10	-	0.82	0.69	0.55
	0.12	0.09	1.08	-	-
3	0.09	0.17	0.02	1.99	-
	0.15	0.14	-	0.63	0.77
	-	-	0.08	-	-
4	0.08	0.14	0.19	0.11	-
	0.10	0.18	0.08	-	0.98
	-	-	-	-	-
5	-	-	-	-	-
	0.12	0.12	0.18	0.21	-
	-	-	-	-	-

*Genetic and phenotypic correlations above and below the diagonal, respectively. Heritabilities on the diagonal.

**In each cell, estimates are 1: total number of pigs born (Bolet and Felgines, 1981; 2: number of pigs born alive (Vidovic, 1982); 3: Johansson and Kennedy (1982).

Table 5. Average values of heritability (%) and of heterosis effects (%) for some reproductive traits in Western breeds of swine.

Trait	Heritability (h^2)	Repeatability (r)	Heterosis	
			Direct	Maternal
Ovulation rate	30	High	0	3
Litter size at birth	10	15	3	8
Litter size at weaning	8	13	6	11
Age at puberty	30	-	0	8
Weaning-fertilization interval	3	8	0	16

However, the amount of heterosis expected depends on the particular breeds or strains involved and we often have inadequate information about this parameter for specific environments. Several studies have investigated the relative value of different crossbreeding systems (two-way, three-way, rotational crosses, etc.) taking into account the merit of each breed for both production and reproduction traits (Sellier, 1976; Wilson and Johnson, 1981). A well known advantage of crossbreeding is to avoid inbreeding depression which can reduce litter size at weaning by 0.34 and 0.23 pigs/per 10% of inbreeding in the progeny and in the dam, respectively (Hill, 1982). Usually, the rate of increase of inbreeding is near 0.5 %/generation so its effect can be considered negligible in practice.

Theoretical rate of response to selection

Expected response to selection for litter size can be calculated using classical formulae of quantitative genetics. These aspects have been discussed by Hill (1982) and Ollivier (1982). A simple mass selection program can be applied with a generation interval of 1 year, with all replacements of both sexes being selected from the first litter. Picking boars from the top 2.5% of first litters; and females from the top 25% yields a theoretical rate of progress of 0.25 piglets/litter/year. A similar theoretical progress can be expected from selection based on the sum of the 2 first litters, with a selection rate of 10% for males and 30% for females, with all replacement males and females being also chosen in first litters. These estimates are obtained using values of 0.10, 0.15 and 2.8 for heritability, repeatability and standard deviation of litter size, respectively. We emphasize that this theoretical gain is rather encouraging and quite comparable to what can be expected from selection for more heritable traits such as growth or backfat thickness.

Under field conditions, we must be more realistic: with a generation interval of 2 years and a selection rate of 50% only applied to females, the expected rate of progress is 5 times lower (0.05 piglets/litter/year).

Usually, the breeder can vary the number of litters (n) upon which selection is made; this increases the selection intensity, the selection accuracy and the generation interval. Ollivier (1974) has shown that an optimum balance between the three above mentioned factors is realized when n=2.

Theoretical estimates of genetic progress due to mass selection are based on the assumptions that successive litters of each sow are expressions of the same characteristic (thus having the same heritability) and that genetic correlations between them are 1. A more realistic approach would be to consider each litter as a different characteristic and to apply selection index theory using the genetic parameters of Tables 3 and 4.

If the breeding goal relates the "age pyramid" of the sows, e. g., each parity being weighted according to its frequency in the population, it appears that the usual criterion which gives the same weight to each parity is quite close to the optimal index. The loss in efficiency is less than 2% when successive litters are assumed to be genetically idnependent (Tartar and Bolet, 1983). Anyway, the expected response to selection is reduced relative to the

predictions described above.

SELECTION EXPERIMENTS

Direct selection

It is interesting to discuss results obtained from actual selection schemes, conducted either in experimental herds or under field conditions. Because of the cost of experimentation with large animals, it is not surprising that data about selection for litter size in pigs are very limited. On the other hand, results from experiments with mice are relatively abundant (see Joakimsen and Baker, 1977, for a review). With 5 lines of mice selected for high litter size, and a number of generations ranging from 11 to 20, the average total response obtained was 3 pups, representing a 35% progress. In all experiments, this gain in litter size was explained by an improvement of ovulation rate, which was enhanced in two cases by a reduction in prenatal losses.

Only two pig experiments similar to the above ones with mice appear to have been reported. The first one was started in 1965 in France and results of the first 10 generations have been published recently (Ollivier and Bolet, 1981). The positive response observed over the first 5 generations (0.15 piglets/litter per generation, Ollivier 1973) was not maintained in the following ones, and the response observed over the whole experiment (including the 11th generation as shown in figure 4) was nil. Among the reasons which may be given for this lack of response, two appear to be most relevant: failure to realize a high selection intensity, and a lower than expected heritability (h^2=0.02). This was estimated from daughter-dam regression for the selection criterion used which is the total number of piglets born over the first two parities. However, a positive trend in ovulation rate was observed, though not significant (figure 5). There was also some positive response in number of surviving embryos recorded on the third liter. In a second experiment, Rutledge (1980) reported on the first 2 generations of selection for litter size in 2 lines, one with litter size reduced at birth to about six pigs and the other unstandardized. While there was a response in the former (figure 4), more generations are needed to establish this in a conclusive manner.

Factors other than selection intensity, heritability and generation interval, can affect the response to selection for litter size. Several studies indicate the existence of maternal effects which may be negatively correlated (either genetically or environmentally) to the direct genetic effect, both in mice and in pigs (Vangen, 1981; Van der Steen, 1983). However, the influence that such effects may have on selection response over a long period of time is sometimes questioned. Standardizing litters at birth is a way of overcoming the difficulty due to maternal effects. In the French experiment with pigs, litter size was not standardized, but heritability was slightly higher for first parity (h^2=0.09 and 0.05, respectively, for the first and second parity). The comparison of prolificacy of F1 sows resulting from reciprocal crosses between the Meishan breed (13.2 piglets per litter at weaning) and the Large White breed (9.7 piglets per litter at weaning) showed a slight and not significant advantage for females raised in the largest litters (15.6 vs 14.6 piglets born per litter).

The plateau in litter size observed under field conditions (figures 2 and

3) is probably due to lack of actual selection applied to this trait. In fact, crossbreeding appears to be an attractive procedure because of its immediate effect. For example, about 20 years of selection (with a theoretical gain of 0.05 piglets/litter/year) are required to equal the improvement obtained after a single generation of crossbreeding.

The "hyperprolific line"

The method was presented and discussed theoretically by Legault and Gruand (1976), and consists in selecting boars in progeny of sows with extreme prolificacy, and then to backcross these boars to sows of similarly extreme prolificacy. By repeating this type of backcross several times, the boars' average genetic merit for prolificacy progressively reaches the genetic level of "hyperprolific sows" used in each generation. In figure 6, the selected sows produced an average of 14.4 piglets over 4 litters and their breeding value was 1.2 piglets over the population mean (10). With the generation interval (from boar to boar) being 1 year, the expected superiority of the daughters of "hyperprolific boars" after 5 years is nearly .6 piglets per litter in purebreeding, and nearly 1.4 piglets per litter in crossbreeding.

The efficiency of such a scheme depends on the size of the sow population screened for prolificacy. Successful screening thus depends on an extensive system of farm litter recording Such a system was implemented in France in 1970, and about 38% of the sow population is presently recorded (over 800,000 litters recorded in 1982). The first hyperprolific sows were detected in 1973, and from then on, 9 generations of boars were selected according to the scheme described above. The results so far obtained have been discussed by Legault et al. (1981) and by Bolet and Legault (1982).

Although "hyper-prolific" purebred progeny in first litters had an ovulation rate of 1.8 corpora lutea higher than the control mean of 14.5, the number of embryos alive at 30 days was only 0.08 higher than in the controls (mean of 9.51). A much larger difference in number of embryos at 30 days (.8) was observed in crossbred gilt progeny. Also, with limited data, numbers born from purebreds increased in second but not in first litters. It was suggested that the uterus of older or of crossbred sows have a greater "carrying capacity". These results illustrate a problem to which we shall return: differences among strains in ovulation rate may not be expressed in terms of litter size because of increased embryonic mortality.

In such a scheme, a very intensive selection can be applied (selection rate between .3 and 1%) and biases from maternal effects on gilts are reduced by using information on several parities. However, the older the selected sows, the larger their genetic lag for production traits. Consequently, systematic performance testing in favor of fast growth and low backfat thickness is advisable for young boars of the strain. A very similar method has been applied in Great Britain, and a significant improvement (about 1 piglet per litter) has been observed in first litters of crossbred gilts (Bichard and Tomkins, 1982).

This method may lead to a complementary improvement of the productivity of sows ranging from 1.5 to 2.5 piglets weaned per year. Main results so far obtained in France and England are summarized in Table 6.

Ovulation rate is a major component of litter size, and heritability estimates for this trait vary from 20 to 45% (see Bolet and Legault, 1982, for a review). We mentioned above that ovulation rate was improved through indirect selection on litter size (Ollivier and Bolet, 1981), or through the development of a "hyperprolific line" (Legault et al., 1981). In a selection experiment for ovulation rate (Cunningham et al., 1979), a marked direct response in ovulation rate was obtained (0.4 eggs per generation) but no correlated response

Table 6. Litter size and its components in progeny of "hyper-prolific" sows.

			Trait:Parity						
			Ovulation rate		Surviving embryos (30 days)		Litter size at birth		
Country	Source of data	Group	1	3	1	3	1	2	3
FRANCE	Research farm	H_1	16.3 (87)	18.0 (27)	9.6 (60)	14.0 (23)	9.6 (42)	11.3 (36)	-
		H_2	15.4 (7)	-	11.2 (60)	-	-	-	-
		C_1	14.5 (212)	16.5 (93)	9.5 (137)	12.5 (83)	10.1 (228)	10.6 (199)	-
		C_2	15.2 (104)	-	10.4 (75)	-	-	-	-
	Field data	H	-	-	-	-	9.7 (83)	11.0 (58)	12.0 (132)
		C	-	-	-	-	9.3 (364)	10.5 (214)	11.5 (250)
GREAT BRITAIN	Selection farms	H_1	-	-	-	-	9.9 (304)	9.9 (212)	-
		C_1	-	-	-	-	9.1 (575)	9.9 (319)	-
	Field data	H_2	-	-	-	-	11.3 (205)	11.5 (124)	
		C^2	-	-	-	-	10.4 (185)	10.3 (103)	

H_1: progeny of hyperpolific boars in purebreeding.
H_2: progeny of hyperprolific boars in crossbreeding.
C_1^2: progeny of contemporaries in purebreeding (control).
C_2: progeny of contemporaries in crossbreeding (control).

in litter size was observed after 10 generations of selection, in spite of a positive tendency over the first 4 generations. It should be noted that the response pattern for litter size in this experiment is similar to the one observed in the French selection experiment (Ollivier and Bolet, 1981; figure 5) although different selection criteria were used in the two experiments. In both experiments the increase in ovulation rate was apparently counter-balanced by a higher embryo mortality. Is that balance limited to first parities, as the results previously reported for "hyperprolific" boars would seem to indicate? In any case, such a balance would limit the effectiveness of selection for ovulation rate (figure 7).

Another criterion of potential interest is testis size, a character which together with ovulation rate responds to selection for litter size in mice (Joakimsen and Baker, 1977). With selection for testis size, ovulation rate was increased but no correlated response in litter size was obtained (Mafizul Islam et al., 1976). In pigs, Proud et al. (1976) reported a more rapid testicular growth in a line selected for high ovulation rate, which indicates a positive genetic correlation between the two characters, as in mice. Moreover, testis size is easy to measure on live boars and it is highly heritable; h^2=0.6 for testis measured by caliper (Legault et al., 1979). The genetic correlation between this trait and litter size is likely to be low; however, this correlation has not been quantified precisely.

OTHER ASPECTS

Boar effects and cytogenetics

Several studies indicate that the "service boar" significantly affects the litter size of his mates, both under artificial insemination (Skjervold 1962) and natural service conditions (Nielsen, 1969; Uzu, 1979). This is probably through the effects of viability genes which are transmitted to the embryo. This effect explains only 5% of the total variance in litter size under artificial insemination conditions. This percentage, however, is largely superior to that explained by the direct genetic effects (Ollivier and Legault, 1967). A particularly striking but relatively rare example of such an effect is given in the case of chromosomal abnormalities such as reciprocal translocations. A boar carrying this defect usually reduces by half the litter size of his mates, and also transmits the abnormality to half of his progeny as illustrated in Figure 8. Popescu (1982) reviewed this problem and Table 7 describes different types of translocations so far identified.

Consequences of selection for growth and carcass traits

Although it is important to know what effects selection programs for growth and carcass traits are likely to have on reproductive performance, relevant information is limited. Analyses of French data by Legault (1971) and of British data by Morris (1975), do not give entirely consistent genetic correlation estimates, and these had high standard errors. In general, genetic correlations were small, except for an unfavorable correlation between killing out percentage and litter size. However, genetic correlations between reproduction traits and growth rate, feed efficiency and leanness tended to be favorable (Hill and Webb, 1982). In a selection experiment in the USA for high and low backfat (Hetzer and Miller, 1970) there was no substantial change in litter size despite large changes in fatness. Several recent studies seem to

Table 7. The 14 reciprocal translocations identified in domestic pigs (*).

Number	Chromosomes involved	Country	Litter size reduction	Authors
1	t rcp (11p+; 15q-)	Sweden	56% 34%	Henricson, Backstrom (1964) Hageltorn, Gustavsson, Zech (1976)
2	t rcp (6p+; 15q-)	Belgium	100%	Bouters, Bonte, Spincemaille, Vandelplassche (1974)
3	t rcp (1p+; 6q-)	Yugoslavia	26%	Locniskar, Gustavsson, Hageltorn, Zech (1976)
4	t rcp (13q-; 14q+)	Sweden	50%	Hageltorn, Gustavsson, Zech (1976)
5	t rcp (6p+; 14q-)	England	100%	Madan, Ford, Polge (1978)
6	t rcp (4q+; 14q-)	France	43%	Popescu, Legault (1979)
7	t rcp (1p-; 16p+)	W. Germany	?	Forster, Willeke, Richter (1981)
8	t rcp (7q-; 11q+)	Sweden	50%	Gustavsson, Settergren, King (1982)
9	t rcp (9p+; 11q-)	Sweden	50%	Gustavsson, Settergren, King (1982)
10	t rcp (1p-; 8q+)	Sweden	?	Gustavsson, Settergren, King (1982)
11	t rcp (1q+; 14q-)	E. Germany	?	Golisch, Ritter, Schwerin (1982)
12	t rcp (7q-; 15q+)	France	45%	Popescu, Boscher, Tixier (1983)
13	t rcp (1p+; 14q-)	Sweden	?	Gustavsson, Settergren (1983)
14	t rcp (1q-; 17q+)	Sweden	?	Gustavsson, Settergren (1983)

(*) Personal communication of P. Popescu (1983)

indicate the existence of an unfavorable genetic correlation between backfat thickness and litter size (Johansson and Kennedy, 1983; Kersey et al, 1983; Laloe and Bolet, 1983).

There is a reduction of about 1 pig/litter in individuals which are homozygotes for the gene causing sensitivity to halothane anaesthesia (Hill and Webb, 1982). Elimination of the halothane gene by testing in breeds where it is at high frequency could lead to improvements in litter size.

The contribution of some Chinese breeds

The main characteristics of Chinese breeds of swin and the French experiment undertaken since 1979 with 3 of these breeds, Meishan (MS), Jiaxing (JX) and Jinhua (JH), were discussed in detail last year by Gianola et al. (1982). The latest available data comprising a total of 486 litters, all born on the Le Magneraud research farm, are presented in Table 8. These data are in very good agreement with previously published results (Legault and Caritez, 1983), and can be summarized as follows:

1) the high prolificacy of Meishan females, both at birth and at weaning was confirmed (14.5 and 13.2 piglets per litter respectively). This was not the case with Jiaxing females (11.0 and 9.8 piglets born and weaned per litter respectively);

2) the prolificacy of F1 MS and F1 JX females is exceptional and slightly better but not significantly, than in pure MS. (15.1 and 14.8 piglets born per litter respectively);

3) this high prolificacy of F1's is accompanied by an excellent mothering ability (12.7 and 13.0 piglets weaned per liter, respectively);

4) excluding JH crosses, the numerical productivity of F1 MS and F1 JX females is about 30% larger than that of European breeds. This is equivalent to 5-8 additional piglets weaned/sow/year;

5) it is suggested that foundation JX animals could have been related and that crossing could have led to recovery from inbreeding depression;

Table 8. Least-squares estimates of litter traits in different types of crosses between European and Chinese breeds*

Genetic type of the dam	N	Litter size		Litter weight (kg)		Food intake
		Birth	Weaning	Birth	21 days	(30 days kg)
LW	19	12.0 a*	9.9 a	15.6 c	58.3 d	163 e
LF	23	10.3 a	9.2 a	14.6 c	57.1 d	163 e
MS	93	14.6 b	13.2 b	15.9 c	58.0 d	103 b
JX	73	10.9 a	9.7 a	9.1 b	38.4 b	82 a
JH	28	11.2 a	9.6 a	7.4 a	30.2 a	74 a
MS.LW	28	14.7 b	12.1 b	20.7 d	65.9 e	128 d
JX.LW	21	14.2 b	12.7 b	13.7 c	61.3 de	132 d
JH.LW	14	10.8 a	9.3 a	10.7 bc	45.0 c	120 cd
MS.LF	22	14.0 b	11.6 ab	17.5 cd	64.0 e	125 d
JX.LF	26	15.3 b	13.1 b	15.6 c	63.6 e	128 d
JH.LF	13	12.2 a	10.4 a	12.4 c	48.3 c	114 c
LW.MS	49	15.6 b	13.0 b	18.2 d	68.1 f	124 d
LW.JX	13	16.0 b	13.6 b	17.2 cd	65.6 e	121 d
1/4 Chinese	63	11.9 a	10.1 a	15.3 d	57.1 d	114 c
European	42	11.0 a	9.5 a	15.1 c	58.3 d	163 e
MS	93	14.5 b	13.2 b	15.8 c	57.7 d	102 b
JX	73	11.0 a	9.8 a	9.3 b	39.1 b	82 a
JH	28	11.4 a	9.7 a	7.7 a	31.1 a	77 a
1/2 MS	99	15.1 b	12.7 b	18.6 d	67.6 f	124 d
1/2 JX	60	14.8 b	13.0 b	14.8 c	63.1 e	130 d
1/2 JH	28	11.3 a	9.7 a	11.2 bc	45.8 c	114 c
1/4 Chinese	63	11.9 a	10.1 a	15.2 c	57.7 d	112 c
Overall	485	13.2	11.4	14.5	55.1	114

N: number of litters; LW: Large White; LF: French Landrace; MS: Meishan; JX: Jiaxing; JH: Jinhua.
*Means with different letters differ significantly ($p<0,05$)

6) preliminary estimates of heterosis (%) involving reciprocal crosses between MS or JX with European breeds are (Bidanel, personal communication):

7) the largest litter weight at 21 days of lacation was observed in F1 MS.

However, heterosis estimates would be biased downwards because the European sows were nursing F1 piglets;

8) the European sows consumed 30-35kg more concentrate during lactation (30 days) than the F1 females. Taking into account the gestation period, the use of F1 sows leads to an economy of at least 100kg of concentrate/sow/year;

	MS x European		JS x European	
	Direct	Maternal	Direct	Maternal
Litter size at birth - 10		17	12	34
Litter size at weaning - 6		13	19	35
Litter weight at 21 days 7		18	19	34

9) the comparison of reciprocal crosses between MS or JX and European breeds suggests that the subsequent prolifcacy of F1 females raised in large litters is not compromised. This would justify using purebred Chinese breeds as maternal lines because the cost of producing F1 gilts would be lower.

GENERAL CONCLUSION

Do pig breeders really want to break the present apparent "plateau" in litter size? Under field conditions, selection pressure on litter size is very low when compared to that on growth and carcass traits. Moreover, crossbreeding and management advances as well as a reduction in preweaning mortality have all contributed very markedly to the improvement of numerical productivity of sows. Hence, an increase in litter size would appear as less urgent.

Some recent developments and research results suggest that we can be rather optimistic regarding the possibilities of increasing litter size in the near future. However, a few comments may be in order here:

- the "mouse model" has been copied too often, and basic or fundamental research on the pig has been neglected;

- much emphasis has been placed on theoretical aspects such as maternal effects, inbreeding depression, highly sophisticated selection indexes, while more simple and efficient methods have been discarded; and

- too many studies have been limited to first parity females, this being particularly true in the U.S.A.

Another potential solution would be to use modern computerized recording systems in order to detect exceptionally prolific animals and then to apply a very strong selection intensity. Using this approach it is then possible to develop a "gene pool" for prolificacy. The computer can also be very useful as an aid in eliminating abnormalities such as chromosomal translocations.

The high prolificacy of some Chinese breeds can also be used to speed up genetic progress for litter size, either through systematic 3-way or 4-way crosses with Western breeds, or by developing "synthetic lines" selected for

heritable traits such as growth rate and backfat. The efficiency of this system might be improved by combining the Chinese breeds with "hyperprolific" lines as mentioned above. All these methods could also be complemented by a search for major genes affecting reproduction and, particularly, litter size. It seems safe to conclude that prolificacy in swine is not refractory to genetic change.

LITERATURE CITED

Aumaitre, A., Morvan, C., Quere, J. P., Peiniau, J., Vallet G. 1982. Potential prolificacy and winter breeding in free-living wild sows. 14 èmes Journèes de la Recherche Porcine en France, Paris, 3-4 fèvrier 1982, 109-124, I.T.P., Paris.

Bichard, M., Tomkins, C. 1982. First results from the pig lines selected for increased prolificacy. (Personal communication).

Bolet, G., Legault, C. 1982. New considerations on genetic improvement of prolificacy in the pig. 2nd World congress on Genetics applied to Livestock Production, Madrid, 4-8 October 1982, 5, 548-567, Editorial Garsi, Madrid.

Bouters, R., Bonte, P., Spincemaille, J., Vandeplassche, M. 1974. Chromosomes of domestic animals. 2. Chromosomal abnormalities associated with reproductive disorders. Het chromosomenonderzoek de huisdieren. II. De afwijkingen in de geslachtschromosomen als oorzalejijke of begeleidende factor van onvruchtbaarheid. Vlaams Diergenees kunder Tijdschrift, 43, 85-99.

Cunningham, P. J., England, M. E., Young. L. D., Zimmerman, R. D. 1979. Selection for ovulation rate in swine: correlated response in litter size and weight. J. Anim. Sci. 48, 509-516.

Desvignes, A. 1968. Proposition de dèfinition de critères zootechniques et èconomiques en matière d'èlevage ovin. 19ème Journèe Etude, Fèdèration Europèenne de Zootechnie, Commission ovine et caprine, Dublin, May 22.

Forster, M., Willeke, H., Richter, L. 1981. An autosomal, reciprocal 1/16 translocation in German Landrace pigs. Eine autosomale, reziproke 1/6 translokation bei Deutschen Landrasse Schweinen. Zuchthyg., 16, 54-57.

Gianola, D., Legault, C., Caritez, J. C. 1982. Evaluation of Chinese swine germ-plasm in France. Proceedings of Pork Industry Conference, University of Illinois, December 2nd, 39-55.

Gustavsson, I. 1983. Reciprocal chromosome translocations and decreased litter size in the domestic pig. J. Dairy Sci., 66, (suppl., 248 [Abstr.]).

Hageltorn, M., Gustavsson, I., Zech, L. 1976. Detailed analysis in a reciprocal translocation (13q-; 15q+) in the domestic pig by G- and Q-staining techniques. Hereditas, 83, 268-271.

Henrickson, b., Backstrom, L. 1964. Translocation heterozygosity in a boar. Hereditas, 52, 166-170.

Hetzer, H. O., Miller, R. H. 1970. Influence of selection for high and low fatness on reproduction performance of swine. J. Anim. Sci., 30, 481-495.

Hill, W. G. 1982. Genetic improvement of reproductive performance in Pigs. Pig News Inform., 3, 137-140.

Hill, W. G., Webb, J. A. 1982. Genetics of reproduction in the pig. Proceedings 34th Nottingham Easter School: Control of Pig Reproduction. Eds. D. J. A. Cole and G. R. Foxcroft (In Press).

Joakimsen, O., Baker, R. L. 1977. Selection for litter size in mice. Acta Agric. Scand., 27, 301-318.

Johansson, K. 1981. Some notes concerning the genetic possibilities of improving sow fertility. Livest. Prod. Sci., 8, 431-447.

Johansson, K. Kennedy, B. W. 1982. Estimates of genetic parameters of fertility traits in pigs. 2nd World Congress on genetics applied to livestock production, Madrid, 4-8 October 1982, 503-508. Editorial Garsi, Madrid.

Johansson, K., Kennedy, B. W. 1983. Genetic and phenotypic relationship of performance test measurements with fertility in Swedish Landrace and Yorkshire sows. Acta Agric. Scand., 33, 195-199.

Kersey, Denise R. S., Irwin, K. M., Swiger, L. A., Plimptom, R. F. 1983. Selection for improved leanness in Yorkshire Swine. IV. Indirect response of the carcass, breeding efficiency and preweaning litter traits. J. Anim. Sci., 56, 551-559.

Laloe, D., Bolet, G. 1983. Corrèlations gènètiques entre les critères de reproduction et de production chez le Porc. (Unpublished data).

Legault, C. 1971. Relationship between reproductive performance and fattening and carcass characters in the pig. Ann. Gènèt. Sèl. Anim., 3, 153-160.

Legault, C. 1978. Analyse des composantes de la productivitè numèrique des truies. Ann. Zootech., 27, 457-470.

Legault, C., Aumaitre, A., Du Mesnil du Buisson, F. 1975. The improvement of sow productivity: A review of recent experiments in France. Livest. Prod. Sci., 2, 235-246,.

Legault, C., Gruand, J. 1976. Improvement of litter size in sows by the cration of a "hyperprolific" line and the use of artificial insemination: Theory and preliminary experimental results. Journèes de la Recherche Porcine en France, Paris, 201-208, I. T. P., Paris.

Legault, C., Gruand, J., Oulion, F. 1979. Developmenmt and genetic value of an "in vivo" method of estimating testis weight in young boars. 11èmes Journèes de la Recherche Porcine en France, Paris, 31 Janvier 1-2 fèvrier 1979, 313-322, I. T. P., Paris.

Legault, C., Gruand, J., Bolet, G. 1981. Results from purebred and crossbred use of a "hyperprolific" line. 13èmes Journées de la Recherche Porcine en France, Paris, 4-5 février 1981, 13, 255-260, I. T. P., Paris.

Legault, C., Caritez, J. C. 1982. First results of experiments with Chinese pigs in France. 1-Reproductive performance in pure and cross breeding with European breeds. 14èmes Journées de la Recherche Porcine en France, Paris, 3-4 février 1982, 127-136, I. T. P., Paris.

Legault, C., Caritez, J. C. 1983. L'expérimentation sur le Porc Chinois en France: I-Performances de reproduction en race pure et en croisement. Génét. Sél., Evol., 15, 225-240.

Locniskar, F., Gustavsson, I., Hageltorn, M., Zech, L. 1976. Cytological origin and points of exchange of a reciprocal chromsome translocation (1p-; 6q+) in the domestic pig. Hereditas, 83, 272-275.

Madan, K., Ford, C. E., Polge, C. 1978. A reciprocal translocation, t (6p+; 14q-) in the pig. J. Reprod. Fertil., 53, 395-398.

Mafizul, Islam A. B. M., Hill, W. G., Land, R. D. 1976. Ovulation rate of lines of mice selected for testis weight. Genet. Res., 27, 23-32.

Morris, C. A. 1975. Genetic relationships of reproduction with growth and with carcass traits in British pigs. Anim. Prod., 20, 31-44.

Nielsen, H. E. 1969. Growth rate, fertility and longevity of boars on different diets during rearing, and some results concerning fertility in sows. 375-Beretning fra forsognslaboratoriet, 47 pp.

Noguera, J. L., Felgines, C., Legault, C. 1983. Evolution de 1972 à 1981 des composantes de la productivité numérique des truies dans 325 troupeaux francais. 15èmes Journées de la Recherche Porcine en France, Paris, 2-3 février 1983, 15, 37-51, I. T. P., Paris.

Ollivier, L. 1973. Five generations of selection for increasing litter size in swine. Genetics, 74, Suppl. 2, part 2, 202-203 (Abstract).

Ollivier, L. 1974. Optimum replacement rates in animal breeding. Animal Production, 19, 257-271.

Ollivier, L. 1982. Selection for prolificacy in the pig. Pig News Inform., 3, 383-388.

Ollivier, L., Legault, C. 1967. The direct influence of the boar on the size and weight of litters obtained by artificial insemination. Ann. Zootech., 16, 247-254.

Ollivier, L., Bolet, G. 1981. Selection for prolificacy in the pig: Results of a 10 generation selection experiment. 13èmes Journées de la Recherche Porcine en France, Paris, 4-5 février 1981, 261`-268, I. T. P. éd., Paris.

Ortavant, R., Thibault, C. 1971. Introduction Bull. Tech. Inf., 257, 77-78, (special issue).

Popescu, C. P., Legault, C. 1979. A new reciprocal translocation t (4q+;14q-) in the domestic pig. Une nouvelle translocation rèciproque t(4q+;14q-) chez le porc domestique (Sus scrofa domestica). Ann. Gènèt. Sèl. Anim., 11, 361-369.

Popescu, C. P. 1982. Reciprocal translocations in pigs and consequences on their performance. Pig News Inform., 3, (3).

Popescu, C. P., Boscher, Jeannine, Tixier, Michèle. 1983. Une nouvelle translocation rèciproque t, rcp 7q-;15q+, chez un verrat "hypoprolifique". Gènèt. Sèl. Evol., (In Press).

Robison, O. W. 1981. The influence of maternal effects on the efficiency of selection: a review. Livest. Proc. Sci., 8, 121-137.

Rutledge, J. J. 1980. Fraternity size and swine reproduction. 1. Effect on fecundity of gilts. 2. Genetical consequences. J. Anim. Sci., 51, 868-874.

Sellier, P. 1976. The basis of crossbreeding in pigs: a review. Livest. Prod. Sci., 3, 203-226.

Skjervold, H. 1962. To what extent do boars affect the litter size? Meldinger fra Norges Landrukshogskole (166).

Tartar, M., Bolet, G. 1983. Application de la thèorie des indices de sèlection à des caractères rèpètès; exemple de la sèlection sur la prolificitè chez le Porc. Gènèt. Sèl. Evol., (In Press).

Uzu, G. 1979. Influence du verrat sur les principaux parametres de la productivitè du troupeau et sur la durèe de gestation. Ann. Zootech., 28, 315-323.

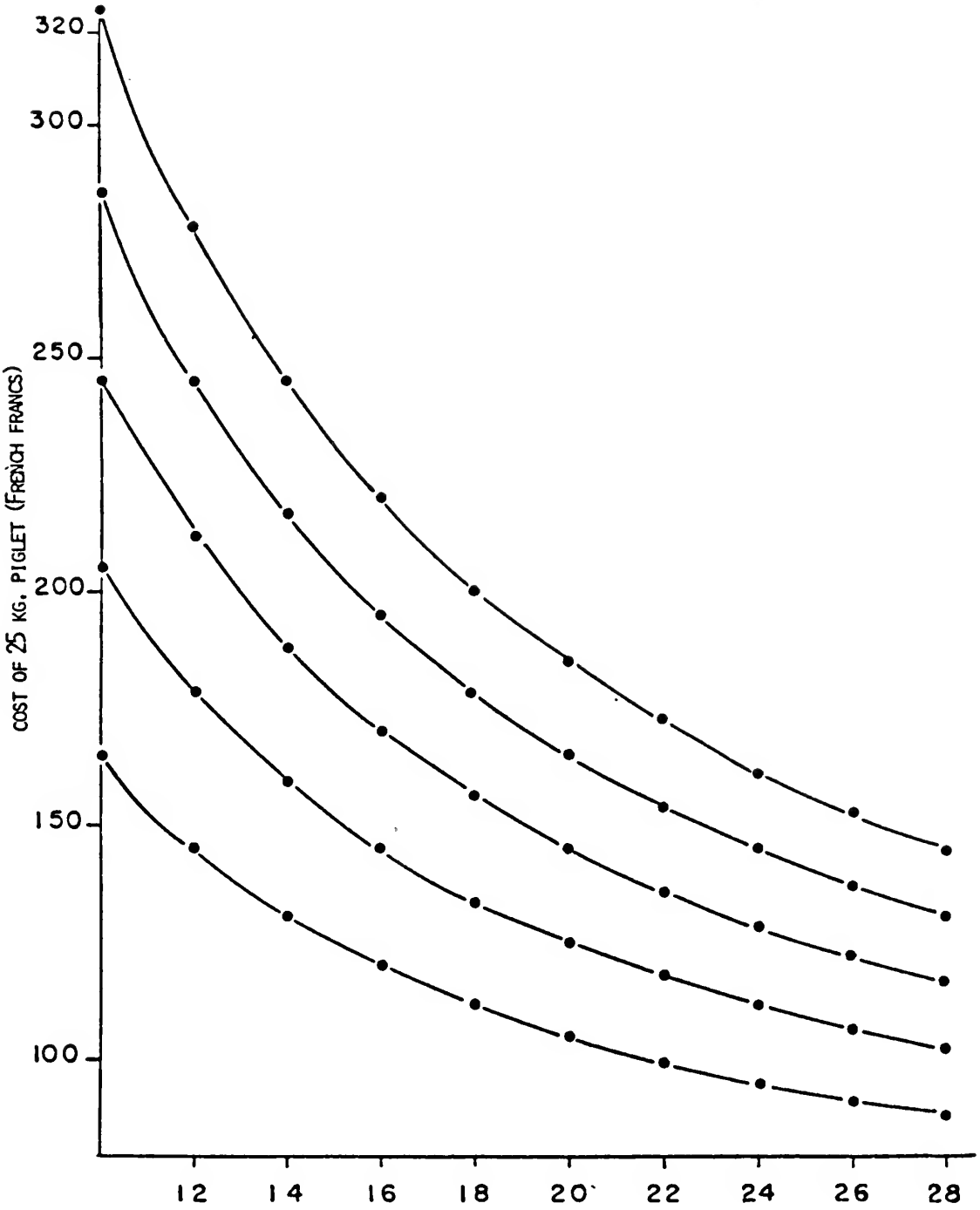
Van der Steen, H. A. M. 1983. Maternal and genetic influences on production and reproduction traits in pigs. Doctoral Thesis, Wagneingen.

Vangen, O. 1981. Problems and possibililities for selection for fecuindity in multiparous species. Pig News Inform., 2, 257-263.

Vidovic, V. 1982. Estimations of genetic parameters for reproductive performances in purebred and crossbred sows. 2nd World congress on Genetics Applied to Livestock Production, Madrid, 4-8 October 1982, 7, 288-292, Editorial garsi, Madrid.

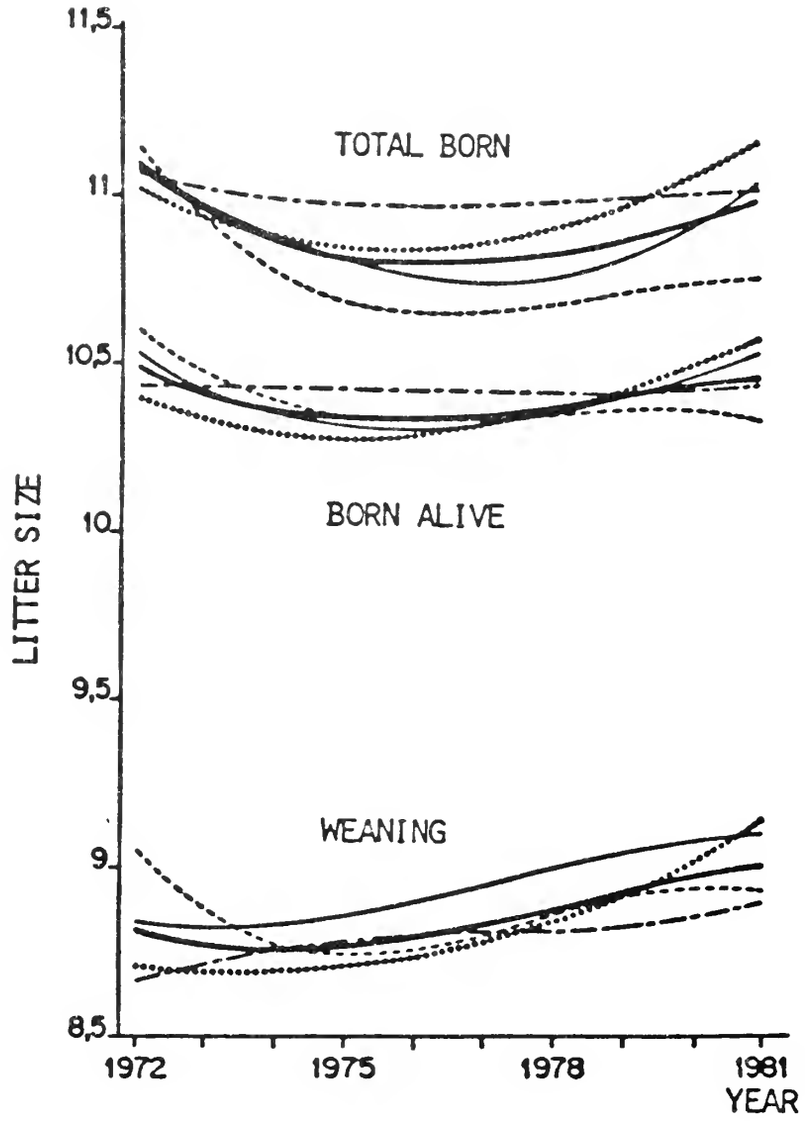
Wilson, E. R., Johnson, R. K. 1981. Comparison of three-breed and backcross swine for litter productivity and postweaning performance. J. Anim. Sci., 52, 18-36.

Figure 1



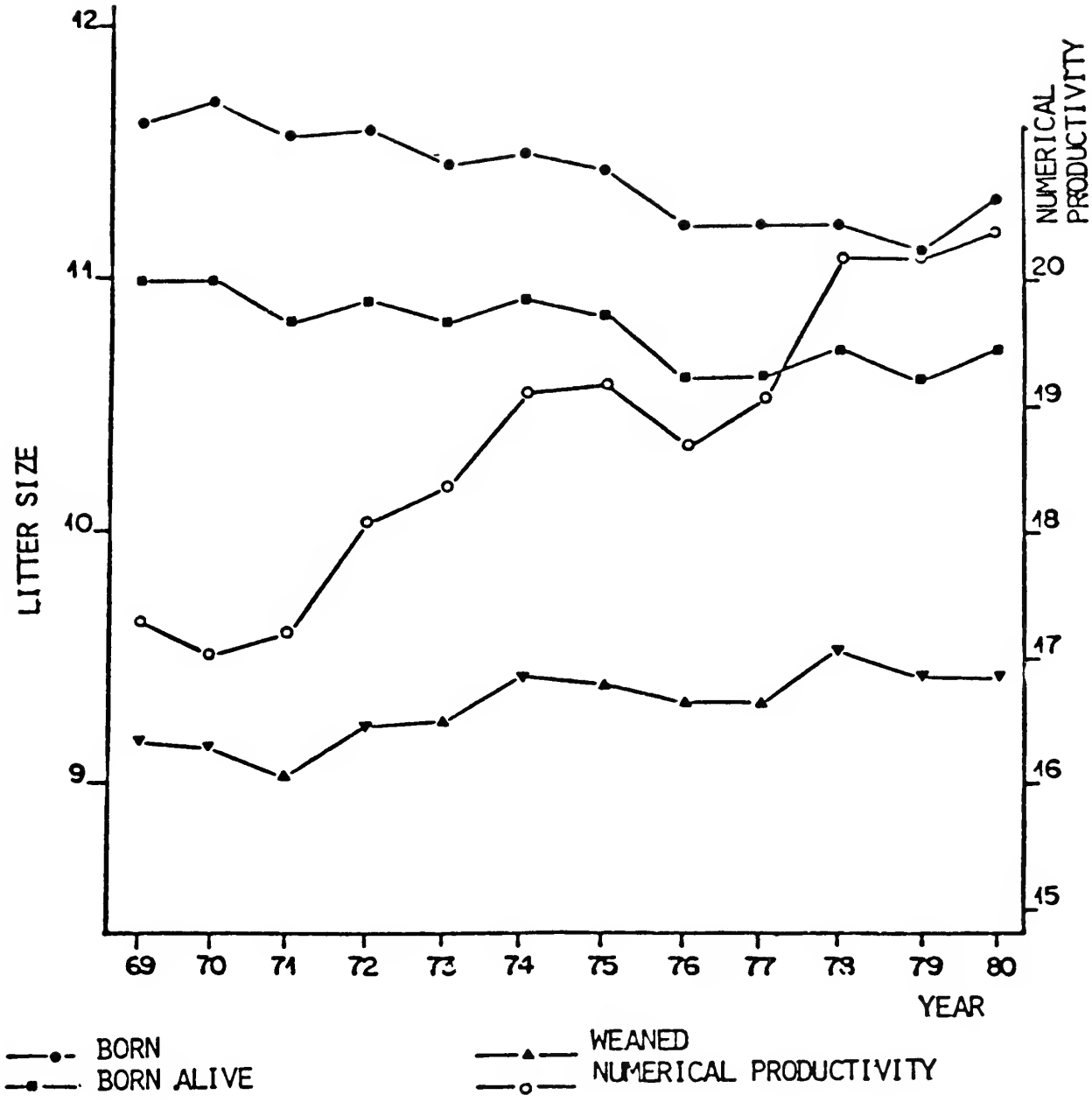
Variation of the cost of 25kg piglets with respect to numerical productivity for five values of C (yearly cost of the sow).

Figure 2



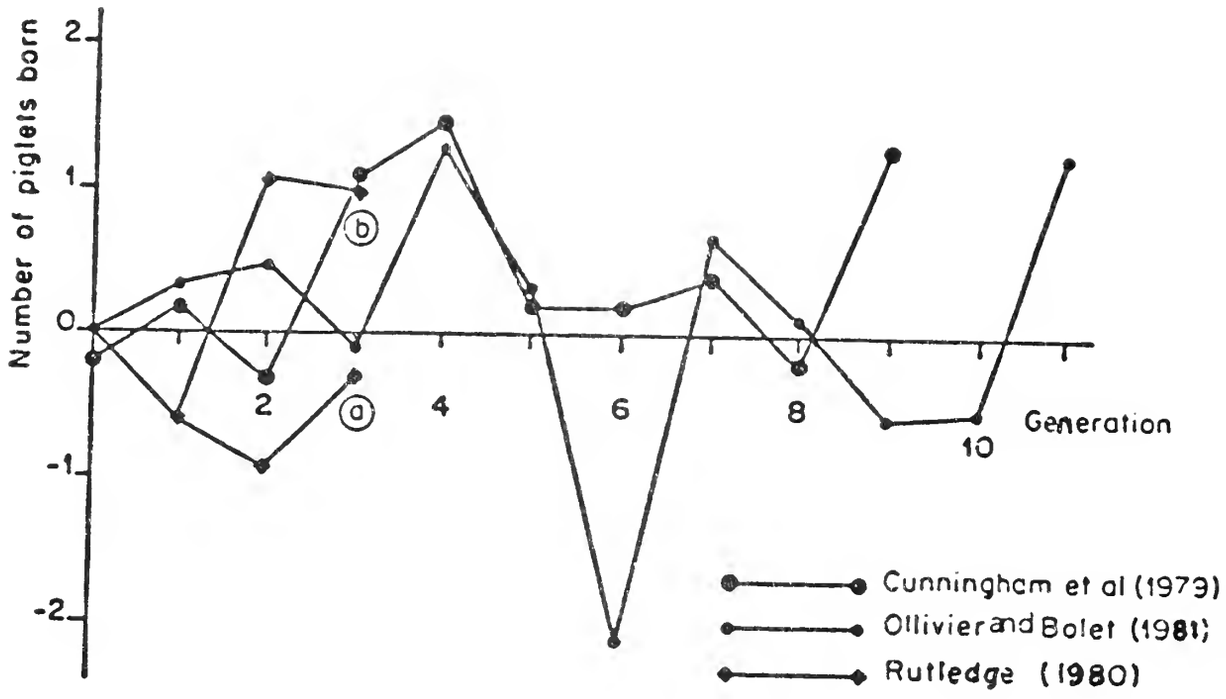
Year effects on litter size in 4 areas of France.

Figure 3



Variation in numerical productivity and prolificacy of Large White sows in France from 1969 to 1980.

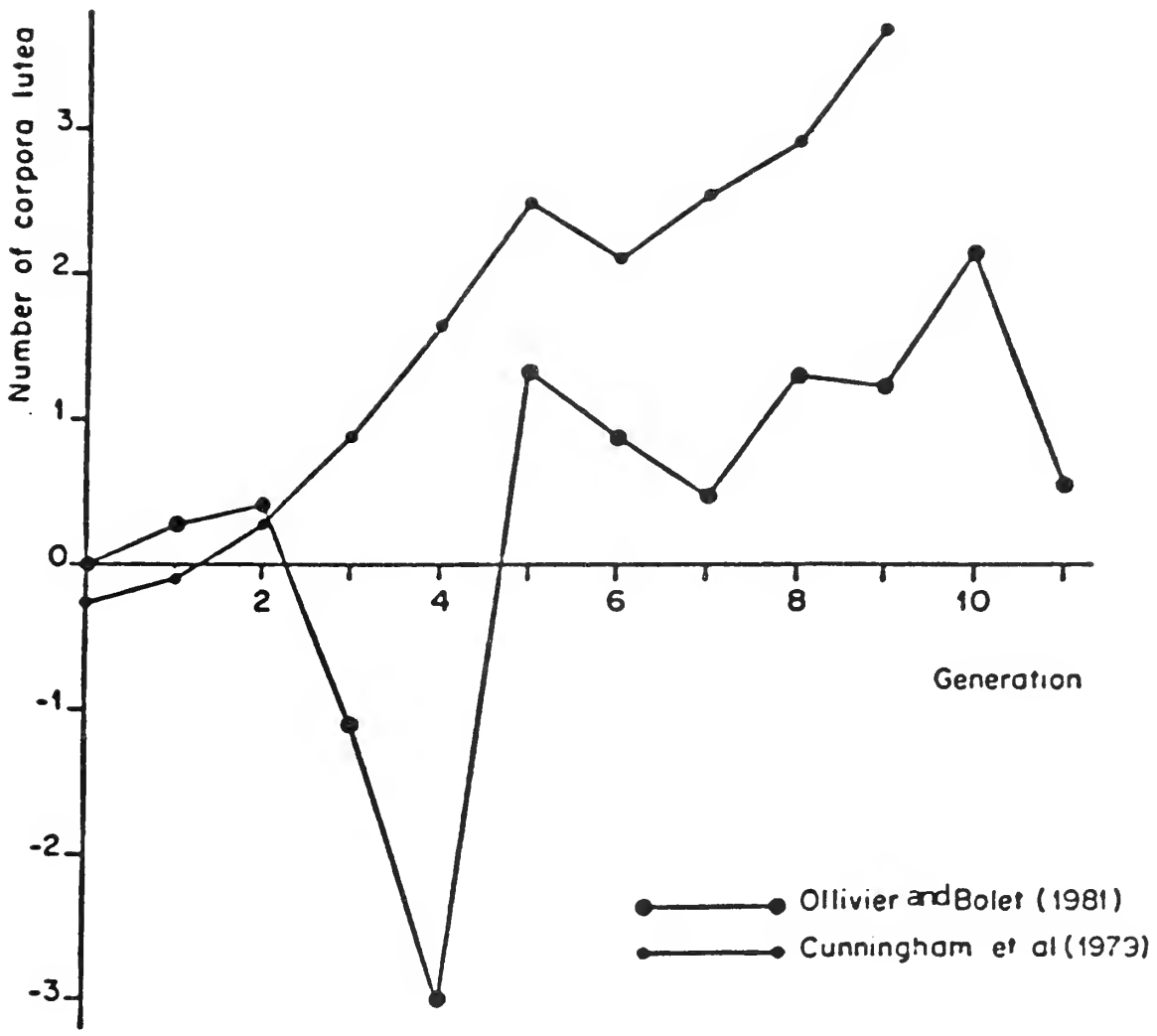
Figure 4



Difference between selected and control lines for number of piglets born in 3 selection experiments:

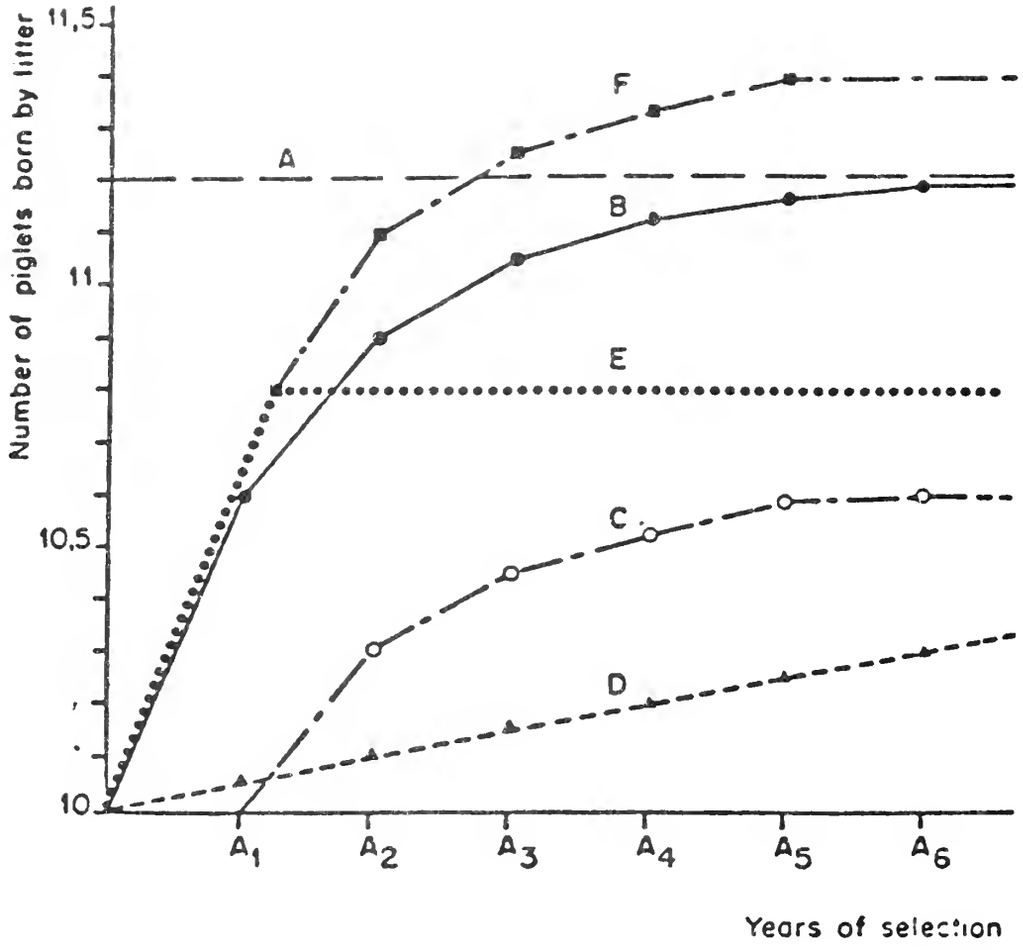
1) *CUNNINGHAM et al.: selection for ovulation rate in the first pregnancy.*
2) *OLLIVER and BOLET: selection for average number of piglets in the first and second farrowings.*
3) *RUTLEDGE: selection for number of piglets in the first farrowing.*

Figure 5



Difference in ovulation rate between selected and control lines in two selection experiments.

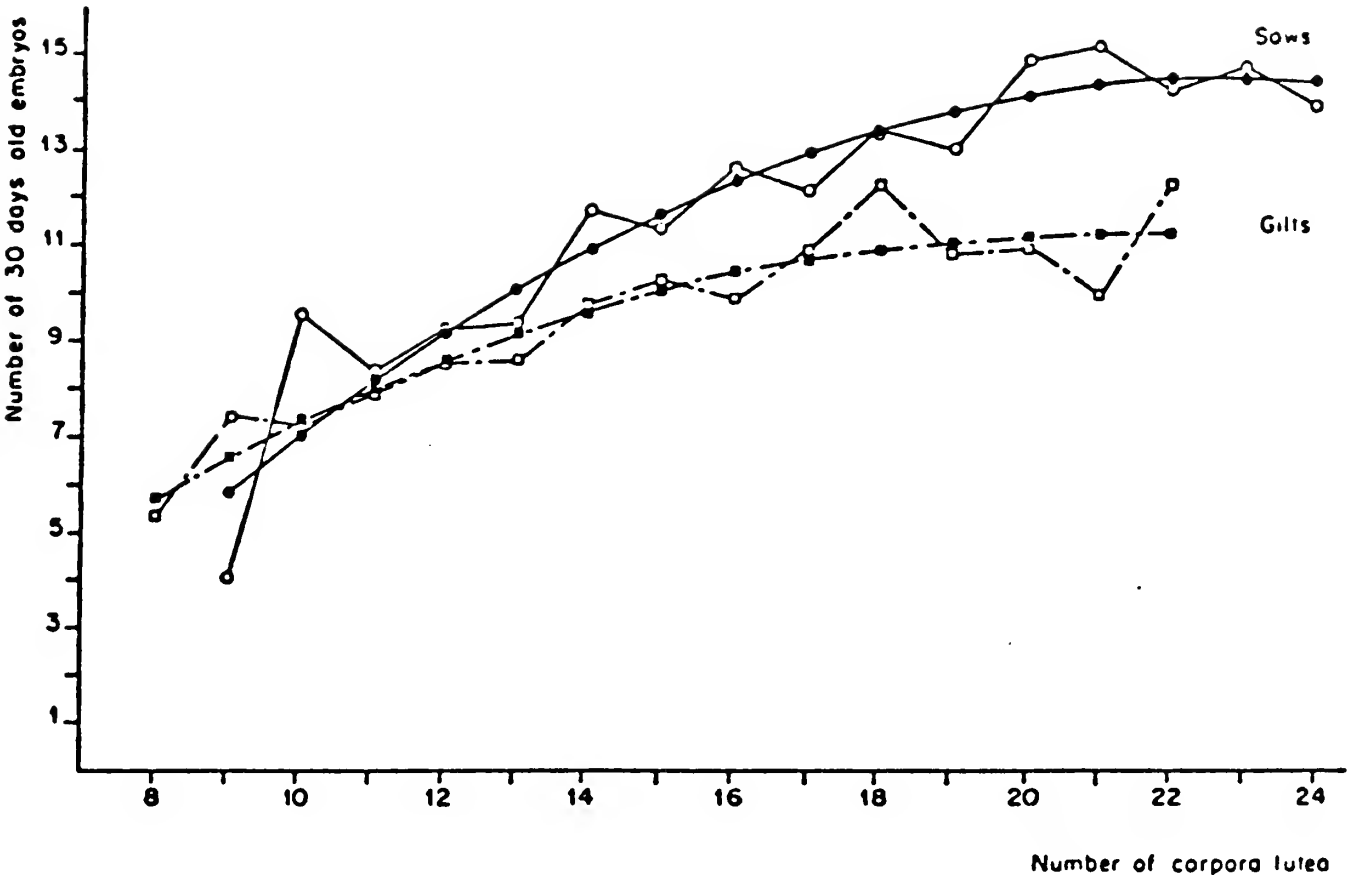
Figure 6



Theoretical variation in the number of piglets born per litter in purebreeding and crossbreeding, with or without use of a "hyper-prolific" line.

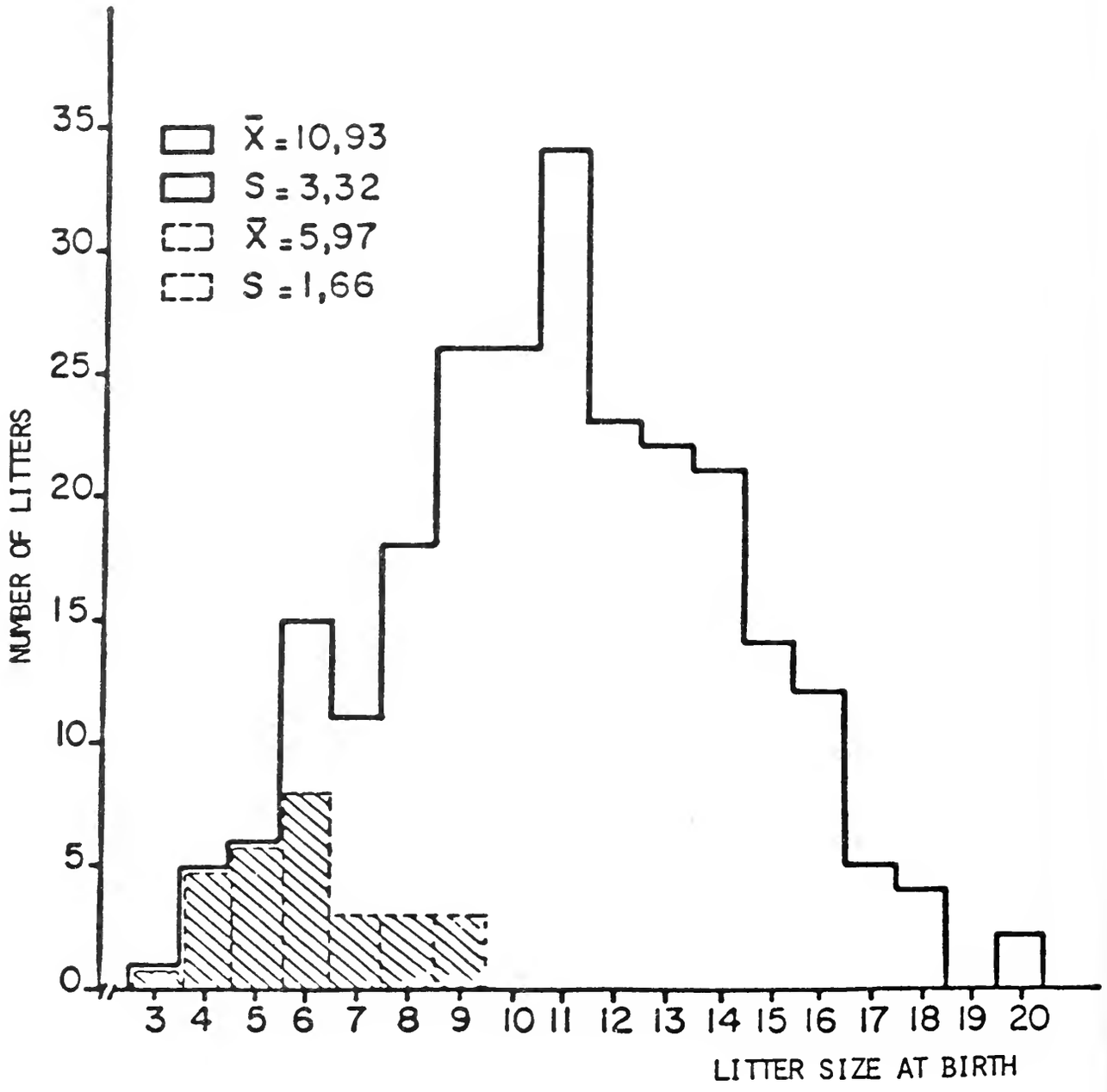
A: Genetic value of "Hyper-prolific" sows
B: Sons of A
C: Grand-daughters of A
D: Intra-herd mass selection
E: Crossbreeding without selection
F: Crossbreeding using B boars

Figure 7



Relationship between ovulation rate and number of 30-day old embryos in gilts and sows: observed and fitted curves.

Figure 8



Histogram of litters sired by T,15+ translocated boars compared to contemporary litters (POPESCU et al., 1983).

Organization of Pig Genetic Improvement in the USA: A Critical Viewpoint

DR. MAURICE BICHARD

INTRODUCTION

It should be made clear at the outset that this title was chosen by the Conference organizers. Few people are truly qualified to give an overall view of improvement in the entire US swine industry - perhaps none. My perspective is bound to be limited by my experience in the USA and contacts over the past 19 years - and I accept this. I do have a particular interest in methods of organizing genetic improvement, and direct experience of several. The organizers specifically asked me to be critical, where I felt this to be necessary, and I saw no reason to shrink from this request. My intention has been to present my own viewpoint in the hope that the audience may perhaps hear some angles with which they are unfamiliar, and to encourage a debate which could be the basis for future changes - in attitudes and in methods.

The approach I have adopted is to first sketch what we know about methods of creating genetic change, both in single herds and whole populations. Then to review what improvement programs I am aware of and measure these against the basic principles. I have had to glean a lot of my information from written articles and reports rather than first hand experience. It is not always obvious what schemes are in operation, what is being planned, and what is merely advice. I have therefore reacted to all of them in some measure.

THE CREATION OF GENETIC CHANGE

Scientists believe that performance in most traits which are economically important in the pig is under the joint control of many different genes and many environmental influences. Genetic improvement therefore involves introducing new and useful genes into a line or increasing the frequency of existing favourable genes or gene combinations. Put in more practical terms this means bringing in samples from different lines or breeds, selecting within existing lines, or crossing them in ways which maximize the productivity of the crossbred stock.

Selection Between Lines

Introduction of new lines which have already been improved is nearly always the most efficient method - where these do in fact exist, and where the veterinary

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regulations allow us to obtain them. Individual breeders have always done this, and all our present day breeds owe their origins to introductions from one or more different populations in the past. Considering how cost-effective such techniques can be it is surprising how little organized effort there is around the world to really examine the pros and cons of the available stocks. New breeds or lines can be used in different ways. In the UK the Swedish Landrace breed was imported in the early 1950's. It was first kept as a separate line for pure breeding, but also used in crossbreeding with the Large White. In addition it was used to improve an existing breed - the Welsh - by controlled immigration and then selection in the new synthetic breed (still called the Welsh).

Crossbreeding

The process of crossbreeding, either to produce fixed terminal crosses (e.g. A x B, or C x (A x B) or continuous rotational systems, is used to exploit two different phenomena. First it permits the combination of lines with complementary traits, and second it allows crossbred gene combinations which are advantageous for fitness traits. The crossbred may well perform even better than the best of the two parent lines.

The relative advantages of fixed and rotational systems are well known. Basically, while continuous rotational crossing may appear simple to organize within a commercial farm, it cannot quite exploit heterosis to the full. More seriously, it cannot so easily assign different roles to the different lines. A good crossing system should exploit complementarity by exploiting the strengths and hiding the weaknesses of each line. In practice this means using the lines best for sow productivity as components of a crossbred sow, and restricting other lines to components of a parent boar.

Within-Line Selection

The final method of creating genetic change, selection within lines, is usually considered first - even though it should logically only be employed when the other two methods have been fully exploited. Of course it is the only way to continue to make progress: selection between lines stops when there are no more good ones to choose; exploiting heterosis through crossing yields the maximum benefit within two or three generations and thereafter remains constant.

Again the theoretical framework for testing and selection is well known. Real economic progress is maximized when:

a) The chosen objectives have a large effect on commercial profits;

b) These traits can be measured accurately, before normal market weight, in both sexes, with a large amount of variation of which a good proportion is heritable;

c) Alternatively that another trait which is closely genetically correlated with the selection objective can be used as a selection criterion (e.g. ultrasonic backfat to predict total carcass fat);

d) As small a proportion as possible of the available candidates is chosen to become parents of the next generation, and these are the candidates with the highest predicted breeding values for the selection objectives;

e) Generations are turned over as quickly as possible according to the reproductive rate being achieved.

The obvious snag about trying to achieve near maximum rates of genetic progress is the cost. Measuring performance, particularly for individual feed intake, involves specialized buildings and equipment. Extra labour, some of it highly trained and motivated, is needed. Carcass evaluation is always costly. Recording and data processing systems usually prove either expensive or else inefficient! The disposal of reject boars may involve discounting apparent carcass values. Running purebreeding herds is inefficient by comparison with good cross-bred commercial units, and this is made worse when most females are disposed of just as they are coming into their most productive periods (3rd to 4th litters).

All in all it is very easy for the improvement programme to cost many times more than the benefits arising from genetically increased production within the herd. The rate of improvement in overall performance is unlikely to exceed 1 to 3% per annum. The only way these costs can be justified is the prospect of selling breeding stock to other pig producers at prices above those prevailing for slaughter hogs. The basis for this is that improvements, once made in the closed Nucleus or Elite herd, can be transferred to many other dependent herds if these routinely derive boars, gilts, semen or embryos from the Nucleus. This fact has been recognised for generations, and so we have a degree of specialization in any swine industry, between breeders and strictly commercial herds - whose output is solely feeder pigs or slaughter hogs. In this way the costs of genetic improvement are shared by the whole group of herds for their mutual benefit, with a cash transfer to the breeder usually in the form of the breeding stock margins he makes on boar sales. This system has a lot to recommend it, since breeding is delegated to a limited number of specialist breeders, while most herds concentrate upon the biological, physical and financial efficiency of production of lean meat.

Unfortunately some of the consequences of such a system do not appear to be well understood and it may be worth setting these out in some detail. For the past 30 years most US producers have bought boars of different breeds in some sort of rotational system. The aim has been to achieve a blend of traits from the available breeds and to cash in on heterosis in both sows and their offspring. Gilts have normally only been purchased to found a herd, to expand it quickly, or to bale it out when it has got into a mess with disease or breeding.

The first point to notice in such a system is the way in which the commercial herd is dependent upon the genetic level achieved in the seedstock herd, or herds, which supply it with boars. It is easy to show this dependence: if we denote genes originating in the seedstock herd as S, and those found in the commercial herd at any particular time as C, then the situation must develop as in Table 1.

Table 1. Dependence of commercial herd upon the seedstock herds from which it buys boars

	Genetic Origin	
Generation	Boars used in commercial herd	Output from commercial herd
0	S	C
1	S	1/2S + 1/2C
2	S	3/4S + 1/4C
3	S	7/8S + 1/8C
4	S	15/16S + 1/16C and so on

Thus after only a few generations of buying boars from a new source, a commercial herd is completely dominated by genes from that source, even though the owner may consider he has a 'closed herd' since he does not purchase gilts.

How then does improvement move from an improving source herd to one which buys its boars exclusively from that source? Again this is very simple to demonstrate. Suppose the breeder's herd manages, somehow, to achieve genetic progress in litter size at a rate of +0.15 pigs weaned per litter per generation of selection. The commercial unit taking boars of two breeds alternately from such an improving source, and running a strict two-breed rotation, might be expected to improve as in Table 2. Assume that it selected its replacement gilts completely at random, with no regard to the litter size (number weaned per litter) of their dam.

Table 2. Predicted results from buying in boars from a seedstock herd with an effective improvement program for sow productivity

Generation	Genetic mean in seedstock herd	Commercial Herd		
		Breed mean	Heterosis	Genetic mean
0	7.00	7.00	0.80	7.80
1	7.15	7.00	0.80	7.80
2	7.30	7.08	0.80	7.88
3	7.45	7.19	0.80	7.99
4	7.60	7.32	0.80	8.12
5	7.75	7.46	0.80	8.26
6	7.90	7.60	0.80	8.40
7	8.05	7.75	0.80	8.55

It can be seen that after an initial delay period, the genetic mean of the commercial herd is pulled forward, at the same pace as the seedstock herd, but lagging two generations of genetic progress behind it. The extra bonus of heterosis from the crossbreds keeps its absolute performance above that of the seedstock herd which is forced to work with purebred stock.

Thus there is an existing mechanism, though not the most rapid, for genetic change made in seedstock herds to be spread throughout the industry.

The example showed the commercial herd lagging by 0.3 piglets per litter, before considering heterosis, but this will depend upon:

a) The annual rate of improvement in the seedstock herd;

b) The rate of generation turnover in the commercial herd; and

c) The degree to which better or worse than average boars are purchased by the commercial herd.

Without going into more complex calculations it can be stated that other commercial considerations are likely to dictate the rate of sow herd turnover, and

that in the case of sow producitivity the purchaser has very little ability to purchase boars from one source with any assurance that they will be above average.

Now many commercial herd owners, and their advisers, appear to find this situation unattractive - in that they do not like to accept that their herd genetic level is in the hands of their boar source. They continue to believe that they can have an equal influence through their selection of homebred gilt replacements.

This is a complete fallacy and it is time that we all appreciated the fact. To demonstrate this we do not require a long selection experiment, or even a complex computer simulation. We may illustrate this by another simple example, using the same assumptions as before. Boars are purchased every generation from a source herd where no genetic progress is being made in sow productivity. Consider the case where all replacement gilts are regularly taken from the best one third of commercial sows, judged on the average of two litters. The genetic superiority of these selected sows would be predicted to be around 0.4 piglets per litter, and the expected consequences are shown in Table 3.

Table 3. Predicted results from selecting for reproductive performance in the commercial herd only.

Generation	Genetic mean in seedstock herd (no selection)	Commercial herd			
		Breed mean	Genetic superiority of dam	Heterosis	Genetic mean
0	7.0	7.00	0.4	0.8	7.80
1	7.0	7.20	0.4	0.8	8.00
2	7.0	7.30	0.4	0.8	8.10
3	7.0	7.35	0.4	0.8	8.15
4	7.0	7.38	0.4	0.8	8.18
5	7.0	7.39	0.4	0.8	8.19
6	7.0	7.39	0.4	0.8	8.19
7	7.0	7.40	0.4	0.8	8.20

The important principle here is not the size of the genetic improvement, but that unlike a gain originating in the seedstock herd, it is not cumulative. The only effect of continuous selection in the commercial herd is to raise reproductive performance over several generations by an amount equal to the average genetic superiority of the selected dams. Once that is achieved no more gain is possible, since the unimproved boars brought in each generation constantly cancel out further change. Nevertheless the selection must be continued or even that gain will disappear!

And of course if the superiority of the selected dams was not really genetic in origin, then no progress would be made at all.

The simple point which has been overlooked so often is that genetic levels in herds which regularly buy in boars are almost totally dependent upon the levels in the source herd.

The final principle which needs stating in this initial section concerns the effectiveness of culling within commercial herds. There is a widespread feeling among herd managers that they can increase their herd mean performance, even in the short term, by culling out those sows which have produced a well-below average performance in their first one, two or three litters. The improvement in mean litter size obtained in the next litter, as a result of culling, depends mainly upon the repeatability of the trait. Now the repeatability of a single record is only of the order of 15%, though this may increase if the mean of two or three records is used as the culling criterion. Calculations of the overall effectiveness of various culling schemes on herd mean productivity give extremely small increases (for example +0.01 to 0.06 piglets weaned per litter) for two main reasons. First, repeatability is low, and second, since gilt litters are smaller than sow litters, the increased proportion of gilt replacements required whenever there is voluntary culling (for any trait) depresses herd mean performance. It can be concluded that culling on the basis of litter size is not to be recommended (Strang and King, 1970).

IMPROVEMENT SCHEMES IN THE USA

So much for what we know about methods of creating genetic change in single herds and disseminating this to whole populations. All of the ideas and results reviewed above have been available for from 10 to 40 years, and should be well known to animal breeding scientists. In spite of this their implications do not always seem to have been appreciated by those who advise practical pig breeders.

No Overall National Scheme

The first thing that would strike many European observers is that there is no overall national scheme for swine improvement, nor has there ever been. By contrast countries like Denmark, Norway, Sweden, Ireland, France and Britain have all had such schemes for between 20 and 80 years. A single agency, which may be part of the Ministry of Agriculture, a quasi-government statutory body funded by a producer levy, or a farmer-controlled cooperative, has often controlled the main, or even the only, recognized improvement scheme in these countries. While the size and separateness of individual States would have been expected to have ruled out such a Federal scheme in the USA, there is not even a State scheme organized along similar lines. Without wishing to get into a strictly political discussion there are certainly strong arguments for a central agency operating so long as the total pig population is divided between thousands of small herds (breeders and commercial units) with none containing sufficient sows to permit a sensible within-herd testing and selection programme. Many of the smaller European nations have been in this situation, and some still are. Where other farms of cooperation were popular then it was obvious that this could be extended into the breeding area. Even in Western Europe, however, this situation is changing, notably in Britain where private schemes (the individual Breeding Companies) have now taken over from the Meat and Livestock Commission's Pig Improvement Scheme as the major source of the nation's seedstock, both boars and gilts. Since there are now plenty of people with the necessary knowledge of how to carry out effective improvement schemes, herd sizes are often large, and the tradition of private enterprise so well developed in the USA, there seems no reason to call for schemes organized by, for example, State Universities in the 1980's, no matter how useful these might have been had they existed back in the 1950's.

State Central Evaluation Stations

Formal testing programmes for swine begain in the USA in the 1950's, and 43 test stations were established in 27 States between 1950 and 1975. Many of these

had their origins in the national and regional stations operated in Denmark (since 1907) and studied by Professor Lush in the 1930's. Now the purposes of centrally testing pigs can be several.

1. Education. To acquaint and educate both breeders and producers with performance records and the idea that effective selection must be based upon measurement and not merely observation.

2. Promotion. To advertize the independently assessed performance levels of individuals or groups from particular breeders, as evidence of the superiority of their stock relative to their competitors.

3. Evaluation of herds. To permit a central agency to rank breeders' herds, and hence recognize some as superior - and perhaps give these status or actual assistance.

4. Evaluation of individual breeding pigs. To permit the comparison of performance levels by individuals from different herds in a situation of equal opportunity (insofar as pre-test environmental effects are unimportant). This can allow breeders to select individuals back into their herds after being ranked more efficiently than was possible had they remained in their herds of birth.

Now the European test stations achieved all four roles because they were an integral part of an overall improvement programme. The only breeders allowed to send stock to these stations were those who continually demonstrated both that samples of their herds performed well, and that their methods of selection etc., conformed to what was thought reasonable by the central agency. As far as I can see the US stations only perform the first two functions - education and promotion, and their genetic impact is therefore indirect at best.

The reason why the US test stations can only have a marginal direct genetic impact is a combination of limited size, and their 'open nature'. In 1981, 26 different stations tested a total of 5013 boars of eight different breeds (National Hog Farmer 15 July 1982). This is an average of fewer than 200 boars per station combined over the different breeds. Admittedly the Iowa station tested 663 boars (with feed efficiency) and the Ohio station 567, but individual breeders generally consign very few boars from each herd. Since there is no requirement for the top ranking boars to return to the testing herds, the system does not provide a means for each herd to identify its best candidates and then use them or their equivalents from other herds, as future boars. As has happened in many other countries - there is a testing programme without an improvement scheme.

Dr. Charles Smith demonstrated many years ago (1958) in his PhD thesis at Iowa State that an industry can only derive maximum genetic benefit from its limited control test facilities when their use is restricted to a pre-determined group of seedstock producers (Smith 1959, 1960). This principle was adopted in 1966 in Britain, and the 5000 boar test places at the four National stations were allocated to some 200 purebred breeders designated Elite, later reduced to only 60 in 1970 and called Nucleus herds. A similar system operated in Denmark. This may all sound too 'élitist' for the USA - but it has certainly been effective in Europe.

NSIF

The National Swine Improvement Federation is the organization which tries to coordinate central test station procedures. Its several broad aims are all to do

with encouraging the collection and use of uniform performance records. But I see very little evidence that it is prepared to grasp this particular nettle and really get States to organize improvement schemes with teeth, based on their central test stations. Perhaps it is already too late to attempt this. In which case the functions of these stations should be clearly identified as being quite different - and concerned mainly with education and example.

On-farm Testing

Few would argue that the actual improvement work of the future must take place in breeders' herds based upon testing done on their own premises. Only in this way can each breeder test sufficient animals to generate a satisfactory selection pressure among his selected herd sires, and avoid all the health risks of open public testing. But such on-farm tests provide no simple evaluation of other sources of stock, so they are really only suitable where the herd size is sufficient to eliminate the need for frequent and extensive purchase of unrelated boars from outside in order to contain inbreeding. And there is no way that on-farm testing can remain synonymous with home-spun, cheap or second best. 'On-farm' merely describes the location of the test, not its precision or complexity.

There is one recent analysis of an on-farm testing scheme which brings out very clearly some of the pitfalls to be avoided. Dr. Phil David's PhD thesis at Lincoln looked at the records of 18 members of the Nebraska SPF program. The results are not encouraging. Genetic progress as we all know depends upon generating positive selection pressures on heritable traits. In the Nebraska herds in the 1970's the low realized selection differentials for backfat, 140-day weight and numbers weaned suggest that other unrecorded criteria were regarded as more important in choosing replacement boars and gilts.

Approximately half the pigs were sired by purchased boars - but there was no evidence that these were superior to homebred boars. In addition the heritability of both fat and liveweight gain when estimated from the farm data was lower than from central-test data elsewhere. This may well have resulted from insufficient control by the breeders of their testing procedures.

As a consequence of all these features Dr. David was unable to demonstrate any important genetic trends in backfat or litter size over the decade, although it seemed that 140-day weight had increased at a modest rate.

Taking all these points together it might seem that the important task for NSIF and the State Central Test Stations is to use their facilities and influence to persuade seedstock herds to adopt efficient on-farm programs. This is a difficult task since they must start by talking to all 13,000 breeders who currently register stock, and many others who do not, knowing that the vast majority are never going to change and adopt really efficient methods. But they must try to be realistic. As I said in the early section, the costs involved in running a good improvement scheme are considerable, and are certainly in excess of the returns which an individual herd can expect from the more efficient production of feeder pigs or slaughter hogs. If the herds which incur these costs, and make a real success, are not then going to make an equal success of marketing breeding stock and thus spreading their influence, then they might as well not embark on the program in the first place.

Selection for Sow Productivity

The theoretical and experimental background to the genetic improvement of sow productivity traits will have been dealt with by Dr. Legault from his vast experience. It is not therefore necessary to repeat this, but rather to examine aspects of the subject in the USA. I believe that some of the same characteristics mentioned in the previous sections on the growth and carcass traits are again evident. Presumably the current great interest in sow productivity in the USA has its origins in two main factors.

First the move towards full confinement systems, in order to achieve greater control and raise output per man and per sow, has brought huge increases in the amounts of fixed capital invested in today's pig farms. In any financial evaluation of these new enterprises this extra capital appears as an ever larger element of fixed costs to be carried by each feeder or slaughter pig. The only way to reduce this item is by increasing sow productivity, and so sharing all those costs associated with a single sow over a larger number of progeny sold.

Second in importance has been the observation by many scientists and farmers that there are breeds or lines of pigs elsewhere, but particularly in Europe, which consistently outperform the stock traditionally kept on farms in the USA. For, example, the 766 herds recorded by the British Meat and Livestock Commission in the years to September 1982, contained some 144,000 sows which averaged 9.05 pigs weaned from each of 2.21 litters per year - giving an annual output of 20.0 pigs per sow per year (where the sow herd excludes unserved gilts) (MLC 1983).

One reaction has, of course, been to start using crossbred sows from the prolific European strains of Large White and Landrace. Where the housing, feeding and management conditions have been similar to those in Europe then performance has been quite equal. The results for 12,600 sows in 26 herds for 1982 (where all females are counted in the breeding herd) show an average of 9.23 pigs weaned from 2.33 litters per year - equivalent to 20.7 pigs per sow per year (PIC Production Control Data 1982). Other breeders have also been using Large White and British Landrace to upgrade their Yorkshire and American Landrace herds, presumably with improvements in proportion to the proportion of genes introduced.

But a different reaction from many has been to ask whether it might be possible to select within the traditional American breeds for improved sow productivity. I confess to being somewhat alarmed by the answers given by many US researchers and advisers. The confidence they show in the results of their theoretical predictions is certainly not yet borne out by any experimental work - anywhere in the world. There is of course the question of whether the heritability of a single record of litter size is around 10%, as believed by most European workers (Strang and Smith 1979) or around 20%, as favoured by some US geneticists (Swiger, Irvin and Isler 1978, Robison, 1981 and Bereskin, 1982). You may think this is merely an academic argument, in every sense of the word, but adopting the higher estimate does increase one's predicted progress by up to a factor of 2! In view of our lack of hard evidence that normal within-herd selection for litter size will produce the predicted responses I would have expected greater caution among advisers. In particular the NSIF might have tried to curb the enthusiasm of those who would like to think that manipulating sow productivity will be simple - but they have actually encouraged it. In their 1981 leaflet on Sow Productivity Indexing, they conclude "This SPI program can be utilized by both commercial and purebred producers and will provide the necessary selection pressure to result in a genetic improvement in sow productivity". Quite a claim in view of the world-wide evidence to date!

Let us accept for the moment that the SPI system might be effective in identifying sows whose genetic superiority can be transferred to their sons and daughters. We must then ask just which herds should be using it? As I understand it, several Universities are actively promoting the system, including the Ohio Sow Records Program (Swiger, Irwin and Isler 1978), Purdue (Jones, Cox and Byrd 1982) and the Kansas State Sow Productivity Program (National Hog Farmer 1980) and probably Iowa State and Michigan also.

But the ineffectiveness of selection for anything within commercial herds, which continue to buy in all their boars, was illustrated in Table 3. The plain facts are that virtually all their genetic change has to come in via these boars, since any genes in their own herds are constantly being replaced by those in the herds supplying boars - however closed they may be to female purchases.

I believe there is likely to be a lot less disappointment in our industry in a few years' time if those breeders who do select have approached the job with realism rather than unfounded optimism. If genetic advisers are to be listened to in the future they would be well advised not to promise today more than they know they can deliver. Certainly commercial hog producers should be wary of expecting any significant improvement at all from taking replacement gilts out of their better sows - particularly if these are constantly changing rotation-crosses.

The organizations which should adopt such a program, if it can be shown to be genetically sound, are the Breed Associations for those breeds likely to be used in future sows on US farms. To this extent the American Yorkshire Club must be congratulated - though I would like to have seen them evaluate the evidence a little more carefully before advising their members to go to the considerable trouble involved in their Sow Productivity Program.

Illinois Swine Progeny Testing Center

The University of Illinois set up a Swine Progeny Testing association in 1980 and Dr. Leif Thompson has described its work in another paper at this conference. I feel somewhat embarassed by having to include my comments on the project while being a guest of the University, but also feel that if a friend does not speak the truth as he sees it then he does a disservice. The facts are that the advantages and disadvantages of progeny testing for swine improvement were threshed out in the 1940's (Dickerson and Hazel 1944) and no developments since then have really altered the conclusions. Progeny testing has appeal since it can give an accurate measure of a boar's breeding value. I suggest that this accuracy is unlikely to be increased significantly for reproductive traits measured in the boars and daughters by making comparisons within litters - which is the new twist in the Illinois technique. The snags have always been that progeny testing restricts selection pressure (since only a few candidate boars can achieve the minimum of 21 test litters), and that it takes a long time to complete a test.

If the purpose of the project is to measure the genetic merit of boars through their progeny in many different herds and locations, and hence to increase our knowledge and awareness of problems in genetics, AI, and reproduction, then it may well succeed. But this is a very different objective from the genetic improvement of our existing breeds through a progeny testing program which identifies superior sires and builds upon their genes. Again I believe we should be well advised not to raise false hopes along these lines.

RESEARCH

So much for some reactions to the existing scene as I understand it. Further genetic improvement must rest upon increased knowledge and research is one of the ways in which this is acquired. I would therefore like to make one or two brief comments on current US research in the area of sow productivity.

A lot of interest has been aroused by the observation in North Caolina, (Revelle and Robison 1973) that the pig suffers from a similar problem to the one previously identified by Falconer in mice (Falconer 1955). This is that young females which are born and reared in large litters do not show their expected genetic advantage when they produce their own first litters, because their early experience gives them an environmental disadvantage. It is clear that we need to understand this phenomenon better, both as it may complicate selection programs and as it may indicate ways in which gilt litter performance may be improved through changes in the rearing system. Van der Steen's (1983) study in the Netherlands, has improved our knowledge, but Dr. Jack Rutledge is pursuing the topic in several ways in Madison (Rutledge 1980).

The disappointing results from two selection experiements for litter size (Ollivier and Bolet 1981) and ovulation rate (Cunningham, England, Young and Zimmerman 1979) have caused the group at Lincoln to re-examine their selection criteria and methods. Dr. Rodger Johnson now has several interesting selection lines established which should certainly provide new results for close study and discussion in the years ahead.

The other group in Nebraska, headed by Dr. Larry Young at Clay Center, but backed up of course by Dr. Gordon Dickerson, has some very interesting populations and lots of ideas. But I wonder how many people realise just how their research at this, the premier USDA Meat Animal Research Center, is being hampered by their chronic lack of facilities? While there are literally thousands of experimental beef cattle and sheep out on the range, and in the feedlots, the facilities devoted to maternal breed research in pigs are now reduced to 96 farrowings/year (which they contrive to increase to 125 maximum)! This is surely no way to tackle the burning questions that face a US industry with some eight to nine million sows!

Finally I return to a point made at the outset of my paper. The easiest way to make genetic improvement is to cash in on what somebody else did yesterday. Yet nowhere can we see the world's pig breeds being adequately compared in a continuous program. The resources needed are considerable, and the veterinary problems quite serious, but the pay-off could be great.

FINAL COMMENT

How then can I summarize my viewpoint on the organization of pig genetic improvement in the USA? There is of course rather little organization - as would be expected in a country which relies so heavily on individual initiative. While this is an undoubted strength, it can be bought at quite a high price. It is the responsibility of the universities, in their roles both of advising the current and educating the next generation of pig producers, to help get the genetic priorities right. Breed substitution will have the major effect on sow productivity in the short term. Proper use of maximum heterosis among the most suitable breeds is the next priority. Relegated to third place must be within-breed selection programs, both because they are so slow and to date many of the techniques are as

Yet unproven. And even then it is only a tiny fraction of the US hog industry which should get actively involved in such selection programs - and this will then influence the remainder bt seedstock sales.

LITERATURE CITED

Bereskin, B. 1982. Genetic evaluation of young boars and gilts for sow productivity. US Department of Ag., Prodn. Res. Rep. 182. 29 pp.

Cunningham, P. J., M. E. England, L. D. Young and D. R. Zimmerman. 1979. Selection for ovulation rate in swine: correlated response in litter size and weight. J. Anim. Sci. 48:509-16.

David, P. J. 1981. Analysis of Nebraska swine field records: selection practices and genetic trends. PhD Thesis, University of Nebraska, Lincoln.

Dickerson, G. E., and L. N. Hazel. 1944. Effectiveness of selection on progeny performance as a supplement to earlier culling in livestock. J. Agr. Res. 69:459-75.

Falconer, D.S. 1955. Patterns of response in selection experiments with mice. Cold Spring Harbor Symp. Quant. Biol. 20:178-96.

Jones, H. W., C. M. Cox and R. H. Byrd. 1982. Sow productivity. Proc. Purdue University Swine Day 17-20.

Meat and Livestock Commission. 1983. Pig Yearbook, MLC Economics, Livestock and Marketing Services, Bletchley, England. 109 pp.

National Hog Farmer. 1980. Productive sow lines identified using pig weights, computers. Natn'l Hog Fmr. Dec. 15, 1980. 12-16.

NSIF. 1981. 'Sow productivity indexing' leaflet.

Ollivier, L. and G. Bolet. 1981. La sélection sur la prolificité chez le porc: Résultats d'une expérience de sélection sur dix générations. Journées Rech. Porcine en france 261-268.

Pig Improvement Company. 1983. Commercial herds with PIC stock. Pig Improver 4. No. 1. p. 5.

Revelle, T. J. and O. W. Robison. 1973. An explanation for the low heritability of litter size in swine. J. Anim. Sci. 37:668-75.

Robison, O. W. 1981. The influence of maternal effects on the efficiency of selection: a review. Livestock Prodn. Sci. 8:121-137.

Rutledge, J. J. 1980. Fraternity size and swine reproduction. 1: Effect on fecundity of gilts. J. Anim. Sci. 51:868-74.

Smith, C. 1958. Efficiency of testing schemes in swine. PhD Thesis. Iowa State University.

Smith, C. 1959. A comparison of testing schemes for pigs. Anim. Prod. 1:113-121.

Smith, C. 1960. Efficiency of animal testing schemes. Biometrics 16:408-15.

Strang, G. S. and J. W. B. King. 1970. Litter productivity in Large White pigs. 2. Heritability and repeatability estimates. Anim. Prod. 12:235-43.

Strang, G. S. and C. Smith. 1979. A note on the heritability of litter traits in pigs. Anim. Prod. 28:403-6.

Swiger, L. A., K. M. Irvin and G. Isler. 1978. Selecting for sow productivity. Mimeo. 5 pp.

Van der Steen, H. A. M. 1983. Maternal and genetic influences on production and reproduction traits in pigs. Doctoral Thesis. Wageningen, Netherlands. 112 pp.

Potential of Progeny Testing

L. H. THOMPSON

INTRODUCTION

A comprehensive look at the swine industry in the United States during the past 10 to 15 years would reveal very little change in any phase of production efficiency. Selection of breeding stock has been carried out primarily by visual appraisal; thus, little selection pressure has been placed on traits of economic values. A significant number of seedstock producers have abandoned their performance testing programs, and in general, the industry has drifted from one showring type to another.

A few dedicated producers are attempting to offer some information on performance traits of their pigs, either in their own on-farm program or through participation in central testing units. Unfortunately for the industry, most seedstock producers use results from their testing program as a sales tool without any serious attempt to employ the same principles in selecting their subsequent herd sires. Thus, their program has very little direction.

The central boar testing stations have lost a lot of the popularity enjoyed in the late 50's and 60's, probably due in part to the potential of disease transmission. The role of the central testing programs, as they are operated presently, is being questioned by geneticists who contend that on-the-farm or within-herd performance testing programs have more potential merit to offer for genetic improvement of swine breeding stock. The capacity of central testing units is not sufficient to evaluate even a portion of the boars needed by the industry, but those centers could serve as a means of making some limited comparisons between herds if adequate numbers of representative pigs were tested by several producers. The concept and use of the central testing program will probably undergo some modification in the near future.

A major problem still remains with regard to the existing testing programs in the swine industry. Commercial producers have been, and continue to be, reluctant to offer financial rewards to seedstock producers commensurate with the genetic quality of performance tested breeding stock. As a result, the seedstock producers write off the cost of their testing programs as part of advertising.

Why haven't the commercial producers been willing to reward seedstock producers for better performance? One reason might be that they have made a lot of money with cheap feed and tax incentives and have not been forced

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financially to consider improving production efficiency. Perhaps they don't believe in the reliability of testing programs or have not been able, in many cases, to realize an improvement in some phase of production efficiency without losses in other areas from the use of tested boars. Many commercial producers have made "improvements" in their production systems through newer and better facilities, better diets and disease control programs. At the same time, genetic improvement has not been stressed. If producers are not aware of deficiencies in their own levels of production at various phases, decisions relative to the most important traits to consider in a selection program involving the purchase of new breeding stock will be more difficult. Perhaps the definition of production goals and their relative economic merit is necessary before a sound genetic program can be pursued.

Apparently, there is a resurgence of interest in obtaining more information on new breeding stock. Also, the advent of indexing sows on the basis of productivity introduces a group of traits that can be considered in addition to those conventional traits such as growth rate, carcass composition and feed efficiency. The dairy industry, while relying primarily on a single trait, probably has much to offer the swine industry in terms of confirming the assets as well as costs of long-term animal breeding programs.

Dr. Lush, who is certainly recognized as one of the fathers of quantitative genetics, was involved with the development and success of that program. Development of a reliable procedure to preserve semen and then use of superior lines in a testing program, combined with a national recording system, provided the avenues by which progeny testing could be successfully applied to the improvement of milk production. Monetary reward was available for those producers who adopted the new animal breeding and selection techniques. At the same time, the structure of dairy cattle production changed. Those breeders who were producing bulls to be used in herds through natural service have almost disappeared, and the use of unproven sires is almost certain assurance of reduced production efficiency or at least no improvement thereof.

The bull, however, is unique when compared to males of other domestic species. Dilution of semen to a rate whereby 200 to 400 females can be inseminated with normal fertility from each ejaculate has resulted in a tremendous increase in breeding capacity and thus genetic impact of superior bulls.

In swine, one collection of semen can be diluted and used for insemination of 10 to 25 females. Since sows should be bred twice in one estrous period, five to 12 litters would actually result from one ejaculate of boar semen. Under conditions where a boar would be used four to five times per week by natural service, extending semen and inseminating sows would increase the yield and thus the genetic impact of a superior boar at least ten-fold. Using a very conservative figure of 20 litters per week, one boar could contribute genetically to the number of offspring equal to approximately 500 sows per year (more than 1000 litters). Most commercial herds of this size would require a battery of 10 to 20 boars and the frequent introduction of several young boars for breeding gilts in a hand-mating system.

These figures include only the existing potential use of AI with present technology at about half of maximum efficiency and do not consider the possibility of significant developments in this area of research. Application of AI in swine has potential for dramatically increasing selection pressure and thus enabling the development of more complete testing procedures to

facilitate the identification of genetically superior sires. At a time when an industry lacks uniformity in testing programs or recording systems, discussion about the relative merits of different types of selection programs becomes almost purely academic. Development of any testing program, as long as it is based on the improvement of economically important traits, would have some merit; however, a large population must be involved.

Several types of testing programs are available for producers. Performance testing programs at central testing facilities have been in existence since the 1950's. Without a doubt, they have served as a means of education and information. However, the genetic value of performance testing programs at central locations is severely limited by the number of animals that can be tested in existing facilities (Robison, 1982).

On-the-farm performance testing is being strongly promoted by the National Swine Improvement Federation as the program of choice. Producers are encouraged to test as many offspring as possible in order to rank animals on performance traits within herds. By selecting the top 5% or fewer young boars and top 10% of the females based on measured performance traits, genetic progress could be realized.

Introduction of outstanding sires from other herds involved in the same type of testing program should enhance performance. Certain production goals must be established and pursued for several generations if a selection program is to yield significant changes in production.

For traits that are exhibited in both sexes and are moderately heritable (30 to 50%), performance records of the individual provide a fairly reliable indication of what that animal can contribute to its offspring under similar conditions. If the performance records of the selected parents and of the original population are known, the expected rate of genetic change in a population can be calculated by the following formula:

$$\text{Rate of change} = \frac{\text{Difference in performance in average of both parents from the population mean} \times \text{heritability (\%)}}{\text{generation interval (days or years)}}$$

For the most rapid improvement, selection of the next generation of breeding stock with the largest deviation from the original herd must be mated at the earliest opportunity. Genetic improvement should be realized in a positive direction but at a decreasing rate as the upper limits of biological capacity within that population or herd are approached. Unless a larger population is screened to maintain a gene source for large differences in performance, progress will be limited.

Breeders should be aware that inferences concerning relative performance cannot be drawn about animals that do not share a common parent or a common environment. A boar that gained 2.5 pounds per day in one population could in fact be superior to a boar that gained 3.0 pounds per day in another population. Herein lies a major barrier to genetic improvement in the swine industry unless a means of comparing animals between different testing units is developed.

For traits that have lower heritability performance records, especially

those based on one observation, are not as reliable as indicators of genetic value. Traits like litter size and milking ability or mothering ability are in this category. Obviously the sire does not express these traits; however, he does contribute genetically to his daughter's capacity to perform. Selection of males for sex-limited traits must then be carried out indirectly by evaluating the performance records of female progeny or female relatives. The reliability or repeatability of maternal traits is low but does improve by nearly 50 percent using records from several litters of the same female (dam) or from several females (siblings).

One must remember, however, that using information only from the dam represents just half of the genetic makeup of an individual. Therefore, both Bereskin (1982) and Schinckel (1983) have recommended that a breeding value estimate of an individual is far more accurate if it is based on the genetic contribution of both parents and perhaps the performance of full siblings. An estimated breeding value, then, is a more precise predictor of genetic merit because it includes data from several relatives for the same trait.

Until a boar sires five to 10 daughters from which maternal performance is recorded, records of his dam, paternal granddam and full siblings provide a reasonable estimate of his genetic value.

Progeny of a sire, especially if they are the result of several different mating combinations, provide the most accurate proof of a sire's genetic merit for sex limited traits of low heritability. Production records from six daughters provide a consistently more reliable basis on which to estimate breeding value than that based on records of both parents. Significant genetic progress has been realized in the dairy industry by the use of sire summaries comprising performance records from daughters in several different herds or environments. The beef cattle testing program is approaching the same type of testing program for both performance traits related to growth rate and maternal traits such as weaning weight and calving ease. The accuracy of these testing programs involving both dairy and beef cattle is greatly increased by the use of one sire in several herds through artificial insemination. A sire used in that fashion becomes a reference sire whose offspring can be the basis on which progeny from sires in several different herds are compared. The reference sire concept allows for evaluation of a much larger group of males than would be accommodated in one central bull stud.

Use of AI in swine provides a mechanism by which reference sires can also be used in different herds (Wilham, 1982). The testing population would be expanded into a much larger group of different herds that would otherwise be a collection of many smaller, isolated populations without a common genetic bond. Rapid genetic progress in an industry as large and diverse as the swine industry in this country cannot be realized through a selection program limited to within-herd comparisons.

The reproductive characteristics of swine probably allow for more diverse types of programs than in other species which do not bear litters or gestate young for a longer period. The polytocous nature of sows offers an opportunity for animal breeders to mate females to two sires simultaneously. Identification of offspring in a heterospermic litter by phenotype such as coat color, ear shape or blood type can be accurate in many cases and can serve as the most precise means of comparing progeny of several sires (Gianola and Thompson,

1983). Since the dam's contribution to her offspring represents a significant source of variation, minimizing or equalizing one side of the pedigree greatly increases the accuracy in comparing performance differences between progeny groups on a within-litter basis. The number of observations needed to make accurate comparisons between sires on a within-litter comparison is smaller by nearly three-fold than where sire groups are single sire litters. Comparing sire groups on a within-litter basis requires fewer offspring for the same degree of accuracy.

Competitive or heterospermic mating, if it is to be used as the basis for a testing program, must be incorporated into an AI program. Two advantages can be gained from such a combination. Pooling semen through natural mating will probably result in an unequal yield of offspring in favor of the most aggressive sire, whereas pooling semen will allow for some adjustment to account for fertility differences. Obviously, the distribution of semen from reference boars from a central location to many cooperating herds greatly enlarges the genetic population concerned and involves many more environments.

Transmission of superior genetic material from the elite herd to commercial utilization has normally involved at least one additional generation through what has been termed the multiplier herd. The amplification of breeding capacity of boars through AI, even with fresh semen, is such that commercial producers could begin to realize the genetic advantage of elite herd sires without either the genetic dilution or delay resulting from the second or third generation in the multiplier herd. Increasing the use of a sire through AI in turn should place greater emphasis on the need for more thorough and accurate evaluation of the genetic potential as a sire. Thus, the prospective use of one tool requires the concomitant development of the other. In the end, combining progeny testing with an AI program effectively reduces the industry genetic lag, that is, the time from which genetic material from the elite herd is actually in production in the commercial herd.

The potential and actual disease problems which are associated with the movement of live animals are a real threat to the seedstock industry. Several systems are in use today for the purpose of minimizing disease transmission. Movement of genetic material from one herd or system to another can be accomplished with the least disease problems through surgical means involved in the transfer of a litter near conception (embryo transfer) or near parturition (Caesarean section). Semen can also be procured with minimal potential for disease problems.

The first alternatives provide the best protection but require much more sophisticated facilities and procedures. The latter case can be conducted in an environment such that disease risk is similar to that of embryo transfer or Caesarean section. Development of some type of a testing program at this time does not appear to be economically feasible with the surgical transfer of piglets, but could be a useful tool in retrieving more ova from hyperprolific sows. Evaluation of progeny of sires involved in a properly controlled AI system is a very realistic situation and has been used advantageously in both the dairy and beef cattle industries. Technology for freezing boar semen has not yielded the results enjoyed by the bull studs but the distribution and use of fresh semen from boars does offer an opportunity for genetic improvement of swine through a model similar to, yet more efficient than, that used in cattle.

Importation of semen actually explodes the gene pool available to both seedstock and commercial producers. Bache (1982) found that most commercial producers were not realizing the benefits of heterosis because of the problems in making the correct matings and the number of boars required for all the necessary mating combinations. Producers could easily use AI to sire a major percentage of their replacement females and then maintain a herd boar battery for production of market hogs (a rototerminal breeding system). Usually, the number of terminal cross-boars used is approximately three to four times the number of gilt-line sires. Management of the breeding program would be simplified and genetic progress would be enhanced by the quaity of sires available through a progeny testing program designed for improvement of maternal performance. Seedstock producers have the most to gain through a program based on the reference sire concept. Without such a system, selection of better herd sires for the next generation will continue to be little more effective than random sampling. Surely the need for a more reliable and integrated approach to genetic improvement is great.

Three reasons justify the development of a progeny evaluation program:

1. More thorough genetic evaluation of sires is needed, especially for sex-limited traits, if selection programs are to be effective. Proof of a sire's genetic merit on the basis of his daughters' performance records is far more reliable than estimating her breeding value on only one of his parent's performance. Estimated breeding values can provide a screening tool to select boars for progeny testing.

2. Use of AI, although not to the magnitude available in the dairy industry, greatly enhances the possible selection pressure on the sire's side of the pedigree. In elite herds, the upper 10 percent or fewer could sire the same number of offspring.

3. By incorporating AI and progeny evaluation with reference sires, realistic genetic comparisons within and between herds would be facilitated. Maximum use of reference sires in an AI program would require a more thorough knowledge of their genetic merit and would greatly enlarge the gene pool under consideration.

SUMMARY

Several types of selection programs are available for swine producers. The use of these tools for genetic improvement of economically important traits will require a lot of time and effort. Widespread acceptance of testing programs will depend largely on the financial support from the commercial sector. As profit margins narrow, the potential for genetic improvement may be realized more quickly. On-farm performance testing is a basic program which is desperately needed. Incorporation of the concept of estimated breeding values is a natural and apparently correct end-product of that system. Development, refinement and implementation of the reference sire concept for progeny testing and genetic communion between different production units is absolutely essential in the maturity of a meaningful selection program. To assume that one phase in the program is not essential is invalid considering the need for more complete and accurate knowledge of true genetic merit of animals. Every producer, regardless of type of operation, will need a more thorough genetic appraisal of a very large gene pool if production efficiency is to

be improved. The futuristic implications of gene transfer and DNA manipulation require nothing short of the application of the most sophisticated and accurate genetic tools available.

LITERATURE CITED

Bache, D. 1982. The real world of swine breeding systems. Proc. Nat. Swine Improvement Fed. Ann. Meet., Des Moines, IA, p. 14.

Bereskin, B. 1982. Genetic evaluation of young boars and gilts for sow productivity. USDA Ag. Res. Sec., Prod. Res. Rep. 182.

Gianola, D. and L. H. Thompson. 1983. Theoretical consequences of heterospermic progeny testing in polytocous species. Mimeo. Dept. of Animal Science, University of Illinois, Urbana.

Robison, O. W. 1982. Role of central test stations in swine improvement. Proc. Nat. Swine Improvement Fed. Ann. Meet., Des Moines, IA, p. 92.

Schinckel, A. P. 1983. Purdue swine improvement program. On-farm Micro Computer Conf., Purdue Univ., West Lafayette, IN.

Wilham, R. L. 1982. Opportunity in swine breeding. Proc. Nat. Swine Improvement Fed. Ann. Meet., Des Moines, IA, p. 8.

Pork Industry Perspective

JACK RUNDQUIST

The pork industry is at a turning point. Beef consumption reached its peak in 1976. Since then, per capita consumption of beef has been declining. Poultry consumption continues to rise and overtook pork consumption on a per capita basis for the first time last year. The reasons for poultry's popularity are two-fold. First, they have improved their feed efficiency to the point where they are extremely competitive in the retail meat case. The other reason has to do with the fact that the poultry industry has come up with a hundred different ways to cut and package their product. We have a vast frontier open to us in this area in the pork industry. Only now are we starting to research and utilize some of this meat cutting, processing and packaging technology.

Pork consumption has been static since 1920, at least when put on a per capita basis. Total pork consumption of course has increased rather substantially in this country, but so has population. So when you consider per capita consumption, there really is not much change over time. We consume all the pork we produce, so, therefore, there are some fairly sharp ups and downs in consumption from year to year. But when leveled off over the long term, there is not much change. In 1980, we had the largest per capita consumption in 20 years. The problem was that there was no profit in pork production in 1980. Therefore, our challenge is not to increase per capita consumption, but rather to increase per capita consumption of pork at a profit to the producer.

The pork industry needs to win back and attract new consumers to pork. The National Pork Producers Council's (NPPC) recent survey shows that we were able to reach nine million new consumers in 1982, as compared to the results of the 1980 study. These studies were done in cities which saw heavy NPPC advertising. Now the question is, what will it take to keep these new consumers and attract more to the pork meat counter? Our NPPC promotion is geared to enhance demand. We have attempted to measure consumers' attitudes and gear our promotion and advertising to fit current lifestyles. By this, I am talking about the fitness boom. Everyone is trying to get healthy. Early in the morning they're out jogging. In the evenings they're out riding their bicycles or roller skating. Or they're playing racquet ball or tennis. Health clubs can barely keep up with their business. Several surveys indicate that perhaps as many as 72 million Americans are participating in some kind of sports or fitness activity on a regular basis. The National Pork Producers Council's "America, You're Leaning on Pork" promotion campaign is directed at this new diet/health/fitness mentality.

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Another great opportunity for expansion in demand for pork in this country has to do with export potential. For meat, it may be limited, although there are still some avenues which we could pursue to increase this export area. For offal, the prospect is quite good. Europe and South America already buy a fair amount of these slaughter by-products. But there is probably some room to increase those exports. The demand for breeding stock is good and has been increasing. Singapore recently bought a large shipment. Haitians and Dominicans are buying breeding stock to repopulate their island after African Swine Fever erradication efforts. The Danes have been making visits looking at several of our meat breeds to improve their meat quantity and quality. There will be an opportunity to export to mainland China and Japan has been buying a fair share of our breeding stock for some time.

National Pork Producers Council is faced with a dilemma about exports of breeding stock. We would certainly rather export value-added products such as pork, or, better yet, processed pork rather than breeding animals. The export of breeding stock only helps that one producer, whereas export of meat increases demand for all pork and therefore helps all producers. On the other hand, the argument is that those countries are going to buy that breeding stock, and if we don't supply it, they'll find it somewhere else.

Our domestic market is by far our biggest and best market for pork. In fact, many other countries see the value of this market too. Canada ships in meat and live hogs. The European economic community ships in canned hams. Italy wants to send us their Parma hams, but because of our concern about African Swine Fever, and other swine diseases, we have not allowed Italians to enter the United States. We are in the process of further researching whether African Swine Fever can be transmitted through processed hams. The foot and mouth disease outbreak in Denmark slowed down Danish exports, but they are about to show some substantial increases again. Denmark raises about 2% of the world's pork, but satisfies about 1/3 of the world's export market.

Japan is the number one importer of U.S. pork and has a great potential for increases of pork and breeding stock imports. Canada, Denmark and Italy, among other countries, want a share of the Japanese market. Tough trade negotiations are needed. We need to study world trade, and to train agricultural leaders of tomorrow in this area. We must develop an awareness for world markets and opportunities and not be limited to our domestic market. We should train people who can understand and be fluent in speaking several foreign languages. We must develop products for specific foreign markets. One of the serious shortcomings in our trade up to this point has been the fact that we've attempted to force our U.S. products on foreign customers. We do need to develop a better foreign trade policy.

Forty-five countries ship meat and meat products to the U.S. United States import restrictions and requirements should be equal to those applied to our domestic meat products. In other words, purity and wholesomeness should be assured. Disease risks should be reduced to the lowest possible level.

Producing pork for domestic and world markets will require further sharpening of production practices, management skills and hard-headed determination to survive with narrow margins, in a low-profit industry. Last summer, Brazil backed out of a contract to supply soybeans on the export market in anticipation of higher prices brought about by our U.S. drought. A high-ranking USDA official reacted to this stating that this action would show that Brazil is not a reliable

supplier. My point here is that we should not just be looking at exporting pork when supplies are high and prices are low, but rather on a continuous basis. We must assure foreign clients that we can supply them with the safe, wholesome, lean pork they want on a consistent basis.

What will it take to really succeed as a U.S. pork producer? We must have a good handle on the three factors which affect our profitability. Those factors for pork production, or for any other business for that matter, are supply, demand and cost of production. On the supply side, we must attempt more self control on numbers as an industry. We all like the free market conditions in our industry, but with more information about trends and projected numbers, we will be in a position to reduce the peaks and valleys associated with our extreme hog cycles. National Pork Producers Council is looking at this area of market analysis as a service to its members.

On the demand side, the industry has a modest advertising program to help enhance demand. Although it is limited by the amount of checkoff received, your National Pork Producers Council has developed an aggressive advertising program which includes TV, radio, newspapers, recipe books, billboards, in-store promotions, and point-of-purchase materials, among other things. My question is, do we really have the product that satisfies that demand? We'll discuss that more later.

The U.S. pork producer in the past marketed corn through his pig crop. In fact, someone said that pork production is really just a part of the corn industry. We must reconsider this approach. We're pork producers, not corn marketers. The most aggressive producers consider pork production as an art and a science in itself rather than just adding value to corn.

What does the U.S. consumer really want for his/her pork dollar? Earlier surveys showed that the four main considerations of a consumer when they buy pork are price, color, fat and bone. Recent surveys showed that there are some other considerations that have come in, but I think we'd be safe in listing the following as things that we should strive for in the industry. Pork should be lean, wholesome, fresh, succulent, free of residues, economically and competitively priced, free from excessive external fat, and with a minimum amount of internal fat. The issue of residues, and the use of antibiotics and hormones in routine feeding of livestock are probably of greatest concern to the general public. Our surveys show that the consumer is more concerned about those than even calories and cholesterol.

Some of the trends that are certainly taking place in the industry include the fact that hogs will be produced where the corn is grown. That was the case during the early years, but when transportation linked the whole country at a cheaper cost, we started seeing proliferation of pork production units in other parts of the country. Now with transportation costs escalating, pork production is again being centered in the corn belt. Another trend that is certainly taking place is that hogs will be slaughtered where they're produced. Again, in conjunction with cheap transportation, some slaughtering plants were built near metropolitan areas. However, it doesn't make much sense to move the whole hog with all its by-products to a distant packing plant. Therefore, the packing plants have been built where the hogs are produced. Another trend which is already taking place is keeping the fat and bone at the packing plant. This has a two-fold efficiency effect. For one, a transportation efficiency is realized because of the reduced weight that has to be shipped to be thrown away at the consumer end. The other efficiency relates to the fact that the fat and bone can be used

in by-products such as lard and bone meal. Therefore, we would see more trimmed boneless products going to the wholesaler and retailer. With this trend, we'll see more ready-to-cook products with little or no water. Vacuum packaging and irradiation will alow us a longer shelf life which will make our product far less perishable.

Pork is cheaper to produce than beef, but probably will continue to be more expensive than poultry meat. The poultry industry achieved a high degree of feed conversion and product development, but beef consumption began to recede in 1976. Pork consumption is the same as pork production in this country as we eat all we produce. Consequently, our decisions concerning expansion and liquidation of the breeding herd ultimately decide how much pork our consumers will buy. Price has a way of clearing the market.

It seems to me we need to become much more sophisticated when making production plans. Somehow we need a game plan that will supply the market with sufficient pork to satisfy the demand but yet allow producers to have a reasonable profit for their efforts. In recent years, the U.S. farmer received about $10 billion regardless of the number of pigs produced. That is to say whether we produce 80 million or 98 million hogs, we still received $10 billion as an industry. Somehow we need to develop strategies that will fulfill the consumer's need at a profit to the producer.

Most everything else I will talk about will have to do with the producer's cost of production. When I'm talking about reducing cost of production, what I really mean is increasing efficiencies. Before any producer can even consider increasing efficiency, he has to know what his current level of operation is. No producer can know where he's going unless he knows where he's been and where he's at. The only way to have this information is to keep records. In fact, I submit that one of the main reasons for the sharp peaks and valleys we experience in our hog cycle is that producers do not have a good handle on their costs of production. Some producers are expanding when they are losing money just simply because they don't realize what their costs are. You'd be amazed if you knew how many producers don't include labor costs in their production records because they say they do all the work themselves. This whole area of record keeping needs further refinement throughout our industry.

One of the basic problems in record keeping on a national basis has to do with the lack of uniformity in formulas and definitions used. If I told you we don't know the definition of a gilt, you would probably laugh. However, as we get into it, we find that there are a number of different times when gilts are added to the breeding herd. This affects the outcome of the calculation of pigs per sow per year, a standard of reproductive efficiency. The figure given for pigs per sow per year by any individual pork production unit means nothing until you know at what time he includes his breeding females in the herd. There are a number of other examples of this. Recently, the NPPC took it upon itself to come up with standardized formulas and definitions. These are in the process of review at the present time and will be published early next year for industry use. Following that publication, consideration will be given to where we go from here to enhance record keeping on a national basis.

Another factor that certainly has a dramatic effect on the efficiency of production and, therefore, the cost of production has to do with disease. We certainly need current health programs to insure minimal or disease-free production. I am very much encouraged by the recent development of the national health program

by the breed associations. I think this is a large step forward and is long overdue. It is estimated that 75% of all the hogs in the country received antibiotics during some phase of the production process. The pork industry therefore carries on its back an entire swine health-related industry which includes veterinarians, drug companies, feed ingredient companies, etc. There is no doubt in my mind that the industry must make efforts to move away from the use of drugs, vaccines, antibiotics, etc., not only because of consumer concerns, but just simply because of the high cost of these products. The SPF concept took many years to gain acceptance, but is finally accepted as the only way to produce healthy breeding stock. Denmark has decided that all breeding herds shall be SPF. My question is why we don't do this in the United States. Our greatest deterrent to exports and the main reason the breeding companies have been so successful domestically is swine disease. We must give this further consideration in the future.

In the reproductive area, the sow productivity index is becoming more widely accepted and used. Some of the breed associations have been the leaders in this area. For example, the American Yorkshire Club has pushed the sow productivity index and shown progress because of it.

I have a real problem with why live performance testing and progeny testing. The breeders say that they would do it if the commercial men wanted the data. I guess my question is why people buy breeding stock without having valid data. We need honest comparable data instead of this mishmash of pedigrees and show winnings which are not very important for long-range breeding programs that will provide the breeding stock we need. It makes a lot more sense to measure the traits you desire and let the hog look like whatever it might, rather than saying what you want it to look like and forcing it to perform from that mold.

Somehow we must improve litter size. A greater number of pigs must be born alive. We need greater survival to weaning. European breeders have established higher prolificacy than American breeders. Landrace and Large White breeding predominates, which are breeds that have been long recognized for their maternal characteristics. But there are indications that European breeders are now looking for breeding stock that will improve carcass qualities. For example, progressive breeders in Denmark are crossing Durocs and Hamps on Danish York X Landrace sows to produce the "Antonius" pig. These Danish pigs show heavier muscling than pure Landrace. This type of approach is being used in other countries that have been using totally white breeds up till now. South Africa and Zimbabwe both have had representatives visit the U.S. recently to buy Hampshires and Durocs to bring some meatiness and durability into their Landrace Large White herds.

Unfortunately, the typical American producer's reaction to improving litter size, especially when prices are low and hog numbers are high, is that "we are producing too many now and increasing litter size would only lead to more pigs". Also, "producers will continue to keep their farrowing capacity full and higher prolificacy will only lead to greater production". This may be true, but because of fixed charges of maintaining a productive female for one year in the breeding herd are practically the same regardless of the number of pigs weaned per sow per year, the cost per pig would be greatly reduced by increasing the number of pigs weaned per litter.

We must come to the realization that we have to do it ourselves. No one else is going to do it for us. We must improve our own level of management and improve our own efficiencies in order to reduce our cost of production. We must continue

to checkoff on a regular basis so that we can each help and enhance the demand for our products. We must have the self control to limit our expansion even when times ahead appear profitable. Too many times in the past we have seen where producers get over ambitious in their expansion plans and soon we are on the downhill slide again.

Back to the discussion of reproduction, we need to find prolific strains in all breeds. Progress will be slow, but we must improve litter size. Sow productivity indexes are useful to differentiate maternal traits. Computers are making the arithmetic easier. We need to practice judicious culling based on several litters with productivity indexes, thereby beginning to indicate better performance. Hopefully, the sow productivity index programs will eliminate poor and/or low milking ability. I think we need to develop an estimated breeding value for the pork industry. This was recently developed and discussed by Dr. Bereskin of USDA. This program is based on sow productivity and progeny performance records so that we are assured that these other characteristics are passed on to successive generations.

Because litter size has a rather low level of heritability, in the range of 15-20%, we have assumed in the past that we really can't do much about changing litter size except by crossbreeding. This must be changed. There is ample evidence that higher prolificacy is the basic cornerstone for a profitable pork enterprise. The producers who realize this are probably still in business. We probably have done more to improve the national averages for reproduction and feed efficiency over the last two or three years than we have done in decades before that. The improvement has not come through any changes in management or environment or genetics, but rather just the weeding out of 100,000 less efficient producers. Although this is difficult if it's your neighbor, your son or you, it is certainly healthy for the overall pork industry.

Feed efficiency defined as feed required per unit of gain is another major concern. Rapid growth to desired market weight is another basic cornerstone. Over the past years, nutrition research has given substantial information to improve production. Corn-soy rations, vitamin supplementation and synthetic amino acid formulations are but a few of these significant factors. These nutritional improvements have resulted in faster growth and less feed.

Ad lib and limited feeding continue to be confounding situations. Liquid or dry feeding is another controversial issue. When producers are routinely awarded for producing lean pork, then some of these feeding methods may be given further consideration because of the fact that they result in greater lean production.

We need carefully formulated rations for all stages of production so that the pig will be able to fully develop his inherent growth potential. National Pork Producers Council has listed as a priority the nutrition of the nursing pig and of the early weaned pig, as well as nutrition of high-producing sows. Actual rations will vary with sources of feed grains and protein supplement available. The precise formulation of on-farm mixing will be achieved by computerized weighing, grinding and mixing.

Among these several factors affecting feed efficiency, one of the most important, but very often overlooked is feeder trough design, feed wastage and spoilage. Going right along with this problem is the problem of moldy feed and toxins which decrease feed consumption. Currently, my personal preference would be a full feed ad lib system with backfat level being controlled through breeding and selection programs.

Evaluating boars for growth rate and feed efficiency are the objectives of the present central boar testing stations. They have helped us to isolate individual boars and strains with the desired characteristics. In spite of this though, central testing has had very limited appeal to breeders. This is partially because of disease risks and probably because of the possibility for poor performance. On-farm performance testing or central testing should be developed as a requirement for boar registration and sale. The National Swine Improvement Federation has developed guidelines for uniform swine improvement programs. These need to be supported by all the breed associations, all breeders whether purebred or breeding companies, and by commercial producers.

Only boars that meet established performance criteria plus soundness and health requirements should be allowed to be sold for breeding purposes. Mandatory performance testing can be developed. Some breeders may not be willing to test and may have to cease operations. Some strains, lines, pedigrees, and perhaps even breeds would be eliminated. Narrowing our genetic base would be a valid criticism, but if that base contains undesirable and poor performance, there is little justification for allowing genetic trash to lower our overall productive efficiency.

The objectives of breeding programs lead eventually to the final product - the market hog ready for slaughter and the resultant carcass ready for consumption. In the past we have had considerable difficulty trying to get recognition through higher pricing for the truly desirable lean meaty hog. We have great expectations that the recent development of the Pork Value Program and the Lean Guide to Pork Value will be accepted by the industry. Basic information on components such as backfat depth, live weight, and muscling is definitive enough to give fairly accurate values.

Backfat probing is an old idea that had great emphasis years ago and really gave the push to get away from the lard-type pig so prevalent 20-30 years ago. We certainly need to continue these measurements in our breeding herd as well as in our hogs going to market.

Hopefully the packing industry will accept the Pork Value concept and develop pricing structures that really give emphasis to meaty lean carcasses. There's good evidence at this point that the packing industry does support the program and that a number of large and small packers have developed their own programs consistent with the Pork Value approach. Hopefully by the end of next year, we will have a large porportion of the hogs in this country bought on that system. Once producers realize that desired hogs are more valuable and undesired hogs are worth substantially less, they will want breeding stock capable of producing the higher-value offspring.

Mandatory identification of all hogs back to the farm of origin is another subject getting a considerable amount of industry attention these days. With modern high-speed electronic computerized equipment, modern packing plants will be able to accurately determine carcass information such as backfat depth, loin muscle area, meat quality, percent fat, percent lean, etc. With identification of individual pigs traced back to the herd of origin and to the sire and dam, the true and ultimate breeding value of the herd can be established.

A very small number of American pork producers have been apprehensive about the identification of slaughter animals. The recent sulfa residue fiasco turned many producers away from identification, but we now have solved the sulfa problem

and we need to reconsider mandatory identification. The greatest advantage will be that of allowing accurate carcass evaluation tied back to the sire and dam of each market animal. Computerized records could accurately determine the true breeding value of the hog industry.

In addition to the carcass evaluation situation are the disease eradication programs. To finally eliminate brucellosis, tuberculosis or pseudorabies from our industry, we must have the ability to go back to the herd of origin and clean up these problems. I earlier stated that residues are the number one concern of consumers. We continue to tell consumers that our product is safe and wholesome and free from residues. Identifiction would give us the opportunity to assure that this is true.

To completely solve the age-old problem of trichinosis, identification will be essential. Because of the unique transmission of trichina through consumption of muscle tissue in both pigs and humans, the original source of infection in the pig must be stopped. Therefore it is essential that pigs infected with trichina be identified and also the farms from which those pigs came must be accurately established so that clean-up procedures can occur. National Pork Producers Council's activities which will lead to a 100% trichina-safe retail pork supply are moving along on schedule.

The next area I want to cover involves breeding stock - who will supply the boars and gilts and who will be the important sources of replacements. There is no doubt that purebred breeders will always be around as they will be necessary for continuation of the pure lines. Even the breeding companies depend on them for sources of pure line boars and sows. However, I think that breeding companies will also continue to be an important source of breeding stock for commercial producers. Because of their substantial investment in sufficiently large numbers of animals and adequately trained technical and sales staff, breeding companies will continue to flourish. The reason that breeding company stock are almost exclusively used in the extremely large units is because these managers do not want to be involved with a genetic program. In addition to livestock, they also buy an entire genetic program from the breeding company.

The purebred producer will continue to provide the industry with purebred stock, but not many have the sufficient size, large enough numbers, and large enough facilities to accurately evaluate several lines or strains of more than one breed. In an attempt to compete with breeding companies, many purebred breeders are trying to keep two or three breeds so they can sell purebred and crossbred breeding stock.

For the purebred breeder to continue to be a viable source of animals for commercial producers, they will have to continue to cooperate among breeds and continue to develop and adhere to programs which enhance their competitive standing. Their health program alluded to earlier is a good example of this. They will also have to encourage more performance testing. And they will have to utilize technical genetic principles more in the future.

To improve our overall efficiency, I would like to see strengthening of our herd health program in all phases of production. This will include breeder herds, commercial farrow to finish, feeder pig producers, and feeder pig finishing herds. We must stop considering segments of our industry and develop an overall plan that puts pressure on all segments to achieve equivalent health status. Somehow diseases must be eradicated, controlled and prevented from spreading to new herds.

Some time in the future genetic resistance to disease may become a reality through genetic engineering and biotechnology, but until then an all-out effort needs to be developed. American animal health standards are not as good as they should be.

Based on recent experiences with pseudorabies funding, it appears that our federal government will not enter into disease eradication programs with indemnity funds supplied by the federal treasury. The alternatives are either to disregard the disease and live with it, or enter into a control and eradication program. In the latter case, we really have two alternatives. One, we could develop our own industry fund in the form of a producer levy to provide indemnity for ourselves, or we could use the benefits from eradication of the disease from a herd as an incentive to go about elimination of the disease. The first alternative here involves a producer levy similar to what was undertaken in the United Kingdom. I question the ability of such a program to be successful because the producer levy from all producers would need to be on the order of at least $1.00 per pig marketed. I am sure that producers would be reluctant to put a dollar in the kitty especially if pseudorabies was not in their area. Therefore the only practical method to support disease eradication programs is to spell out the economic returns to a producer if he should decide to eradicate the disease from his herd.

I have attempted to give an overview of where I think this industry is heading in this talk on pork industry perspectives. There is no doubt that we can all take a moment to reach around and pat ourselves on the back for a great deal of progress over the last 20 or 30 years. However, to remain in that position for very long would be a big mistake. There is a lot of work to be done and a lot of improvements that can still be made. Even though this industry has accepted many technological advances over the past few years, I still feel that the future is a vast frontier. We have so much to learn so much yet to do.

Environment and Health in the Hog House

STANLEY E. CURTIS AND KEITH W. KELLEY

INTRODUCTION

Swine are adaptable to a large range of environments. They can be produced successfully in many facilities and climates, even some that provide suboptimal conditions. Swine respond adaptively to environmental stresses in order to survive and reproduce in adverse surroundings. These reactions take the form of changes in functions, structures, and behaviors. Unfortunately, some of them are counterproductive in terms of swine health and performance.

The surroundings in which most swine are raised today differ greatly from those prevalent as recently as 20 years ago. A pork producer's decision as to which production plan to use is based on several factors in addition to the health and productivity of the swine themselves. Of course, fundamental defects of the feeder pig finishing system (viz., mixing pigs from several sources) and of the continuous farrow-to-finish system (viz., the temptation to use facilities continuously, without sanitation breaks) are reflected to some extent in traditional economic analyses. Not addressed adequately in such approaches, though, are the differences in technological requirements of different production systems. This is especially so in regard to environment and disease. For a given farm, management ability is fixed, and differences in productivity among similar operations owes more to variation in management level than any other factor (Backstrom, 1973).

Given the opportunity, swine seek shelter and alter their microenvironments and schedules insofar as possible to achieve comfort. The more intensive the production system, the fewer options and resources the pigs ordinarily have in these regards. Thus, in modern systems, the manager must know the animals' needs and how to meet them.

Intensification of swine production has been followed by new challenges in environmental and disease management. Deficiencies in some operations are immediately apparent. However, first appearances can be deceiving; e. g., the healthy, productive environment for pigs provided by the lowly ventilated, crowded "sweat house" still used in Ireland. Thus, we must learn more, as well as apply more of what we know and learn, about the ecology and ethology of swine.

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ENVIRONMENTAL EFFECTS ON SWINE HEALTH

Direct environmental effects on performance of healthy swine are well-recognized. Most attention has been paid to effects of the thermal environment on survival, growth, and feed-conversion efficiency (Curtis, 1981, 1982). Some direct pathologic consequences of stresses--such as the porcine stress syndrome (Marple and Judge, 1976), cardiac infarcts (Johansson et al., 1974), and fundic gastric ulcers (Backstrom and Bjorklund, 1974)--have been characterized, too. Hence, the environment can affect swine performance not only directly, but indirectly via influences on health, as well (Backstrom and Curtis, 1981).

A new class of diseases has also arisen: the syndromes of multiple etiology, known variously as production diseases or factorial diseases. These diseases are epitomized by the finding that piglets born to specific-pathogen-free sows and kept under environmental conditions classified as poor, fair, or optimal, respectively, developed pneumonia, the severity of which depended on environmental quality (Table 1) (Kalich, 1970). Clearly, clinical manifestations of infectious diseases of swine can be affected by the microenvironment in which the animals reside.

Table 1. Effect of environmental quality in farrowing house on incidence and severity of pneumonia lesions in piglets born to specific-pathogen-free sows and aged 4 to 12 weeks.

Environment of farrowing house										
Air temperature (^{o}F)										
Group	House	Nest	Relative humidity	Air speed (ft/sec)	No. of piglets	Incidence of pneumonia -	±	+	++	+++
A	43-48	54-57	90-95%	.7-1.0	18	0	0	4	10	4
B	48-54	68	70-85%	.7	18	0	4	11	3	0
C	61	86	70%	.3	18	16	2	0	0	0

Source: Kalich, 1970.

Health as Harmonious Balance

Swine can function normally only when they are living in harmonious balance with other living forms and the physical and chemical factors in the environment. Each of these three--animal, biologic environment, and physical environment--affects the others. Thus, when one or more changes, imbalances can arise. Such imbalances sometimes lead to abnormal function, a sign of disease. Hence, in the broad sense, the physical and biologic environments and the pig itself--together--are codeterminants of infectious diseases.

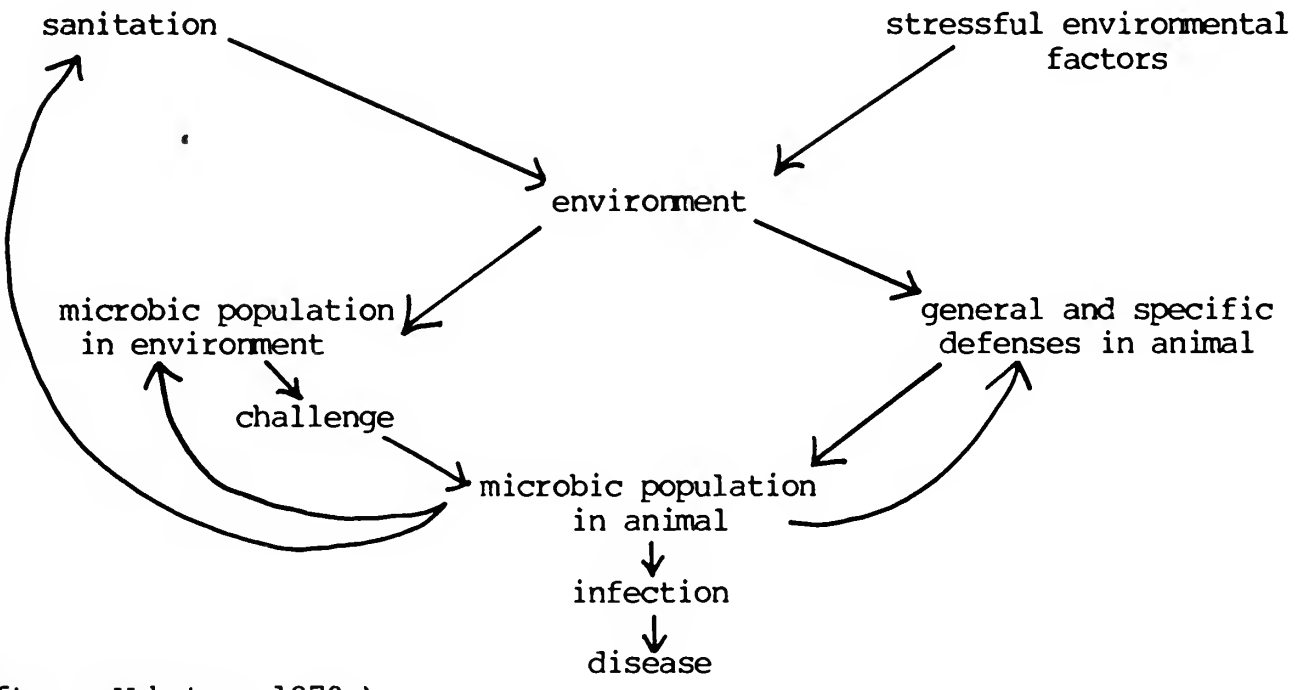
More specifically, disease results from the action of a primary pathogenic agent on a given animal in a given environment. Again, three factors--primary pathogenic agent, environment, and animal--combine to determine whether disease

occurs or not. The etiology of a specific case of disease--the information necessary to reduce chances of its occuring in the future--is complete only when all three components are considered.

The primary pathogenic agent is itself a part of the animal's environment. It might be a toxic substance, e. g., a noxious gas; a thermal-environmental factor, e. g., solar radiation; or a parasite, e. g., a bacterium, a virus, or a worm. The action of the primary pathogenic agent represents a direct challenge of the environment to the animal's continued existence. The animal resists such challenges with a variety of defense mechanisms, all of which are costly in terms of the animal's energy and nutrient balances. Disease ensues if these defenses are impaired or otherwise inadequate. If the defenses are successful, either disease does not occur or it is eventually overtaken.

The other environmental factors combine to influence both the specific primary challenge and the animal's defenses. These are secondary pathogenic agents. They either enhance the primary agent's challenge or impair the animal's defenses. Either way, in effect, they predispose the animal to the debilitating effects of the primary agent. Via these effects, the total environment influences the frequency and the severity of infectious diseases. Secondary pathogenic agents might be any sort of environmental factor: a living vector can aid in transmitting a pathogenic microbe to a susceptible animal; dust can carry into the lungs an irritating gas that otherwise would have been absorbed in the mucus lining the upper respiratory passages; cold stress can reduce an animal's defenses so an opportunistic microbe that had been inhabiting the animal without rendering harm during a nonstressful period can fulfill its pathogenic potential.

Figure 1. Factors associated with infectious disease in an animal.



(After: Webster, 1970.)

The equilibrium between an animal and a population of infectious microbes, and the ways the surroundings can affect this equilibrium, are complicated (Figure 1). Since thousands of years ago, environmental stresses have been known to alter disease resistance. In the meantime, such casual observations have not often been substantiated or refuted by scientific experimentation. Yet Louis Pasteur himself--since deified as a founding father of the germ theory of infectious disease--recognized that environmental stresses could influence the course of a bacterial infection. He demonstrated that the resistance of young chickens against Bacillus anthracis was reduced when body temperature was lowered from 108° to 100° F.

The Challenges and the Environment

The ultimate source of a microbic agent of disease is an infected animal. The environment transports pathogenic microbes between animals and also determines the microbe's survival outside a host animal. Crowding the animals tends to enhance the spread of infectious agents via direct contact. Environmental management is even more crucial in indirect transmission, in which the microbes must survive an environmental excursion with virulence remaining intact to fulfill their mission. The environment affects the challenges of microbic pathogens to swine health (Backstrom and Curtis, 1981). Environmental factors which are important in this respect include aerial vapor pressure, utraviolet radiation, and various animate and inanimate vectors (Curtis, 1983).

One critical, often neglected facet of disease management in swine production is basic sanitation. There is striking evidence in favor of a sanitation break in farrowing rooms, for example. In one study, during the 6 months preceding such a break, the incidence of diarrhea in newborn piglets was 33 percent, and deaths from it almost 4 percent; during the first 6 months after the sanitation break, these had fallen to 11 percent and less than 1 percent, respectively (Nielsen et al., 1976). Other researchers depopulated and thoroughly disinfected a farrowing house after collecting data on the last 30 previous litters in a continuous series of farrowings (Pepper and Taylor, 1977). Then they collected data on the first 30 and second 30 litters farrowed in the facility after the sanitation break. Depopulation and disinfection led to markedly improved piglet performance (Table 2). But there also was evidence that disease build-up started immediately after the break, suggesting that sanitation breaks should be made as frequently as feasible.

Table 2. Results in terms of piglet performance of a depopulation/disinfection-break in a farrowing house.

	30 litters preceding break	First 30 litters after break	Second 30 litters after break
Ave. litter size (no. at 8 wk)	7.6	8.2	8.0
Ave. pig weight (kg at 8 wk)	11.2	13.9	12.3
Mortality (percent at 8 wk)	18.3	13.7	15.2

Source: Pepper and Taylor, 1977.

The Defenses and the Environment

It is practically impossible to eliminate stress in animal production. The problem of stress-altered disease resistance must be dealt with continually. Although it is generally agreed that stress influences an animal's defenses against infectious agents, unequivocal quantitative evidence is still difficult to obtain. Indeed, little is yet available, because the phenomenon involves relations among complex entities--environment, immunity, and infectious diseases.

The focus of the rest of this paper will be the effects of environmental elements on the resistance side of the infectious disease teeter-totter. A variety of common environmental stressors--including cold and hot environments, crowding, mixing, weaning, limit-feeding, noise, and movement restraint--are known to alter animals' defenses against infectious microbes (Kelley, 1980). In particular, the susceptibility of swine to specific infections, and the courses and effects of these infections, are known to be influenced by specific stressors (Kelley, 1982).

Pulmonary Bacterial Clearance and Various Stressors

Swine normally can clear bacteria that have deposited in their lungs and thus keep the lungs relatively sterile. Respiratory-tract defenses include alveolar phagocytes and the mucociliary apparatus. However, various stressors --including cold exposure, aerial ammonia, and the migration of ascarid larvae through the lungs--depress pulmonary bacterial clearance processes, presumably predisposing the pigs to pulmonary infections (Curtis et al., 1976; Drummond et al., 1978; Tisch et al., 1980).

Aerial Ammonia and Specific Infections

The courses and effects of respiratory diseases are also influenced by atmospheric ammonia. For example, effects of stresses from ammonia and early ascarid infection on the growth of young pigs were additive (Drummond et al., 1981b). Either condition alone depressed body-weight gain by around 30 percent; when they were combined, growth rate was reduced by 60 percent, even though the air pollutant did not intensify the liver and lung lesions left by the migrating ascarid larvae. On the other hand, the severity of nasal-turbinate and lung lesions due to infection of young pigs by a pneumotropic strain of Bordetella bronchiseptica was directly related to the aerial concentration of ammonia, but growth rate--which was depressed by the infection alone--was not affected additionally by the ammonia (Drummond et al., 1981a).

Some Principles of Immunology: A Digression

As a prelude to characterizing some of the known environmental influences on specific immune events, it will be useful to review a few of the essential terms and phenomena in immunology. Several books--including Essential Immunology by Ivan Roitt (1977)--give fuller treatment of the following and additional related topics.

Immunity is a special facet of an animal's defenses against infectious diseases. It depends on three traits of the immune system: memory, specificity, and recognition of "non-self". Immunity is adaptive; it is developed in response to a challenge from the environment. When an animal first contacts an antigen (something, such as a toxin or a microbe, capable of eliciting an immune response), its primary response is to produce some antibodies, which can combine with the antigen so as to eliminate it. At the same time, the animal's immune system responsiveness is primed as the antigen's nature is committed to the system's memory. Then, the next time the animal's lymphoid cells contact that antigen appropriately, they mount a secondary response—a much faster, much greater production of antibodies than at first. This phenomenon is used in vaccination schemes: the initial contact takes place in a carefully controlled way and is intended mainly to prime the immune system's memory; then, if and when the animal contacts the antigen again, it can respond quickly, specifically, and powerfully.

The immune system's memory is indeed specific; it can differentiate precisely among different antigens. And, finally, the animal's immune system learns what is "self", which it must tolerate if it is not to destroy itself. Everything else is recognized as "non-self", and is more or less antigenic.

Antibodies are special proteins called immunoglobulins. There are several classes and subclasses of immunoglobulins, based on general structure. Some structural variations are related to where in the body the immunoglobulin occurs, others to antibody specificity.

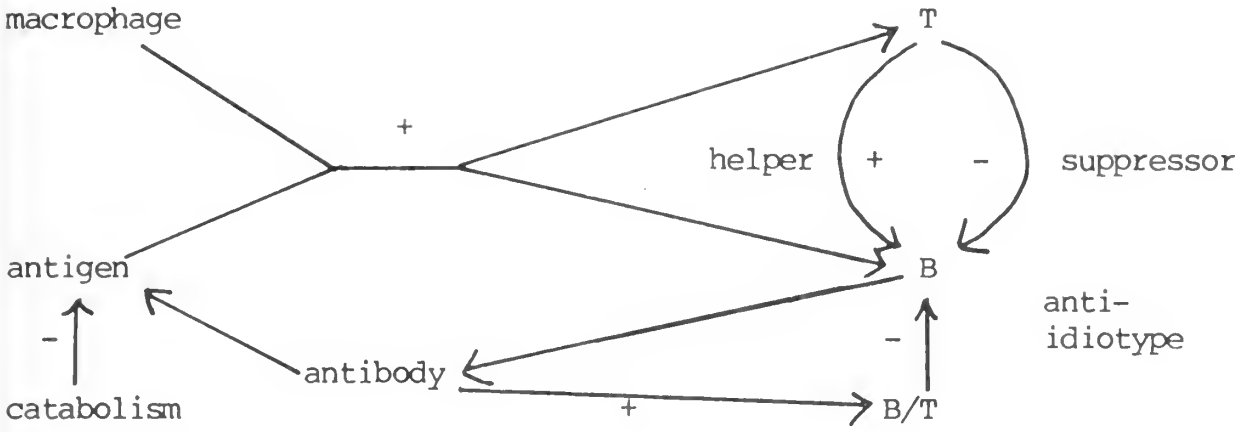
When an antigen contacts an animal's lymphoid cells in an appropriate way, one of two kinds of immunity occurs. Humoral or antibody-mediated immunity is the synthesis and release of antibodies by plasma-cell lymphocytes into body fluids, where they may react directly with antigen. Cell-mediated immunity, on the other hand, is the production of sensitized lymphocytes, which have antibody-like molecules on their surface, and which are involved in immune reactions such as (a) rejection of transplanted tissues and (b) delayed hypersensitivity, as in the tuberculin test.

The two kinds of immunity just mentioned involve two populations of lymphocytes, called B and T, respectively. Stem cells are undifferentiated lymphoid cells produced in the bone marrow. They are the precursors of all blood and reticuloendothelial cells, including both B- and T-lymphocyte lines. Those which differentiate and mature in the bone marrow (mammals) or bursa of Fabricius (birds) become B-lymphocytes, while those dependent for transformation on the thymus gland become T-lymphocytes. When stimulated by an effective antigen, B-lymphocytes proliferate and develop into a series of plasma cells, which are responsible for the production and secretion of circulating and secretory antibodies. Antigen-stimulated T-lymphocytes, on the other hand, proliferate and transform to a series of lymphoblasts, which are responsible for cell-mediated immunity and participate in the regulation of immune function, but secrete little if any free antibody. Division and transformation of T- and B-lymphocytes can also be stimulated nonspecifically by appropriate artificial mitogens.

Immune phenomena must be regulated, and this sometimes takes the form of collaboration between T- and B-cells. It is known, for example, that separate T-cell subpopulations play helper and suppressor roles, respectively, in regard to antigenic stimulation of certain B-lymphocytes. Other relations have been

characterized, too (Figure 2). Environmental stressors seem to act at several points of interaction among lymphoid cell subpopulations.

Figure 2. Some features of the regulation of immune function.



(After: Roitt, 1977.)

Cold Stress and Passive Immunity in Piglets

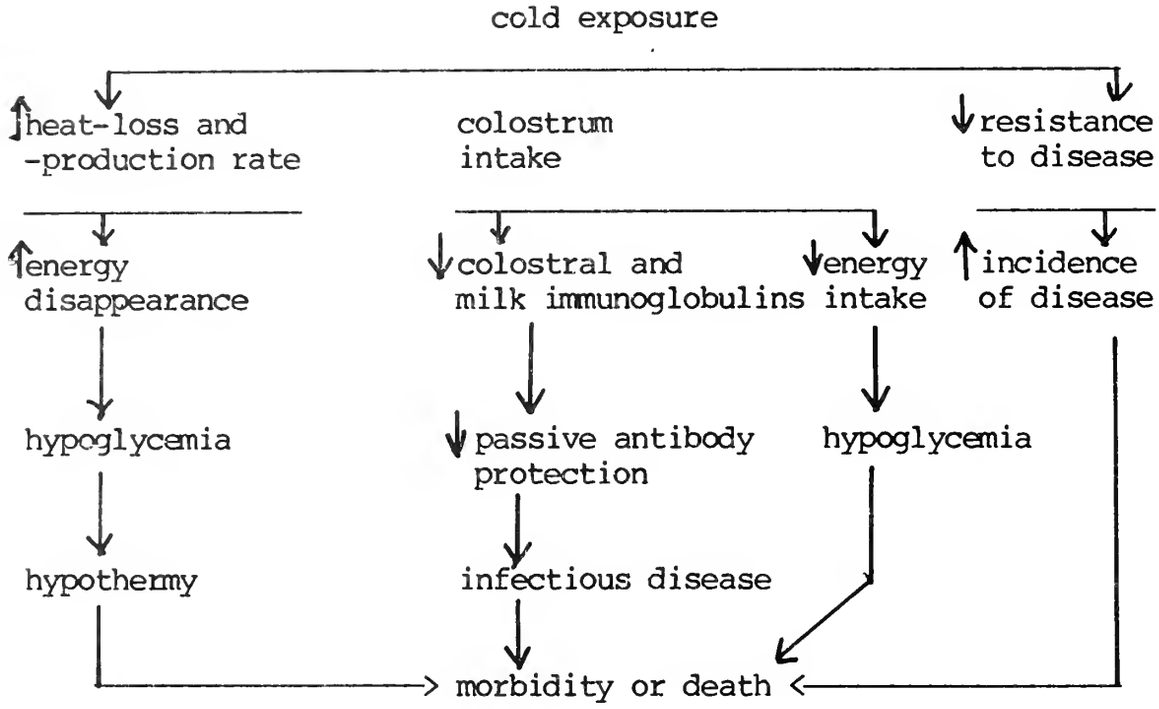
Piglets are born having a very low blood concentration of antibodies (immunoglobulins). They raise the blood antibody concentration via immunoglobulins from sow colostrum ingested during the first postnatal day. These large colostral-immunoglobulin molecules are localized, internalized, and transported through intestinal epithelial cells and discharged into the blood by way of the lymphatic system. Many of the piglets that die before weaning had blood colostral-immunoglobulin concentrations that were significantly lower than normal a couple of days after birth (Hendrix et al., 1978; Yaguchi et al., 1980).

Furthermore, when newborn piglets are exposed to a cold environment and then returned to the sow, they have lower levels of circulating immunoglobulins than do those kept in thermoneutral surroundings (Parker et al., 1980; Blecha and Kelley, 1981a). Subsequent research has revealed that this cold-induced reduction in passively derived serum immunoglobulins is not due to any defect in the piglet's ability to transport immunoglobulins from the intestinal lumen into the blood (Kelley et al., 1982a). Instead, it seems that exposure to a cold environment simply reduces the amount of colostrum a piglet takes in (LeDividich and Noblet, 1981).

Newborn piglets have a high lower critical temperature of around 93°F (Curtis, 1982). They are fragile during the first day after birth, but thermostability improves markedly during the second and third postnatal days (Curtis et al., 1967). The immunoglobulin concentration in colostrum falls in a much shorter period. By 4 to 6 hours after parturition, it is only half that at the start of farrowing (Bourne, 1969). Also, the ability of the newborn piglet to absorb colostral immunoglobulins decreases rapidly with age (Lecce, 1973). Therefore, to obtain maximal blood concentration of colostral immunoglobulins, piglets must nurse soon after birth. If they are cold at birth, they are less likely to nurse vigorously. The result is a lower concentration of colostral immunoglobulins in both the gastrointestinal tract and the blood.

A theoretical scheme of effects of cold exposure on piglet mortality is shown in Figure 3. It is well-known that cold stress increases a piglet's heat-loss and -production rates. If unremitted, cold stress eventually leads to a lowering of body temperature, depletion of energy reserves, and death. But cold stress also reduces the piglet's intakes of colostrum and milk. This has two crucial effects in the piglet: (a) a reduction in energy intake, which

Figure 3. How cold stress increases morbidity and mortality in newborn pigs.



(After: Kelley, 1982.)

contributes to the morbid condition of the cold-stressed piglet, and (b) a reduction in the amounts of protective colostral and milk antibodies in the small intestine and the blood. The second effect can occur even when the sow has been vaccinated and is carrying high titers of protective antibodies in the colostrum. Finally, cold stress can reduce the piglet's resistance to infectious diseases directly.

Heat Stress and Passive Immunity

Heat stress during pregnancy has deleterious effects on the passive immunity the sow provides her young (Machado, 1980). Sows were subjected to moderate heat stress during the last two weeks of gestation, but not during or after parturition. The concentrations of cortisol in the blood and colostrum of stressed sows were higher than normal, while those of immunoglobulins in colostrum and milk were lower. The significantly lower immunoglobulin concentration in the blood of day-old piglets from heat-stressed sows might owe to higher colostral cortisol concentration, lower colostral immunoglobulin concentration, or both. In any case, piglets born to sows which have experienced heat stress during late gestation apparently have subnormal immunity against certain infectious diseases.

Cold Stress and Infectious Diseases

When 21-day-old pigs were kept at 82°F and infected with enterotoxigenic Escherichia coli, around a third of them developed clinical symptoms of diarrhea (Armstrong and Cline, 1977). But when the pigs were exposed to a cold environment (50°F) and infected, the number that developed diarrhea doubled. Also, the rate of body-weight gain in cold-stressed diarrheic pigs was a third less than in control pigs. These findings confirm field observations.

The susceptibility of pigs to transmissible gastroenteritis (TGE) virus is also increased by cold stress (Shimizu et al., 1978). When 8- to 12-week-old pigs were held in an 86°F environment, they were resistant to TGE virus. But when infected with the virus and held at 39°F, the pigs became highly susceptible to the virus. When environmental temperature fluctuated between 68°F and 39°F, there was a similar reduction in resistance to TGE virus.

It is not known why cold exposure reduces the resistance of pigs to certain infectious diseases. However, there is substantial evidence indicating that cold exposure affects an animal's immune system (Kelley, 1980). It has been postulated that cold exposure causes changes in critical hormonal parameters. These endocrine changes might then alter the mechanisms that control the induction or the expression of immune processes. The glucocorticoids, for example, are immunosuppressive when present in blood at very high concentrations. Such changes in immune function might then alter the immune status of the young pigs, and thus their susceptibility to infectious diseases.

Cold Stress and Immunoglobulins

Since cold stress increases the susceptibility of pigs to certain infectious diseases, it might be expected that the pig's immune defense mechanisms would be affected negatively by cold weather or sudden downward fluctuations in environmental temperature. Paradoxically, it was found that serum neutralizing-antibody titers to TGE virus were generally higher in cold-stressed, infected pigs than in infected pigs kept in a thermoneutral environment (Shimizu et al., 1978). This puzzling result led to speculation that the TGE virus simply proliferated to a greater extent in cold-stressed pigs. The postulated greater proliferation of the virus, in turn, would provide a greater amount of antigen to stimulate antibody synthesis, which might explain the higher antibody titers found in cold-stressed, infected pigs.

If cold stress increases the antigenic challenge simply by increasing viral proliferation, it would be expected that the cold would not affect antibody titers in pigs vaccinated with killed vaccines or other nonreplicating antigens. Consequently, experiments were conducted with cold-stressed pigs that were vaccinated with a commonly used nonreplicating antigen, sheep red blood cells (SRBC). Five-week-old pigs were put in a 32°F environment immediately after such vaccination and kept there for 4 days (Blecha and Kelley, 1981b). However, even with thus nonreplicating antigen, cold stress caused a 360-percent increase over control pigs at thermoneutrality in passive hemagglutination titers to the SRBC. Most of this increase was mediated by IgM-specific anti-SRBC antibodies. Another interesting finding was that the concentration of immunoglobulin in blood increased by 60 percent in cold-stressed pigs. These results indicated that cold stress had a direct, stimulatory effect on the pig's ability to synthesize antibodies.

The physiologic reasons for these cold-stress-induced increases in serum immunoglobulin concentration and antibody titer are unknown. Neither is it known whether this change in antibody-mediated immunity contributes to pathological lesions in infectious diseases, as in certain autoimmune and hypersensitivity states. But it is clear that cold stress affects antibody-mediated immunity in pigs and other animals, probably by altering regulatory circuits among the particular lymphoid cell subpopulations that control antibody synthesis.

Weaning stress and immune responses. In the experiment just described, it was of interest to learn whether newly weaned pigs were more susceptible to changes in immune responses caused by cold stress than were control pigs weaned previously. Littermate pigs weaned at 21 or 35 days of age were exposed to a cold environment for 4 days at 35 days of age. It was found that cold exposure caused significant increases in antibody titer to SRBC and in circulating immunoglobulin concentration as compared to thermoneutral conditions, regardless of age at weaning. In other words, newly weaned pigs were no more subject to cold-induced changes in antibody immunity than were their littermates that had been weaned two weeks earlier (Blecha and Kelley, 1981b). However, although time of weaning and cold stress did not interact, pigs that were injected with SRBC immediately after weaning at 35 days did have significantly lower antibody titers than did their littermates that were injected at 35 days, but had been weaned at 21 days of age. This change was caused mostly by a reduction in IgM-specific antibody titer to SRBC--a T-cell dependent, B-cell-mediated phenomenon. Clearly, weaning pigs at the time of vaccination can impair their ability to synthesize antigen-specific antibodies.

The reduction in antibody synthesis caused by weaning could be related to changes in T-cell-mediated immune events. A new series of experiments (Blecha et al., 1983a) have demonstrated that the cell-mediated immmune response is suppressed by the weaning of pigs before 5 weeks of age: (a) a significant reduction in vitro of both phytohemagglutinin- and pokeweed-mitogen-stimulated lymphoblastogenesis at 24 hours after weaning and (b) a significant suppression of in vivo responses of newly weaned pigs to phytohemagglutinin. Interestingly, when pigs were weaned at 5 weeks of age, there was very little suppression in either of these two types of immune response.

Taken together, these results suggest that: (a) weaning pigs younger than 5 weeks of age impairs T-cell-mediated immune responses, (b) a slight but significant reduction in B-cell-mediated synthesis of antigen-specific antibody occurs when young pigs are injected with antigen at the time of weaning, and (c) effects of cold stress and weaning on synthesis of antibodies are additive. From a practical point of view, it would appear that at least some of the problems in management of early weaned (e. g., 3-week-old) pigs have to do with suppressed immune functions in these animals.

Cortisol and Lymphoid Cells

It is well-known that acute stressors, such as cold exposure, increase the concentration of the pig's predominant glucocorticoid, cortisol, in the blood. Removing nursing piglets from the sow also increases blood cortisol concentration (Worsaae and Schmidt, 1980). At pharmacologic doses, glucocorticoids have a wide range of effects on lymphoid cells. For instance, cortisol can cause

lysis of lymphocytes, but this lytic effect differs considerably among species. Therefore, there has been speculation as to whether physiologic concentrations of cortisol could be responsible for immune system changes that have been observed in stressed pigs.

Two experiments have been conducted to learn more about this possibility (Kelley et al., 1982b). First, porcine splenocytes, thymocytes, and peripheral blood mononuclear leukocytes were incubated with cortisol at concentrations ranging from 0.04 to 7 X 10^{-6}M (the higher value is the maximum physiologic concentration). Cortisol caused a moderate but significant lysis of splenocytes (16 percent) and thymocytes (11 percent). Viability of mononuclear leukocytes was not affected at this concentration.

In the second experiment, these same porcine lymphoid cells were incubated in the presence of cortisol plus a mitogen--phytohemagglutinin (which seems to stimulate transformation of helper T-cells preferentially) or Concanavalin A (which seems to stimulate transformation of suppressor T-cells preferentially). After 72 hours in culture, mitogenic responses were estimated by measuring the uptake of tritiated thymidine in the DNA of lymphoid cells. At 0.4 X 10^{-6}M, cortisol caused a nearly 50-percent reduction in mitogenesis in splenocytes and thymocytes stimulated by Concanavalin A. Responses of splenocytes to phytohemagglutinin were also significantly reduced by cortisol at this dose.

These results show that physiologic concentrations of cortisol can impair the ability of the pig's lymphoid cells to proliferate in vitro. If there is a similar effect in vivo, then the stress-induced increase in cortisol secretion rate might be related to changes in immune function known to be associated with adverse environmental stimuli.

FUTURE RESEARCH DIRECTIONS

High social stress decreases the chicken's resistance to Newcastle disease virus, Marek's disease virus, and Mycoplasma gallisepticum, while it increases the bird's resistance to Staphylococcus aureus, Escherichia coli, Northern fowl mite, and Eimeria necatrix (Gross and Colmano, 1969, 1971; Gross, 1972; Hall and Gross, 1975; Gross, 1976). How can these opposite effects be explained? Perhaps social distress elevates blood heterophil concentration, thus enhancing the bird's defenses against bacterial and coccidial infections. Another possibility is differential effects of such a psychosocial stress on various lymphoid-cell subpopulations.

Normal regulation of the immune system is an active, dynamic process, controlled by lymphoid cells which either enhance or inhibit various antigen-specific immune processes. Many cell interactions are involved in the immune response. It has been proposed that the function, differentiation, or perhaps the number of these regulatory lymphoid-cell subsets is affected differentially by environmental stressors (Kelley, 1983). This notion is supported by the fact that certain adaptation hormones alter specific functional immune events at physiologic concentrations in vitro. Also, the relative importances of respective lymphoid-cell subsets depends on the nature of the pathogenic insult present.

Only little is known about the undoubtedly complex mechanisms involved in lymphoid-cell regulation and about how antigen-specific subsets of lymphoid cells become involved in specific infectious diseases. It now appears to be most likely that hormonal influences lie at the center of these regulatory phenomena. If the general notion that hormones affect the differentiation and function of lymphoid cells eventually is proven to be correct, then it will still remain to be learned how different components of the immune system can respond differentially to the same stressor, how the same component can respond differentially to different stressors, and even how the same component can respond differentially to the same stressor at different times.

Unless and until the results of basic research unlock more natural secrets about these phenomena, we will not understand the pig's immune system well enough to be able to accurately predict its function under any particular set of environmental conditions.

Current Research and Ideas

Several recent avenues of scientific investigation have yielded results pertaining to ecological/immunological questions in swine production. Brief synopses provide glimpses of what the future might hold as more is learned about the ecology of the immune response.

Movement restraint and immune function. Bodily movements of swine are more or less limited by certain production systems used nowadays in the pork-production industry. Movement restraint affects the clinical outcome of certain infectious diseases. Does it affect cell-mediated immune events in vivo? Two different ways of probing this question have been employed in experiments with mice (Blecha et al., 1982a): (1) delayed hypersensitivity to SRBC antigen and (2) contact sensitivity reaction to dinitrofluorobenzene (DNFB) antigen.

The mice were treated with either SRBC or DNFB one day and then left undisturbed for four days. At day 4, the mice were treated with the respective agents again and their reactions to this re-exposure were measured. In the cases of both SRBC and DNFB, some of the mice were partially immobilized for 2 hours prior to the first treatment, some for 2 hours prior to the retreatment four days later. The results indicate that physical restraint affected cell-mediated immune events in specific, complicated ways: (a) immobilization just prior to vaccination suppressed the response to the subsequent antigen challenge in the case of SRBC, but enhanced it in the case of DNFB, and (b) similarily, immobilization just prior to the challenge four days after the initial treatment reduced the reaction to SRBC, but enhanced that to DNFB. This was the first demonstration that an environmental stimulus can enhance one type of T-cell-mediated event, and at the same time suppress another such event. It might well be that the glucocorticoids are involved in these differential effects (Blecha et al., 1982b).

There is now evidence that several functions of the immune system of the young pig, too, are affected by movement restraint (Mertsching and Kelley, 1983). Further, restraint of the sow via tethering before and after farrowing both (a) suppressed the sow's antibody synthesis in response to SRBC and (b) reduced the quantity of antigen-specific antibodies transferred from colostrum

to the piglets' blood (Oosterlee et al., 1980). The possible practical implications of these findings are obvious.

Thermal stress and immune function. There is now ample evidence that cold stress and heat stress influence immune functions. Either cold stress or heat stress reduced the contact sensitivity reaction to DNFB in chickens. Apparently, thermal stresses can directly inhibit one component of the bird's T-cell-mediated immune response while having little effect on antigen-specific B-cell-mediated immunity (Regnier and Kelley, 1981; Regnier et al., 1980).

Similar phenomena have been found in ungulates. Heat stress supressed the calf's immune reaction to tuberculin greatly. Cold stress affected this reaction differently at different times; acute cold stress caused immunoenhancement, while chronic cold stress resulted in immunodepression (Kelley et al., 1981a; Kelley et al., 1982c).

Cold stress enhanced young pigs' antibody-synthesizing capability and increased their blood immunoglobulin concentration (Blecha and Kelley, 1981). Heat stress reduced in vivo proliferation of peripheral lymphocytes in response to T-cell mitogens in pigs (Jensen et al., 1983).

Shipping stress and immune function. It has been found that transportation inhibits certain T-cell-mediated immune phenomena and phagocytic functions in calves (Kelley et al., 1981b; Blecha et al., 1983b).

Other recent avenues of investigation. The regulation of immune functions, and the implications and ramifications of environmental stressors in this regard, are being scrutinized in many laboratories around the world today (Kelley, 1983). Immune parameters are even being proposed as measures of animal strain. Involvements of numerous entities in the regulation of immune function are being investigated. Among these are neural and neuroendocrine components of the central nervous system, adrenocorticotropin and the adrenocortical steroids, the catecholamines, and β-endorphin. New biotechnological methods have been developed to make it possible to conduct more sophisticated research on lymphoid cell subpopulations. These include monoclonal antibodies, useful in identifying the subpopulations; fluorescence-activated cell sorting; and cloning.

A wide variety of environmental stressors--including biological, physical, chemical, even psychological stressors--can change an animal's resistance to infectious diseases. Exactly how this happens is not known. The use of basic scientific techniques developed in recent years--coupled with more traditional neurobiological, endocrinological, and immunological approaches--holds much promise for gaining in the near future a better understanding of how the environment influences regulatory signals among lymphoid cell subpopulations.

LITERATURE CITED

Armstrong, W. D., and T. R. Cline. 1977. Effects of various nutrient levels and environmental temperatures on the incidence of colibacillary diarrhea in pigs: intestinal fistulation and titration studies. J. Anim. Sci. 45:1042.

Backstrom, L. 1973. Environment and animal health in piglet production: a field study of incidences and correlations. Acta Vet. Scand. Supp. 41.

Backstrom, L., and N. -E. Bjorklund. 1974. Gastric ulcers in swine: a study of incidences under different environmental conditions at abbatoir before slaughter. Proc. XII Nord. Vet. Cong.

Backstrom, L., and S. E. Curtis. 1981. Housing and environmental influences on production. In: Diseases of Swine. Iowa State Univ. Press, Ames.

Blecha, F., and K. W. Kelley. 1981a. Cold stress reduces the acquisition of colostral immunoglobulin in piglets. J. Anim. Sci. 52:594.

Blecha, F., and K. W. Kelley. 1981b. Effects of cold and weaning stressors on the antibody-mediated immune response of pigs. J. Anim. Sci. 53:439.

Blecha, F., et al. 1982a. Stress-induced alterations in delayed-type hypersensitivity to SRBC and contact sensitivity to DNFB in mice. Proc. Soc. Exp. Biol. Med. 169:239.

Blecha, F., et al. 1982b. Adrenal involvement in the expression of delayed-type hypersensitivity to SRBC and contact sensitivity to DNFB in stressed mice. Proc. Soc. Exp. Biol. Med. 169:247.

Blecha, F., et al. 1983a. Weaning pigs at an early age decreases cellular immunity. J. Anim. Sci. 56:396.

Blecha, F., et al. 1983b. Influences of shipping on immunocompetency in feeder calves. J. Anim. Sci. 57(Supp. 1):384. (Abst.)

Bourne, F. J. 1969. Studies on colostral and milk whey proteins in the sow. 1. The transition of mammary secretions from colostrum to milk with natural suckling. Anim. Prod. 11:337.

Curtis, S. E. 1981. Pig energetics--the diet and the environment. Proc. 1981 Pork Industry Conf., Univ. of Illinois at Urbana-Champaign.

Curtis, S. E. 1982. Baby pig management: birth to weaning. Proc. Symp. Management of Food Producing Animals. Purdue Univ., West Lafayette, Indiana.

Curtis, S. E. 1983. Environmental Management in Animal Agriculture. Iowa State Univ. Press, Ames.

Curtis, S. E. et al. 1967. Age dependent changes of thermostability in neonatal pigs. Am. J. Vet. Res. 28:1887.

Curtis, S. E., et al. 1976. Effects of cold stress and age on pulmonary bacterial clearance in young pigs. Am. J. Vet. Res. 37:299.

Drummond, J. G., et al. 1978. Effects of atmoshperhic ammonia on pulmonary bacterial clearance in young pigs. Am. J. Vet. Res. 39:211.

Drummond, J. G., et al. 1981a. Effects of atmospheric ammonia on young pigs experimentally infected with Bordetella bronchiseptica. Am. J. Vet. Res. 42:963.

Drummond, J. G., et al. 1981b. Effects of atmospheric ammonia on young pigs experimentally infected with Ascaris suum. Am. J. Vet. Res. 42:969.

Gross, W. B. 1972. Effect of social stress on occurrence of Marek's disease in chickens. Am. J. Vet. Res. 33:2275.

Gross, W. B. 1976. Plamsa steroid tendency, social environment and Eimeria necatrix infection. Poul. Sci. 55:1508.

Gross, W. B., and G. Colmano. 1969. The effect of social isolation on resistance to some infectious diseases. Poul. Sci. 48:514.

Gross, W. B., and G. Colmano. 1971. Effect of infectious agents on chickens selected for plasma corticosterone response to social stress. Poul. Sci. 50:1213.

Hall, R. D., and W. B. Gross. 1975. Effect of social stress and inherited plasma corticosterone levels in chickens on populations of Northern fowl mites, Ornithonyssus sylviarum. J. Parasitol. 61:1096.

Hendrix, W. F., et al. 1978. Porcine neonatal survival and serum gamma globulins. J. Anim. Sci. 47:1281.

Jensen, M. A., et al. 1983. Effect of fluctuating hot temperatures on performance and immunity in finishing hogs. J. Anim. Sci. 57(Supp. 1):173. (Abst.)

Johansson, G., et al. 1974. Severe stress-cardiopathy in pigs. Am. Heart J. 87:451.

Kalich, J. 1970. Untersuchungen ueber die Beziehungen zwischen enzootischer Pneumonie (Ferkelgrippe) und Umwelt. Muench. Tieraerztl. Wschr. 83:289.

Kelley, K. W. 1980. Stress and immune function: a bibliographic review. Ann. Vet. Res. 11:445.

Kelley, K. W. 1982. Environmental effects on the immune system of pigs. Pig News and Information 3:395.

Kelley, K. W. 1983. Immunological consequences of changing environmental stimuli. Proc. Int. Conf. Animal Stress: New Directions in Defining and Evaluating Effects of Stress. Univ. of California, Davis. (In press.)

Kelley, K. W., et al. 1981a. Acute cold stress in zeranol-implanted steers. J. Anim. Sci. 53 (Supp. 1):184. (Abst.)

Kelley, K. W., et al. 1981b. Whole blood leukocyte vs. separated mononuclear cell blastogenesis in calves: time-dependent changes after shipping. Can. J. Comp. Med. 45:249.

Kelley, K. W., et al. 1982a. Cold exposure and absorption of colostral immunoglobulins by neonatal pigs. J. Anim. Sci. 55:363.

Kelley, K. W., et al. 1982b. Physiologic concentrations of cortisol suppress mitogenesis of porcine lymphoid cells. J. Anim. Sci. 55 (Supp. 1):19. (Abst.)

Kelley, K. W., et al. 1982c. Effect of chronic heat and cold stressors on plasma immunoglobulin and mitogen-induced blastogenesis in calves. J. Dairy Sci. 65:1514.

Lecce, J. G. 1973. Effect of dietary regimen on cessation of uptake of macromolecules by piglet intestinal epithelium (closure) and transport to the blood. J. Nutr. 103:751.

LeDividich, J., and J. Noblet. 1981. Colostrum intake and thermoregulation in the neonatal pig in relation to environmental temperature. Biol. Neonate 40:167.

Machado, R. 1980. Effects of elevated environmental temperature on passive immunity in swine. Ph.D. Thesis, Univ. of Illinois at Urbana-Champaign.

Marple, D. N., and M. D. Judge. 1976. Porcine stress syndrome. Pork Industry Handbook, PIH-26.

Mertsching, H. J., and K. W. Kelley. 1983. Restraint reduces size of thymus gland and PHA skin swelling in pigs. J. Anim. Sci. 57 (Supp. 1): (Abst.)

Nielsen, N. C., et al. 1976. Prevention and Therapy of Swine Diseases. Royal Veterinary and Agricultural Univ., Copenhagen.

Oosterlee, C. C., et al. 1980. Influence of housing systems on the immune response of sows, colostral transfer of antibodies and behaviour of piglets. Paper No. M2.6, 31st Ann. Meeting Europ. Ass. Anim. Prod.

Parker, R. O., et al. 1980. Serum concentration changes in protein, glucose, urea, thyroxine and triiodothyronine and thermostability of neonatal pigs farrowed at 25 and 10°C. Can. J. Anim. Sci. 60:503.

Pepper, T. A., and D. J. Taylor. 1977. Breeding record analysis in pig herds and its veterinary applications: experience with a large commercial unit. Vet. Rec. 101:196.

Regnier, J. A., and K. W. Kelley. 1981. Heat- and cold-stress suppresses *in vivo* and *in vitro* cellular immune responses of chickens. Am. J. Vet. Res. 42:294.

Regnier, J. A., et al. 1980. Acute thermal stressors and synthesis of antibodies in chickens. Poul. Sci. 59:985.

Roitt, I. 1977. Essential Immunology. Blackwell, Oxford.

Shimizu, M., et al. 1978. Effects of ambient temperatures on induction of transmissible gastroenteritis in feeder pigs. Infec. Immun. 21:747.

Tisch, D. A., et al. 1980. Effects of Ascaris suum larval migration on pulmonary bacterial deposition and clearance in the weanling pig. J. Anim. Sci. 51 (Supp. 2):159. (Abst.)

Webster, A. J. F. 1970. Environmental and physiological interactions influencing resistance to infectious disease. In: Resistance to Infectious Disease. Modern Press, Saskatoon.

Worsaae, H., and M. Schmidt. 1980. Plasma cortisol and behavior in early weaned piglets. Acta Vet. Scand. 21:640.

Yaguchi, H., et al. 1980. Studies on the relationship between the serum gamma globulin levels of neonatal piglets and their mortality during the first two months of life: an evaluation for the ammonium sulphate reaction. Brit. Vet. J. 136:63.

Feeding Antimicrobials to Control Atrophic Rhinitis in Swine

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Introduction

Atrophic rhinitis (AR) was first described in Germany in 1830 (Pond and Maner, 1974), then was thought to have spread globally from Europe through the practice of importing breeds of swine for genetic improvement. AR was not reported in the United States until 1940, but at present is widespread. Dunn (1969) reported a 25% prevalence of AR in over 1600 swine slaughtered in Nebraska from 1962 to 1969. A 25 - 50% infection level is not uncommon in many commercial herds throughout the United States. Ross et. al. (1967) demonstrated that AR could be successfully transmitted to pigs from rats, rabbits, and cats. This observation adds to the difficulty encountered in eradicating the disease in infected herds. Many European countries including the United Kingdom have observed periodic outbreaks of AR, but rigid quarantine followed by slaughter of infected animals was successful in reducing the disease to a manageable level.

Experimental evidence links the bacterium Bordetella bronchiseptica with AR in swine. Other agents have also been incriminated (Pasteurella sp., Hemophilus sp., viruses) and environmental irritants may increase severity of lesions (ammonia, pit gases).

At lease two explanations can be offered to account for the severe snout distortion. In young pigs the nasal turbinates may fail to attain normal size and structure as a result of infection. This condition is referred to as turbinate hypoplasia. When pigs are infected with Bordetella later in life, a true atrophy or shrinkage of normally developed turbinate bones may occur. In either event, Bordetella produces toxins that inhibit or disrupt normal bone mineralization in the turbinates.

Bordetella has been sensitive to the sulfonamide drugs, sulfamethazine, and sodium sulfathiazole (Switzer, 1963), and vaccines prepared against some specific strains of the bacterium. Unfortunately, neither sulfonamide drugs nor specific vaccines are completely effective in eliminating AR in infected herds. The effectiveness of vaccination has been extremely variable and post-vaccination problems with absesses in sows and piglets have occurred sporadically. Resistance to sulfonamides also seems to be increasing (Table 1) and this has been postulated as a factor in treatment failure.

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Table 1. Antibiotic Sensitivity of 138 B. bronchiseptica from 19 Herds (11 with Clinical AR)

Antimicrobial agent	No. Sensitive/Resistant	% Resistant
Potentiated Sulfa	79/59	42.8
Sulfamethoxazole	64/74	53.6
Tetracycline	138/0	0
Erythromycin	137/1	0.7
Chloramphenicol	135/3	2.2
Penicillin	0/138	100

Smith, I.M., et.al., (1980), Vet. Rec., 106, 462-463.

Data measuring the effects of feed antimicrobials on AR are limited. Feed antimicrobials are generally not absorbed intact from the intestine and are not expected to function systemically although they favorably affect the health of swine when used according to recommendations. There are a wide variety of these drugs and each carries FDA clearance together with experimentally proven efficacy against one or more diseases. All beneficial antimicrobials combat disease producing agents for which they are specific, but some drugs appear to affect or control diseases for which they are not specific. This occurrence may be termed as a favorable side-effect.

Experiments have been conducted over the past ten years to evaluate the effectiveness of several feed antimicrobial compounds in controlling the occurrence and severity of AR in pigs infected by natural exposure to Bordetella. These experiments also tested the hypothesis that drugs nonspecific for Bordetella might control the harmful effects of Bordetella.

General Experimental Design

Approximately 96 weaned pigs averaging 25 pounds were allotted to one of 4 treatments on the basis of litter, origin, sex and bodyweight. Each of the 4 treatments contained 6 replicate pens with 4 pigs assigned per pen. Treatments were assigned at random to complete blocks for statistical comparison and consisted of the following:

Group 1. Basal corn-soybean meal control diet containing no antibiotics/antimicrobials. The protein levels in the basal diet were 19% (starting), 16% (growing), and 14% (finishing).

Group 2, 3, 4. Basal diet plus various feed antimicrobials. Label restrictions and withdrawal times were observed.

Starting, growing and finishing diets were fed until pigs assigned each treatment had attained 75, 120, 220 pounds bodyweight, respectively.

At approximately 40 pounds bodyweight, nasal swabs were made from one pig in each pen. Clinical tests revealed the presence of Bordetella bronchiseptica in approximately 30% of the random group sampled. Following slaughter, the snout from each pig was transected at the level of the first upper premolar.

Hypoplasia and/or atrophy of the turbinates in the cross-sections were quantitated by measuring the space (mm) between the turbinates and the adjacent wall of the nasal passage. Measurements reflecting turbinate shrinkage or absence are reported according to the following criteria:

Hypoplasia and/or Atrophy (mm)	Turbinate Classification	Severity Score
1-3	Normal	0
3-6	Mild	1
6-9	Moderate	2
>9	Severe	3

A snout cross-section revealing greater than 3 mm of turbinate shrinkage was accepted as positive diagnosis of AR. When the snouts from all pigs within a treatment group were measured, a corresponding AR index was calculated using the following equation:

$$I = \frac{\Sigma X}{N}$$

where: I = AR index
ΣX = sum of severity score (0, 1, 2, 3)
N = number of animals in the treatment

Data from the randomized complete-block design were subjected to an analysis of counts chi-square using 4 x 2 contengency (Little and Hills, 1972).

Results and Discussion

Tables 2-7 summarize data from nasal turbinate measurements. Generally, the number of pigs with severe turbinate hypoplasia/atrophy was higher ($P<.025$) in groups receiving the unmedicated diet when compared with those fed medicated diets. The number of animals receiving normal turbinate scores and those displaying mild or moderate turbinate damage did not differ ($P<.05$) among treatments. AR indices did not differ ($P<.05$) among medicated treatments.

Table 2. Effect of Lincomycin (40 and 100 Gm/T) on AR Lesions

	Control (%)*	40 Gm/T. (%)	100 Gm/T. (%)
Average AR Index	1.75 (93.8)	1.35 (76.5)	1.19 (68.8)

Hammell and Becker (1975).

Table 3. Effect of Mecadox Antimicrobials on AR Lesions

	Control(%)*	Mec/Oleando(%)	Mec/Terra 50(%)	Mec/Terra 150 (%)
Average AR Index	1.5 (88.9)	1.04 (64.7)	0.89 (61.1)	1.25 (68.8)

Hammell and Becker (1976).

* (%) Percent of pigs with mild, moderate or severe AR lesions.

Table 4. Effect of Various Antimicrobials on AR Lesions

AR Score	Control N	Control (%)*		Mecadox/Terramycin N	Mecadox/Terramycin (%)		Virginiamycin N	Virginiamycin (%)		Lincomycin N	Lincomycin (%)	
0	6	(26.2)		9	(37.5)		7	(30.4)		8	(33.3)	
1	7	(30.4)		11	(45.8)		11	(47.8)		11	(45.8)	
2	3	(13.0)	73.8	4	(16.7)	61.8	4	(17.4)	69.5	4	(16.7)	66.7
3	7	(30.4)		0	(0)		1	(4.3)		1	(4.2)	
Average AR Index	1.48			0.79			0.96			0.92		

Becker and White (1980)

Table 5. Effect of Chlorachel-250 and CSP-250 on AR Lesions

AR Score	Control N	Control (%)*		Chlorachel-250 N	Chlorachel-250 (%)		CSP-250 N	CSP-250 (%)	
0	9	(37.5)		9	(40.9)		7	(29.2)	
1	6	(25)		7	(31.8)		8	(33.3)	
2	5	(21)	62.7%	5	(22.7)	59.1%	8	(33.3)	70.8%
3	4	(16.7)		1	(4.6)		1	(4.2)	
AR Index	1.17			0.91			1.13		

Becker and White (1981).

Table 6. Effect of Mecadox and ASP-250 on AR Lesions

	Control (%)*	Mecadox/Tetracycline (%)		ASP-250 (%)
Average AR Index	2.18 (80.2)	1.46	(62.6)	1.69 (61.1)

Farrington and Shively (1980)

* (%) Percent of pigs with mild, moderate or severe AR lesions.

Table 7. Effect of Bacitracin, Tylan Sulfa, and Flavomycin on AR Lesions

AR Score	Control N	Control (%)*		Bacitracin N	Bacitracin (%)		Tylan/Sulfa N	Tylan/Sulfa (%)		Flavomycin N	Flavomycin (%)	
0	3	(13.0)		6	(26.1)		8	(33.3)		8	(33.3)	
1	12	(52.2)		11	(47.8)		9	(37.5)		10	(41.7)	
2	5	(21.8)	87%	4	(17.4)	73.9%	7	(29.2)	66.7	6	(25.0)	66.7%
3	3	(13.0)		2	(8.7)		0	(00.0)		0	(00.0)	
Average AR Index	1.35			1.09			0.96			0.92		

* (%) Percent of pigs with mild, moderate or severe AR lesions.
Becker and White (1982)

Tables 8 and 9 summarize six experiments representing over 800 pigs. Medicated pigs had an average turbinate score of 0.44 lower then non-medicated groups. Medicated pigs had 14.4% fewer lesions of AR than non-medicated controls.

Table 8. Summary of Turbinate Scores (Six Experiments)

Experiment	Average Turbinate Scores: Non-medicated Controls	Medicated Groups	Difference+
1	1.75	1.27	0.48
2	1.50	1.06	0.44
3	1.48	0.89	0.59
4++	2.18	1.58	0.60
5	1.17	1.02	0.15
6	1.35	1.00	0.35
	9.43	6.82	2.61
Average	1.57	1.14	0.44

+ Difference between control and treated group averages.

++ Non-Florida data (see Table 6).

Table 9. Summary of Percent of Pigs with Mild, Moderate or Severe AR Lesions (Six Experiments)

	% of Pigs with AR Lesions		
Experiment	Non-medicated Controls	Medicated Groups	Difference[+]
1	93.8	72.7	21.1
2	88.9	64.9	24.0
3	73.8	66.0	7.8
4[++]	80.2	61.9	18.3
5	62.7	65.0	-2.3
6	87.0	69.6	17.4
	486.4	400.1	86.3
Average	81.1	66.7	14.4

[+] Difference between control and treated group averages.
[++] Non-Florida data (see Table 6).

The pigs used in experiments 1, 2, 3, 5, and 6 were free of pneumonic lesions. This undoubtedly explains the normal growth and feed:gain performance observed. Straw, 1981, reported no correlation between turbinate scores and average daily gain, but lesions of pneumonia have been correlated with reduced performance.

Vaccination has been suggested as a means of increasing/maximizing immunity to B. bronchiseptica and/or Pasteurella multocida. Various bacterin combinations and vaccination schedules have been devised. Results of vaccination have been variable and somewhat confusing. Cassler and Hill, 1982, reported favorable results using a bacterin produced in Switzerland. Sows/gilts were vaccinated 5 and 2 weeks prior to farrowing. Piglets were not vaccinated. Diluted bacterin and bacterin in oil were also used for comparison. The pigs were slaughtered at 13-14 weeks of age (72-77 days post challenge) and turbinates examined for damage. Results of turbinate scoring are shown in Table 10. The reduction in average AR index is similar to that seen when antimicrobials are used in the feed. This and similar research tends to confirm the growing opinion that AR vaccination should be considered as an adjunct or supplement to overall AR control.

In addition to vaccination and feeding antibiotics, several other management techniques are available that will help control not only AR but respiratory diseases in general. These should be considered and/or used and include 1) maintaining an "old" sow herd, 2) all in-all out farrowing/nursery units, 3) nasal swabbing, culturing and culling of carrier sows, 4) medicated early weaning (MEW), 5) addition of only Specific Pathogen Free (SPF) or Minimal Disease (MD) breeding replacements, and 6) control of piglet diarrhea.

Maintaining a sow herd that is as old as possible will take advantage of the natural nasal clearance of B. bronchiseptica that occurs with age (Table 11). Older sows will also provide higher quality colostrum and are usually better mothers than gilts.

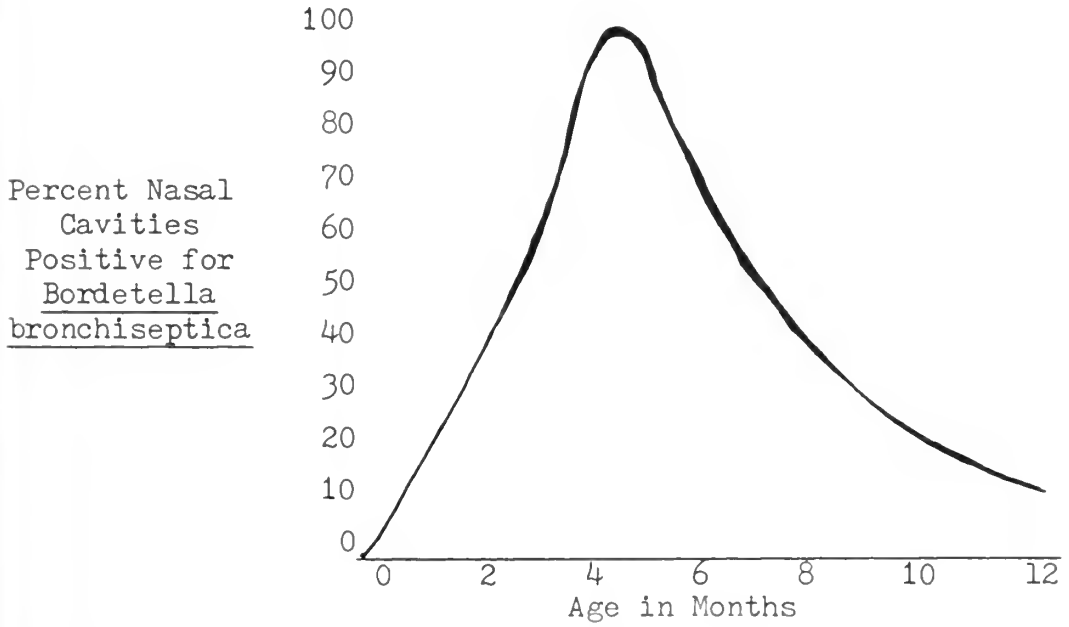
Table 10. Vaccination Effect on AR Lesions

Group	Control			Rhinipig (TM)			Bacterin (Dil. 1:5)			Bacterin (oil)		
	N	(%)*		N	(%)		N	(%)		N	(%)	
0	10	(16)		32	(52)		20	(41)		23	(42)	
1	11	(18)		20	(32)		20	(41)		23	(42)	
2	25	(40)	84%	10	(16)	48%	9	(18)	59%	9	(16)	58%
3	16	(26)		0	(0)		0	(0)		0	(0)	
	109/62			40/62			38/49			41/55		
Average AR Index	1.76			0.65			0.78			0.75		

* ( ) represents % of pigs receiving various AR score.

Cassler and Hill (1982) (modified)

Table 11. Expected Clearing Rate of Bordetella bronchiseptica from the Nasal Cavity of Swine



At 4-10 weeks after farrowing the highest B. bronchiseptica culture positive results occur. By market weight this may be reduced to 50% and by 1 year of age to 10-15%. (Switzer & Farrington, Iowa State Univ., in Hog Farm Mgt., June, 1977).

Mefford et.al. (1983) reported very favorable results when vaccination of sows, gilts and piglets with AR bacterin was combined with injection of piglets with long-acting oxytetracycline (Table 12). Death loss, gain/pig, feed:gain ratio, AR index, lung score and gross profit were all favorably influenced. This represents a form of medicated early weaning and combines the advantages of immunity with those of early medication.

Table 12. Effects of Injectable LA-200™ and BB+PM Bacterin in an Endemic Atrophic Rhinitis Herd

Post Weaning Nursery Results

	G-1	G-2	G-3	G-4
Death Loss %	5.5^{ab}**	8.5^{a}	4.0^{ab}	1.5^{b}
Av. Gain/Pig	25.8^{a}	24.0^{b}	29.8^{c}	35.3^{d}
F/G Ratio	2.31^{a}	2.59	2.1^{a}	2.21^{a}

Marketing, Slaughter Check and Economic Results

	G-1	G-2	G-3	G-4
Av. Turbinate Score	5.25^{a}	5.46^{a}	4.23^{b}	1.9^{c}
Av. Lung Score	3.37^{a}	2.75^{b}	0.65^{c}	0.81^{c}
Market Value of Pigs($)	13,476	14,156(105%)	15,229(113%)	16,775(125%)
Treat. Cost/Pig($)	0	0.78	1.20	2.01
Gross Profit/Control($)	0	7.14	12.30	33.63

* G-1 = Controls (Non-medication, non-vaccinated).
G-2 = Vaccinates (Sows, gilts vaccinated 5 & 2 weeks prefarrowing; piglets vaccinated at 7 & 21 days of age).
G-3 = LA-200 (Piglets injected SC with 1 ml. of LA-200 on 1, 7, 14 and 21 days of age).
G-4 = G-2 + G-3 (Vaccination plus LA-200).

** figures with differing superscripts are significantly different ($P < 0.05$).

Mefford et.al., (1983)

Summary

Many management decisions and techniques are available to the pork producer wishing to control or minimize the effects of AR. Data generated during ten years of research with feed antimicrobials indicates that feeding antimicrobials to swine favorably reduces the severity and extent of AR lesions while enhancing average daily gain and feed:gain ratio. Furthermore, combining vaccination and appropriate medication of the piglet may further reduce the effects of not only AR but pneumonia as well.

Bibliography

Becker, H.N. and Hammell, D.L. 1975. Effect of Lincomycin on Atrophic Rhinitis Lesions. Live Oak ARC Research Report 75-04.

Becker, H.N. and Hammell, D.L. 1976. Effect of Various Antimicrobials on Atrophic Rhinitis Lesions. Live Oak ARC Research Report 76-1.

Becker, H.N. and White, C.E. 1980. Effect of Various Antimicrobials on Atrophic Rhinitis Lesions. Live Oak ARC Research Report 80-2.

Becker, H.N. and White, C.E. 1981. Effect of Chlorachel-250 and CSP-250 on Atrophic Rhinitis Lesions. Live Oak ARC Research Report 81-1.

Becker, H.N. and White, C.E. 1982. Effect of Bacitracin, Tylan/Sulfa and Flavomycin on Atrophic Rhinitis Lesions. Live Oak ARC Research Report 82-1.

Cassler, R.W. and Hill, H.T. 1982. A New Approach to Rhinitis Vaccination. Proc. G.A. Young Conf. Veterinarians, pg. 79-88.

Dunn, J.W. 1969. Pneumonia and Atrophic Rhinitis in Nebraska Swine. Proc. G.A. Young Conf. Veterinarians, pg. 45.

Farrington, D.O. and Shively, J.E. 1980. Effect of Carbadox on Swine Performance in Three Herds with Infectious Atrophic Rhinitis. Proceedings of International Pig Veterinary Society, pg. M15.

Little, T.M. and Hills, F.J. 1972. Statistical Methods in Agricultural Research. University of California Ag. Ext. Service, Berkley, CA.

Mefford, P.E., Swafford, W.S. and Pinkston, M.L. 1983. Proceedings of the Amer. Assoc. Swine Pract., pg. 21-28.

Pond, W.G. and Maner, J.H. 1974. Swine Production in Temperate and Tropical Environments. W.H. Freeman & Co., San Francisco, CA.

Ross, R.F., Switzer, W.P. and Duncan, J.R. 1967. Comparison of Pathogenicity of Various Isolates of B. bronchiseptica in Young Pigs. Can. J. Comp. Med. Vet. Sci., 31, pg. 53-57.

Smith, I.M., Oliphant, J., Baskerville, A.J., Giles, C.J. 1980. High Prevalence of Strains of B. bronchiseptica Resistant to Potentiated Sulphonamide in English Pig Herds in 1978-79. Vet. Record. 106, pg. 462-463.

Straw, B. 1981. Atrophic Rhinitis, Proceedings of Minnesota Swine Herd Health Programming Conference, pg. 11-15.

Switzer, W.P. 1963. Elimination of B. bronchiseptica from the Nasal Cavity of Swine by Sulfonamide Therapy. Vet. Med., 58, pg. 571.

Managing Out Disease

RUSSELL PERKINSON

INTRODUCTION

Tolerance of some level of disease in the swine herd is a necessity, but the degree of tolerance and the adjustment to that situation will be different for each individual swine producer.

As an industry, we have eliminated cholera and kept other diseases out of this country. But as individuals, we must contend with diseases which are common in our hog population. Our success in producing hogs is related, to a large degree, on how well we meet the challenge of disease to our herd.

The presence of disease makes it necessary for the producer to make adjustments in management practices. As I remember my high school physics, to every action, there is an equal and opposite reaction. So, too, disease level in a herd requires those adjustments necessary to make that herd economically competitive.

Many books and scientific treatises have been written on hog management practices. My purpose is to relate one pork producer's efforts to reduce the incidence of disease in his herd. I will tell you something of our farm history and relate some of the problems we have or have not had.

Our herd developed as an ordinary commercial crossbred herd, using several different breeds. We purchased high-indexing boars from test stations and purebred boars from seedstock producers. We never experience a serious disease outbreak or high death loss, but routinely vaccinated our breeding animals for cholera and erysipelas. We also had some arthritic and lung problems, but little or no rhinitis.

I believe it was 1963 when we changed our herd to SPF. Dr. Jason James had started his facility in Sullivan and Dr. Starkey had a similar unit in Waynesville. I took sows from my herd to both places to obtain females for our new breeding herd. I purchased purebred sows to obtain primary boars. My brother had hog facilities but no hogs. I took the young primary pigs to his place and kept them there until shortly before they were to begin farrowing. I had continued normal production on my farm. About two months before the new gilts were to begin farrowing, we began to dispose of the older animals for slaughter and the younger pigs as feeders. We cleaned up the premises and brought our new herd home a week before they were to begin farrowing.

Russell Perkinson is General Manager of Porkville Pork Farm, Thawville, Illinois.

From the time our pigs left the veterinary laboratory, we were diligent in our efforts to keep disease out. Several pairs of overshoes, changing clothes, isolation of contaminated equipment, etc. became a way of life. Boars were purchased from SPF seedstock producers or were taken by C section by Dr. James at Sullivan.

Twenty years later, our herd at Perkinson Pork Farm is still a clean herd--no mange, no lice. My son, Steve, who manages Perkinson Pork Farm, is currently using rhinitis and erysipelas bacterin. Internal parasites are controlled by worming the breeding herd and bacterin to aid in the control of E. coli scours in baby pigs is used in the sow herd. Other diseases have not been a problem. Rhinitis has not been a serious problem, but control measures are probably worthwhile. Cats have been kept for rodent control and possibly have been the source of rhinitis infection.

The development of the technique of interrupting the disease cycle by taking the baby pig from its mother by hysterectomy or C section is a major step toward reducing disease level in a hog herd. Also, improvements in artificial insemination techniques and the development of embryo transfer are significant tools which aid the pork producer in the fight against disease.

Because of our successful experience with the SPF program, there was no doubt about the source of our breeding herd when we started our new unit. Steve's operation at Perkinson Pork Farm did not require my expertise. Steve's two younger brothers, Dave and Doug, and I organized a partnership called Porkville. We drilled a well, found water, called Dr. James to arrange for laboratory facilities, and began construction.

Bred sows from Perkinson Pork Farm were taken to Dr. James at Sullivan to obtain primary SPF gilts and primary boar pigs were obtained by taking sows from a purebred herd to Dr. James. The young pigs were taken from the veterinary facility at 4-5 weeks of age and placed at three different building sites on nearby farms. The pigs were brought to our own buildings as our construction permitted.

We closed our herd to the addition of any stock except primary lab pigs or by embryo transfer. I feel the use of such stock will eliminate one source of disease contamination. There are many excellent herds of registered SPF hogs, and, with the exception of primary C section and embryo transfer, they are the cleanest source of breeding stock available.

Freedom from communicable disease does not eliminate all problems, but it does significantly reduce costs of material and labor for medication and helps us to pinpoint the cause of the problems when they do exist. Of course, E. coli problems are still with us and biologicals to aid in the control of baby pig scours are routinely used. We are not only free of many diseases and external parasites, but fecal examinations have verified that our hogs are free of internal parasites.

When I started planning facilities, it was decided to screen our waste for refeeding to sows and to recycle water for flushing our buildings. There was concern about disease and parasite build-up. We have experienced no such problem, probably because of a low incidence of disease in the recycled flush water. Of course, feeding screened waste to the gestating

sows helps them to give colostrum immunity to the newborn pigs.

Reduced disease level accommodates more variance in management. Our experience indicates that pigs not stressed by normal levels of disease are more able to tolerate other stresses such as poor ventilation, overcrowding, chilling and mixing.

Basic strategy is (1) get a clean herd, and (2) keep it clean. However, most producers may not need or desire to go SPF. The strategy is then (1) good management, and (2) be diligent and disease-conscious. If the management situation is such that repopulation is not being considered, it is desirable to stabilize the disease at a minimum level.

Good hog producers have learned to live with a certain level of disease in the herd, and under good management, undesirable effects of disease can be minimized. Veterinary science has done wonders in the development of products to minimize effects of disease, but good hog care will reduce the need for such products. Disease outbreaks often occur when pigs are stressed by some failure in our management.

I have indicated a few thoughts on having and managing a certain disease level. How do we keep disease out? Disease can be introduced in several ways: (1) hogs, (2) people, and (3) other, e.g., feed, water, rodents, birds, equipment, etc.

(1) Hogs. As we move to confinement, fenceline contact is not the problem it used to be. If you have fenceline contact, then your neighbor's problems may also be yours. For many, however, the purchase of breeding stock can be a major source of new infection. Sources of breeding stock in order of freedom of disease are (1) primary SPF pigs taken by C section under sterile conditions and pigs produced by embryo transfer, (2) registered SPF herds, and (3) non-SPF herds. Use of artificial insemination also has merit in a disease control program. Remember, when you buy a boar or gilt, genetics and disease come in the same package.

(2) People and equipment. Traffic of people in and around hog facilities needs to be restricted for sanitary reasons. Clothing, shoes and equipment can readily spread disease from one farm to another or from market to farm. Salesmen, truckers, neighbors, delivery trucks, etc. should be kept as far as possible away from buildings and lots. Perkinson Pork Farm and Porkville are one mile apart, but we have no traffic between. When I go to Perkinson Pork Farm, I shower and change clothes before returning to Porkville.

Our facilities are arranged so that incoming feed, supplies, etc. are easily segregated from the hog facilities until we feel they are safe to use. Deliveries of bagged material stay in our entrance building several days before being taken further. Bulk feed ingredients are stored several days before being used as feed.

Load-out facilities for market animals are arranged so that contamination from truck loading is prevented. Market hogs are stage-gated as they are loaded; that is, as the hogs proceed out the loading ramp to the truck, gates are dropped behind so that a contaminated animal cannot return to the building.

Waste handling is accomplished without facility contamination. The use of a two-stage lagoon, refeeding of screened waste, and irrigation of flush effluent makes hauling waste and possible contamination by vehicle unnecessary.

Birds, rodents and water are not thought to be a serious threat. Our curtain wall buildings are screened with hardware cloth, and, while we have some sparrow population, starlings have never been a problem. When snow is on the ground, we have no place for them to feed. We have an occasional skunk in our utility building.

Hog production for me, and for many of you, is a way of life. If we were not the kind of people who sought continued improvement, we would not be here today. To raise the healthiest pigs, consistent with our location, our facilities and other constraints, is probably the goal of every pork producer.

Primer in Toxicology

JOHN E. GARST

INTRODUCTION

We are each subjected to a myriad of chemical substances. Over 60,000 chemicals have some present commercial use and countless others are naturally present in our own bodies and the foods we consume (Ames, 1983). Regardless of its source, the existence of any substance in an animal is governed by four processes (Table 1). Distribution, metabolism, and excretion seem designed to diminish toxicity arising from these substances. These processes probably arose in animals from the need to regulate the levels of endogenous, highly

Table 1. Processes through which any substance must pass

1. Absorption into the animal.
2. Distribution throughout the animal.
3. Metabolism to new products.
4. Excretion of the substance.

water-insoluble compounds, like the sex steroids, because of the evolutionary recognition that ANY SUBSTANCE CAN BE TOXIC IN ANY ANIMAL SPECIES, IF THE DOSAGE IS SUFFICIENTLY HIGH. Distribution, metabolism, and excretion can also reduce the concentrations of foreign chemicals, which are encountered through absorption from the environment. It should be evident, however, that toxicity from these exogenous agents can be easily and completely eliminated by simply preventing exposure and the resultant absorption process. However, because of the need for food and because accidents inevitably occur, total prevention of exposure to such substances is simply impossible.

TOXICOLOGY has been defined as the SCIENCE OF POISONS, although the SCIENCE OF ADVERSE BIOLOGICAL SUBSTANCES is perhaps more appropriate today. Traditionally, toxicology has involved the study of TOXICOKINETICS or those processes of ABSORPTION, DISTRIBUTION, METABOLISM and EXCRETION. Increasingly today, much of academic toxicology involves TOXICODYNAMICS, a subdiscipline concerned with elucidation of the mechanisms by which the adverse effects of substances are actually mediated. In analogy, PHARMACOLOGY can be defined as the SCIENCE OF BENEFICIAL BIOLOGICAL SUBSTANCES. Likewise, it is composed of PHARMAKOKINETICS AND PHARMACODYNAMICS. The two sciences share a common network of principles and methodology. Moreover, because we all like to make a "silk purse from a sow's ear," what we discover in TOXICOLOGY has often led to a later pharmacological utility of many naturally-occurring "toxic" substances (Table 2). Indeed, a chemical labelled as a "toxicant" is not necessarily something to fear, since

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EVERYTHING IS TOXIC AT SOME DOSAGE LEVEL IN SOME ANIMAL SPECIES. The two essential questions are: toxicity occurs at what dosage and toxicity occurs in which animal species?

Table 2. Five naturally-occurring toxicants which possess or which have led to beneficial compounds

1.	Colchicine	A plant alkaloid useful in treatment of gout and to produce experimental polyploidy of cells (Shen, 1981).
2.	d-Tubocurarine.	A potent South American arrow poison, which led to development of neuromuscular blockers used in surgery (Stenlake, 1981).
3.	Cocaine.	Although frequently abused today, this South American plant alkaloid led to the development of local anesthetics (Takman and Adams, 1981).
4.	Morphine.	Although also abused, this poppy alkaloid has probably alleviated more pain than it has caused (Johnson and Milne, 1981).
5.	Zearalenone.	The estrogenic effects of this mycotoxin in pigs led to the synthetic congener zearanol, which is being evaluated as a livestock growth promotant (Staigmiller et al., 1983).

This PRIMER introduces the principles of TOXICOKINETICS; the oral presentation will briefly describe the toxicodynamics of selected compounds and will utilize some of these toxicokinetic concepts in discussions of those compounds. There are several good general toxicology books available that discuss these principles in greater detail (Doull et al., 1980; Hodgson and Guthrey, 1980; Timbrell, 1982). Other textbooks deal with more specific agricultural interests, such as mycotoxins (Uraguchi and Yamazaki, 1978; anonymous, 1979a, 1979b) or pesticides (Hayes, 1975). Rather than provide extensive detail, emphasis in this PRIMER has been placed on general information and recent review papers.

TOXICOKINETICS

A. ABSORPTION.

Lien (1981) has recently reviewed physical factors affecting disposition of many chemical agents.

1. Routes of absorption.

   a. Accidental routes of toxicant entry.
      1. Skin has a multilayered structure, which slows absorption (Scheuplein, 1977).

2. Lungs facilitate absorption of carbon monoxide and other gaseous toxicants. Morris et al., (1982) recently reported studies of carbon monoxide toxicosis in swine.

3. Oral administration often affords a lower toxicity than administration by intentional routes. Such quantitative differences depend on the extent of absorption and which tissue first receives the toxicant.

The neutral form of an ionizable compound is preferred for absorption. Thus, acids, which tend to be neutral molecules at low pH, are better absorbed from the stomach. Bases, which tend to be neutral molecules at higher pH, are better absorbed from the intestine.

b. Four intentional routes of toxicant entry.

1. Intraperitoneal-into the peritoneal cavity (i.p.).

2. Intramuscular-into a muscle mass (i.m.).

3. Subcutaneous-just under the skin (s.c.).

4. Intravenous-into the blood supply (i.v.)

2. The physics of the absorption process is discussed by Scheuplein (1977). Passage through membranes leading to absorption involves four mechanisms.

a. Filtration occurs through pores.

b. Carrier-mediated entry processes.

1. Facilitated diffusion proceeds with the concentration gradient.

2. Active transport proceeds against the concentration gradient.

c. Pinocytosis is an engulfment of particles, generally by macrophages.

d. Passive diffusion through lipid membrane constituents is defined by physics.

1. The rate of diffusion is controlled by Fick's Law. The diffusion rate = $kA(C2-C1)/d$, where A is the surface area for the transfer, C2-C1 is the concentration gradient across the membrane, d is the membrane thickness, and k is the diffusion constant (Scheuplein, 1977).

2. Three factors control the diffusion constant k.
   a. Molecular weight and size.
   b. Steric and spatial factors.
   c. Toxicant partition coefficient, P, and the degree of ionization. The importance of this factor is reflected by the

the excellent correlations found between an oil:water log P and anesthesia in the tadpole (Table 3).

Table 3. Minimum alcohol concentration for tadpole anesthesia

Alcohol	*Anesthetic concentration in aqueous media, molarity*	*Log P Cottonseed oil:water*	*Octanol:water*
Methanol	0.57	-2.015	-0.71
Ethanol	0.29	-1.447	-0.28
n-Propanol	0.11	-0.807	+.20
i-Butanol	0.045	-0.231	+.78
Correlation coefficients:		R = -0.964	R = 0.96

Taken from Goldstein et al., (1974) with modifications.

B. DISTRIBUTION

Distribution is controlled by the following four factors.

1. Blood or distribution media uptake rates. Epinephrine contracts blood vessels and delays toxicant and drug uptake.

2. Binding to plasma proteins for distribution is governed by the toxicant octanol:water (o:w) partition coefficient as log P(o:w). Better than a 0.96 correlation coefficient was obtained for 42 chemicals of varied structure and functionality between log P(o:w) and their ability to bind serum albumin (Helmer et al., 1978).

3. Plasma protein bound toxicants are in equilibrium with the free form. Competitive displacement from the binding protein by other preferentially bound agents can create high blood concentrations quickly with toxic consequences.

4. Storage sites in distribution-a protective mechanism and a time-bomb waiting to be released!

   a. Plasma-protein (p.p.) bound. Dieldrin is 99% p.p. bound prior to deposit in fat for longer term storage.

   b. Liver-stores lead and other minerals.

   c. Kidneys-stores cadmium and other minerals.

   d. Fat-rapid mobilization of fat such as occurs in starvation can release toxicants and produce whatever effects are caused by the substance (Hayes, 1975). This approach can also be used to detoxify smaller animals, where starvation time isn't a major factor. Fat animals retain hydrophobic (e.g. water-fearing) agents longer than lean animals.

e. Bone-inorganic and mineral storage. Lead, fluoride, and strontium are extensively bone bound. Concentration in bone is a fairly simple, but reversible ion exchange process. Degradation of bone, which often occurs in old age, can reverse toxicant accumulation and actually release toxic levels of certain minerals.

f. Brain and its age-dependent barrier to toxicant entry (THE BLOOD-BRAIN BARRIER).

1. Capillary endothelial cells are tightly joined with few pores to minimize filtration of lower molecular weight toxicants.

2. Capillaries are surrounded with a low fat, thick, fibrilrich, glial connective tissue, which effectively minimizes hydrophobic diffusion and impedes diffusion.

3. The distribution media of the central nervous system, the cerebral spinal fluid, has a low protein level relative to other tissues. This inhibits protein-binding toxicant concentration processes.

g. The placenta is similarly protected from toxicants. Animals such as the pig, horse, and donkey have six tissues separating fetal and maternal blood, while the rodents and rabbits have only one (Doull et al., 1980). The thicker this barrier, the less likely that toxicants will passively diffuse through it.

C. METABOLISM OF FOREIGN CHEMICAL SUBSTANCES.

Introduction

Over the past thirty years, it has become increasingly obvious that the effect of a toxicant depends in large part upon the animal species subjected to the agent. For instance, rabbits can consume belladonna leaves containing atropine, a potent inhibitor of neural transmission, without ill effect. Nicotine, a tobacco alkaloid, is a potent toxicant to some insects, yet others can consume the tobacco plant without ill effect. These remarkable differences in susceptibility to certain compounds can occur even within an animal species, since toxicants affect various animal strains and sexes differently. Table 4 is a list of some of the variables which can affect toxicity.

Table 4. Variables affecting susceptibility to toxicants

Species	Time of day	Diet	Dose
Strain	Altitude	Bedding	Dose vehicle
Sex	Gravity	Lighting	Route of administration
Age	Fever	Pregnancy	Prior chemical exposure

Caldwell, 1980

Explanation of these effects on toxicant susceptibility resides in metabolism and metabolic differences between animals. The word METABOLISM refers to the

sum of all physical and chemical processes by which living organized substance is produced, maintained, and utilized. BIOCHEMISTRY deals in large part with the enzymatic metabolism of substances ENDOGENOUS to life. In contrast, TOXICOLOGY deals in large part with those substances EXOGENOUS to life. Such substances are called XENOBIOTICS; the term originates from XENO, which refers to "foreign," and from BIOTIC, which refers to "life."

Phase I and Phase II Metabolism

Xenobiotic metabolism generally involves two distinct types of biological reactions. The first metabolic reactions involve what are known as FUNCTIONALIZATION or PHASE I REACTIONS. They enzymatically incorporate one oxygen functionality into a molecule and ultimately achieve two main purposes. Firstly, PHASE I METABOLISM reduces the concentration of the initial toxicant by subdividing that toxicant dosage into other metabolites. Secondly, PHASE I METABOLISM enables the metabolites to participate in a variety of conjugation reactions with various endogenous agents, such as sulfate, glutathione, and glucuronic acid. These metabolic CONJUGATION processes are collectively referred to as PHASE II REACTIONS. The possibility that a PHASE I METABOLITE can undergo multiple conjugation reactions further reduces the concentration of the initial agent. Overall, the dosage of the original toxicant can be substantially and often quickly reduced by these xenobiotic metabolic pathways.

PHASE I reactions generally afford compounds with an increased water solubility. Quantitatively, the xenobiotic agent will usually display a decreased octanol:water partition coefficient, P(o:w), when compared to the parent compound. While metabolism tends to detoxify by expediting the PHASE II process of cleansing of these substances from the body, PHASE I reactions do not necessarily reduce toxicity. In fact, in recent years it has been discovered that many PHASE I transformations produce highly reactive metabolic products having far greater toxicity than the original xenobiotic. N-hydroxyamides, epoxides (carbon-carbon-oxygen 3-membered rings), phosphooxythiranes (phosphorous-sulfur-oxygen-3-membered rings), free radicals (atoms bearing an unpaired elecron), carbenes and carbonium ions (two electron deficient reactive carbon atoms), and other reactive metabolites can be formed during PHASE I FUNCTIONALIZATION REACTIONS. Increasingly, toxicology is involved with determining the nature, bioselectivity, and toxicities associated with these reactive metabolites, for it is often these species that cause the harm. Furthermore, by better understanding the molecular aspects of these reactive metabolites, it is possible to find nutritional ways to assist enzymatic protection mechanisms which can reduce and even eliminate the danger.

Phase I Metabolism Enzymes

There are several enzymes responsible for what is termed PHASE I metabolism. Chief amongst these enzymes are the heme-containing cytochromes P-450, which were given this name because of the unique 450 nm spectral absorption maxima apparent when the cytochrome binds carbon monoxide. Although originally studied in bacteria extensively by I. C. Gunsalus in the Biochemistry Department at Illinois, these enzymes have since been found in virtually all the major organs of animals and in some plants as well (Wilkinson, 1980). Their universal nature affords them a general role in xenobiotic metabolism. Chapters (Johnson, 1979; Coon and Persson, 1980) and several monographs have been written about this enzyme system (Sato and Omura, 1978; Gustafsson *et al.*, 1980).

Most animal tissues, particularly the liver and to lesser extents the adrenal gland, kidney, and lungs, can contain high levels and multiple forms of these cytochromes P-450 (Wilkinson, 1980). Although evolution has finely tuned metabolic systems for the formation, utilization, and disposal of endogenous substances, xenobiotic metabolism by the cytochrome P-450 enzymes involves minimal steric specificity and relatively slow substrate turnover. Because of this broad substrate specificity, the cytochromes P-450 are often referred to as mixed function oxidases (MFO). They are located on the cellular endoplasmic reticulum along with another oxidase called cytochrome C reductase, which was given that name originally because it can donate NADPH-derived electrons to cytochrome c, and another enzyme called cytochrome b_5. The name cytochrome c reductase is somewhat misleading for it can also donate NADPH-derived electrons to cytochromes P-450. This has now afforded it the name NADPH-cytochromes P-450 reductase (Masters, 1980). Cytochrome b_5 is also capable of donating one electron to cytochromes P-450, although it must be NADH-derived. Both these enzymes are useful as internal standards during enzyme isolation.

Multiplicity of cytochromes P-450

When microsomal suspensions containing the cytochromes P-450 are subjected to polyacrylamide gel electrophoresis (PAGE) in the presence of the detergent sodium dodecyl sulfate (SDS), the proteins migrate differently and multiple enzyme forms or isozymes of the cytochromes P-450 can be identified. This electrophoretic technique indicates only exterior differences between these molecules. It says nothing about the ability of the isozymes to catalyze the metabolism of different substrates. Some of these isozymes are very adept at transforming steroids, prostaglandins, and other natural biochemicals to substances critical to control functions such as reproduction. In this regard, they often have very high turnover rates and strict specificities for these substates. A given isozyme may alter, for example, one compound and not another. However, foreign compounds metabolized by this enzyme system are often metabolized at considerably different and far slower rates by the multiple isozymes (Figure 1).

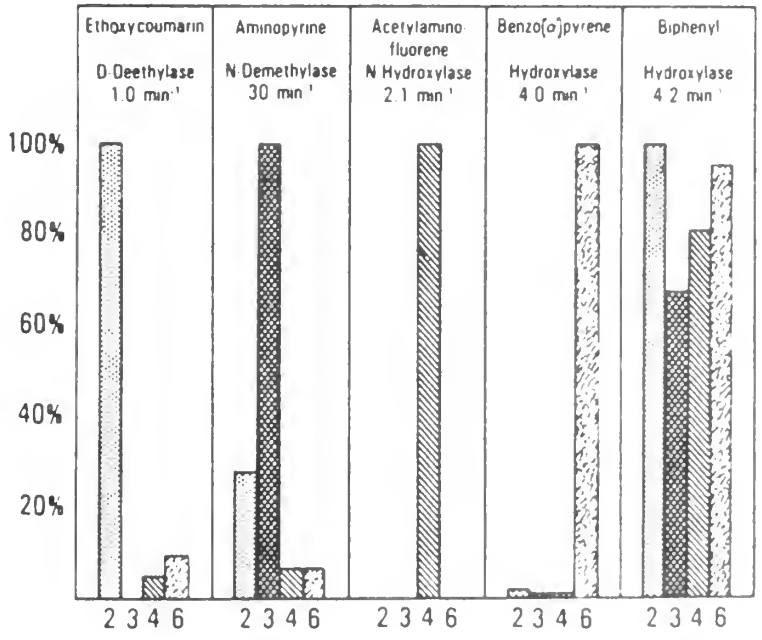

Figure 1. Different catalytic capabilities of four reconstituted liver cytochrome P-450 isozymes from the rabbit. Turnover rates are presented as a percent of the maximum measured with any one substrate. (From Johnson, 1980).

Interactions between chemicals

An early step toward understanding drug interactions arose from studies with hexobarbital, a potent barbiturate which produces a dose-dependent sleep in animals, and the Smith, Kline, and French product, SKF 525A. Although, SKF 525A was found to be without any action of its own, it could dramatically prolong the hexobarbital sleeping times of rodents (Axelrod *et al.*, 1954). This compound acts initially as a substrate, but later begins to display a complex irreversible inhibition. Now it is known to undergo a N-deethylation reaction, affording a secondary amine, which apparently interacts irreversibly with the ferric heme (Goldstein *et al.*, 1974). As such SKF 525A became one of the first *in vivo* tools for the study of cytochromes P-450 metabolism. Today we are aware of many cytochromes P-450 inhibitors. One class of inhibitor having agricultural importance contains the methylenedioxybenzene structure (Hodgson and Philpot, 1974). One such substance called piperonyl butoxide is added to many insecticides to inhibit the insects metabolism by cytochromes P-450 and effectively lengthen the lethal interval of carbamate and organophosphate anticholinesterase inhibitors.

Aside from inhibition another rather unusual aspect of cytochromes P-450 enzymology involves its ability to be induced. Certain chemical substances can actually increase the amount of this enzyme, measured as the carbon monoxide binding pigment. Originally, two such agents were identified as inducers of the enzymes. They are phenobarbital (PB), and numerous related barbiturates, and 3-methylcholanthrene (3MC), one of many carcinogenic polynuclear aromatic compounds (PNA's). Since discovery of these two inducing agents, many other substances have been found to have this action, including compounds as diverse as ethanol, steroids, and even the herbicide contaminant tetrachlorodibenzodioxan (TCDD), which has been linked to the ills of Vietnam veterans. The extremely low quantities of the agent required for enzyme induction by this most toxic of all man-made chemicals has led to the speculation that its effects may stem from induction of the enzyme and enhanced metabolism of many naturally present, but otherwise less-toxic agents (Poland and Glover, 1980; Poland and Knutsen, 1982). Clearly, human effects mediated by this mechanism would be very difficult, if not impossible to prove.

Today it is quite common to see animals pretreated with phenobarbital, 3MC, TCDD, and other agents to induce one or more cytochromes P-450 prior to toxicant exposure or sacrifice for *in vitro* studies. Foods and nutrients can also affect these enzyme levels (Cohen and Van Dyke, 1977). Induced animals look no different than control animals, but their response to certain chemicals, even otherwise innocuous agents, can be dramatically different.

Induction represents a fascinating series of events initiated at the gene level: (1) there is increased protein synthesis markedly raising the amount and activity of many enzymes, (2) including the cytochromes P-450, NADPH cytochrome P-450 reductase, and epoxide hydratases amongst those enzymes producing or metabolizing reactive compounds, and (3) there is increased overall rate of metabolism of substrate. Genetic mechanisms controlling induction of the polysubstrate monoxygenases have been recently described (Nebert *et al.*, 1981). Induction modified metabolism can translate into an increase or a decrease in toxicity, depending on whether a reactive intermediate is involved or not, respectively. The relevance of induction is that animals are afforded a mechanism with which to respond to a wide chemical environment. Since this response is genetically controlled, certain species, strains, and even individuals can, however, respond

differently. While there will be a tendency to select for survival over a population, that need not be true for a given individual's exposure to a specific substance. Often, it is a result of these genetically encoded changes in metabolic pathways that bacteria, insects, and even animals quickly become resistant to antibiotics, to pesticides, and even to drugs and toxicants.

Other phase I metabolic enzymes

1. Although the cytochromes P-450 are a major enzyme system for hydroxylating carbon, nitrogen, and sulfur, they are only one of several enzyme systems involved in toxicant metabolism. The microsomal flavoprotein monooxygenase is another major enzyme system found in the tissues of many animals, especially the pig (Ziegler, 1980; Poulsen, 1981). Its ability to specifically hydroxylate nitrogen and sulfur atoms in xenobiotics has made it an important enzyme in the metabolism of many man-made pesticides and environmental substances. Although certain N-oxides can be quite reactive, in general, this enzyme seems more important to detoxification than toxification, since it does not produce epoxides.

2. Alcohol dehydrogenase is an enzyme which uses zinc as a cofactor to oxidize alcohols to carbonyl compounds. By virtue of the reverse reaction they can also convert carbonyl compounds to alcohols (Bosron and Li, 1980). Aldehyde dehydrogenases and oxidases can further convert aldehyde products to the corresponding acids (Bosron and Li, 1980; Weiner, 1980). Both enzymatic reactions tend to further disperse the source toxicant and effectively reduce its toxicity. In this regard, inhibition of this enzyme system can significantly increase toxicity. Zinc chelators can inhibit the alcohol dehydrogenase. The drug disulfiram, an inhibitor of aldehyde dehydrogenase, can make animals and humans quite ill after consuming alcohol.

3. Many amine oxidases can catalyze the deamination of primary, secondary, and tertiary monoamines and primary diamines (Tipton, 1980). Their inhibition can also have significant toxicological impact (Kaiser and Setler, 1981).

4. Nitro and azo reductases utilize NADH and NADPH for the reduction process, but these enzymes are inhibited by oxygen. In effect these enzymes reverse the oxidations achieved by the cytochromes P-450 and Ziegler's enzymes.

5. Esterases are enzymes which cleave esters; certain cholinesterases are specifically designed to cleave acetylcholine and related neurologically active esters. Both enzymes tend to reduce toxicity and biological activity by preventing access of the lipophilic ester to critical receptor sites. Acetylcholinesterase inhibition is the major mechanism of action for many insectidal carbamates and organophosphates, for such inhibition leads to an overabundance of the naturally-produced ester, an over-stimulated nervous system, and death (Silver, 1974). Certain of these pesticides have, in addition to cholinesterase inhibition, the ability to alkylate a neurological receptor, termed a neuroesterase; this alkylation produces a generally permanent paralysis, especially in chickens and in man (Abou-Donia, 1981; Johnson, 1982).

Phase II metabolism enzymes

Whether from administration or from phase I metabolism, once molecules are available which contain suitable functional groups, then a variety of phase II enzymes can catalyze several different types of conjugative reactions. These reactions generally utilize an endogenous substance produced by normal, but energy requiring biochemical reactions.

Two types of conjugation reactions are possible: Type I conjugation involves the combination of a high energy biochemical substance with a low energy xenobiotic substrate in glycosidation, sulfation, methylation, or acetylation reactions. Type II conjugation combines a high energy substrate with an amino acid to afford a peptide product.

1. Glycoside formation reaction can occur with a number of sugars, although glucuronic acid is the most often encountered conjugate of this class found to add to amines, thiols, and hydroxyl groups present in organic xenobiotics. The most basic group in a molecule seems affected first (Kasper and Henton, 1980; Burchell, 1981).

2. Sulfation reactions, catalyzed by sulfotransferases, utilize adenosine-3'-phospho-5'-phosphosulfate (PAPS) as the sulfate donor. Sulfate or sulfamate conjugates, formed by sulfation of hydroxyl groups or amines, are ionized at physiological pH and are readily excretable. (Jakoby et al., 1980b).

There are some significant differences in the extent of glucuronidation and sulfation reactions between species, as is evident from Table 5. While enzyme deficiencies are the cause, the real significance of the missing enzymes to the affected species remains unclear.

Table 5. Excretion of phenol conjugates by different species

Species	phenylsulfate	phenylglucuronide
Cat	93%	1%
Rat	45%	40%
Pig	2%	95%

From Caldwell (1980).

3. Many methyl transferases catalyze the methylation of amines, thiols, and aromatic hydroxy groups. Although the methyl donor S-adenosylmethionine can be formed from methionine, the S-adenosylhomocysteine product is remethylated by other biochemical reactions enabling a cyclic pathway (Borchardt, 1980: Jakoby and Habig, 1980a; Weisiger and Jakoby, 1980). The most basic functionality is generally favored when multiple possiblities for conjugation exist. A separate enzyme, catechol-O-methyltransferase (COMT), catalyzes the methylation of neurologically important catechol derivatives, such as epinephrine and norepinephrine (Triggle, 1981).

4. Acylation of amines, thiols, and hydroxyl groups is simply another transformation spreading a given dose of a chemical throughout the animal. Deacylation is the reverse of the acylation reaction, although separate enzymes are required. Various interesting species and strain differences

appear in acylation and deacylation reactions and the specific acyl and amino acid conjugates (Caldwell, 1980; Killenberg and Webster, 1980; Weber, 1980).

Phase I, Phase II, and enzyme reactions affording protection from various reactive metablites.

1. Epoxide hydratases. An epoxide is a reactive three membered ring, often produced in vivo by cytochromes P-450. Their irreversible alkylation of biological amines, phosphate, and sulfhydryl groups in proteins, enzymes, and DNA, can lead to death and (or) tumor production. Epoxide hydratases catalyze the addition of water to the epoxide (Guengerich, 1982; Liu and Miwa, 1982). This enzyme can often dramatically reduce the toxicity of the epoxide. Enzyme inhibitors exist, but seem to have little relevance in vivo.

2. Glutathione transferases. Glutathione is a low molecular weight protein containing a free sulfhydryl group as the essential part of its chemistry (Kosower and Kosower, 1976). By virtue of that functionality, glutathione combines, either chemically or often faster via glutathione transferase catalysis, with alkyl halides, epoxides, and other electrophilic alkylating agents. This glutathione reaction tends to protect critical protein, enzyme, and DNA groups from alkylation and tissue alteration (Reed and Beatty, 1980). As a result, toxicity is often decreased (Motoyama, 1980). In this regard, toxicants which deplete such tissue glutathione as methyl chloride and diethyl maleate, which deplete tissue glutathione, can dramatically increase susceptibility to other agents. Glutathione adducts to these reactive functionalities are further metabolized and appear in the urine or the bile as mercapturic acids (Tate, 1980).

3. Glutathione peroxidases. Oxidants may convert glutathione to the corresponding disulfide, which is then unavailable for cellular protection. This enzyme system, which requires selenium as a cofactor, regenerates glutathione by reduction and can afford substantial protection from oxidants (Wendel, 1980).

4. Superoxide dismutase. This enzyme chemically disproportionates (i.e. dismutates) the free radical-anion superoxide (O_2^-) forming hydrogen peroxide and oxygen (Hassan and Fridovich, 1980). Superoxide anion radical is often produced by uncontrolled electron transfer to oxygen. The toxicity of many chemicals has been found to involve this free-radical intermediate. Without the enzyme catalyzed dismutation, superoxide anion could decompose by hydrolytic reactions to the high energy, reactive singlet form of oxygen. Virtually all oxygen utilizing organisms have one or more forms of superoxide dismutase, but anaerobic bacteria lack this enzyme system. Superoxide anion radical is one of several very important radicals also produced from oxygen by radiation exposure. Monographs have appeared on this enzyme system and the role of superoxide anion and related oxygen free radicals in biology (Cohen and Greenwald, 1983; Greenwald and Cohen, 1982).

5. Catalase. This long known enzyme system decomposes hydrogen peroxide. It has gained considerable importance lately because of the danger of the hydrogen peroxide hydroxy radical homolytic cleavage product. Indeed it is likely that the hydroxy radical is a major cause of many toxic events (Cohen and Greenwald, 1983; Greenwald and Cohen, 1983). Perhaps even diabetes may be produced by this radical (Garst et al., 1983).

D. EXCRETION OF CHEMICAL SUBSTANCES

1. Kidney excretion involves urine formation. Poorly volatile, xenobiotics and (or) their conjugates with molecular weights less than about 325 are typically excreted in the urine.

a. Glomerular filtration through 40 angstrom pores removes all substances with molecular weights less than about 60,000, unless they are bound to larger proteins.

b. Passive tubular diffusion enables reabsorption of those filtered substances as functions of their oil:water partition coefficients and their degree of ionization. Stronger bases, which are more protonated at urinary pH are less well reabsorbed (i.e. better excreted) than are weaker bases. Stronger, more ionized acids are less well reabsorbed (i.e. better excreted) than are weaker acids. Adjustment of urinary pH is a useful way of enhancing the excretion of certain acids or bases.

c. Active secretion requires energy to pump passively reabsorbed agents back into the tubules for excretion. Bases will generally be protonated and excreted at the pH of urine. Acids, however, can exist in the neutral form at that pH, so depending on their lipophilicity, they may be passively reabsorbed. An active transport system has evolved to reexcrete these acids. Probenecid is a sulfanilic acid which can occupy and block that acid pump; by minimizing the active renal tubular secretion, probenecid can prevent the reexcretion of acids and can reduce the toxicity of the acidic mycotoxin citrinin in rodents (Berndt and Hayes, 1982). Should this research be applicable to citrinin and ochratoxin, it may be possible to delay or even prevent the porcine nephropathy associated with these mycotoxins.

2. Liver excretion involves toxicant disposal in the bile. Biliary concentration relative to serum is found with bile salts, lead, and xenobiotics or their conjugates having molecular weights about about 325.

Factors which affect metabolism and liver function can change the conjugation of xenobiotics and change the excretory route of that substance. No wonder then that different species may excrete the same compound differently. Liver damage or removal of the bile duct as a route of toxicant excretion can have dramatic effects. For instance, the acute dose killing 50% of the rats administered diethylstilbesterol (i.e. LD 50) decreased from 100 mg/kg of body weight to 0.75 mg/kg of body weight upon bile duct ligation (Doull et al., 1980).

3. Lungs tend to excrete substances having a substantial volatility at body temperature, although many poorly volatile substances may be metabolized by lung tissue. Excretion via the lungs involves a simple diffusion mechanism.

4. Gastrointestinal tract excretion occurs for those agents which are: (1) incompletely absorbed in the stomach or intestine, (2) excreted via the bile or other body secretions into the intestine, or (3) which enter the intestine by diffusion from the tissues.

5. Excretion of toxicants can occur in milk. Indeed, tremetol and tremetone from the white snakeroot plant, can make cow's milk quite dangerous (Evers and Link, 1974). There are numerous stories indicating that milk poisoning by this Midwestern plant killed Lincoln's mother and was the factor leading the Lincoln family to move to Illinois from Kentucky.

TOXICODYNAMICS

Other Pork Industry Conference Reports describe the influence of mycotoxins on the growth and reproduction of swine (Hollis and Jones, 1983: Diekman, 1983). The oral presentation will briefly describe the TOXICODYNAMICS (i.e. toxicity mechanisms) for several agents of interest to swine producers. Where possible, insights as to how these effects might be alleviated by nutritional mechanisms will be offered. The reader is referred to literature pertinent to this presentation (Cohen and Van Dyke, 1977; Swenson, 1982; Essigmann, 1982; Ames, 1983).

LITERATURE CITED

Abou-Donia, M. B. 1981. Organophosphorous ester-induced delayed neurotoxicity. Ann. Rev. Pharmacol. Toxicol. 21:511.

Ames, B. N. 1983. Dietary carcinogens and anticarcinogens. Science. 221:1256.

Anonymous. 1979a. Interactions of Mycotoxins in Animal Production. National Academy of Science. Washington, D.C.

Anonymous. 1979b. Aflatoxin and Other Mycotoxins: An Agricultural Perspective. Council for Agricultural Science and Technology. Publication 80.

Axelrod, J., J. Reichenthal, and B. B. Brodie. 1954. Mechanism of the potentiating action of beta-diethylaminoethyl diphenylpropylacetate. J. Pharmacol. Exp. Therap. 112:49.

Berndt, W. O. and A. W. Hayes. 1982. Effect of probenecid on citrinin-induced nephropathy. Toxicol. Appl. Pharmacol. 64:118.

Borchardt, R. T. 1980. N- and O-Methylation. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 2. Academic Press. New York.

Bosron, W. F. and T-K, Li. 1980. Alcohol dehydrogenases. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 1. Academic Press. New York.

Burchell, B. 1981. Identification and purification of multiple forms of UDP-glucuronosyl transferase. Rev. Biochem. Toxicol. 3:1.

Caldwell, J. 1980. Comparative Aspects of Detoxication. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 1. Academic Press. New York.

Cohen, G. and Greenwald, R. A. 1983. Oxy Radicals and Their Scavenger Systems. Vol. I. Molecular Aspects. Elsevier/North-Holland. New York.

Cohen, E. N. and R. A. Van Dyke. 1977. Metabolism of Volatile Anesthetics: Implications for Toxicity. Addison-Wesley. Reading, MA.

Coon, M. J. and A. V. Persson. 1980. Microsomal cytochrome P-450: A central catalyst in detoxification reactions. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 1. Academic Press. New York.

Diekman, M. 1983. Effects of toxins on swine reproduction. Pork Industry Conference, University of Illinois, Urbana, IL.

Doull, J., C. D. Klaassen, and M. O. Amdur. 1980. Toxicology, The Basic Science of Poisons. (2nd Ed.). MacMillian Book Co. New York.

Essigmann, J. M. 1982. Metabolic activation of aflatoxin B1: patterns of DNA adduct formation, removal, and excretion in relationship to carcinogenesis. Drug Metabol. Rev. 13:581.

Evers, R. A. and R. P. Link. 1972. Poisonous Plants of the Midwest. Univ. Ill. Agric. Exp. Stat. Publicn. 24, Urbana, IL.

Garst, J., P. Stapleton, and J. Johnston. 1983. Mutagenicity of alpha-hydroxy ketones may involve superoxide anion radical. In: R. A. Greenwald and G. Cohen (Ed.). Oxy Radicals and Their Scavenger Systems. Vol. II: Cellular and Medical Aspects. Elsevier/North Holland.

Goldstein, A. L. Aronow, and S. M. Kalman. 1974. Principles of Drug Action: The Basis of Pharmacology, 2nd ed. Wiley, New York.

Greenwald, R. A. and G. Cohen. 1983. Oxy Radicals and Their Scavenger Systems. Vol II. Cellular and Medical Aspects. Elsevier/North-Holland. New York.

Guengerich, F. P. 1982. Epoxide hydrolase: properties and metabolic roles. Rev. Biochem. Toxicol. 4:5.

Gustafsson, J-A., J. Carstedt-Duke, A. Mode, and J. Rafter. 1980. Biochemistry, Biophysics, and Regulation of Cytochrome P-450. Elsevier/North-Holland. New York.

Hassan, H. M. and I. Fridovich. 1980. Superoxide dismutases: detoxication of a free radical. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 1. Academic Press. New York.

Hayes, Jr., W. J. 1975. Toxicology of Pesticides. Williams and Wilkins Co. Baltimore, MD.

Helmer, F., K. Kiehs, and C. Hansch. 1968. The linear free-energy relationship between partition coefficients and the binding and conformational perturbation of macromolecules. Biochemistry. 7:2858.

Hodgson, E. and R. M. Philpot. 1974. Interaction of methylenedioxyphenyl (1,3-benzodioxole) compounds with enzymes and their effects on mammals. Drug Metab. Rev. 3:231.

Hodgson, E. and F. E. Guthrey. 1980. Introduction to Biochemical Toxicology. Elsevier/North Holland. New York.

Hollis, G. R. and R. W. Jones. 1983 Pork Industry Conference, University of Illinois. Urbana, IL.

Jakoby, W. B. and W. H. Habig. 1980a. Glutathione transferases. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 2. Academic Press. New York.

Jakoby, W. B., R. D. Sekura, E. S. Lyon, C. J. Marcus, and J-L. Wang. 1980b. Sulfotransferases. In: W.B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 2. Academic Press. New York.

Johnson, E. F. 1979. Multiple forms of cytochrome P-450: criteria and significance. Rev. Biochem. Toxicol. 1:1.

Johnson, E. F. 1980. Contrasting properties of cytochrome P-450 isozymes; characterization, identification, and significance of multiple forms of rabbit liver microsomal cytochrome P-450. In: Gustafsson, J-A., J. Carstedt-Duke, A. Mode, and J. Rafter (Eds.). Biochemistry, Biophysics, and Regulation of Cytochrome P-450. Elsevier/North-Holland. New York.

Johnson, M. K. 1982. The target of initiation of delayed neurotoxicity by organophosphorous esters: biochemical studies and toxicological applications. Rev. Biochem. Toxicol. 4:141.

Johnson, M. R. and G. M. Milne. 1981. Analgetics. In: M. W. Wolff (Ed.). Burger's Medicinal Chemistry 4th Ed. Part 3. Wiley-Interscience. New York.

Kaiser, C. and P. E. Setler. 1981. Antidepressant agents. In: M. W. Wolff (Ed.). Burger's Medicinal Chemistry 4th Ed. Part 3. Wiley-Interscience. New York.

Kasper, C. B. and D. Henton. 1980. Glucuronidation. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 2. Academic Press. New York.

Killenberg, P. G. and L. T. Webster, Jr. 1980. Conjugation of peptide bond formation. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 2. Academic Press. New York.

Kosower, N. S. and E. M. Kosower. 1976. The glutathione-glutathione disulfide system. Free Radicals Biol. 2:55.

Lien, E. J. 1981. Structure-activity relationships and drug dispostion. Ann. Rev. Pharmacol. Toxicol. 21:31.

Liu, A. Y. H. and G. T. Miwa. 1982. Molecular properties and biological functions of microsomal epoxide hydratase. Ann. Rev. Pharmacol. Toxicol. 22:523.

Masters, B. S. S. 1980. The role of NADPH-cytochrome C (P-450) reductase in detoxication. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 1. Academic Press. New York.

Morris, G. L., S. E. Curtis, and J. Simon. 1982. Studies of carbon monoxide toxicosis in swine. Swine Res. Rep. University of Illinois, Urbana, IL.

Motoyama, N. and W. C. Dauterman. 1980. Glutathione S-transferases: their role in the metabolism of organophosphorous insecticides. Rev. Biochem. Toxicol. 2:49.

Nebert, D. W, H. J. Eisen, M. Negishi, M. A. Lang, L. M. Hjelmeland, and A. B. Okey. 1981. Genetic mechanisms controlling the induction of polysubstrate monooxygenase (P-450) activities. Ann. Rev. Pharmacol. Toxicol. 21:431.

Poland, A. and E. Glover. 1980. 2,3,7,8-Tetrachlorodibenzodixan: studies on the mechanism of action. In: H. R. Witschi (Ed.). The Scientific Basis of Toxicity Assessment. Elsevier/North-Holland. New York.

Poland, A. and J. C. Knutsen. 1982. 2,3,7,8-Tetrachlorodibenzodioxan and related halogenated aromatic hydrocarbons: examination of the mechanism of toxicity. Ann. Rev. Pharmacol. Toxicol. 22:517.

Poulsen, L. L. 1981. Organic sulfur substrates for the microsomal flavin-containing monooxygenases. Rev. Biochem. Toxicol. 3:33.

Reed, D. J. and P. W. Beatty. 1980. Biosynthesis and regulation of glutathone: toxicological implications. Rev. Biochem. Toxicol. 2:213.

Sato, R. and T. Omura. 1978. Cytochrome P-450. Academic Press. New York.

Scheuplein, R. J. 1977. Permeability of the skin. In: S. R. Geiger (Ed.). Handbook of Physiology. Amer. Physiol. Soc. Bethesda, MD. pp. 299-322.

Shen, T. Y. 1981. Nonsteroidal anti-inflammatory agents. In: M. E. Wolff (Ed.). Burger's Medicinal Chemistry 4th Ed. Part 3. Wiley-Interscience. New York.

Silver, A. 1974. The Biology of Cholinesterases. Elsevier/North-Holland. New York.

Staigmiller, R. B., R. A. Bellows, and R. E. Short. 1983. Growth and reproduction traits in beef heifers implanted with zeranol. J. Anim. Sci. 5:527.

Stenlake, J. B. 1981. Neuromuscular blocking agents. In: M. E. Wolff (Ed.). Burger's Medicinal Chemistry 4th Ed. Part 3. Wiley-Interscience. New York.

Swenson, D. H. 1981. Metabolic activation and detoxification of aflatoxins. Rev. Biochem. Toxicol. 3:155.

Takman, B. H. and H. J. Adams. 1981. Local anesthetics. In: M. E. Wolff (Ed.). Burger's Medicinal Chemistrey 4th Ed. Part 3. Wiley-Interscience. New York.

Tate, S. S. 1980. Enzymes of mercapturic acid formation. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol 2. Academic Press. New York.

Timbrell, J. A. 1982. Principles of Biochemical Toxicology. Burgess Publishing Co. Ft. Lee, N. J.

Tipton, K. F. 1980. Monoamine oxidase. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 1. Academic Press. New York.

Triggle, D. J. 1981. Adrenergics: catecholamines and related agents. In: M. E. Wolff (Ed.). Burger's Medicinal Chemistry 4th Ed. Part 3. Wiley-Interscience. New York.

Uraguchi, K. and M Yamazaki. 1978. Toxicology-Biochemistry and Pathology of Mycotoxins. Halstead Press. New York.

Weber, W. H. and I. B. Glowinski. 1980. Acetylation. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 2. Academic Press. New York.

Weisiger, R. A. and W. B. Jakoby. 1980. S-Methyltransferases. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 2. Academic Press. New York.

Weiner, H. 1980. Aldehyde oxidizing enzymes. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 1. Academic Press. New York.

Wendel, A. 1980. Glutathione peroxidase. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 1. Academic Press. New York.

Wilkinson, C. F. 1980. The metabolism of xenobiotics: a study in biochemical evolution. In: H. R. Witschi (Ed.). The Scientific Basis of Toxicity Assessment. Elsevier/North-Holland. New York.

Ziegler, D. M. 1980. Microsomal flavin-containing monooxygenase: oxygenation of nucleophilic nitrogen and sulfur compounds. In: W. B. Jakoby (Ed.). Enzymatic Basis of Detoxication. Vol. 1. Academic Press, New York.

Mycotoxins and Reproduction in Swine

MARK A. DIEKMAN AND GERALD G. LONG

Introduction

Of the 200,000 known species of molds, only 50 or 60 are known to be harmful to humans or livestock. Certain fungi that grow on grains and grasses can produce chemical substances called mycotoxins that adversely affect reproduction in swine. Most mycotoxins that are of economic concern in the United States are produced by the genera Pennicillium, Aspergillus, and Fusarium. The common antibiotic Penicillin is obtain from the Pennicillium mold and alfatoxin, a mycotoxin produced by Aspergillus mold and found primarily in the southeastern United States, will not be covered in this report. Emphasis will be placed upon the most common toxin-producing fungus of corn in the midwest Fusarium roseum (Gibberella zeae). F. roseum produces two mycotoxins, zearalenone and deoxynivalenol, that are associated with decreased performance in swine. Zearalenone is not a toxin in the sense of causing deaths, but the substance mimics the action of the reproductive hormone estradiol-17β. Deoxynivalenol is a true toxin and pigs are reluctant to eat it. Hence, the most common sign of deoxynivalenol in swine feed is feed refusal or decreased consumption. Usually F. roseum infected corn will contain both mycotoxins. Therefore, it becomes difficult to establish the specific actions of each mycotoxin on reproduction in swine. Utilization of purified zearalenone or deoxynivalenol in the experiments described in this paper have helped to clarify their involvement in the disruption of pregnancy in swine. In this paper we have elected to include procedural details of the experiments in order that the reader will know how the experiments were conducted.

EFFECT OF FUSARIUM ROSEUM CORN CULTURE CONTAINING ZEARALENONE AND DEOXYNIVALENOL ON EARLY PREGNANCY IN SWINE

An ideal environment for Fusarium roseum to synthesize zearalenone occurs when the corn plant is subjected to cool wet weather during flowering and a short drying season in the fall. Improper storage conditions will also allow the fungus infected corn to continue production of the mycotoxin. Moisture content of the grain must be lowered to 13-14% to insure that mold growth has been halted.

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Ingestion of moldy corn containing zearalenone has its most profound effects in prepubertal gilts. In these animals, the estrogenic effects are quite marked (Kurtz et al., 1969; Christensen, 1979). Prepubertal gilts exposed to zearalenone will show signs of hyperestrogenism, including swollen vulvas and mammary development. In severe cases, there may be prolapse of the vagina and rectum. Prepubertal gilts are quite sensitive to zearalenone on a dosage basis and may show hyperestrogenism at relatively low concentrations of the compound in the feed (i.e., 1 ppm or less in the feed). Concentrations required to cause reproductive problems in mature sows are much higher. Hence, prepubertal gilts are sensitive indicators for contamination of feed with zearalenone.

Moldy feed in the diet of pregnant sows has been associated with abortion, weak pigs, stillbirths, decreased litter size, and fetal mummification (Sharma, 1974; Mirocha et al., 1977; Shreeve, 1978; Christensen, 1979). Concentrations of zearalenone in the feed were not determined in most of these cases. Zearalenone added to the diet of pregnant sows at 25 and 50 ppm caused sows to farrow smaller litters and smaller pigs (Christensen, 1979). Lower concentrations of zearalenone in the feed appear to have little effect on the pregnant sow. A diet with 2 ppm zearalenone caused no overt differences in farrowing performance (Etienne and Jemmal, 1979) while 3.6 ppm zearalenone caused a decrease in fetal weight at 80 days of gestation but had no effect on embryonic mortality (Vanyi et al., 1976). To determine whether moldy corn containing known amounts of zearalenone affects early pregnancy, the following experiment was conducted at the Baker-Purdue Swine Center, (Long et al., 1982).

Fusarium roseum (Gibberella zeae) #1693 was grown on sterile popcorn. Popcorn (410 gm) and 220 ml water were autoclaved for 1 hour at 121°C. Cultures were grown at 23°C for 9 days, followed by 16°C for 4 weeks, followed by 12°C for 3 weeks. Corn cultures were then dried at 38°C with forced aeration and ground in a Wiley mill. Ground cultures were mixed and divided into sublots, and each sublot was analyzed for zearalenone.

Concentrations of zearalenone were determined by using a chloroform/water extract of the feed, which was partially purified on a silica gel column then further purified by liquid-liquid partition using hexane and acetonitrile (Official Methods of Analyses, 1980). The quantitation was accomplished by thin-layer chromatography plates scanned densitometrically at an excitation wavelength of 327 mμ and emission at 455 mμ. Deoxynivalenol concentration of the feed was determined by using a methanol extract purified on a silica gel column with chloroform and methanol. Column elute was analysed by gas liquid chromatography. Deoxynivalenol was also quantitated by gas chromatography:mass spectrography (Vesonder et al., 1978; Vesonder and Hesseltine, 1980/81).

The ground F. roseum corn culture was added to a standard 14% protein corn-soybean swine gestation ration. Concentrations of zearalenone were 7, 38 and 64 ppm for the low, medium and high dosage groups, respectively. Deoxynivalenol concentrations were calculated to be approximately 0.4, 2.4, and 4.2 ppm for the low, middle and high dosage groups, respectively. Feed from the same lot without added F. roseum culture served as control feed.

Mature, 100-110 kg crossbred gilts were bred by natural service. Each gilt was served by 2 different boars of proven fertility within a 24 hour period. On day 3 postbreeding, gilts were randomly assigned to one of three treatment groups or to the control group. Gilts were given 1.8 kg feed per day, the

infected feed being given from days 3 through 34 postbreeding. Blood samples were taken at weekly intervals from days 11-42 postbreeding for determination of serum concentrations of progesterone by radioimmunoassay (Niswender, 1973). All gilts were sacrificed between days 38 and 43 postbreeding. Reproductive tracts were examined for number and size of corpora lutea, number and size of fetuses, and appearance of endometrium.

None of the gilts demonstrated complete feed refusal, but gilts in the high dosage group did not consume the entire ration during the first few days of experimental feeding. Two gilts in the high dose group showed signs of hyperestrogenism from days 19-35 postbreeding.

Number of corpora lutea and fetuses in each gilt at slaughter are shown in table 1. No significant differences in the number of corpora lutea among treatments were found. The low dose group did not have a significantly different number of fetuses from the control group. Two of the low dose gilts had abnormally small litters (1 and 3 pigs). In the middle dose group, there were two nonpregnant gilts and one gilt with one fetus. None of the gilts were pregnant in the high dose group. There was no effect of fetal size in treated gilts that were pregnant.

TABLE 1. EFFECT OF ZEARALENONE ON NUMBER OF CORPORA LUTEA AND FETUSES

Treatment	No. of Gilts	No. of Corpora Lutea[a]	No. of fetuses[a]
Control	8	11.0 ± .8	6.6 ± 1.1
7 ppm	4	9.7 ± .5	5.5 ± 2.1
38 ppm	4	10.8 ± .8	2.2 ± 1.9
64 ppm	4	10.5 ± 1.0	0 ± 0

[a]Mean ± Standard Error.

All zearalenone-fed gilts had constant serum concentrations of progesterone during the experiment indicating that the corpora lutea had persisted in these animals (figure 1). Even though the number of fetuses were reduced considerably in some gilts, these gilts did not return to estrus. The small litters probably would have been carried for a normal gestation. Likewise, the gilts fed the high dose of zearalenone would not have recycled until the corpora lutea had regressed several months later.

The microscopic appearance of the endometrium in the low and middle dose group gilts was the same as that in the pregnant control gilts. The endometrium of the nonpregnant treatment gilts had an appearance similar to the endometrium of control gilts in areas where there was no placental attachment. The endometrial morphology in the high dose group gilts had less stacking of cells, but was more like that of the pregnant controls than the nonpregnant control. The mucosa of the fallopian tubes in the low and middle dose groups resembled that of the pregnant controls. The mucosa of the uterine tube in the high dose group was intermediate in appearance between the pregnant and nonpregnant controls.

In summary, the primary effect of bred gilts consuming feed containing zearalenone and deoxynivalenol was to prevent early fetal development while

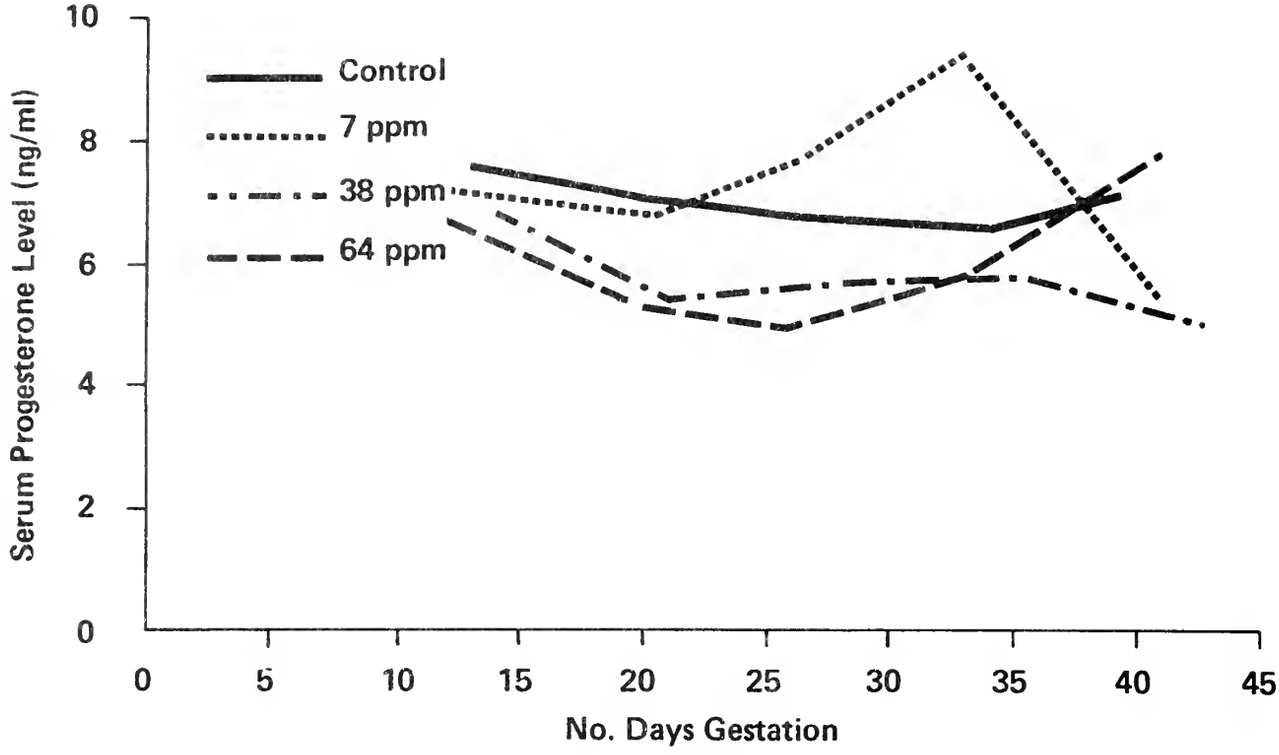

FIGURE 1

causing persistence of corpora lutea and continued secretion of progesterone. The estrogenic effects of zearalenone became obvious only in some of the gilts that received the high doses. Previous studies on the effects of mycotoxins on reproduction in swine involved continuous feeding throughout pregnancy or during late gestation. In the present study, it appears that the ingestion of F. roseum infected corn by bred gilts during a limited time near implantation resulted in a loss of pregnancy. The problem would be compounded in a field situation because the persistent corpora lutea would delay the gilt from returning to estrus. Gilts in the pseudopregnant condition may not be detected until they approached their anticipated farrowing date.

CONSUMPTION OF FUSARIAN ROSEUM CONTAMINATED CORN DURING DAYS 7-17 POSTESTRUS BY CYCLING AND PREGNANT GILTS

A second experiment was conducted to determine if a diet containing 10% F. roseum molded corn caused pseudopregnancy in gilts (Long et al., 1983). The final mixed diet contained 100 ppm zearalenone and 8 ppm deoxynivalenol. As mature crossbred gilts exhibited estrus, they were assigned to one of three groups. In treatment group 1, 10 gilts were fed the F. roseum infected corn from days 7-16 post estrus. In treatment group 2, five gilts were observed to be in estrus, but were not bred. These gilts were fed the F. roseum ration days 7-17 post estrus. In treatment group 3, four gilts were bred and fed the control ration. Blood samples were drawn from the jugular vein at weekly intervals during week 1-5 postestrus from all gilts and at biweekly intervals during weeks 7-20. Gilts were examined visually for signs of estrus during the first 5 weeks postestrus. Each gilt was tested twice for pregnancy by 3 people on different days between 40 and 50 days postbreeding with an ultrasonic pregnancy testing device.

Five of the gilts in treatment group 1 had swelling of the vulva and/or demonstrated lordosis for variable periods during the time they were consuming the F. roseum diet. Two gilts in treatment group 2 exhibited estrus between 19 and 22 days postestrus and one gilt in treatment group 3 exhibited estrus 17 days postestrus. Pregnancy testing of the group 1 gilts resulted in 6 being called pregnant, one nonpregnant, and 3 questionable. Of the gilts in treatment group 2, 2 appeared to be nonpregnant and the other 3 were questionable. Three gilts in treatment group 3 tested as pregnant, and one as nonpregnant.

Three trends were evident in the serum concentrations of progesterone in the gilts in treatment group 1. In 3 gilts, corpora lutea were maintained throughout the 20 week period, as indicated by high concentrations of serum progesterone (> 10 ng/ml). Serum concentrations of progesterone fell to < 1 ng/ml in 4 gilts from weeks 4-14 postestrus. In these gilts, the serum progesterone concentration remained low for the remainder of the test period, indicating an absence of corpora lutea. The pattern of concentrations of serum progesterone in one gilt indicated that she failed to develop corpora lutea on the first cycle, and returned to estrus during week 3-4. When the corpora lutea from this cycle regressed, she did not recycle. The remaining gilts in treatment group 1 initially had high concentrations of serum progesterone (> 20 ng/ml) which fell and rose at various times from 10-20 weeks postestrus.

Serum progesterone concentrations of the gilts in treatment group 2 showed the same inconsistencies as the gilts in treatment group 1. Maintenance of serum concentrations of progesterone was observed in the gilts in treatment group 3 except for a sharp drop and rebound in progesterone concentrations of one gilt at 3 weeks postestrus.

To summarize the results, none of the bred gilts fed F. roseum diet farrowed. However, several of these gilts were judged to be pregnant on days 40 to 50 by ultrasound. In addition, serum concentrations of progesterone were highly variable regardless of when the fetuses died during the gestation period. Certainly, the moldy corn containing zearalenone and deoxynivalenol disrupted pregnancy and produced symptoms of pseudopregnancy.

EFFECT OF PURIFIED ZEARALENONE ON EARLY PREGNANCY IN GILTS

The experiments described above indicated that ingestion of F. roseum molded corn containing zearalenone and deoxynivalenol was detrimental to fetuses during early pregnancy. However, it was not possible from these studies to discern the specific actions of each mycotoxin on the reproductive tract. To determine the effect of purified zearalenone on early pregnancy, the following experiment was conducted (Long and Diekman, 1983).

Purified zearalenone (> 95% pure) was added to a standard corn-soybean swine gestation ration to give concentrations in the feed of 5, 15, 30, 60 and 90 ppm (mg/kg). Feed from the same batch without added zearalenone served as control. The feed was tested by gas-chromatography/mass-spectrometry procedures and found to be free of other mycotoxins.

Crossbred gilts (100-120 kg) were handmated to 2 different boars during a 12-16 hour period. The day of breeding was designated as day 0. After breeding, gilts were individually penned and assigned to a treatment group. Five gilts each were assigned to groups that received 0, 15, and 30 ppm zearalenone in the diet, and 3 gilts each to the 5, 60 and 90 ppm zearalenone treatment groups. Gilts were fed the zearalenone treated feed on days 2 through 15 after breeding and control feed for the remainder of the experimental period. Each gilt was fed 1.8 kg feed/day. Gilts used in this experiment were either vaccinated with a killed porcine parvovirus vaccine 30 days before breeding or shown to be free of porcine parvovirus during the experimental period by serologic testing.

After breeding, blood samples were obtained weekly from each gilt by puncture of the jugular vein. On days 13-15 and 40-43 postmating, gilts were cannulated nonsurgically and blood samples were drawn at 20 minute intervals for 4 hours. All blood samples were immediately placed on ice. Serum was harvested the following morning by centrifugation at 2,500 X g for 30 min and stored at -20°C until assayed for luteinizing hormone (LH), follicle stimulating hormone (FSH) and prolactin (PRL) by radioimmunoassay. Serum concentrations of LH, FSH and PRL were averaged for each gilt during the 4-hour sampling period (13 samples/gilt). Any value greater than one standard deviation above the mean during the sampling period of each gilt was identified as a secretory spike. A nonparametric statistic, the Kruskal-Wallis test, was used to test for differences in the frequency of identified spike among the treatment groups (Hollander and Wolfe, 1973).

All gilts were killed 40-43 days after breeding. Gross observations included degree of fetal development (fetal length and weight) and fetal survival (number live fetuses:number corpora lutea). Pituitary glands were collected and stored at -80°C until assayed for LH and FSH.

The number of corpora lutea and number of live fetuses observed at slaughter are presented in table 2. The number of corpora lutea were similar among the treatment groups. The number of live fetuses were similar among the gilts that consumed 0, 5, 15 or 30 ppm zearalenone. Bred gilts that ingested 60 and 90 ppm zearalenone did not maintain any of their fetuses. Fetal weights were similar in gilts fed 0, 5, 15 or 30 ppm zearalenone.

TABLE 2. NUMBER OF CORPORA LUTEA AND LIVE FETUSES IN BRED GILTS FED PURIFIED ZEARALENONE.

Zearalenone PPM	No. Gilts	Corpora Lutea[a]	Fetuses[a]
0	5	13.0 ± 1.6	10.2 ± 1.5
5	3	12.0 ± 1.0	10.3 ± 1.8
15	5	11.2 ± 1.4	6.5 ± .9
30	5	12.0 ± .7	8.6 ± 1.6
60	3	9.6 ± 1.3	0
90	3	12.0 ± 2.0	0

[a]Mean ± standard error of mean.

Serum concentrations of progesterone measured at weekly intervals after breeding are presented in figure 2. Serum concentrations of progesterone were similar in gilts fed 0, 5, 15 and 30 ppm zearalenone but lower in gilts fed 60 and 90 ppm zearalenone at weeks 2, 3 and 6.

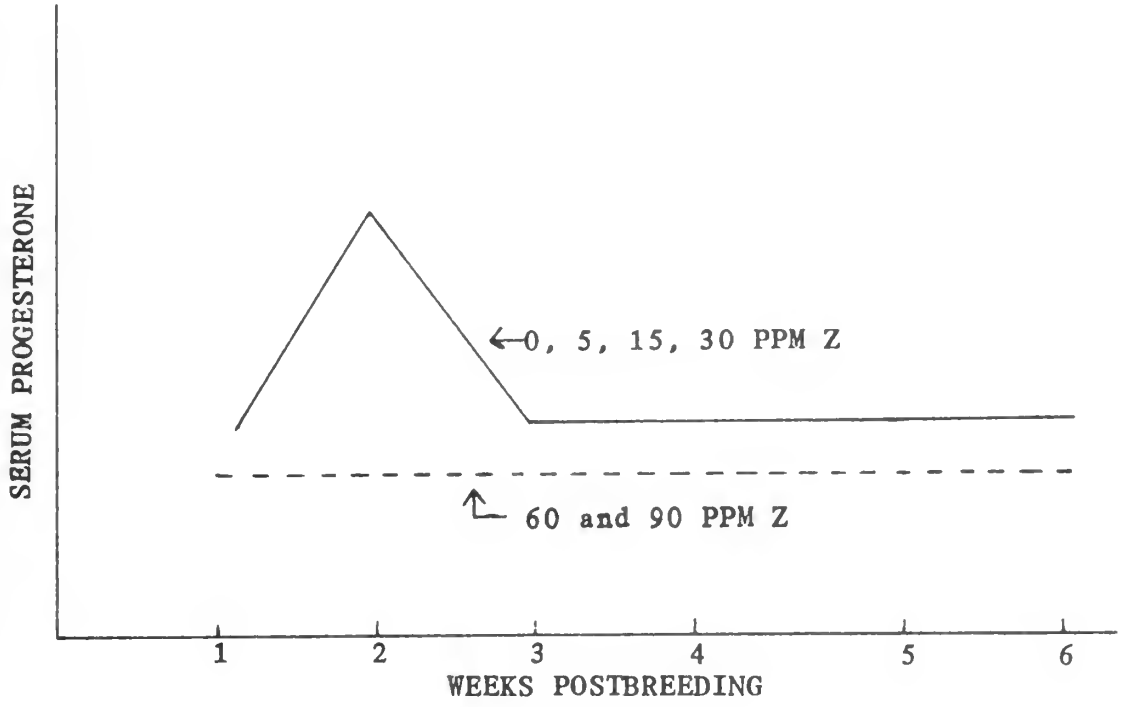

FIGURE 2

The number of identified secretory spikes for serum LH, FSH and PRL during the two sampling periods are shown in table 3. When the secretory patterns of LH, FSH and PRL were examined on days 13-15 postmating while the gilts were ingesting zearalenone daily, no differences in the number of hormone spikes were found. Likewise, no differences in the number of secretory spikes were observed for LH, FSH and PRL during the sampling period 40-43 days postmating. By this date gilts in the zearalenone treatment groups had been consuming the control feed for approximately 4 weeks. Serum concentrations of LH, FSH and PRL in the weekly samples were also similar among treatment groups.

TABLE 3. NUMBER OF IDENTIFIED SERUM SECRETORY SPIKES FOR LH, FSH AND PRL DURING CONSUMPTION OF FEED CONTAINING Z (DAYS 13-15) AND FOLLOWING FOUR WEEK WITHDRAWAL PERIOD (DAYS 40-43)

Z	No.	LH Peaks/4 H/Gilt		FSH Peaks/4 H/Gilt		PRL Peaks/4 H/Gilt	
PPM	Gilts	13-15	40-43	13-15	40-43	13-15	40-43
0	5	.6	.8	.8	1.0	.8	.8
5	3	1.0	2.0	.5	1.0	1.0	.5
15	5	1.5	1.0	1.0	.8	1.2	1.0
30	5	1.0	1.0	1.2	.6	1.2	.6
60	3	1.0	1.0	1.0	.6	1.0	.6
90	3	.6	1.3	.3	.6	.3	1.0

The influence of various concentrations of zearalenone on anterior pituitary gland concentrations of LH and FSH is presented in table 4. When zearalenone was fed to bred gilts from days 2-15 postmating, no differences in anterior pituitary gland weight or concentrations of LH and FSH were found when the gilts were killed on days 40-43 postmating.

TABLE 4. EFFECT OF ZEARALENONE ON PITUITARY WEIGHT AND CONCENTRATIONS OF LH AND FSH.

Zearalenone PPM	No. of Gilts	Pit Weight[a] (mg)	LH[a] (ug/mg)	FSH[a] (ug/mg)
0	5	217 ± 9	2.24 ± .58	1.35 ± .14
5	3	200 ± 13	2.92 ± .58	1.64 ± .08
15	5	174 ± 14	2.47 ± .43	1.76 ± .20
30	6	207 ± 24	2.74 ± .31	1.65 ± .10
60	3	181 ± 14	1.92 ± .66	1.67 ± .24
90	3	181 ± 12	1.41 ± .73	2.37 ± .73

[a]Mean ± Standard Error.

In summary, bred gilts that consumed feed containing 60 or 90 ppm of purified zearalenone failed to maintain their embryos. In addition, the corpora lutea were still present in these gilts several weeks after withdrawal of the zearalenone. Not only is reproductive efficiency reduced if bred gilts consume zearalenone during early pregnancy because embryos die, but it may be several months before these females will return to heat and can be bred again successfully (Cantley et al., 1982; Etienne and Jemmali, 1982; Young and King, 1983).

The lack of embryonic development in the gilts fed 60 or 90 ppm zearalenone may have been related to the alteration in serum concentrations of progesterone. Serum concentrations of progesterone normally rises rapidly after mating in the pig peaking at about day 12 postmating, and then declines about 30 percent (Guthrie et al., 1972). In this experiment, the treatment groups that had significantly lower concentrations of progesterone 2-3 weeks postmating also lacked fetal development. Zearalenone causes morphologic changes in the reproductive tract consistent with estrogenic stimulation (Kurtz et al., 1969; Chang et al., 1979) and most likely also causes changes in the secretory activity of the endometrium and hence the environment in the uterine lumen. In addition, it appears that the detrimental effects of zearalenone are mediated locally on the reproductive tract (ovaries and uterus) since no changes in the secretory pattern of pituitary hormones LH, FSH and PRL were observed.

EFFECT OF PURIFIED DEOXYNIVALENOL ON EARLY PREGNANCY IN SWINE

To determine if purified deoxynivalenol was detrimental to embryos during early pregnancy, an experiment was conducted similar to the one described above utilizing purified zearalenone. Purified deoxynivalenol was added to a standard corn-soybean gestation ration to give concentrations of 2, 4 and 8 ppm. Feed from the same batch without added deoxynivalenol served as control.

Crossbred gilts were handmated to 2 different boars during a 12-16 hour period. After breeding, gilts were individually penned and assigned to a treatment group. Four gilts each were assigned to groups that received either 0, 2, 4 or 8 ppm deoxynivalenol from days 2 through 15 postbreeding. All gilts were killed 40-43 days after breeding and reproductive tracts were examined.

The number of corpora lutea and number of live fetuses observed at slaughter are presented in table 5. No effect of deoxynivalenol was found on either the number of corpora lutea or fetuses. Based on data presented earlier, it appears that the detrimental effect of moldy corn containing mycotoxins on pregnancy in pigs is caused by zearalenone. However, it is possible that concentrations of deoxynivalenol used in this experiment were too low to exert a deleterious effect on pregnancy. Currently, an experiment is being conducted to determine if greater concentrations of deoxynivalenol are toxic to embryos during early development.

TABLE 5. NUMBER OF CORPORA LUTEA AND LIVE FETUSES IN BRED GILTS FED PURIFIED DEOXYNIVALENOL.

Deoxynivalenol, PPM	Corpora Lutea[a]	Fetuses[a]
0	13.2 ± 1.1	10.0 ± 0
2	14.3 ± .8	11.7 ± .8
4	13.0 ± 1.7	9.2 ± 1.4
8	13.7 ± 1.4	11.0 ± 2.1

[a]Mean ± Standard Error.

INFLUENCE OF PREPUBERTAL CONSUMPTION OF ZEARALENONE ON SEXUAL DEVELOPMENT OF BOARS

In 1971, Bristol and Djurickovic observed decreased libido in mature boars fed moldy corn, but Ruhr et al. (1979) found no effect of consumption of 60 ppm zearalenone for 8 weeks on serum concentrations of testosterone, libido or semen traits. In immature boars fed moldy corn containing 500 to 600 ppm zearalenone, growth and testis weight as a percentage of body weight were reduced compared to the corresponding measures for control animals (Christensen et al., 1972). To examine the effects of prepuberal consumption of purified zearalenone on plasma concentrations of reproductive hormones and reproductive development of boars, the following experiment was conducted (Berger et al., 1981).

Eight littermate pairs of crossbred boars (Hampshire x Duroc x Yorkshire) were divided at 11 weeks of age, with one member of each pair randomly assigned to the control group and the other to the treatment group. Control boars received a corn-soybean meal growing diet (16% crude protein) supplemented with minerals and vitamins until reaching 45 kg of body weight (17 weeks of age), and were fed a 14% crude protein finishing diet thereafter. Pigs were fed ad libitum until average weight reached 100 kg (25 weeks of age). Subsequent feed intake was limited to an average of 2.2 kg/boar daily. Boars in the treatment

group were fed the same diet in a similar manner, except that 40 ppm zearalenone (> 97% pure) was mixed with the diet fed from 14 to 18 weeks of age. Every other week from 12 to 32 weeks of age, boars were bled between 1000 and 1300 hr by puncture of the jugular vein.

Libido was examined biweekly from 21 to 35 weeks of age. Boars were assigned a libido score based upon their behavior during 5 minute exposure to an ovariectomized gilt. Estrus had been induced in the gilts 3.5 days earlier with an intramuscular injection of 1.5 mg estradiol benzoate in sesame seed oil. Libido scores were: 0, no sexual interest; 1, some sexual interest; 2, a great deal of sexual interest; 3, one or more false mounts; 4, one correct mount; 5, repeated correct mounts; 6, penis extension; 7, intromission, and 8, ejaculation. Mating was allowed to continue if begun during the 5 minute exposure.

The sexual behaivor of boars fed a control diet or a diet containing zearalenone is presented in table 6. Boars in the control group had higher libido scores than boars fed zearalenone. Libido in the control boars was greater than libido in the boars fed zearalenone at 29 and 31 weeks of age. Libido scores for boars fed zearalenone began to increase at 33 weeks of age.

TABLE 6. SEXUAL BEHAVIOR OF BOARS FED A CONTROL DIET OR A DIET CONTAINING ZEARALENONE.

	Libido[a]		% of animals mating	
Age, weeks	Control boars	zeara-lenone	Boars fed Control boars	Boars fed zeara-lenone
21	2.3	1.1	0	0
23	1.3	.2	0	0
25	2.3	.6	0	0
27	2.3	.5	12	0
29	3.5*	.7	25	0
31	5.0**	.6	50*	0
33	5.1	3.0	62	25
35	5.1	3.1	50	12

[a]Values listed are least-squares means for eight observations; SEM = .7.
*Greater (P<.05) than the corresponding value for the boars fed zearalenone.
**Greater (P<.01) than the corresponding value for the boars fed zearalenone.

Before zearalenone was fed, there was no difference (P>.25) between the two groups of boars in plasma concentrations of testosterone or LH (table 7). The feeding of zearalenone from 14 to 18 weeks depressed (P<.05) plasma concentrations of testosterone (.86 ng/ml) to levels below those in control boars (1.68 ng/ml). After withdrawal of zearalenone, plasma concentrations of testosterone in the treated boars tended to be lower (P<.10) than concentrations in the control boars (1.67 vs. 2.50 ng/ml). During the zearalenone

feeding period, there was a tendency (P<.10) for plasma LH to be depressed. There was no difference due to treatment following the feeding period.

TABLE 7. PLASMA CONCENTRATIONS OF TESTOSTERONE AND LUTEINIZING HORMONE IN BOARS FED A CONTROL DIET OR A CONTROL DIET CONTAINING ZEARALENONE.

Hormone	Observation period	Control boars[a]	Boars fed zearalenone[a]	SEM	pb
Testosterone, ng/ml	Prefeeding	.66	.89	.15	>.25
	During feeding	1.68	.86	.22	<.05
	Postfeeding	2.50	1.67	.30	<.10
LH, ng/ml	Prefeeding	1.89	2.14	.60	>.25
	During feeding	1.54	.79	.23	<.10
	Postfeeding	.85	.72	.25	>.25

[a]Values listed are least-squares means for eight observations.
[b]Probability estimate.

In summary, boars fed 40 ppm zearalenone from 14 to 18 weeks of age had reduced libido scores and depressed concentrations of serum testosterone. Recently, Ruhr et al. (1983) have reported that mature boars consuming feed with 200 ppm zearalenone had normal libido scores and semen concentrations of sperm. Further experimentation will be needed to determine if prepubertal and postpubertal boars react differently to diets containing zearalenone.

TREATMENT OF MOLDY CORN

Various treatments have been tried over the years to remove mycotoxins from infected corn in order that swine would consume it. F. roseum contaminated corn has been treated with propionic acid, ascorbic acid or anhydrous ammonia with some success but the cost is somewhat prohibitive. Since zearalenone and deoxynivalenol are water soluble, corn can be washed free of the mycotoxins but this procedure is very expensive and time-consuming. Adding aflafla to increase dietary fiber helps to slow absorption of the toxins (James and Smith, 1982). Additions of zeolite and folic acid have also been reported to slow absorption of the compounds into the bloodstream of swine. However, at the present time the most efficient way to utilize moldy corn infected with mycotoxins is to blend it with sound corn in such a proportion that the animal cannot detect it. Such blended corn can be used for market gilts and barrows. Based on data obtained in the experiments presented in this paper, Fusarium roseum infected corn should never be fed to pregnant, lactating, cycling or replacement gilts or to boars. Reproductive problems certainly would occur.

LITERATURE CITED

Bristol, F.M. and S. Kjurickovic. Hyperestrogenism in female swine as the result of feeding moldy corn. Can. Vet. J. 12:132, 1971.

Cantley, T.C., D.A. Redmer, R.D. Osweiler and B.N. Day. Effect of zearalenone mycotoxin on luteal function in gilts. Abstract at Midwestern Animal Science Meetings, 1982.

Chang, K., H.J. Kurtz, and C.J. Mirocha. Effects of the mycotoxin zearalenone on swine reproduction. Am. J. Vet. Res. 40:1260, 1979.

Christensen, C.M., C.J. Mirocha, G.H. Nelson and J. F. Quast. Effect of young swine of consumption of rations containing corn invaded by Fusarium roseum. Appl. Microbiol. 23:202, 1972.

Christensen, C.M. Zearalenone in Conference in Mycotoxins in Animal Feeds and Grains Related to Animal Health. William Shimoda, ed. p. 1-79. Food and Drug Administration, Rockville, Maryland, 1979.

Etienne, M. and M. Jemmali. Consequences de lingestian de Mais fusarie par la Truie reprodectrice. C.R. Acad. Sc. Paris. Serie D, 288:779-782, 1979.

Etienne, M. and M. Jemmali. Effects of zearalenone (F2) on estrous activity and reproduction in gilts. J. of Anim. Sci. 55:1, 1982.

Guthrie, H.D., D.M. Henricks and D.L. Handlin. Plasma estrogen, progesterone and luteinizing hormone prior to estrus and during early pregnancy in pigs. Endocrinology 91:675, 1972.

Hollander, M. and D.A. Wolfe. Nonparametric Statistical Methods. John Wiley and Sons, New York, 1973.

James, L.J. and T.K. Smith. Effect of dietary aflafla on zearalenone toxicity and metabolism in rats and swine. J. of Anim. Sci. 55:110, 1982.

Kurtz, H.J., M.E. Nairn, G.H. Nelson, C.M. Christensen and C.J. Microcha. Histologic changes in the genital tracts of swine fed estrogenic mycotoxin. Am. J. Vet. Res. 30:551, 1969.

Long, G.G. and M.A. Diekman. Effect of purified zearalenone on early pregnancy in gilts. Submitted to J. of Anim. Sci., 1983.

Long, G.G., M.A. Diekman, J.F. Tuite, G.M. Shannon and R.F. Vesonder. Effect of Fusariun roseum corn culture containing zearalenone on early pregnancy in swine. Amer. J. of Vet. Res. 43:1599, 1982.

Long, G.G., M.A. Diekman, J.F. Tuite, G.M. Shannon and R.F. Vesonder. Effect of Fusariun roseum (Gibberella zeae) on pregnancy and the estrous cycle in gilts fed molded corn on days 7-17 postestrus. Vet. Res. Comm. 6:199, 1983.

Mirocha, C.J., S.V. Pathre and C.M. Christensen. Zearalenone. In Rodricks, J.V., C.W. Hesseltine, and M.A. Mehlam (ed): Mycotoxins in Human and Animal Health. Forest Park South, Ill. Pathotox Publications, Inc, 1977, pp. 345.

Niswender, G.D. Influence of the site of conjugation on the specificity of antiobodies to progesterone. Steroids 22:413, 1973.

Official Methods of Analyses, 13th Edition, 1980. AOAC, Washington, D.C., USA.

Ruhr, L.P., G.D. Osweiler and C.W. Foley. Effect of mycotoxin zearalenone on reproductive potential in the boar. Am. J. Vet. Res. 44:483, 1983.

Ruhr, L.P., G.D. Osweiler and C.W. Foley. The effect of the mycotoxin zearalenone on fertility in the boar. J. Amer. Vet. Med. Assoc. 1975:608, 1979. (Abstr.).

Sharma, V.D., R.F. Wilson and L.E. Williams. Reproductive performance of female swine fed corn naturally molded or inoculated with Fusarium roseum. Ohio Isolates B and C. J. An. Sci. 38:598, 1974.

Shreeve, B.J., D.S.P. Patterson, B.A. Roberts and A.E. Wrathall. Effect of moldy feed containing zearalenone on pregnant sows. Br. Vet. J. 134:421, 1978.

Vanyi, A., A. Azeky, S.E. Rurnvaryne. Fusariotoxicoses. V. The effect of F-2 toxin on the sexual activity of female swine. Magy Allatorv Lapja 29:723, 1976.

Vesonder, R.F., A. Ciegler, R.F. Rogers, K.A. Burbridge, R.J. Bothast and A.H. Jensen. Survey of 1977 crop year preharvest corn for vomitoxin. App. Environ. Microbiol., 36:885, 1978.

Vesonder, R.F. and C.W. Hesseltine. Vomitoxin: Natural occurrence on cereal grains and significance as a refusal and emetic factor to swine. Process. Biochem., 16:11, 1980-81.

Young, L.G. and G.J. King. Feeding of low levels of zearalenone in diets of puberal gilts. Abstract at Annual Meeting of American Society of Animal Science, 1983.

The Effects of Mycotoxins on the Growth of Pigs

G. R. HOLLIS AND R. W. JONES

Introduction

Throughout human history, moldy feedstuffs have been used in many diverse and unique ways. More than 3,400 years ago, the Gibeonites tried to beguile Joshua by using moldy bread and an unthrifty appearance as symbols of their sincerity. Joshua, however, exposed the Gibeonites' deceit and made them his servants (Joshua, Chapter 9). Today's pork producer is faced with a problem somewhat similar to that of the Gibeonites. If he can beguile his stock into consuming mold-tainted corn and not become a servant to unacceptable pig performance, he can turn a moldy burden into useable feedstuff.

Molds produce toxic metabolites, and it is these compounds that may affect the growth of pigs. The molds most commonly found in feedstuffs and now receiving much attention are Aspergillus flavus, which produces aflatoxin, and Fusarium roseum, which produces the toxin zearalenone. Other mycotoxins found in feedstuffs include those produced by several Fusarium species; the tricothecene toxins (T-2 and vomitoxin); the ochratoxins, produced by several species of Aspergillus and Penicillum; and ergot, produced by Claviceps purpurea.

This review will focus chiefly on the effects of aflatoxin and zearalenone on the growth of pigs and on methods for safely including moldy corn in diets for growing and finishing swine. The growth-depressing effects of vomitoxin, T-2 toxin, ochratoxin, and ergot will also be examined briefly. Although not well researched with swine, two nutritional methods for reducing the depression in performance of swine that are fed mycotoxins as well as the practical application of those methods will be briefly discussed. Two reviews to be presented at this conference will discuss the mode of action of mycotoxins (Garst, 1983) and the effects of mycotoxins on the reproduction of swine (Diekman, 1983); therefore, those topics will not be covered here.

Aflatoxins

Aflatoxins affect all farm livestock, but swine are particularly sensitive. In fact, of all animals, only the duckling, trout, and cat are more sensitive than pigs to aflatoxins (Pier, 1981). As expected, younger pigs are more suscep-

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tible to aflatoxin poisoning than older pigs because the detoxification mechanisms of the liver are not fully developed (Pier, 1981). Four types of aflatoxin are produced by Aspergillus flavus: B1, B2, G1, and G2. Aflatoxin B1 is the most potent and, therefore, has been the most comprehensively investigated of the four as it has the most striking effect on the growth of pigs.

Effects on growth. Although numerous studies on the effects of aflatoxins on the liver and the other body organs have been reported, the quantitative effects of these toxins on growth rate and feed efficiency are not well investigated. In the few published studies on this subject, the method of recording the amount of aflatoxin ingested by pigs has varied and some have been reported incompletely. Results from several studies on feeding low doses of aflatoxin in growing and finishing swine are summarized in Table 1.

Studies from the United Kingdom (Duthie et al., 1966) have indicated that 280 parts per billion (ppb) of aflatoxin B1 in the diets of 40 to 140-pound pigs will not significantly impair performance, and in pigs from 140 to 200-pounds, the aflatoxin B1 level may be increased to 410 ppb without adversely affecting growth. Hintz et al (1967) concluded that growing and finishing pigs perform adequately on diets containing up to 480 ppb of total aflatoxin (450 ppb B1). In more recent work, however, Southern and Clawson (1979) have suggested that the maximum tolerance level of finishing swine to aflatoxin is about 385 ppb.

Table 1. Summary of the Effects of Aflatoxin on the Growth Rate (ADG), Feed Intake (ADF), and Feed Efficiency (G/F) of Growing and Finishing Swine

Aflatoxin content[a]	Initial weight or age	Final weight or age	ADG	ADF	G/F	Reference
(ppb)	(lb)	(lb)	(lb)	(lb)		
2	40	200	1.32[c]	-[b]	0.31[c]	Duthie et al
140	40	200	1.30[c]	-	0.30	(1966)
280	40	200	1.28[d]	-	0.30[d]	
410	40	200	1.25[d]	-	0.29[d]	
	(weeks)	(weeks)				
<6	13-14	29-30	1.56	-	0.26	Hintz et al
480			1.56	-	0.26	(1967)
765			1.50	-	0.27	
1005			1.03	-	0.23	
	(lb)	(lb)				
20	116	229	1.69[c]	6.31[c]	0.27[c]	Southern and
385	116	213	1.47[d]	5.57[d]	0.26[c]	Clawson
750	116	200	1.25[d]	4.73[e]	0.27[c]	(1979)
1480	116	176	0.90	3.54[f]	0.25[d]	

[a]Total aflatoxin, B1, B2, G1 and G2.

[b]Limit fed to 3.5 to 5.0 percent of body weight.

[c,d,e,f]Means with different superscripts in the same column differ ($P<.05$).

From the data in Table 1 and from the results of several qualitative studies (Gange et al., 1968; Duthie et al., 1968), a level of 400 ppb in the diets of

of growing and finishing swine does not impair performance.

Effects of feed efficiency. Reduced growth rate and feed intake are obvious subclinical signs of aflatoxin poisoning; however, it is interesting to note that the gain-to-feed ratio is not adversely affected by aflatoxin intake (Southern and Clawson, 1979). Although the depression in gain-to-feed ratio was statistically significant when finishing pigs were fed 1,480 ppb of aflatoxin, the decrease was nevertheless very small -- about 0.02 pound. The importance of this observation will be discussed later.

Zearalenone

Zearalenone, often noted as F-2 toxin and produced by Fusarium roseum, is commonly found on corn and hence is a major toxin with which pork producers in the Midwest must contend. It is an estrogenic compound, and it has been noted that its principal effect is on sows and their reproduction efficiency. Despite the growth-enhancing effects of zearalenone and its derivatives in cattle and chickens, the growth of swine is affected dissimilarly.

Effects on growth. Reported research has been directed, as one would expect, towards the effects of zearalenone on reproduction in mature swine. Therefore, the effects of the estrogenic compound on growing and especially on finishing swine have been largely overlooked. The results of several studies on young growing pigs are presented in Table 2.

The performance of the young growing pig (from 13 to 15 lb) is highly influenced by zearalenone. Studies by Young et al. (1981, 1982) indicate that daily gain, intake, and feed efficiency is depressed by a dietary zearalenone concentration of less than 10 parts per million (ppm) (Table 2). On the other hand, James and Smith (1982) and Smith (1980), using slightly heavier pigs taken to a heavier weight (from 37 to 50 lb), found no relation between dietary zearalenone and growth even when appreciably higher levels of the compound were fed. In one case (James and Smith, 1982), a linear increase in growth rate appears to be attributable to zearalenone.

Table 2. Summary of the Effects of Zearalenone on the Growth Rate (ADG), Feed Intake (ADF), and Feed Efficiency (G/F) of Growing and Finishing Swine

Zearalenone content	Initial weight	Final weight	ADG	ADF	G/F	Reference
(ppm)	(lb)	(lb)	(lb)	(lb)		
0	15.4	30.1	0.70[a]	1.89	0.57	Young et al
3	14.5	25.7	0.53[b]	1.63	0.50	(1981)
6	14.5	25.3	0.51[b]	1.96	0.40	
9	13.6	21.3	0.37[c]	1.19	0.44	
0.0	14.5	21.6	0.33	0.95	0.35	Young et al
2.1	15.0	25.1	0.48	1.36	0.35	(1982)
3.7	15.2	18.4	0.20	0.81	0.20	
4.8	14.5	17.8	0.15	0.77	0.19	

Continued on next page

Zearalenone content	Initial weight	Final weight	ADG	ADF	G/F	Reference
	(weeks)	(weeks)				
10	8-11	18-23	0.81	1.65	0.49	James and
10			0.84	1.65	0.51	Smith (1982)
20			0.86	1.78	0.49	
40			0.97	1.80	0.53	
	(lb)	(lb)				
0	15.4	37.4	0.73	1.25	0.58	Smith (1980)
50			0.70	1.25	0.56	
0	15.4	37.4	0.97	1.72	0.57	
50			0.81	1.32	0.55	

[a,b,c]Means with different superscripts in the same column differ (P<.05).

These apparent contradictions may be related to the length of the studies: the studies conducted over a period from 14 to 21 days showed growth depression, whereas studies conducted over a 28-day period did not. The diets used by Young et al. (1981) were contaminated with small amounts of vomitoxin, which is known to reduce intake and growth (Forsyth et al., 1977), but the authors argued that the presence of vomitoxin did not affect the results. In addition, the contradictory results could be explained by a more mature detoxification system in older, heavier pigs.

The effects of zearalenone on seven-month-old gilts were quite different. Weight gains from first estrus to farrowing were significantly higher in gilts fed zearalenone (2.1 to 4.8 ppm). Moreover, gilts fed zearalenone lost significantly less weight from the time of farrowing to 14 days post-partum that those fed a zearalenone-free diet (Young et al., 1982). It is not immediately clear whether this decrease in post-farrowing weight loss is due to an increase in the weight of reproductive tissue. Unfortunately, no studies have been reported on the effects of zearalenone on finishing pigs. Data from finishing barrows would be helpful in explaining how age relates to zearalenone toxicity in swine.

Because of the conflicting evidence about the effect of zearalenone on the growth of young pigs, it is difficult to specify the amount of zearalenone that can be safely included in the diet. It appears that zearalenone concentrations up to 50 ppm do not appreciably affect growth (Smith, 1980), but more work in this area is certainly needed.

Effects on feed efficiency. Experiments conducted by James and Smith (1982) and Smith (1980) did not demonstrate that zearalenone produces a decrease in feed efficiency, but those of Young et al. (1981, 1982) showed a significant reduction in feed efficiency. Unlike aflatoxin, which produces a decrease in feed intake but no corresponding decrease in gain-to-feed ratio (Table 1), zearalenone decreases efficiency as it reduces feed intake (Young et al., 1981, 1982). The effect of zearalenone on the feed efficiency of older pigs remains unclear.

Other Mycotoxins

As the science of toxicology benefits from improved technology we are able to isolate and identify toxins in feedstuffs with greater precision and in smaller concentrations. As a result, toxins other than aflatoxin and zearalenone, although

not new to the toxicologist, have only recently come to the attention of the pork producer.

Vomitoxin. Produced by several Fusarium species, vomitoxin has been found to contaminate corn alone and in combination with zearalenone. The presence of vomitoxin (deoxynivalenol) -- the most common tricothecene toxin in the United States -- in corn has been reported to cause decreased feed intake, feed refusal and vomiting when consumed by Pigs (Vesonder et al., 1976; Pier, 1981).

Pollman et al. (1983) fed vomitoxin-contaminated wheat diets to starter and finisher pigs and noted a decrease in gain and feed intake. They concluded that wheat-based diets containing more than 1 ppm vomitoxin will result in reduced weight gains and feed intake in growing and finishing swine.

A drastic decrease in intake of corn-based diets contaminated with vomitoxin and a resulting decrease in gain has also been noted by Forsyth et al. (1977). A vomitoxin concentration of 3.6 ppm decreased intake by 20 percent from 1.41 to 1.13 kg per day in 20-kg pigs. Almost complete (90 percent) refusal of feed occurred when 40 ppm vomitoxin was added to the diet. As feed intake decreased exponentially, so did the rate of gain and the gain-to-feed ratio. The authors state that feeding naturally contaminated corn rather than adding pure vomitoxin to the diets resulted in even greater depressions in feed intake. They suggested that the presence of other mycotoxins, such as zearalenone, may be responsible for the increased refusal of feed.

In fact, vomitoxin is often found in combination with zearalenone. Together, these toxins have a significant influence on growth rate, feed efficiency, and feed intake. The results reported by Young et al. (1981) and summarized in Tables 3 and 4 represent the situation that often occurs when moldy corn is fed to swine.

A decrease in feed consumption was observed when pigs were fed diets containing 6.7 ppm zearalenone plus 3.6 ppm vomitoxin; however, feed consumption appeared increased when the diets contained 4.4 ppm zearalenone and 1.7 ppm vomitoxin.

Further results by Young et al. (1983) showed that a dietary level of approximately 20 ppm vomitoxin caused vomiting, 12 ppm vomitoxin caused almost complete feed refusal and 1.3 ppm caused a significant depression in feed intake and rate of gain. If it is assumed that older swine are able to metabolize zearalenone in greater amounts than reported in this study, it must be concluded that vomitoxin has very serious effects on production.

Table 3. The Effects of Zearalenone and Vomitoxin on the Growth Rate (ADG), Feed Intake (ADF), and Feed Efficiency (G/F) of Gilts Fed Corn-Soy Diets for 21 Days

Level of zearalenone (ppm)	Level of vomitoxin (ppm)	ADG (lb)	ADF (lb)	G/F
0.0	0.0	2.07	6.64	0.32
1.5	2.9	1.69	5.98	0.29
3.0	5.7	0.57	2.82	0.14
4.5	8.7	0.44	3.78	0.18

Source: Young et al., 1981. (Trial 1).

Table 4. Performance of Young Pigs (14.5 lbs) Fed Moldy Corn[a]

Level of zearalenone (ppm)	Level of vomitoxin (ppm)	ADG (lb)	ADF (lb)	G/F
0.0	0.0	.70	1.26	.57
2.5	<1	.53	1.08	.50
4.4	1.7	.50	1.30	.40
6.7	3.6	.37	0.78	.44

[a]21-day trial.
Source: Young et al., 1981 (Trial 3).

T-2 toxin. Cognate to zearalenone and vomitoxin, T-2 toxin is produced by several Fusarium species but is unique because it is formed during storage when the temperatures fall to between 43° and 65°F. T-2 toxin and diacetoxyscirpenol (DAS), both tricothecene toxins, have been found to reduce growth in swine, but quantitative data are limited. Weaver et al. (1977), as reported by Pier (1981), indicated that the rate of gain in swine was decreased by feeding from 1 to 10 ppm DAS, but 8 ppm T-2 toxin had no effect on weight gain. Feed refusal was observed at levels of 16 ppm T-2 toxin and 10 ppm DAS.

Ochratoxin. Produced by several species of Aspergillus and Penicillum, ochratoxin can infest barley, oats, corn, and wheat. It is a problem primarily on the Southeastern coast. As expected, the first observable signs of ochratoxicosis in swine are slow growth and reduced feed intake. The primary target organ for ochratoxin is the kidney (Rutqvist et al., 1978), to which this toxin can cause irreparable damage. Although no reports indicate the response of pigs to subchronic levels of ochratoxin in the diet, Szczech et al. (1973) reported that three-to-four-week-old pigs fed 28 ppm of ochratoxin died within three weeks. Clearly, ochratoxin is very toxic to pigs. Further work is needed to determine the ochratoxin tolerance level in pigs.

Ergot. According to Pier (1981), ergot is the mycotoxin that has longest been recognized to affect animal and human health through its presence in food and feedstuffs. Documented epidemics of human ergotism in central Europe occurred as early as 837 A.D. Ergotism is caused by a variety of alkaloids and lysergic acid derivatives produced by Claviceps purpurea and may be divided into two types: nervous and gangrenous. Nervous ergotism is characterized by vertigo, staggers, convulsions, temporary posterior paralysis, drowsiness, and eventual death. In gangrenous ergotism, the blood supply to the periphery -- limbs, ears, and tail -- is decreased resulting in loss of these extremities (Burfening, 1973). Swine have been affected by both types of ergotism. Burfening (1973) reported reduced growth rates, the loss of tails, and lowered reproductive efficiency in sows. The growth rate reduction, he postulated, was due to the reduced palatability of the ergot-containing feed. The effect on feed efficiency was not reported, and quantitative data on growth rate are not available.

Dietary Methods for Reducing Mycotoxin Injury

Mycotoxins can be fed at certain levels without appreciably altering the growth rate of swine. Two nutritional methods can be used to insure against reduced growth rates when diets containing tolerable amounts of mycotoxins are fed

or to reduce growth depression when it is necessary to feed diets containing more than the tolerable level. Although not tested in practical situations, these methods could play a major role in treating aflatoxin and zearalenone mycotoxicosis.

Protein concentration. It is reasonable to assume that nutritional stress will aggravate toxin-induced stress. Less than optimum concentrations of dietary protein have indeed been shown to influence the response of pigs to aflatoxin and zearalenone. Using miniature swine, Sisk and Carlton (1972) found that the decrease in growth was accentuated when the crude protein was decreased from 17.0 to 11.4 percent in the diets of 10-week-old pigs given small doses of aflatoxins. Comparisons of pigs fed diets containing 17 percent crude protein with and without the administration of aflatoxins showed no significant differences in weight gain.

Likewise, the protective effects of dietary crude protein on zearalenone toxicosis in pigs have been indicated. In a recent study (Smith, 1980) the growth rate of immature gilts was slightly depressed by 50 ppm of zearalenone. By increasing the dietary crude protein concentration from 16.2 to 29.4 percent, the effects of zearalenone on growth rate, feed intake, and feed efficiency were completely eliminated.

The practical significance of these studies remains tenuous. The increases in the concentration of dietary protein are substantial and in most cases would not be economically feasible for the producer. However, these studies indicate the importance of proper diet formulation when feeding mycotoxin-tainted grains. Protein concentration is especially important because most mycotoxins impair the utilization of dietary protein. The interferences due to aflatoxin and zearalenone may be lessened by increasing dietary protein, whereas protein supplements appear to have little effect on tricothecene mycotoxicosis (Smith, 1982).

Riboflavin concentration. Although the interactions between riboflavin, aflatoxin, and sunlight have only been explored in the rat, some interesting results have been reported (Joseph-Bravo et al., 1976). It has been shown *in vivo* that aflatoxin will cause blood riboflavin levels to decrease, an effect which is enhanced by the irradiation of aflatoxin and/or riboflavin. This suggestion that photosensitized riboflavin and aflatoxin form a complex is complemented by *in vitro* studies indicating that riboflavin quenched aflatoxin photodegradation by complexing with perhaps aflatoxin. The authors concluded from their extensive studies that low, carcinogenic doses of aflatoxin may complex with endogenous, photosensitized riboflavin, inhibiting its degradation into carcinogenic metabolites.

Compared to other vitamins, riboflavin is relatively inexpensive and may prove to be economically feasible for the producer to use as an anti-aflatoxin additive. However, positive results must first be shown in the pig before unwarranted trust is given to riboflavin as a possible treatment for aflatoxicosis. The effects of combining riboflavin and dietary protein are also unknown.

Conclusion

Mycotoxin-contaminated grain does not have to be the burden it once was. Although the Gibeonites could not fool Joshua with their moldy bread, growing and finishing swine may be fooled into consuming moldy grain. If the level of each mycotoxin is kept at or below the pig's tolerance level, performance -- daily gain, feed intake, and feed efficiency -- need not suffer. However, the producer must correctly formulate the diet to insure that no more than the tolerable amount of mycotoxin is present and that the proper amount of protein is included. In the

future it may be possible to reduce the toxicity of mycotoxins by forming complexes with other compounds such as irradiated riboflavin, but further work in this area is greatly needed.

LITERATURE CITED

Burfening, P. J. 1973. Ergotism. J. Amer. Vet. Med. Assoc. 163:1288.

Diekman, M. 1983. Effects of toxins on swine reproduction. 1983. Pork Industry Conference, University of Illinois, Urbana, Illinois.

Duthie, I. F., M. C. Lancaster, J. Taylor, D. C. Thomas, C. A. Shacklady, P. H. Attfield and E. Fuller-Lewis. 1966. Toxic groundnut meal in feeds for pigs. Part 1. A trial made at two laboratories with pigs from about 40 to 200 lb live-weight fed to a restricted scale. Vet. Rec. 79:621.

Duthie, I. F., M. C. Lancaster, J. Taylor, E. B. Lomax and H. M. Clarkson. 1968. Toxic groundnut meal in feeds. Part 2. The effect of consuming groundnut meal during part of the growing period or during the finishing period. Vet. Rec. 82:427.

Forsyth, D. M., T. Yoshikawa, N. Mooroka and J. Tuite. 1977. Emetic and refusal activity of deoxynivalenal to swine. Appl. Environ. Microbiol. 34:547.

Gange, W. E., D. L. Dungworth and J. E. Moulton. 1968. Pathological effects of aflatoxin in pigs. Pathol. Vet. 5:370.

Garst, J. E. 1983. A primer in toxicology. 1983 Pork Industry Conference, University of Illinois, Urbana, Illinois.

Hintz, H. F., A. N. Booth, A. F. Cacullu, H. K. Gardener and H. Heitman, Jr. 1967. Aflatoxin toxicity in swine. Proc. Soc. Exp. Biol. Med. 124:266.

James, L. J. and T. K. Smith. 1982. Effect of dietary alfalfa on zearalenone toxicity and metabolism in rats and swine. J. Anim. Sci. 55:110.

Joseph-Bravo, P. I., M. Findley and P. M. Newberne. 1976. Some interactions of light, riboflavin, and aflatoxin Bl *in vivo* and *in vitro*. J. Toxicol. Environ. Health 1:353.

Pier, A. C. 1981. Mycotoxins and animal health. Adv. Vet. Sci. Comp. Med. 25:185.

Pollman, D. S., B. A. Koch and L. Sietz. 1983. Effect of vomitoxin-contaminated wheat in swine diets. J. Anim. Sci. Suppl. 1, 57:94 (abstract).

Rutqvist, L., N. E. Bjorklund, K. Hult, E. Hokby and B. Carlsson. 1978. Ochratoxin A as the cause of spontaneous nephropathy in fattening pigs. Appl. Environ. Microbiol. 36:920.

Sisk, D. B. and W. W. Carlton. 1972. Effect of dietary protein concentration on response of miniature swine to aflatoxins. Am. J. Vet. Res. 33:107.

Smith, T. K. 1980. Influence of dietary fiber, protein and zeolite on zearalenone toxicosis in rats and swine. J. Anim. Sci. 50:278.

Smith, T. K. 1982. Influence of mycotoxins on protein and amino acid utilization. Fed. Proc. 41:2828.

Southern, L. L. and A. J. Clawson. 1979. Effects of aflatoxins on finishing swine. J. Anim. Sci. 49:1006.

Szczech, G. M., W. W. Carlton, J. Tuite and R. Caldwell. 1973. Ochratoxin A toxicosis in swine. Vet. Path. 10:347.

Vesonder, R. F., A. Ciegler, A. H. Jensen, W. K. Rohwedder and D. Weisleder. 1976. Co-identity of the refusal and emetic principle from Fusarium infected corn. Appl. Environ. Microbiol. 31:280.

Weaver, G. A., H. J. Kurtz and C. J. Mirocha. 1977. Proc. 81st Ann. Mtg., U.S. Animal Health Assoc., pp. 215-218.

Young. L. G., L. McGirr, V. E. Valli, J. H. Lumsden and A. Lun. 1983. Vomitoxin in corn fed to young pigs. J. Anim. Sci. 57:655.

Young, L. G., G. J. King, L. McGirr and J. C. Sutton. 1982. Moldy corn in the diets of gestating and lactating swine. J. Anim. Sci. 54:976.

Young, L. G., R. F. Vesonder, H. S. Fennell, I. Simmons and B. Wilcock . 1981. Moldy corn in diets of swine. J. Anim. Sci. 52:1312.

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December 6-7, 1984
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WELCOME TO ANOTHER UNIVERSITY OF ILLINOIS PORK INDUSTRY CONFERENCE! This is an opportunity for us to present our current thinking on a number of very important facets of our industry that so importantly affect the bottom line in your record books or tax returns.

Today some rather new terms will be bandied about and you may as well get used to hearing them for they have already been seen in the popular press and undoubtedly will become as common as such terms as crossbreeding, balanced rations, hedging, swine sales or feed supplies. Biotechnology, repartitioning agents, prostaglandins, monoclonal antibodies, irradiation, behavior, genetic engineering, computers and the list goes on and on. It is certainly not the purpose of the speakers today to impress you with their expanded vocabulary but rather to tell you of basic research results as well as of some already applicable management practices that can increase litter size, for example, hence letting us capitalize on the improved genetics we worked so hard to build into our herds.

The whole field of nutrition-reproduction interactions is receiving renewed attention in all farm species. Sexual maturity, libido, postweaning estrus and rebreeding, litter size, pig birth weight and vigor and still more factors determining the reproductive performance are so vitally affected by the nutritional program in the herd and this will be a highlight of today's program.

Enjoy yourselves and take this opportunity to become better acquainted with our staff, both old and new.

A. L. Neumann
Acting Department Head
Department of Animal Science

The Department of Animal Science at the University of Illinois

THE DEPARTMENT OF ANIMAL SCIENCE IS CONCERNED WITH THE MULTIDISCIPLINARY ACTIVITIES associated with the production, care, and utilization of animals useful to mankind. It includes primary fields of study in behavior, genetics, environmental physiology and management, meat science and muscle biology, nutrition, and reproductive physiology. Beef cattle, horses, poultry, sheep, swine, and various companion and laboratory animals are studied to assist animal producers and owners to obtain more efficient performance, more economical production and other such improvements which ultimately benefit the general public.

Our staff includes about 140 people of whom 50 are academic staff members; 40 are nonacademic staff members in the offices, research farms, and laboratories and about 50 are part-time teaching or research assistants who are pursuing graduate study. Several of the senior academic staff members have received national and international recognition for their accomplishments.

The work of the Department is divided into extension, research, and teaching. Extension or off-campus teaching is handled by eight full-time staff members. Each extension specialist conducts seminars, clinics, field days, etc., for livestock or poultry producers. Their primary work is to apply new research findings to the business of animal production or product processing.

Over the years the Department has had very active and productive research programs. Some of the more notable research accomplishments include the discovery of the value of antibiotics in livestock feeding, the elucidation of the amino acid needs of swine and poultry, the utilization of inorganic nitrogen by ruminants, the development of simplified corn-soybean meal rations for swine feeding, and the development of confinement production technics, particularly the use of slatted floors. Current research studies pertain to such topics as recycling of animal wastes, decreasing prenatal mortality in gestating sows, the inheritance of blood groups in swine, exercise physiology in horses, alcohol treatment of soybean meal, factors affecting amino acid needs of domestic animals, and animal behavior.

With the recent increase in student numbers, the teaching load in Animal Science increased significantly. At present our Department provides instruction for about 2,800 students a year in the classroom, and staff members advise 400 undergraduate (mostly juniors and seniors) and 90 graduate majors. Our graduates have many opportunities for employment.

The future of the Department of Animal Science appears bright. Meat consumption and the use of animals for companionship and in recreational activities are high and seem likely to increase further. Such an expanding animal industry will require an educational program to produce well-trained animal scientists and research programs geared to produce new and improved technology.

Dedication

The 1984 University of Illinois Pork Industry Conference is dedicated to Dr. D. E. Becker who was instrumental in founding this conference in 1977. Dr. Becker retired as Head of the Department of Animal Science in August of this year.

Except for a year served at the University of Tennessee, Dr. Becker spent his entire 35-year professional career at the University of Illinois. A brilliant researcher and teacher, Gene rose through the ranks rapidly, becoming Professor of Swine Nutrition in 1958. He was named Head of the Department of Animal Science in 1967, a position he held until his retirement in August of this year. For his innovative research work in the area of protein-amino acid and carbohydrate nutrition and metabolism, Gene was awarded the prestigious American Feed Manufacturers Award in 1957. In the early sixties, Gene led the fight for acceptance of the simplified corn-soybean meal diet for swine of all weights and ages. Through his efforts, this diet became and is today the standard of excellence, not only in the United States but throughout the world. For this and other work, Dr. Becker was given the American Society of Animal Science's Morrison Award in 1977, the most prestigious award obtainable in the Animal Sciences. He also received the University of Illinois Paul A. Funk Award in 1972, an award for lifelong service to Illinois Agriculture. He held positions of Associate Editor and then Editor of the Journal of Animal Science. He also served the American Society of Animal Science in several executive positions, including a two-year term as President.

Dr. Becker put forward the first complete set of amino acid requirements for growing-finishing swine. He, together with his graduate student trainees, were also among the first to establish protein and amino acid requirements for pregnant and lactating sows. Many of his former Ph.D. students have become distinguished scientists in their own rights and, in fact, have gone on to win national and international awards themselves. Many today hold prominent positions in both academia and industry.

Gene Becker had an unswerving dedication and loyalty to the pork producers of Illinois. He knew most of them personally. He was active in the Illinois Pork Council, serving as its Secretary-Treasurer from 1961 to 1967. He and leaders of the pork industry established the Fighting Illini Pork Club in 1973. Through the efforts of this organization, one football game each year is designated "Pork Day".

As Department Head, Dr. Becker built his department into one of the finest in the nation. He recruited outstanding young faculty members and then dedicated himself to helping them become productive teaching, research and public service leaders. He raised the necessary funds to institute a unique departmental awards program. He also believed strongly in the comparative approach to research and, as such, defended use of rabbits, horses, cats and dogs in animal science experiments. His vision also extended to nontraditional disciplines. Thus, the Animal Science Department at the University of Illinois was either first or among the first to enter areas such as environmental physiology, companion animal biology, animal behavior and ethology, toxicology, immunology and biotechnology. Dr. Becker demanded dedication and excellence in himself and he expected his faculty to follow suit. The Swine Research Center at the U of I is generally considered to be the finest swine research establishment in the world. This, too, is a tribute to his leadership, dedication and vision.

People of Gene Becker's intellectural capacity, purposefulness and vision come along once or twice in a lifetime. For his many contributions to the swine industry of Illinois, the 1984 Illinois Pork Industry Conference is dedicated to Dr. D. E. Becker.

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Genesis of a Pig–Conception to Parturition

PHILIP J. DZIUK

So, you would like to be a piglet and also be a live one. You would like to know what it takes to achieve that position. The process is quite involved and requires about 114 days and you must go through all the steps and stages correctly. Some start but don't finish. The miracle is that so many do finish and develop into everyday pigs.

First, pick out two healthy parents, one needs to produce viable sperm and the other must produce eggs capable of being fertilized. Your father should produce about 40 to 50 billion sperm per ejaculation. Not that more than 15 sperm are needed to actually fertilize the eggs, but that fertilization is a case of chance or probability. A bird in flight is easier to shoot with a shotgun with 200 missiles than a rifle with one missile. While it is important to have at least one sperm fertilize each egg it is equally important to limit the number of sperm that penetrate the egg to just one. If more than one sperm penetrates the egg, polyspermy results and the embryo will not develop. Too many cooks spoil the soup. The number of sperm reaching the site of fertilization are usually very restricted by the valve-like structure between the uterus, where the sperm are deposited, and the oviduct where fertilization occurs. The outer layer of the egg reacts chemically to the penetration of the first sperm and prevents subsequent sperm from penetrating all the way. Many sperm may later attach to the surface of the egg but do not penetrate the outer layer.

Up to this point your father and mother have met, sperm were deposited and were waiting. A few hours after the end of heat the egg is ovulated and fertilized. The genetic information from your two parents is fused or combined to provide all the genetic instructions you will need for the remainder of your life. You will be only one cell for about the first 22 hours after fertilization. At 24 hours you will be two cells and at 48 hours you will be 4 to 8 cells. Each of your 8 cells retains the ability to develop a complete individual. The cells have not reached the stage of differentiating and dividing the responsibilities of organs and tissues. At 48 hours after fertilization you leave the narrow oviduct and enter the uterus together with your siblings arising from eggs from the same ovary you came from. On the other side of your mother another group of siblings that come from the other ovary are entering the other end of the "V" shaped uterus. At this stage of development each of your cells now has lost its ability to develop into a complete individual and some cells are now destined to become

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the placenta, some will form the fetus. Until day 6 you will remain near the tips of the uterine horns where you entered orginally. By day 7 the contractions of the uterus have moved some of you and your siblings away from the top and by day 8 your brothers and sisters from both sides of the uterus have just about met at the bottom of the "V" shaped uterus. You will continue to be moved farther for the next 4 days until some embryos from the ovary on the right side are now in the left side of the uterus and vice versa. It is interesting that embryos from each ovary can meet and pass one another going in opposite directions in the uterus. At day 11 you now consist of several hundred cells in the form of a hollow ball that resembles a miniature collapsed football. You are visible as a white bit of tissue about 1/4 to 1/2 inch long. At day 12 you can no longer move throughout the uterus but attach and begin to elongate at a very rapid rate. By day 14 the trophoblast consisting of the embryo and placenta is 12 inches or more in length and there is a slight fold and thickening of the long filamentous tissue where you as the fetus will form. The remainder will form the placental membranes. On day 17 you are suspended in the fluid in the amniotic sac. You have formed blood and your beating heart is visible. Your limbs, eyes, liver and general form is established by day 25. With a little imagination one could identify you as a pig even though you are just slightly over an inch long. You and your placenta are producing a very high level of hormones at this time. These hormones pass into the circulation of your mother and can be measured. Some estrone is produced by each of your siblings so that measurement of a metabolite, estrone sulfate, in blood from your mother will give an indication of the number of brothers and sisters you have. Earlier at day 12 and 13 each of you was also sending a message to your mother that you were present and that she should not develop a new set of follicles and return to heat, but rather she should maintain the corpora lutea on the ovary to continue the secretion of progesterone essential for gestation. These same corpora lutea formed from the rupture of the follicles producing each egg from which you are derived. The number of corpora lutea throughout gestation is a very good indication of the number of eggs ovulated. The proportion of embryos or fetuses surviving can be determined by counting the corpora lutea and the fetuses.

The uterus of your mother began at heat as a long "V" shaped tube, with each arm of the "V" about 34 inches long. The hormones from each of your litter mates have caused the uterus to nearly double this length to 70 inches. Each fetus needs a minimum of about 10 inches of space to grow properly and survive. If your litter mates distributed uniformly at day 12 and each had approximately equal space then each uterine horn could accommodate seven fetuses. If one embryo grows more slowly and is crowded into a smaller space it may either be a runt at birth or may not survive throughout gestation and be resorbed. If you, as this little pig, are to survive be certain to establish your position by day 12, grow rapidly and "stick your elbows out" to occupy enough space to grow and survive. From day 25 to day 112 it is only really necessary to grow, develop and differentiate. Your skin color and hair color are distinguishable at day 85 whereas your sex was obvious at about day 35. Boars in particular produce quite high levels of testosterone as fetuses at days 35 to 70 to assist in converting the embryo into a male.

When the end of gestation approaches you cause the level of estrogen to rise in your mother, her corpora lutea become less functional thus the level of progesterone declines. Because progesterone keeps the uterus quiet and estrogen causes it to be sensitive to stimuli, the stage is now set for uterine contractions. You are about to leave your snug, protected home for the outside world. Prostaglandins and their analogues cause the corpora lutea to regress and

progesterone to decline while causing a peak of relaxin. This phenomenon is caused by some unknown signal from you and your litter mates. When progesterone is low enough, relaxin has caused the cervix to relax to allow passage of the fetuses from the uterus to the vagina and estrogen level is high, labor beings. Your litter mates in each horn are born more or less alternately. Your siblings at the bottom of the "V" are born first and those at the tip of the "V" are born last and are more likely to be stillborn than others. Giving injections of progesterone to your mother beginning about day 112 will prevent parturition. This delay is usually associated with a greater proportion of dead fetuses. Fetuses that implant in the upper part of the uterine horn that have one or no other siblings in that horn will nearly always be stillborn. Having a sibling previously occupy the uterus that you must transverse during birth is beneficial to your survival. Good Luck in your quest to be a live pig. There are many hazards but many achieve success.

REFERENCES

Baker, R. D., P. J. Dziuk and H. W. Norton. Polar body and pronucleus formation in the pig egg. J. Exp. Zool. 164:49-498. 1967.

Baker, R. D., P. J. Dziuk and H. W. Norton. The effect of volume of semen, number of sperm and drugs on transport of sperm in artificially inseminated gilts. J. Animal Sci. 27:88-93. 1968.

Dhindsa, D. S., P. J. Dziuk and H. W. Norton. Time of transuterine migration and distribution of embryos in the pig. Anat. Rec. 159:325-330. 1967.

Dickmann, Z. and P. J. Dziuk. Sperm penetration of the zona pellucida of the pig egg. J. Exp. Biol. 41:603-608. 1964.

Dziuk, P. J. Effect of number of embryos and uterine space on embryo survival in the pig. J. Animal Sci. 27:673-676. 1968.

Dziuk, P. J. Control and mechanics of parturition in the pig. Animal Reproduction Sci. 2:335-342. 1979.

Dziuk, P. J. and B. G. Harmon. Succession of fetuses at parturition in the pig. Am. J. Vet. Res. 30:419-421. 1969.

Dziuk, P. J., C. Polge and L. E. Rowson. Intra-uterine migration and mixing of embryos following egg transfer. J. Animal Sci. 23:37-42. 1964.

Polge, C. and P. J. Dziuk. Time of cessation of intrauterine migration of pig embryos. J. Animal Sci. 31:565-566. 1970.

Sherwood, O. D., C. C. Chang, G. W. BeVier, J. R. Diehl and P. J. Dziuk. Relaxin concentrations in pig plasma following the administration of prostaglandin $F_2\alpha$ during late pregnancy. Endocrinology 98:865-879. 1976.

Webel, S. K. and P. J. Dziuk. Effect of stage of gestation and uterine space on prenatal survival in the pig. J. Animal Sci. 38:960-963. 1974.

Nutrition to Support Critical Reproductive Functions

R. A. EASTER

INTRODUCTION

Reproduction can be viewed as a process of converting nutrients into a host of biological tissues, the sum of which is a healthy pig at weaning and a sow ready for rebreeding. The problem is to provide the proper nutrients at the right time during the process and in the correct amounts. Much has been done in the past 25 years to define the essential components of a successful feeding program for the brood sow thoughout her reproductive cycle. This is a brief review of some of the knowledge that has led to the development of current recommendations.

A discussion of nutrition and reproduction must always begin with the assertion that successful nutrition of the sow is a continuous process. It begins with the developing female and continues so long as she remains a part of the herd. Thus, successful lactation is dependent on good nutrition during pregnancy and successful rebreeding must be preceded by a sound feeding program during lactation.

ENERGY

Reproduction, like all biological processes is dependent on sufficient energy to drive essential reactions. Simply stated, energy intake must be sufficient to meet metabolic needs for maintenance, synthesis, work and to balance losses to the environment. Because of the complexity of the these needs it is impractical to state daily requirements in quantitative terms. Thus, the animal caretaker must understand the basis for establishing the daily caloric needs of individual animals.

A gilt consuming a diet inadequate in energy will experience a delay in the onset of first estrus by as much as 20 to 30 days (Cunningham et al., 1974; Friend, 1977). Gilts that are fed ad libitum reach puberty at a younger age but, interestingly, at about the same body weight. This suggests that weight is likely more important in the the achievement of puberty than is chronological age. It is believed in some circles that ad libitum feeding of prepubertal gilts will shorten their reproductive life. Recent evidence from Virginia does

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not support this notion (Arthur et al., 1983). Thus, the recommendation that developing gilts be fed free-choice until puberty appears to be consistent with available knowledge.

The discovery by Zimmerman et al. (1960) that ovulation rate could be improved by a substantial increase in energy intake for several days prior to breeding created much interest in the practice of "flushing" the breeding gilt. The research has been repeated many times worldwide with, frequently, contradictory results, c.f., Kirkpatick et al.,(1967). It is difficult to advocate the practice on the basis of available data. Dziuk (personal communication) has suggested that the increase in ovulation rate may be unrelated to energy intake but rather due to an effect on rate of digesta passage and thus hormone reabsorption from the lower bowel.

The possible adverse effect on embryo survival of excess energy intake, or "flushing" during early pregnancy has been suggested by several (Robertson et al., 1951, Frobish and Steele, 1970). Recent data (Toplis et al., 1983) does not substantiate a negative effect. However, excess caloric intake throughout the preganancy will precipitate farrowing difficulty, reduced litter size at weaning, impaired rebreeding and increased culling of sows. Inadequate energy intake results in smaller pigs at birth and weaning and the liklihood of poor sow body condition at the time of rebreeding (Vermedahl et al., 1969; Baker et al., 1969).

It is generally assumed that sows housed in a thermoneutal environment require between 5,500 and 6,500 kcal per day of metabolizable energy. This represents four to five pounds of a corn-soybean meal diet. Environmental conditions as well as sow condition can greatly alter this requirement as is indicated by the data in table 1. The values are calculated from several experiments and show that "fat" sows require less feed to remain comfortable at lower temperatures than do "thin" sows. The difference is largely due to the thermal insulation provided by a thicker layer of subcutaneous fat. This is not intended to suggest that sows should be "fat" just so the energy need can be reduced during pregnancy.

Table 1. Effect of Temperature on Feed Required for Maintenance of Pregnant Sows (grams)1

	Degrees (F) below thermal neutrality2			
Sow type	0	9	18	27
Fat	0	178	355	711
Thin	0	282	564	846

1 Holmes and Close (1977)
2 Lower critical temperature, 40-50 degrees F.

The practical implications of the data in table 1 are obvious. Feed intake must be increased during periods of cold stress. As the energy required for maintenance is increased the calories remaining for production decrease.

Although it has not been demonstrated experimentally, there is field evidence that prolonged inadequate energy intake by sows individually stalled in a cold barn can result in abortions.

It has been suggested by several investigators, c.f., Jensen et al.(1972), that a good index of energy status of the sow is weight gain during the pregnancy. First-litter gilts should achieve approximately 70 pounds of maternal gain, i.e., weight immediately after farrowing minus weight at breeding, while sows should gain approximately 40 pounds during each pregnancy. Composition of the sow's body changes gradually with maturity. European scientists have recommended that an increase in sow weight of about 45 pounds between the end of the first lactation and the end of the third lactation is desirable. After this the weight at the end of each successive lactation should remain constant.

If scales are unavailable for measuring weight gain during pregnancy, the descriptions in table 2 can be used as a rough estimate of sow condition. In general, an appearance corresponding to a score of 3.5 at the end of the pregnancy is desirable.

Table 2. Sow Condition Scores1

Condition score	Appearance
0	Emaciated, skin and bones
1	Backbone clearly visible
2	Aitchbone easily felt
3	Aitchbone felt with pressure
4	Not possible to feel aitchbone
5	Sow bulging with fat

1G. M. Hillyer (personal communication)

One justification offered for modest overfeeding of sows during gestation has been to provide a reserve of calories in the form of body fat to be utilized during lactation. Although this is possible, the energetic efficiency is not favorable. Bowland (1969) calculated the energy of cost of depositing fat in depots during pregnancy followed by mobilization of this fat during milk production. He concluded that feed calories are much more efficiently utilized to produce milk directly than to first produce fat which is subsequently used for milk production.

The sow's requirement for energy increases dramatically during lactation. Unless dietary sources are adequate there will be excessive weight loss by the sow. In the extreme case this can result in the so-called "thin sow syndrome" (MacLean, 1968). Sows in this condition are severely emaciated and anestrus. Hardy and Lodge (1969) reported a significant reduction in ovulation rate among sows which had experienced severe weight loss during lactation. The effect of moderate energy restriction during lactation may be less obvious in the short-term. O'Grady (1973) conducted a three-parity study wherein sows were fed either 12,200 kcal or 18,250 kcal of diet per day during a 42 day lactation. This is roughly equivalent to feeding eight or twelve pounds per day of a corn-soybean meal diet. In the first lactation milk yield, composition and pig

weaning weights were unaffected by treatment; however, by the end of the third lactation, milk yield had been reduced by 32% and litter weaning weights by as much as 21%.

Thus, free-choice, or near free-choice, feeding of sows during lactation is a sound recommendation. The feed allocation should be judiciously reduced in those instances where the sow is nursing a small litter. There has been concern that free-choice feeding during early lactation may precipitate the MMA syndrome. A recent experiment at the University of Kentucky compared free-choice feeding from farrowing to a scheme of limited intake during the first week of lactation. There was no difference in sow performance. Voluntary feed intake by the sow may be reduced by elevated farrowing house temperatures. The data in table 3 depict the effect of heat stress on feed intake by lactating sows.

Table 3. Effect of Environmental Temperature on the Sow and Litter[1]

	Temperature (F)			
	Experiment 1		Experiment 2	
Item	81	70	81	60
No. sows	20	20	16	16
Sow feed/day, lbs	10.1	11.4	9.2	12.3
Pig wt @ 28 days, lbs	13.6	15.4	14.1	16.1

[1]Lynch (1978).

Note that an 11 degree increase in temperature caused a 1.3 lb per day reduction in feed intake. But, more importantly the reduction in feed intake resulted in smaller weanling pigs.

Much recent attention has been given to the practice of adding fat to the diet of lactating sows to increase the caloric density of the feed. The information in table 4 represents a summary of some 31 experiments wherein fat was added to the sow's diet prior to farrowing and during lactation. From these data one can suggest that fat addition increases pig survival and to some extent

Table 4. Effect on Reproductive Performance of Fat Addition to the Sow Diet[1]

Item	Control	(No. litters)	+ Fat	(No. litters)
Pigs born alive/litter	10.0	(677)	9.9	(814)
Pigs Weaned/litter	8.1	(677)	8.4	(814)
Survival,%	82.0	(736)	84.6	(938)
Average birth weight, lbs	3.1	(677)	3.0	(814)
Average 21 d weight, lbs	12.25	(356)	12.45	(432)
Fat in colostrum, %	7.3	(360)	9.1	(512)
Fat in milk, %	9.1	(322)	10.1	(506)

[1]Moser and Lewis (1981).

the weight of pigs at weaning. Both field experience and research point to 7.5% added fat as a minimum required to elicit a beneficial response. Also, it appears important to start the fat addition at least a week prior to farrowing and continue the diet for a minimum for two weeks post-farrowing. The response is, admittedly, variable and the most value for fat addition appears to be in those herds with poor piglet survival.

A practical problem with utilization of fat is the "sticky" character which it gives to ground diets. This affects movement of feed through feeding systems and feed-down in self-feeders. Jensen at the University of Illinois has demonstrated that comparable lipid levels and positive responses can be achieved by use of oil seeds such as sunflower seed, soybeans and high-oil corn.

PROTEIN AND AMINO ACIDS

The present University of Illinois recommendations for protein and amino acid nutrition of the breeding female are shown in table 5.

Table 5. Protein and Amino Acid Recommendations for Gestating & Lactating Swine[1]

Item	Gestation, %	Gestation, g/day	Lactation, %	Lactation, g/day
Crude protein	12.0	216.	14.0	770.
Arginine	-0-	-0-	.34	18.7
Histidine	.13	2.7	.26	14.3
Isoleucine	.37	6.7	.39	21.4
Leucine	.40	7.6	.79	43.5
Lysine	.42	7.7	.60	33.0
Methionine	.28	5.0	.36	19.8
Phenylalanine	.37	6.7	.40	22.0
Threonine	.34	6.1	.51	23.7
Tryptophan	.08	1.5	.13	6.6
Valine	.46	8.3	.55	30.2

[1]Adapted from NRC (1979). Calculations based on 4 lbs/day feed intake during gestation and 12 lbs/day feed intake during lactation.

Note that the requirements are expressed both as a percentage concentration and as absolute quantities, i.e., grams per day of amino acid. It is convenient to use percentage values to express requirements and, in free-choice feeding programs, usually does not present a problem. In the case of the limit-fed, gravid sow it may lead to under formulation and an inadequate daily intake of a key nutrient. For example, if a diet is formulated to .42% lysine and the sow is fed five pounds of that diet daily, the daily lysine intake will be 9.5 grams but if feed intake is reduced to 3.5 pounds the daily lysine intake will only be 6.7 grams, an inadequate amount.

Adequate amino acid intake is essential during both gestation and lactation, but the consequences of a deficiency may not be immediately apparent. Swine nutritionists were shocked by data (Pond et al., 1968) which demonstrated that the sow is able to produce viable piglets even when fed a protein-free diet during gestation. This finding was solid evidence that the sow is capable of providing amino acids for the development of the fetal pig by catabolizing her own body protein. As one might expect, depletion of maternal tissue eventually results in reproductive failure by either the second or third parity (Mahan, 1977; Mahan, 1979). An inadequate diet not only affects the reproductive process but may also affect the potential for growth of the progeny. In the studies of amino acid deprivation during gestation, Pond and his associates found that, progeny of amino acid deprived sows had a reduced loin eye area and a reduced rate of weight gain post-weaning in comparison to pigs from sows fed adequately during gestation.

No one would propose to feed a protein-free diet under practical circumstances, but it may often be economically attractive to feed corn alone during gestation with only vitamin and mineral supplements. Corn has been shown to be deficient in both lysine and tryptophan for the gestating sow (Allee and Baker, 1970). The typical effect of feeding an all-corn diet during gestation is shown in table 6. Neither litter size nor piglet weights were affected at birth; but, sows fed the all-corn diet weaned smaller litters with lighter pigs than sows fed any other diet. The opaque-2 corn treatment is of interest due to the elevated concentration of lysine and tryptophan, the two amino acids most deficient in normal corn.

Table 6. Effect of Dietary Protein Quantity and Quality on Reproductive Performance and Progency Development1

		Opaque-2	Corn-soybean meal		
Criterion	All-corn	corn	12% CP	16% CP	20% CP
No gilts started	54	54	53	54	54
No gilts farrowing	41	39	38	34	39
No pigs born/litter	8.7	8.8	8.8	9.4	9.3
Live pigs born/litter	7.3	7.5	7.6	8.1	8.1
No pigs weaned/litter	6.2	6.9	6.9	7.5	7.3
Birth wt. per pig, lbs	2.73	2.77	2.75	2.68	2.75
Birth wt. live pigs, lbs	2.77	2.79	2.79	2.73	2.82
Litter wt. 21 days, lbs	61.68	75.43	75.72	80.91	75.41
Pig wt. at 21 days, lbs	10.07	10.49	10.69	11.19	10.49
Gestation gain, lbs2	52.32	72.22	80.36	88.02	80.69
Lactation gain, lbs3	9.81	-.63	-5.14	-9.633	-7.12

1Baker, et al. (1970)
2Gestation gain was: weight after farrowing - weight at breeding.
3Lactation gain was: weight after farrowing - weight at weaning.

In addition to the effects on sow performance during lactation, the all-corn diet resulted in a significant reduction in maternal weight gain during the pregnancy. The data fail to provide conclusive evidence that more than 12% crude protein is required to achieve maximum reproductive performance though there are trends for improvement with the higher levels. In subsequent work the

Illinois group found that an amino acid deficiency created by feeding an all-corn diet during early pregnancy could be overcome by feeding a diet containing an excess of amino acids during the final third of the gestation (Baker et al. 1970b). It was logical to ask if sows fed an adequate diet during the first two-thirds of the gestation would benefit from an excess of amino acids during the final third of pregnancy. Either a 12% crude protein diet was fed for the entire pregnancy or the 12% diet was fed for the first two-thirds of the pregnancy followed by a 16% diet for the final one-third. There was no as improvement in any criteria measured as a result of the extra protein fed during late gestation (Easter, 1980). Thus, when feeding a corn-soybean meal diet, it appears that 12% crude protein is adequate for the entire gestation.

Recent experiments in our laboratory provide evidence that the reduced weight gain and survival seen in piglets suckling sows deprived of amino acids during gestation is related to reduced milk production and, possibly, to reduced capacity to provide immuno-proteins to the piglet via colostrum. The results of an experiment conducted to study this phenomena are shown in table 7. In order to create specific deficiencies the diets were formulated with sorghum, a cereal grain that is deficient in both lysine and threonine for the gravid sow. To the all-sorghum diet, both lysine and threonine were added.

Table 7. Effect of Lysine and Threonine Supplementation of an All-sorghum Gestation Diet on Milk Yield and IgG Concentration in Sow Plasma1

Item	Sorghum-soybean meal	Sorghum	Sorghum + Threonine	Sorghum + Lysine	Sorghum + LYS + THR
Plasma IgG, mg/ml	15.85	12.26	14.43	12.08	14.27
Milk yield g/pig/d	890.	550.	670.	880.	790.

1Cuaron (1983)

In this experiment, the addition of lysine to the gestation diet resulted in a marked increase in milk production during lactation, a time when all sows were being fed a common, adequate diet. It may be that the deficiency of this essential amino acid during late gestation impairs the preparation of the mammary tissue for milk production. Thus, during lactation the potential for milk production is reduced and ultimately piglet weight at weaning is affected. The effect of the amino acid deficiency on the production of immuno-proteins is more difficult to evaluate. It is apparent that a deficiency results in a reduction of the circulating levels of IgG. It would be expected that the ultimate levels of IgG in piglet plasma would also be reduced. This has not been found to be the case (Cuaron et al., 1984). Consequently an experiment was conducted wherein pigs were farrowed into either a warm environment or a cold environment. The intent was to increase dependence of the piglets on colostral antibodies for protection against disease. The results are presented in table 8.

Pigs suckled in the cold environment acquired IgG at a slower rate than did those nursed in a warm environment. This phenomena has been previously reported by French scientists (Le Dividich and Noblet, 1981.) The acquisition rate was futher reduced when the pigs were suckling sows that had been fed a diet inadequate in amino acids during the gestation. The environmental treatments were continued until the pigs were weaned at day 28 of lactation. Piglet survival tended to reflect the effects of the early post-weaning acquisition of colostral antibodies, despite the fact that the ultimate levels achieved did not differ among treatment groups. These data are preliminary but substantiate the need for adequate amino acid nutrition during gestation.

Table 8. Effect of Lysine and Threonine Supplementation of Sorghum Diets on IgG Acquisition and Piglet Survival1

Environment	Time after birth	Sorghum soybean meal	Sorghum	Sorghum + threonine	Sorghum + lysine	Sorghum + Lys + Thr
		IgG, mg/ml				
warm	Ø hrs	-Ø-	-Ø-	-Ø-	-Ø-	-Ø-
	3 hrs	9.62	8.36	8.9Ø	9.ØØ	9.Ø4
	6 hrs	13.98	13.Ø9	13.Ø7	13.27	13.61
cold	Ø hrs	-Ø-	-Ø-	-Ø-	-Ø-	-Ø-
	3 hrs	7.22	5.36	4.74	7.Ø1	7.99
	6 hrs	12.74	1Ø.84	1Ø.63	11.48	12.69
		Survival, number of pigs alive				
warm	Birth	9.5Ø	1Ø.75	9.5Ø	9.5Ø	11.25
	Day-14	8.5Ø	9.ØØ	7.5Ø	8.25	9.25
	Day 28	8.25	8.75	7.5Ø	8.25	9.25
cold	Birth	11.25	11.25	1Ø.5Ø	9.25	9.75
	Day-14	9.ØØ	6.5Ø	3.75	7.ØØ	8.ØØ
	Day-28	9.ØØ	6.25	3.75	7.ØØ	7.75

1Cuaron (1983)

The requirements for individual amino acids for lactating sows have been exhaustively investigated in a series of experiments conducted over the past 15 years at Iowa State University, c.f., Lewis and Speer (1974). The most immediate consequence of a deficiency is a depression in milk production with reduced piglet weaning weights following.
There are also negative effects on the milk quality and nitrogen status of the sow.

Attention has been given to the problem of defining the appropriate crude protein level in corn-soybean meal diets for optimal reproductive performance. A large expermiment involving both first and second parity sows was conducted in our laboratory a few years ago with levels of crude protein of 12%, 14%, 16% and 18%. In the case of second-litter sows there was no apparent advantage of feeding more than 12% crude protein during the four-week lactation. There was, however, a definite improvement in piglet weaning weights when the level of crude-protein in gilt diets was increased from 12% to 14%. There was no additional improvement in any criteria due to higher levels of crude protein. The results of the experiment with first-litter gilts are shown in table 9.

Table 9. Effect of Dietary Protein Level in Lactation on Reproductive Performance of First-litter Giltsl

Criterion	12% CP	14% CP	16% CP	18% CP
Number of gilts	50	48	46	50
Total pigs farrowed	10.2	9.6	9.9	9.8
Live pigs farrowed	9.0	8.7	8.4	8.6
Pigs weaned	7.8	7.2	7.8	7.7
Litter wt at birth, lbs	25.74	26.95	26.26	26.62
Pig wt. at 28 days, lbs	13.28	14.10	14.54	14.14
Lactation wt loss, lbs	21.86	34.78	29.24	30.14
Lactation feed consumption, lbs	237.3	242.8	242.7	243.1

lEaster (1980)

MINERALS

The corn-soybean meal diet should be supplemented with salt and calcium,. phosphorus, iron, zinc, iodine, selenium and manganese. The actual requirements for calcium and phosphorus has received much research attention over the years and is still a subject of controversy. The University of Illinois calcium recommendation for gestation and lactation is .75% and .75%, respectively, with the caveat that the level should not exceed 1.0% of the diet. These concentration values are based on the assumption that the sow will be fed 4 to 5 pounds per day of a corn-soybean meal diet during gestation and will be permitted to eat free-choice during lactation. The phosphorus recommendations are .50% for gestation and .50% for lactation. These recommendations are based on thorough evaluation of the available literature and are considered adequate.

Since the publication of the National Research Council recommendations in 1979, the recommendations for calcium and phosphorus for breeding swine have been roundly debated. A recent series of experiments conducted at Virginia Polytechnic Institute and State University by Dr. E. T. Kornegay and associates has done much to give validity to those values (Kornegay and Kite, 1983).

Iron supplementation is crucial to the well-being of suckling piglets. But, recent information supports the hypothesis that excess iron may increase the susceptiblity of young pigs to bacterial disease (Knight et al., 1983). In the body the iron is tightly bound to proteins as a means of limiting the supply of this essential nutrient to bacteria. Without iron, bacterial growth may be reduced. From a practical perspective excess iron, beyond the capacity of the proteins to sequester it, may increase that quantity available to pathogens. Hopefully, on-going research will define the maximum upper dose.

VITAMINS

The occurrence of a frank vitamin deficiency in swine is probably rare and likely results from improper diet mixing or loss of vitamin potency due to improper storage. The vitamins which should be added to a corn-soybean diet for breeding swine are: vitamin A, vitamin D, vitamin E, vitamin K, riboflavin, niacin, vitamin B-12, pantothenic acid and choline. Nutritionists are not in

agreement with regard to the necessity of either biotin or vitamin C addition to sow diets. Research conducted at the US Meat Animal Research Center has failed to demonstrate any beneficial effects of vitamin C addition to diets (Yen and Pond, 1983). Bryant et al. (1983) reported some tendency for improvement in reproductive performance with biotin addition to the diet, however, the the effect was not significant (P >.10). Hamilton and Veum (1984) found an increase in number of pigs weaned in only one parity of a five parity study where biotin supplemented and nonsupplemented diets were compared.

LITERATURE CITED

Allee, G. L. and D. H. Baker. 1970. Limiting nitrogenous factors in corn protein for adult female swine. J. Anim. Sci. 30:748.

Arthur, S. R., E. T. Kornegay, H. R. Thomas, H. P. Veit, D. R. Notter, K. E. Webb, Jr., and J. L. Baker. 1983. Restricted energy intake and elevated calcium and phosphorus intake for gilts during growth. IV. Characterization of metacarpal, metatarsal, femur, humerus and turbinate bones of sows during three parities. J. Anim. Sci. 57:1200.

Baker, D. H., D. E. Becker, H. W. Norton, C. E. Sassee, A. H. Jensen and B. G. Harmon. Reproductive performance and progeny development in swine as influenced by feed intake during pregnancy. J. Nutr. 97:489.

Baker, D. H., D. E. Becker, A. H. Jensen and B. G. Harmon. 1970. Protein source and level for pregnant gilts, a comparison of corn, opaque-2 corn and corn-soybean meal diets. J. Anim. Sci. 30:364.

Baker, D. H., B. A. Molitoris, A. H. Jensen and B. G. Harmon. 1974. Sequence of protein feeding and value of alfalfa meal and fish meal for pregnant gilts and sows. J. Anim. Sci. 38:325.

Bowland, J. R. 1977. Energetic efficiency of the sow. J. Anim. Sci. 26:533.

Bryant, K. L., E. T. Kornegay, J. W. Knight, H. P. Viet and D. R. Notter. 1983. Influence of supplemental biotin on reproductive performance and various biochemical criteria in gilts and sows fed corn and wheat based diets. J. Anim. Sci. 57(Suppl. 1)237.

Cuaron, J. A. 1983. Lysine and threonine supplementation of sorghum gestation diets for swine. Ph.D. Thesis, University of Illinois.

Cuaron, J. A. 1984. Effect of lysine and threonine supplementation of sorghum gestation diets on nitrogen balance and plasma constituents in first-litter gilts. J. Anim. Sci. 58:631.

Cunningham, P. J., C. H. Naber, D. R. Zimmerman and E. R. Peo, Jr. 1974. Influence of nutritional regime on age at puberty in gilts. J. Anim. Sci. 39:63.

Easter, R. A. 1980. Amino acids in swine nutrition. Feedstuffs: 52 (26) 20.

Friend, D. W. 1977. Effect of dietary energy and protein on age and weight at puberty in Yorkshire gilts. J. Anim. Sci. 44:601.

Frobish, L. T., N. C. steele and R. J. Davey. 1973. Long term effect of energy intake on reproductive performance of swine. J. Anim. Sci. 36:293.

Hardy, B. and G. A. Lodge. 1969. The effect of body condition on ovulation rate in the sow. Anim. Prod. 11:505.

Hamilton, C. R., and T. L. Veum. 1984. Response of sows and litters to added dietary biotin in environmentally regulated facilities. J. Anim. Sci. 59:151.

Jensen, A. H., B. G. Harmon, G. R. Carlisle and A. J. Muehling. 1972. Management and housing for confinement swine production. Univ. of Ill. Coop. Ext. Serv. Circ. 1064.

Kirkpatrick, R. L., B. E. Howland, N. J. First and L. E. Casida. 1967. Ovarian and pituitary gland changes in gilts on two nutrient energy levels. J. Anim. Sci. 26:358.

Kornegay, E. T. and B. Kite. 1983. Phosphorus in swine. VI. Utilization of nitrogen calcium and phosphorus and reproductive performance of gravid gilts fed two dietary phosphorus levels for five parities. J. Anim. Sci. 57:1463

Le Dividich, J. and J. Noblet. 1981. Energy metablosim of the newborn pig during the first 24h of life. Biol. Neonate. 40:175.

Lewis, A. J. and V. C. Speer. 1974. Tryptophan requirement of the lactating sow. J. Anim. Sci. 31:778.

Lynch, P. B. 1978. Environment and the breeding sow. Moorepark Pig Farming Conference. Dublin, Ireland. An Foras Taluntais.

Mahan, D. C. 1977. Effect of feeding various gestation and lactation protein sequemces on long term reproductive performance in swine. J. Anim. Sci. 45:1061.

Mahan, D. C. 1979. Effect of dietary protein sequences on long term sow reproductive performance. J. Anim. Sci. 49:514.

Moser, B. D. and A. J. Lewis. 1981. Fat additions to sow diets - A review. Pig News and Information. 2:265.

MacLean, C. W. 1968. The thin sow problem. Vet. Rec. 83:308.

NRC. 1979. Nutrient Requirements of Domestic Animals, No. 2. Nutrient Requirememts of Swine. Eighth Revised Ed. National Academy of Sciences - National Research Council, Washington, DC.

O'Grady, J. F., F. W. H. Elsley, R. M. MacPherson and I. McDonald. 1973. The response of lactating sows and their litters to different dietary energy allowances. Anim. Prod. 17:65

Pond, W. G., W. C. Wagner, J. A. Dunn and E. F. Walker. 1968. Reproduction and early postnatal growth of progeny in swine fed a protein-free diet during gestation. J. Nutr. 94:309.

Robertson, G. L., L. E. Casida, and A. B. Chapman. 1951. Age at puberty and related phenomena in Chester White and Poland China gilts. J. Anim. Sci. 10:647.

Toplis, P., M. F. J. Ginesi and A. E. Wrathall. 1983. The influence of high food levels in early pregnancy on embryo survival in multiparious sows. Anim. Prod. 37:45.

Vermedahl, L. D., R. J. Meade, H. E. Hanke and J. W. Rust. 1969. Effects of energy intake of the dam on reproductive performance, development of offspring and carcass characteristics. J. Anim. Sci. 28:465.

Yen, J. T., and W. G. Pond. 1983. Response of swine to periparturient vitamin C supplementation. J. Anim. Sci. 56:621.

Zimmerman, D. R., H. G. Spies, H. L. Self and L. E. Casida. 1960. Ovulation rate in swine as affected by increased energy intake just prior to ovulation. J. Anim. Sci. 19:295.

Principles to Practice–The Management Challenge

A. H. JENSEN

The multi-faceted nature of a swine production unit provides many decision-making challenges to the management. Decisions relative to routine matters are usually rather straightforward. But a decision to introduce new, or to significantly modify ongoing, practices as a result of recently acquired information should be given careful consideration. This new information needs to be evaluated in terms of its (1) relative advantage - what positive effects would result, (2) compatibility - will it fit into the overall program, (3) complexity - is the principle(s) fully understood and can it be effectively used, (4) trialability - can its effects be identified by a small-scale application, and (5) observability - will effects be measurable or evident.[a]

One of the more obvious examples of putting "principle to practice" was the program initiated to change from fat-type to lean-type hogs. The motivation to change was generated by such factors as consumer negative reaction to fat pork, competition of lard with vegetable oils, and economic outlook for the producer. The "principle" -- backfat thickness was a good indicator of the amount of fat in a carcass -- was put into "practice" with the use of the backfat probe as a tool to aid in selecting breeding stock. In adopting this practice there was (1) relative advantage - leaner carcasses would provide leaner pork for the consumer which should stimulate consumption and enhance profit for the producer, (2) compatibility - the use of the backfat probe was easily incorporated into selection and sorting of animals, (3) complexity - there was no problem in relating backfat thickness to fat content of carcass, (4) trialability - probing quickly identified overly fat hogs and (5) observability - changes in type were evident in the offspring of the selected breeding animals.

Change in type of hog produced was a dramatic effect from "principle to practice". But what about reproductive efficiency? Statistics show that the average number of pigs weaned per litter has hovered around seven for at least the last quarter-century. This implies an average death loss of 20 to 30% of live-born piglets. Yet, individual units have nine or more pigs weaned per litter. Why the difference? Perhaps it is due, in part at least, to the degrees to which "principles have been put into practice", the appropriate application in the production program of those principles, generated and confirmed by research, which

[a]Adapted from Rogers, E. M. 1983. Diffusion of innovations. 3rd Ed., Macmillan Publishing Company, Inc., New York.

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could reasonably be expected to improve reproductive efficiency. What new, or not previously practiced, ideas or information or factors affecting breeding efficiency, embryo survival, environment, animal management and nutritional adequacy as discussed in this conference, could be put into practice to improve reproductive efficiency?

Putting some of these principles into appropriate practice at the swine production unit should enhance biological efficiency. And a likely benefit would be in number of pigs weaned per litter. Thus, if adopted statewide, these improved practices would result in fewer sows needed to produce a given number of pigs. For example, if eight pigs, instead of seven, were weaned per litter, 178,571 fewer litters would be required for the annual production of ten million pigs. This would mean a savings of 71,240 tons of sow feed. On an individual herd basis, if a well-managed unit produces 6,000 pigs annually and currently weans eight pigs per litter, increasing to 8.75 pigs per litter would mean 64 fewer litters needed per year. This would save about 26 tons of sow feed, and other costs of production would also be favorably affected. This would certainly testify to positive benefits from putting "principles into practice."

Since profit is the intent for each production unit, any modification of or addition to a management program that would increase profit potential would be highly motivating. What, then, are some of the reasons for not adopting new information? Responses frequently include: the research wasn't carried out under farm conditions - not enough time - not enough labor - don't have needed facilities - none of my neighbors are doing it - don't believe it applies to me - I'm not doing as good now as I know how!

Results of extensive studies indicate that those who are alert to new ideas, do not resent change, use records effectively in decision-making, and can conceptualize the possible impact of adopting new information are the first to put new "principles to practice". These "innovators" may evaluate, decide and implement within a few months. Others may follow suit only after several years. But it should be cautioned that acceptance and early adoption of information based on ill-conceived experimentation and unproven merit would probably be only an expensive experience.

Acceptance and speed of adoption of new principles by individual swine producers will be significantly affected by the producer's perceived merits of the information and the impact of its adoption. These will often reflect the effectiveness with which the information is communicated. It thus behooves the provider of the information to communicate understandingly, honestly and convincingly.

Making good decisions is a continual challenge to management. And sources of information are abundant - university researchers and extension specialists, special conferences (Pork Industry Conferences), agricultural experiment station publications, producer and commercial organizations, professional society meetings and publications, and a wide variety of popular press magazines. Certainly a prerequisite for good "principle-to-practice" decisions is to evaluate carefully, sort out the valid from the invalid, the objectively determined from the subjectively stated and the fact from the fad.

Effective "principle-to-practice" results from successful interplay between the provider and the adopter of new information. The appropriate information must be presented and understood, there must be persuasion that it is valid, it

must be adequate to make a sound decision, it must be suited to effective introduction into the production program and its adoption portends positive results.

Support Your Land Grant University

JOHN HUFTALIN

Whenever one is asked by a friend for a favor, it is so easy to say "Sure, I can handle it. Be glad to do it." Soon thereafter the sweat period begins, and you realize you have just committed yourself to an assignment which not only is challenging, but downright scary.

I am proud to be here as a spokesman for agriculture, however. You may think because of my close ties to the College of Agriculture, the Alumni Association and the University that I'm speaking for them. You're right! But do you know what? I'm speaking for you in a louder voice, with even more interest and concern because I am first, and foremost, a farmer and pork producer. In addition, I have two young sons in the operation at home and another who is a sophomore in agricultural economics here at Illinois. It is their future, and your son's and daughter's futures, that we're talking about.

My grandad, like most grandads, was a wise man. I learned much from him, and I'm glad he taught me that if you are going to dance, you are going to have to pay the fiddler.

I have a friend in Virginia, formerly with the USDA during the days of the beef boycott. One evening, while he was addressing a group of housewives in New York City, one of the women stood up, and in a loud voice announced that she did not need farmers because she got her food at the supermarket.

You ask what does paying the fiddler and the beef boycott have to do with what we're about? It's like the bumper sticker we've all seen which says, "Don't complain about the cost of food with your mouth full."

Several years ago my wife and I, along with 36 other pork producers and friends from Illinois, spent three weeks in Europe, including visits in Russia, Romania, and Hungary, along with three non-communist countries. That experience allows me to honestly say we should not take for granted what we have in this country.

For many years we have all sat in the shade of trees that someone else planted. Many of us got our start in business through 4-H Club work. I can remember how excited I was when my grandad gave me a Poland China gilt to launch me into the hog business.

John Huftalin, Pork Producer and President, University of Illinois College of Agriculture Alumni Association.

We progressed through 4-H, FFA, rural youth, went on to college, farming, business or whatever we chose. We didn't think too much about who set up the programs or why we used certain methods. We didn't question the value of clean pastures for hogs, or the value of the corn-soybean ration or a host of ideas we used in our production units. If we had a question, we called the farm adviser. If our pigs were sick we called the veterinarian. If our corn blew over because the root system had been eaten away, we called Don Kuhlman or before him Steve Moore and Pete Petty .

When someone started saying animals had rights,we said, "Where's Stan Curtis?"

We had trouble setting up good financial records and suddenly Tom Frey was there.

We needed help in setting up and conducting barrow shows and progeny testing and there stood Gilbert Hollis and Leif Thompson and before them Dick Carlisle and Harry Russell.

What about Lowell Hill's efforts in international grain trade--that's part of our market too.

Even when the burden of stress seems overwhelming, Jerry Robinson is prepared to come in and get us on the right track.

Finally, let's consider the director of our research station Don Holt and our great new dean, John Campbell.

What's the point behind all the name dropping? It's very simple. These people were all trained at our land-grant universities across this country, and for the most part, our citizens have enjoyed and benefitted from this without realizing or appreciating how darn good they have had it.

As Paul Harvey would say, "And now here is the rest of the story."

We're in a new ball game. Some of the players have changed. Some of the rules have been changed. Even the centerfield fence has been moved back and it's harder to hit a home run. Remember, if you will, how we used to play ball on Sunday afternoon in the cow pasture. Some of those grounders were pretty hard to handle--as was running down a fly ball with one eye in the air and one eye on the ground.

Now the things now we can't afford to step into are drug residues, animal rights, over-production, poor marketing, high input costs, red meat scares, competition from fish and poultry, vegetarianism and complacency.

Let me ask you, "Who's going to pay for research help and assistance to avoid these spots in the cow pasture? Who's going to benefit from all of this?" The answer's the same. We all are, and we'd better get the idea across real soon to the other 97 percent of the American public.

We need to tell them about the Hatch Act of 1887 enacted to fund agricultural experiment stations in each state throughout the United States, and how each year the funds represent a smaller percent of the federal budget.

The general public and the private sector are missing tremendous opportunities by not investing more heavily in agricultural research, in development, and in education activities. The annual return to investments in agricultural research ranges from 10 to more than 200 percent, and averages between 30 and 60 percent. The marginal product of agriculture research and development is estimated to be more than $10 for the last dollar invested in research on commodities such as corn, soybeans, and wheat.

Again, a bumper sticker: "Farming is everbody's business." In agriculture we tend to think farming is only our business. But, that is not so. We used to think all we had to do was produce and somone else would dispose of our produce. The law of supply and demand is still valid and will always be important in a free society. However, we have a new kid in town, and his name is "the world market."

There are farmers around the world who can produce pork, ship it to our country, and still make a profit. (Witness these past few months with our neighbors to the north.) We all make funny sounds when imports come to our markets and hold our prices down. We've even heard other industries such as the auto, steel, and textile people express displeasure about imports.

Now don't start throwing things because I am on your side. I'm only pointing out the fact of the world market. Like it or not, that's where we'll be selling our products. I don't believe we can afford to build fences around our markets because other folks might build higher and stronger ones than we would.

You ask, "How do we cope with bearish pig crop reports, drought, low prices, high interest,and disease?" The list goes on. I don't have all the answers, but certainly I'm convinced we're not going to solve the problem by ourselves or by turning our heads the other way. If we are to going to gain the competitive edge we'll have to use every tool in our tool box, including the research we gather at our land-grant universities.

It was 122 years ago that the land-grant universities were signed into being by President Lincoln. At that time no individual had the money or facilities for conducting meaningful research on his own. The same holds true today and the need for unbiased information is just as accute as it was then. But now there are increased demands on the state revenues that account for half the University of Illinois' total budget. The balance has to come from federal and private sources.

The reason for private gift support relates to the issue of maintaining quality--the quality of graduates, the quality of the faculty and the curriculum, the quality of research, and the quality of the institution's services to the public.

We must help John Q. Public understand that the tremendous quality and quantity of food in this country is no accident. From time to time we complain about the FDA. We think they're too stringent and take too long to make their decisions. But then, isn't it fortunate the consumer can go into the supermarket, buy what he or she wants, and be assured of its safety and wholesomeness? This makes our marketing job a whole lot easier.

Let me suggest a couple of ideas for you to help understand the dilemma we're facing. As you know about 97 percent of the people of this country are non-farmers. This means 97 percent of the voters are nonfarmers. Sometime when your wife goes grocery shopping, go with her. Check out the meat counter for quality and quantity. If we are to gain the understanding of the consumer so they will support our research programs, then we need to see things from their side of the counter.

Get to know your elected legislators whether you voted for them or not. Any way you cut it the end is the same, as these are the people who write and pass the laws and grant the money for research. If you think we have too much research now, you may never attain the competitive edge you'll need to succeed in the future.

Another very important consideration is your decision to attend this conference and others like it. These sessions strengthen not only the students, which we are, but also encourage our researchers to probe for even greater findings.

Speaking of students, in this country we are experiencing a steep decline in the numbers of high school graduates available for college. That means there are fewer to choose from to fill our classrooms, conduct our research, and fill all those position in agri-business that require a sound, well-rounded degree in agriculture. We all need to spend some time with the ag teachers, counselors and extension workers in our respective counties, encouraging them to send their most promising students to the U of I. We can ill afford to let this important task go undone.

Many of you are graduates of the College of Agriculture. I hope you are also members of your Alumni Association. I could speak for a week on what an important part you have played in the support of this campus. Look around. The new Veterinary Medicine complex, the Agricultural Engineering Sciences Building, and the soon-to-be Animal Sciences building are examples of what we can do when we go about it right. Any of you who helped with the Food For Century III project can be proud of your efforts. This is a prime example of why you need a strong commodity organization and a good relationship with your legislators.

Another fact worth noting is that this very night we will be awarding seventy-three $2,500 scholarships to incoming freshmen on a merit basis. No other college of agriculture in the country is coming close to this effort in the pursuit of excellence. Dean Campbell started this program and many of you have enhanced it with your gifts to the College of Agriculture.

These are a few of the commitments we have made--collectively. We are proud of our past, we are concerned about our future.

The buzz word of the times is "the competitive edge." It can only be obtained through the pursuit of excellence. There are tough times ahead and there will be casualties. I firmly believe only the strong shall survive, and we all know what it takes to be strong.

Your land-grant university recognizes and appreciates your support in the past, and will welcome it in the future.

Integrated Control of Reproduction

PHILIP J. DZIUK

Fifty years ago chickens were produced in groups of 10 or 15 as they were incubated, hatched and mothered by a broody setting hen. The hatches were scattered over several weeks until the poultry producer had accumulated as many chicks as he wanted-or was going to obtain. The artificial incubator and brooder has changed that, so at the present the poultry producer can order a certain number of chickens delivered on a certain calendar date during normal working hours. The number of eggs that a hen will lay can be predicted with some confidence.

The timing of events associated with reproduction in pigs occurs more or less at random. This randomness does not permit scheduling or appointment of activities but necessitates completion of tasks on a demand basis, something similar to the chicken enterprise of 50 years ago. To say the least, this is no way to run a modern business. With effort and persistence some of the promise of integrated control of reproduction in pigs may be realized. Such control would include, but not be limited to, appointment of the date and hour for insemination of a group of gilts or sows and scheduling of farrowings during usual daylight working hours. Over the period of the past 20 years, there has been a number of materials and procedures tested for efficacy, cost, and convenience and safety to accomplish control. Thus far most have not reached the producer because of one or more of a combination of factors. What are the possibilities of control of reproduction and what might we strive to attain? There are many more possibilities than there are realities. Induction of parturition by prostaglandin is a reality, the use of prostaglandin or a derivative to induce parturition is now approved and reasonably effective. Administration of prostaglandin at about 112 days of gestation will cause the corpora lutea to cease functioning and parturition will occur in about 24 to 30 hours. When the prostaglandin is given in the morning most farrowings will occur during the next day during daylight hours. There are studies underway to help make the control even more certain and precise. To avoid the problems arising from the sows that farrow earlier than day 113 and to delay parturition, a number of materials are being tested that will hold parturition in abeyance until it is wanted. On the other hand, tests are underway for procedures to induce parturition at very precise times. Supervision of farrowing could then be convenient and feasible.

The control of the time of ovulation is essential for timed inseminations and would facilitate timed farrowings. If all sows in a group had been mated on the

Philip Dziuk, Professor, Department of Animal Science, University of Illinois.

same day, treatments to control parturition could be applied at the same time 112 days later. Procedures and materials for control of ovulation have yet to be approved in the United States. Materials are approved and used in eastern Europe and in some western European countries. The biology has been studied well enough to know which procedures are effective but safety must still be established and approval for use obtained. If and when materials are approved, the way will be opened for the timing aspect of planned reproductive management.

Pregnancy detection at relatively early stages of gestation is now possible by a number of ultransonic devices. By measuring hormones in the blood of the gilt between days 20 and 30 after breeding, it is possible to not only determine pregnancy with 100% accuracy but also to estimate potential litter size. A non-pregnant animal or one with a small litter could then be detected and appropriate action taken. Because the pig embryo signals the mother of her pregnancy as early as 12 days after mating it may be possible in the future for the manager to interpret that signal and make decisions based on that interpretation.

Animals are selected for the breeding herd for one purpose - to reproduce. If they fail to reproduce or do so at a very low level, the genes that they are expected to pass on to the next generation are lost. Presently there are no available methods for accurately estimating the potential fertility of a replacement gilt or boar. The ability to predict the potential litter size of a gilt before she is selected from a larger group would be an integral part of controlled reproduction. On the basis of limited experimental evidence it appears that gilts will ovulate greater number of eggs after puberty are also more sensitive prepuberally to hormones that cause ovulation than other gilts ovulating fewer eggs. By applying this test to all gilts and selecting for those with a greater ovulation rate, increases in average litter size may be realized.

A goal to strive for would include artificial insemination of all gilts on an appointed hour and date, who would in turn all conceive and produce large litters with pigs of uniform size. Because the boar has a significant influence on conception rate and litter size, the potential fertility of this boar should be determined accurately. Presently available methods are relatively ineffective in distinguishing between males of high, medium or low fertility. If the boar is nearly infertile, microscopic or chemical examination may in some cases be helpful in detecting this low fertility. Methods employing test-tube fertilization appear to be much more useful and accurate in predicting fertility of a male.

Because control of reproduction is a relatively new aspect of biology, the necessary information for complete control is just beginning to be accumulated. As more information is gathered even greater control may be exerted to the extent that several hundred full brothers and sisters could be produced on one day by ovulation control, embryo transfer and induced parturition. Before we are willing to accept the situation as is, we should ponder on the possible fate of a poultry producer with only setting hens to incubate his future flock.

Control of Reproduction Now and in the Future

STEPHEN K. WEBEL

INTRODUCTION

Pork production differs greatly today from ten or twenty years ago and it will also differ in the future. Operation of pork production factories will become even more highly organized and management activities more precisely scheduled with the application of currently available technology and new products being developed. The day or even the precise hour of breeding and farrowing will be scheduled for individual animals, perhaps even months in advance.

Although many facets of production will see vast changes, this presentation is limited to control of the reproductive process and discussion of products for regulation of breeding and farrowing. There is, of course, much research directed at developing anabolic or growth promoting products as well as products for control or treatment of disease and improved nutritional products. However, each of these could be topics for extensive presentations and will not be discussed further.

This presentation will focus on two specific areas of production and the products to be used with management systems for control of reproduction in gilts and sows. "To be used with management systems" is an extremely important concept because these products are useful only in conjunction with excellent management. From my experience, few of the tools for controlling reproduction have been successful unless the management and people involved were highly competent.

The first area we will discuss involves the control of the estrous cycle or control of the time of breeding. The second subject area deals with control of the time of farrowing. We will review how these functions are naturally regulated, then discuss how compounds or products can be incorporated into management programs to schedule the time for these important events in a swine operation. Control of the time for breeding and subsequently farrowing sets the stage for other events to fall into place and facilitates management of routine activities. If the breeding and farrowing phases of production are well organized and managed, then other phases of an operation become easier to manage. For example, scheduling of labor for moving pigs, processing newborn litters, washing pens, and weaning pigs becomes much easier.

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REVIEW OF THE REPRODUCTIVE CYCLE

The reproductive cycle of the sow requires approximately 140 days. This includes 5 days to develop follicles and come into estrus, 114 days for gestation and 21 days for lactation. This translates into approximately 2.5 litters per sow per year. To better understand how and why products which regulate reproduction work, let's briefly review the normal events of the sows reproductive cycle.

The follicular phase is approximately five days in length and is the period of follicular development just before estrus. Two gonadotophic hormones, follicle stimulating hormone (FSH) and luteinizing hormone (LH) are secreted by the pitituitary gland. These hormones promote follicle growth and ovulation. The growing follicle secretes estrogen which stimulates sexual behavior or estrus, and prepares the reproductive tract for breeding. Near the onset of estrus a surge of LH is released which stimulates the follicle to reach maturity and release the egg into the reproductive tract for fertilization. The timing of these events and amounts of hormone must be precise, or the entire process will be disrupted. Indiscriminate injection of hormones may do more harm than good. However, years of basic research have provided much of the information needed to develop products to control this intricate system.

Following ovulation the eggs are fertilized if sufficient sperm is deposited into the reproductive tract at the proper time. The hormonal environment then changes and becomes dominated by progesterone which is secreted by the corpus luteum (CL). This is the luteal phase of the estrous cycle and lasts for 14 to 15 days, in the non-pregnant sow, before a new follicular phase begins. If the sow is pregnant progesterone continues to be produced during pregnancy. Continous production of progesterone is necessary for the sow to maintain pregnancy and prevents the sow from coming into estrus. Progestone has been produced synthetically and forms the basis for artificial control of estrus, which will be discussed in greater detail in the next section of this presentation.

The events that lead up to, and signal farrowing to begin, are also very complicated and not totally understood. However, it is known that the fetus sends a signal to the mother which causes the release of prostaglandin. The CL regresses or is killed by prostaglandin and progesterone production is stopped. Following the decline in blood progesterone the farrowing process can be initiated. Although this greatly over simplifes the physiology, it illustrates how researchers have determined what compounds are useful for artifical control of reproduction. For example, prostaglandins are widely used to induce and control the time of parturition. In addition, it has been known for many years that oxytocin will stimulate uterine contractions and lead to expulsion to the fetus.

Estrus does not occur during lactation even though progesterone levels are low. There apparently is a different mechanism involved for estrus suppression than during the luteal phase or pregnancy. Although the mechanism is not understood, it is well established that the reproductive cycle is restarted and repeated in the same cyclic manner after piglets are weaned.

An understanding of the basic physiology of the reproductive cycle has provided the information necessary for scientists and pharmaceutical companies to develop products for the swine producer to use in managing his operation.

We will next explore how the hormones, such as progesterone, FSH, LH and prostaglandin are being used to regulate the reproductive cycle of the sow.

CONTROL OF THE TIME OF BREEDING

The estrous cycle of the gilt or sow frequently does not fit well into our modern production practices. With a 21 day estrous cycle, the day of estrus frequently does not coincide with the desired time for breeding. Sows are easier to schedule than gilts, because they generally return to estrus 4 to 6 days following weaning. However, this regularity of estrus is frequently absent in some of the sow lines in use today. Heavy milking sows may not return to estrus promptly after weaning. This appears to be a greater problem after the first litter and during certain times of the year, especially late summer when the temperatures are elevated and day length is decreasing. Breeding gilts has long been and continues to be a problem and frustration, not only for introducing gilts into the herd in synchrony with sows, but also due to delays in attaining puberty and the difficulty of estrus detection in closely confined environments. Products to control estrus in the gilt and sow are of two basic types, either suppressive or stimulatory in nature. Each of these types will be discussed briefly. More detailed reviews have previously been written and provide further background information (Webel,1979; Webel and Day, 1982).

Products for Regulation of Estrus Following Suppression of the Cycle

Progesterone or synthetic progestins are the most widely used class of compounds for suppression of the estrous cycle. Progestins prevent the sow from developing follicles or coming into estrus, just as if she were in the luteal phase of the cycle or pregnant. This type of hormone suppresses estrus during administration, then following withdrawal the sow comes into heat at a predictable time. These compounds are very useful if the gilt or sow is cycling, but are not effective in the anestrous or non-cycling animal. Although numerous compounds have been tested during the past 15 or 20 years there appears to be only one potential product currently under development. This is a compound known as altrenogest. This product is a synthetic progesterone like compound. Numerous trials have been conducted in the U.S. and internationally. In fact, it is available commercially to the swine producer in France. The standard procedure for using altrenogest is to mix the compound into the feed and administer it for 14 or 18 days. Estrus is suppresed during feeding, then following withdrawal of the product the normal events of the estrus cycle are allowed to proceed and the sow comes into estrus on the 5th or 6th day. The product is very effective for regulating and scheduling estrus in gilts to coincide with recently weaned sows. Altrenogest is also useful for synchronizing or regrouping sows. The previous estrous record is not needed to use the product because feeding can be initiated during any part of the estrous cycle. To synchronize estrus simply begin feeding altrenogest the appropriate number of days before the date selected for breeding.

Trials have been conducted for over ten years, with more than 8,000 animals being treated. No reductions in fertility or litter size have been observed. On individual farms conception rates and litter size similar to pretreatment or control groups are generally observed. Whether a producer is obtaining 95 % or 50% conception before synchronization, the same results

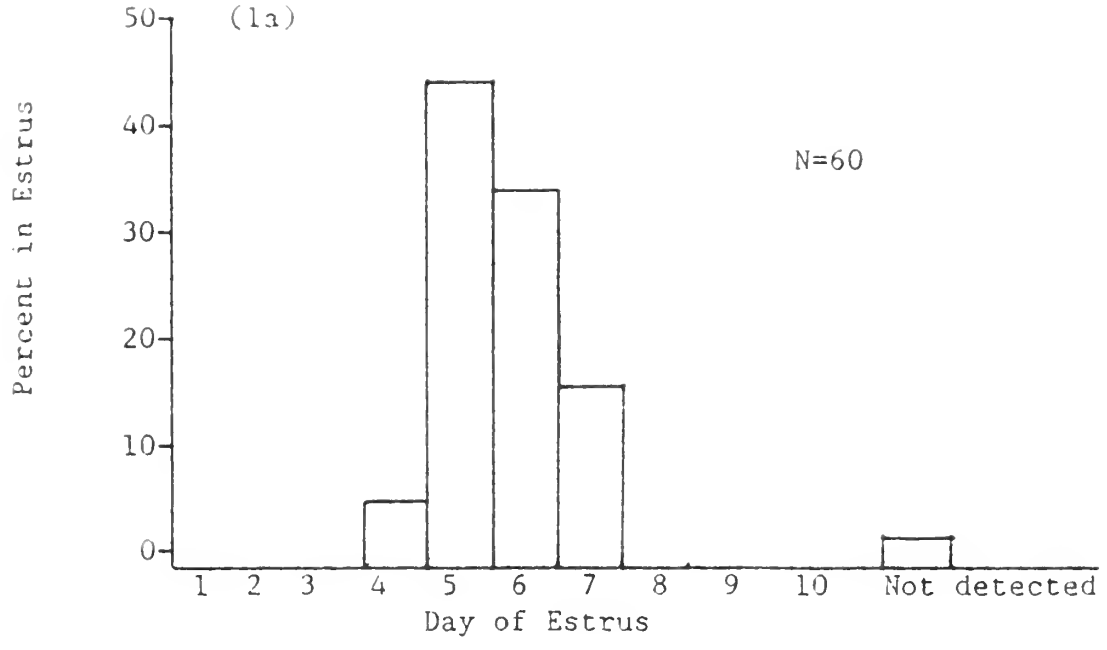
should be expected following synchronization. There does appear to be some increase in litter size. In large scale farm trials, approximately a one half pig increase in average litter size was observed. However, the primary application for the product is regulation of estrus in the gilt or sow.

On most swine farms it is not known for certain whether gilts are cycling or when their previous estrus occurred. The percent of gilts in estrus each day following altrenogest is presented in figure 1 (on page 5). Figure 1a illustrates estrous synchronization for gilts cycling before treatment and figure 1b illustrated the results in herds where the gilts were old enough but no information was available on previous estrous cycles. A higher percent were not detected in estrus in the second group presumably because they were not cycling before treatment. Data from other trials has shown that following treatment of cycling gilts over 90% can be expected in estrus during a three day period. For gilts on commercial swine farms over 7 months of age, but without records of previous estrus 80% can be expected in estrus within three days. For prepuberal gilts less than 6 months of age approximately 60% can be expected in estrus between days 5 and 10 post-treatment. The response varies from farm to farm and between seasons of the year.

Table 1. Fertility following synchronization of estrus with altrenogest in gilts

Treatment	Number of gilts bred	Percent farrowed	Litter size	Reference
Control	145	83	10.0	Jobard et al. Unpublished
Altrenogest	175	83	10.7	
Control	70	60	10.0	Webel,1978
Altrenogest	68	75	11.3	
Control	68	74	9.1	Pursel et al. 1981
Altrenoget	58	71	10.5	
Control	29	86	9.8	Britt, 1980
Altrenogest	48	85	9.9	

Adapted from Webel and Day, 1982
1. Significantly ($P<.05$) different from control group.



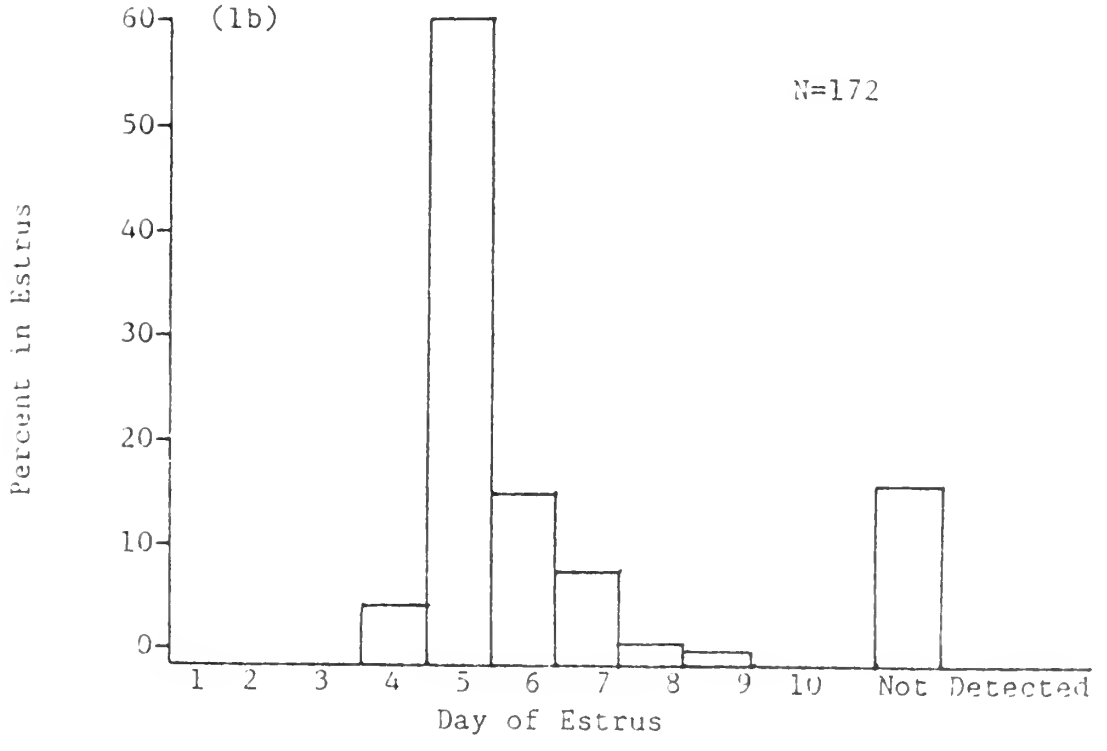


Figure 1. Percent in estrus by day following altrenogest withdrawal for gilts which were previously cycling (1a) and for gilts on commercial farms without previous records of estrus (1b).

Representative results from fertility comparisions are presented in table 1. In general little difference has been observed in either farrowing rate or litter size. As was observed previously and can be noted in table 1, there has been a tendency for increased litter size following altrenogest. Although the differences have been statistically significant in some trials they have been small and variable ranging up to one pig per litter.

Altrenogest has been a very effective product for regulation of estrus in the gilt or sow. Estrus has been fairly precisely scheduled when the animals were cycling before treatment. However, altrenogest has not been very effective for treating non-cycling gilts (Kraeling et al., 1982). This product is most effective for synchronizing estrus in gilts to coincide with estrus in weaned sows. Fertility has not been impaired and perhaps litter size may have been slightly increased. Although this compound is available in France, it has not yet been approved by the FDA. Research and development on the product is continuing with good prospects that it will be available to the U.S. producer in the future.

Products for Induction of Estrus

In contrast to progestins like altrenogest, which are useful for estrous cycle regulation because of their suppressive activity, another class of compounds, the gonadotrophins, are useful because they are stimulatory. These are hormones that act to induce or stimulate follicle development, ovulation and estrus. The normal hormones which control this function are follicle stimulating hormone (FSH) and luteinizing hormone (LH). These natural hormones have been used to induce estrus, but are not practical under farm conditions because frequent injections of precise doses are required. Two compounds that have been substituted for FSH and LH are pregnant mare serum gonadotrophin (PMSG) and human chorionic gonadotrophin (HCG). PMSG is obtained from the blood of pregnant mares and contains both FSH and LH activity whereas HCG is obtained from urine of pregnant women and has primarily LH activity.

PMSG is not a new compound, but rather has been available and widely used outside the U.S. for many years. It was commercially available several years ago in the U.S., but was withdrawn from the market presumably because of limited sales and regulatory requirements. However, at the present time, several firms are actively pursuing production and registration of PMSG, therefore I believe we can discuss it as a product that will be available to the swine producer.

PMSG is effective for stimulation of follicle development and induction of estrus in anestrous gilts or sows. Therefore, it is most useful for prepuberal or non-cycling gilts and for sows which have not returned to estrus following weaning. This product is not effective for inducing or synchronizing estrus if the animal is cycling. When PMSG is injected during the luteal phase of the cycle, follicle development is stimulated, but estrus does not occur. This is due to the inhibitory effects of progesterone, which block estrus. Altrenogest should be the product of choice for cycling animals. Injection of PMSG to prepuberal gilts or sows at weaning, when progesterone levels are low, generally induces estrus in 4 to 6 days. An example of the response for non-cyclic gilts and early weaned sows is presented in table 2.

Table 2. Response of seasonally acyclic gilts and primiparous sows to PMSG

	Gilts		Sows	
	Control	PMSG	Control	PMSG
No. Animals	26	26	31	21
No. in estrus	22	24	29	21
Days to estrus	38	16	12	5
Percent in estrus within 10 days	15	69	58	95
Percent farrowed	86	87	67	86
Litter size	7.7	8.8	10.6	9.5

Adapted from Britt et al., 1984

The mean days to estrus were 16 and 5 respectively for PMSG treated gilts and sows compared to 38 and 12 for controls. The percent in estrus within 10 days was increased in PMSG treated animals to 69 for gilts and 95 for sows compared to 15 and 58 in controls. These results are typical of and in agreement with other data reported following use of PMSG (Webel and Day, 1982). This trial represents fairly typical applications for this product which are induction of estrus in gilts and stimulation of estrus in sows at weaning. Injections of PMSG should be given on the day of weaning or to recently weaned anestrous sows. Gilts should be of the size and near the age of puberty, but not cycling for best results.

HCG has been used both in combination with and subsequent to PMSG. A product called PG 600 is widely used outside the U.S. This product contains a combination of 400 IU of PMSG and 200 IU of HCG. It is being used for the same applications as PMSG. With PG 600 a lower dose of PMSG is required compared to doses of 800 to 1000 IU when PMSG is used alone. HCG has also been used subsequent to PMSG to induce ovulation. For this application HCG is injected 72 to 96 hour following PMSG and ovulation is induced approximately 40 hours later. This sequential treatment may permit a fixed time insemination without estrus detection.

Another product that is marketed for treatment of cystic ovaries in cattle, gonadotropin releasing hormone (GNRH), has been evaluated in gilts and sows. GNRH has been effective for inducing estrus and ovulation however frequent injections over long periods of time are required. At the present time this is not a feasible product to consider for control of estrus on the farm. Perhaps as new delivery methods are developed or further research is completed GNRH may be of use in the future.

PMSG, HCG and PG 600 are all available in most countries outside the U.S. HCG is currently available in the U.S. and several firms are now developing PMSG products. Perhaps within a reasonable period of time these management tools will be available to the swine producer.

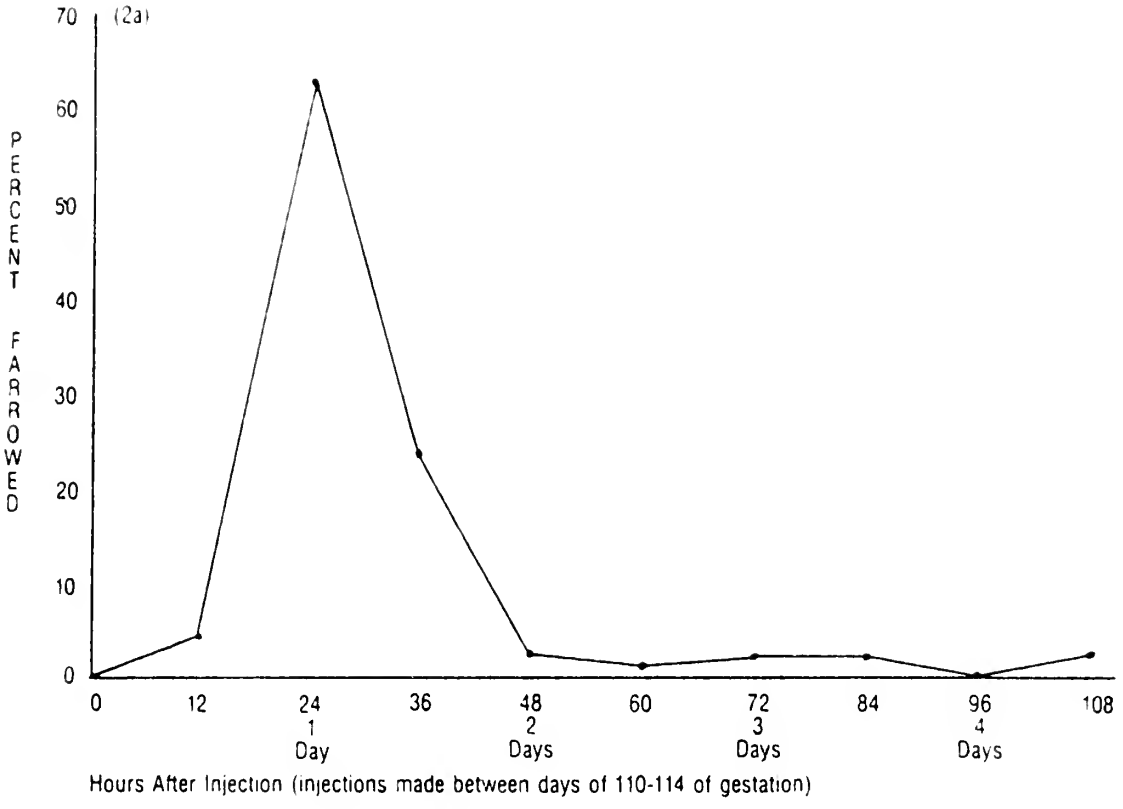
To summarize the potential products for controlling the time of breeding we have discussed two basic types of compounds. Altrenogest functions by suppressing the estrous cycle during administration. Then following withdrawal of altrenogest from the feed, normal cyclic activity resumes and gilts are in estrus 5 or 6 days later. PMSG or PG 600 stimulate follicle development and ovulation. Estrus is induced 4 to 5 days following injection. HCG can be injected 3 or 4 days following PMSG to precisely control the time of ovulation. Altrenogest is the drug of choice when animals are cycling and PMSG is the drug of choice for anestrous animals. These products can also be used in combination to synchronize estrus and ovulation at a precise time to permit a fixed time insemination.

CONTROL OF FARROWING

Parturition or abortion can easily be induced with prostaglandins at any stage of gestation in the sow. However, to successfully produce viable live pigs, the sow must be within 3 or 4 days of term (First, Lohse and Nara, 1982). This product is perhaps more exciting than the estrous regulation products because it is presently available. Several firms have a product under development and at least one has obtained FDA approval for marketing. From data available it appears that each product which has been tested is similar in effectivness. Therefore, we will discuss results for the product that is currently approved by the FDA and is available for use.

Prostaglandins injected on day 111, 112 or 113 of gestation have induced parturition in 75 to 90 percent of treated sows within 30 to 36 hours. Generally, if sows are injected between 6 and 11 in the morning they will farrow during working hours the following day. Figure 2 depicts the pattern of farrowing observed in one large field study following injection of Lutalyse on days 110 to 114 of gestation. The Lutalyse injected sows farrowed at a mean of 30 hours following injection with 92 percent farrowing by 36 hours after injection. Control sows farrowed in a normal pattern over an 11 day interval between day 110 and 120. The duration of farrowing, proportion of pigs born live and weaned were not different between the two groups, demonstrating that prostaglandin effectively induced farrowing without an adverse effect on piglet survival (McAllister et al., 1982).

A comparision of the time of farrowing following treatment with prostaglandin or prostaglandin plus oxytocin with controls is presented in table 3. The objective of this trial was to maximize the number of sows that farrowed by the end of the work day on the day following Lutalyse injection. Oxytocin was injected 20 to 24 hours following Lutalyse in part of the sows to determine if farrowing could be more closely synchronized. Previous research has shown that injection of oxytocin 24 hours following Lutalyse induced farrowing in 86% of sows within 3 hours compared to only 33% for controls (Gall and Day, 1981). Synchronized farrowing was desired to facilitate attended farrowing, cross-fostering of piglets and processing and handling the litters of piglets as a group.



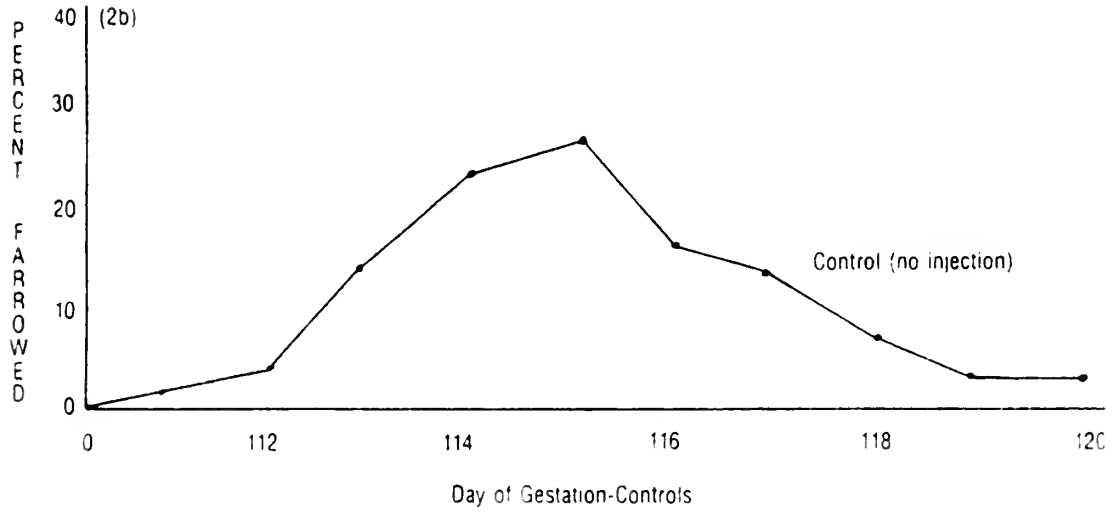


Figure 2. Percent farrowed by day of gestation for control (2b) and hours after 2 ml Lutalyse injected once between days 110 and 114 of gestation (2a).

(Adapted from McAllister et al., 1982)

The results, in table 3, are presented as the proportion farrowed by 30, 36 or greater than 36 hours. When Lutalyse was injected at 7 or 8 o'clock in the morning, 30 hours later fell in the afternoon of the second day and 36 hours after the end of the normal work day. Control sows farrowed from day 112 to 118 with a peak on day 114. Following Lutalyse injection about 80% of the sows had started farrowing by 30 hours and 90% by 36 hours. Following oxytocin injection the proportions farrowing were increased to 92 and 94% at 30 and 36 hours respectively.

Table 3: Comparision of natural and scheduled farrowing

Treatment	Number treated	Time of farrowing				
		Percent farrowing (day)				
		111-112	113	114	115	116-119
Control	50	14	18	22	16	30

Treatment	Number treated	Percent farrowing (hour)		
		0-30	31-36	36+
Lutalyse	38	79	10.5	10.5
Lutalyse + oxytocin	65	92	2.0	6.0

1) Lutalyse was injected on days 112, 113 or 114 of gestation.
2) Nine percent of the sows assigned farrowed before the scheduled injection and were not included in the analysis.

Cross-fostering and processing pigs was greatly facilitated when 80 to 90% of the sows had farrowed by 3 o'clock in the afternoon. Following the injection of oxytocin nearly all sows had farrowed by 12 to 1 pm. This facilitated assigning a person to be in attendence at the farrowing. Although some sows farrowed during the night prior to 24 hour following injection and were not attended, these litters were available for cross-fostering with litters that farrowed during the second day. For example, in one farrowing group of 24 sows, 20 were injected on the same day. Two of the treated sows farrowed during the night and two had not farrowed by 30 hours. Thus cross-fostering of pigs was done among the 18 litters. This permitted allotting similar numbers and size of pigs within litters for each of the 18 sows. Normally only 3 to 5 litters were available for crossfostering. The results presented in figure 1 and table 3 demonstrate that Lutalyse effectively synchronized farrowing and

that oxytocin further synchronized farrowing when injected 20 to 24 hour following Lutalyse.

The effect of induced farrowing on the number of piglets born and weaned is presented in table 4. Because of differences in the total number of pigs born, the parameters born live and born dead are expressed as percentages in addition to the actual numbers. Differences in total pigs born were not due to treatments since assignments were random. Observation of such differences between groups of sows in number of pigs born emphasizes the need for properly controlled experiments when farm trials are conducted. For the Lutalyse + oxytocin + attendance group there was a person in the farrowing room continuously during farrowing to both observe and assist delivery. For the other groups, sows were observed periodically but not attended.

Table 4: Comparision of farrowing performance with natural or induced parturition

Treatment	Number treated	Number born		Percent born live	Number weaned	Percent weaned
		Total	Live			
Control	49	11.2	10.6	94	8.6	77
Lutalyse	38	10.3	9.8	96	8.0	78
Lutalyse + oxytocin	32	10.2	8.9	88	7.8	77
Lutalyse + oxytocin + attendance	32	11.3	10.8	95	9.0	80

1) Nine percent of sows assigned to the Lutalyse groups farrowed before injection.
2) Ten percent of the sows assigned to the oxytocin groups farrowed before injection.

There were no statistically significant differences between any of the treatments in percent of pigs born live or weaned. However, the decrease in percent born live in the Lutalyse + oxytocin group should be noted. Following oxytocin some sows would farrow one or perhaps two pigs and then uterine contractions would essentially stop and delivery would be prolonged. It was also noted in some sows that uterine contractions would move piglets into the birth canal but expulsion would not be completed. Increased numbers of pigs born dead were observed in these litters unless action was taken either in the

form of manually assisting in delivery or administration of additional oxytocin. Similar reports of problems associated with oxytocin treatment following prostaglandin have been received from researchers, and also from veterinarians and producers in the field. Therefore, until further research is completed oxytocin should only be injected if milk can be expressed from the mammary gland and when highly competent personnel are attending the farrowing.

In summarizing the control of farrowing, we have seen that time of farrowing can be precisely controlled by injection of a prostaglandin when the sow is within 3 or 4 days of term. Treatment with oxytocin 24 hours later will further synchronize farrowing. Piglet survival has not been adversly affected by synchronization of farrowing. The primary advantage from synchronized farrowing is consolidation of activities associated with farrowing and improved opportunity for cross-fostering of piglets.

OTHER POTENTIAL NEW PRODUCTS FOR THE FUTURE

There are certainly other potential new products under development, many of which may only be known to a few of the researchers or firms involved in their development. Looking into the crystal ball I forsee products to improve the precision for controlled farrowing. For example, one current area of investigation is for a product to delay farrowing in sows which farrow before the scheduled injection of prostaglandin. Improved methods for detecting pregnancy and predicting litter size early in pregnancy are being tested and will surely be available in a few years. Other developments may include improved delivery systems for currently available hormones to improve their efficacy or ease of administration. In association with products for controlling reproduction, will be products for easy and permanent sow identification, and perhaps indwellng sensors to monitor the health and physiological activities of the animal. For example, the herdsman sitting in front of a computer terminal may be able to identify precisely the best time to inseminate a sow, when farrowing will begin or which sows may need assistance during farrowing.

SUMMARY AND CONCLUSIONS

The products which are now or will soon be available to assist in controlling the time of breeding and farrowing in gilts and sows have been reviewed and discussed. For cycling gilts or sows altrenogest is very effective for regulating the time of estrus, but is not effective in anestrous or non-cycling animals. Groups of animals can be synchronized or estrus controlled in individual animals by administering altrenogest in the feed for 14 to 18 days. Estrus is generally observed on the 5th or 6th day following withdrawal. For anestrous or non-cycling animals, such as prepuberal gilts or sows at weaning, PMSG is effective for inducing estrus. HCG can be combined with PMSG as a single injection or given 72 to 96 hours later to more precisely control the time of ovulation. Fertility and litter size are not impaired following the use of these products.

The time of farrowing can be controlled by injection of prostaglandin within three days of expected farrowing. Approximately 90 percent of treated sows will farrow within 36 hours. Farrowing can be further synchronized with the proper use of oxytocin 24 hours following prostaglandin. However, oxytocin should not be used unless milk can be ejected from the mammary gland.

With the proper application of technology the producer of the future will select the date for breeding in advance. The time of estrus will then be programmed and synchronized with insemination at an appointed hour. Pregnancy status and litter size will be determined. The animals retained to farrow will then be selected and farrowing scheduled for an appointed hour on a preselected day several weeks in advance. The precise farrowing schedule for groups of sows will be programmed weeks or even months in advance. Certain aspects of this type of program are currently being done on an experimental basis on several commercial swine farms. When the level of management is excellent such programs work very well but under poorly managed conditions this technology can be counter productive.

REFERENCES

Britt, J.H., K.L. Esbenshade and K. Heller. 1984. Response of seasonally acyclic gilts and weaned primiparous sows to pregnant mare's serum gonadotrophin (PMSG) and Regumate. J. Anim. Sci., 59:20, (Suppl. 1)

First, N.L., J.K. Lohse and B.S. Nara. 1982. The endocrine control of parturition. In, D.J. Cole and G.R. Foxcroft (Ed.); Control of Pig Reproduction. Butterworths, London.

Gall, M.A. and B.N. Day. 1981. The induction and synchronization of parturition in sows with prostaglandin F2 alpha and oxytocin. J. Anim. Sci., 53:319, (Suppl. 1).

Kraeling, R.R., G.B. Rampacek and T.E. Kiser. 1982. Failure of the orally active progestin, Regu-mate to overcome confinement-induced delayed puberty in gilts. Theriogenology, 17:183.

McAllister, J.F., J.W. Lauderdale, R.W. Thomas and S.K. Webel. 1982. Effect of 10 mg PGF2 alpha (Lutalyse Sterile Solution) on induction of parturition in the pig. Technical Report No. 631-9665-82-002, The Upjohn Company.

Webel, S.K. 1979. Ovulation control in the pig. In, D.B. Creighton, N.B. Haynes, G.R. Foxcroft and G.E. Lammimg (Ed.); Control of ovulation. Butterworths, London.

Webel, S.K. and B.N. Day. 1982. The control of ovulation. In, D.J. Cole and G.R. Foxcroft (Ed.); Control of Pig Reproduction. Butterworths, London.

Requirements for Drug Approvals in Food Producing Animals

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Food and Drug Administration (FDA) on the basis of conclusive evidence that it is both effective and safe. The responsibility to develop and present that evidence belongs to the drug sponsor. When the drug is intended for use in food-producing animals, the safety issues become extremely complex because it is not simply a matter of demonstrating that the drug is safe for use in the target animal, but also that the food products derived from the treated animal do not contain unsafe drug residues. It is this latter issue that presents the greatest challenge to a drug sponsor, but it is certainly not the only issue that must be considered.

THE REGULATORY REQUIREMENTS

The animal drug industry is regulated by several sections of the Federal Food, Drug and Cosmetic Act (FFD&CA). While the first law enacted by Congress for the purpose of protecting the public against adulterated or unwholesome food went into effect in 1890 (Wiley Act), being significantly strengthened in 1906 and 1912, it was in 1938 that the basic Federal Food, Drug, and Cosmetc Act as we know it today came into being. Since that time, there have been several modifications, the most important to our business being the Kefauver-Harris Amendments of 1962 and the Animal Drug Amendments of 1968. The enactment of these latter two amendments has resulted in the basis for the regulation of animal drugs in the manner we know today.

When federal agencies become involved in regulating some aspect of our society, they obtain their authority from the specific laws passed by Congress. They then develop regulations to flesh out the law and provide more detail as to how the law will be interpreted and implemented by the Agency. The regulations developed by FDA, as a federal agency, to implement the FFD&CA are contained in a multi-volume document called Title 21 of the Code of Federal Regulations(21 CFR).

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The regulations contained in this document are, in essence, as binding as law. The procedures for adding, deleting, or revising regulations contained in the CFR's, are very precise, in many cases being as laborious as those involved with the enactment of legislation by Congress.

The laws and regulations, however, seldom define precisely what the Agency requires in specific situations for their actual implementation. When it appears to be desirable to better define how the Agency plans to specifically interpret various requirements, it will develop and make available guidelines. Guidelines are intended to be just what their name implies. They are not laws or regulations, but specific suggestions as to how the Agency believes a specific issue should be satisfied. However, while they are not as binding as law, one needs to have an acceptable alternative if one is to deviate from them. Guidelines then become the real criteria a drug sponsor must satisfy when developing a new drug for animals. When those animals are food-producing animals such as swine, the guidelines become very specific and generally quite rigid. The reason for this, of course, is that FDA's greatest responsibility to the public is to protect its health by making sure its food supply is as safe and wholesome as the public believes it to be. The public's confidence that the food it eats is safe is the critical issue and will be elaborated on later.

THE DATA REQUIREMENTS

The data that are necessary to develop before a drug is approved for use in animals are defined in 21 CFR. Specifically, the requirements are spelled out in 21 CFR Part 514 and are included in an official FDA document called the Form FD 356V, or New Animal Drug Application (NADA). The NADA contains eleven (11) major sections, and dozens of sub-sections, all of which have to be satisfied before a drug may be approved.

Following are the major sections of the NADA, along with a brief summary of the information called for in each, and thus, a description of what is required for drug approvals in animals, specifically those used for food:

1. **Identification** - This simply identifies what kind of application the sponsor is submitting, including a brief description of the drug and the sponsor's name and address.

2. **Table of Contents and Summary** - Describes the organization of the application and a summary of what it contains, including the scientific rationale and purpose of the drug, highlights of the research conducted, evaluation of the data, and the conclusions reached. In short, upon reading this section, one should be able to understand what the sponsor is proposing and on what basis the proposal is made.

3. **Labeling** - The basis for any drug approval is its label. This is the information which the user will have to depend on to properly use the drug. Therefore, it must be complete, accurate, clear, and totally consistent with the data from the research conducted to support the approval.

4. **Components and Composition** - This defines the ingredients that make up the drug product and how they are put together to make the final formulation.

5. **Manufacturing Methods, Facilities, and Controls** - This is a very complex section which provides a complete description of how the product is produced, the facilities and equipment used, the qualifications of the people involved, the specifications and test methods for all the raw materials and finished product, description of and test methods for all the packaging components, stability data, and an environmental analysis of the manufacturing process.

6. **Samples** - This section is not always used, but provides for the submission of samples of raw materials or finished product for testing purposes when requested by FDA.

7. **Analytical Methods for Residues** - This is one of two major sections of the new drug approval process which are unique to food producing animals. Basically, it requires the development of an analytical procedure to detect minute levels of residues of a drug or its metabolites, and the establishment of a withdrawal period to assure the public does not inadvertently consume potentially harmful chemicals. While these are the primary goals for this section, the research to achieve them is very complex and expensive, and will be addressed again later.

8. **Evidence to Establish Safety and Efficacy** - The research designed to determine if the drug has the effect claimed on the label and is safe to the animal treated, is conducted to satisfy this section of the requirements. It is through this work that a dose is established, the treatment period determined, the margin of safety determined, and the nature of side effects and/or adverse reactions identified. Most of the information included on a product's labeling comes from these studies.

9. **New Animal Drugs Subject to 512(n) of The Act** - This section is now obsolete and may be addressed by a simple statement that it is not applicable.

10. **Environmental Impact Analysis Report** - This is the second section which is, in most cases, unique to drugs for food producing animals. A comprehensive report must be prepared, detailing the potential effect of the subject drug on the environment. Generally, this will involve research studies which are designed to evaluate the effects of a drug, when used as labeled, on the soil, groundwater, lakes and streams, plants, normal environmental bacteria, wildlife etc. It involves a detailed understanding of the fate of a drug in the animal and the exposure of the environment to the drug through the animal's excretions. Depending on the drug and its use, this can be an extremely complicated issue to resolve.

11. **Confidentiality of Data and Information in a New Animal Drug Application** - To comply with relatively recent regulations based on the public's "right to know" whats going on in regulatory agencies, a Freedom of Information (FOI) summary is prepared for public disclosure. This FOI summary becomes probably the single most important piece of the NADA, since it is what is made available to the public for its use. It must include rather detailed summaries of all the data on which approval of a product is

based, and therefore must be legally defensible. For this reason, it is highly scrutinized by FDA lawyers, making sure that, if challenged, its contents may be defended in court. Since the FOI regulations came into existence in the mid 70's, the requirements have become increasingly more comprehensive, so that at the present time, a typical summary could be anywhere between 25 and 100 pages in length.

The Review Process

Once all the work completed and is the NADA assembled, a process that for drugs intended for food animals will take an average of 5-8 years, it is ready for submission to FDA for review. FDA is man dated by regulation to respond to any NADA within 180 days. While in the past this was not often adhered to, the Agency is now quite sensitive to its time commitments. However, when that first response is received in 180 days, it invariably is in the form of an "incomplete letter". This means FDA has not accepted the sponsor's data, and that specified deficiencies must be rectified.

Depending on the nature of those deficiencies, the response from the sponsor may occur in from a few weeks to years. Many times, additional research may be required, in which cases, the response will take months to years to prepare. This whole process may be repeated a number of times until finally, FDA accepts the sponsor's application and approves it. From the time it's discovered or speculated that a drug for food animals has a particular activity, is developed for a specified use and approved by FDA, it is not unusual for ten years to elapse, and an expenditure of one to several millions of dollars to have occurred. What is responsible for all this time and money?

Human Food Safety

When you and I as consumers buy animal food products at the super market, we assume them to be safe and wholesome. We do not want them to contain residues of drugs that may be harmful if consumed. You as producers, do not wish to market livestock which may contain harmful drug residues. We as drug manufacturers want to know the safety and residue picture of our products so they may be labeled for safe and effective use. And, of course, FDA wants to be sure the public is protected and its confidence in the wholesomeness of its food supply maintained.

The key factor to consider is the <u>assumption</u> by the consumer that its food supply is safe. If we buy cigarettes, we buy them with the full knowledge, because of mandatory labeling, that they are harmful to our health. When we use drugs directly ourselves, we are told either by our doctor or pharmacist, or by the labeling of the drug, what risk we are taking along with the drug. In both of these cases, we make an informed decision with full knowledge of the risks. However, we partake of our food supply with a different attitude. We <u>assume</u> it's safe. We acknowledge no risk. Therefore, the producer, processor, feed and additive suppliers, and the regulatory people all have a responsibility to see to it that it is in fact safe, and that the consumer's trust is not violated.

As mentioned previously, the drug manufacturer's responsibility involves the development of an accurate and adequately sensitive analytical method to detect very small levels of drug in meat and milk, and then the establishment of an adequate withdrawal period to insure against inadvertent consumption of harmful residues. These two activities are tied closely together and both relate to the basic problem of establishing what is a safe tissue level of residues of a given drug.

In dealing with the challenge of establishing a safe level of a drug in tissue, and ultimately an appropriate withdrawal period, several activities are systematically addressed:

1. **Threshold Assessment (TA)** - The chemical is subjected to several assessments to determine the extent of testing necessary to assure safe use. In general, the decision becomes one of whether or not the chemical has the potential to produce tumors (carcinogen). If it does, then it is handled in a much more severe manner, and may never survive the rigid criteria established. The purpose or intended use of the chemical also plays a major role in its assessment (e.g. a drug used on a herd basis for routine use as a growth promotant will have more rigid requirements than one used on individual animals to treat a specific disease. The third part of the assessment involves the determination of total drug residues through the use of radioactive drug studies. This measures the total of both the drug itself and its metabolites. The higher the total residue, the more severe the requirements.

2. **Toxicological Testing** - Depending on the outcome of the Threshold Assessment, a series of toxicological tests are conducted in various species of laboratory animals such as mice, rats, dogs, sub-human primates, or other special species as indicated. These studies may range from specialized short term studies to 30-day, 90-day, 1-year, 2-year, and lifetime studies, depending on the perceived need based on the TA. It's important to recognize that the purpose of these studies is not to determine safety in the species tested, but to try to predict the expected safety concerns for man, attempting to establish the highest level which causes no effects. Once the highest no-effect level is determined, then a 100X or 1000X safety factor is applied to further assure safety to man. Laboratory animals must be used in these studies because we certainly can't run such experiments on humans.

3. **Metabolism Studies** - These become some of these most complicated and expensive studies to conduct and interpret. The objective is to determine what happens to a chemical in the animal. Is it broken down to metabolites or does it pass through the animal unchanged? The purpose is to identify what chemical should be used for toxicology testing and as the marker compound for residue testing? Sometimes a metabolite is more toxic than the parent compound, so it obviously would not do any good to conduct the toxicology studies with the parent drug. Also, the target tissue is identified. This is the tissue (e.g. liver, kidney, fat etc.) in which the highest and longest residue level is expected.

4. **Establishing a Tolerance** - Based on the results of the metabolism studies, the toxicology studies, the highest no-effect levels, the safety factors applied, the target tissue, and an arbitrary consumption value, a tolerance, or safe tissue level is established.

The conclusion is that the consumption of food products containing the tolerance level of a drug and it metabolites would be completely safe.

5. **Method of Analysis for Residues** - Once the marker compound, target tissue, and tolerance are established, then a method of anlaysis is developed so that the food supply can be monitored for residues of the subject drug. This allows the USDA (FSIS) to follow a particular drug so we can be assured that violative tissue residues are not contaminating our meat and milk supply.

6. **Establishment of a Withdrawal Period** - The final question to be answered is, how long must a drug be withdrawn from an animal prior to that animal being slaughtered for food to assure that no residues in excess of an established tolerance level will exist? Using the established method of analysis, tests are conducted on animals sacrificed at various times after withdrawal. The withdrawal period then becomes the first kill-time that all samples are either negative or below the approved tolerance. This period then become part of the approved product labeling, that when followed, assures safe use of the drug.

Conclusion

We have all read the negative reports of the toxic substances contaminating our food supply. The media and certain other groups seem to revel in such reports. We all have a very serious responsibility to see to it that these negative reports are unfounded, and that our food supply remains the most abundant, nutritious and safest in the world. I hope that this discussion gives added confidence to you, and ultimately to the public at large, that the drugs used to medicate our livestock, are completely safe from a food residue standpoint as long as all label directions are followed. The regulatory and scientific process required to obtain approval of these drugs for use in food producing animals mandate this to be the case.

Application of Biotechnology Tools to Animal Agriculture: Monoclonal Antibodies and Transfection

KEITH W. KELLEY

Imagine the changes in pork production that would occur if producers could buy cloned pigs. Some operations might purchase fast-growing pigs, others might use pigs that were resistant to certain bacterial infections while other producers might select pigs that had large loin eyes. Tremendous changes would have to occur in managing this "biotech" pig operation. New types of decisions would have to be made. Selecting pigs would be like choosing seedcorn varieties.

Fifty years ago, most dairy farmers could not imagine a good dairy farm without a bull in the pasture. At that time, scientists were studying the possibilities of artificial insemination and frozen semen. Today, buying frozen semen and inseminating cows is an accepted management practice. An entire industry has been built to serve the needs of livestock producers who are interested in using artificial insemination. Land grant universities, such as the University of Illinois, played a leading role in developing and implementing these tools for use in animal agriculture. In those times, agricultural scientists were doing research that would be needed by animal producers 25 years later. Those scientists were thinking and planning for the future of the animal agriculture industry.

The same thing is happening today. Agricultural scientists have acquired new tools to conduct research. Approaches that were not available just five years ago can now be used to solve problems. Researchers are only now learning to apply these techniques in their research programs. They are just beginning to understand how approaches such as these can be used to answer questions that they feel are important keys to the profitable production of domestic livestock.

The excitement about biotechnology in agriculture has not been generated because of what it has already done for agriculture, but rather for the promises it brings for the future. This excitement is well-justified when one considers the advances that have been made in a short period of time with humans and mice. However, there are great challenges that must be met before applying these techniques to animal agriculture. The philosophical issues of engineering new organisms must be discussed and reconciled by our society. The application of basic scientific protocols to domestic animal problems will require time.

In general, we do not understand the basic biology of domestic livestock nearly as well as the more common experimental subjects, rodents and humans. Therefore, agricultural scientists must "catch up" to what is already known about other

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animals. Reagents that can be purchased for standard research subjects are not generally available for domestic animals. Before genes are transferred, animal scientists must know which genes of domestic animals are important for enhancing animal production. This type of fundamental research with domestic animals has not yet seriously begun. Therefore, spectacular results with biotechnology tools will be an exception, rather than the rule, during the next decade. Some significant advances have already been made, and these will continue. However, it is more realistic to view the efforts in biotechnology as an investment for the future. These tools will be used initially to develop a more complete understanding of the basic biology of domestic animals. This information may not be applied to animal production until possibly after the turn of this century.

BIOTECHNOLOGY

Modern-day biotechnology really represents significant advances in methodological tools that are available to the research scientist. Researchers are excited about applying these tools to agricultural problems. From a broad prospective, biotechnology is generally divided into three areas: recombinant DNA technology, monoclonal antibodies and embryo manipulation.

Two years ago, Walter Hurley from the Dairy Science Department at the University of Illinois presented a primer on recombinant technology to Illinois Pork Producers (Hurley, 1982). Dr. Hurley explained that genes from animals, which were once considered to be the smallest unit of inheritance, are really portions of a molecule known as deoxyribonucleic acid (DNA). Recombinant DNA technology as applied to animal agriculture involves cutting-up the genetic information that exists in the chromosomes of living organisms, finding the DNA that is important for enhancing animal production, and introducing these DNA molecules into living cells where they can grow (i.e., artificially recombining DNA).

Through centuries of evolution, nature has done an excellent job of "recombining DNA." Scientists who are interested in recombinant technology are now trying to unravel nature's mysteries to enhance animal production and growth. Embryo manipulation will be an important part of this research effort. These possibilities were highlighted about one year ago in Science magazine, where a group of researchers transferred a gene for human growth hormone into mouse embryos (Palmiter et al, 1983). The embryos were transferred to recipients, and the resulting "supermice" grew significantly faster than controls. They were also capable of transfering this gene to their offspring. These techniques, when combined with embryo sexing and splitting, are important biotechnological advances that need to be applied to agricultural problems.

WHAT ARE MONOCLONAL ANTIBODIES?

Monoclonal antibodies are important tools that constitute another major portion of biotechnology. Monoclonal antibodies are made from cells that have also been artificially engineered - these cells are called hybridomas. These genetically-engineered cells are hybrids of a certain type of cancer cell line and a normal lymphocyte. Lymphocytes are a type of white blood cell. There are a variety of different types of lymphocytes, and each type has a special function. One of these types, called a B lymphocyte, can develop into an antibody-producing cell. These B lymphocytes are activated when a foreign substance, called an antigen, enters an animal's body. An antigen can be a virus, parasite, bacteria, fungus or

even cells from another animal. These antigens carry certain shapes on their surfaces, and these shapes are called epitopes or antigenic determinants. Each B cell recognizes only the epitope that initated its synthesis. When the B cell is stimulated by this epitope, it develops into a plasma cell that secretes one and only one type of antibody that is unique to that determinant. Because each antigen may have many different epitopes, many B cells are stimulated to differentiate and become antibody-secreting plasma cells. Therefore, if one searched for antibodies to a given antigen in the blood of a pig, one would find many different kinds of antibodies that bind to different determinants on the same antigen. These are called polyclonal antibodies.

Monoclonal antibodies are identical copies of antibody protein produced from the progeny of a single B cell. Since all of the progeny cells are identical, they are called a clone. To be able to make monoclonal antibodies, researchers must be able to isolate and grow individual B cells. This was not possible in a practical sort of way until 1975 (Kohler and Milstein, 1975). On October 15 of this year, these two immunologists shared the Nobel Prize in Physiology and Medicine for developing this technique (along with another immunologist by the name of Jerne). It is now known as hybridoma technology, and these hybrid cells secrete monoclonal antibodies.

WHY MONOCLONAL ANTIBODIES?

Monoclonal antibodies are already finding an important place in modern-day animal agriculture. For instance, they are being used: (1) in the development of new diagnostic procedures, (2) as research keys to unlock doors leading to an understanding of fundamental factors controlling growth and reproduction and (3) as a therapeutic aid to prevent symptoms of an important diarrheal disease in young animals known an colibacillosis.

All antibodies have an amazing capability of recognizing substances that differ only slightly in chemical structure. Therefore, scientists have exploited this property and have used antibodies to measure substances in the brain and blood at levels as low as one trillionth of an ounce! However, problems sometimes arise when scientists try to use polyclonal antibodies found in whole blood or colostrum of animals. Additionally, many problems occur when researchers attempt to isolate specific antibodies from the blood. The process is time-consuming, it does not yield identical, homogeneous antibodies and the supply of these purified antibodies is limited.

Monoclonal antibodies have many advantages over polyclonal antibodies because they: (1) offer scientists a way to consistently produce antibodies that react with a single determinant (2) are biochemically identical (3) can be used in very sensitive assays (4) can be produced in unlimited quantities and (5) may detect biological compounds that have gone undiscovered using conventional techniques. Of course, monoclonal antibodies have some problems, too - they are expensive to make, they are not useful for some types of diagnostic tests and their biological function may be somewhat limited. However, most evidence indicates that monoclonal antibodies are going to revolutionize diagnostic products, ranging from test kits for pregnancy to sexually-transmitted diseases. They will be used as vaccines, in genetic screening, in cancer detection and treatment, and in the isolation of high-potency biological compounds such as the interferons and cytokines. However, to understand how monoclonal antibodies work and how they may

ultimately be useful in the profitable production of pork, one must first appreciate how the pig normally makes antibodies.

ANTIBODY SYNTHESIS IN THE PIG

When pigs are inoculated with a foreign substance, this antigen gives rise to the synthesis of antibodies by lymphoid cells of the pig. These antigens which induce the immune response must be fairly large (Molecular Weight > 10,000), must exhibit a certain degree of complexity and must be administered at an appropriate dose. sometimes in potentiating compounds (adjuvants). When pigs are first exposed to the antigen, a variety of antibodies are synthesized which will combine with epitopes on the antigen. This specific combination of antigen and antibody leads to the removal of antigen from the pig's body. For example, erysipelas is an infectious disease in pigs that may cause death. Vaccination with killed bacterins or live-culture strains of Erysipelothrix rhusiopathiae of low virulence is sufficient to stimulate antibody synthesis to the organism. If the vaccinated animal is reexposed to the virulent bacteria, the pig rapidly synthesizes antibodies that bind to the bacteria. The antibody and bacteria then nonspecifically bind to lymphoid cells that engulf and kill the bacteria (opsonization). Thus, the antibody is important for guiding the invading organism to specialized cells in the pig's body that subsequently limit growth of the bacteria. Antibodies also serve many other important functions, such as activation of complement (a system of serum proteins involved in many antigen-antibody reactions), neutralization (blocking viral penetration or binding of toxins to receptors on their target cells) and some forms of lymphoid-mediated cell killing.

Scientists can use the antibodies that pigs synthesize for analytical and histochemical techniques. However, pigs make a variety of different kinds of antibodies to different kinds of determinants that exist on the single large antigen molecule.. Some of these antibodies cross-react in an unpredictable manner with other antigens that are almost identical in structure but found on different tissues. These newly-synthesized antibodies can be composed of at least five different kinds of immunoglobulins (IgG, IgM, IgA, IgE and IgD) that each have their own physiochemical properties and biological functions. The specific antibodies can be very important in preventing some important infectious diseases of pigs.

Unfortunately, it is virtually impossible to isolate antibodies that are identical in chemical structure and antigen-binding characteristics from biological fluids of pigs (i.e., blood, milk, colostrum, nasal secretions). While the heterogeneity of the immune response is of benefit to the pig, it has hampered attempts by scientists to understand how lymphoid cells become activated to secrete immunoglobulin, how specific antibodies are involved in preventing disease, how genes from different breeds of pigs might control synthesis of specific antibodies or how other biologic factors might affect synthesis of antibodies. Without answers to these questions, it will be impossible to manipulate the immune response to provide the most efficacious vaccination programs. Therefore, to thoroughly understand the process of antibody synthesis, scientists developed ways of studying antibody production in the test tube where environmental factors can be controlled. These studies were instrumental in leading to the development of monoclonal antibody and transfection technology.

MONOCLONAL ANTIBODIES FOR DIARRHEA PROBLEMS

It is now clear that construction of cells which secrete monoclonal antibodies really use the normal cell machinery that exist for making antibodies in pigs. It permits researchers to isolate B lymphocytes from immunized amimals and immortalize them in tissue culture by fusion with a cancerous cell line called a plasmacytoma. Unfortunately, tissue-culture adapted strains of plasmacytomas from pigs are currently unavailable. Therefore, most monoclonal antibodies are made with plasmacytoma cells from mice, rats or humans.

A monoclonal antibody has been made recently that could have substantial impact on reducing the incidence of diarrheal disease caused by enterotoxigenic Escherichia coli (Sherman et al, 1983). These strains of bacteria possess pilus (fimbriae) proteins. These small proteins permit enterotoxigenic E. coli to adhere to intestinal epithelial cells. After these bacteria attach in the small intestine, they grow and produce various types of proteins called enterotoxins. These enterotoxins cause secretion of fluid into the small intestine and reduce absorption of nutrients. This leads to clinical symptoms of diarrhea and disease. Therefore, if the attachment of bacteria to the small intestine could be blocked, bacteria could not attach, proliferate, produce enterotoxins and cause diarrhea. Scientists reasoned that an antibody against the pilus proteins might be effective in blocking attachment of enterotoxigenic E. coli in the gut. They therefore made a monoclonal antibody against one type of pilus protein (K99). They hoped this monoclonal antibody would prevent symptoms of colibacillosis.

These workers vaccinated mice with the K99 protein. Several weeks later the mice had circulating antibodies to K99, which indicated there were stimulated B lymphocytes that were the source of these antibodies. Spleen cells from the mice were mixed with mouse plasmacytoma cells. This cell line was a mutant that was susceptible to a drug called aminopterin. The cell membranes and nuclei of of these two types of cells were fused with a chemical called polyethylene glycol. The mixture of normal cells, fused cells and unfused cancer cells was put in a growth medium containing the drug aminopterin. As expected, the normal lymphocytes died after a few days in tissue culture. The unfused lymphocyes also died due to the drug aminopterin. However, the fused lymphocytes lived for two reasons: (1) they had a normal gene that was resistant to the effects of aminopterin and (2) they received a gene from the cancer cell line that prevented them from dying as did their normal, unfused counterparts. Therefore, this type of medium selected for the growth of only fused cells which are called hybridomas. The researchers then searched for the original parent B cell that was secreting antibody to K99. This cell line was found, cloned and then grown in large quantities to produce massive amounts of monoclonal antibody against K99.

This antibody was then fed to calves, and the calves were infected with the diarrhea-causing bacteria. The incidence of diarrhea in both the control- and antibody-treated calves was similar. However, treated calves lost half the weight of the control animals. Clinical symptoms of diarrhea were significantly reduced. More importantly, only 29 percent of the calves treated with the monoclonal antibody died, but 82 percent of the control calves died. These results indicated that the passive administration of murine monoclonal antibodies that were specific for a pilus protein of E. coli was effective in protecting calves against death caused by diarrhea. This is the first monoclonal product that has been used for domestic livestock production.

A similar approach could be used for preventing diarrhea in pigs, and perhaps antibodies against other pilus proteins (K88, 987P) could be included in the passive immunization mixture. Furthermore, monoclonal antibodies might be useful for defining changes in specific antipilus antibodies within each of the five immunoglobulin isotypes. This approach has not yet been explored, but some progess is now being made on this problem (Kissinger et al, 1983). Monoclonal antibodies are also being used to help clarify the heterogeneous subpopulations of leukocytes in pigs (Kelley et al, 1984). These reagents may be useful in the diagnosis of other important pig diseases. Finally, progress has been made in fusing mouse plasmacytoma cells with bovine lymphocytes (Srikumaran et al, 1983, Davidson et al, 1982). This is important because these particular hybridomas secrete bovine antibodies rather than mouse antibodies, which might be better in passive immunization schemes for cattle. This advancement now opens the door for the possibility of making antigen-specific antibodies within each immunoglobulin class for each domestic animal species.

CELL LINES MAKING PIG ANTIBODIES--TRANSFECTION

An important aspect of the above discussion is that foreign substances (mouse immunoglobulin proteins) may be absorbed into the blood of calves. If the calves mount an antibody response to these proteins, a second dose of the monoclonal antibodes could lead to pathologic problems such as anaphylaxis or serum sickness. It would be much better if calf monoclonal antibodies against the pilus proteins could used. A similar situation exists with the pig. Some progress has been made towards solving this problem. For example, USDA researchers recently fused mouse plasmacytoma cells with bovine spleen cells. The resulting hybridomas secreted bovine immunoglobulins. This approach now offers the possibility of creating bovine immunoglobulins with a specificity against important antigens on pathogens important in the cattle industry. A similar approach could be used with the pig. Another technique could also be used--it is called transfection.

Transfection is really an important offshoot of recombinant DNA technology (Davis et al, 1985). The technique is very similar to making hybridomas, but the principle differs. With transfection, genetic material is transferred directly into cells without packaging DNA into some inert vector. Indeed, genomic DNA can be transferred directly into mammalian cells. This process is enhanced by using calcium phosphate crystals and polyethylene glycol to shock the cells. This year, one of the first papers was published in which this procedure was used to immortalize mouse B cells that secreted antibodies that were specific for the antigen that was used to inoculate the mice (Jonak et al, 1984). Transfection has even been used to produce tissue-culture adapted lymphocytes from pigs, but possible secretory products from these cells have not yet been identified (Davis et al, 1984). This procedure is exciting because it offers the possibility of directly transferring DNA into mammalian cells, without the tedious techniques involved in embryo manipulation. Theoretically, it should now be possible to directly transfer DNA that codes for some desired protein into cells of domestic animals. However, we must know what DNA should be transferred. Answering these questions, isolating and inserting the appropriate DNA and obtaining expression of these genes will take years of research. But the possibility is exciting to scientists, and it will certainly have far reaching effects in animal agriculture.

SUMMARY

Biotechnology really represents the acquisition of new tools that are needed by agricultural scientists to explore the biology of domestic animals. These

research tools include recombinant DNA technology, monoclonal antibodies, transfection and embryo manipulation. These techniques can be used to more completely understand how nutrition, genetics and reproductive physiology are involved in the profitable production of pork. In the short term, there will be advances that can be immediately applied on the farm. However, the real advances to be derived from this technology will take years of research by several scientists. These types of studies are needed to prepare animal agriculture for the 21st century.

REFERENCES

Dain, A., and E. Tucker. Intra- and interspecific hybridomas for the production of monoclonal antibodies and for gene mapping in cattle. J. Physiology 343:8, (Abstract), 1983.

Davidson, I., S. Katzav, H. Ungar-Waron, Z. Eshhar, J. Haimovich and Z Trainin. Somatic cell hybridization between bovine leukemia virus-infected lymphocytes and murine plasma cell tumors: Cell fusion studies with bovine cells. Mol. Immunol. 19:893-899, 1982.

Davis, W., S. Seifert and S. Ristow. Establishment of continuous cell lines by transfection. Fed. Proc. 43:1605 (Abstract No. 1101), 1984.

Davis, W., T. Yilma, L. Perryman and T. McGuire. Perspectives on the application of monoclonal antibody and transfection technology in veterinary microbiology. Progress in Veterinary Microbiology. (In Press, 1985).

Hurley, W. A primer on genetic engineering. University of Illinois Pork Industry Conference. pp. 62-74. 1982.

Jonak, Z., V. Braman and R. Kennett. Production of contiuuous mouse plasma cell lines by transfection with human leukemia DNA. Hybridoma 3:107-118, 1984.

Kelley, K. W. Davis, H. Salmon, J. Aynaud and M. Olivier. Monoclonal antibodies against porcine leukocyte antigens. Fed. Proc. 43:1681 (Abstract No. 1545), 1984.

Kissenger, C., K. Kelley and W. Davis. Production of monoclonal antibodies against the major porcine immunoglobulin isotypes. J. Anim. Sci. 57 (Suppl. 1): 350 (Abstract No. 527), 1983.

Kohler, G., and C. Milstein. Continuous cultures of fused cells secreting antibody of predefined specificity. Nature (London) 256:495-497, 1975.

Palmiter, R., G. Norstedt, R. Gelinas, R. Hammer and R. Brinster. Metallothionein-human GH fusion genes stimulate growth of mice. Science 222:809-814, 1983.

Sherman, D., S. Acres, P. Sadowski, J. Springer, B. Bray, T. Raybould and C. Muscoplat. Protection of calves against fatal enteric colibacillosis by orally administered Escherichia coli K99-specific monoclonal antibody. 42:653-658, 1983.

Srikumaran, S., A. Guidry and R. Goldsby. Production and characterization of monoclonal bovine immunoglobulins G1, G2 and M from bovine x murine hybridomas. Vet. Immunol. Immunopath. 5:323-342, 1983.

Pork Production at Ronon Farms

ROBERT G. HUNSBERGER

INTRODUCTION

History of the Farm

Very few people get a start in life without some assistance from Mom and Dad. Our farm is no exception to that. The farm on which our sow herd is housed was purchased by my father in 1948. The house in which I grew up is now the farm office as well as an apartment for the sow herd manager. In 1965, Dad bought the farm that I now live on. At that time, it was a 75 sow farrow to finish hog operation. From 1968 to the present time, we have modified and expanded the farm to its present state. It is now a family owned company with my parents, brother and sister still being shareholders although my wife and I now have a controlling interest in the business.

Description of the Farm

a) Physical Layout

The farm is located in Waterloo County in Southern Ontario. If a line is drawn from Boston to Milwaukee, our farm is on that line at about the midway point. The climate in the area is strongly influenced by the Great Lakes. This results in moderate temperatures in both winter and summer and a significant snow-fall with normally adequate moisture. Our soils are a medium-light loam with an open bottom and adequate natural drainage.

The locations of the land that we own and rent are shown on the map. The net result is that there is one major parcel of land with about 250 workable acres, an 80 acre parcel with no buildings or livestock, and a 35 acre parcel with my house and one feeder hog barn. The major parcel has a gestation barn, a farrowing and breeding barn, a weaned pig nursery barn and a modified open front finishing barn. We farrow about 950 litters annually. This produces about 8000 feeder pigs which are distributed as follows:

Robert G. Hunsberger is president of Ronon Farms Inc., Breslau, Ontario, Canada.

3000 finished to market in the NOF (barn #3)
1500 finished to market in barn #1
1000 finished to market in barn #2
4500 sold as feeder pigs at about 50 pounds.

The feeder barns will be described in more detail later.

The farrowing barn is a concrete building with insulation sandwiched in the walls. It was constructed in 1978 by precasting the wall panels horizontally and lifting them into place with a crane. This building is a tight, well-insulated, rodent-proof structure. Most of the equipment and concrete work was done on the farm in our shop.

Weaner pigs are kept in a conventional stud wall, steel-clad building. The gestating sows are housed in groups in an open-front shed.

b) Staff

The farm is operated with a permanent staff of six people. They are

Jim Conley--general sow herd management and breeding
Shelley Winsor--farrowing house and piglet management
Pete Dumart--feed production and distribution as well as shipping and trucking of pigs
Joe Kunderman--pressure washing and general cleaning
Tony Plein--feeder barns and growing pigs
Bill Swijters--crops, construction projects and general maintenance.

In addition, we have a high school student who spends considerable time on week-ends, summers and holidays helping out in various areas. I have been known to do one or two jobs myself.

Dr. George Charbonneau, a veterinarian who is a partner in another hog operation with me and several others, also spends some time with us reviewing herd health and general production performance and records.

c) Objectives and Philosophies

All members of the staff are encouraged to develop personal goals and objectives for themselves and their families. These can and do vary widely. The degree of commitment and ambition is different for each individual. Our objective is to fit each person into a slot that is, as much as possible, compatible with personal goals. It is useless, for example, to put a person in a very responsible position such as sow herd manager if one of his/her personal goals is to be home by 5:30 each day to coach baseball.

Each person should also understand the criteria by which success in his/her job will be measured. This could be production levels in some area under his/her control or a time-frame for completing a project.

Our major production philosophy is what I refer to as the Closed Biological Cycle. In short, this means that we grow crops to feed hogs and we feed hogs to grow crops. The major feed cost on our farm is energy so we attempt to grow as much of our energy requirement as possible. Similarly, the manure produced by the hog operation is a major nutrient source for our cropping program. This has some strong implications for various areas of the farm. When we are spreading manure, for example, we are fertilizing a crop not only emptying a pit. The distribution and handling practices of manure are important. Similarly, the storage, handling, and processing of corn and grain on the farm are important details. Avoiding the costs of transporting corn aroung the country and the costs of disposing of manure leads us to believe that if the price of corn is too low to make any money growing corn and the price of pork is too low to make a profit feeding hogs, we can buy the groceries by changing our corn into pork.

With this general outline, then, I shall describe the production system in more detail.

MANAGING PRODUCTION

Breeding and Gestation

One of the problems of confinement sow operations is that they amplify the importance of sow productivity. With a limited number of sow places, a shortfall in sow productivity can mean missing output targets. This productivity begins at or before weaning. We believe that lighting in the farrowing rooms and breeding area is important. Our barns have about 65 to 70 foot candles of light in these areas. The lights in the farrowing rooms are on a time clock set to provide 17 hours of light.

The second area we consider important in order to achieve acceptable breeding performance is the condition of the sows at weaning. This is particularly important for gilts. We, therefore, spend considerable time and effort feeding sows during lactation. First litter gilts may be fed extra energy if they are under pressure from a large litter.

On weaning, the sows are moved to the breeding room and penned in groups of 2 or 3 adjacent to boars. This program seems to have been quite successful. It is perhaps the one area of our operation that works as we had hoped. Sows are routinely bred at 5 to 7 days post-weaning.

The breeding of sows is, of course, only the beginning. After that, the sows must be managed to maintain pregnancy and produce a

healthy litter. When we built our barn, our plan was to hold the sows post-breeding for 4 to 5 weeks in individual stalls where they would be fed carefully, pregnancy tested and then grouped in large pens in the gestation barn. That plan, however, was based on 300 sows producing 600 litters per year. We are now keeping 420 sows and producing 950 litters per year. This leaves several options for the individual holding stalls.

1) Keep the sows there for 3 weeks, then group them.
2) Move some immediately post-breeding and some at over 40 days.
3) Move open sows to the gestation barn and use the individual stalls for late gestation animals.

At various times, we have tried all of these and more. For a period of time, we moved all sows being bred for their second litter into the group pens open and bred them with gilts. This allowed us to hold older sows longer in the stalls. Moving the sows at 3 weeks post-breeding seemed wrong initially since it is very near the time of implantation. We briefly played with this practice and ruled it out. More recently, we have begun moving all sows within the first week post-breeding. The stalls are then used for late gestation animals as a sort of pre-farrowing area. This seems to be one of our best options based on our limited experience to date.

Farrowing rate varies seasonally as is common in the industry. One of our strategies is to adjust herd size according to expected farrowing rate dropping the size slightly through the winter and increasing it in the summer to allow us to breed more animals. We also use a spray cooler in the summer and are careful about unnecessary stresses at critical times.

Farrowing and Nursing Piglets

Our targets for financial performance are based on a minimum of 150 pigs per week out of the sow system as 50 pound feeders. At current performance levels, this indicates that about 180 pigs must be born each week. This number of 150 feeder pigs per week is, in our opinion, the most critical number to watch. We, therefore, adjust our breedings and farrowings to achieve this target. The implications of this are considerable. The number of litters, for example, is less important than the number of pigs born. We, consequently, pay less attention to litter size than to total pigs born. A factor such as sow productivity is a fine-tuning detail and we feel it should not receive much attention until the final marketing targets have been reached. In simple terms, our philosophy on this point is that 100 sows producing 2000 pigs are less profitable than 125 sows producing 2100 pigs. All this is, of course, dependent on having enough gestation room to keep the extra sows since, as I said earlier, confinement sow housing amplifies the importance of sow productivity.

After the piglets are born, the object of the exercise becomes to keep them alive and healthy. This is not an easy task, at least not on our farm. The major causes of piglet mortality

and morbidity are crushing, scours, and starvation. The three are closely related. We, therefore, vaccinate sows for E. coli scour prevention and are religious in treating scours in piglets with the appropriate antibiotics. Coupled with this is a sanitation program of washing sows and crates between litters. Piglets are cross-fostered according to weight and colostrum supply. This is done as early as possible in the piglet's life as is castration and tail-docking. After the initial few days starting period, we begin the race to weaning time. Sows are fed all we can possibly give them and piglets are offered creep feed daily after the first week of age. Warmth, absence of draughts, and dryness are all well-documented factors in piglet performance and we monitor them daily.

Weaner Pigs

a) Nursery Rooms

Following the nursing procedures outlined above allows us to wean piglets at 18 to 24 days of age and between 5 and 6 kg. The weaned pigs are moved into double-decked cages, normally in litter groups. They are fed a 20% ration containing milk proteins and sugar. After a week or ten days they are changed to a home-made corn and soymeal ration. We hope that 4 weeks in the cages will get us a 25 pound pig to move to the grower rooms. I say "hope" because it doesn't always happen. This occurs, I believe, for a variety of reasons. The sanitation is again important as scours at this stage can be severe. Moreover, the scours can be nutritionally caused as well as bacterially caused. I suspect that North Americans know less about handling pigs at this stage than do Europeans. We have a lot of work to do in this area. About all I can say at the moment is that the theory of "Brilliance on the Basics" applies here more strongly than in most places and it's always "heads up hockey". We always experience a post-weaning growth check and it appears that sanitation, feed quality, feed quantity, and general husbandry skills are all important in reducing that check.

b) Grower Rooms

When pigs on our farm are moved out of the cages, another race against time begins. The pigs are moved to grower pens which are about 4 feet by 12 feet. The grower area is divided into separate rooms which are cleaned between groups. At this time the pigs are sorted according to size. The time allowed in this area depends on the numbers in the pipeline but averages about 4 weeks. We want as heavy a pig as possible to go into the feeder barns and these conditions set the terms of the race. Since we normally sell some feeder pigs, one of our strategies is to sell those pigs at 40 to 45 pounds, thereby gaining some space to hold the ones we want to finish ourselves to 65 to 70 pounds.

Finishing Hogs

a) Barn #1

Barn #1 is a controlled environment, fan-ventilated barn with an in-barn pit. It has a capacity of 550 hogs. The pigs are self-fed on total slats. In 6 years of operation, it has averaged about 3 turns per year and a feed efficiency of 3.3 to 3.5.

b) Barn #2

Barn #2 is a renovated barn. It has total slats over a shallow gutter system. It is also fan-ventilated. The pigs are self-fed. It has a capacity of 300 hogs. In 4 years of operation, it has averaged about 3 turns per year and a feed efficiency of 3.3 to 3.5.

c) Barn #3

Barn #3 is a mono-slope modified open front building. It has a capacity of 1200 hogs. The hogs are floor fed by a pneumatic delivery system. The system is controlled by a microprocessor which starts the auger and switches the valves to put a specified quantity of feed in each pen. The barn is equipped with a platform scale on which we can weigh pigs in pen lots. This equipment allows us to record feed consumption and average daily gain by pen. From this system, we have been able to learn some interesting things about the barn and animal performance. The most important of these is that after 3 years ofoperation, the barn has averaged about 3 turns per year and a feed efficiency of 3.3 to 3.5.

RECORDING AND MONITORING SYSTEMS

Sow Herd Performance

Sow herd performance is recorded and monitored on a computer-based system. Data is collected on inventory forms printed by the computer. These include a farrowing barn inventory sheet, open sows and breeding sheet, and a gestation barn inventory. These three work sheets go to the barn for daily record keeping chores. The results of farrowing and breeding activities are penciled in as they happen. Each Friday afternoon, the records are brought to the office and entered into the computer. From this data, the computer prints new work sheets for the next week as well as various production reports. The production reports include summaries of farrowing and weaning performance, inventories, expected farrowings and pregnancy test results. Most reports compare the current week's results with 13 and 26 week moving averages. These moving averages are watched closely and selected ones are graphed on a wall chart in the office. This gives us a ready grasp of certain key variables.

Weaner Pigs

Very few computer-based swine systems allow the option of monitoring performance beyond the sow herd. We, therefore, have developed a system of recording that is a combination of manual and computerized methods. The basis of this system is recording weights of pigs and feed. Piglets are weighed at weaning as well as out of cage and grower rooms. The weight of feed used in the cage rooms is recorded. These records are kept manually on a simple daily log sheet. The log sheets are kept in the barn and brought in to the office weekly.

In the early stages of this system, we began to summarize this data monthly. We have now changed to four week periods. The summary sheet is prepared on the computer. It includes the total and average weights into and out of each area for both the pigs and feed. From this, we can calculate weight added, feed efficiency, average daily gain, and mortality rates.

Finishing Barns

The basis of feeder barn records has been a truck scale. All feed is weighed in truck load lots. Pigs entering feeder barns are also weighed on this scale. These weights are recorded by filling in a weight ticket at the scale. These tickets, together with shipping information, allow us to prepare a four week summary similar to the one done for nursery and grower areas.

In addition, we have some records being developed from the computer-controlled system previously described. The pig movement and feeding activities in this barn are logged in the computer memory. The memory of the feeding computer is dumped weekly onto disk and analysed by the office computer. This analysis produces a "life history" for each pen of pigs in the barn. This history includes starting date and weight as well as number of hogs in the group. The feed consumption is then recorded on a daily basis and at any time that a weight for the group is entered, the history will produce a figure for average daily gain and feed efficiency. The exciting aspects of this system are the ability to vary feed consumption over the life of the growing animals as well as according to sex or some other criteria. The ability to control and record feed intake and animal performance on 3000 hogs per year offers some interesting possibilities. I believe that this technology will be the basis of some new concepts in feeding procedures and ration variation. The cost of development can, however, be substantial.

Making Decisions, Assessing Performance and Risk

a) Assessing Performance

From these records, we prepare a final summary which we call

the Production Statistics Summary. This sheet has three sections--one for sow herd performance, one for weaner pig and grower rooms, and one for feeder barns. Each section has three columns--one for the target performance level, one for the current performance level, and one for the alarm point. The figures in the sow herd section are updated weekly, the other sections are done every four weeks. Each week as the new sheet is prepared, it is posted on the office wall and sent out on the clipboards of personnel in the hog barns. This allows the opportunity for the entire staff to be kept informed regarding over-all production performance. Any area that drops below the alarm point becomes an area of concern.

The information on the sheet includes such things as farrow to farrow interval, days from weaning to breeding, number of litters born per week, litter size, stillbirth rate, and nursing mortality. In the growing pig and feeder barn sections, it includes inventories in each area, mortality levels, feed efficiency, and average daily gain.

b) Models, Partial Budgets and Projections

Perhaps the most confounding question on a hog farm is the one regarding economics. Should we be concerned about a production short-fall in a particular area? Will the cost of the cure be more expensive than the disease? We have attempted to address these questions with a computer model of a farm. This model allows us to describe to the computer various production levels as well as capital structure and cash flow parameters. The computer uses this information to calculate a cost of production. By changing any of these variables, we can assess the potential affect on the bottom line. This can be a valuable tool for several purposes. We can use the program as a partial budgeting tool by changing the capital structure to fit the project being considered as well as the production levels affected. The program can be used to reflect historical performance by using the production and financial data from the last year. This use is of particular interest to bankers since they love historical performance figures. A more interesting use for a capitalist is the future projection function. We can anticipate the potential profit or loss in a project or deal under certain performance and price structures. The project can be studied under several production and price structures. By this method, it is possible to make some assessment of the risk involved.

c) Increasing Technical Depth of Knowledge

One of the most obvious short-falls in the hog business is what I call a lack of technical depth. This is certainly the case with us and I suspect it is true for most farms. We understand a little about a lot of areas but the quality of the decisions made is not as good as it might be. The role of the farmer is to take the information and advice given to him by the various disciplines involved in agriculture and integrate this into a productive and

profitable system. It is not an easy job. Our goal, therefore, is to integrate a research enterprise into our system. We attempt to do this in several ways. First, we try to hire people who have some specialized training and make them an active part of our system. In order to do this, we must have challenging jobs and positions within our farm or organization. Secondly, we attempt to maintain contact with people who are specialists in their areas. By doing this, we can have a pool of knowledge available that exceeds our own understanding. I do not expect anyone to make decisions for me, but my own thoughts can be greatly clarified by the exposure to the findings and ideas of others. It is for this reason that conferences such as this one are important for all of us in the industry and it is why I take great pleasure in participating in it.

International Trade Prospects in Grain, Oilseeds, and Livestock

STEPHEN C. SCHMIDT

The value of U.S. agricultural exports after reaching a record $43.8 billion in fiscal 1980/81 declined for two consecutive years to a low of $34.8 billion in 1982/83. The forecast is that the value of U.S. agricultural exports in fiscal 1983/84 will rise to around $38 billion (Table 1). For fiscal 1984/85, U.S. agricultural exports may increase modestly in volume, but show a small decline in value.

As in previous periods, U.S. agricultural exports in coming years will be affected by 1) world economic conditions; 2) supplies in main importing and competing exporting countries; 3) trade policies of importing and exporting countries; 4) availability of foreign exchange; and 5) strength of the U.S. dollar and, hence, competitiveness of U.S. farm commodities.

The short-term outlook for world economic conditions is low inflation and relatively slow growth. World economic growth will be slower in 1985. World economic growth in 1985 is projected to slow to 2.7 percent from about 4.5 percent this year and that of the industrialized countries to 2-2.5 percent. The strength and duration of the current world recovery is open to question.

The pace of U.S. economic revival is expected to decelerate in 1985 and even out at about 3.2 percent - still somewhat above the 1972-1982 average of 2.2 percent. Over the same period growth in the European economies is expected to top out and hold in the 2-2.5 percent growth range. The pace of Japan's economic growth, will probably be faster than in other industrial countries, holding in the range of 3.75-4 percent in 1985. Soaring U.S. imports and the resultant $130 million deficit in 1984 have helped the rest of the world far more than foreseen.

Economic growth in the developing world is expected to average 3 percent. In the developing areas, the Asian countries, excluding the Philippines, show the most promise with growth averaging 6 percent.[1] The Latin American nations are likely to experience some slow improvement, with the growth of average real output advancing from 0.5 percent in 1984 to 2.7 percent in 1985 and 4 percent in 1986. The Middle Eastern region, with oil prices remaining flat should experience a moderate 4 percent economic growth. Africa, the weakest region in the world, may experience a 1.5-2 rate of growth. The East European economies are expected to pick up slightly on average over the next few years.

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1. South Korea, Taiwan, Hong Kong, Singapore, Thailand, Malaysia, and Indonesia.

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Table 1--Agricultural exports: Value by commodity 1982/83-1983/84

Commodity	Fiscal years 1/ 1982/83	1984 Forecast
	-Billion Dollars-	
Grains and feed	15.2	17.0
Wheat & products	6.2	6.3
Rice	.9	.9
Coarse grains 2/	6.6	8.4
Corn 3/	5.7	7.1
Oilseeds and products	8.9	9.1
Soybeans	5.9	6.1
Soybean cake and meal	1.4	1.2
Soybean oil	.5	.6
Livestock products	3.0	3.4
Poultry & products	.4	.4
Dairy products	.3	.4
Horticultural products	2.7	2.6
Tobacco	1.5	1.5
Cotton & linters	1.7	2.4
Other	1.3	1.2
Total	34.8	38.0

Table 3--U.S. Agricultural exports: Volume by commodity 1982/83-1983/84

Commodity	Fiscal Years 1/ 1982/83	1984 Forecast
	-Million metric tons-	
Wheat	36.7	37.7
Wheat flour	1.5	1.2
Coarse grains 2/	53.8	55.4
Corn 3/	47.1	47.0
Feeds, ingredients & fodders	7.0	7.0
Rice	2.3	2.0
Soybeans	24.5	20.6
Soybean cake & meal	6.5	4.9
Soybean oil	.9	.8
Sunflowerseed	1.4	1.0
Sunflowerseed oil	.2	.2
Other oilcakes & meals	.2	.3
Beef, pork & variety meats	.4	.4
Poultry meat	.3	.2
Animal fats	1.4	1.4
Tobacco	1.2	.2
Cotton & linters	1.4	1.5
Horticultural products	3.0	2.9
Other	3.2	3.3
Total	144.7	141.0

1/ Year Beginning Octoberl; 2/ Includes corn, oats, barley, sorghum, rye, and mixed grains.
3/ Excludes products.

Constraining the growth of demand for U.S. food and agricultural exports are 1.) the slow pace of economic growth in most of the rest of the world; 2.) debt problems in many developing countries; 3.) continued strength of the U.S. dollar; 4.) subsidized competitor exports, especially from the European Community (EC).

The prospects for growth in the export earnings of the developing countries is weak. Many of the middle-income developing countries are deep in debt. So the extra export earnings they get from higher primary commodity prices are used to service debt rather than to buy new machines from abroad. Shortage of foreign exchange, for example, has forced Mexico and Brazil and the East European countries to reduce demand for agricultural imports. At the same time, the fall in the international price of oil is depressing demand from oil-exporting countries. Their share of world imports and exports declined from a peak of 15 percent in 1980 to 12 percent in 1982 and is expected to diminish further in 1984.

The restrictive policies required to deal with the debt problems and without a strong upsurge in export earnings will force many developing countries to restrict their farm imports in the years ahead. What course international lenders take with regard to debt servicing will have a major impact on commodity trade. Debt adjustment policies under consideration provide no cause for great optimism for a strong increase in total world demand for agricultural products in 1984/85.

Import restrictions by developing countries hits debtor countries especially hard. According to the General Agreement on Tariffs and Trade (GATT) estimates 30-40 percent of exports from oil-importing developing countries are subject to some form of restraint in the markets of rich countries. These restrictions on imports in the United States, Europe and Japan are provoking debtors to impose controls on imports of their own. Most forecasters predict that the U.S. dollar will remain strong in 1984. However, in 1985 and thereafter, the U.S. dollar is expected to decline. If the dollar falls relative to other currencies, the value of our exports will rise because of both price and volume effects.

GLOBAL COMMODITY MARKET SITUATION AND OUTLOOK
OILSEEDS AND THEIR PRODUCTS

Oilseed Production

The outlook at this point is for world oilseed production to increase to 185.9 million tons in 1984/85, 13 percent more than in 1983/84.[1] This large increase in global output is due to prospects for record or near-record outputs for every major oilseed except flaxseed and copra (Appendix Table 1).

The current outlook for world oilseeds is dominated by the 17.7 percent increase in the global soybean crop. Total production of soybeans is forecast at 93.8 million tons accounting for 50.5 percent of prospective 1984/85 world oilseed output (Table 2). The 1984/85 world cottonseed and product situation will likely see a significant improvement. World cottonseed production in 1984/85 is expected to rise to 30.6 million tons, up 12.1 percent from a year earlier. Cottonseed constitutes the second largest oilseed crop representing 16.5 percent of world output (Appendix Table 1).

1. The world production of oilseeds, even though increasing faster in the last decade than for grains, is still only about 12 percent the level of grain crops.

Table 2. Shares of Oilseeds, Protein Meals and Vegetable and Marine Oils in Total Production and Exports, 1984/85.

	Oilseeds	
	Production	Export
Soybean	50.5 %	79.7 %
Cottonseed	16.5	0.4
Peanut	10.7	3.3
Sunflowerseed	9.3	5.8
Rapeseed	8.2	7.6
Flaxseed	1.3	1.8
Copra	2.5	0.8
Palm Kernel	1.1	0.5

	Protein Meals	
	Production	Export
Soybean	60.0 %	71.6 %
Cottonseed	11.2	2.8
Rapeseed	8.4	3.1
Sunflowerseed	6.9	4.5
Fish	5.2	7.8
Peanut	4.8	2.5
Copra	1.4	3.0
Linseed	1.3	2.1
Palm Kernel	1.0	2.5

	Vegetable and Marine Oils	
	Production	Export
Soybean	29.6 %	24.9 %
Palm	14.8	30.3
Sunflowerseed	13.5	11.0
Rapeseed	11.1	6.8
Cottonseed	8.3	2.8
Peanut	7.6	2.4
Coconut	5.9	8.3
Olive	3.7	2.1
Fish	2.6	5.3
Palm Kernel	2.0	3.9
Linseed	1.5	1.9

Source: Appendix Tables 1-3.

World peanut production in 1984/85 will probably increase to 19.9 million tons, 5.3 percent over 1983/84. Large crops in the U.S., India, China and Indonesia are seen to contribute to the increase. Peanuts are the third most important oilseed crop, supplying 10.7 percent of world output in 1984/85. The 1984/85 sunflowerseed crop should rise to 17.5 million tons, up 11.6 percent from a year earlier. Sunflowerseed should account for 9.3 percent of world oilseed production in 1984/85. Major producers in descending order of importance are the USSR, Argentina, East Europe, and the United States.

World rapeseed production is forecast at 15.2 million tons for 1984/85, slightly less than the previous year's level. Rapeseed should provide 8.2 percent of 1984/85 world oilseed production (Table 2). China is the worlds's largest producer and user of rapeseed. Other major producers are Canada and India. World copra production in 1984/85 is expected to increase to 4.6 million tons, 9.5 percent over 1983.84. The Philippines is the leading copra producer accounting for more than 40 percent of the total. Indonesia produces about one-fourth of world output. Other important producing countries are India, Malaysia, and Sri Lanka.

Total production of flaxseed in 1984/85 is expected to rise to 2.5 million tons but still below the 1982/83 level. World production of palm kernels-essentially a by-product - in 1984/85 is forecast to remain at previous year's level of around 2 million tons (Appendix Table 1).

Larger oilseed supplies in prospects for 1984/85 will allow an estimated 5 percent boost in crush and a significant rebuilding of stocks. Record crushings, in turn, will yield increased protein meal and vegetable and marine oil outputs. Despite a near two-third increase in ending stocks, they will still remain well below 1982/83 levels (Appendix Table 1).

Trade in Oilseeds

The volume of world oilseed trade is expected to increase by about 400,000 tons to around 33 million in 1984/85 (Appendix Table 1). The rebound is seen to result from a prospective 600,000 ton gain in soybean exports which would more than compensate for reduced cottonseed, rapeseed, flaxseed and copra shipments. World exports of rapeseed are likely to post a decline in 1984/85 to 2.5 million tons from 2.6 million in the previous year. Canada and France are the largest exporters. Trade in sunflowerseed, the third ranking export category, are forecast to increase marginally over the preceding year's level to 1.9 million tons. Total peanut and palm kernel exports may hold at 1983/84's level. The U.S. is the world's largest peanut exporter and the EC the largest import market. Relatively little copra and palm kernels are traded on world markets (Appendix Table 1).

Protein Meal Production

Total protein meal production in 1984/85 is to expand 5.4 percent to around 100 million tons and total meal consumption is forecast to be up 5 percent, reaching a record 99.1 million ton level (Appendix Table 2). Soybean meal dominates the world protein meal markets accounting for roughly 60 percent of total production and consumption and providing about 72 percent of world exports in 1984/85. The 11.2 million tons of cottonseed meal is second to soybean meal, and the 8.4 million tons of rapeseed meal ranks third in world protein meal production. Sunflowerseed meal provides nearly 7 percent of the total and fish meal, somewhat

irregular in supply, contributes around 5 percent to the overall volume. Total world production of fish meal in 1984/85 has been forecast to reach a record 5.2 million tons, 2 percent above the 1983/84 level. Nine countries account for approximately 80 percent of world fish meal production.[1] World copra meal production in 1984/85 is forecast at 1.4 million tons, 9.1 percent over 1983/84. In the same period, world palm kernel output is expected to match the previous year's level of 1 million tons. Production of copra meal is concentrated in the Philippines and Indonesia (Appendix Table 2).

Trade in Protein Meals

World protein meal exports in 1984/85 are expected to recover from the depressed 1983/84 level expanding to 30.7 million tons (Appendix Table 2). Of this total, soybean meal is expected to account for 71.6 percent and fish meal for 7.8 percent (Table 2). World fish meal exports in 1984/85 are forecast at 2.4 million tons, just 100,000 tons above last year's volume. The members of the Fishmeal Exporter's Organization (FEO - Chile, Iceland, Norway and Peru) account for nearly two-thirds of exports. Chile is by far the largest exporter. Germany is the largest importer of fish meal and Taiwan the second largest. Fish meal imports are also high into Sweden and Yugoslavia. Sunflowerseed meal constitutes the third largest protein meal export category. Global exports are forecast at nearly 1.4 million tons, 13.8 percent above 1983/84. World rapeseed meal exports in 1984/85 are expected to stay at the previous year's level of 958,000 tons. India, Canada and the EC are the major exporters. EC trade is traditionally among member countries. Germany, the Netherlands and Denmark are the largest EC markets for rapeseed meal. Total peanut meal exports for 1984/85 are forecast at 756,000 tons, 61.5 percent above 1983/84. The bulk of the increase in world peanut meal exports resulted from India's exports to the USSR.

World cottonseed meal exports for 1984/85 are forecast at 855,000 tons, 16 percent over the previous year's exports. India is the world's largest cottonseed meal exporter and Denmark the world's largest importer of cottonseed meal. Total palm kernel meal exports in 1984/85 are expected to total 770,000 tons and copra 920,000 tons. The Philippines and Indonesia dominate the export market for copra meal while Western Europe provides the major market for copra meal.

Vegetable and Marine Oils

World production of vegetable and marine oils is forecast to rise about 6 percent in 1984/85 to 46 million tons. Of this total, soybean oil is expected to contribute about 13.6 million tons, nearly 30 percent of the total (Appendix Table 3).[2] Palm oil and sunflowerseed oil production, in particular, are expected to rise rapidly in 1984/85 totaling 6.8 million tons and 6.2 million, respectively. Malaysia dominates palm oil production with 58 percent of the world total. By far the second most important producer is Indonesia and Nigeria the third largest. Rapeseed oil production in 1984/85 at 5.1 million tons is expected to be slightly less than a year earlier. World coconut oil production in 1984/85 is forecast to

1. Japan is the largest producer of fish meal followed by Chile, the USSR, Peru and the United States.
2. It should be recognized that soybean oil, while being the most important vegetable oil, accounts for less than half of the total than for soybean meal. This fact is, as expected, given that a bushel of soybeans includes over four-times as much meal as oil.

rise to 2.7 million tons compared with 2.4 million in 1983/84. Of this, the Philippines and Indonesia normally account for about two-thirds of the total.

Significant quantities of oil crops are produced in almost all parts of the world thus the production of vegetable oils is more dispersed geographically than the production of protein meals. Total vegetable oil and marine oil consumption is expected to rise 3 percent reflecting a recovery from the very tight supply situation in 1983/84, particularly for palm oil (Appendix Table 3).

World trade in vegetable and marine oils in 1984/85 will likely see a significant expansion reaching a record 14.5 million tons. More than half of the 1.1 million ton increase in total exports forecast for 1984/85 will be provided by increased shipments of palm and coconut oils (Appendix Table 3). Malaysia, the leading exporter of palm oil, is forecast to supply about three-quarters of the 1984/85 total of 4.4 million tons. The developing countries of Asia, the Middle East and North Africa are the main markets for palm oil. World coconut oil exports for 1984/85 are estimated at 1.2 million tons, some 200,000 tons above the year earlier level. More than three-quarters of these shipments originate in the Philippines. The United States is the world's leading importer of coconut oil and Western Europe, particularly Germany, is the second largest importer.

Sunflowerseed oil is the third ranking vegetable oil export with 1.6 million ton volume forecast for 1984/85. Major exporters are the U.S., Argentina, South Africa and Eastern Europe. World rapeseed oil, olive oil, and fish oil exports in 1984/85 are forecast to remain at their previous year's level. World peanut oil exports in 1984/85 are expected to increase by 29 percent to 354,000 tons. Major exporters are Senegal, Sudan, Brazil, and Argentina. France is the world's largest importer of peanut oil.

SOYBEAN MARKET SITUATION

Production and Consumption Prospects

Much of the prospective growth in the world oilseed and product markets is expected to come from the expansion in the soybean sector. USDA estimates that world soybean production in 1984/85 will reach a record 93.8 million tons primarily resulting from a sharp increase in U.S. production (Appendix Table 1).

Most of the world's supply of soybeans is produced in just four countries. In 1984/85 the United States, Brazil, China and Argentina accounted for 92.7 percent of world outpus. The U.S. crop of 55.4 million tons equaled 59 percent of the forecast 1984/85 world production. Brazil, the second largest producer of soybeans in the world grew an estimated 16 million tons representing about 17 percent of world total. Chinese production should reach 9.8 million tons. China is the third major producer of soybeans, accounting for about 10 percent of world production.[1] Argentina, the fourth major producer of soybeans, is a relative newcomer to the world soybean picture. As a newcomer, however, its entrance has been quite remarkable and its role today is an important one. Argentina's production has grown from about 1 percent of the world total in 1976/77, when it only produced 695,000 tons, to over 6 percent in 1984/85, when its production is forecast to rise to around 5.8 million tons.

1. China was the second largest producer of soybeans in the world for some time. Production has remained relatively stable at 7.6 million tons between 1978 and 1980 and trended upward since then.

World crush of soybeans in 1984/85 is expected to rebound from last year. The bulk of the anticipated 3 million ton increase in soybean crush will come in Brazil and Argentina.[1] EC soybean crushings too are expected to show a small revival after declining during 1982/83. World soybean mean and soybean oil production is shared by a larger number of countries than is world soybean production. This arises, however, because soybean importers became soybean product producers as the imported soybeans are processed. World soybean meal output in 1984/85 is forecast at 60.0 million tons, 5.5 percent above the 56.9 million tons in 1983/84 (Appendix Table 2). U.S. soybean meal production is estimated to rise to 21.6 million tons, about one million tons above last year's level. The combined soybean meal production of the U.S. and Brazil is forecast to account for 53.5 percent of world soybean meal production.

World consumption in 1984/85 is forecast to rise from the much reduced 1983/84 level of 56.9 million tons to 59.3 million tons (Appendix Table 2). Slowing the pace of prospective growth in soybean meal consumption are programs to reduce milk production in the European Community and in the United States. Nevertheless, lower meal prices in relation to other feedstuffs would strengthen demand and encourage meal consumption.

Overall soybean oil production for 1984/85 is forecast at 13.5 million tons, 4.7 percent over the 1983/84 level (Appendix Table 3). U.S. soybean oil production in this period is forecast to rise from 4.91 million tons to 5.08 million. At 13.6 million tons prospective world soybean oil consumption is slightly above the previous year's level but still below 1982/83.

Trade Prospects

The export outlook for soybeans is not very bright. World soybean exports for 1984/85 are forecast at 26.3 million tons, only slightly above last year's level (Appendix Table 1). The United States, Argentina and Brazil are the major suppliers in world trade. Total U.S. soybean exports for 1984/85 are projected at 21.8 million tons but this volume still falls short of the high levels achieved during 1981-83 (Table 4). Much of the prospective U.S. export gains are attributable to reduced Argentine sales. The U.S. is by far the largest supplier of soybeans in the world market, providing 82.9 percent of the soybeans entering world trade in 1984/85. Nonetheless, the U.S. is also the residual supplier. The U.S. share of world soybean exports has varied from year-to-year reflecting, in large part, changes in foreign availabilities (Table 4).

Nearly all of th soybeans produced in the PRC are consumed within the country thus leaving small amounts for exports. In 1984/85 China is forecast to export around a half million tons of soybean meal. Argentina is the world's second largest exporter of soybeans and Brazil the third largest. In 1984/85 Argentina is expected to provide 7.2 percent of the world supply of soybeans moving into world markets. The two countries are the principal competitors of the United States in soybeans and key factors in world oilseed and protein meal markets.

The EC is the world's leading importer of soybeans taking nearly 40 percent of world soybean exports in recent years (Table 4). Within the EC, Germany is the largest buyer and the Netherlands ranks second. Mainly because of larger sunflowerseed and rapeseed crops EC imports are anticipated to rise only by 400,000

1. Brazil and Argentina have expanded crushing facilities and used a number of policies designed to promote the export of value-added products instead of the seeds.

Table 4. Trade In Soybeans, Soybean Meal and and Soybean Oil, 1983/84 and 1984/85.

	Exports			Imports	
	1983/84	1984/85		1983/84	1984/85
	(million tons)			(million tons)	
Soybeans			Soybeans		
U.S.	20.7	21.8	EC	9.5	9.9
Brazil	1.3	1.5	Eastern Europe	.6	.6
Argentina	2.8	1.9	Japan	4.7	4.8
EC	.1	.1	Spain	2.9	3.0
World	25.8	26.3	USSR	1.1	1.1
Soybean Meal			Soybean Meal		
U.S.	4.8	5.0	EC	10.9	11.5
Brazil	7.6	7.9	Japan	.2	.3
Argentina	1.8	2.3	USSR	1.2	1.5
EC	4.3	4.5	Eastern Europe	3.1	3.3
World	20.2	21.5	Spain	.2	.3
Soybean Oil			Soybean Oil		
U.S.	.8	.7	EC	.5	.5
Brazil	.9	.9	India	.6	.5
Argentina	.3	.5	Pakistan	.3	.4
EC	.9	.9	USSR	.2	.2
Spain	.4	.4	Mid-East/ N. Africa	.7	.7
World	3.5	3.5	Latin America	.4	.5

* Source: U.S. Department of Agriculture, FAS, Foreign Agriculture Circular Oilseeds and Products FOP 8-84, August 1984, pp. 13-15.

tons to 9.9 million in 1984/85.[1]

Asia provides the second largest market with Japan the chief buyer. Japan is now the world's largest single market for soybeans. Japanese soybean imports are forecast to increase slightly in 1984/85 to 4.85 million tons (Table 4). Taiwan and Korea are also expected to increase their soybean imports to 1.35 million tons and 810,000 tons, respectively. Elsewhere, Eastern Europe has been a comparatively small and volatile market for soybeans. The region's imports in 1984/85 may slightly decline to around 600,000 tons. Romania and Yugoslavia are the largest markets in the region. The buildup in Soviet livestock numbers points to a rise in requirements. Imports into the USSR are likely to rise above the previous years level of 1.1 million tons. Reducing the pace of import growth is an expected recovery in domestic oilseed production, up 3 percent from 1983/84.

Spain is the leading non-EC-West European market for soybeans. Spanish imports are expected to increase slightly in 1984/85 to 3.03 million tons (Table 4).

World ending stocks of soybeans in 1984/85 are projected at 15.5 million tons, up 39 percent from the low 1983/84 level, but still below the record set in 1982/83. The United States is expected to hold 6.8 million tons of the 1984/85 total.

Overall, the growth in trade in soybeans has benefited from 1.) increased demand for red meat and poultry; 2.) certain distinct and inherent advantages;[2] and 3.) liberal import policies. Soybean import demand has shown responsiveness to changes in their a.) own prices relative to prices of competing oilseeds (or meals); b.) livestock prices; and c.) price and availability of surplus commodities like wheat and nonfat dry milk.

World trade in soybean meal is forecast to increase by 1.3 million tons to 21.6 million in 1984/85 (Table 4). Imports into the EC, the major importer of soybean meal, are estimated to increase by 5.5 percent to 11.5 million tons. This increase is connected with some recovery in the use of soybean meal and a slight increase in EC meal exports. Dampening prospects for EC soybean meal imports are increases in production of field peas and beans in France. Moreover, pressure on the use of soybean meal in the EC can be expected from increased grain supplies. Soybean meal will have to compete heavily against grain to gain the projected 4 percent increase in consumption.

Imports into Eastern Europe, the second largest regional market, have stagnated since 1981/82. Imports are forecast to post a moderate recovery in 1984/85 but at 3.3 million tons are still around one million tons short of 1980/81's level. Soviet soybean meal imports, despite some increase in domestic oilseed meal production, are expected to register a small expansion in 1984/85, totaling about 1.5 million tons compared with 1.2 million a year earlier. This import volume, however, falls short of the 1982/83 import level by about one million tons.

Japan has not been a major market for meal, due mainly to the maintenance of a restrictive soybean meal import policy (Table 4).

1. The EC is forecast to increase sunflowerseed production by 29 percent and rapeseed production by 11 percent.
2. Soybeans, when compared with most oilseeds, have a relatively high meal to oil ratio and supply a high quality protein. Soybean protein has a highly desireable amino acid balance for all classes of livestock, particularly for poultry and hogs and has a high degree of digestibility.

Most of the prospective increases in exports will be supplied by foreign competitors. Brazil has assumed the role of the leading meal exporter supplying 7.9 million tons in 1984/85. These shipments represent 36.6 percent of world trade.

U.S. soybean meal exports for 1984/85 are forecast at 5.04 million tons, 4 percent above the previous year's depressed level (Table 4). At this export level the U.S. share of the meal market would be only 23.1 percent against 27.6 percent in 1982/83.

Foreign demand for meal, used primarily for feed, is shaped by factors operating in the feed-livestock economy. Livestock-feed prices and feeding techniques are related factors that exert a strong influence on demand. Constraining demand for protein meals this past year (1983/84) was poor livestock feed profitability. In 1984/85 utilization will again be affected by the profitability of livestock feeding determined by feed and livestock product prices. Forecast consumption and trade expansion is based on the expectation that feed prices will decline from year ago levels and thus result in improved feed profitability.

On the demand side for livestock products, consumption of meat is expected to increase in developed countries in response to a gradual improvement in economic conditions. Relatively strong economic growth in several Asian developing countries, particularly Taiwan, South Asia, Singapore and Malaysia, leads to increased demand for livestock products and thus for increased protein meal demand. Growth in meal consumption and hence in trade has been markedly affected by year-to-year shifts in the price ratio of protein meals relative to other sources such as feed grains, pulses and grain by-products. In countries where grain prices are high relative to those of meals, some substitution of grains by these is in evidence. This was often the case in the EC and Spain.

There is also a high degree of substitution among protein meals themselves. The relative price attractiveness and ample supplies of soybean meal since the 1970's have been important factors contributing to increased use and trade.

With record oilseed crops and expanded palm oil production, world exports of soybean oil are forecast to increase only slightly during 1984/85. Export prospects may also be influenced by smaller availabilities of competing animal fats. World exports are anticipated at 3.54 million tons compared with 3.47 million in 1983/84. Of the 1984/85 total the U.S. is seen to provide 658,000 tons, about 100,000 tons less than in the previous year (Table 3). Brazil and Argentina are expected to benefit from the prospective growth in demand. As with meals, Brazil also dominates the soybean oil export trade. In 1983/84, Brazil supplied about one-fourth of all soybean oil exports. The EC is both a substantial exporter and importer of soybean oil. EC soybean oil exports in recent years were only slightly below the level of Brazilian shipments (Table 4).

On a regional basis the Middle EAst and North Africa provide the largest market for soybean oil. India is currently the largest country importer followed by the EC.

Overall the demand for vegetable oils varies from country to country and the extent to which this demand is satisfied with soybean oil depends upon 1.) local tastes and cooking habits; 2.) relative prices of competing oils and fats; and 3.) degree of price protection afforded to local oilseed producers.

Current per capita consumption of food fats and oils is generally depressed in

developing countries because of limited consumer buying power, and unfavorable balance of payments. In contrast, per capita consumption in the developing countries is already high and consumption has been expanding slowly. Underlying the leveling off in per capita consumption of edible oils and fats are 1.) the low income elasticity of demand for these products and 2.) changing eating habits.[1] The demand for edible oils tends to be more inelastic with respect to price than for meal. Because of its small impact on consumer budgets, price increases of edible oils do not much affect oil sales in the retail markets of the developing countries. The relative inelasticity of demand for vegetable oil has kept world consumption from declining despite high prices.

Because of the high substitutability among oils and fats, the volume of individual oils and fats consumed has shown considerable yearly variation in response to changing price relationships. Both consumers and edible oil processors tend to change the mix of their edible and/or products purchases in a way that minimizes expenditures and costs.

Peanut, rapeseed, cottonseed and sunflowerseed oil are the major competitors of soybean oil in Western Europe.

GRAIN MARKET SITUATION: PRODUCTION AND CONSUMPTION PROSPECTS

Total Grains

World grain production (wheat, rice and coarse grains) for 1984.85 is forecast at 1.59 billion tons, an increase of 7 percent on last year. The increase reflects record food grain and coarse grain crops. Larger harvests in the U.S., Western Europe and China contributed most to the growth in global grain output (Table 5).

The EC is forecast to harvest a record grain crop of 144 million tons in 1984/85, 16 percent larger than last year's drought reduced crop and a 9 percent increase from the previous record harvest in 1982/83. China too is facing a record grain harvest of more than 296 million tons, compared with 289 million tons in 1983/84 [2] (Table 5).

Soviet 1984/85 grain output is estimated at 164 million tons, 31 percent below target. The Soviet national plan for 1984/85 has set 238 million tons as the target for grains.[3]

Utilization of all grains is expected to be up by more than 30 million tons, to a record 1.58 billion tons. Over half of the increase will be accounted for by coarse grains, the bulk of the rest by wheat with rice output marginally above last year's crop (Table 5).

1. The income elasticity of demand in developing countries for fats and oils is .55 and for vegetable oils .56; the corresponding estimates for developed countries are .14 and .05, respectively. These coefficients indicate the percentage change in consumption associated with a 1 percent change in income.
2. Includes rice, wheat and coarse grains. Soybeans, pulses and miscellaneous grains are included in China's definition of total grains, which gives a total output of 404 million tons.
3. The Soviet record of 237 million tons was harvested in 1978 and annual output has been near or below the 200 million figure since then.

Table 5. Total Grains, Rice and Wheat: Production, Consumption, Net Trade and Ending Stocks, 1983/84 and 1984/85.

	1983/84				1984/85			
	Production	Consumption	Net Trade	Ending Stks	Production	Consumption	Net Trade	Ending Stks.
	(million tons)				(million tons)			
Total Grains								
World	1,483.7	1,547.5	204.4	188.3	1,592.6	1,580.6	213.3	200.4
U.S.	205.7	180.7	95.6	71.3	307.8	191.0	105.6	83.1
EC	124.4	117.1	10.7	14.3	144.5	121.7	16.7	20.3
USSR	183.0	209.5	-31.5	n.a.	164.0	210.0	-44.0	n.a.
Rice								
World	305.4	305.4	12.4	16.9	307.5	307.7	11.9	16.8
China	114.9	117.7	.6	n.a.	114.9	117.8	.6	n.a.
India	60.2	56.8	-.7	6.0	58.5	57.3	-.4	6.5
Thailand	12.8	9.3	4.3	.8	12.6	8.0	4.0	.8
U.S.	3.1	1.8	2.2	1.5	4.2	2.0	2.2	1.8
EC	.7	.7	0	n.a.	.7	1.0	-.2	n.a.
Wheat								
World	488.7	484.2	101.6	101.3	499.6	498.7	103.0	102.2
U.S.	65.9	30.3	38.9	37.9	70.0	29.0	41.5	37.5
EC	59.3	49.4	12.9	8.8	72.3	51.7	14.8	14.3
China	81.4	91.4	-10.0	n.a.	84.0	95.0	-11.0	n.a.
USSR	78.0	95.0	-20.0	n.a.	78.0	101.0	-23.0	n.a.
E.Europe	35.4	37.1	-2.2	n.a.	37.6	38.8	-1.6	n.a.
Canada	26.6	5.9	20.4	9.0	20.5	5.3	16.2	7.0
Australia	21.8	3.3	11.6	7.5	16.5	3.4	15.0	5.6
Argentina	12.0	4.5	9.6	.6	9.7	4.5	6.0	.5
India	42.5	43.3	-2.5	11.5	65.9	n.a.	0	n.a.

* Source: U.S. Department of Agriculture, FAS, Foreign Argriculture Circular. Grains. FG-12-84, September 1984, pp 8-11.

Global grain trade in 1984/85 will likely see a 10 million ton improvement to 213 million tons after increasing only slightly in 1983/84 (Table 5). Ending grain stocks could be up 7 percent in 1984/85, with most of the increase in the U.S. Virtually all of the anticipated increase in world stocks will be in coarse grains, as wheat and rice stocks are expected to change little from their 1983/84 level (Table 5 and Table 6).

Rice

World rice production in 1984/85 is placed at a record 307.5 million tons (milled basis) with consumption expected to be slightly ahead. About 90 percent of the world's rice crop is produced in Asia. In 1984/85 China, India and Indonesia together are forecast to account for two thirds of world rice production (Table 5). World trade in 1984/85 shows lack of strong demand for rice and prospects are that volume will remain essentially unchanged from 1983/84 level. Thailand is the world's largest rice exporter and the U.S. the second largest. The major importers are the EC, Indonesia, Nigeria and Iran. Import demand in many developing countries will continue to be limited by the lack of foreign exchange.

Wheat

In 1984/85 world wheat production is set to reach a new record of 499.6 million tons, 11 million tons higher than last year's production (488.7 million tons). The growth reflects record wheat harvests in the EC, India, Spain and China and good outturns in the U.S. and Eastern Europe. Wheat output alone in the EC will climb 22 percent to 72 million tons in 1984/85. China has reaped a 84 million ton bumper harvest while the Indian wheat crop is estimated to be 44.6 million tons (Table 5).

The prospective increases are expected to more than compensate for smaller harvests anticipated in the major exporting countries, notably Canada, Australia, and Argentina. The 1984/85 Canadian wheat crop is estimated at only 20.5 million tons, 6 million tons less than the previous year's record production of 26.6 million tons. The USSR has experienced another poor harvest estimated at 78 million tons. The crop was about the same size as in 1983/84 (Table 5).

World utilization of wheat in 1984/85 is forecast to be 498.7 million tons, about 14 million tons more than last year. Increased consumption of wheat is predicted in the EC, the USSR, and China. Because world utilization of wheat is expected to be less than world production, world wheat stocks could increase in 1984/85 by about one million tons to 102.2 million tons (Table 5).

Coarse Grains

World production of coarse grains in 1984/85 is forecast at a record 785.8 million tons, 14 percent higher than last year's crop (689.5 million tons). Of major importance to this increase are the return to a more normal crop in the U.S., a record EC coarse grain outturn of 72 million tons and larger corn crops in South Africa and Thailand (Table 6). Barley contributes nearly 42 million tons to EC's 1984/85 coarse grain output.

In the USSR and Australia, 1984/85 coarse grain crops will be considerably smaller than in the previous year. Total USSR production of coarse grains,

Table 6: Coarse Grains, Corn and Barley: Production, Consumption, Net Trade and Ending Stocks, 1983/84 and 1984/85.

	1983/84				1984/85			
	Prod.	Consump.	Net Tr.	End. Stk.	Prod.	Consump.	Net Tr.	End. Stk.
	(million tons)				(million tons)			
Coarse Grains								
World	689.5	757.9	90.0	70.1	785.4	774.2	98.4	81.3
U.S.	136.7	148.6	54.4	31.9	233.4	159.9	61.9	44.0
EC	64.0	67.6	-1.6	5.5	71.2	69.0	1.6	6.0
USSR	105.0	114.5	-11.5	n.a.	86.0	109.0	21.0	n.a.
E. Europe	67.3	68.2	-.8	n.a.	67.5	68.0	-.6	n.a.
Canada	21.0	18.5	5.1	4.6	21.9	n.a.	3.7	n.a.
Argentina	18.3	6.9	12.0	.3	18.5	7.0	11.2	n a.
Japan	.4	18.8	20.5	2.0	.4	19.0	21.2	n.a.
Corn								
World	349.1	410.6	59.6	34.2	439.0	431.5	66.9	41.7
U.S.	105.8	120.4	46.6	18.1	191.8	130.8	53.3	25.8
EC	19.5	24.0	4.5	n.a.	19.5	24.0	4.5	n.a.
USSR	16.5	24.2	10.0	n.a.	13.3	29.7	15.4	n.a.
Japan	0	14.6	14.6	1.3	0	14.4	14.8	n.a.
Barley								
World	170.0	175.3	14.8	15.0	167.7	165.6	16.3	17.1
EC	36.1	34.0	1.9	n.a.	41.7	36.0	5.0	n.a.
Canada	10.3	8.0	4.5	2.0	10.2	6.9	3.2	1.9

* Source: U.S. Department of Agriculture, FAS, Foreign Agriculture Circular, Grains FG-12-84, September 1984, pp 10-14.

including miscellaneous grains, is estimated to have dropped to 86 million tons while Australia's coarse grain production is estimated at 7.8 million tons. In Eastern Europe, output of coarse grain production is estimated to have reached a level similar to that of 1983/84 (Table 6).

World coarse grain consumption is expected to reach 774.2 million tons, a new record, and since production is expanding at a faster pace, we should see a certain rebuilding of global stocks. Global ending stocks could rise from the present low level of 70.1 million tons to 81.3 million tons. Of this about 44 million tons are expected to be held in the U.S. (Table 6).

Corn is by far the most important coarse grain and has been responsible for the growth in world coarse grain output. For 1984/85 world corn output is projected at 439 million tons, about 90 million tons above 1983/84. The outstanding feature of world corn production is the dominance of the U.S., providing about 44 percent of prospective global 1984/85 output (Table 6).

Global utilization is to rise from 410.6 million tons in 1983/84 to a forecast 431.5 million tons in 1984/85. With prospective production exceeding utilization world corn stocks are expected to be partly replenished from the abnormally low 1983/84 level (Table 6).

TRADE PROSPECTS

Wheat

World trade in wheat is projected to remain at around last year's level of 102 million tons. The U.S., the world's leading exporter, will probably experience an increase in exports expected to reach 41.5 million tons (Table 5). This would represent an increase of 2.6 million tons from last year (38.9 million tons) with the largest gains in Hard Red Winter wheat.

The prospective gains in U.S. wheat exports are due to expectations of lower export availabilities in Canada and Argentina and substantial increase in Russian, Bangladeshi and Pakistani import requirements. According to Canadian outlook reports, a significant reduction in Canadian wheat exports can be expected in 1984/85. An export volume of about 16.2 million tons is anticipated, approximately 4 million tons below 1983/84 (Table 5). Argentine wheat exports are forecast to fall to 6 million tons from 9.6 million tons.

USDA analysts have estimated Soviet wheat import requirements at 24 million tons during 1984/85. This would be nearly 4 million tons more than last year. Because of drought conditions wheat imports into several Near Eastern countries (Iran and Syria) and Africa will probably be considerably above 1983/84 levels. China, the second largest wheat importer, will, at best, import the same amount of wheat in 1984/85 than the year before because of increased domestic wheat production. China has in 1984/85 reaped its fourth consecutive record wheat harvest and doesn't need to purchase additional supplies. Usually, China secures its imports under long-term agreements with various exporting countries (Argentina, Australia, Canada and the U.S.). China is committed to buying 6 million tons of U.S. grain annually, but its purchases in 1983 fell 2.2 million tons short of the

stipulated level.[1] The 2.2 million ton amount was carried over into 1984, leaving the assumption that 8.2 million tons would be bought this year. So far in 1984, China bought only 4.2 million tons of U.S. grain and it has threatened to retaliate against the new U.S. textile controls which took effect September 7, 1984.[2] China's grain purchase agreements with the U.S., Australia and Argentina expire at the end of 1984, while its grain pact with Canada ends in June 1985. On present indications China has no interest in renewing any of the current grain agreements.

The Latin American region, India, Eastern Europe and the EC are expected to import less wheat in 1984/85 than in the previous year. Requirements, however, are expected to increase in Brazil approximating at least 4.5 million tons (Table 5)

Total EC wheat imports are forecast to decline from 3.1 million tons in 1983/84 to 2.7 million tons in 1984/85 while total wheat shipments are forecast by the Council at 17.5 million tons, compared with 16 million tons in 1983/84. These figures cover common wheat and flour, durum, and deliveries of 1 million tons of food aid. The EC has a voluntary limit of 14 million tons per year on its soft wheat and flour exports aided with export subsidies. The dollar's strength permits the EC to export wheat without any export subsidy which do not count against the voluntary limit.

The projected 1984/85 EC wheat imports of 2.7 million tons is the lowest level since the Community's formation. The bulk of imports consist of high protein hard and durum wheats used for blending and which come primarily from the U.S. and Canada.

Coarse Grains

World coarse grain trade in 1984/85 is expected to recover from its depressed 1982-84 level but at 98.4 million tons it still will remain below the 1980/81 volume (Table 6). Inhibiting any appreciable expansion in global coarse grain trade are continued substitution of feed wheat for coarse grains in developed areas and balance-of-payments constraints in many developing countries.

Most of the prospective increase in world exports is attributable to increased U.S. exports, forecast at 62 million tons for 1983/84. Shipments by major foreign exporters are expected to stay at their 1983/84 level. Expansion of Soviet import demand provided the main impetus for the growth in world coarse grain trade. Soviet import demand for 1984/85 is forecast at 21 million tons, up 8 million tons from 1983/84. Record livestock numbers and poor feed grain and forage crops are forcing the Soviets to cover its requirements with purchases from all the major exporting countries, and from small suppliers.

Continued strong demand in major Asian markets is anticipated, particularly in Japan, Taiwan and Korea. Japanese import demand for coarse grains will expand in 1984/85 reaching over 21 million tons. Prospects are for a modest revival in East European domestic coarse grain use which, in turn, could raise import require-

1. In 1983, China halted purchases of U.S. wheat, soybeans and cotton when the two countries could not agree on terms of textile trade, and even after a textile pact was signed, China did not fulfill the annual minimum purchase obligation.
2. The regulations prevent a country from circumventing U.S. import quotas by shipping mostly completed garments to another country to be assembled before shipments to the U.S. Finished textile products will be charged against China's quotas instead of the country port of shipment.

ments in 1984/85 by about one million tons, to 4.4 million tons (Table 6).

The EC is projected to become a net exporter of coarse grains in 1984/85 (Table 6). Imports of coarse grains by the EC in 1984/85 are expected to continue their long-term decline, down from over 23 million tons, to a forecast level of 5 million tons. With prospects for a large barley harvest and efforts to expand feed wheat usage at the expense of domestic feed grains, pressures could intensify to export up to 5.5 million tons of barley.

China is expected to increase its 1984/85 coarse grain imports to 1.1 million tons posting a substantial expansion over the previous year's level. Nonetheless this import volume is less than half the 1982/83 amount.

World trade in corn is forecast to increase to 66.9 million tons compared with 59.6 million in 1983/84. Most of the increase in trade will be supplied by the U.S. with shipments totaling 53.3 million tons. This puts forecast U.S. exports 6.7 million tons above 1983/84. Reports of lower-than-expected corn production in France could mean additional EC import demand for third-country exporters like the United States in 1984/85. U.S. corn shipments to the EC have steadily declined from over 16 million tons in 1976/77 to about 3 million tons in 1983/84. U.S. corn is generally favored for industrial uses (Table 6).

World barley trade in 1984/85 is expected to rise over 16 million tons, 1.5 million tons above last year's level. The increae in exports is due to a significant increase in EC's exportable supplies turning the Community into the world's largest barley exporter. Canada and Australia will see a significant drop in their export volumes due to crop shortfalls. Increased imports by Saudi Arabia and the USSR are expected, while Japanese and East European imports of barley should remain close to last year's level (Table 6).

MEAT MARKET SITUATION: PRODUCTION, CONSUMPTION AND TRADE PROSPECTS

Production

World meat production in 1984 is estimated at 107.7 million tons, 1 percent above 1983; production is forecast to grow another 1 percent in 1985 totaling 108.5 million tons. The 1985 increase in meat supplies will come from continuing expansion in poultry production. Beef is the principal meat category accounting for 38.8 percent of world meat production and pork for 34.6 percent.

Consumption

Demand for meat in 1984 has risen in response to recovery in the world economy, particularly in Latin America and Africa. In North America and Western Europe, where consumption is already high and population growth rates are low, little change is recorded. Average EC per capita consumption stagnated at around 89 kilograms. In general, consumption of mutton and lamb increased in the EC at the expense of beef.

U.S. per capita consumption in 1983 averaged 95 kilograms compared to 92 kilograms the previous year. Japanese consumption at 31 kilograms is still well behind that in the industrial countries. Consumption in the USSR increased to 63 kilograms from 60 kilograms in recent years thanks to higher domestic production.

Consumption is likely to stagnate in Eastern Europe due to inadequate supplies and high consumer prices.

Consumption throughout the world is expected to concentrate more on pork and poultry meat in 1985 which could result in stagnation in the beef sector.

Trade

World trade in meat in 1985 is forecast to increase only slightly. Growing domestic production is likely to lead to a fall in imports by the USSR, the EC and North America, while the other countries of Eastern Europe and Africa, south of the Sahara, will continue to need to limit purchases because of economic difficulties.

Oil revenues and growing demand for red meats, in particular on the part of foreign workers, have made the countries of the Middle East and North Africa an important outlet for meat exports. Meat import requirements are expected to continue to rise in 1985 and beyond. Despite growing home production, several Far Eastern countries still need to import meat and no significant change is likely in the near future.

BEEF AND VEAL

Production

World beef and veal production may increase about 2 percent in 1984 to 41.8 million tons. Much of the increase in 1984 is expected to occur in the EC and the USSR.

Production of beef and veal in the Community is very closely linked to milk production. Indeed, some 80 percent of all the beef and veal produced in the EC comes from herds which produce both milk and meat. One can not, therefore, examine what is happening in the beef-veal sector without noting developments on the dairy side. The marked rise in EC beef supplies (cow beef) in 1984 is closely attributable to changes in the common dairy policy which provides for the imposition of quotas at levels approximately 7 percent below 1983 milk production.

Mexico, Eastern Europe and Argentina have also experienced increases in beef and veal output during 1984 while Australia recorded a reduction in output. Beef and veal production in 1984 in the U.S., Canada, New Zealand, Uruguay and Japan is expected to remain at previous year's level. Apparently, the impact of the U.S. dairy program announced in the Fall of 1983 on actual cow slaugher has not been as great as initially anticipated.

World beef and veal production in 1985 is anticipated to be virtually the same as in 1984 or possibly could even slip below the 1984 level. In contrast, indications point to production advances in the USSR, Brazil and Mexico and to declines in the EC and the U.S. Little or no change in production is projected in Canada, Eastern Europe, Argentina, Australia and New Zealand.

By far the sharpest increase in production, 4 percent, is projected for the USSR totaling 7.5 million tons. Production in the EC is projected to drop 3 percent to 7.1 million tons primarily because of the introduction of dairy quotas and

resultant earlier cow slaughterings.

Consumption

Many factors affect the demand for meat and consumer preferences for meat. Historical spending patterns indicate that in periods of weak income growth demand for beef tends to be sluggish relative to other meats, notably poultry. That is, the demand for beef has been found as more income sensitive than the demand for poultry meats.

Meat consumption in the EC is generally accepted as having reached saturation levels, particularly in the case of beef. High prices have encouraged increased beef and veal production and dampened consumption. Although production has steadily increased, consumption has declined in recent years. The EC has long gone beyond the point of self sufficiency in beef, reaching 108 percent (excluding veal) in 1983/84. Any growth in meat expenditure in recent years has been largely on cheaper pork and poultry products. However, beef consumption does respond to relative price changes and an increase in beef supplies and lower prices could favor a resumption in the growth of beef consumption. Hence, any changes in the level of beef consumption will be as a result of changes in the relative prices of beef and other meats. Prices also influence the pattern of meat usage in the processing sector. France is the Community's largest beef market followed by Italy, Germany and the U.K.

Trade

World beef and veal exports are forecast to rise to around 5 million tons in 1984 mainly because of larger shipments from the EC and Brazil. Export availabilities in other supplier countries, notably Australia, Argentina and New Zealand, are expected to be lower than a year earlier.

The EC became a net exporter of beef and veal for the first time in 1980, and is likely to stay in such a net position, at least for the foreseeable future. Exports of beef and veal from the EC are expected to increase to 650,000 tons in 1984 while imports are expected to fall. As a result, the EC could even become the world's largest beef exporter in 1984 and remain one of the largest exporters in years ahead. Historically, Australia has been the world's leading exporter of beef shipping about 672,000 tons in 1984. Argentina has been the second or third largest exporter of beef. Argentine beef exports were 522,000 tons in 1982, 420,000 tons in 1983 and are estimated to amount to between 350,000 and 375,000 tons in 1984. The EC's accumulation of enormous stocks of meat was charged as threatening the development of Argentina's beef industry. To stimulate beef exports Argentina removed the export duty effective October 1, 1984.[1]

U.S. beef and veal exports have risen about 13 percent in 1984 to about 105,000 tons, partially because of the new beef agreement with Japan. Japan, the largest market for U.S. beef will increase its quota for high-quality beef from its current level of 30,6000 tons during the next four years to 58,400 tons.

The U.S. is by far the world's largest market for beef. U.S. imports of beef and veal for 1984 are expected to be around 860,000 tons, or slightly below the

1. At the same time the two meat-free days a week in restaurants are to be retained so as to give no additional impetus to domestic consumption.

1982/83 level.[1] The U.S. Meat Import Act limits imports of fresh, chilled and frozen beef, veal, mutton and goat meat to adjusted quotas based on average 1966/67 meat imports. Imports cannot be restricted below 1.25 billion pounds. The law provides that a trigger point of 110 percent of the adjusted quota of imports be computed. The 1983 trigger point was 1.231 billion pounds (110 percent of the adjusted import quota of 1.119 billion).

The EC is the second largest beef import market with Italy and the U.K. the major country buyers. EC imports from third country suppliers have hovered around 250,000 - 400,000 tons for several years. Roughly one-third of all beef-veal coming into the Community is under concessionary schemes. Another 20 percent or so of EC beef-veal imports are manufacturing grade beef. The EC has a 29,800 tons quota for high quality beef of which the U.S. share is 10,000 tons.

Import demand for beef in the USSR is expected to remain high in 1984; imports by the USSR are expected to be about 420,000 tons, 170,000 tons above 1983 levels. The USSR is likely to continue to maintain the 1984 beef import level in an effort by the leadership to appease consumers.

PORK

Production

World pork production is expected to remain relatively stable in both 1984 and 1985 at around 37.6 million tons. Lower U.S. output should be compensated by gains in the EC, Eastern Europe and the USSR. EC pork production is to grow as a result of reduced hog inventories whereas growth in Soviet and East European pork production is sustained by record inventories. The EC production levels, second only to China in output, will likely increase only marginally. In Eastern Europe, most rebound is expected in Poland after four years of decline.

Trade

World pork trade is not likely to expand in 1985 and approximate the 1984 level of 3 million tons. The EC is the world's leading pork exporter but the bulk of trade is among member countries. Denmark and the Netherlands are the major exporting nations. Pork exports from the EC should gain in 1984 as importers have lifted their ban on Danish exports.[2]

Exports of fresh and frozen pork account for over 80 percent of total U.S. pork exports. Japan is the largest market for U.S. pork and pork variety meat exports and Canada the second largest market for pork. Pork variety meats make up the bulk of U.S. pork exports to the EC which provides the second largest outlet for this meat category.

Germany, the U.S., Italy, France and Japan are the major pork import markets. The U.S. is a net importer of pork. Traditionally, processed pork (primarily canned hams and shoulders from Europe) was the major U.S. pork import category.

1. Through the 1960-1982 period, U.S. beef and veal imports averaged 7.6 percent of U.S. production and 7.2 percent of U.S. consumption.
2. Because of the outbreak of foot-and-mouth disease in March 1982, imports from Denmark were banned until September 1, 1983.

In recent years fresh or frozen pork and live hogs, primarily from Canada, have gained in importance and accounted for more than half of total U.S. pork imports.

While pork imports to Japan have fluctuated widely from year to year, they have generally been on the rise. Imports reached a record high in 1981, followed by a sharp decline in 1982 and leveling off in subsequent years. Canada was the major exporter of pork to Japan followed by the U.S. and Denmark.

POULTRY MEAT

Production

World poultry meat production is expected to expand 1.6 percent in 1984 despite a decline in Brazilian and French output (the third and fourth largest single country producers). France is the EC's largest producer followed by Italy and the U.K. Brazilian poultry meat production is expected to drop by 1.49 million tons in 1984, 6 percent below 1983. Declining export prospects, higher production costs along with weaker domestic demand are the main factors behind the forecast decline in Brazilian and French output during 1984. Production in Hungary too may decline in 1984 as a result of unfavorable export markets.

World poultry meat production may show a 3 percent increase in 1985 totaling 24.4 million tons as each of the large producers are forecast to expand production. The U.S. is expected to lead the advance in 1985's poultry meat production with output reaching about 7.68 million tons, 4.6 percent above the 1984 level. Production in Brazil and Hungary is forecast to recover in 1985, output, however, is not expected to reach 1983 levels. After years of steady growth, output has more than doubled since 1978, Brazilian poultry meat production expansion would seem to be coming to an end. Marked expansions are anticipated in the USSR, North Africa and the Near East. Soviet production should reach 2.7 million tons, while the Five Year Plan provides for an even greater growth rate. Hungary's poultry meat production is expected to grow about 2.8 percent to 370,000 tons. Iran is the principal poultry producer in the Third World with output possibly reaching 250,000 tons in 1985. All countries in North Africa and the Middle East have steadily expanded production in recent years. Estimates for 1985 point to this trend continuing.

Consumption

Most of the anticipated growth in output is likely to go into domestic consumption rather than exports. There is evidence that at given incomes and prices, demand has shifted away from beef in favor of poultry products during the last decade. Indeed chickens have gained an increasing share of meat consumption. Positive trends are reported in EC broiler consumption. In 1983 consumption reached about 14.3 kilograms (kg) per capita, 0.6 kilograms more than in 1980. Highest consumption was in Italy (17.4 kg) followed by France (17.3 kg), Greece (16.3 kg) and Ireland (16.1 kg). U.K. and Belgium-Luxembourg come about midway.

Worldwide, Israel with 41.4 kilograms has the highest per capita intake of poultry meat and the U.S. with 29.8 kg the second highest. Apart from Israel, considerable growth rates have been recorded in the Middle and Near East. Saudi Arabia, Kuwait and Bahrain have the highest per capita consumption at over 25 kg. Poultry meat is also popular in Asia with per capita consumption at 26.7 kg in

Hong Kong and 17 kg in Taiwan. Per capita consumption at 11.5 kg is still low in Japan and even lower in the USSR (10.5 kg).

Trade

World poultry meat exports have at 1.7 million tons stagnated in 1984 and are forecast to increase only marginally in 1985. Slowing the growth in exports is the reaching of saturation point on the world market as important customers in North Africa and the Middle East are trying to increase their own production.

The EC is the world's largest exporter of poultry meat and Brazil the second largest. The U.S. ranks third as poultry meat exporter but its shipments have been declining since 1982. With over 400,000 tons France accounted for nearly half of EC poultry meat exports in 1984. Next comes the Netherlands with 290,000 tons.

Both EC and Brazilian poultry meat exports in 1985 are likely to stay near their 1984 level. U.S. poultry meat exports are forecast to continue to drop in 1985 due to strong competition from other major suppliers, such as the EC, Brazil, and Hungary. In 1983, about 75 percent of U.S. poultry meat exports were chicken parts. Brazilian and French trade is mainly in whole chickens. U.S. broiler exports have suffered the largest losses. Exports of whole turkeys and turkey pieces also fell sharply. The USSR is the world's largest poultry meat buyer with imports of around 260,000 tons. Germany is the second largest poultry meat importer and Saudi Arabia the third largest. Japan imports around 110,000 tons of poultry meat and ranks fourth among importers. The top markets for U.S. chicken parts are Japan, Hong Kong, Singapore and Canada, whereas the Middle East and Western Europe provides the main market for U.S. turkey meat. Japanese imports of U.S. broiler meat for 1984 are estimated at 55,000 tons, roughly 9,000 tons less than in 1983. The decline is attributed to rising U.S. prices and the continued strengthening of the dollar.

Deteriorating trade relations with Middle Eastern countries were mainly responsible for the heavy setbacks suffered by U.S. exports in these countries. The EC and Brazil are the main suppliers of the Arab countries and the USSR. France, in particular, enjoys traditional commercial links with the Near and Middle East.

DAIRY PRODUCTS

Milk Production

Global milk production is expected to remain steady in 1984 and should decline in 1985 as EC and U.S. production control programs have an impact. The number of cows has shown little change in 1984, but plans point to a decline in 1985.

U.S. milk production in 1984 is likely to decline to 61.6 million tons from the record 63.5 million tons a year earlier. The smaller output will result from the paid diversion program and lower effective price for milk. Some recovery may occur in 1985 after the paid diversion program ends (March 31, 1985) with production advancing to around 62 million tons.

EC milk production may decline from its 1983 level of 112.3 million tons of

110.3 million tons in the following year due to lower cow numbers.[1] Among the other major producers, Eastern Europe is the only area expected to show a sharp decline.

Large gains should be recorded in the USSR. Milk production in the USSR is expected to reach 97.5 million tons during 1984 and should continue to rise in 1985 to about 99 million tons. With cow numbers stable for the past two years improved productivity is the main explanation for the increase. Canada's 1984 and 1985 milk production is forecast to stay at the 1983 level of around 8 million tons.

Milk production in both New Zealand and Australia is expected to increase in 1984 to 7.2 million tons and 6.1 million tons respectively. For 1985, New Zealand milk production is forecast to remain near 1984's output whereas declining cow numbers should bring a production decline in Australia.

Butter and Cheese Production

With declining global milk output, the relative strength of demand will largely determine the mix of products produced. Cheese output will likely increase somewhat in 1984 and 1985, while butter and nonfat-dry milk will decline. The EC and the U.S. largely account for this decrease. Production in the EC, the world's largest butter producer, is forecast to drop 5 percent in 1984 to 2.17 million tons and by a similar amount in 1985. U.S. production is projected down 12 percent in 1984 and is expected to fall again in 1985.

With more milk, Soviet butter production in both 1984 and 1985 is expected to increase about 2 percent reaching 1.62 million tons in 1985. Increased milk production in 1984 will force butter production up in Australia and New Zealand. Production is forecast to decline in both countries during 1985.

World cheese production is expected to follow an upward trend through 1985. The expansion reflects mainly the growth in EC cheese production expected to reach 3.73 million tons by 1985. Cheese production in the U.S. will likely drop 3 percent in 1984 because of smaller milk supplies. There is likely to be a small recovery in U.S. cheese production in 1985 but output will fall short of the 1983 level.

Soviet cheese production is forecast to make some modest gains through 1985. Canadian and Australian cheese production by contrast is expected to remain essentially unchanged in 1984 and 1985. Cheese production in New Zealand is forecast to fluctuate with 1985 output below the 1983 level.

World production of nonfat dry milk is forecast to follow the trend in butter production and is expected to fall in 1984 and 1985. The decline in world output reflects sharp drops in EC and U.S. production. Australia and New Zealand are forecast to expand production substantially in 1984 but will likely scale down production somewhat during 1985.

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1. In an effort to slow the growth of milk production the EC set a production threshold of 99.57 million tons of milk for 1984/85. The threshold for the next four years was set at 98.36 million tons.

Trade

Despite the projected decrease in world butter and nonfat dry milk production in 1984 and 1985 international demand will be insufficient to absorb the projected outputs. Little imporvement in trade in dairy products is foreseen in 1985 because of continued sluggish and stagnant demand. The weakness of international demand for dairy products stems from the continuing financial difficulties of several importing developing countries, the decline in revenue of oil-exporting countries, international food aid programs and changes in consumer attitudes about fat content. The wide price differentials between butter and margarine is another factor that discourages butter consumption.

Given the huge EC and U.S. stocks of dairy products and prospects for further accumulations the world outlook is for intensified competition on international markets between exporting countries.[1]

1. The new EC dairy policy will not solve the surplus problem in the short-term as the 99.5 million ton threshold exceeds current consumption of dairy products by 13.5 million tons.

Appendix Table 1: Major Oilseeds: World Production, Crush, Exports and Ending Stocks, 1983/84 and 1984/85.

	1983/84 (million tons)				1984/85 (million tons)			
	Prod.	Crush	Exp.	End. Stk.	Prod.	Crush	Exp.	End. Stk.
Soybean	79.7	73.5	25.7	5.8	93.8	76.0	26.3	9.9
Cottonseed	27.2	21.6	.2	.1	30.6	23.9	.1	.5
Peanut	18.9	11.3	1.0	.6	19.9	11.8	1.1	.6
Sunflowerseed	15.4	13.6	1.9	.3	17.3	15.2	1.9	.5
Rapeseed	14.6	13.8	2.6	.4	15.2	13.8	2.5	.7
Flaxseed	2.3	2.2	.7	.4	2.5	2.1	.5	.4
Copra	4.2	4.0	.3	.1	4,6	4.3	.3	.1
Palm Kernel	2.0	1.9	.1	.1	2.1	2.0	.2	.1
TOTAL	164.3	141.7	32.6	7.9	185.9	149.1	33.0	12.9

*Source: U.S. Department of Agriculture, FAS, Foreign Agriculture Circular, Oilseeds and Products FOP 8-84 August 1984, p. 7.

Appendix Table 2: Major Protein Meals: World Production, Consumption, Exports and Ending Stocks, 1983/84 and 1984/85.

	1983/84 (million tons)				1984/85 (million tons)			
	Prod.	Consp.	Exp.	End. Stk.	Prod.	Consp.	Exp.	End. Stk.
Soybean	57.5	56.9	20.9	1.8	60.2	59.2	22.0	2.0
Cottonseed	10.0	10.0	.7	.1	11.2	11.1	.8	.1
Rapeseed	8.3	8.4	.9	.2	8.4	8.4	.9	.2
Sunflowerseed	6.1	6.2	1.2	.1	6.9	6.7	1.4	.1
Fish	5.1	4.8	2.4	.6	5.2	5.1	2.4	.6
Peanut	4.6	4.6	.5	0	4.7	4.8	.7	0
Copra	1.3	1.3	.8	0	1.4	1.4	.9	0
Linseed	1.4	1.3	.7	0	1.3	1.3	.7	0
Palm Kernel	1.0	.9	.7	0	1.0	1.0	.8	0
TOTAL	95.2	94.5	28.9	3.0	100.3	99.1	30.7	3.2

*Source: U.S. Department of Agriculture, FAS, Foreign Agriculture Circular, Oilseeds and Products FOP 8-84 August 1984, p. 8.

Appendix Table 3: Major Vegetable and Marine Oils:
Production, Consumption, Exports and Ending Stocks, 1983/84 and 1984/85.

	1983/84 (million tons)				1984/85 (million tons)			
	Prod.	Consp.	Exp.	End. Stk.	Prod.	Consp.	Exp.	End. Stk.
Soybean	13.0	13.3	3.5	.9	13.6	13.5	3.6	.9
Palm	6.3	6.0	4.0	.5	6.8	6.5	4.4	.6
Sunflowerseed	5.5	5.6	1.5	.2	6.2	6.0	1.6	.2
Rapeseed	5.2	5.2	1.0	.2	5.1	5.0	1.0	.2
Cottonseed	3.4	3.4	.3	.1	3.8	3.7	.4	.1
Peanut	3.3	3.4	.3	0	3.5	3.5	.3	0
Coconut	2.4	2.6	1.0	.1	2.7	2.7	1.2	.1
Olive	1.4	1.7	.3	.7	1.7	1.7	.3	.8
Fish	1.1	1.1	.7	.2	1.2	1.1	.8	.2
Palm Kernel	.8	.8	.5	0	.8	.8	.6	0
Linseed	.7	.6	.3	0	.7	.6	.3	0
TOTAL	43.4	43.8	13.4	3.1	46.0	45.2	14.5	3.3

*Source: U.S. Department of Agriculture, FAS, Foreign Agriculture Circular, Oilseeds and Products FOP 8-84 August, 1984, p. 9.

Agricultural Applications of Genetic Engineering

DAVID H. BAKER

The potential of genetic engineering is enormous. It has been labeled one of the four major scientific breakthroughs of the century--on a par with unlocking the atom, escaping earth's gravity and the computer revolution. Activity in this fledgling industry in the U.S. has been great in the last four years, primarily as a result of the U.S. Supreme Court's decision in 1980 to allow patenting of artificially developed organisms. The market potential is greatest and probably most immediate in agriculture. Worldwide estimates of the market value of genetically engineered products by the year 2000 range between 50 and 100 billion dollars.

Because genetic manipulation conjures up all sorts of risks and monstrous images, it would be well to review certain applications being widely discussed by the lay public.

TEST-TUBE BABIES

In July of 1978, Lesley Brown of Lancashire, England, gave birth to the first so-called "test-tube baby". An abbreviated outline of the specific procedures involved is given below:

1. The mature egg is removed from a female ovary.
2. The egg is placed in a petri dish containing a culture medium similar to that found in the fallopian tube.
3. Sperm is collected from the male and added to the culture medium; the medium is then incubated for 18 hours at 98.6° F.
4. The fertilized egg is transferred to a different culture medium (simulating the uterine environment) and incubated for another 18 hours; cell division occurs resulting in blastocyst (i.e., embryo) formation.
5. The blastocyst is placed (by way of the vagina) into a recipient female uterus after the female has first been prepared via hormone treatments to accept the embryo.

This is a very difficult procedure in that timing of the collection of the egg when it is just mature is critical. Also, often after everything else has succeeded, the recipient uterus rejects the embryo. Nonetheless, in the U.S.

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alone, an estimated 300,000 married women have blockage of the fallopian tubes; roughly five times this number of men have a condition known as oligospermia (lack of sufficient sperm count to effect conception). Hence, this procedure of embryo culture and transfer offers hope to infertile couples who would like to conceive and bear their own (genetic) children.

EMBRYO TRANSFER IN ANIMALS

This procedure, unlike the ova transfer procedure described above for humans, involves removing the embryo from the uterus of a donor (e.g., a cow) at about seven days postconception and transferring it to the uterus of a recipient cow for the remaining nine months of gestation. Hence, by this technique, a valuable cow, e.g., one with excellent milk production records, could theoretically be the genetic mother of ten or more valuable calves, all born within a few days of one another. In practice, the donor cow is treated with a fertility drug such as follicle-stimulating hormone to effect superovulation, thus permitting production of eight to ten embryos instead of the usual one. The embryos are then flushed from the uterus and stored (or frozen in liquid nitrogen) until transfer time. For high pregnancy rates, the surrogate recipient mother must be at about the same stage of pregnancy as the donor.

Embryo transfer currently costs about $1,000 per pregnancy. An estimated 50,000 calves were born in North America in 1982 using this technology. Embryo transfer of this type has been done in horses, swine and humans, in addition to cattle. A limited amount of research work has also been done with sheep, goats and rabbits.

EMBRYO SPLITTING AND SEXING

The science of embryo manipulation has recently been extended to include what has become known as embryo splitting and sexing. These procedures offer exciting potential in animal production. Using a procedure called "micromanipulation", reproductive cell biologists have been able to take a bovine embryo, preferably at the four- to eight-cell stage, and split it into several embryos. Thus, this procedure facilitates production of "phenocopies" (e.g., identical twins). Hence, herds of "super cows" may be on the horizon.

Another active area of investigation involves sperm and embryo sexing. Although this technique is not yet perfected for use in practice, there is little question that once perfected, widespread application will quickly follow. The procedure involving sperm would include separating the sperm into those containing a Y-chromosome (male offspring production) and those containing an X-chromosome (female offspring production). The procedure would use electrophoretic techniques and, also, antibodies that recognize the "H-Y" antigen on a Y-bearing sperm. The dairy industry desires females while the beef cattle industry desires male calves. Thus, engineering the desired sex in the bovine species would involve a tremendous breakthrough with far-reaching implications.

Offspring sexing can also be accomplished by sexing embryos prior to transfer. Tests are being conducted in which antibodies that recognize the H-Y antigen are used to "kill" the male embryo. As an alternative to killing the male embryos, the antibody-tagged male embryos can be sorted out. Time will tell whether this antibody-tagging procedure will prove commercially feasible.

CLONING

A clone is the asexual progeny of an individual; i.e., an exact replica of the organism from which it came. Were a mammal to be cloned, the female nucleus (23 chromosomes in humans) of the egg would have to be removed. The nucleus of a body cell (46 chromosomes) would then be inserted into the egg. The egg with its new nucelar material (from the donor) would then be transferred into the uterus of a recipient host animal and allowed to grow to maturity. If all went well, the resulting offspring would be an exact replica (genetically and in outward appearance) of the animal who donated the body cell. While progress has been and is being made in this technology in frogs and mice, there is little question that no human has ever been cloned, despite certain claims to the contrary.

David Rorvick's controversial book, In His Image: The Cloning of a Man, (Lippincott) purported that a 67-year-old millionaire by the name of Max had himself cloned. Recently, a lawsuit was won against the publisher who had publicized the book as nonfiction. The Lippincott Company thus recanted and admitted that it now believes the book to have been a fraud and a hoax.

Ira Levin's best-selling novel, The Boys from Brazil, later a movie, contended that several clones of Adolf Hitler had been produced from cellular material from Hitler's dead body. This, too, is pure science fiction. Frog eggs have been cloned for almost 40 years, but frog eggs are 20 times larger than human eggs, and even with very young tadpole donors, the success rate is only about one in 100 tries. Hence, it is foolish to believe that an adult human body cell could have been used to clone a man, particularly a man produced using cells from a dead person.

MONOCLONAL ANTIBODIES (HYBRIDOMAS)

The science of producing specific disease-fighting antibodies offers exciting potential for the future. A hybridoma is a cell which produces one, and only one, very specific antibody. They can be made in the laboratory by fusing an antibody-producing cell from the spleen with a tumor cell (myeloma). The fused product will then rapidly reproduce in tissue culture. The spleen cell, hence, provides for the design of the product while the tumor cell allows mass production of that product--a product called a monoclonal antibody. Because of their specificity, those antibodies can be used in producing new vaccines, in improving the diagnosis of disease and in fighting disease itself.

A new development from monoclonal antibody technology involves the transfer of genes from tumor cells directly into mammalian cells. This new procedure is known as transfection. What makes this technology exciting to the scientific community is that it will permit lymphoid cells from domesticated animals to be literally immortalized in tissue culture. Such cells could be used in the production of a wide range of useful animal biologics. The transfection technology represents a major advance in genetic engineering wherein both monoclonal and recombinant DNA procedures are required.

RECOMBINANT DNA APPLICATIONS

The classical recombinant DNA experiment is depicted in Figure 1. An Escherichia coli bacterium is shown with several DNA molecules: one very large

one, the chromosome, and several small DNA pieces called plasmids. Plasmids can be removed from the E. coli cell, purified and then cut with a special kind of enzyme known as a restriction nuclease. The cut plasmid from E. coli is then combined with a cut (exactly fitting) from a segment of DNA from, say, human pancreatic tissue. In this way, recombinant DNA has been produced. The recombinant DNA is next inserted into another (prolific) E. coli bacterium which begins reproducing. Thus, E. coli can be used to manufacture valuable proteins such as human insulin which is identical to the insulin produced by human pancreatic cells. Obviously, the potential in biomedicine and agriculture from this technology is enormous.

ANIMAL AGRICULTURE APPLICATIONS

Products produced from recombinant DNA technology for use in domesticated animals will likely reach market before products used in plant production. For the most part, this is due to the fact that many products used in animal production are derived from fermentation--and fermentation, using bacteria or yeast cells, is ideally suited to recombinant DNA procedures. Genetic transfer from one animal species to another is difficult; practical applications are certainly years down the road. Noteworthy, however, is that species-to-species gene transfer in mammals has been done; i.e., the gene for hemoglobin production in rabbits has been transferred to mice. More recently, the gene for growth hormone production in humans has been transferred to mice. Work is currently underway to accomplish a similar transfer of the growth hormone gene to swine. This demonstrates that interspecies gene transfers are possible.

Fermentation Products

Literally any product produced normally via fermentation can potentially be produced more efficiently using recombinant DNA technology. Examples include single cell, e.g., yeast protein, a key enzyme necessary for mass production of high-fructose corn sweeteners, antibiotics, vitamins (e.g., riboflavin and vitamin B-12), amino acids (e.g., lysine), hormones (e.g., insulin, growth hormone), inhibitors of viral replication (e.g., interferon), a multitude of vaccines and products such as lymphokines (interleukin I and II) which have the potential of making vaccines more effective.

Vaccines

Vaccines against viral diseases will probably reach market first. These will be followed by antibacterial vaccines and then antiparasitic vaccines. Products that have already been developed or are in final stages of testing are: (1) foot-and-mouth-disease vaccine, an improved and potentially less expensive version of the conventional vaccine; (2) bovine interferon for use against shipping fever in cattle; (3) a vaccine for neonatal diarrhea in young pigs, and another for treatment of colibaccilosis diarrhea of piglets.

Hormones

Most exciting in this area is growth hormone. Experimentally, daily injections of bovine growth hormone have been shown by Cornell workers to bring about dramatic increases in milk production in dairy cows, particularly in low-producing cows. Porcine growth hormone has been shown by Monsanto scientists to improve feed efficiency and carcass leanness in pigs as well. Some form of implant will

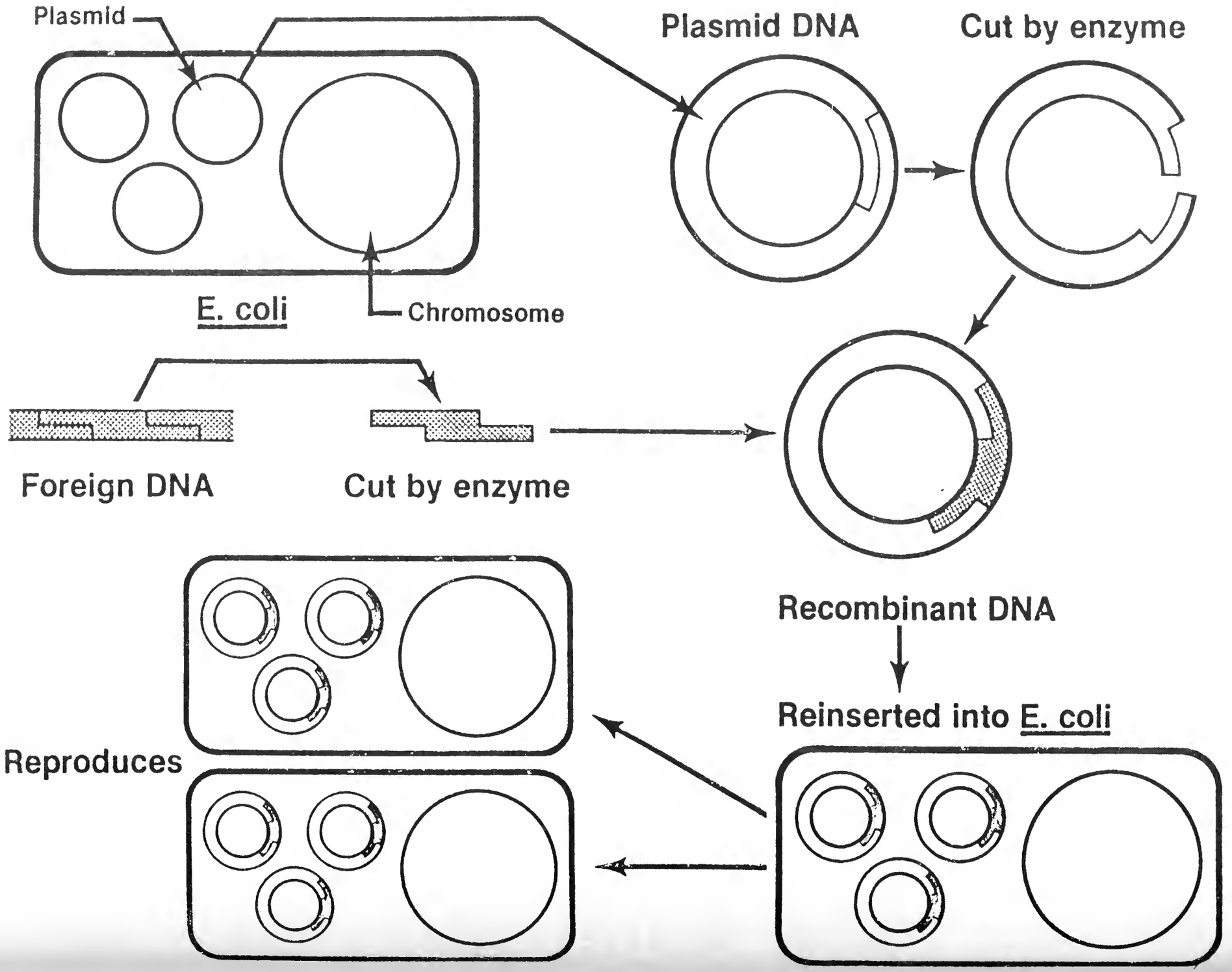
have to be developed to circumvent the labor of giving daily injections of growth hormone. Certainly, it would be far better if we could put "amplifying genes" directly into the animal to code the animal and its offspring for, say, rapid growth rate, high milk production or maximum disease resistance. Indeed, it would be better, for example, to be able to buy a special strain of high-producing Holstein than to have to inject or implant growth hormone produced through recombinant DNA production. But the amplifying gene idea is not as yet a reality, and probably won't be for some time. Fermentation-produced growth hormone, on the other hand, is on the immediate horizon and probably will be available commercially very soon.

PLANT AGRICULTURE APPLICATIONS

Realistically, in the near term, genetic engineering technology will likely supplement, rather than supplant, the more conventional methods used in plant breeding. Also, crops such as wheat, soybeans and peanuts will likely receive more attention from genetic engineering procedures than will corn. Corn is thus very adaptable to conventional plant breeding and plant pathology applications. In the last 20 years, U.S. corn yields have doubled, and further yield increases are expected in the future.

It is conceivable that somewhere in the future we may see (a) corn and wheat plants that will fix atmospheric nitrogen; (b) crops that will have so much salt tolerance that they will be capable of being irrigated with sea water and (c) corn, wheat and rice plants whose grain will have a protein quality (i.e., amino acid balance) similar to that of meat, milk and eggs. Shorter term, we will see recombinant DNA methodology used to develop more pest resistance in cereal grain and oilseed crops, more cold tolerance in a variety of plants, more disease and saline resistance, more efficient photosynthesis and nitrogen fixation, and greater capability for plants to respond to growth regulators.

There is little question that nitrogen fixation is the big prize. Over two billion dollars annually are spent on nitrogen fertilizers applied to the soil. With corn, for example, it is known that nitrogen fixation potential can and will eventually be installed--but there will be considerable problems to overcome. Thus, nitrogen-fixing varieties of corn may not yield well, they may not fix nitrogen efficiently in the presence of nitrogen fertilizers, and they may be less tolerant to disease, drought and salinity.


Plasmid
Plasmid DNA
Cut by enzyme
E. coli
Chromosome
Foreign DNA
Cut by enzyme
Recombinant DNA
Reinserted into E. coli
Reproduces

Repartitioning Agents: Feed Additives of the Future to Improve Performance and Carcass Composition

RONALD H. DALRYMPLE

The discovery of exogenous agents that stimulate the production of lean muscle in the body while limiting the synthesis of subcutaneous and internal fat will result in the anticipated marketing of a new class of feed additive products by the late 1980's. In addition to, and probably as a result of, the repartitioning of nutrient utilization and deposition, these agents also produce a growth response and, most importantly, improve feed conversion.

These efficiency improvements result from the energy spared when depositing muscle instead of fat. In spite of the approximately equal amount of energy required to synthesize fat and protein (van Es, 1977), there is a considerable net savings in energy when comparing adipose tissue versus muscle tissue due to the fact that a unit of muscle tissue contains 70 to 75% water while an equal weight of adipose tissue contains less than 20%. Furthermore, a unit of fat contains twice the calories of a unit of protein. Based on this, it is little wonder that animals fed repartitioning agents are more efficient at converting feed to weight gain.

The use of repartitioning agents will greatly accelerate the changes in carcass composition currently being brought about by genetic, nutritional and management means. The importance of reducing excess fat on the swine, cattle, sheep and poultry we produce is clear to all of us. An example of the magnitude of the problem can be seen from the figures of Allen, et al (1976) who calculated that about four billion pounds of excess fat was produced yearly on our meat animals. The amount of feed consumed by the animals to produce this waste fat is even more monumental and represents a tremendous squandering of our resources. While the industry is concentrating on reducing fat, it must not lose sight of the real objective of raising meat animals, the production of muscle. Two examples of what can potentially go wrong in breeding programs, when attempting to alter composition, are the Porcine Stress Syndrome problem brought about by extreme increases in muscle and the "meatless wonder" type animal produced when leanness, without regard to muscling, is over-emphasized. Studies, to date, have shown us that the use of repartitioning agents would allow producers to make significant changes in their market animals and, at the same time, allow them to maintain emphasis on production factors, such as litter size, in their breeding herds.

DISCOVERY OF REPARTITIONING AGENTS

Given the need to emphasize leanness and muscling in meat animals and the limitations of genetic and nutrition solutions, there appears to be considerable commercial

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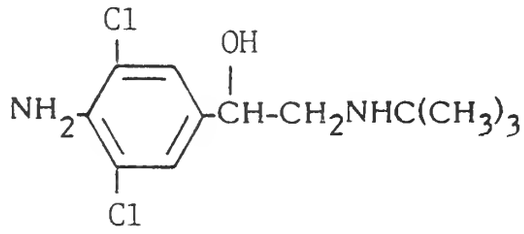
potential for an exogenous agent which would bring about the desired compositional changes. Based on the remarkable shifts in nutrient partitioning that take place with normal changes in physiological state, such as the onset of lactation (Bauman and Currie, 1980) or during rapid increases in growth (Bauman et al, 1982), it appeared possible that the systems controlling these physiological changes could be manipulated using exogenous chemical agents or hormones. About ten years ago, based on this general idea, we initiated a discovery project with the emphasis on screening large numbers of random compounds for their ability to increase the accretion of lean muscle and limit the deposition of adipose tissue in growing animals. Recently, using a rodent model system, we have identified a series of compounds which possess the desired activity in the major meat animal species. These compounds appear to be exerting their effects directly on the metabolism of muscle, adipose and liver cells such that the normal partitioning of nutrients and energy into muscle and fat tissue is altered. Hence, we have coined the term "repartitioning agents" to describe this series of compounds.

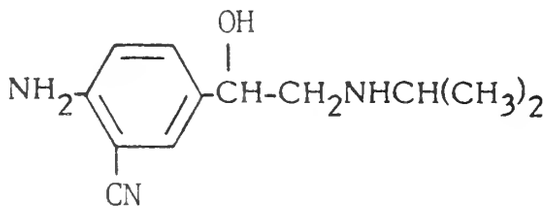
The initial active compound discovered in this series was clenbuterol (Figure 1) which we subsequently have evaluated in major meat animal species including swine (Ricks et al, 1984a), poultry (Dalrymple et al, 1983, 1984b), sheep (Baker et al, 1983, 1984) and cattle (Ricks et al, 1983, 1984b). More recently, we have begun to evaluate another compound in this series identified, to date, as AC 263,780 (Figure 1) and have reported on its evaluation in both poultry (Dalrymple et al, 1984c) and swine (Dalrymple et al, 1984a and Moser et al, 1984).

Figure 1

Structure of Repartitioning Agents, Clenbuterol and AC 263,780



Clenbuterol



AC 263,780

MEAT ANIMAL EVALUATION OF REPARTITIONING AGENTS

Swine

The initial evaluation of repartitioning agents in swine was carried out in finishing swine with clenbuterol (Ricks et al, 1984a). The repartitioning effects are clearly evident in the results of this titration study (Table 1).

Table 1
Performance and Carcass Characteristics of Barrows and Gilts Fed Various Levels of Clenbuterol from 50 kg to Market Weight (110 kg)

Observation	Clenbuterol Level (ppm in feed)			
	0	.05	.1	1.0
Avg. daily gain (g)	765	776	741	717*
Gain/feed	.311	.319	.314	.293
Dressing %	74.7	74.7	75.1	75.6*
Avg. backfat thickness (cm)	3.59	3.41	3.20**	3.34
Longissimus area (cm^2)	34.7	37.8*	38.5*	42.1*
Fat depth over loin (cm)	3.39	3.14	3.16	2.99*
Carcass Composition				
Fat %	31.04	30.01*	29.98*	28.51**
Protein %	15.78	16.16	16.77**	16.66**
Water %	49.45	51.26*	51.33*	52.09**

* and ** Significantly different from control, P = .05 and .01, respectively.

Although no improvements were found in animal performance, there was evidence of repartitioning even at the .05 ppm level since longissimus muscle area was increased by almost 10% and percent carcass fat was reduced when the whole ground carcass was analyzed by proximate analysis. The extreme potency of these compounds is evident from the active levels (.05 to 1 ppm) used in this study. Importantly, no evidence was seen that these agents had a detrimental effect on meat quality at any level tested.

Further reported evaluation of this series of repartitioning agents in finishing swine was conducted with AC 263,780. The initial studies demonstrated that AC 263,780 did produce a repartitioning response (Dalrymple et al, 1984a), which was comparable to that seen in the above clenbuterol study.

As seen with clenbuterol, performance of the pigs fed AC 263,780 was not improved (Table 2) although there was a trend toward improved feed conversion. The highest level tested (1 ppm) reduced daily gain, possibly as a result of lower feed consumption.

Table 2
Performance of Barrows and Gilts Fed Various Levels of AC 263,780 from 60 kg to 105 kg

Observation	AC 263,780 (ppm)			
	0	.05	.2	1.0
Final number of pigs	47	46	46	43
Avg. daily gain (g)	780	789	789	739*
Avg. daily feed (kg)	2.92	2.86	2.85	2.70
Gain/feed	.267	.276	.277	.274

* Significantly different from control, P = .05

Carcass evaluation at the termination of the study indicated that AC 263,780 treatment produced a dramatic effect on carcass parameters related to repartitioning (Table 3).

Table 3
Carcass Characteristics of Barrows and Gilts Fed Various Levels of AC 263,780 from 60 kg to 105 kg

Observation	AC 263,780 (ppm)			
	0	.05	.2	1.0
Dressing %	74.1	74.1	73.8	74.8
Avg. backfat (cm)	3.20	3.00	3.02	2.90*
10th rib fat depth (cm)	2.87	2.67	2.69	2.49**
Leaf fat weight (g)	1175	1116	1146	928*
Longissimus area (cm^2)	36.9	37.9	38.9**	40.2**
% Carcass muscle[a]	55.5	56.4	57.0*	58.2**
Longissimus color score[b]	2.52	2.54	2.48	2.48
Longissimus marbling score[c]	2.53	2.39	2.17	2.32
Belly thickness (cm)	4.3	4.2	4.3	4.2

* and ** Significantly different from control, P = .05 and .01, respectively

[a] Estimated using National Pork Producers Council formula (1983)

[b] Color score: 1 = pale to 5 = dark

[c] Marbling score: 1 = traces to 5 = abundant

Based on the results of this study the active levels of AC 263,780 are in the range of .2 to 1 ppm. As noted with the clenbuterol study (Table 1) and in this study (Table 3), the most sensitive parameter to the repartitioning activity appears to be an increase in longissimus muscle area. Dose dependent responses were noted and levels as low as .05 and .2 ppm were needed to obtain a significant (P = .05) response for the respective trials. Measures of adipose tissue response to repartitioning agents were evident in both studies; however, were somewhat inconsistent with regard to dose levels.

With these encouraging results, we initiated a series of field trials at several universities to confirm the results we had seen, to date, and to evaluate these and additional parameters under different conditions from our in-house studies.

The first of these trials has been reported recently (Moser et al, 1984). In this study at the University of Minnesota a narrower range of drug levels (.25, .5 and 1 ppm) were examined in finishing pigs. The only significant effect of AC 263,780 on animal performance (Table 4) was a depression in feed intake. However, there was also a tendency for the treated pigs to be more efficient than the controls. No effect of treatment on the soundness of the feet and legs of the live animals was found.

Table 4
Performance of Finishing Pigs Fed Various Levels of AC 263,780 at the University of Minnesota

	AC 263,780				Treatment
	0	.25	.5	1.0	Significance
Avg. daily gain (kg)	.767	.780	.762	.748	NS
Avg. daily feed intake (kg)	2.67	2.51	2.58	2.48	.02
Gain/feed	.288	.302	.294	.302	NS
Days on test	59.0	56.1	55.3	57.7	NS

Carcass evaluation of these pigs indicated a substantial repartitioning effect at all levels tested (Table 5). Dressing percent was improved by up to .6 percentage points. There appears to be a consistent increase in dressing percent in pigs displaying meaningful repartitioning. This is opposite to the general belief that fatter pigs yield higher. It is presumed that the dressing percent benefits from repartitioning are the result of a greater increase in carcass muscle weight than the loss of carcass weight due to a decrease in fat, thereby resulting in an overall increase in ratios of soft tissue (muscle and fat) to bone, gastrointestinal tract, etc. which determines yield. Responses of both adipose tissue and muscle to the repartitioning effects of AC 263,780 were seen and are consistent with those noted in the previous study (Dalrymple et al, 1984a) reported above.

Table 5
Carcass Characteristics of Pigs Fed Various
Levels of AC 263,780 at the University of Minnesota

	AC 263,780				Treatment
	0	.25	.5	1.0	Significance
Dressing %	78.1	77.9	78.6	78.7	.03
Leaf fat (kg)	1.77	1.71	1.69	1.70	NS
Avg. backfat thickness (cm)	3.45	3.23	3.10	3.12	.001
P_2 fat depth (cm)	2.41	2.16	2.06	2.16	.02
Fat depth over loin (cm)	2.95	2.69	2.46	2.54	.0002
Loin muscle area (cm^2)	36.7	38.2	38.1	39.9	.04

Poultry

We have conducted approximately 20 broiler floor pen trials with clenbuterol and AC 263,780, to date, including trials at several locations and with different strains of broilers. Early work indicated that repartitioning agents only need to be administered during the finisher period from about four to seven weeks of age to produce their maximum benefits. A drug withdrawal period of three to five days is also used in the studies since this is common industry practice and it may be a registration requirement for these agents. Feeding 1 ppm clenbuterol to broilers following this regimen results in dramatic improvements in performance and carcass parameters (Dalrymple et al, 1983, 1984b). An improvement of 3.3% in gain and 3.0% in feed conversion was demonstrated (Table 6). At slaughter we noted a .8 percentage point increase in carcass yield but apparently clenbuterol had no effect on the abdominal fat pad. Proximate analysis of the ground whole carcass indicated alterations in carcass composition in the direction of repartitioning.

Table 6
Summary of Performance and Carcass Characteristics of Broilers Fed
Clenbuterol from 28 to 46 Days of Age in Three Trials (sexes combined)

Observation	Clenbuterol Level (ppm in feed)	
	0	1
Weight gain, (g) 28-49 days	1156	1194**
Feed/gain, 28-49 days	2.30	2.23**
Carcass yield at 50-51 days (%)[1]	69.91	70.71**
Abdominal fat pad (%)[2]	3.76	3.61
Carcass composition		
Fat %	16.78	15.88
Protein %	18.83	19.17
Water %	61.83	62.90**

** Significantly different from control, P = .01

[1] Eviscerated weight/live weight x 100 (without abdominal fat pad)

[2] Abdominal fat pad/eviscerated weight x 100

Paralleling the research conducted in swine, we next conducted several studies to evaluate AC 263,780 in poultry following a similar feeding regimen to that of clenbuterol (Dalrymple et al, 1984c). In one of these studies we titrated AC 263,780 at .125, .25, .5 and 1.0 ppm and used clenbuterol at 1 ppm as a positive standard. Gains from 28 to 49 days of age were improved by administration of AC 263,780 and clenbuterol (Table 7). Improvements in feed conversion paralleled those seen in gain. Carcass yield increases in the AC 263,780 treatment groups ranged from .53 to 1.02 percentage points while clenbuterol improved yield by .74 percentage points. The abdominal fat pad showed no response to the repartitioning agents even though percent fat in the ground whole carcass was reduced in all treated groups. The abdominal fat pad appears to be insensitive to the action of repartitioning agents which is consistent with the general unresponsiveness of the abdominal fat pad to catecholamines (Carlson et al, 1964), which are structurally and physiologically related to both clenbuterol and AC 263,780.

Table 7
Performance and Carcass Characteristics of Broilers Fed AC 263,780 and Clenbuterol from 28 to 46 Days of Age (sexes combined)

	Treatment (ppm)					
	AC 263,780					Clenbuterol
Observation	0	.125	.25	.5	1	1
Weight gain, 28-49 days (g)	1245	1281*	1301**	1295**	1268	1284*
Feed/gain, 28-49 days	2.24	2.19**	2.18**	2.16**	2.21	2.18**
Carcass yield at 50-51 days(%)[1]	70.29	70.82	70.94*	71.31**	71.04*	71.03*
Yield % point increase		.53	.65	1.02	.75	.74
Abdominal fat pad (%)[2]	2.82	2.77	2.82	2.75	2.80	2.83
Carcass composition						
Fat %	19.19	19.98	19.60	19.47	19.71	19.65
Protein %	17.56	16.18*	16.44*	15.98**	16.22*	16.26*
Water %	61.03	61.79	61.86	62.50*	62.35*	62.21*

* and ** Significantly different from control, P = .05 and .01, respectively

[1] Eviserated weight/live weight x 100 (without abdominal fat pad)

[2] Abdominal fat pad weight/eviserated weight x 100

Several studies in feedlot lambs have indicated that repartitioning agents can produce a tremendous improvement in performance and carcass characteristics (Baker et al, 1983, 1984). The results of one study in which 2 ppm clenbuterol was fed to feedlot lambs is summarized in Table 8. Gain and feed conversion were improved by 24 and 19%, respectively. Carcass data demonstrate the dramatic changes repartitioning agents can produce. Longissimus muscle area was increased by 40% while subcutaneous and internal fat were decreased by almost 40%. Yield grade was improved in the clenbuterol fed lambs by one grade point as a result of the repartitioning, while quality grade was unaffected. Based on the data collected, to date, with the repartitioning agents in all species, lambs are the most responsive specie to their repartitioning and growth promoting effects.

Table 8
Performance and Carcass Characteristics of Wether Lambs Fed Clenbuterol for Eight Weeks

Observation	Clenbuterol Level (ppm in feed) 0	2
Average daily gain (kg)	212	263**
Gain/feed	.120	.149**
Dressing %	54.6	57.2*
Longissimus area, (cm^2)	16.85	23.85**
Fat depth, 12th rib (mm)	5.9	3.7**
% Kidney and pelvic fat	3.6	2.3**
USDA quality grade (1-12)[1]	9.6	10.1
USDA yield grade (1-5)	3.5	2.5**

* and ** Significantly different from control, P = .05 and .01, respectively

[1] 12 = Prime+, 11 = Prime average, 10 = Prime-, 9 = Choice+, etc.

Only one study has been conducted, to date, in feedlot cattle and the results are presented in Table 9 (Ricks et al, 1983, 1984b). No performance improvements were found in this study which was designed, primarily as a safety study, to examine tolerance to a high drug level (500 mg/head/day or approximately 50 ppm). This high feeding level may have been responsible for the depression in gain. However, excellent repartitioning activity was seen at both low and high dose levels, with the higher level showing a slightly greater response. Improvements in longissimus muscle area of 11 to 17% and in yield grade of 1 to 1.5 grade points were noted along with a 40% decrease in 12th rib fat depth and a 25-35% decrease in internal fat.

Table 9
Performance and Carcass Characteristics of Hereford Feedlot Steers Fed Clenbuterol for 98 Days

Observation	Clenbuterol Level (mg/head/day) 0	10	500
Number	8	8	8
Average daily gain (kg)	1.098	1.007	.868*
Gain/feed	.084	.083	.084
Slaughter weight (kg)	447	436	424
Dressing %	63.7	64.5	63.5
Kidney, pelvic and heart fat (%)	2.52	1.93*	1.68*
Longissimus area (cm^2)	79.6	88.1*	92.6**
Fat depth, 12th rib (cm)	1.29	.83**	.75**
USDA yield grade (1-5)	2.7	1.7**	1.2**

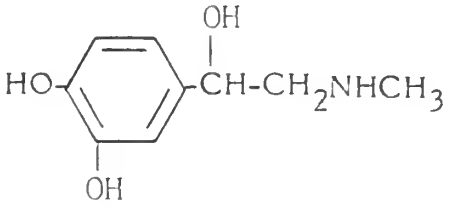
* and ** Significantly different from control, P = .05 and .01, repectively

MODE-OF-ACTION OF REPARTITIONING AGENTS

Compounds of the current series of repartitioning agents, including clenbuterol and AC 163,780, are pharmacologically classified as beta agonists and are structurally classified as substituted catecholamines. Although they are chemically synthesized, they possess many similar structural and functional features to the natural catecholamine epinephrine (Figures 1 and 2).

Figure 2

Structure of Epinephrine



Epinephrine

Natural catecholamines exert their many metabolic and physiological effects through interaction with cell membrane receptors called alpha and beta receptors. Beta agonists act specifically by binding to the beta receptors located on the cell membrane which initiates an intracellular response through the stimulation of cyclic adenosine monophosphate (c-AMP) production (Fain and Garcia-Saing, 1983). Beta receptors have been further divided by Lands et al, (1967) into two distinctive subtypes known as beta 1 and beta 2. The former are physiologically typified by those mediating effects on cardiac muscle contractibility, while the latter cause relaxation of tracheal and uterine smooth muscle. The proportion of beta 1 or 2 receptors found within any tissue or organ are characteristic of that tissue or organ and can differ from specie to specie. Recently, the idea was put forth that there exists in tissues, other than cardiac and smooth muscle, further subtypes of beta receptors such as a beta 3 receptor in brown adipose tissue (Arch et al, 1984).

In human and veterinary medicine, clenbuterol and other beta 2 agonists are used as bronchodilators for the treatment of bronchial asthma (Del Bono et al, 1977) and also are used to stop uterine smooth muscle contractions for control of parturition (Wolfe, 1983). Recently, certain agents with beta agonist activity have been examined as potential human antiobesity agents since in obese rodents they produce a dramatic loss of adipose tissue with little apparent effect on lean body mass (Massondi et al, 1983). Increased fatty acid mobilization from fat depots, coupled with a stimulation of thermogenesis in brown adipose tissue whereby the mobilized fat is converted to heat, may explain how these agents produce their antiobesity effect. What sets our series of compounds apart from these thermogenic beta agonists is that instead of converting fat into waste heat, the repartitioning agents are diverting this energy into the production of skeletal muscle. It is known that catecholamines and beta agonists stimulate the release of fatty acids from meat animal adipose tissue both in vivo and in vitro (Mersmann, 1979). It remains to be elucidated whether these repartitioning agents also have an effect on fat accretion by producing an inhibition of fatty acid synthesis in adipose tissue.

Obviously, repartitioning agents are having a dramatic effect on the accretion of skeletal muscle protein. It has been shown that epinephrine and certain beta agonists reduce the rate of muscle protein degradation (Hill and Malamud, 1974; Li and Jefferson, 1977; Tischler, 1981). Recently, clenbuterol has been found to increase in vivo muscle protein synthesis (fractional rate) in chronically treated rats (Emery et al, 1984). It would seem, therefore, that repartitioning agents act to stimulate muscle accretion by altering the protein turnover rate through both a stimulation of protein synthesis and an inhibition of protein degradation. The increased synthesis is supported by the diverting of energy and nutrients away from adipose tissue. The overall effect is a repartitioning of both protein and energy utilization and deposition in the body.

IMPACT OF REPARTITIONING AGENTS AS FEED ADDITIVES

Looking into the future, the commercialization of repartitioning agents will have a dramatic effect on the livestock industry. They will be the basis of a new class of feed additives which will allow producers to raise meat animals having a composition desired by the meat packers and the consumers of their products. At the same time, it will improve the efficiency of feed conversion to salable product for the producer and, in most cases, improve the rate of gain of his livestock. The activity of the repartitioning agents would also appear to be additive to that of the other drugs which the livestock producer will use.

References

Allen, E. A.; Beitz, D. C.; Cramer, D. A.; Kauffman, R. G. 1976. Biology of fat in meat animals. North Central Regional Research Publication No. 234. Research Division, College of Agricultural and Life Sciences, University of Wisconsin, Madison, Wisconsin.

Arch, J. R. S.; Ainsworth, A. T.; Cawthorne, M. A.; Piercy, A.; Sennitt, M. V.; Thody, V. E.; Wilson, C. and Wilson, S. 1984. Atypical beta-adrenoceptor on brown adipocytes as target for anti-obesity drugs. Nature 309:168.

Baker, P. K.; Dalrymple, R. H.; Ingle, D. L.; Ricks, C. A., 1983. Use of an adrenergic agonist to alter muscle and fat deposition in lambs. Fed. Proc. Soc. Exp. Biol. 42:816 (Abstr.).

Baker, P. K.; Dalrymple, R. H.; Ingle, D. L.; Ricks, C. A., 1984. Use of a beta-adrenergic agonist to alter muscle and fat deposition in lambs. J. Anim. Sci. (in press).

Bauman, D. E.; Currie, W. B., 1980. Partitioning of nutrients during pregnancy and lactation: A review of mechanisms involving homeostasis and homeorhesis. J. Dairy Sci. 63:1514.

Bauman, D. E.; Eisemann, J. H.; Currie, W. B., 1982. Hormonal effects on partitioning of nutrients for tissue growth: Role of growth hormone and prolactin. Fed. Proc. Soc. Exp. Biol. 41:2538.

Carlson, L. A.; Liljedahl, S.-O.; Verdy, M.; Wirsen, C., 1964. Unresponsiveness to the lipid mobilizing action of catecholamines in vivo and in vitro in the domestic fowl. Metabolism 13:227.

Dalrymple, R. H.; Ricks, C. A.; Baker, P. K.; Pensack, J. M.; Gingher, P. E.; Ingle, D. L., 1983. Use of the beta-agonist clenbuterol to alter carcass composition in poultry. Fed. Proc. Soc. Exp. Biol. 42:668 (Abstr.).

Dalrymple, R. H.; Baker, P. K.; Doscher, M. E.; Ingle, D. L.; Pankavich, J. A.; Ricks, C. A., 1984a. Effect of repartitioning agent CL 263,780 on muscle and fat accretion in finishing swine. 1984 J. Animal Sci. 59:212 (Suppl. 1) Abstr. 164.

Dalrymple, R. H.; Baker, P. K.; Gingher, P. E.; Ingle, D. L.; Pensack, J. M.; Ricks, C. A., 1984b. A repartitioning agent to improve performance and carcass composition of broilers. J. Poultry Sci. (in press).

Dalrymple, R. H.; Gingher, P. E.; Ingle, D. L.; Maddock, H. M.; Pensack, J. M.; Ricks, C. A.; Goodwin, T. L.; Waldroup, P. W., 1984c. Use of the repartitioning agent AC 263,780 to improve performance and carcass characteristics in broilers. Poultry Sci. 63:85 (Suppl. 1) Abstr.

Del Bono, N.; D'Aula, S.; Vibelli, C., 1977. Dose-response of patients with reversible bronchospasm to oral clenbuterol: Four doses compared. Curr. Therap. Res. 22:376.

Emery, P. W.; Rothwell, N. J.; Stock, M. J.; Winter, P. D., 1984. Chronic effects of beta-agonists on body composition and protein synthesis in the rat. Bioscience Reports 4:83.

Fain, J. N.; Garcia-Sainz, J. A., 1983. Adrenergic regulation of adipocyte metabolism. J. Lipid Res. 24:945.

Hill, J. M.; Malamud, D., 1974. Decreased protein catabolism during stimulated growth. FEBS Lett. 46:308.

Lands, A. M.; Arnold, A.; McAuliff, J. P.; Luduena, S. P.; Brown, Jr. T. G., 1967. Differentiation of receptor systems activated by sympathomimetic amines. Nature 214:597.

Li, J. B.; Jefferson, L. S., 1977. Effects of isoproterenol on amino acid levels and protein turnover in skeletal muscle. Amer. J. Physiol. 232:E243.

Massondi, M.; Evans, E.; Miller, D. S., 1983. Thermogenic drugs for the treatment of obesity, screening using obese rats and mice. Anim. Nutr. Metab. 27:26.

Mersmann, H. J., 1979. Endocrinology of adipose tissue and fat cell metabolism. Reciprocal Meat Conference Proceedings, 32:93.

Moser, R. L.; Dalrymple, R. H.; Cornelius, S. G.; Pettigrew, J. E.; Allen, C. E., 1984. Evaluation of a repartitioning agent on the performance and carcass traits of finishing pigs. J. Animal Sci. 59:255 (Suppl. 1) Abstr. 269.

National Pork Producers Council, 1982. Procedures to evaluate market hog performance. 2nd Edition. National Pork Producers Council, Des Moines, Iowa.

Ricks, C. A.; Dalrymple, R. H.; Baker, P. K.; Ingle, D. L., 1983. Use of a beta-agonist to alter fat and muscle deposition in steers. Fed. Proc. Soc. Exp. Biol. 42:816 (Abstr.).

Ricks, C. A.; Baker, P. K.; Dalrymple, R. H.; Doscher, M. E.; Ingle, D. L.; Pankavich, J. A., 1984a. Use of clenbuterol to alter muscle and fat accretion in swine. Fed. Proc. Soc. Exp. Biol. 43:857 (Abstr.).

Ricks, C. A.; Dalrymple, R. H.; Baker, P. K.; Ingle, D. L., 1984b. Use of a beta-agonist to alter fat and muscle deposition in steers. J. Anim. Sci. (in press).

Tischler, M. E., 1982. Hormonal regulation of protein degradation in skeletal and cardiac muscle. Life Sci. 28:2569.

van Es, A. J. H., 1977. The energetics of fat deposition during growth. Nutr. Metab. 21:88.

Wolfe, D. F., 1983. Manipulation of cattle for daylight calving. Mod. Vet. Pract. 64:21.

Trichina-safe Pork from Irradiation Processing

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INTRODUCTION

For a number of years the American swine industry has been concerned with the problems associated with trichinae in pork. Although trichinae in pork is not a serious health problem in this country, it disproportionately affects consumer behavior. Recently, the use of irradiation to inactivate trichinae in pork has been proposed as a mechanism to produce trichina-safe pork. The purpose of this paper is to discuss this process and examine some of the implications to the swine industry.

THE TRICHINOSIS PROBLEM

Trichinosis is a disease caused by a very small parasitic nematode called Trichinella Spiralis. The parasite can be found in many wild animals, carnivorous domestic animals and humans. The incidence of trichinosis in the United States population peaked in the 1930's and 1940's. It has been estimated that in 1947 there were 20 million cases of human trichinosis. This number has decreased greatly and in 1981, only 188 human cases were reported with one associated death. It should be noted that many of the human infections are probably not severe and therefore, go unreported. The incidence of trichinae infected pigs went from 9.5 per 1,000 in the 1930's to 1.25 per 1,000 in the 1960's. Given this incidence of infection, there could be 100,000 infected pigs slaughtered per year (Ref. 5).

The life cycle resulting in infection with Trichinella Spiralis is shown in Figure 1. The infected muscle contains encysted trichinae larvae, and after the muscle is eaten by another animal, the cyst wall is broken down releasing the larvae. The released larvae burrow into the intestine wall and go through maturation and reproduction in two to four days, resulting in thousands of larvae which will continue to reproduce until the host animal builds up an immunity to the organisms. Some young larvae will enter the circulatory systems and be dispersed to all tissues; however, only those in striated muscle will survive. Larvae in the muscle will increase in size and become encysted. Once in the encysted state, larvae are alive but dormant. The cycle begins again when the muscle containing the encysted larvae is eaten by another host. Infection can

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cause muscle pain and weakness in the host.

Some of the losses associated with trichinosis are listed in Table 1.

Table 1. Losses associated with trichinosis

1. Problems associated with human suffering, such as loss of human productivity, loss of quality of life, and medical treatments.
2. Reduced demand for American Pork in both domestic and foreign markets.
3. Educational expense for proper cookery of pork.
4. Swine morbidity.
5. Costs associated with obtaining trichina-safe pork (heating, freezing, irradiation, etc.).
6. Inspection program of trichina regulations.

From Ref. 5

CHARACTERISTICS OF AMERICAN PORK INDUSTRY

In America, more hogs are produced than in any other nation. It was estimated that 88 million hogs were slaughtered in 1983 with a cash receipt value of approximately 9.7 billion dollars. The number of hogs slaughtered per year has remained relatively constant for the past 20 years but the amount of pork from these animals increased slightly (Table 2).

Table 2. Hogs slaughtered and pork production

Year	Slaughter animal number in thousands	Meat Production Pounds in millions
1963	87,117	12,427
1968	86,417	13,064
1973	77,890	12,751
1978	78,417	13,393
1983	88,084	15,202

From Ref. 8

Hog production is concentrated in the midwest as is hog slaughter. Most of these activities are centered in Iowa, Illinois, Indiana, Minnesota, Nebraska and Missouri.

The number of plants slaughtering hogs has decreased with large plants slaughtering over 100,000 animals per year accounting for approximately 90% of all hogs slaughtered. In 1983, 104 federally inspected plants (7.8% of total) slaughtered 92% of all hogs (Ref. 8). The obvious trend has been that fewer large plants slaughter an increasingly greater percentage of the animals. Currently, major changes are underway with several progressive companies investing heavily in hog slaughter operations.

PORK CONSUMPTION

The consumption of retail pork has been relatively constant over the past 20 years (Table 3), while beef has shown small gains. The greatest changes have taken place in poultry consumption which has shown a steady increase resulting in 70% more poultry used in the past two decades. These trends indicate that more emphasis should be placed on increasing and maintaining the pork market share.

Table 3. Per capita disappearance of meat in pounds on a retail weight basis

Year	Pork	Beef	Lamb	Poultry	Fish	Total
1963	61	70	4	38	11	184
1968	61	81	3	45	11	201
1973	57	81	2	49	13	202
1978	56	87	1	56	13	213
1983	62	79	2	65	13	221

From Ref. 8

Several unusual features concerning pork utilization should be mentioned. Pork products have a rather unique role as the meat most commonly served with breakfast (Ref. 7). Second, a very large percent of pork (as much as 70%) is further processed and sold as hams, bacon, sausage, etc.

Table 4 lists the price per pound of beef, pork, chicken and turkey over the past 17 years. Because of the methods used by the U. S. Government to compile these numbers, a direct comparison of 1965 to 1983 prices is not totally valid; however, the trend may be correct. This table indicates the price of poultry has not increased as rapidly as red meat. Again, a direct comparison of poultry

Table 4. Meat price per pound in dollars.

	1965	1975	1983	% Increase over 1965
Beef	.82	1.55	2.38	190
Pork	.65	1.35	1.70	162
Broilers	.39	.63	.73	87
Turkey	.48	.73	.92	91

From Ref. 8

prices to red meat prices may not be totally valid, but it is obvious that poultry is cheaper on a per pound basis than either pork or beef.

FACTORS ADVERSELY AFFECTING CONSUMER DEMAND FOR PORK

In Table 5, four factors affecting the utilization of pork are listed. Of these concerns, the one dealt with in this paper is trichinae infection. Although trichinae infection is not a major health problem in America today, the American public has such an aversion to parasites that it has a disproportionately large negative effect on pork consumption attitudes.

Table 5. Factors adversely affecting consumer demand for pork

1. Religious custom.
2. Trichinosis
3. Health concerns associated with consumption of animal fat.
4. Health concerns associated with consumption of nitrite and excess salt.

From Ref. 7

CONTROL OF TRICHINOSIS

Control of trichinae infection in pork or its elimination is a very desirable goal with a number of positive economic incentives which could include increased consumer demand for pork and possible increased pork exports.

Table 6. Methods of eliminating trichinae in the pork supply

1. 100% elimination of trichinae from swine by blocking a vector.
2. Guarantee trichinae free pork carcasses by inspection of each animal at slaughter.
   A. Microscopic evaluation.
   B. Blood sampling.
3. Destruction of trichinae in pork.
   A. Freeze meat under specific conditions.
   B. Heat meat to specified temperatures.
   C. Irradiation of pork.

There is a need for a simple, cheap method of inactivating trichinae in pork that would not greatly change production practices and would allow the industry to satisfactorily handle the small number of animals that are infected. Table 6 lists some of the methods used to combat trichinosis. Several of these methods are not economically feasible or are technically difficult to complete. Of the methods listed to inactivate trichina, it would not be desirable to freeze or cook all pork; however, irradiation of pork carcasses does hold promise as an inexpensive and safe procedure to meet these objectives.

FOOD PRESERVATION BY IRRADIATION

Food preservation by irradiation normally uses a radioactive source such as cobalt-60 to provide high-energy gamma rays that penetrate tissue and can be fatal to living tissue such as bacteria, parasites and insects. At a food irradiation facility, the gamma rays at a controlled dose pass through food, much like microwaves pass through food. A dose of 100 Krad will pasteurize most food (a chest x-ray is less than 1 rad). These gamma rays do not induce any radioactivity in food and do not release heat or change the foods gross physical state. Benefits are many and vary with the food, but generally, the number of microorganisms are reduced, shelf life extended, and food loss due to spoilage is reduced (Ref. 6). In other foods, insects can be destroyed and in the case of pork, parasites can be inactivated.

It is estimated that 28 countries including Japan, Israel, France, Holland

and the Soviet Union have some level of clearances for commercial irradiation of more than 50 food stuffs. The foods range from vegetables, poultry, fish and grain to nuts. It is estimated that about 100 food irradiation plants are operated world wide (Ref. 2). In America, only one food irradiation facility exists and it is used to treat export foods. Currently, our country has regulations that allow for irradiation of spices, natural flavorings and dehydrated vegetable seasonings. The FDA is currently reviewing its policy on use of low doses of gamma radiation to treat citrus, grapes, cherries and meat.

IRRADIATION OF MEAT

Food irradiation can be divided into three categories according to the dose applied (high, medium and low). High dose is above 1000 Krad, a medium dose is 100 to 1000 Krad and low dose is below 100 Krad.

Some potential uses of irradiation of meat products are listed in Table 7.

Table 7. Potential applications of irradiation for meat products.

1. Reduction of salmonella on meat or poultry.
2. Control of pork trichinosis and toxoplasmosis.
3. Control of beef parasites.
4. Reduction of nitrite levels in cured meat while maintaining botulism protection.

From Ref. 3.

The use of high-dose irradiation for the sterilization of meat, poultry and fish is of much interest because of the reduced energy requirements. The estimated irradiation dose required to sterilize meat and have an acceptable safety factor varies from 2550 Krad for pork sausage to 4370 Krad for pork. Currently, research is focused on high-dose treatment of poultry where detailed toxicity and wholesomeness studies are now being performed. Also, research has been done on irradiating cured meats in order to provide protection against Clostridium botulism and also as a means to reduce sodium nitrite. Experimental results indicate that irradiation of vacuum packaged bacon at a dose of 1000 to 1500 Krad at 5°C with 40 ppm nitrite will afford significant protection (Ref. 3).

Treatment with medium irradiation doses of fresh meats and poultry prior to refrigerated storage can reduce bacteria and other microorganisms resulting in extended shelf life. For example, beef irradiated under the proper set of conditions with 100 to 200 Krad can be maintained in a retail case for much longer periods of time, up to 21 days. Also, medium irradiation doses can be used to control salmonella in fresh chicken and turkey. Studies of this nature using both poultry and fish are being conducted in Canada (Ref. 3).

The use of low doses of irradiation by the red meat industry has focused on control of trichinae in pork. Experiments have indicated that irradiation will effectively inactivate the trichinae in pork thus preventing transmission to humans. As a generality, most irradiated products are wholesome and acceptable, nontoxic, and nutritionally unaltered.

CONTROL OF TRICHINOSIS BY IRRADIATION

Several university and government groups have worked on these problems and results indicate that irradiation of ground pork with 20 Krad will substantially inactivate most trichinae and a 30 Krad dose will result in complete inactivation. Doses as low as 10 Krad will inhibit production of most second generation larvae in pork and 20 Krad will reduce maturation of encysted larvae in infected pork. In other studies using cobalt-60 irradiation of hog carcasses, 11 Krad was sufficient to sterilize female trichinae. Also, low level irradiation of hog carcasses can inactivate first and second generation larvae. Low level irradiation of hog carcasses also inactivates Toxoplasma gondii, a microorganism causing brain and eye lesions.

Design of an irradiation facility for hog carcasses needs to provide for the irradiation properties of the source, product response to radiation, and protection for workers. The primary component of a food irradiation system is either a cobalt-60 or cesium-137 source. Other components in addition to the radiation source include a conveyor to transport the pork through the flux field, shielding of the source to protect workers and a control system to allow the safe use of the facility. A schematic of a pork carcass irradiation facility is shown in Figure 2.

ESTIMATED COST OF IRRADIATION OF PORK

A cost analysis study has been undertaken from the limited numbers currently available (Ref. 5). The costs of irradiation per hog are listed in Table 8. In construction of this table, a number of assumptions were made for a carcass irradiation facilitiy operating 16 hours per day and yielding a 25% return per year on capital investment.

Table 8. Cost of irradiation per animal.

Plant capacity hog/day	Capital cost $/hog	O&M Cost $/hog	Total cost $/hog
1,000	.614	.340	.954
4,000	.285	.100	.385
10,000	.219	.052	.271

From Ref. 5

With capital costs being recovered in 4 years, the numbers in Table 8 for total cost per hog are for the first four years of operation. As listed for the smaller 1000 hog per day plant, the total cost per hog for the first four years would be 95 cents per animal, but beginning the fifth year, the cost would drop to 34 cents per animal provided the O&M costs remain constant. This cost analysis indicates the total cost per animal for irradiation is small; however, the initial capital outlay is large and thus would be of significance to the packer-processor.

CONSUMER ACCEPTANCE OF IRRADIATED PORK

To date, evaluation of consumer demand for irradiated pork has been difficult to quantitate. Table 9 is a partial list of both positive and negative factors

associated with consumer acceptance of irradiated pork.

Table 9. Potential factors affecting consumer acceptance of irradiated pork.

Potential positive factors:

1. Consumer belief that product is safer, no active parasites
2. Fewer cooking requirements, more variety available
3. Less negative publicity concerning pork
4. Increase export market

Potential negative factors:

1. Consumer belief that irradiation of food is unhealthy
2. Negative publicity by anti-nuclear groups
3. Problems associated with uneducated public

From Ref. 5.

Although there is little data concerning consumer acceptance, there has been some evaluation of the problem (Ref. 5). Various scenarios are possible for the implementation of irradiated pork. In scenario 1, elimination of trichinae results in an immediate 2% increase in demand for pork and a 1% long term increase and an expansion of exports. This could result in increased profits of $493 million per year in the short run, primarily through increased price. In the long run, the quantities of pork sold would increase resulting in a yearly $130 million increase.

In scenario 2, the domestic consumer reacts negatively to irradiation by reducing demand 2% and long term demand remains down but pork export is up. This results in a short run loss of about 393 million dollars to the industry. The industry group absorbing most of this short term loss would be producers. In the long run, there may be a small total dollar gain but this would go primarily to the packer. When one compares the above two scenarios, it becomes evident that if either is true, the producer will reap most of the short term gain or loss while the packer could profit under either scenario.

PORK EXPORT

Currently, America is a net importer of pork, much of which can be attributed to canned hams coming from Denmark and other European nations and fresh or frozen pork from Canada. Table 10 shows the major pork importing countries in the world and also the countries importing significant amounts of American pork. The export of American pork has been about 3-4% of production for the past five years. The 1983 pork exported had a value of about $183 million and the imported pork that year had a value of $610 million (Ref. 8). Several European countries do not import much American pork because it does not meet their trichinae regulations. It should be noted that irradiation of foods is approved in several of these countries. It is possible that an irradiation program could remove a hurdle to exporting pork; however, even if this hurdle is removed, many others including foreign subsidies to the pork industry and the stron dollar remain major obstacles.

Table 10. Countries importing pork.

Major Pork Importing Countries	Major Importing Countries of U. S. Pork
United Kingdom	Japan
West Germany	Mexico
Italy	Canada
France	Dominican Republic
Japan	Venezuela
U.S.A.	
Soviet Union	

From Ref. 5

SUMMARY

There does not appear to be any major obstacle to stop the use of irradiation of pork to inactivate trichinae. The technology involved has been proven and the low dose level would not effect meat quality. The FDA has shown a willingness to consider irradiation of pork; however, the labeling requirements for irradiated products is unclear (Ref. 9 and 10).

What is clearly needed now is a better analysis of the potential benefits and drawbacks of this technology. The producers could benefit from a larger market; however, major questions remain about the consumer acceptance of irradiated pork. Two such consumer areas are the perception of safety associated with irradiated food (pork) and the value of irradiation technology. At another level is the acceptance of pork irradiation by the packer. The packers' costs, etc. need to be documented so they can assess the risks and benefits.

Presently, a number of things are happening on the national scene that affect the potential application of irradiation to pork. First, the FDA is evaluating low dose irradiation of foods, including pork, and it is possible rules governing the use of irradiation to inactivate trichinae will be issued. Another very productive development has been the efforts put forth by the National Pork Producers Council, USDA and the U.S. Department of Energy to support evaluation of the pork irradiation process. These efforts have led to a detailed preliminary study [(Ref. 1 and 5) (much of which is reported in this paper)] and the potential construction of a demonstration facility. There are plans to construct a small but functional demonstration pork irradiation facility at a university or commercial plant. The purpose of the demonstration facility would be to assess all aspects of pork irradiation on a much larger scale than has been possible in the past.

REFERENCES

1. Consumer Reaction to the Irradiation Concept. (1984) Prepared by Wiese Research Assoc., Inc. for the U. S. Dept. of Energy and the National Pork Producers Council.
2. Food Irradiation: A Fresh New Look. (1983) Prepared by Atomic Industrial Forum, Inc.
3. Meat Irradiation Is Here But Is It Now? (1983) By J. S. Sivinski. The National Provisioner, Nov. 12, 1983.
4. Facts About Low-Level Radiation. (1981) Prepared by the International Atomic Energy Agency, World Health Organization.
5. Trichina-Safe Pork By Gamma Irradiation Processing, A Feasibility Study. (1983) Prepared by CH_2M Hill Central, Inc., for U. S. Dept. of Energy.
6. Preservation of Food by Ionizing Radiation. (1983) Vol. 2 & 3. Editors: E. J. Josephson and M. S. Peterson, CRC Press.
7. Pork in the American Fast Food Industry (1983) by P. J. Bechtel and F. K. McKeith. In: Pig News and Information 4: 35.
8. Meat Facts 1984 Edition, prepared by American Meat Institute.
9. Irradiation in the Production, Processing and Handling of Food; Proposed Rule (1984). In: Federal Register, Feb. 14, 1984.
10. Legal Considerations Concerning Food Irradiation (1983) by E. L. Korwek. In: Food Technology 37: 38.

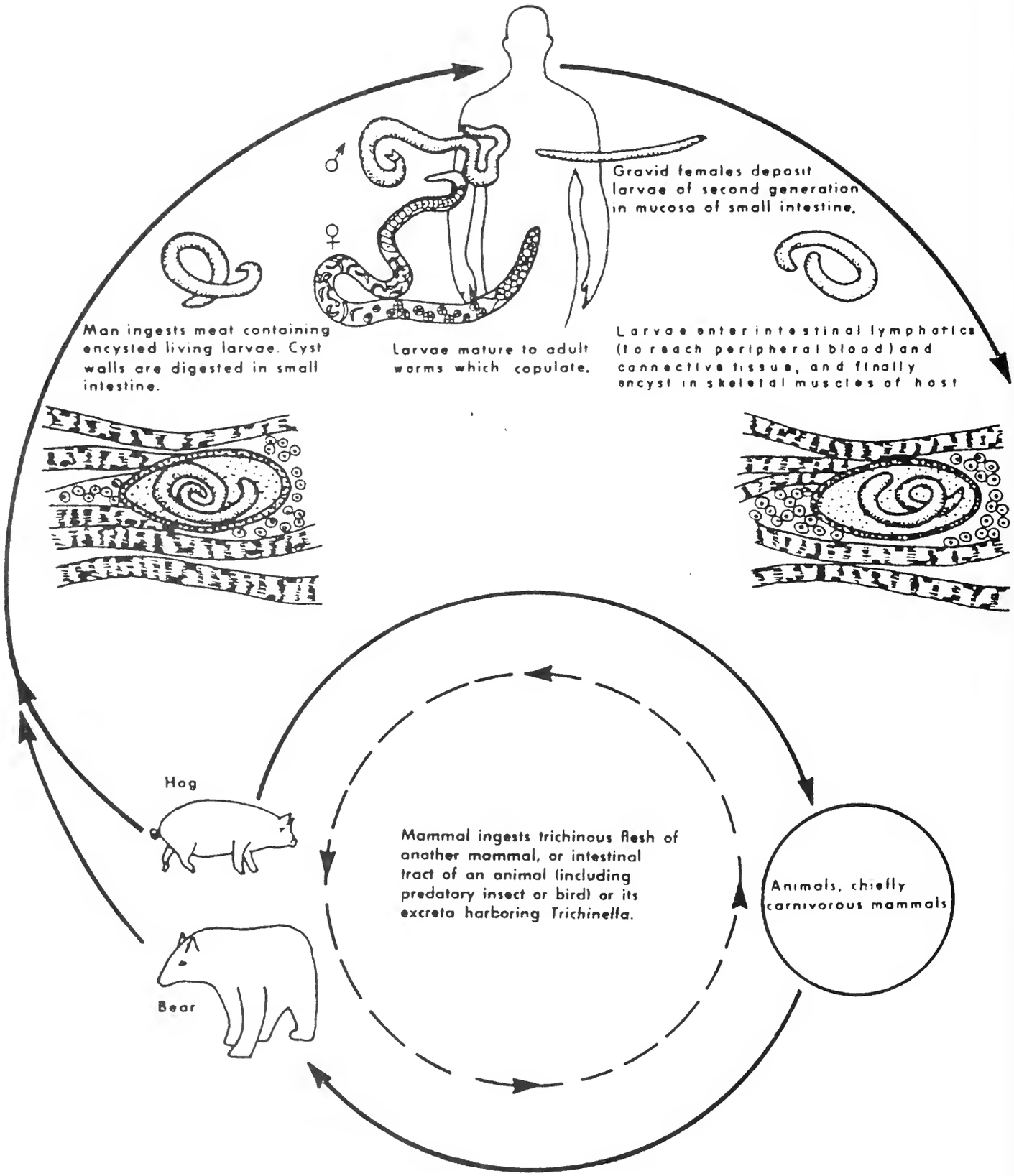

Figure 1. The Trichinosis Cycle
(Figure from Ref. 5)

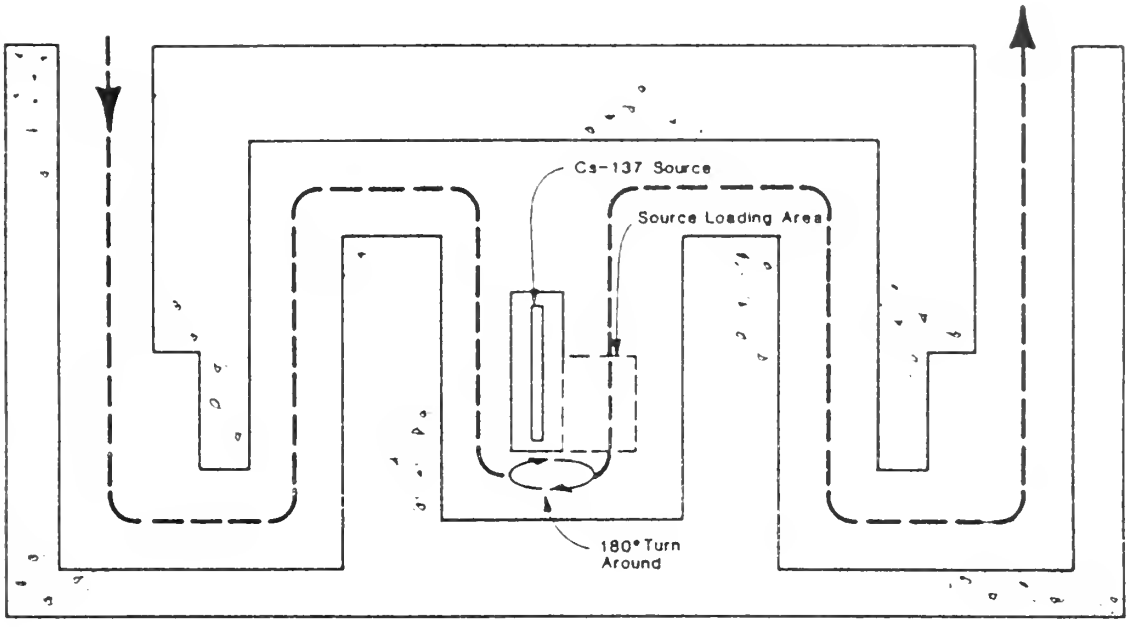

Figure 2. Possible Pork Irradiator Design
(Figure from Ref. 5)

When the Facts Are Made Available: Physician and Public Perceptions of Red Meat in the Diet

BURDETTE C. BREIDENSTEIN, Ph.D.

A recent issue of a publication put out by a well-known consumerist organization contained an article which said, "Most cuts of fresh pork derive more than 50% of their calories from fat."(1) This is right as far as it goes, of course, at least in the case of untrimmed pork. Because fat contains twice as many calories as protein, a cut of pork need only be 13% fat to derive 50% of its calories from the fat component.

The article also says that this is a "a poor comparison to a truly lean meat, such as chicken which gets only around 19% of its calories from fat when the skin is removed," and elsewhere states, "The average untrimmed three-ounce serving of pork supplies 284 calories."(2)

How many health-conscious consumers will notice, as I'm sure you all did, that the comparison is being made between untrimmed pork and skin-off chicken, and will understand that most of the fat in chicken is in or attached to the skin?

Furthermore, how many consumers will be able to go to USDA Handbook 8-5 and learn that, even with the skin removed, the average over the entire roast chicken carcass is 35%, not 19%, of calories from fat? (The 19% figure is for breast meat only, with the skin removed; for comparison, the thigh portion with the skin removed derives 47% of its calories from fat.)(3)

Unfortunately, consumer perceptions of the role of red meat in the diet rarely are based on Handbook 8. This is readily understood, and the avenues which nutrition misinformation travels from its source---diet/health extremists---to the innocent consumer are obvious. Two advertisements which the article in Nutrition Action criticized, one from the Meat Board and one from the National Pork Producers Council, based their figures on Handbook 8 and USDA Handbook 456. But when a reporter is looking for a story on nutrition and health, he almost certainly will go to a consumerist publication for his data and not to a magazine ad bought and paid for by the meat industry. After all, everyone knows that consumerists exist solely for the purpose of protecting the public, and industry exists solely for the purpose of making money any way it can.

Thus the consumer is misled. When it comes to the health professional, on the other hand, one would expect more assiduous checking of data, certainly less reliance on hearsay, resulting in a better overall understanding of the issues at hand.

However, this is not necessarily the case. The Meat Board presently is conducting an advertising campaign in such professional publications as the Journal of the American

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Medical Association and the New England Journal of Medicine. The ads present some basic data on the fatty acid profile and nutrient composition of red meats, and offer further information to interested parties who send in a request.

We have been gratified at the interest shown in the types of information carried in the ads. But it also may be viewed as a sort of "there's some good news and some bad news" situation. That is, I'm very happy that so many physicians have written in asking for reliable information on the nutrient profile of meat. I also am dismayed at the tone of some of the letters. These physicians often appear extremely skeptical of information which has been readily available in the nutrition literature for decades.

In fact, a few months ago I received a letter from a physician who had seen some of our Meat Board literature. The letter expressed considerable surprise at such claims made in our publications as that beef fat is only about 48% saturated and pork fat is only about 36% saturated. The letter politely asked that I provide the author with such citations as I might have that would support these claims.

I responded that the fatty acid profile of beef can be derived from information published on pages 177-186 of USDA Handbook #456, Nutritive Value of American Foods in Common Units, which publication has been one of the two or three main information sources for nutritionists since 1975.

That an individual physician did not know about this publication is not necessarily all that shocking. In this particular instance, however, one further fact should be mentioned: The physician was the head of the Department of Clinical Nutrition at a major hospital which had acted as one of the Lipid Research Clinics in the enormous National Heart, Lung, and Blood Institute Coronary Primary Prevention Trial which was reported (erroneously) in the press throughout the country earlier this year as having provided the final link between fat consumption and heart disease. I feel it is virtually certain that he has had a copy of Handbook 456 conveniently shelved within a few feet of his desk for the past nine years.

Of course, I was pleased to provide the man with information on the fatty acid breakdown of red meats. I might have wished he had looked into the matter before becoming one of the two or three dozen physicians who engineered the Coronary Primary Prevention Trial, however. I most assuredly wish he had investigated the fat content of red meats before being quoted in media across the country as recommending that people reduce their meat intake because of all the fat in meat.

On exactly which issues surrounding red meat and health are consumers and physicians most confused? I am afraid that I would have to say exactly all of them. However, there are some specific areas in which perceptions are radically divergent from the facts.

Among these, of course, are the total fat content, saturated fat content, and cholesterol content of red meats as compared with other flesh-source foods. One of the most commonly offered physician recommendations is that the patient cut back on red meat consumption. (From what level we are supposed to cut back, I will deal with presently.) The most common prescription probably is to replace a lot of that red meat with chicken or fish, thereby reducing total fat, saturated fat, and cholesterol in the diet.

Three ounces of cooked lean pork provides about 11 grams of fat, representing 99 calories from fat. Of this, 36% is saturated fat and the rest is monounsaturated and polyunsaturated. This fraction is the equivalent of sightly less than 4 grams of saturated fat, or about 36 calories.(4)

(It should be pointed out here that the American Heart Association recommends we get no more than 30% of our total daily calories from fat, and no more than 10% from saturated fat.(5) The fat in the serving of pork referred to here provides less than five percent of an already low 2,000-calorie diet, and the saturated fat part provides less than 2%.)

What if we were to change from pork to chicken---roast chicken with the skin off, say? How much of a savings in calories from fat and from saturated fat would we accomplish?

Well, three ounces of chicken with the skin removed provides 6.3 grams of fat, or 57 calories' worth. The fat in chicken is 28% saturated, as compared with 36% in pork, resulting in a total of 16 calories' worth of saturated fat in the serving of chicken.(6)

This represents a savings of a total of 42 calories from fat by switching from pork to chicken, and a savings of 20 calories from saturated fat.

To get more than 30% of a 2,000-calorie diet from the fat in cooked lean pork, a person would have to eat six three-ounce servings of pork a day. To get more than 10% of a 2,000-calorie diet from the saturated fat in cooked lean pork, he would have to eat 5.6 servings.

Which brings us to another widely accepted fallacy: Americans consume far too much meat. It is on the assumption that we eat giant steaks and huge slabs of ribs and six hot dogs at a sitting that doctors so often tell their patients to eat less red meat. Well, less than what?

The red meat industry finds carcass weight and retail weight figures extremely useful in a variety of production contexts. Only relatively recently did we open our eyes to the way these figures had been used to calculate the dietary contribution of red meat in America. This use is very misleading, of course. To say that Americans consumed a carcass weight of 170.8 pounds of red meat per capita in 1983 says very little about the actual nutritional contribution of that amount after bones and fat are removed in the processing facility, and more bones and fat are removed at the retail level, and, often, still more bones and fat are removed in cooking and serving. Assuming that we all consume fully 50% of the fat remaining on every cut of beef, pork, lamb and veal we're served (probably an overstatement of trimmable fat consumption), we see that the average per capita red meat consumption in this country is 3.99 ounces.

This 3.99 ounces---which includes all processed meat, too---contributes, on the average, only 214 calories from fat, of which only 100 are calories from saturated fat. It contributes only 87 milligrams of cholesterol, or 29% of the 300 milligrams the American Heart Association has identified as a recommended upper limit.

It also contributes the following percentages of the Recommended Dietary Allowances for an adult male: iron, 25%; thiamin, 21%; vitamin B-12, 71%; protein, 46%; zinc, 32%. These contributions must be weighed against a contribution of calories from fat of only 10.7% of a 2,000-calorie diet and calories from saturated fat of only 5% of a 2,000-calorie diet.(7)

It is my belief that very few of the doctors who automatically tell their patients "Cut down on your meat consumption" understand that, on the average, Americans are eating 4 ounces of red meat per day, while the accepted dietary recommendation for a healthy adult is two 2-3 ounce servings from the meat group.(8)

That is, a doctor who urges his patients to cut back on meat consumption might reconsider if he understood that, of all the broiling steaks produced in America from all the steers, all the heifers and half of all the cows, stags and bulls were cooked to 10-ounce boneless weight and were divided among the population, it would amount to one steak per person every five weeks. Eliminating children under five, half the children aged 5-13 years, and half the over-65 group still won't get the total over one such steak every four weeks.

What other misunderstandings cause problems when it comes to the diet/health issues and red meat consumption? Sodium and hypertension is a very good example. I think there probably is not one person in this room who doesn't know somebody who has quit eating salt or has reduced salt intake for health reasons, even though he or she has never shown any signs of high blood pressure. Since salt is a component of most processed meat products, avoiding salt to many people means avoiding processed meats. (To many, it also means avoiding meat, even though fresh meats contain so little sodium they can be included on strict low-sodium regimens.)

However, the best available scientific evidence on the subject is represented by a study by the Federation of American Societies for Experimental Biology. The only conclusion in this study is that about 20% of Americans are hypertensive or borderline hypertensive, and that 15% of these individuals are salt-sensitive. Fifteen percent of 20% equals a total of 3% of the populace that should restrict their intake of salt.

Consumption of fat has been linked with cancer. Few people understand, however, that the expression "has been linked with" is not synonymous with "has been shown to cause." Nowhere in the public media did I notice any reference to articles which appeared in the New England Journal of Medicine and the Journal of the American Medical Association earlier this year. One of these articles concluded, "...information about specific dietary factors is generally inconsistent or incomplete. It is our own belief that available data are not sufficient to serve as a basis for strong specific dietary recommendations. The preliminary recommendations of the National Research Council...that people should generally eat less fat and more fruits, vegetables, and whole-grain products (in addition to moderating alcohol intake and minimizing the consumption of certain processed foods) seem sensible, not so much because of the firmness of any expectation that cancer rates will be lowered by such actions, but"---and here I add my own emphasis---"because the changes are unlikely to be harmful and may well be beneficial in the context of other diseases."(9)

The other article arrived at virtually the same conclusion, and the author added a reference to a side of scientific controversy that the layman rarely sees: "Ordinarily, it is the responsibility of those advocating change to prove that the proposed changes will lead to a desired result. In this case, however, those who are not convinced that specific dietary changes are advisable have been put on the defensive. This is so even though there is no consensus among knowledgeable scientists on the issue of diet, nutrition and cancer."(10)

In other words, segments of the scientific community who support certain diet/cancer hypotheses happen to be holding the reins just now.

The public cannot be expected to pore over the pages of the Journal of the American Medical Association to determine whether they should or should not change their diet in the hope of averting cancer or heart disease. All they know is that the National Cancer Institute (11) and the American Cancer Society (12) have issued dietary recommendations to that effect. Similarly, all the public knows about diet and heart disease is what they read in the papers, and in the papers they have read that the American Heart Association has issued dietary guidelines intended to reduce the likelihood of heart disease.(13)

However, an article by an eminent biochemist in a recent issue of the American Journal of Clinical Nutrition takes the AHA severely to task. According to the author, the Heart Association, in the publication in which it intended to present the bases for its dietary recommendations, relied heavily on obsolescent research and out-of-context quotes. Often the AHA apparently even cited in support of its dietary recommendations studies in which the original researchers specifically stated that the dietary changes urged by AHA are ineffectual in controlling serum cholesterol levels.(14)

And so it goes. The public cannot be expected to derive from scientific journals its perceptions about the alleged relationships between meat and these chronic disease problems. Bulletins from the Federation of American Societies for Experimental Biology are not exactly gripping. Self, New Woman, Ladies' Home Journal, USA Today, Runner's World, Good Morning, America, these constitute the wellspring to which the public repairs to drink of the waters of nutritional wisdom. If a female tennis pro appears on a TV talk show and says that the leanest steak you can buy is 40% fat or the bouncy little host of a TV exercise program says you should never eat pork because it's mostly fat or a well-known consumerist publication makes inappropriate dietary comparisons between pork and chicken, these wil be incorporated by the public into its beliefs about the role of meat in the diet.

The New England Journal of Medicine? Boring. Besides, how could a publication that doesn't even have a picture of a woman in exercise garb on its cover possibly have anything important to say about nutrition?

A study commissioned by the American Meat Institute and the National Live Stock and Meat Board resulted in a segmentation of the consumers surveyed into five groups. One of these was identfied as "health-oriented," and constituted 17% of respondents. It was the second-highest group in income, and had the worst perception of red meats. The segment identified by the name "active lifestyle," constituting 16% of respondents, had the second worst attitude toward red meat; these respondents were the highest-paid of all.

The two groups also were far better educated than the rest of the people surveyed.(15)

In other words, the two groups best able to afford meat, and presumably best equipped to evaluate the diet/health claims, most wholeheartedly have accepted the allegations regarding the role of red meat in chronic disease.

In my opinion, and in the opinions of the authors of most of the scientific papers I have mentioned today, among others, this is not because the diet/health hypotheses are so evidently true that they cannot be rejected. Forces which have influenced this educated, affluent segment of our population to accept these unproven hypotheses as simple fact are many and complex, but neither the demonstrable accuracy of the hypotheses nor the consensus of scientific opinion is among them.

It remains the role of responsible scientists to work in the direction of making the public aware of sources of scientific knowledge beyond lady tennis players and TV game-show hosts. In an era when more and more consumers are seeking maximum nutrition in a relatively low-calorie package, the red meat industry's products are among the most nutrient-dense in the food marketplace. This is the sort of simple, direct fact that the public seems to prefer. It is my hope that, when the simple facts are made available, the public will have the wisdom to act appropriately.

Bibliography

1. Zuckerman, S., Pigging out---the 'lean' way. Nutrition Action 11(7):5, Sep. 1984.

2. Ibid.

3. USDA/SEA, Composition of foods/poultry products/raw, processed, prepared, agriculture handbook No. 8-5. Washington: GPO, Aug. 1979.

4. USDA/SEA, Composition of foods/pork products/raw, processed, prepared, agriculture handbook No. 8-10. Washington: GPO, Aug. 1983.

5. Grundy, S.M., et al., Rationale of the diet-heart statement of the American Heart Association, Report of the Nutrition Committee. Circulation 1982;65:839A-54A.

6. USDA/SEA, Composition of foods/poultry products/raw, processed, prepared, op. cit.

7. Breidenstein, B.C., Contribution of Red Meat to the U.S. Diet. Chicago: National Stock and Meat Board, 1984.

8. USDA/SEA, Food. Home and Garden Bulletin No. 228, Washington, D.C., 1979.

9. Willet, W.C., and MacMahon, B., Diet and cancer---an overview (second of two parts). N. Engl. J. Med., 310(11):697-703, Mar. 15, 1984.

10. Pariza, M.W., Perspective on diet, nutrition, and cancer. JAMA 251(11):1455-1458.

11. National Cancer Institute, Diet, nutrition and cancer prevention: a guide to food choices. NIH Publication No. 84-2711, Washington, D.C., Sep. 1984.

12. American Cancer Society, Eating the right stuff: ACS anti-cancer dietary recommendations. Cancer News 38(2):4-6, Spring/Summer 1984.

13. Grundy, S.M., et al., op. cit.

14. Reiser, R., Commentary on the rationale of the diet-heart statement of the American Heart Association. Am. J. Clin. Nutr. 40:Sep. 1984, 654-658.

15. Yankelovich, Skelly and White, Inc., Consumer Climate for Meat Products. Chicago: National Live Stock and Meat Board/American Meat Institute, Sep. 1984.

Swine Behavior and How It Affects Total Reproductive Efficiency

STANLEY E. CURTIS AND HAROLD W. GONYOU

Profitable pork production depends on efficient swine reproduction. The sows' and boars' behavior makes reproduction possible. The animals' behavior also can reduce reproductive rate. Weak libido and poor mating behavior in boars and fighting between dry sows necessitate either extra capital investments or special attention by stockmen. On most farms, they demand both. But behavioral problems in swine reproduction do not end there.

Effects of the sorts just mentioned come before farrowing, sometimes long before. We shall emphasize here behavioral constraints on swine reproduction that come at or after farrowing instead. These include problems in both piglets and sows. Postnatal and postpartal constraints are relevant because more than one of every five liveborn piglets dies before weaning, thus accounting for a large part of the swine industry's total reproductive inefficiency.

THE PRINCIPLES

Sow Behavior Before and During Farrowing

The sow is strongly driven to build a maternal nest a day or so before farrowing. The nest's purpose is to provide a warm, safe niche for the piglets soon to be delivered. Even in an environment--such as an unbedded farrowing crate--devoid of nest material, most sows engage in vigorous phantom nest-building for several hours sometime during the day before farrowing begins. During the two days preceding farrowing, the sow becomes restless. This restlessness peaks and then subsides as farrowing is approached. Shortly before the first piglet is born, the sow begins to lie quietly on her side nearly all the time. Savage sows sit and stand longer than do others, although the cause(s) of savaging are still unknown. Sows in pens sit up or stand up less often than do those in crates. Also, crated sows show more rooting, pawing, and other stereotyped behaviors, than do penned ones. The

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presence of straw affects neither the times sows get up nor the time they spend sitting or standing. The question has been raised as to whether behavioral differences between sows in these two environments--pens and crates--are accompanied by endocrine phenomena which result in the delivery of weak piglets.

Piglet Behavior Just After Birth

In comparison with other newborn mammals, piglets are delivered quite easily but then left on their own to escape the birth membranes and locate a teat. Most piglets are vigorous and quickly break out of the placental membranes. They ordinarily take their first breaths within a few seconds after delivery. Some piglets are sluggish at birth, and may die before breathing or finding a teat. These are often piglets that have experienced a long or difficult delivery. In these cases the umbilical cord often shows signs of ruptured blood vessels, suggesting the piglet was cut off from the vital placenta for too long a period during the birth process. This happens mostly in piglets born in the latter half of the litter.

Once the piglet has taken its first breath, it soon stands and then immediately tries to find a teat. A few piglets suckle the first time within five minutes after birth; others may take several hours, and a few never do locate the mammary glands. Most piglets will be at the udder within 30 minutes of birth. Locating a teat early is important for several reasons. First, the piglet does not have much energy reserve at birth. If it does not find a teat quickly, it can become fatigued and chilled. Once a teat is located, the piglet can restore its energy status with the nutrients in milk. Piglets that take more than an hour to locate a teat for the first time often lie down during that time and display only short bursts of teat-seeking behavior afterward. Second, the supply of colostrum lasts for only a short time. The last few piglets in a litter may obtain less immunity from the sow, especially if littermates born earlier have been very active sucklers. Third, the sow's lacteal secretion may not be readily available if the piglets wait too long. During parturition and for a short time afterwards, colostrum or milk is available from the teats continuously. But as soon as 30 minutes after the sow completes delivery of the litter, the secretion may cease flowing freely, and thereafter be available for only seconds during short nursing bouts about once an hour.

Once standing, the piglet uses its nose to search for a teat. The piglet investigates any vertical object it comes in contact with by several pushes with its snout. Piglets also open and close their mouths at this time, as if groping for a teat. Nosing and gaping are particularly evident when a soft object, such as the sow's rump or udder, is touched. As the piglet improves its coordination, nosing activity increases until two to four hours after birth, after which time the piglet begins to lose interest. It is not clear what stimuli, if any, play major roles in directing the piglet's search toward a teat. Some believe the hair patterns on the sow's belly, her grunts, or the warmth of her udder are important. It appears that soft, wet surfaces maintain the piglet's interest for some time, but this is probably of more importance once the udder is found than during the search.

If one watches several piglets, it is clear that some problem areas exist which delay the finding of a teat. Piglets spend a great deal of time nosing

the sow's rump and often suckle the tip of her vulva. It is difficult for piglets to get around the rear legs of the sow. Frequently, they return to the vulvar region rather than proceed around the legs to the udder. Piglets that wander to a side of the pen or crate may follow the solid wall around its periphery. Occasionally, a piglet begins to nose the corner of the pen and seems to be reluctant to move on.

Once a piglet has found a teat and suckled, it proceeds to sample other teats on the udder. In searching for a second or subsequent teat, piglets tend to restrict their nosing behavior to the same height at which they located the first one. During this teat-sampling phase, fighting usually occurs among the littermate piglets.

After a few hours, the piglets begin forming preferences for teats. This continues beyond the time of constant milk availability in some cases. The sow's front teats are generally preferred over the rear, and there is some evidence that very large or very small nipples tend to be avoided. But considerable variation in teat selection is left unexplained by current theories. Research is now being conducted on the actual milk yield of each teat and the possible relation between this and teat selection.

Sow Behavior After Farrowing

The postpartal sow spends most of the time lying down, provided she need not forage to fulfill her appetite. She must stand several times each day to facilitate ingestive and eliminative behaviors. As she lies down again, the chances are great that the sow will crush a piglet to death. The lactating sow in unrestraining quarters shows special care in the act of lying down; she appears to try to maximize her stability. An unusual hindquarter "squat" movement by sows in a pen has been contrasted to a hindquarter "flop" movement in a crate. Thus, sow behaviors protective of the piglets seem to have evolved, but some farrowing accommodations might interfere with them and thus jeopardize the piglets' safety.

The way the sow exposes her udder to the piglets at nursing time is also crucial to the survival of her young. Again, farrowing accommodations vary in regard to facilitating udder exposure by the sow.

Piglet Behavior During the Nursing Phase

During the first day or two after birth, piglets spend much time in contact with the sow. This is of concern to producers who want to reduce the incidence of piglets being crushed by the sow. Providing a heat source reduces piglet mortality during the first three postnatal days, but this effect may be due to a better thermal environment, not the attraction of piglets away from the sow. Most observations on radiant heat sources, creep areas, and brooder boxes suggest that piglets use these areas extensively beginning only two or three days after birth.

This two-day adjustment period accords with observations on piglets' defecation patterns. Although piglets rarely defecate in the creep area after two days of age, they will do so before that time. Later, when uses of various areas of the pen are better defined, piglets defecate along the walls, in corners, and over perforated areas rather than on solid floor areas.

However, piglets less than two days of age do seem to prefer certain types of floors on which to lie. The preference order is plastic-coated expanded metal most, then perforated metal, fiberglass slats, and woven wire least. This same order remains after weaning. Whether variable floor type is a means of moving piglets away from the sow during the first two days after birth is unknown.

Piglets sleep about half of the time up to five weeks of age. While piglets are on the sow, sleep usually occurs in 20-minute bouts. Growing pigs and adults often sleep for two-hour periods. The paradoxical or "dream" sleep decreases as a portion of total sleep from birth to five weeks of age. What a newborn piglet has to dream about, if in fact it does dream in any sense resembling the human experience, is open to speculation.

General activity levels of piglets increase from about 20 percent of the time during the first week after birth to around 50 percent during the fifth week. Eating creep feed does not usually occur at a significant rate until the piglets are four weeks old. Drinking is rare during the first postnatal week, but fairly common beginning in the second. However, several factors, such as the sow's milk yield and environmental temperature, can influence the development of both creep-eating and drinking behaviors.

Suckling and Associated Behaviors

Perhaps the most interesting aspects of piglet behavior involve teat order and nursing sequence. By three to four days of age, most litters' teat orders have been established. From this time until weaning time, around 90 percent of the piglets will suckle only from their own respective teats. A few piglets will switch teats up to a week of age, and some routinely suckle from two teats. Udder sections that are not used regress and stop producing milk within a few days. This is one reason it is difficult to cross-foster piglets onto a sow after a few days postpartum.

A piglet probably recognizes its own teat by several stimuli. Odor is likely to be important, but piglets can readily locate their own teats even after the udder has been scrubbed thoroughly. Other cues must be used, also. Piglets on front or rear teats may use the sow's legs as reference points, and their littermates may in turn use them as landmarks if odors have been removed. Piglets locate their own teats in a matter of seconds, and once a piglet has located its teat, it is very difficult for a littermate to take it away.

A stable teat order, which results in very little fighting over teats at the udder, is necessary for the type of nursing sequence pigs observe. Nursing occurs about once an hour. It involves a complex series of signals and stimuli between the sow and her piglets. The sequence begins with either the sow or the piglets indicating intent. The other respond(s) accordingly. The sow lies down and begins a series of slow grunts. She exposes her udder to her young as the piglets assemble, begin nosing the udder, and locate their respective teats. While the piglets continue to stimulate the sow by rooting the udder, the sow responds with a gradual increase in grunting rate. In about one minute there is a rapid increase in the sow's grunt rate, and oxytocin is released from her pituitary gland, resulting in milk release within 20 to 30 seconds. Meanwhile, the piglets stop nosing and begin to

slowly suckle the teats. When milk flow actually starts, the piglets suckle rapidly, and the sow's grunt frequency increases again. Milk flow lasts for only 10 to 20 seconds. If a piglet is not settled on its teat at this time, it will not get any milk during that bout. Perhaps this is the primary reason for the teat order: to allow piglets to locate a teat quickly and surely and not have to fight during the very short period of milk availability from the sow (10 to 20 seconds every hour). Once milk flow stops, the sow's grunting declines once again, and the piglets begin to suckle very slowly. Then they usually proceed to fall asleep.

Several important features have been discovered about the nursing sequence. In order for a sow to be stimulated to the point of milk letdown, the front half of the udder must be massaged. If piglets are removed from these teats, the sow will not nurse successfully. In addition, after a successful nursing, she will not be able to let milk down again for at least 15 minutes. Nursing is often synchronized among litters in a farrowing room. The sounds associated with nursing by one litter frequently seem to stimulate nursing by others. It might be possible to stimulate sows to nurse by playing back the sounds of a nursing litter through a speaker, but conflicting results have been found in experimental attempts to increase nursing frequency in this way.

THE PRACTICES

We can meet any behavioral need(s) of the sow and her litter of piglets only if we know what these needs are. How can we identify the behavioral needs of swine? How can this information be used in designing improved swine equipment and facilities?

A Digression on Behavioral Needs of an Animal

A well-accepted theory of motivation in humans is based on the idea that our needs are arranged in a hierarchy according to relative priority or potency. More potent needs dominate an organism until they are satisfied; then, in turn, the less potent ones arise and become motivational forces. A brief review of Abraham Maslow's hierarchy of human needs might supply a useful first approach to assessing the well-being of animals in different settings.

The most prepotent needs of humans are the physiological needs, including those for adequate food and a tolerable thermal environment. Once these have been satisfied, those next higher--the safety needs--emerge: security and freedom from fear and anxiety, among others. Most humans develop additional needs in approximately the following order: the belongingness/love needs, the esteem needs, the need for self-actualization (that is, what one can be, one "must" be), the desires to know and to understand, and finally the aesthetic needs.

Application of Maslow's scheme for humans to animals might result in a hierarchical organization of animals' needs along the following lines, from lowest to highest: the physiological needs, the safety needs, and the behavioral needs. What, specifically, are these needs? What do they imply as to agricultural animals' well-being?

Animals' Physiological Needs

A great deal is known about farm animals' physiological needs, and, as these nutritional, environmental, and health needs are now understood, most are being met most of the time.

Animals' Safety Needs

Safety needs stand next in the hierarchy suggested for domestic animals. It is obvious, firstly, that physical maltreatment of animals by humans is not only inhumane, but also anathema in efficient production. It occurs rarely, but--because human nature is what it is--abuse of animals is unlikely to ever disappear completely. On the other hand, there is increasing scientific evidence that confirms conventional wisdom: agricultural animals respond positively in terms of health and productivity to supportive social contacts with humans.

In practice, the safety needs are tended somewhat less rigorously than are the physiological needs, even though laxity in this respect often results in physical injury or even death. Three kinds deserve mention.

Many animals in natural environments are hurt or lose their lives in weather accidents. The number would be much greater had the industries not taken elaborate steps to protect against severe impingements by the meteorological elements. Occasionally, animals kept in closed houses succumb indirectly to stormy weather, as when a power outage leads to ventilation failure and environmental temperature rises to the lethal point. But otherwise animals provided housing, windbreaks, shades, or other environmental modifications are generally better protected from the various extremes of weather than are their counterparts which must depend on natural features in outdoor settings.

Predators kill large numbers of sheep and lambs, calves, young pigs, and poultry, and seriously injure even more, despite the availablity of effective and acceptable methods of control. Animals' chances of falling prey to coyotes and other wild carnivores ordinarily are decreased when they are shifted from extensive production systems to intensive ones.

The design of equipment and facilities with respect to animal safety requires interdisciplinary collaboration. There is much scope for improvement. Fortunately, problems of this sort are amenable to scientific inquiry and to the application of findings to the immediate benefit of the animals, as the following three examples indicate.

First, vast improvements in the hen's safety in terms of bodily entrapment and foot health have come about during the past decade due to design changes in commercial laying cages that resulted directly from carefully controlled comparative studies of special features of then-available commercial cages in a simulated production setting. This project is discussed further below.

Second, a headgate is an essential piece of restraining equipment for beef-cattle operations. Each of the four basic kinds of headgate was designed

for a distinct purpose and has special advantages. Each also has drawbacks in terms of cattle safety--including tendencies to cause choke or head, shoulder, or leg injuries--when used for some purpose other than that for which it was intended.

Third, the nature of the floor surface in a dairy facility affects the health of the cows' feet and legs greatly, and slipperiness frequently leads to falls and serious injuries. The ideal requirements that a floor for hoofed animals possess softness, a friction coefficient high enough to minimize the animals' chances of slipping, and low abrasiveness, are very difficult to meet simultaneously and economically, but strategies for compromise are evolving.

Animals' Behavioral Needs

Any instance of animal suffering due to human action or inaction falls, according to Roger Ewbank, into one of three categories: abuse, neglect, or deprivation. Abuse refers to obvious cruelty, such as beating an animal with a stick. Neglect occurs when an animal is confined and then denied a physiological or safety need, such as feed or water, health care, or shelter. As already indicated, abuse and neglect occur infrequently in animal-agricultural operations managed according to modern norms.

Deprivation, on the other hand, involves the denial of certain, often less vital aspects of the environment. It is the form of cruelty most difficut to assess. Many of the needs in this last category are behavioral and have not yet been ascertained, let alone characterized well enough to be useful in the design of animal quarters. Their presence is signalled by demonstrations--often subtle--of frustration, fear, and discomfort. It is here that the most controvewrsy lies as to whether agricultural animals experience well-being or suffering.

Hughes's Unitary Model of Motivation

Barry Hughes has proposed a unitary model of motivation which might be applied usefully in the assessment of animals' behavioral needs. He theorized that the traditional psychohydraulic and mixed-motivation models occupy different ranges in a continuum of the ratio between external and internal contributions to the causation of behavior, and that each behavior pattern lies at a specific point in this continuum.

The Hughes model can be partitioned into three regions for the sake of discussion: category 1--environmental stimuli of behavior are far more important than internal motivations near one end of this continuum, category 2--internal and external factors both contribute significantly as behavioral triggers in the middle, and category 3--internal factors are the chief releasers of behavior near the other end. Examples in the sow for the three respective categories might be: category 1--escape and fighting behaviors, category 2--the mating stance, and category 3--maternal nest-building.

Hughes suggests that, to ensure animal well-being, it is desirable to provide for some of the behavior patterns in category 2, and essential to provide for all of those in category 3. The decision as to whether it is desirable for a category-2 behavior to be accommodated by environmental manipulation depends on the relative contributions of external and internal

factors to the release of that particular behavior. Those which surface even without significant environmental stimulation should be accommodated. For example, the sow's mating stance depends on a distinct environmental releaser (pressure on the rump, for example) as well as an appropriate hormonal state; hence, this behavior would not be considered a need. Phantom nest-building, on the other hand, occurs in many sows even in a relatively barren environment, such as a farrowing crate, where the normal external stimuli for this behavior are presumably weak. Thus, it might be desirable to provide sows the opportunity to build a maternal nest, even though engagement in vacuum activities such as phantom nest-building does not necessarily mean the sow is distressed. She can, after all, perform the behavior in the barren environment. Likewise, sometimes animals are able to cope by performing displacement behaviors and in this way preserve their own welfare for themselves.

For a behavior pattern to be considered an essential behavioral need (category 3), Hughes would require clear evidence of either frustration (such as stereotyped pacing preceding oviposition in some caged hens) or distortion of the behavior pattern, which of course can be ascertained only if the behavior's normal limits are known in fine detail.

Before any behavior should be designated a need, the animal must be subjected to careful, thorough behavioral analysis in the very environment of interest. Moreover, to establish that well-being indeed exists on the part of the animal, it is not necessary that the animal's behavior be the same in the environment of interest as in another one arbitrarily set as the standard.

Consequences for Environmental Design and Management

Mike Baxter has pointed out that although "performance of behaviour by an animal will simultaneously accommodate its requirements for both agricultural productivity and animal welfare....productivity and welfare are the outcome of 2 separate behaviour subsystems, concerned with the function of behaviour and the control of behaviour, respectively. Thus, to describe animal requirements as the need to perform normal behaviour is an oversimplification." He goes on to say that sometimes both productivity and welfare can be accommodated without the animal ever performing a certain behavior.

With reference to nesting behavior in the prepartal sow, for instance, Baxter suggests that its productive functions (protecting piglets from cold and from being crushed by the sow) could be replaced by appropriate husbandry techniques, while its welfare function (postulated by Baxter to be furnishing the sow's highly sensitive udder a comfortable contract surface) might be achieved simply by providing a comfortable floor. Consequently, the absence of nesting behavior--real or phantom--in a sow would not necessarily mean the sow was suffering from behavioral deprivation; it might mean merely that nest-building behavior is in Hughes's category 1 or category 2, and that environmental manipulation had eliminated the releaser of the behaviour (uncomfortable floor) as well as the usefulness (piglet protection and udder comfort).

Baxter's line of thinking can be taken a step further to embrace environmental richness. Sows characteristically wallow in mud to enhance evaporative heat loss during hot weather. However, in outdoor environments the amount of wallowing behavior performed is related directly to environmental temperature,

and sows do not wallow at all at air temperatures below 12°C. These observations can be interpreted to mean that sows wallow primarily to achieve thermal comfort and that, even at high environmental temperatures, so long as the sow is made comforable by some means, it is unlikely that she is either deprived or frustrated when her environment is devoid of a wallow.

Alex Stolba and David Wood-Gush have taken a different tack in designing an artifical production environment for swine that would be minimally equivalent, in accommodating the animals' behavior, to an 11,000-m^2 natural setting outside Edinburgh they arbitrarily designated as the standard. The scientists carefully characterized behavior patterns--in terms of frequencies, sequences, orientations, contexts, and "clue factors" (which provide releasing, orienting, and situational stimuli)--and level of arousal of swine in (a) the standard natural setting described above; (b) natural paddocks of varyng environmental richnesses and measuring either 1,000 m^2 or 300 m^2; and (c) a variety of pens, some relatively barren, others enriched in various ways.

Their behavioral observations led to the development of several generations of artificial swine-rearing environments which incorporated those elements of the standard natural setting which were deemed necessary to support standard behavior. Finally, an enriched housing system for matriarchal swine families was designed. It contains nesting, activity, and rooting areas as well as pieces of furniture, including partitions, feeding stalls, farrowing rails, rubbing post, straw rack, and levering bar. A husbandry plan befitting the system has been developed, and from initial results the animals' reproductive and productive performance levels would seem to be above commmercial minima.

The approach of Stolba and Wood-Gush is attractive because it exhaustively explores the relations between an animal's environment and its behavior. But it presumes that in any environment accepted as satisfying behavioral needs the animals should behave as they do in some standard environment. This notion (a) was called "an oversimplification" by Baxter, (b) does not abide by Hughes's more rigorous criteria for establishing animals' behavioral needs and (c) presumes that any departure from some standard behavior is unacceptable.

Our Approach to Farrowing-Crate Design

Although sows crush or savage fewer piglets in crates than in pens, today's total preweaning mortality rate remains similar to that 25 years ago when pens were in vogue. Farrowing crates or certain of their specific design features might have deleterious effects on sows, piglets, or both.

Farrowing-crate designs have been developed by innovative producers and equipment manufacturers. Dozens of models have come onto the commercial market. Swine-industry trade magazines list over 90 farrowing-equipment manufacturers in this country nowadays. Most of them offer farrowing crates, many more than one model.

From evaluations of animal equipment other than the farrowing crate, we know that small design differences can affect both animals and production markedly. But there have been neither scientific evaluations of various farrowing crates nor scientific confirmations of alleged benefits of specific

design features. Results of controlled farrowing-crate comparisons simply have not been available to pork industry decisionmakers.

There seem to be limits to fitting animals genetically to artificial environments. Environments must be fitted to the animals, too. The variety of farrowing equipment on the market is wide. Apparently, (a) sows and piglets are plastic creatures, adaptable to a wide range of environments, so some design differences are insignificant in terms of animal health or performance, although they might be important economically, and (b) some manufacturers ignore present knowledge of sows' and piglets' environmental requirements as they design attractively priced farrowing crates.

A theoretical framework for the design of farrowing environments has been developed by Seaton Baxter. In regard to spatial analysis, Baxter identifies three zones--(a) a safe zone for piglets, (b) a piglet-sow interaction zone, and (c) a sow zone--and discusses their implications regarding total farrowing environment design. But by no means is our basic knowledge in this area now adequate to the task of designing the ideal farrowing environment a priori. We are just now beginning to gain a quantitative notion of the animals' specific requirements in each zone (for example, from data such as Mike Baxter's on the sow's space requirement).

Nevertheless, standards are needed for the design and manufacture of farrowing crates. Most other products farmers use--seeds, feeds, herbicides, feed additives, biologicals, to name a few--have stronger histories of scientific research and development. Indeed, farmers now expect scientific documentation of manufacturers' claims of efficacy for many products. Other industries--electric devices, milking machines, farm tractors--have voluntary programs of standards establishment and product testing. Multidisciplinary teams of scientists should establish initial standards for farrowing crates now. At the same time, we need to mount energetic research programs to describe sows' and piglets' environmental requirements and development programs to learn how to fulfill them.

Outstanding reseach and development on layer-cage design is flourishing in Sweden under Ragnar Tauson. Tauson's successes with hen cages are encouraging to those concerned about sow crates. The program was established largely in response to pressures from animal welfarists, but the egg industry has reaped substantial dividends, too. Numerous parameters are evaluated in relation to specific cage design features, including various aspects of the hens' external condition, behavior, health, and egg production; egg quality; operating reliability and maintenance; and ergonomics considerations.

This work has occurred in three stages. In phase 1, pluses and drawbacks of specific features of popular designs were identified during three years of testing. Major differences among designs were found for accidental trapping, external body condition, feed wastage, external egg quality, and technical reliability. These findings were used to improve design features, such as solid partitions, reduced floor slope, smoother floor coating, rounded manure deflector, elimination of gaps and uneven spacing in floor and walls, changes in front design, horizontal bars at trough, and smooth trough lip.

These features listed above and others as well were incorporated in several experimental cages created and assessed in phase 2. The various

modifications resulted in marked improvements in the parameters monitored. In fact, phase 2 was so successful that most of its benefits were achieved in only three years. Cooperation and collaboration between Tauson cage manufacturers also commenced during phase 2, and these relations have since grown stronger.

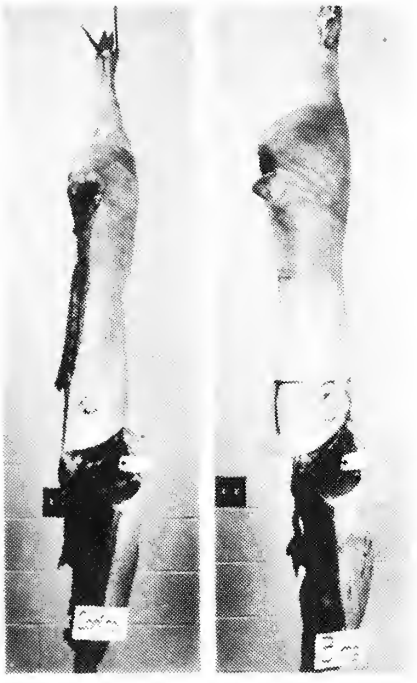
Phase 3 involves a search for unconventional solutions to remaining cage design problems. This basic research is slower, partly because, in the cages developed in phase 2, some problems occur so infrequently that it will be difficult to decrease them further. For example, the frequency of accidental trapping has been reduced by 90 percent, and foot-health score improved from .95 to .47 (0 = no lesions, 3 = severe).

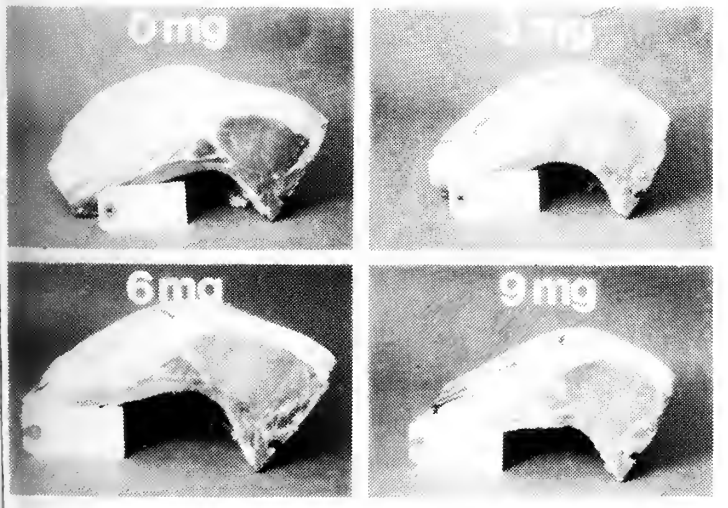
Tauson's program is unique. For other livestock and poultry equipment, no one has yet completed phase 1, let alone phases 2 or 3, employing such an holistic, integrated, effective approach. But at the University of Illinois, animal scientists are collaboraing with specialists in agricultural engineering, aricultural economics, and veterinary medicine in a thoroughgoing examination of the farrowing crate that began in the summer of 1984.

Design features being evaluated include: lower bar--straight, bowed, fingered; height of lower bar--8 and 10 inches; width of sow zone--22 and 24 inches; back-gate design; and sliding bar feature to restrain sow upon lying down. The floor is the same across crate models--plastic-coated expanded metal.

Productive performance parameters being measured include sow weight in and out, number of piglets born alive and dead, birth weight, age at death, cause of death, number weaned, and weaning weight. All health observations and medical/surgical treatments are being registered and taken into account, including lesions on sows in and out and on piglets at specific ages. Also, immune-function tests are being conducted. Behavioral observations, made both directly and via videotape recordings, are being made on adequate samples of animals and include sows' reactions to crates upon introduction: sows' maintenance activities, including postures, phantom nest-building, and parturient behavior; piglets' time to first suckle; piglets' suckling behavior, including assembly at udder; and physical interactions between piglets and of piglets and sows at times other than nursing. Ergonomics observations include herder safety records, and objective evaluations of different crates by herders, and time required to perform standard tasks, such as collecting all piglets. Maintenance requirements are being determined from detailed records of all maintenance efforts and assessments of crate condition at the end of every farrowing cycle. Production economics will be analyzed completely at the end of the project.

During phase 2 of the Illinois farrowing-crate project, problems which persist in the prototype crate(s) developed on the bases of phase 1 results would be identified. Future objectives are (a) to determine the behavioral, physiological, or design features contributing to these problems and (b) to develop and test features designed de novo to reduce these specific problems.





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PORK INDUSTRY CONFERENCE

December 10-11, 1987
College of Agriculture
Department of Animal Sciences
Cooperative Extension Service
Agricultural Experiment Station
University of Illinois at Urbana-Champaign

University of Illinois
Pork Industry Conference

The Repartitioning Revolution: Impact of Somatotropin and Beta Adrenergic Agonists on Future Pork Production

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The University of Illinois Department of Animal Sciences, College of Agriculture and Cooperative Extension Service acknowledges the contributions and support of the following companies:

AMERICAN CYANAMID COMPANY,
Princeton, NJ

IMC PITMAN-MOORE, INC.,
Terre Haute, IN

LILLY RESEARCH LABORATORIES,
Greenfield, IN

MONSANTO AGRICULTURAL COMPANY,
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Contents

Development and Status of Repartitioning Agents for Use in the Swine Industry

David G. Topel

HISTORICAL BACKGROUND

The swine industry in the United States has historically played a major role in providing high quality protein sources for the consumer. With the major emphasis placed on reducing heart problems in the last 30 years, however, pork consumption has decreased in the United States and the consumer is selecting, at an increasing rate, other muscle foods (fish, poultry) for quality protein sources in their diet.

Because pork is still considered a high calorie food by many consumers, it may be of value for the participants of this conference to review the composition of typical market weight pigs in the United States as they grow from 2 to 250 pounds (Table 1). At 200 pounds, a pig with 1.6 inches backfat has approximately 35 percent fat and 46 percent muscle in its carcass. A pig with 1.0 inches backfat has approximately 28 percent fat and 53 percent muscle. At 250 pound, both the fat and meat type pigs are expressing high quantities of fat in the carcass (41.7 vs 37.7 respectively).

Because the consumer will not purchase fat pork products at the retail level, the majority of the pork carcass fat is removed at the packing plant and some is trimmed at the retail stores. This increases the cost of lean pork for the United States consumer.

Even though good progress has been made by the U.S. Swine Producers in the last 30 years in reducing fat through genetic

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selection (Table 2) the average market pig still has more than 30 percent fat in its carcass (Topel, 1986). Therefore, the swine industry needs more "tools" than just genetic selection to reduce carcass fat. The theme (Impact of Somatotropin and Beta-adrenergic Agonists on Future Pork Production) for this conference addresses the most recent "tools" associated with biotechnological research which have the potential to greatly reduce fat deposition and increase muscle deposition in market pigs. Some of these new "tools" can repartition nutrients into muscle tissue rather than fat tissue in pigs.

Several of these new compounds were developed for weight control programs for people, but also have great commercial value for controlling body composition in domestic animals produced for meat production. This reflects the direct application of fundamental, biotechnological research for improving quality pork products and profits for swine producers.

DEVELOPMENTS FOR THE SWINE INDUSTRY FROM BIOTECHNOLOGY RESEARCH

Biotechnology, the use of living organisms or their components in industrial processes, is not new to farmers or the agricultural industry. We have used technology of this type since the early 1940's. Recent publicity in the popular press and legal challenges from organizations aligned with the humane treatment of animals, however, has brought new biotechnology research discoveries to television talk shows, congressional hearings and court rooms around the country. Challenges of this type reflect the significant concerns by a small number of people about the biotechnology research in the 1980's on altering the growth process and metabolic functions of animals. We must do a better job of informing the public about the major advantages of biotechnology research and the contributions it can provide our society. If we don't overcome these negative challenges, the application of new biotechnology concepts may be limited for the agricultural industry. Despite the recent challenges for applying biotechnology concepts to production agriculture, research efforts of thousands of scientists throughout the world continue to enhance our understanding of genes, their chemical composition, function and their performance potential. Our understanding of these basic phenomena has developed to the extent that we can chemically describe genes, and they can be isolated and replicated in the laboratory outside the parent organism. Genes can be transferred from the parent to another organism that is not necessarily closely related to the parent organism. For example, the antiviral substance interferon which is produced in animal tissues in response to virus infection is controlled by a gene which has been chemically defined, reproduced in the laboratory and transferred to bacterial cells. The animal gene for interferon causes the bacterial cells to produce interferon. Several additional genes, for example, those controlling the synthesis of growth hormone and insulin

production have also been successfully transferred to bacterial cells with the resultant production of these compounds by the bacterial cells.[1]

Porcine growth hormone prepared with biotechnology methods was injected into pigs and the encouraging results were reported by Pennsylvania State University researchers (Etherton, et al. 1986, 1987). The application of this research to the swine industry was reported in the October, 1986 issue of Hog Farm Management. An article prepared by Kathy Hohmann stated that growth hormone administration to growing pigs can result in a 2.3 feed conversion when producing 260 pound pigs, increase growth rate by 16 percent, reduce backfat by 70 percent, and increase muscling by 25-30 pounds. The porcine growth hormone injection improved feed efficiency 35 percent. If this figure is applied to the current swine industry, Etherton stated that the industry could save two to six billion a year in feed costs.

Cornell University researchers (Boyd et. al. 1986) also studied growth hormone influences on carcass composition as well as lactation in sows. They found that growth hormone increased milk production in 2-4 weeks of lactation by as much as 30 percent during a 21 day treatment period. In many swine herds, the baby pig has much more potential to grow than the sows' milking ability. Therefore, growth hormone has the potential to increase baby pig growth by stimulating more milk production in the lactating sow.

Before growth hormone, however, has commercial application to the swine industry, a simple method to administer the hormone must be developed. The compound is destroyed in the digestive tract so it can't be added to feed. The current injection methods are not practical for commercial use of the growth promoting hormone. Commercial companies which plan to merchandise growth hormone to the livestock industry have active research programs associated with the development of low cost and simple delivery systems for the administration of growth hormone to livestock. In a few years, I would expect commercial use of this product by the swine industry.

The production of porcine growth hormone with new biotechnology techniques associated with recombinant DNA is just one example of the application of this powerful mechanism for improving the efficient production of pork.

Monoclonal antibodies and hybridoma techniques will also be used to develop a better understanding of various hormonal systems associated with reproduction and disease.

A 1982 report from the Biotechnology Committee of the National Association of State Universities and Land-Grant Colleges provided an excellent summary of the role required of scientists at the Land-Grant Universities if we plan to take advantage of the current scientific

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[1]Emerging Biotechnologies in Agriculture: Issues and Policies.

knowledge associated with recombinate DNA and the control centers for cellular functions in domestic animals. These new research technologies are all related to molecular and cellular genetics. The genetic material (DNA and RNA) of all living organisms does not change from animal to animal, plant to plant or animal to plant. Therefore, every organism is related at the molecular level. Scientists at the Land-Grant Universities have a great opportunity to take advantage of this concept and develop strong research teams for the biotechnology area.

If significant productivity from biotechnology research is to occur at the Land-Grant Universities, integration of the basic sciences such as molecular biology, biochemistry, microbiology and biophysics with the applied sciences (entomology, plant pathology, animal science, soil science, etc.) must occur at the scientists level. The integration of basic science with the applied agricultural sciences will require different kinds and degrees of programs coordination depending on the administrative structure of the research program. The very nature and structure of the Land-Grant Universities, however, provides an ideal setting for establishing biotechnology research teams. Several of the research teams will use swine as the experimental model and this will result in major contributions for advancing swine production techniques. The pig is an excellent experimental model for human medicine research. Therefore, we can expect a large amount of biomedical research to be centered around the pig in future years. This will further develop new concepts for improving practical production methods for the swine industry.

The theme of this conference is not centered on traditional genetic selection for production and carcass traits, but this area should not be forgotten when we discuss methods to repartition nutrients into specific components of the pig. We are just identifying key processes governing growth in pigs which contribute to the natural variation in animal traits of commercial importance. Kuhlers and Jungst (1987) have developed growth lines with the Duroc and Landrace breeds. These lines provide excellent animals to study nutritional requirements for pigs with different growth potential (Prince, 1987) or reproductive traits. Research results reported by Kuhlers and Jungst (1987) reflect larger litter size from sows representing the rapid growth line. Lines of this type should be useful to biotechnology researchers as they can compare cellular functions and identify which control centers are responsible for the genetic differences. When this information is established, genes can be identified and biotechnology concepts applied to the commercial swine industry.

In addition, swine producers must continue to use good genetic selection methods after the new repartitioning agents are commercially available. The new repartitioning agents should be used to further stimulate growth, efficiency and muscle deposition on pigs which are produced under excellent management and genetic selection criteria.

It would be a major mistake if swine producers use repartitioning agents as a substitute for good, efficient management. After this conference, you will probably realize it may take even better than average management skills to obtain maximum benefits from the various types of repartitioning agents.

CURRENT DEVELOPMENT STATUS OF REPARTITIONING AGENTS

Currently, only three or four repartitioning agents are undergoing intensive review, evaluation and development for possible use by the swine industry. You can expect more in the next 10-15 years, but the complexity of these compounds will limit the number available for commercial utilization.

One of the recent repartitioning agents (Ractopamine) developed by Lilly Research Laboratories was tested at Auburn University (Prince et al. 1987). Ractopamine increased daily gain when fed at 5 ppm and improved feed:gain at 5, 10, 20 and 30 ppm levels. Carcass leaf fat decreased, backfat decreased and loin eye area increased. Carcass dissection data showed that percent lean increased by 4.4 percent and fat decreased by 3.2 percent when 20 ppm Ractopamine was fed (Tables 3, 4, 5). The response illustrated in Table 3 from feeding Ractopamine reflect the economical significance of feeding one of the repartitioning compounds which is still in the development phase. I used this example to reflect the current status of commercial development of repartitioning agents for pigs. You will be exposed to several others during this conference.

The participants of this conference will have the unique opportunity to be informed in much detail about the most current information available on repartitioning agents for the swine industry. These agents are not commercially available at this time, but they will be in the near future.

Information presented at this conference, however, will have a significant impact on the swine industry over the next 5 years.

LITERATURE CITED

Boyd, R. D., D. E. Bauman, D. H. Beermann, A. F. De Neergaard, L. Souza and W. R. Butler. 1986. Titration of the porcine growth hormone dose which maximizes growth performance and lean deposition in swine. J. Animal Sci. 63:218 (Suppl. 1).

Etherton, T. D., J. P. Wiggins, C. S. Chung, C. M. Evock, J. F. Rebbun and P. E. Walton. 1986. Stimulation of pig growth performance by porcine growth hormone and growth hormone-releasing factor. J. Animal Sci. 63:1389.

Etherton, T. D., J. P. Wiggins, C. M. Evock, C. S. Chung, J. F. Rebbun, P. E. Walton and N. C. Steele. 1987. Stimulation of pig growth performance by porcine growth hormone: determination of the dose-response relationship. J. Animal Sci. 64:433.

Hohmann, K. 1986. One giant step for the pork industry. Hog Farm Management. 23:8.

Kuhlers, D. L. and S. B. Jungst. 1987. Four generations of mass selection for growth to 200 days of age in Duroc pigs. I. Growth traits. J. Animal Sci. 65:209 (Suppl. 1).

Prince, T. J., D. L. Huffman and P. M. Brown. 1987. Effects of ractopamine on growth and carcass composition of finishing swine. J. Animal Sci. 65:309 (Suppl. 1).

Prince, T. J. 1987. Effects of dietary fat addition and nutrient density on performance and carcass composition of swine. J. Animal Sci. 65:301 (Suppl. 1).

Topel, D. G. 1986. Future meat-animal composition: industry adaptation of new technologies. J. Animal Sci. 63:633.

TABLE 1. RELATIONSHIP BETWEEN BODY WEIGHT AND COMPOSITION OF SWINE [a]

Live	Percent Muscle		Percent Fat		Percent Bone		Percent Skin	
Wt. lbs.	Meat Type	Fat Type	Meat Type	Fat Type	Meat Type	Fat Type	Meat Type	Fat Type
2.2	48.6	48.3	0.0	0.0	31.8	31.9	19.3	19.5
50	60.8	59.1	8.9	9.6	18.8	19.1	11.2	11.9
100	63.1	60.2	11.5	13.8	15.9	16.0	9.4	9.9
150	58.0	54.3	20.0	24.4	14.9	14.0	7.0	7.3
200	52.8	46.4	28.8	35.2	12.0	11.5	6.2	6.7
250	45.9	42.3	37.7	41.7	10.6	10.0	5.7	5.9

[a]Meat type at 200 had 1.1 inches backfat and the fat type had 1.6 inches backfat.

TABLE 2. PERCENTAGE OF PORK CARCASSES IN EACH GRADE[a]

Grade	1968	1980
1	8.0	71.7
2	42.0	24.2
3	36.0	3.7
4	12.0	.3
Utility	2.0	.1

[a]From Topel, 1986.

TABLE 3. GROWTH PERFORMANCE FROM RACTOPAMINE

Ractopamine Dosage (ppm)	No. of Exptl. Units	Total No. of Pigs	Average Initial Wt (lbs)	Least Square Means			
				Average Final Wt (lbs)	Performance		
					ADG (lbs)	ADF (lbs)	F/G
0.0	3	17	141	220	2.03	6.84	3.39
2.5	3	17	141	226	2.18	7.13	3.27
5.0	3	18	141	227	2.20	6.83	3.10
10.0	3	18	140	225	2.13	6.49	3.04
20.0	3	18	140	225	2.17	6.64	3.05
30.0	3	17	142	222	2.03	6.29	3.10

From Prince et al., 1987.

TABLE 4. CARCASS MEASUREMENTS FROM PIGS FED RACTOPAMINE

Ractopamine Dosage (ppm)	No. of Exptl. Units	Live Sltr. Wt (lbs)	Least Squares Means: Hot Carcass Wt (lbs)	Average Dressing (%)	Carcass Length (in)	Leaf Fat (lbs)	Average Backfat (in)	Average 10th Rib Fat Depth (in)	Average 10th Rib Loin Eye (sq in)	Estimated Muscle[a] (%)	Muscling[b]	Color[c] of Lean	Marbling[d]	Firmness[e]
0.0	3	226	161	71.4	31.7	3.12	1.06	0.99	5.05	50.3	3.8	2.4	1.6	2.6
2.5	3	232	166	71.4	31.7	3.28	1.13	1.02	5.27	50.6	3.9	2.4	1.6	2.8
5.0	3	229	164	71.7	31.3	2.95	1.10	0.96	5.71	52.2	3.9	2.2	1.6	2.6
10.0	3	230	164	71.4	31.5	2.54	1.09	0.85	5.79	53.4	4.1	2.4	1.5	2.5
20.0	3	226	164	72.6	31.2	2.73	1.10	0.86	5.76	53.2	4.3	2.0	1.8	2.5
30.0	3	227	165	72.7	31.0	2.82	1.06	0.85	5.77	53.3	4.4	2.2	1.8	2.8

[a]Estimated Fat Free Muscle Percentage = 44.4 + [2.73 x 10th Rib Loin Eye Area] - [8.06 x 10th Rib Fat Depth]
[b]Muscling Score: 1 = thin, 5 - very thick
[c]Color of Lean Score: 1 = pale, 5 = dark
[d]Marbling Score: 1 = traces, 5 = abundant
[e]Firmness Score: 1 = soft, 5 = very firm

From: Prince et al., 1987.

TABLE 5. CARCASS DISSECTION DATA FROM PIGS FED RACTOPAMINE[a]

TABLE 5. CARCASS DISSECTION DATA FROM PIGS FED RACTOPAMINE[a]

Ractopamine Dosage (ppm)	No. of Exptl. Units	Total No. of Pigs[b]	Least Squares Means: Chilled Side Wt.[c] (lbs)	Lean (%)	Fat (%)	Bone (%)	Skin (%)	Lean (lbs)	Fat (lbs)	Bone (lbs)	Skin (lbs)
0.0	3	12	76.80	53.6	26.3	13.8	6.5	41.2	20.2	10.6	5.0
5.0	3	12	80.94	54.8	25.5	13.1	6.5	44.4	20.6	10.6	5.3
20.0	3	12	79.94	58.0	23.1	12.7	6.2	46.3	18.5	10.2	5.0

[a] Data were collected from the 0, 5, and 20 ppm levels only.
[b] Sex ratio was 50/50 (barrows/gilts) for each treatment level.
[c] The right side of the pork carcass was used for physical separation.

From: Prince et al., 1987.

Potential for Inserting the Somatotropin Gene into the Swine DNA: Long Term Goals and Implications

Vernon Pursel

INTRODUCTION

The dramatic advances in molecular biology of the past decade and the development of micromanipulative techniques with living embryos were combined in 1980 to provide the means for transferring cloned genes into mice (see review by Brinster and Palmiter, 1986). The transfer of genes was immediately recognized as an important scientific achievement, but the creation of "super" mice by the transfer of a rat or human somatotropin gene provided the exciting experimental evidence that demonstrated the potential applications of gene transfer (Palmiter et al., 1982). As a consequence, research on gene transfer was quickly expanded to include farm animals.

The purpose of this report is to review the goals of research directed toward the insertion of a somatotropin gene into the pig genome to make "transgenic" pigs, some of the things that have been learned while pursuing these goals, and some of the problems that must be overcome to achieve the long-term goal.

LONG-TERM RESEARCH GOAL

The long-term goal of this research is to produce transgenic boars that will transmit a controllable somatotropin gene into the genome of their progeny. When the somatotropin gene is externally stimulated in these transgenic progeny they would be expected to exhibit a superior rate of growth, produce high-quality lean pork and be able to convert feed efficiently into meat.

The ideal external stimulus would be some component that could be added to the feed. When the transgenic pigs reached a certain size, the farmer would merely add the component to the feed to activate the gene, causing somatotropin secretion to elevate, which would enhance growth. Just before market, the component could be removed from the feed if that was necessary to comply with possible regulations regarding either the component or the elevated somatotropin.

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Although somatotropin genes from other species have been used in much of the preliminary research, a cloned pig somatotropin gene would probably be the structural gene of choice for producing transgenic pigs that would be used on the farm. This would totally sidestep problems with the hormone being compatible for all aspects related to growth in the pig.

In order for all the progeny to be transgenic, boars would need to be homozygous for the somatotropin gene. However, activation of the gene in these boars would not be necessary if the elevation of somatotropin had a detrimental effect on either the boar's longevity or capacity for sperm production, which would be their primary function.

Homozygous transgenic boars would be too valuable to be limited to use for natural service. Therefore, they would be used primarily to produce semen for artificial insemination (AI) of sows to produce market hogs. This would dramatically increase the use of AI in the swine industry. Of course, a farmer could rather quickly produce a line of homozygous boars by a father-daughter cross or by a half-sib cross, which could then be used in his own herd. Whether purebred seedstock producers could produce boars for sale to other farmers would depend upon whether the original sires were patented and on court decisions that will be handed down regarding recent rulings by the U.S. Patent Office regarding transgenic animals.

Before transgenic pigs harboring the somatotropin gene reach the farm any special nutritional or management requirements will have to be determined. Transgenic pigs will probably not have special requirements until elevated somatotropin secretion is stimulated. After stimulation one might expect protein, mineral and vitamin requirements to differ considerably from the requirements of normal pigs. One can assume that nutritional and management requirements of transgenic pigs would be comparable to pigs that receive exogenous somatotropin; such studies are currently in progress.

The research has been organized into several specific goals that must be achieved in sequential order if we are to successfully achieve the long-term goal. The progress being made on each of these goals will be reviewed.

GOAL 1: MICROINJECTION OF A GENE INTO THE PRONUCLEUS

Most transgenic mice have been produced by the microinjection of a gene into the pronucleus of the fertilized egg. The dense lipid granules present in pig eggs prevent visualization of pronuclei by microscopy. While this problem initially impeded progress, we overcame the problem by stratifying the cytoplasm of the egg by high-speed centrifugation for about 3 minutes. The centrifugation leaves the pronucleus of one-cell eggs and nuclei of two-cell eggs discernible by interference-contrast microscopy (Wall et al., 1985). The centrifugation treatment did not significantly reduce the ability of pig eggs to develop to the blastocyst stage when they were transferred back into the oviducts of recipient gilts.

GOAL 2: INTEGRATION OF SOMATOTROPIN GENES INTO THE PIG GENOME

Several groups of researchers have successfully used the centrifugation procedure and microinjected somatotropin genes into pronuclei of pig eggs. The somatotropin genes used in these studies were fusion genes, i.e., the regulatory or "promoter" portion of one gene fused to the coding or "structural" portion of another gene. The efficiency of gene transfer was extremely low with only about 1% or less of the injected eggs resulting in birth of a pig with an integrated gene (Table 1). When the same fusion genes were microinjected into mouse eggs, the efficiency of integration was about 3%.

The number of gene copies that integrated in transgenic pigs varied from less than one (in cases of mosaic integration only some of the cells have the gene) to 490 copies per cell (Hammer et al., 1985). Most gene integrations occur at a single chromosomal site.

Table 1. Efficiency of transferring somatotropin genes into pigs

Fusion gene	Eggs injected (no.)	Offspring (no.)	Offspring (%)	Transgenic (no.)	Transgenic (%)[a]	Expressing (no.)	Expressing (%)[b]
MThSt[c]	268	15	5.6	1	.37		
MThST[d]	2035	192	9.4	20	.98	11/18	61
MTbST[e]	2198	149	6.8	11	.50	8	73
MTpST[f]		17		6		1	17

[a] Percentage of injected eggs resulting in a pig with gene integration.
[b] Percentage of transgenic pigs expressing the fusion gene.
[c] Brem et al. (1985).
[d] Hammer et al. (1985).
[e] Pursel et al. (1987).
[f] Vize (1987).

GOAL 3: EXPRESSION OF THE INTEGRATED SOMATOTROPIN GENE

When the intact human somatotropin (hST) gene, i.e., both the hST promoter portion and hST structural portion, was transferred into mice only low levels of human somatotropin were found and growth was not enhanced (Wagner et al., 1983). In contrast, transgenic mice, which harbored the fusion gene consisting of the promoter portion of mouse metallothionein gene (MT) fused to the structural portion of hST or rat somatotropin (rST) genes, had high concentrations of these somatotropins in their serum (indicating gene expression) and grew faster and larger than littermate control mice (Palmiter et al., 1982,

1983). The use of the MT promoter resulted in the foreign somatotropin being produced in the liver and other tissues where metallothionein is normally produced instead of only in the pituitary as would be the case if the rST or hST promoter had been used.

Because the MT promoter was quite effective in the mouse, this promoter thus far has been used for somatotropin genes transferred into pigs (Table 1). The MThST (mouse MT promoter fused to hST structural gene) and MTbST (mouse MT promoter fused to bovine ST structural gene) fusion genes were expressed in more than 60% of the transgenic pigs (Hammer et al., 1985; Pursel et al., 1987). In contrast, Vize (1987) reported only one of six transgenic pigs expressed the fusion gene MTpST (human MT-IIA promoter fused to porcine ST structural gene).

Expression of the inserted gene does not appear to be related to the number of copies that integrate into the genome. The circulating concentrations of hST in plasma of expressing transgenic pigs varied from 3 ng/ml to over 5,000 ng/ml. The plasma concentrations of somatotropin were unrelated to the number of gene copies per cell (Pursel et al., 1987).

Transgenic pigs that produced hST or bST grew slower than their littermates on a total body weight basis (Pursel et al., 1987). However, the lack of subcutaneous fat in transgenic pigs became evident by 2 months of age; by 90 kg body weight the average backfat thickness at the 10th rib was significantly lower for hST pigs than for littermate control pigs (7.0 ± 2.1 vs 18.5 mm ± 1.8; Pursel et al., 1987). Therefore, growth on a lean-tissue basis was similar for transgenics and their littermates.

In contrast, the single transgenic pig that expressed the MTpST gene was reported to gain 953 g/day versus 806 g/day for sex-matched littermates, an increase of 15.4% in daily weight gain from 50 to 120 days of age (Vize, 1987). Although these results are based on only one pig, the importance of somatotropin specificity for enhanced growth in the pig is suggested.

Evidence that hST and bST were physiologically active in transgenic pigs were: 1) pigs expressing the MThST gene rarely had detectable plasma pST after they reached one week of age, which suggests normal function of feedback mechanism (Bolt et al., 1986); 2) plasma insulin-like growth factor and insulin were significantly elevated in transgenic pigs (Miller and Pursel, unpublished data); 3) transgenic pigs had much less subcutaneous fat than control pigs as mentioned above; 4) pigs producing bST were significantly more efficient in converting feed to body weight gain (Mitchell and Pursel, unpublished data); 5) mammary gland stimulation was observed in pigs producing hST but not bST, which is consistent with the known lactogenic properties of hST (Pursel et al., 1987).

GOAL 4: TRANSMISSION OF SOMATOTROPIN GENES TO PROGENY

Transgenic offspring have been obtained from both expressing and non-expressing transgenic pigs (Pursel et al., 1986). Five of six founder transgenic pigs transmitted the MThST fusion gene to one or more progeny. Transgenic progeny of non-expressing parents also failed to express the gene. All three transgenic progeny of one expressing boar expressed the gene.

Reproductive capacity was seriously impaired in transgenic pigs that expressed the fusion gene. Transgenic gilts that expressed the gene failed to exhibit estrus; corpora lutea or corpora albicans, which would provide evidence of ovulation, were not present on their ovaries at autopsy even after gilts were one year old (Pursel et al., 1987). The transgenic boars were totally devoid of libido, so spermatozoa were recovered by electroejaculation or by flushing them from the epididymis at necropsy in order to test for germline transmission of the gene. The reproductive problem in females is not unique to pigs. Female transgenic mice that express the MThST or MTbST genes were also sterile (Hammer et al., 1984).

GOAL 5: REGULATION OF THE INTEGRATED SOMATOTROPIN GENE

Continuous secretion of high levels of somatotropin is as detrimental to pigs as it is to humans in which the medical condition known as acromegaly results. Notable characteristics observed in transgenic pigs that expressed the hST and bST genes included extreme lethargy, general muscle weakness, incoordination of rear limbs beginning about 6 months of age, development of arthritis, susceptibility to stress, parakeratosis, anestrous in gilts, and lack of libido in boars (Pursel et al., 1987). Whether transgenic pigs harboring a pST fusion gene would encounter identical problems is equivocal at present. However, Bryan et al. (1987) reported that prolonged daily administration of pST to gilts caused muscle weakness and incoordination of the rear legs comparable to those we observed in transgenic pigs.

Based on the detrimental effects resulting from over-expression of the somatotropin genes, we believe that tight control over the level of somatotropin production by the cells activated by the gene is essential to obtain only the positive effects of elevated somatotropin on growth performance. The possibility of accomplishing this in the future is excellent even though gene regulation in higher animals is poorly understood at present.

Promoter sequences, which are located on the chromosome adjacent to the first segment of the structural gene sequences, are responsible for a continuous level of the gene product being secreted or produced. This constant level of production varies greatly among promoters, so the choice of promoter used to construct a fusion gene is extremely important. In addition, the promoter determines which cells within the body will produce the gene product.

Another major factor involved in the regulation of gene activity is a part of the gene known as the enhancer. Enhancer sequences can be located at various distances before or after the structural sequences of the gene. The binding of specific proteins to the enhancer somehow alters the activity of the promoter and stimulates the rate of gene transcription.

As knowledge of promoters and enhancers increases, molecular biologists will be able to select promoters and enhancers to use in constructing fusion genes that can be stimulated externally to control the level of gene expression.

CONCLUSIONS

Recent research has clearly demonstrated that somatotropin genes can be integrated into the pig genome by microinjecting the gene into a pronucleus of fertilized pig eggs. The integrated genes were expressed, the somatotropins were biologically active, and many of transgenic pigs were able to transmit the gene to their progeny. Although substantial progress on the specific goals has been made, the long-term goal of producing transgenic boars to sire market hogs with enhanced growth performance cannot be achieved until we learn how to regulate expression of the somatotropin gene in transgenic pigs.

REFERENCES

Bolt, D. J., V. G. Pursel, R. E. Hammer, R. J. Wall, R. D. Palmiter and R. L. Brinster. 1986. Plasma concentrations of human growth hormone and porcine growth hormone in transgenic pigs. J. Anim. Sci. 63:(Suppl. 1):220 (Abstr.).

Brem, G., B. Brenig, H. M. Goodman, R. C. Selden, F. Graf, B. Kruff, K. Springman, J. Hondele, J. Meyer, E.-L. Winnaker, H. Krausslich. 1985. Production of transgenic mice, rabbits and pigs by microinjection into pronuclei. Z.F. Zuchthygiene 20:251.

Brinster, R. L. and R. D. Palmiter. 1986. Introduction of genes into the germ line of animals. The Harvey Lectures, Series 80, pp 138. Alan R. Liss, Inc.

Bryan, K. A., D. E. Carbaugh, A. M. Clark, D. R. Hagen and J. M. Hammond. 1987. Effect of porcine growth hormone (pGH) on growth and carcass composition of gilts. J. Anim. Sci. 65 (Suppl. 1):244.

Hammer, R. E., R. D. Palmiter and R. L. Brinster. 1984. Partial correction of murine hereditary growth disorder by germ-line incorporation of a new gene. Nature 311:65.

Hammer, R. E., V. G. Pursel, C. E. Rexroad, Jr., R. J. Wall, D. J. Bolt, K. M. Ebert, R. D. Palmiter and R. L. Brinster. 1985. Production of transgenic rabbits, sheep and pigs by microinjection. Nature 315:680.

Palmiter, R. D., R. L. Brinster, R. E. Hammer, M. E. Trumbauer, M. G., M. G. Rosenfeld, N. C. Birnberg and R. M. Evans. 1982. Dramatic growth of mice that develop from eggs microinjected with metallothione-in-growth hormone fusion genes. Nature 300:611.

Palmiter, R. D., G. Norstedt, R. E. Gelinas, R. E. Hammer and R. L. Brinster. 1983. Metallothionein-human growth hormone fusion genes stimulate growth of mice. Science 222:809.

Pursel, V. G., R. E. Hammer, D. J. Bolt, R. J. Wall, R. D. Palmiter and R. L. Brinster. 1986. Transgenic swine transmit foreign gene to progeny. J. Anim. Sci. 63 (Suppl. 1):203.

Pursel, V. G., C. E. Rexroad, Jr., D. J. Bolt, K. F. Miller, R. J. Wall, R. E. Hammer, C. A. Pinkert, R. D. Palmiter and R. L. Brinster. 1987. Progress on gene transfer in farm animals. Vet. Immunol. Immunopathol. In Press.

Wagner, E. F., L. Covarrubias, T. A. Stewart and B. Mintz. 1983. Prenatal lethalities in mice homozygous for human growth hormone gene sequences integrated in the germ line. Cell 35:647.

Wall, R. J., V. G. Pursel, R. E. Hammer and R. L. Brinster. 1985. Development of porcine ova that were centrifuged to permit visualization of pronuclei and nuclei. Biol. Reprod. 32:645.

Vize, P. D. 1987. Expression of porcine growth hormone in bacteria and transgenic animals. Ph.D. Thesis, University of Adelaide, Adelaide, South Australia.

Primer on Beta Adrenergic Agonists and Their Effect on the Biology of Swine

Harry J. Mersmann

INTRODUCTION

The goal of swine, cattle and sheep producers is to efficiently provide a palatable source of protein for human consumption. Because of the low cost of feed and little concern about the effects of fat on human health, there has not been much emphasis on reduction of fat in carcasses of pigs. Certainly the modern hog is a much leaner animal than its predecesor of 50 to 75 years ago. This change to a longer, leaner, faster growing and more efficient pig was achieved almost entirely, in the U.S.A. by genetic selection. Little progress has occurred in the last 10 to 15 years to improve the real efficiency of porcine meat production, i.e., increased lean muscle mass accompanied by decreased fat mass.

In the U.S.A., consumer preferences for reduced fat content of the retail meat product are having impact on the red-meat producing industry, including pig production. Product fat may be reduced by trimming of the product, a costly procedure, or by further alteration of swine production practices to efficiently produce lean meat. It is important to emphasize at this point that efficiency of animal production must be measured in terms of the product produced, i.e., muscle not animal weight relative to the amount and cost of feedstuffs ingested. Traditional approaches using animal weight to estimate efficiency are not appropriate because they include an expensive waste product, fat that is not only costly to produce but when produced, costly to eliminate, by trimming. Furthermore, fat has negative health conotations to the human consumer in the U.S.A. if it is present in the product beyond extremely limited levels.

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The young animal deposits considerably more muscle mass than fat mass and is very efficient at using feedstuffs to produce the desired product, lean meat. As the animal continues to grow relatively more fat mass is produced causing a decrease in the efficiency of production of product (Etherton and Walton, 1986). This natural change in compartmentalization of the mass of the animal from predominantly muscle to an increasing proportion of fat is what is usually called repartitioning of growth. The desired effect for efficient lean muscle production is to change this pattern of compartmentalization in the latter stages of growth toward more muscle and less fat production. There are several endocrinologically mediated approaches to achieve this response, namely, sex steroid administration to cattle or sheep but not to pigs, somatotropin and β-adrenergic agonist administration to cattle, pigs or sheep (reviewed in Mersmann, 1987a).

I will summarize the biology of β-adrenergic agonists, the effects of feeding such compounds to pigs and some possible mechanisms by which such effects may be achieved.

BIOLOGY OF β-ADRENERGIC AGONISTS

Endogenous adrenergic agonists. The catecholamines are structurally related compounds (figure 1) that function in mammals as hormones or as nervous system neurotransmitters. Norepinephrine or noradrenaline is the chemical neurotransmitter for portions of the brain and for the sympathetic nervous system. The adrenal medulla produces as an end product the hormone, epinephrine or adrenaline which is secreted to the blood stream. Dopamine is a neurotransmitter for both central and peripheral nerves. Dopamine is a biosynthetic precursor of norepinephrine, the latter produced by attachment of a hydroxyl group to the sidechain carbon adjacent to the ring. Epinephrine is biosynthetically derived from norepinephrine by addition of a methyl group to the sidechain nitrogen. Epinephrine is considered a hormone because it is secreted by the adrenal medulla into the bloodstream to be delivered to various tissues peripheral to the adrenal gland. Dopamine and norepinephrine, the neurotransmitters, are likewise present in the blood stream, suggesting that these substances also may have hormonal activity at peripheral sites. In mammals, the circulating concentration of norepinephrine is greater than that of epinephrine whereas dopamine concentration is greater, less or equal to epinephrine but less than norepinephrine concentration (Buhler et al., 1978). The synthesis, degradation and biological function of adrenergic agonists has been reviewed many times. The reader is referred to several such reviews for details of the biochemistry, physiology and pharmacology of catecholamines and other adrenergic agents (Gilman et al., 1980; Martin, 1985; Cooper et al., 1986).

Adrenergic receptors. Observations on the physiology and pharmacology of catecholamines predominantly have been with isolated organs or tissues in vitro but there also have been many studies with

intact animals. Because the catecholamine hormones and neurotransmitters have a multitude of effects in mammals, the functions in vivo and sometimes in vitro are complex and intertwined. Many of these complex and multitudinous observations are comprehensively reviewed (Szekeres, 1980). In order to simplify these observations, Ahlquist (1948) developed the concept of two types of adrenergic receptors, the α- and β-adrenergic receptor. Functions such as heart contractility and rate, fat cell lipolysis and bronchodilation are stimulated by β-adrenergic receptors. Functions such as contraction of gut sphincters or arterioles in the skin or cerebrum are controlled by α-adrenergic receptors. The categorization of adrenergic function was helped by the synthesis of many analogs of norepinephrine. The rather simple structure allowed changes in substituents on the ring, changes in substituent position on the ring, changes in ring structure lengthening and substitution on the sidechain and simple to complex substitution on the sidechain amine. Some of the analogs are more effective or more potent than norepinephrine or epinephrine, some are relatively selective for α- or β-adrenergic receptors, some are relatively tissue specific regardless of receptor specificity and some are inhibitory, i.e., antagonize or block adrenergic receptor function. Literally thousands of such compounds have been synthesized in the last 50 years. One of the simplest and most useful norepinephrine analogs is isoproterenol (figure 1) because it stimulates only β-adrenergic receptors, i.e., it is a pure β-adrenergic agonist.

The increase in physiological and pharmacological observations over time coupled with the availability of many norepinephrine analogs increased the complexity of adrenergic receptor function. The concept of β_1- and β_2-subtypes of the β-adrenergic receptor was proposed by Lands et al. in 1967 and much later classification of the α-adrenergic receptor into α_1- and α_2-subtypes was proposed (Johansson, 1984). The receptor classification into subtypes has helped interpretation of the complexity of adrenergic physiology and pharmacology and has been the impetus for synthesis of agonists and antagonists specific not only for α- and β-adrenergic receptors but also for the subtypes. The concept that the heart and lungs had different β-adrenergic receptors, β_1- and β_2-, respectively, led to attempts to find agonists that would stimulate bronchodilation (β_2) with little effect on heart contractility (β_1). Thus, a norepinephrine analog such as terbutaline was developed to treat asthmatics without the cardiac side effects of the traditional therapy with epinephrine or isoproterenol. A brief summary of adrenergic subtypes and function is indicated in table 1.

Norepinephrine and epinephrine stimulate both α- and β-adrenergic receptors. Epinephrine is more potent toward α- and β_2-adrenergic receptors than norepinephrine whereas norepinephrine is more potent toward β_1-adrenergic receptors than epinephrine. Isoproterenol has essentially equal potency and efficacy toward β_1- and β_2-adrenergic receptors but does not interact with α-adrenergic receptors. There are specific dopamine receptors, distinct from the α- and β-adrenergic receptors.

Classification of adrenergic receptors into subtypes and of norepinephrine analogs as specific agonists or antagonists for α- or β-adrenergic receptors or their subtypes has been augmented by measurement of specific binding of radiolabelled antagonists or agonists to membrane receptors (Williams and Lefkowitz, 1978). Regardless of the approach, biological function or receptor binding, classification of receptors into subtypes and of individual agonists or antagonists as specific for receptor subtypes is complex and requires cautious interpretation. Classification of norepinephrine analogs as specific for β_1-adrenergic receptors usually has used rat or guinea pig heart whereas specificity for β_2-adrenergic receptors has used lung from guinea pig or other mammalian species. These heart and lung tissues have exclusively or at least predominantly β_1- and β_2-adrenergic receptors, respectively (Minneman et al., 1979; O'Donnell and Wanstall, 1981; Kenakin, 1984). These tissues may be the exception rather than the rule for the same tissues in other species or other tissues in these same species appear to have varying proportions of both β_1- and β_2-adrenergic receptors. The use of a single norepinephrine analog designated as specific for β_1- or β_2-adrenergic receptors to classify β-adrenergic receptor activity in another species or tissue is not by itself valid. Furthermore, the β-adrenergic receptor in a specific cell type may have different specificity for the chemical structure of norepinephrine analogs than the receptors in the cell types used for classification. For example, clenbuterol has been classified as a β_2-adrenergic agonist using such heart and lung preparations (O'Donnell, 1976) but the effects observed in cattle, pigs and sheep when clenbuterol is fed may extend far beyond interaction with β_2-adrenergic receptors in these species. The adrenergic receptor classification into subtypes and the related classification of norepinephrine analogs as specific for receptor subtypes probably will become less useful as more tissues in more species are investigated because of species and tissue dictated receptor specificity coupled with varied proportions of receptor subtypes.

Function. The cell membrane bound β-adrenergic receptor is coupled through a cascade type system to activation of the enzyme, adenylate cyclase to synthesize cyclic-AMP that in turn activates an enzyme, protein kinase that subsequently phosphorylates intracellular proteins (figure 2). The phosphorylation of intracellular enzyme proteins either activates or inactivates them, e.g., the enzyme, hormone sensitive lipase is activated by phosphorylation so that interaction of a β-adrenergic agonist with its receptor on an adipose tissue cell causes phosphorylation and activation of this enzyme to increase lipolysis or the degradation of fat within the adipocyte.

Both β_1- and β_2-adrenergic receptors are coupled to adenylate cyclase in the same manner to cause increased cyclic-AMP production whereas α_2-adrenergic receptors are coupled to adenylate cyclase to inhibit cyclic-AMP production (Birnbaumer et al., 1985). For example, in adipose tissue stimulation of β-adrenergic receptors increases cyclic-AMP production and, consequently, lipolysis whereas stimulation of α_2-adrenergic receptors decreases cyclic-AMP production and reduces lipolysis (Fain and Garcia-Sainz, 1983). The α_1-adrenergic receptor

operates by coupling to phosphoinositol metabolism using intracellular Ca^{++} and diacylglycerol as intracellular mediators; it does not couple to adenylate cyclase (Downes and Michell, 1985).

Adrenergic receptor function may be modified by cell-surface receptor density. The number of receptors on the cell surface subject to interaction with adrenergic agonists can be lowered (or raised) so that a common observation is desensitization to agonists or down-regulation of receptors. Desensitization is observed in many cell types after chronic exposure to adrenergic agonists and is usually rather rapid (Sibley and Lefkowitz, 1985). This is one mechanism to diminish continuous hormone effects during periods of chronic exposure to elevated hormonal concentration.

There is a plethora of physiological function controlled by adrenergic mechanisms because there are adrenergic receptors on most cell types, epinephrine and norepinephrine are present in the bloodstream and there is sympathetic nervous system innervation of most tissues (Gilman et al., 1980; Szekeres, 1980; Martin, 1985). Such functions as heart rate and force of contraction, bronchial constriction or dilation, blood vessel constriction or dilation, motility of the gut and uterus, skeletal muscle contraction and many others are changed with the status of stimulation of adrenergic receptors. Major metabolic functions such as lipolysis, lipogenesis, glycogen metabolism, glycolysis and gluconeogenesis are modulated by adrenergic control (Ellis, 1980). Effects of an exogenous norepinephrine analog are difficult to predict because of the multitudinous possibilities for physiological and metabolic regulation and also because of divergent receptor subtype distribution, specifity and density in individual tissues within a species and between species within a given tissue. Specifies specificity for pharmacodynamics and pharmacokinetics including absorption, distribution, metabolism and excretion of the exogenous norepinephrine analog can further modify the effects observed in a given species.

ORAL ADMINISTRATION OF β-ADRENERGIC AGONISTS TO PIGS

Effects. Several analogs of norepinephrine have been fed to pigs during the latter stages of growth. The typical approach is to mix the β-adrenergic agonist with feed and administer it to pigs fed ad libitum beginning at 50 to 60 kg live weight. The first reports on such an approach to change swine growth were in 1984 by scientists at American Cyanamid Company (Ricks et al., 1984) using clenbuterol (figure 1). Work by this company on clenbuterol was discontinued in favor of another analog, cimaterol. Summarization of the effects of the oral administration of either of these compounds (table 2) to pigs indicates small or no improvement in average daily gain, probably a modest reduction in feed intake and probably a modest improvement in feed efficiency. There is an unquestionable and substantial increase in muscle mass and a decrease in fat mass. Thus, evaluation of cimaterol using classical assessment of growth would not yield impressive effects but if the efficiency of growth were calculated as the

efficiency of product production, these animals would have greatly improved carcass composition and efficiency of lean meat production. The red-meat producing industry must develop approaches to reward the producer for the product. The live weight basis of marketing hogs is unacceptable when the consumer demands a lean pork product. The use of β-adrenergic agonists as demonstrated with clenbuterol and then cimaterol is an approach to production of pigs to match the current and projected niche in the marketplace.

Recently, effects of another norepinephrine analog, ractopamine (table 1), have been presented. Ractopamine, produced by Eli Lilly and Company, when orally administered to pigs, has major effects on muscle and fat accretion but in contrast to clenbuterol or cimaterol, ractopamine substantially increases average daily gain and improves classical feed efficiency (table 2). Calculation of efficiency of lean muscle production would indicate a remarkable change in production efficiency. Finally, the initial information on a β-adrenergic agonist, L644,969 (table 1) produced by Merck, Sharp and Dohme Research Laboratories indicates this norepinephrine analog also will improve classical feed efficiency and muscle accretion rates as well as decrease fat accretion rates (table 2).

Utility. It is too early to compare these compounds in regard to their utility for improving growth in pigs. Valid comparison will be to test simultaneously two or more analogs at the manufacturers recommended dose level in the same animal trial. Utility will be evaluated ultimately from a combination of factors in addition to the specific effects on growth rate, efficiency of production and body composition. The price will largely reflect not only the level needed in the feed but also the cost of organic synthesis and formulation of product. Although the norepinephrine analogs fed to pigs are somewhat similar in structure (figure 1), the chemical approaches to synthesis are undoubtedly different and could vary considerably in cost. The stability of the compound when mixed in feed could influence the comparative utility of compounds. Finally, the price charged must be balanced against the effects produced. Feed levels of cimaterol currently being used for experimental trials are about 1 mg·kg diet^{-1} or less, those for ractopamine about 20 mg·kg diet^{-1} or less and those for L655,969 about 1 mg·kg diet^{-1} or less (see references in table 2).

A major hurdle to utility of any of these compounds is the overall safety which can affect the ultimate approval for use in meat-producing animals. At this time, none of the norepinephrine analogs is approved for use in pigs or for that matter in cattle, poultry or sheep. Because there is preliminary approval of cimaterol and ractopamine as investigative new drugs, it probably can be projected that safety to the human consumer of meat products from treated animals will present few problems. The short withdrawal times for cimaterol, ractopamine and L644,969 (personal communication from R. H. Dalrymple, D. B. Anderson and E. M. Convey, respectively), indicate that any residues from these particular compounds are rapidly dissipated. The questions pertaining to type and amount of residues as well as toxicity and rate of dissipation of residues will vary with different

norepinephrine analogs. Some analogs could present major residue problems but these analogs would not be expected to reach the marketplace or even the stage of investigative drug experimentation.

There could be three problems with β-adrenergic agonists used to enhance efficient porcine growth. Firstly, hoof lesions and/or lameness may be a problem with some of these compounds. Lameness or locomotor unsoundness has been reported for clenbuterol (unpublished data in Moser et al., 1986), cimaterol (Cromwell et al., 1987) and L644,9696 (Wallace et al., 1987). Hoof cracks are observed with cimaterol (Jones et al., 1985; Cromwell et al., 1987). The seriousness and extent of these effects remains to be determined when large numbers of pigs are exposed under a variety of husbandry conditions. To this point, lameness and hoof lesions do not seem to be a problem with ractopamine (D. B. Anderson, personal communication). Secondly, the meat produced may have somewhat less desirable organoleptic attributes because of reduced marbling and/or changes in muscle fiber structure to change tenderness. There is no data published on organoleptic properties of porcine muscle from pigs treated with a β-adrenergic agonist. Sheep (Hamby et al., 1986) and cattle (S. B. Smith, personal communication) fed clenbuterol have increased shear force (i.e., less tender) in the longissimus muscle. However, another report from the same laboratory with cattle (Miller et al., 1987) indicates no change in shear force. It is not known how extensive such observations will be, whether they will be pertinent to other norepinephrine analogs, whether they will be detectable by the consumer, or whether they will translate to pigs. Finally, there could be reversal of effects on growth rate and efficiency or on carcass composition during a withdrawal period. One study in pigs (Jones et al., 1985) indicated no change in these parameters during a seven day withdrawal from cimaterol except that carcass fat measurements returned toward control levels. This would not appear to be a real problem for cimaterol because its current withdrawal time is considerably less than seven days. Whether withdrawal time will have any negative effects on the enhancement of growth or improvement of carcass composition achieved with other β-adrenergic agonists remains to be demonstrated.

Overall, it can be concluded that β-adrenergic agonists provide an effective approach to efficient production of lean porcine muscle. The compounds used to this time also appear to be safe with few residue problems as indicated by the short withdrawal times. Considering that investigative drug withdrawal times usually are established conservatively, withdrawal times may be even shorter after final approval for marketing. The impact of potential side effects such as hoof and leg soundness or meat tenderness remain to be established on large and varied populations of pigs.

MECHANISMS

The mechanisms involved in β-adrenergic agonist enhancement of body weight gain, reduction in feed intake, increased carcass muscle mass or decreased carcass fat mass are not known. Furthermore, they

may never be known and the mechanism for each norepinephrine analog may be different in different species or the mechanism of different analogs may be different within a given species. Speculation about the mechanism of any biologically active material should remain within the demonstrated activities of that material. Consequently, I will discuss potential mechanisms of β-adrenergic agonists to change growth of pigs primarily from the perspective of what is known about such agonists in the pig. Because so little is known about these agonists in pigs, information obtained in cattle, poultry, rats and sheep will be included to broaden the perspective. Until demonstrated in the pig, such projected mechanisms are even more speculative.

Adipose tissue. Direct interaction of norepinephrine analogs with adipose tissue β-adrenergic receptors could be a major mechanism to yield decreased fat deposition in animals fed such analogs (figure 3). There are receptors on adipose tissue cells for β-adrenergic agonists (Lafontan and Berlan, 1985); bovine (Jaster and Wagner, 1981) and porcine adipose tissue (Bocklen et al., 1986) appear to have such receptors. Stimulation of lipolysis or fat degradation in adipose tissue would provide a mechanism for decrased deposition of fat. Interaction of β-adrenergic agonists with their adipose tissue receptor via the activation cascade (figure 2) phosphorylates and activates the adipose tissue enzyme, hormone sensitive lipase that cleaves adipose tissue triacylglycerol, the storage fat to free fatty acids. Fatty acids are to a large extent, transported to the blood stream to be used by other organs as an energy source or for biosynthetic activities (Fain and Garcia-Sainz, 1983; Vernon and Clegg, 1985). Incubation of porcine adipose tissue with some β-adrenergic agonists in vitro stimulates lipolysis (Mersmann et al., 1974) so that this could be a mechanism for the reduction of fat in pigs fed β-adrenergic agonists. Porcine adipose tissue has rather stringent specificity for β-adrenergic agonists in vitro (Mersmann, 1984). Thus, this mechanism may not be appropriate for some norepinephrine analogs in pigs. The specificity of porcine adipose tissue for β-adrenergic agonist structures has been verified to some degree by acute infusion of agonists into pigs using plasma concentration of free fatty acids as an indicator of lipolysis in vivo (Mersmann, 1987b). A concrete example of a β-adrenergic agonist that decreases fat deposition when fed to pigs but does not change lipolysis or adipose tissue cyclic-AMP concentration in vitro is clenbuterol (Hu et al., 1987; Mersmann, 1987b). Acute infusion of clenbuterol into pigs does increase plasma free fatty acid concentration so that other mechanisms than direct stimulation of adipose tissue β-adrenergic receptors are implied. Clenbuterol very effectively stimulates lipolysis in vitro in adipose tissue from poultry (Campbell and Scanes, 1985) and sheep (Thornton et al., 1985) so that in these species the observed decrease in carcass adipose tissue might result from direct stimulation of adipose tissue lipolysis.

Adipose tissue fat synthesis represented by the synthesis of long-chain fatty acids and the esterification of fatty acids into triacylglycerol is inhibited by β-adrenergic agonists (Saggerson. 1985). Inhibition of either or both of these anabolic pathways could

contribute to the decreased adipose tissue deposition observed when animals are fed β-adrenergic agonists. When porcine adipose tissue is incubated with epinephrine, isoproterenol, clenbuterol or fenoterol in vitro, there is no inhibition of glucose incorporation into long-chain fatty acids or into total lipids (Rule et al., 1987). This observation is unexpected because of previous demonstrations of such inhibition in adipose tissue from rodents (Saggerson, 1985), sheep (Thornton et al., 1985) and of lipid synthesis in chicken hepatocytes (Campbell and Scanes, 1985), the site of fatty acid biosynthesis in this species.

Triacylglycerol biosynthesis is inhibited in rat adipose tissue by norepinephrine analogs (Saggerson, 1985). This pathway is inhibited also in porcine adipose tissue by isoproterenol (Rule et al., 1987) but only under very specific conditions of incubation in vitro. If the triacylglycerol biosynthetic pathway is inhibited by norepinephrine analogs in pigs in vivo, at least some of the decrease in adipose tissue accretion would be accounted for. Clenbuterol, one of the norepinephrine analogs that decreases fat deposition when fed to pigs, does not inhibit triacylglycerol biosynthesis in vitro in porcine adipose tissue. Thus, clenbuterol, a compound that changes carcass composition in pigs, does not affect any aspect of porcine adipose tissue lipid metabolism in vitro. This may not be surprising in light of the extreme structural specificity porcine adipose tissue demonstrates for β-adrenergic agonists to stimulate lipolysis in vitro (Mersmann, 1984). Presumably, this specificity resides in the porcine adipose tissue β-adrenergic receptor.

Recent reports suggest rates of fatty acid synthesis in vitro are inhibited in adipose tissue obtained from pigs fed ractopamine (Merkel et al., 1987; Williams et al., 1987). To my knowledge, comparable studies with clenbuterol or cimaterol have not been reported in pigs. Ractopamine does not inhibit acetate incorporation into lipids when added to porcine adipose tissue in vitro (A. C. Williams, personal communication) as observed for clenbuterol (Rule et al., 1987). Adipose tissue from pigs fed ractopamine also appears to have decreased rates of lipolysis (Merkel et al., 1987), but incubation of porcine adipose tissue with ractopamine in vitro did not stimulate lipolysis (A. C. Williams, personal communication), as observed with clenbuterol (Rule et al., 1987).

The attempts to demonstrate effects of norepinephrine analogs on porcine adipose tissue in vitro have to a large extent been negative with those analogs that when fed to pigs reduce carcass fat deposition. This type of observation may imply that porcine adipose tissue does not function, even qualitatively, in vitro as it functions in vivo or the more likely implication that many of the effects obtained from feeding β-adrenergic agonists to pigs result from indirect effects rather than direct interaction with the porcine adipose tissue β-adrenergic receptor. This conclusion cannot be extrapolated to all β-adrenergic agonists because some are expected to interact with the receptor (Mersmann, 1984). The conclusion cannot be extrapolated to adipose tissue from other species because clenbuterol has been shown

to stimulate lipolysis in ovine and chicken adipose tissue in vitro and inhibit lipogenesis in ovine adipose tissue and chicken liver in vitro (Thornton et al., 1985; Campbell and Scanes, 1985). Ractopamine (Hausman et al., 1987), clenbuterol and several other β-adrenergic agonists (Duquette and Muir, 1985) inhibit lipogenesis and stimulate lipolysis in rat adipose tissue preparations in vitro.

Muscle. Norepinephrine and its analogs were thought to be catabolic on skeletal muscle because they produced degradative effects such as breakdown of glycogen by stimulation of the enzyme, phosphorylase. Only more recently has stimulation of anabolic processes been demonstrated in muscle. There are receptors for β-adrenergic agonists on skeletal muscle membranes (Williams et al., 1984) including pig muscle membranes (Bocklen et al., 1986). Norepinephrine analogs stimulate anabolic processes such as amino acid transport and protein synthesis in skeletal muscle in vitro (Deshaies et al., 1981; Nutting, 1982) and also decrease catabolism or protein degradation (Garber et al., 1976; Li and Jefferson, 1977). Thus, the observations of increased muscle mass in red-meat producing animals fed norepinephrine analogs may result from direct interaction with the skeletal muscle cell (figure 4).

The demonstration of direct effects on muscle of those norepinephrine analogs fed to meat producing animals is limited. Cimaterol increases the myofibrillar fraction of chicken muscle cells in culture; the mechanism to achieve this change is not yet known (Young et al., 1987). Cimaterol does not change apparent rates of protein synthesis in ovine skeletal muscle in vitro (Wilson et al., 1987) nor does it change apparent rates of protein synthesis or degradation in cultured L6 myotubes (Roeder et al., 1987). There are more evidences, including some in pigs, of effects of norepinephrine analogs on skeletal muscle when the analog is fed. Skeletal muscle protein synthesis is increased in rats given clenbuterol (Emery et al., 1984), in pigs given ractopamine (Bergen et al., 1987) and possibly in sheep given cimaterol (Wilson et al., 1987). Other studies have not been able to demonstrate increased muscle protein synthesis in rats given clenbuterol (Reeds et al., 1986), in sheep given clenbuterol (Bohorov et al., 1987) and in pigs given ractopamine (Johnson et al., 1987). Likewise, skeletal muscle protein degradation seems to be decreased in some studies, for example, in rats given clenbuterol (Reeds et al., 1986), in sheep given clenbuterol (Bohorov et al., 1987) and in pigs given ractopamine (Bergen et al., 1987). Other studies cannot demonstrate an effect on protein degradation, for example, sheep given cimaterol (Wilson et al., 1987) and pigs given ractopamine (Johnson et al., 1987). The messenger RNA for bovine skeletal muscle myosin light chain is increased in cattle fed ractopamine suggesting an increased synthesis of specific myofibrillar proteins (Smith et al., 1987). At this time, the data generated is of a mixed nature so that it is not possible to conclude whether β-adrenergic agonists produce increased muscle mass by direct effects on muscle cells. It should be noted that the data generated to this time are with several different norepinephrine analogs, which may have different mechanisms and,

furthermore, represent studies in several species, each of which may react differently to a given β-adrenergic agonist.

The increase in size observed in muscle from animals fed norepinephrine analogs seems to be the result of muscle cell hypertrophy. Muscle fiber diameter is increased in sheep (Beermann et al., 1985, 1987; Hamby et al., 1986; Kim et al., 1987) and in cattle (Miller et al., 1987) fed either clenbuterol or cimaterol. It is not clear whether the increase in muscle fiber size is restricted to a particular type of fiber; in sheep fed cimaterol there is hypertrophy of both Type I and Type II myofibers (Beermann et al., 1985, 1987; Kim et al., 1987) whereas in cattle fed clenbuterol, hypertrophy is observed only in Type II myofibers (Miller et al., 1987).

Blood Flow. In addition to direct action on adipose tissue and skeletal muscle, the two tissues most affected when pigs are fed a β-adrenergic agonist, several other potential mechanisms could contribute to the observed effects in vivo. One of the most obvious mechanisms is that β-adrenergic agonists could stimulate blood flow to various tissues including muscle and adipose tissue. Most β-adrenergic agonists have slight to very large effects on the circulatory system; they increase the rate and force of cardiac contraction and they cause vasodilation. These types of change would be expected to increase blood flow to various tissues. The ultimate controls on blood flow are complex but suffice it to say that blood flow may be differentially regulated in individual tissues. Infusion of isoproterenol to rats causes increased blood flow to heart, skeletal muscle and brown fat but decreased blood flow to kidney, liver and white fat (Wickler et al., 1984). Stimulation of the sympathetic nervous system in which the major chemical mediator is norepinephrine, likewise causes decreased blood flow to white adipose tissue (Fredholm, 1985). It is not known whether any of the β-adrenergic agonists including those currently fed to meat-producing animals cause differential blood flow changes in skeletal muscle or adipose tissue of cattle, pigs or sheep. Clenbuterol increases heart rate and hind limb blood flow when fed to cattle (Williams et al., 1986; Eisemann et al., 1987) whereas cimaterol increases hind limb blood flow when fed to sheep (Beermann et al., 1986). Similar studies have not been done in pigs but heart rate is increased in acute experiments when clenbuterol is infused intravenously into pigs (Mersmann, 1987b). It should be noted that although increased blood flow may be a viable mechanism to explain at least part of the effects of orally administered β-adrenergic agonists, it is expected there will be major species differences in the detailed observations. Animals must adapt to chronic administration of an agonist that has large effects on physiological function because organ, tissue or cellular mechanisms cannot function at extreme rates over an extended period. Animals adapt by various desensitization processes. Animals must adapt to an agonist that greatly stimulates heart rate; for example, heart rate and hind limb blood flow are increased 70 and 85% in cattle initially fed clenbuterol but 9 days later these percentages are 25 and 40%, respectively (Eisemann et al., 1987). Cimaterol-fed sheep have elevated hind limb blood flow of 44 and 14% after 2 and 4 weeks of feeding (Beermann et al., 1986). The blood

flow studies indicate that cattle and sheep and presumably pigs do adapt to chronic feeding of β-adrenergic agonists but that blood flow remains elevated for an extended period.

Hormone Release. Secretion of many metabolic hormones may be influenced positively or negatively by adrenergic agonists and this may vary depending on the agonist and the species (Huang et al., 1983). Insulin release by the pancreas is inhibited by β-adrenergic agonists and has been demonstrated in the pig (Hertelendy et al., 1966). This could provide a mechanism to decrease fat accretion because lower insulin concentration in the blood stream and, thus, at the level of the adipose tissue cell might be expected to decrease lipogenesis or synthesis of fat and to increase lipolysis or degradation of fat. Insulin control of adipose tissue metabolism has been demonstrated in many mammalian species including cattle, pigs and sheep (Vernon, 1981; Mersmann, 1986a). Acute effects of insulin on porcine adipose tissue fatty acid or triacylglycerol synthesis in vitro are difficult to demonstrate consistently (Mersmann and Hu, 1987; Rule et al., 1987) but in at least one laboratory larger and more consistent stimulation of anabolic processes is observed (Walton and Etherton, 1986). Inhibition of lipolysis, the degradative process is readily observed in porcine adipose tissue in vitro and circulating free fatty acids concentration is depressed during acute insulin infusion in pigs (Mersmann, 1986b; Mersmann and Hu, 1987; Rule et al., 1987). There are no reports on circulating hormone concentrations in pigs fed β-adrenergic agonists. Studies in sheep fed cimaterol by Beermann et al. (1985; 1987) indicate changes in plasma concentration of several metabolic hormones; insulin and somatomedin are decreased, somatotropin and thyroxine are increased and prolactin, triiodothyronine and cortisol are unchanged. Even these studies with one species and one β-adrenergic agonist indicate great complexity regarding effects on circulating hormones. The complexity can only increase with other β-adrenergic agonists with altered specificity and pharmacodynamics compared to cimaterol and other species with different receptor specificity and distribution on various endocrine gland cells.

Other mechanisms. Although there are many possible mechanisms that could be speculated about, one that may be operative with some β-adrenergic agonists is a decrease in feed intake as a result of interaction with the central nervous system (Garattini and Samenin, 1984). Cimaterol and ractopamine appear to decrease feed intake in pigs (table 2). Slightly depressed feed intake would not be expected to decrease muscle mass but it would decrease fat deposition that is most closely associated with intake of dietary energy (Henry, 1985).

Because of the extensive distribution of β-adrenergic receptors in essentially all tissues in the mammalian body, there are many possible ways in which an orally administered β-adrenergic agonist could affect growing pigs. The importance of some of these interactions will be slight but others will have major impact. The mechanism for a given β-adrenergic agonist will most likely be a mosaic effect involving both positive and negative effects on many tissues.

Depending on the specificity of the agonist, there could be direct effects on adipose tissue or muscle, increased or decreased blood flow to specific tissues, changes in circulating concentration of one or many metabolic hormones and central nervous system effects including change in appetite. Given one, or more likely several of these direct effects, an asundry of indirect effects will result yielding altered metabolic function in a variety of tissues. Species specificity of receptors, their distribution and of the metabolism and distribution of the agonist will further complicate the operative mechanism(s) for a given β-adrenergic agonist to repartition growth in pigs as well as cattle, poultry and sheep.

ACKNOWLEDGEMENTS

Mention of trade names, proprietary products or specific equipment does not constitute a guarantee or warranty of the product by the USDA and does not imply its approval to the exclusions of other products that may also be suitable.

I thank M. M. Bierman for secretarial assistance.

The literature review for this paper was terminated September 1987.

LITERATURE CITED

Ahlquist, R. P. 1948. A study of the adrenotropic receptors. Am. J. Physiol. 153:586.

Anderson, D. B., E. L. Veenhuizen, W. P. Waitt, R. E. Paxton and D. H. Mowrey. 1987. Effect of ractopamine on nitrogen retention, growth performance and carcass composition of finisher pigs. J. Anim. Sci. 65(Suppl. 1):130 (Abstr. #109).

Beermann, D. H., W. R. Butler, V. K. Fishell, E. N. Bergman and J. P. McCann. 1986. Preliminary observations on the effects of cimaterol on heart rate, blood flow, plasma insulin concentration and net glucose uptake in the hindquarters of growing lambs. J. Anim. Sci. 63(Suppl. 1):225 (Abstr. #160).

Beermann, D. H., W. R. Butler, D. E. Hogue, V. K. Fishell, R. H. Dalrymple, C. A. Ricks and C. G. Scanes. 1987. Cimaterol-induced muscle hypertrophy and altered endocrine status in lambs. J. Anim. Sci. (In press)

Beermann, D. H., D. R. Campion and R. H. Dalrymple. 1985. Mechanisms responsible for repartitioning tissue growth in meat animals. Proc. Recip. Meat Conf. 38:105.

Bergen, W. G., S. E. Johnson, D. M, Skjaerlund, R. A. Merkel and D. B. Anderson. 1987. The effect of ractopamine on skeletal muscle metabolism in pigs. Fed. Proc. 46:1021 (Abstr. #4105).

Birnbaumer, L., J. Codina, R. Mattera, R. A. Cerione, J. D. Hildebrandt, T. Sunyer, F. J. Rojas, M. G. Caron, R. J. Lefkowitz and R. Iyengar. 1985. Structural basis of adenylate cyclase stimulation and inhibition by distinct guanine nucleotide regulatory proteins. In: P. Cohen and M. D. Houslay (Eds.) Molecular Mechanisms of Transmembrane Signalling. pp 131-182. Elsevier Science Publishers, New York.

Bocklen, E., S. Flad, E. Muller and H. von Faber. 1986. Comparative determination of beta-adrenergic receptors in muscle, heart and backfat of Pietrain and Large White pigs. Anim. Prod. 43:335.

Bohorov, O., P. J. Buttery, J. H. R. D. Correia and J. B. Soar. 1987. The effect of the β-2-adrenergic agonist clenbuterol or implantation with oestradiol plus trenbolone acetate on protein metabolism in wether lambs. Brit. J. Nutr. 57:99.

Buhler, H. U., M. Da Prada, W. Haefely and G. B. Picotti. 1978. Plasma adrenaline, noradrenaline and dopamine in man and different animal species. J. Physiol. 276:311.

Campbell, R. M. and C. G. Scanes. 1985. Adrenergic control of lipogenesis and lipolysis in the chicken in vitro. Comp. Biochem. Physiol. 82C:137.

Cooper, J. R., F. E. Bloom and R. H. Roth. 1986. The biochemical basis of neuropharmacology. Oxford University Press, New York. 5th Edition. Chapters 9 and 10.

Crenshaw, J. D., P. M. Swantek, M. J. Marchello, R. L. Harrold, R. C. Zimprich and R. D. Olson. 1987. Effects of a phenethanolamine (ractopamine) on swine carcass composition. J. Anim. Sci. 65(Suppl. 1):308 (Abstr. #276).

Cromwell, G. L., J. D. Kemp, T. S. Stahly, J. R. May and H. H. Monegue. Efficacy of cimaterol as a repartitioning agent for swine. J. Anim. Sci. 65(Suppl. 1):300 (Abstr. #258).

Dalrymple, R. H., P. K. Baker and C. A. Ricks. 1984. Repartitioning agents to improve performance and body composition. Proc. Geo. Nut. Conf. pp 111.

Deshaies, Y., J. Willemot and J. Leblanc. 1981. Protein synthesis, amino acid uptake, and pools during isoproterenol-induced hypertrophy of the rat heart and tibialis muscle. Can. J. Physiol. Pharmacol. 59:113.

Downes, C. P. and R. H. Michell. 1985. Inositol phospholipid breakdown as a receptor-controlled generator of second messengers. In: P. Cohen and M. D. Houslay (Eds.) Molecular Mechanisms of Transmembrane Signalling. pp 3-56. Elsevier Science Publishers, New York.

Duquette, P. F. and L. A. Muir. 1985. Effect of the beta-adrenergic agonists isoproterenol, clenbuterol, L-640,033 and BRL 35135 on lipolysis and lipogenesis in rat adipose tissue in vitro. J. Anim. Sci. 61(Suppl. 1):265 (Abstr. #141).

Eisemann, J. H., G. B. Huntington and C. L. Ferrell. 1987. Effects of dietary clenbuterol on metabolism of the hindquarters in steers. J. Anim. Sci. (In press)

Ellis, S. 1980. Effects on the metabolism. In: L. Szekeres (Ed.) Andrenergic Activators and Inhibitors, Handbook of Experimental Pharmacology. Vol. 54/I pp 319-352. Springer-Verlag, New York.

Emery, P. W., N. J. Rothwell, M. J. Stock and P. D. Winter. 1984. Chronic effects of β_2-adrenergic agonists on body composition and protein synthesis in the rat. Bioscience Rep. 4:83.

Etherton, T. D. and P. E. Walton. 1986. Hormonal and metabolic regulation of lipid metabolism in domestic livestock. J. Anim. Sci. 63 (Suppl. 1):76.

Fain, J. N. and J. A. Garcia-Sainz. 1983. Adrenergic regulation of adipocyte metabolism. J. Lipid Res. 24:945.

Fredholm, B. B. 1985. Nervous control of circulation and metabolism in white adipose tissue. In: A. Cryer and R. L. R. Van (Eds.) New Perspectives in Adipose Tissue: Structure, Function and Development. pp 45-64. Butterworths, London.

Garattini, S. and R. Samanin. 1984. d-Fenfluramine and salbutamol: Two drugs causing anorexia through different neurochemical mechanisms. Int. J. Obesity 8 (Suppl. 1):151.

Garber, A. J., I. E. Karl and D. M. Kipnis. 1976. Alanine and glutamine synthesis and release from skeletal muscle. IV. β-adrenergic inhibition of amino acid release. J. Biol. Chem. 251:851.

Gilman, A. G., L. S. Goodman and A. Gilman. (Eds.) 1980. In: Goodman and Gilman's The Pharmacological Basis of Therapeutics. MacMillan Publishing Co., Inc., New York. Chapters 4, 8, 9 and 12.

Hamby, P. L., J. R. Stouffer and S. B. Smith. 1986. Muscle metabolism and real-time ultrasound measurement of muscle and subcutaneous adipose tissue growth in lambs fed diets containing a beta-agonist. J. Anim. Sci. 63:1410.

Hancock, J. D., E. R. Peo, Jr. and A. J. Lewis. 1987. Effects of dietary levels of ractopamine (a phenethanolamine) on performance and carcass merit of finishing pigs. J. Anim. Sci. 65(Suppl. 1): 309 (Abstr. #277).

Hanrahan, J. P., J. F. Quirke, W. Bomann, P. Allen, J. C. McEwan, J. M. Fitzsimons, J. Kotzian and J. F. Roche. 1986. β-Agonists and their effect on growth and carcass quality. In: W. Haresign and D. J. A. Cole (Eds.) Recent Advances in Animal Nutrition. pp 125-138. Butterworths, London.

Hausman, D. B., R. J. Martin, E. L. Veenhuizen and D. B. Anderson. 1987. Effect of the phenethanolamine (ractopamine) on in vitro insulin sensitivity and responsiveness of isolated rat adipocytes. Fed. Proc. 46:1178 (Abstr. #5017).

Henry, Y. 1985. Dietary factors involved in feed intake regulation in growing pigs: A review. Livestock Prod. Sci. 12:339.

Hertelendy, F., L. J. Machlin, R. S. Gordon, M. Horino and D. M. Kipnis. 1966. Lipolytic activity and inhibition of insulin release by epinephrine in the pig. Proc. Soc. Exp. Biol. Med. 121:675.

Hu, C. Y., J. E. Novakofski and H. J. Mersmann. 1987. Hormonal control of porcine adipose tissue fatty acid release and cyclic AMP concentration. J. Anim. Sci. 64:1031.

Huang, X. Y. and S. M. McCann. 1983. Effect of β-adrenergic drugs on LH, FSH, and growth hormone (GH) secretion in conscious, ovariectomized rats. Proc. Soc. Exp. Biol. Med. 174:244.

Jaster, E. H. and T. N. Wegner. 1981. Beta-adrenergic receptor involvement in lipolysis of dairy cattle subcutaneous adipose tissue during dry and lactating state. J. Dairy Sci. 64:1655.

Johansson, P. 1984. α-Adrenoceptors: recent development and some comparative aspects. Comp. Biochem. Physiol. 78C:253.

Johnson, S. E., W. G. Bergen, R. A. Merkel and M. A. Stachiw. 1987. The effect of Ractopamine (RAC) on skeletal muscle in swine. J. Anim. Sci. 65(Suppl. 1):277 (Abstr. #203).

Jones, R. W., R. A. Easter, F. K. McKeith, R. H. Dalrymple, H. M. Maddock and P. J. Bechtel. 1985. Effect of the β-adrenergic agonist cimaterol (CL 263,780) on the growth and carcass characteristics of finishing swine. J. Anim. Sci. 61: 905.

Kenakin, T. P. 1984. The classification of drugs and drug receptors in isolated tissues. Pharmacol. Rev. 36:165.

Kim, Y. S., Y. B. Lee, C. R. Ashmore and R. H. Dalrymple. 1987. Effect of cimaterol on skeletal muscle characteristics of lambs. J. Anim. Sci. 65(Suppl. 1):278 (Abstr. #205).

Lafontan, M. and M. Berlan. 1985. Plasma membrane properties and receptors in white adipose tissue. In: A. Cryer and R. L. R. Van (Eds.) New Perspectives in Adipose Tissue: Structure, Function and Development. pp 145-182. Butterworths, London.

Lands, A. M., A. Arnold, J. P. McAnliff, F. P. Ludvena and T. G. Brown. 1967. Differentiation of receptor systems activated by sympathomimetic amines. Nature 214:597.

Li, J. B. and L. S. Jefferson. 1977. Effect of isoproterenol on amino acid levels and protein turnover in skeletal muscle. Am. J. Physiol. 232:E243.

Martin, C. R. 1985. Endocrine Physiology. Oxford University Press, New York. Chap. 8.

Merkel, R. A., P. S. Dickerson, S. E. Johnson, R. L. Burkett, R. J. Burnett, A. L. Schroeder, W. G. Bergen and D. B. Anderson. 1987. The effect of ractopamine on lipid metabolism in pigs. Fed. Proc. 46:1177 (Abstr. #5015).

Mersmann, H. J. 1984. Adrenergic control of lipolysis in swine adipose tissue. Comp. Biochem. Physiol. 77C:43.

Mersmann, H. J. 1986a. Lipid metabolism in swine. In: H. C. Stanton and H. J. Mersmann (Eds.) Swine in Cardiovascular Research. Vol. 1, pp 75-104. CRC Press, Inc., Boca Raton, Florida.

Mersmann, H. J. 1986b. Acute effects of metabolic hormones in swine. Comp. Biochem. Physiol. 83A:653.

Mersmann, H. J. 1987a. Nutritional and endocrinological influences on the composition of animal growth. Prog. Food and Nutr. Sci. 11:175.

Mersmann, H. J. 1987b. Acute metabolic effects of adrenergic agents in swine. Am. J. Physiol. 252:E85.

Mersmann, H. J., Brown, L. J., Underwood, M. C. and Stanton, H. C. 1974. Catecholamine-induced lipolysis in swine. Comp. Biochem. Physiol. 47B:263.

Mersmann, H. J. and C. Y. Hu. 1987. Factors affecting measurements of glucose metabolism and lipolytic rates in porcine adipose tissue slices in vitro. J. Anim. Sci. 64:148.

Miller, M. F., D. K. Garcia, M. E. Coleman, P. A. Ekeren, F. Y. Wu, D. K. Lunt, K. A. Wagner, M. Prockner, T. H. Welsh, Jr. and S. B. Smith. 1987. Adipose tissue, longissimus muscle and anterior pituitary growth and function in clenbuterol-fed heifers. J. Anim. Sci. (In press)

Minneman, K. P., A. Hedberg and P. B. Molinoff. 1979. Comparison of beta adrenergic receptor subtypes in mammalian tissues. J. Pharmacol. Exp. Ther. 211:502.

Moser, R. L., R. H. Dalrymple, S. G. Cornelius, J. E. Pettigrew and C. E. Allen. 1986. Effect of cimaterol (CL 263,780) as a repartitioning agent in the diet for finishing pigs. J. Anim. Sci. 62:21.

Nutting, D. F. 1982. Anabolic effects of catecholamines in diaphragm muscle from hypophysectomized rats. Endocrinology 110:307.

O'Donnell, S. R. 1976. Selectivity of clenbuterol (NAB 365) in guinea-pig isolated tissues containing β-adrenoceptors. Arch. int. Pharmacodyn 224:190.

O'Donnell, S. R. and J. C. Wanstall. 1981. Pharmacological approaches to the characterization of β-adrenoreceptor populations in tissues. J. Auton. Pharmac. 1:305.

Prince, T. J., R. H. Dalrymple and D. N. Marple. 1985. Effects of feeding cimaterol (CL 263,780) on performance and carcass characteristics of finishing pigs. J. Anim. Sci. 61(Suppl. 1): 301 (Abstr. #230).

Prince, T. J., D. L. Huffman and P. M. Brown. 1987. Effects of ractopamine on growth and carcass composition of finishing swine. J. Anim. Sci. 65(Suppl. 1):309 (Abstr. #278).

Reeds, P. J., S. M. Hay, P. M. Dorwood and R. M. Palmer. 1986. Stimulation of muscle growth by clenbuterol: lack of effect on muscle protein biosynthesis. Brit. J. Nutr. 56:249.

Ricks, C. A., P. K. Baker and R. H. Dalrymple. 1984. Use of repartitioning agents to improve performance and body composition of meat animals. Proc. Recip. Meat Conf. 37:5.

Roeder, R. A., N. L. Hackmann, J. M. Arnzen and C. W. Hunt. 1987. Effect of β-adrenergic agonists on protein turnover in muscle cell cultures. Fed. Proc. 46:1177 (Abstr. #5010).

Rule, D. C., S. B. Smith and H. J. Mersmann. 1987. Effects of adrenergic agonists and insulin on porcine adipose tissue metabolism in vitro. J. Anim. Sci. 65:136.

Saggerson, E. D. 1985. Hormonal regulation of biosynthetic activities in white adipose tissue. In: A. Cryer and R. L. R. Van (Eds.) New Perspectives in Adipose Tissue: Structure, Function and Development. pp 87-120. Butterworths, London.

Sibley, D. R. and R. J. Lefkowitz. 1985. Molecular mechanisms of receptor desensitization using the β-adrenergic receptor-coupled adenylate cyclase system as a model. Nature 317:124.

Smith, S. B., D. K. Garcia, S. K. Davis, M. A. Patton and D. B. Anderson. 1987. Specific gene expression in longissimus muscle of steers fed ractopamine. J. Anim. Sci. 65(Suppl. 1): 278 (Abstr. #207).

Szekeres, L. (Ed.). 1980. Adrenergic activators and inhibitors. Handbook of Experimental Pharmacology Vol. 54/I. Springer-Verlag, New York.

Thornton, R. F., R. K. Tume, G. Payne, T. W. Larsen, G. W. Johnson and M. A. Hohenhaus. 1985. The influence of the β_2-adrenergic agonist, clenbuterol, on lipid metabolism and carcass composition of sheep. Proc. New Zealand Soc. Anim. Prod. 45:97.

Veenhuizen, E. L., K. K. Schmiegel, W. P. Waitt and D. B. Anderson. 1987. Lipolytic, growth, feed efficiency, and carcass responses to phenethanolamines in swine. J. Anim. Sci. 65(Suppl. 1):130 (Abstr. #108).

Vernon, R. G. 1981. Lipid metabolism in the adipose tissue of ruminant animals. In: W. W. Christie (Ed.) Lipid Metabolism in Ruminant Animals. pp 279-362. Pergamon Press, New York.

Vernon, R. G. and R. A. Clegg. 1985. The metabolism of white adipose tissue in vivo and in vitro. In: A. Cryer and R. L. R. Van (Eds.) New Perspectives in Adipose Tissue: Structure, Function and Development. pp 65-86. Butterworths, London.

Wallace, D. H., H. B. Hedrick, R. L. Seward, C. P. Daurio and E. M. Convey. 1987. Growth and efficiency of feed utilization of swine fed a beta-adrenergic agonist (L-644,969). EEC Seminar. Brussels, Belgium, May 19-20.

Walton, P. E. and T. D. Etherton. 1986. Stimulation of lipogenesis by insulin in swine adipose tissue: antagonism by porcine growth hormone. J. Anim. Sci. 62:1584.

Wickler, S. J., B. A. Horwitz, S. F. Flaim and K. F. LaNoue. 1984. Isoproterenol-induced blood flow in rats acclimated to room temperature and cold. Am. J. Physiol. 246:R747.

Williams, A. C., P. M. Brunjes, R. J. Martin, E. L. Veenhuizen and D. B. Anderson. 1987. Influence of feeding ractopamine to finishing swine on the in vitro responsiveness of adipose tissue to ractopamine and insulin. J. Anim. Sci. 65(Suppl. 1):249 (Abstr. #139).

Williams, L. T. and R. J. Lefkowitz. 1978. Receptor binding studies in adrenergic pharmacology. Raven Press, New York.

Williams, P. E. V., L. Pagliani and G. M. Innes. 1986. The effect of a β-agonist (clenbuterol) on the heart rate, nitrogen balance and some carcass characteristics of veal calves. Livestock Prod. Sci. 15:289.

Williams, R. S., M. G. Caron and K. Daniel. 1984. Skeletal muscle β-adrenergic receptors: variations due to fiber type and training. Am. J. Physiol. 246:E160.

Wilson, M. A., N. E. Forsberg, C. Zhong, R. H. Dalrymple, and C. A. Ricks. 1987. Protein synthesis and degradation in sheep skeletal muscle: Effects of maturity and of dietary and medium cimaterol. J. Anim. Sci. 65:251 (Abstr. #143).

Young, R. B., C. E. McGee, D. M. Moriarity, H. E. Richter, M. R. Campbell, J. A. Maupin and J. R. Hudson. 1987. Protein metabolism in chicken muscle cell cultures treated with cimaterol. Fed. Proc. 46:1020 (Abstr. #4101).

Table 1. Alpha- and beta-adrenergic function[a]

Receptor subtype	Function	Agonist
α_1	constrict arteries	methoxamine
α_2	inhibit lipolysis	clonidine
β_1	increase heart rate	dobutamine
β_2	bronchodilate	terbutaline

[a]Adrenergic function summarized in Gillman et al., 1980; Martin, 1985; Cooper et al., 1986.

Table 2. Approximate percentage response of pigs to oral β-adrenergic agonists

Agonist	Daily gain	Feed consumption	Feed efficiency	Protein/or muscle mass	Fat mass
clenbuterol[a]	0		0	+ 9	-10
cimaterol[b]	+2	-5	+ 5	+ 7	-10
ractopamine[c]	+7	-4	+10	+10	-12
L 644,969[d]			+10	+10	- 9

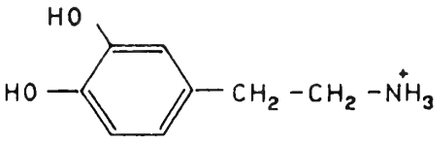
[a]Dalrymple et al., 1984.

[b]Dalrymple et al., 1984; Jones et al., 1985; Prince et al., 1985; Hanrahan et al., 1986; Moser et al., 1986; Cromwell et al., 1987.

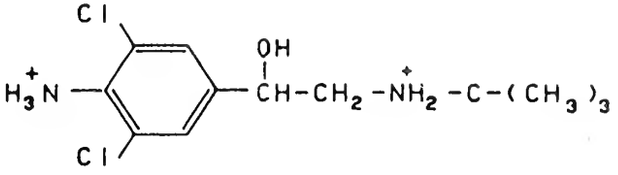
[c]Anderson et al., 1987; Crenshaw et al., 1987; Hancock et al., 1987; Prince et al., 1987; Veenhuizen et al., 1987.

[d]Wallace et al., 1987.

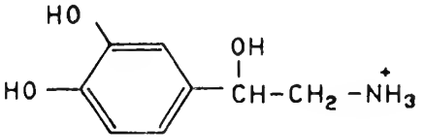
dopamine



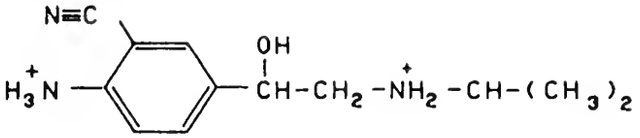
clenbuterol



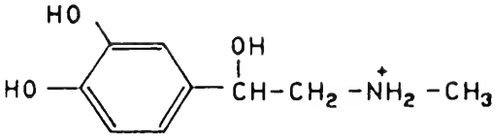
norepinephrine



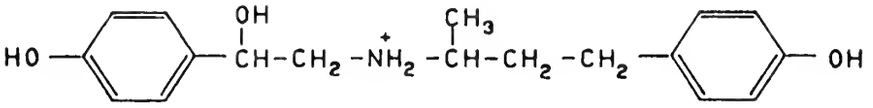
cimaterol



epinephrine



ractopamine



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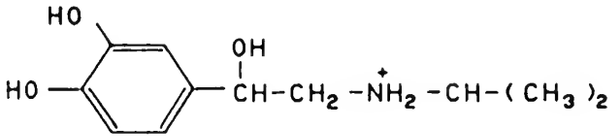

L644,969

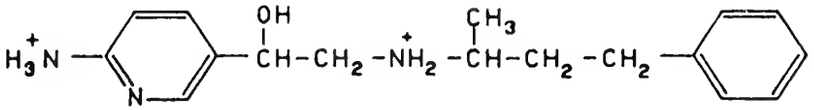

Figure 1. Structure of adrenergic hormones and selected β-adrenergic agonists.

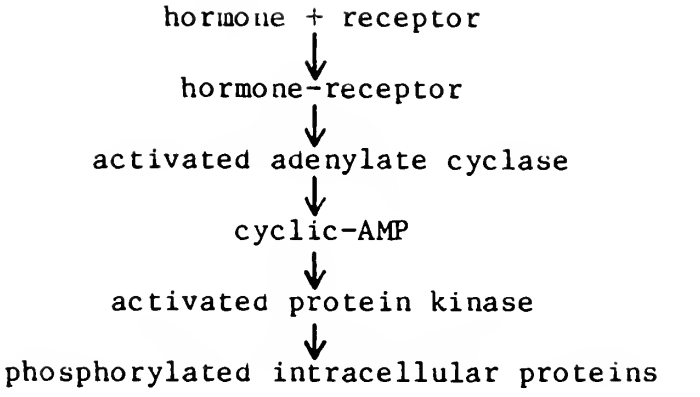

Figure 2. Adrenergic activation cascade.

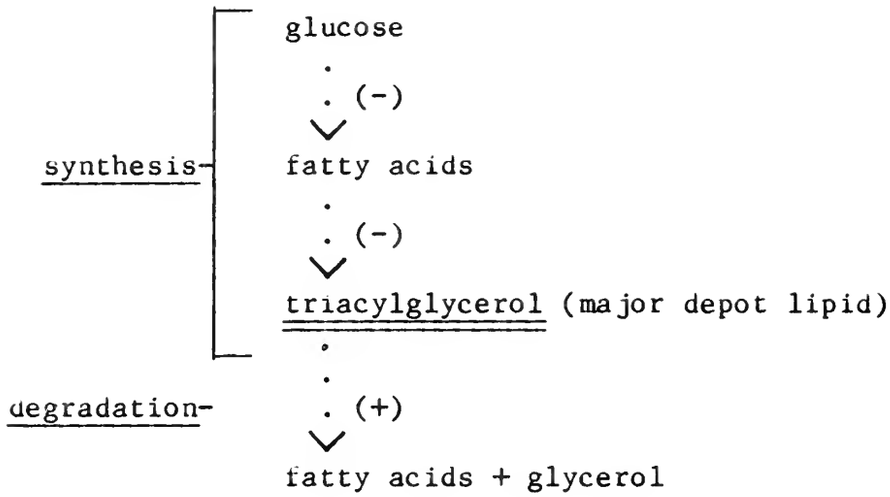

Figure 3. β-Adrenergic agonist control of adipose tissue metabolism.

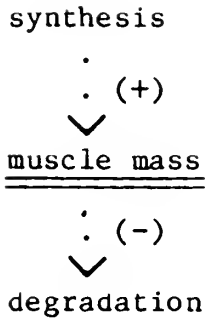

Figure 4. β-Adrenergic control of muscle metabolism.

Primer on Somatotropins and Their Effect on Finishing and Lactating Swine

Peter J. Bechtel

INTRODUCTION

The recent advances in molecular biology have resulted in new methods of producing protein hormones in large amounts. Although these new methods are relatively expensive, they allow commercial production of proteins that are normally not available for scientific studies. Proteins produced by these methods include a number of human hormones, such as human growth hormone and insulin, as well as some selected enzymes. The production of animal protein hormones which were previously not widely available is of use to the animal industry. The availability of recombinant bovine and porcine somatotropin has resulted in scientific studies on how these proteins alter an animal's metabolism, production characteristics and body composition. With a number of scientific studies behind us, commercial uses of recombinant bovine and porcine somatotropin may be on the horizon. Of special interest to this conference is the use of recombinant porcine somatotropin (rPST) to alter the production characteristics and composition of finishing swine.

Somatotropin

The biochemistry, physiology and endocrinology of somatotropin have been the subject of a number of reviews including Daughaday (1985), Laron (1983) and Malarkey (1980). Human somatotropin is a small 191 amino acid polypeptide which is synthesized and secreted by the pituitary gland (Fig. 1). The human somatotropin gene is located on the long arm of chromosome 17. Somatotropin has a molecular weight of about 21,500 daltons and contains two disulfide bonds which are necessary for the hormones biological activity. Somatotropin is first synthesized in the pituitary as a larger pre-somatotropin protein (220 amino acids) which is then proteolytically cleaved to the 21,500 dalton protein. The amount of somatotropin in an adult human pituitary is 5-15 mg and it

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appears that similar amounts of somatotropin are in the porcine pituitary (Etherton et al., 1986).

Recombinant porcine somatotropin has been produced in microorganisms by a number of the industrial firms, many of which are associated with the large multinational pharmaceutical companies. There are subtle differences between natural and recombinant somatotropin, some of which are associated with the formation of the two disulfide bonds after synthesis of the polypeptide and an additional methionine attached to the N-terminus. As a result, recombinant somatotropin is routinely compared to natural somatotropin by multiple criteria (Mayne et al., 1984). Multiple forms of somatotropin have been reported and reviewed by Lewis (1984).

Growth Hormone Releasing Factor

Growth hormone releasing factor (GHRF) has been the subject of several recent reviews (Gelato and Merriam, 1986; Ling et al., 1985). In normal humans, GHRF is produced in the hypothalamus and secreted as a 44 amino acid polypeptide. Porcine GHRF differs in only three amino acid residues from human GHRF. GHRF is thought to be released from the hypothalamus into the portal vessels where it travels to the pituitary. Doses in humans as low as 0.5 µg/kg result in near maximum elevation of somatotropin within 30 to 60 min. Administration of GHRF to pigs in order to elevate somatotropin levels and eventually alter the growth rate has been examined by Etherton et al. (1986). Their results indicate that a growth response can be obtained, but the response is less than that obtained by administering PST.

Somatostatin

Somatostatin is a small peptide that has a regulatory role on secretion of growth hormone. When hypothalamine concentrations of somatostatin are elevated, somatotropin release from the pituitary is decreased. Somatostatin levels are regulated by a variety of factors including feedback mechanisms for somatotropin and somatomedin. A novel early approach to increasing growth hormone release was to remove somatostatin from the animal active immunization against somatostatin. Although some early results looked promising in sheep, recent studies with other animals are less positive.

Somatotropin Secretion in Pigs

In humans and most animals somatotropin is secreted in a pulsatile manner. This type of data requires a lot of samples to be collected under conditions which will not alter the concentration of somatotropin (avoid stress). Recently some evidence has been found for a blood somatotropin carrier protein (Baumann et al., 1987); however, the significance remains to be determined. A recent study completed in our laboratory indicates that finishing pigs have very low baseline levels of less than 2 ng/ml PST and peaks of less than 7 ng/ml. We have typically seen three to five peaks per day. An analysis from one pig sampled over 1/2 hour time periods is shown in Figure 3. When compared to other animals this is a low level of growth hormone which certainly

has a number of metabolic consequences to the animals. A list of some of the factors known to alter growth hormone secretion are listed in Table 1. The somatotropin receptors have been reviewed by Hughes et al. (1985).

Table 1. Selected Factors Affecting Growth Hormone Secretion.

Increase	Decrease
Sleep	Elevated FFA
Exercise	Somatostatin
Stress	Glucocorticoids
GHRF	Selected beta-adrenergic agonists
Estrogen	Obesity
Hypoglycemia	Old age
Selected neurotransmitters	Birth
Anorexia nervosa	
Acromegaly	

Effects of Somatotropin on Intermediate Metabolism

The effects of somatotropin on carbohydrate and lipid metabolism have recently been reviewed by Davidson (1987). Experiments administering somatotropin to somatotropin-deficient animals permit the most straight forward interpretation. Under these conditions somatotropin accelerates fat mobilization from adipose tissue to the liver. Also, fatty acid oxidation is increased in liver and muscle. From a carbohydrate standpoint, glucose utilization by muscle is improved, probably due to the increased availability of free fatty acids for oxidation. In the liver, carbohydrate metabolism is altered resulting in an increase in fasting blood glucose. Major effects are seen on protein intermediate metabolism. The major effect is an increase in protein accumulation resulting from increased amino acid transport, increased ribosomal number, increased mRNA and increased protein synthesis components. Also, the incorporation of amino acids into protein away from oxidative pathways leads to a decrease in urea formation.

Somatomedin

The actions of somatotropin on animals are derived from two separate pathways. The first has been described in the previous section and deals with the direct effects of somatotropin on tissues altering their metabolic pathways. The second route is for somatotropin to bind to the liver which then makes a small insulin-like hormone called somatomedin C (reviewed by Hall and Sara, 1983). Somatomedin C (Sm-C) is secreted from the liver and travels to the target tissues bound to a carrier protein. Somatomedin C binds to the target tissue membrane and will then elicit various responses (Fig. 4).

There are two somatomedins. Somatomedin C (or IGF-I) is a 70

amino acid peptide with a slightly basic isoelectric point (Fig. 2). The other somatomedin is called IGF-II and is a 67 amino acid polypeptide and is slightly acidic in nature. In humans and several other species, it has been shown that somatomedin-C is secreted from liver in response to growth hormone during postnatal growth. The other somatomedin (IGF-II) is often thought to be involved in prenatal growth regulation. The receptors for the somatomedins have been intensively investigated and recently reviewed by Jacobs and Cuatrecasas (1985) and Rechler and Nissley (1985). The concept that IGF-I is a postnatal growth regulator and IGF-II a prenatal growth regulator is rather simplistic and does not adequately describe the complexities of growth regulation. However, it is clear that in growing animals including finishing pigs, somatomedin-C plays a very important role in growth regulation.

Table 2. Effect on Somatomedin-C on Mesenchymal Cells

Growth effects	Metabolic effects
Protein accumulation increase	Insulin-like effects
RNA increase	Increased glucose metabolism
DNA increase	Increased lipogenesis

The role of Sm-C in regulation of growth has been examined using hypophysectomized animals. As expected, when somatotropin is administered to hypophysectomized animals they grow larger. When hypophysectomized rats were administered somatomedin-C, they also grew, but not as well as with somatotropin (Skottner et al., 1987). This type of analysis is used to show that many of the growth regulating properties of somatotropin eventually work through modulation of somatomedin levels.

The effects of Sm-C on tissues are varied and quite complicated. Sm-C has insulin-like properties which include increases in glucose uptake and lipogenesis in adipose tissue. Also in skeletal muscle glucose transport, glycogen synthesis, amino acid transport and protein synthesis are increased with Sm-C. However, the most important effect of Sm-C is its effect on mesenchymal cells (Table 2). Mesenchymal derived cells include those of muscle, adipose, bone and connective tissue. These tissues which are targets for manipulation in meat animals such as pigs respond to somatomedin by increasing the amount of DNA in the tissues. Eventually the new DNA increases the protein content of these tissues and we have enlarged tissues.

In swine, the somatomedins have not been adequately characterized. While it is evident that a Sm-C polypeptide is present in postnatal pigs, our results (Table 3) show that Sm-C activity is increased several fold in response to somatotropin administration to finishing swine.

Table 3. Least-squares Means of Blood Chemistry from pST Treated Finishing Pigs*

Variable	Level of PST (mg/d)				
	0	1.5	3	6	9
Blood urea nitrogen (mg/dl)	16.3	13.9	11.9	9.3	8.4
Triglyceride (mg/dl)	23	26	28	32	37
Glucose (mg/dl)	82.4	79.6	98.2	97.9	98.4
Insulin (μU/ml	15	18	22	28	35
T3 (ng/dl)	64	80	91	95	75
T4 (μg/dl)	3.0	2.7	2.5	2.0	1.5
RIA Somatomedin C (U/ml)	0.35	0.49	0.62	0.89	1.16

*From Novakofski et al. (1987).

Effects of Somatotropin on Finishing Swine

I now want to address how somatotropin injected into finishing swine alters composition and feed efficiency. There is general consensus that the administration of somatotropin at the appropriate level and length of time to swine in their finishing phase of production results in major alterations in feed efficiency, carcass fat content, organ weights, lean carcass content with smaller effects on bone growth (Grebner et al., 1987; Boyd, 1987; Steele et al., 1987; Etherton et al., 1987).

The actions of somatotropin working through somatomedins contribute to the increase in cell number associated with the larger organ weight. Also, the somatomedins will have effects on bone growth and increase total DNA content of muscles. The effect of somatotropin on adipose deposits of these animals is more complex. There is clearly less fat (up to 40% less) in the PST treated animals.

Our studies indicate the effects of somatotropin on adipose are mediated by several mechanisms. Somatotropin has a lipolytic effect and furthermore, chronic administration increases sensitivity to other lipolytic stimuli. Other somatotropin effects such as elevated thyroid hormones probably impact directly on the enhanced lipolytic sensitivity to augment repartitioning.

The increase in efficiency of PST treated animals can be partially explained by the change in animal composition. The decrease in fat content of PST treated animals combined with the large water content of the increased lean tissue mass result in the animal having a lower caloric content.

LITERATURE CITED

Baumann, G., Auburn, K. D., and Buchanan, T. A. (1987) The effect of circulating growth hormone-binding protein on metabolic clearance,

distribution, and deregulation of human growth hormone. J. Clin. Endocrinol. Metab. 64:657.

Boyd, R. D. (1987) Somatotropin and productive efficiency in swine. Animal Health & Nutrition. Feb. 1987, p. 23.

Daughaday, W. H. (1985) Prolactin and growth hormone in health and disease. In. Contemporary Endocrinology, Vol. 2. Ed. S. H. Ingbar. Plenum Publishing Co., New York, NY.

Davidson, M. B. (1987) Effect of growth hormone on carbohydrate and lipid metabolism. Endocrine Reviews 8:115.

Etherton, T. D., Wiggins, J. P., Evock, C. M., Chung, C. S., Rebhun, J., Walton, P. E., and Steele, N. C. (1987) Stimulation of pig growth performance by porcine growth hormone: Determination of the dose-response relationship. J. Anim. Sci. 64:433.

Etherton, T. D., Wiggins, J. P., Chung, C. S., Evock, C. M., Rubhun, J. F., and Walton, P. E. (1986) Stimulation of pig growth performance by porcine growth hormone and growth hormone releasing factor. J. Anim. Sci. 63:1389.

Gelato, M. C. and Merriam, G. R. (1986) Growth hormone releasing hormone. Ann. Rev. Physiology 48:569.

Grebner, G. L., McKeith, F. K., Novakofski, J., Easter, R. A., McLaren, D. G., Brenner, K., Bechtel, P. J., Jones, R. W. and Dalrymple, R. H. (1987) Carcass characteristics of pigs injected with different levels of natural porcine somatotropin from 57 to 103 kg weight. J. Anim. Sci. 65:245. #130.

Grossbeck, M. D., Parlow, A. F. and Daughaday, W. H. (1987) Stimulation of supranormal growth in prepubertal, adult plateaued, and hypophysectomized female rats by large doses of rat growth hormone: physiological effects and adverse consequences. Endocrinology 120:1963.

Hall, K. and Sara, V. R. (1983) Growth and somatomedins. Vitamins and Hormones 40:175.

Hughes, J. P., Elsholtz, H. P., and Friesen, H. G. (1985) Growth hormone and prolactin receptors. In. Polypeptide Hormone Receptors. Ed. B. I. Posner. Marcel Dekker, Inc., NY.

Jacobs, S. and Cuatrecasas, P. (1985) The mobile receptor hypothesis and polypeptide hormone action. In. Polypeptide Hormone Receptors. Ed. B. I. Posner. Marcel Dekker, Inc., NY.

Laron, Z. (1983) Deficiencies of growth hormone and somatomedins in man. Special Topics in Endocrinology and Metabolism 5:149.

Lewis, U. J. (1984) Variants of growth hormone and prolactin and their posttranslational modification. Ann. Rev. Physiol. 46:33.

Ling, N., Zeytin, F., Böhlen, P., Esch, F., Brazeau, P., Wehrenberg, W. B., Baird, A., and Guillimin, R. (1985) Growth hormone releasing factors. Ann. Rev. Biochem 54:403.

Malarkey, W. B. (1980) The pituitary and hypothalamus. In. Textbook of Endocrinology. Ed. E. L. Mazzaferri. Elsevier Science Publishing Co., Inc., New York, NY.

Mayne, N. G., Hsiung, H. M., Baxter, J. D., and Belagaje, R. M. (1984) Direct expression of human growth hormone in Escherichia Coli with the lipoprotein promoter. Recombinant DNA products. Ed. A. P. Bollon. CRC Press.

Novakofski, J., Brenner, K.-V., McKeith, F. K., McLaren, D. G., Easter, R. A., Grebner, G. L., Bechtel, P. J., Dalrymple, R. H., and Jones, R. W. (1987) Performance enhancement by natural porcine

somatotropin (PST): Hematology and endocrinology. J. Anim. Sci. 65:246. #113.

Rechler, M. M. and Nissley, S. P. (1985) The nature and regulation of receptors for insulin-like growth factors. Ann. Rev. Physiol. 47:425.

Skottner, A., Clark, R. G., Robinson, I. C., and Fryklund, L. (1987) Recombinant human insulin-like growth factor: testing the somatomedin hypothesis in hypophysectomized rats. J. Endocr. 112:123.

Steele, N. C., Campbell, R. G., and Caperna, T. J. (1987) Update on porcine growth hormone research: practical and biological implications. October 27, 1987, Cornell Nutrition Conference.

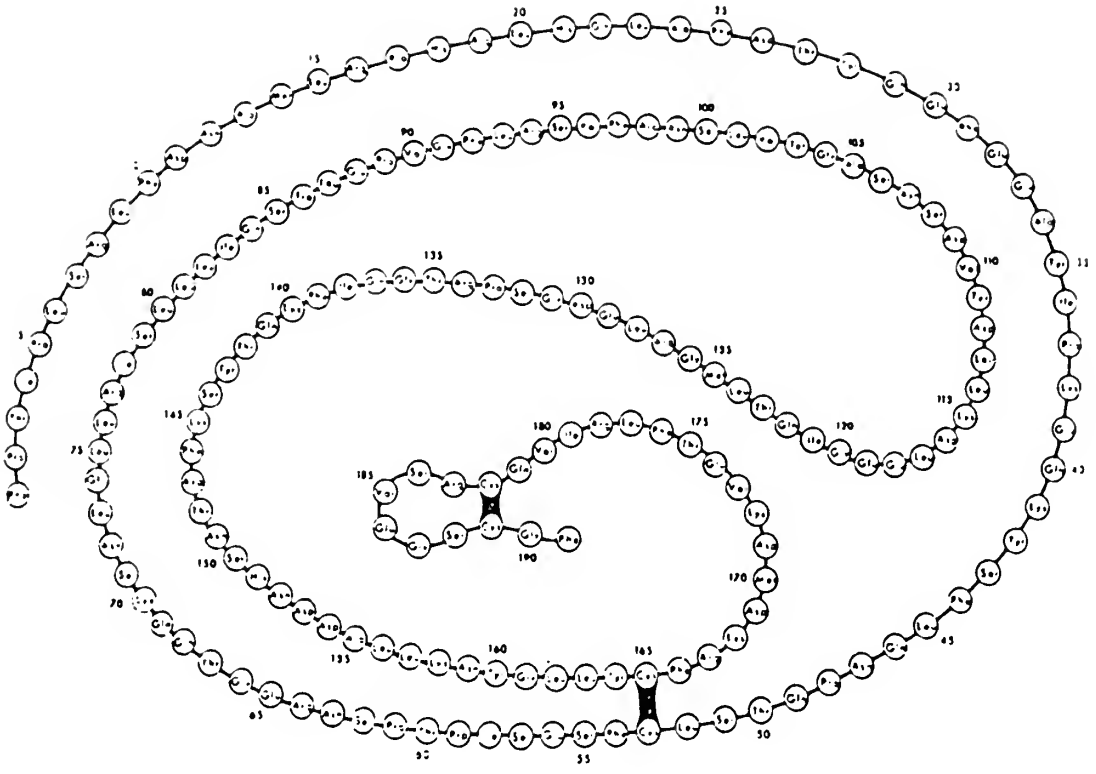

Figure 1. Structure of human somatotropin from Harper's Review of Biochemistry.

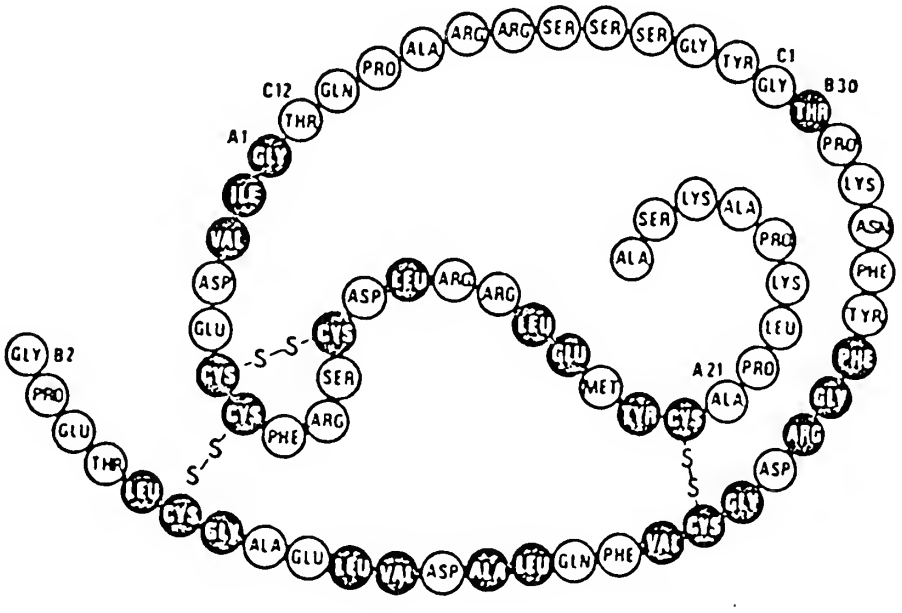

Figure 2. Structure of human somatomedin-C from Rinderknecht and Humbel.

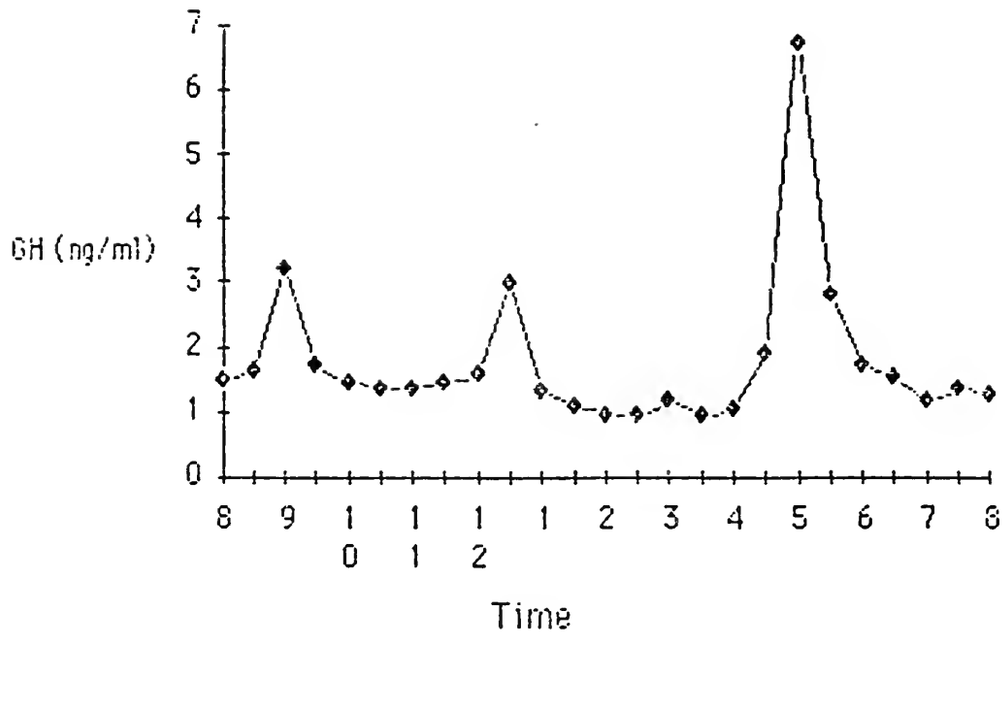

Figure 3. Somatotropin secretion pattern of a single pig over a 12 hour time period (from K. Brenner, University of Illinois).

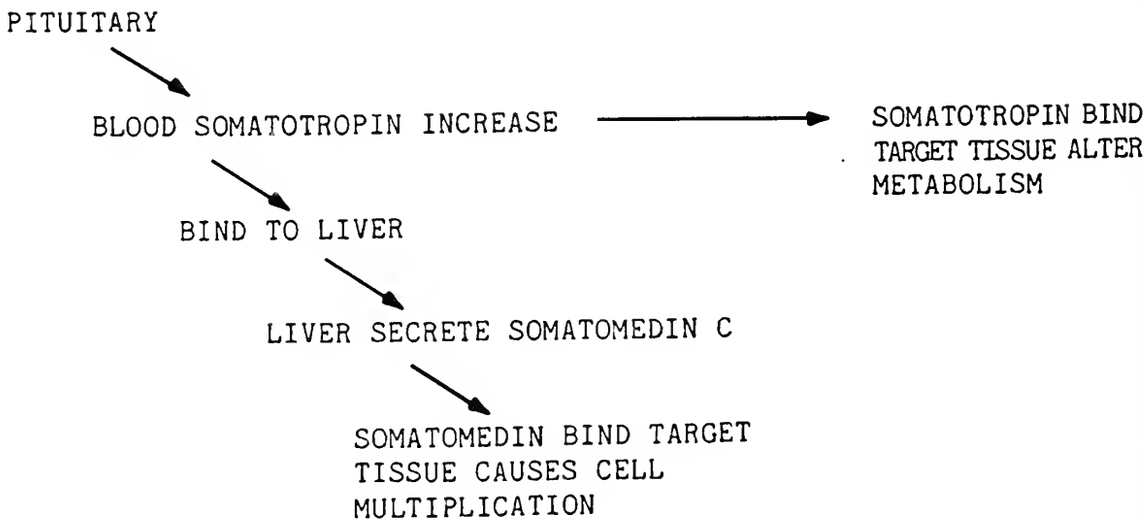

Figure 4. Diagram of somatotropin mechanism of action.

The Orderly Integration of New Technology into a Changing Swine Industry

Orville Sweet

As you have heard this morning from the animal scientists, both government and university research, the dramatic advances being made with somatotropins to improve the quality and nutritional values of meat animals has reached the point where all in America can benefit. The task of feeding America has historically been the province of farmers and ranchers, but now they have a powerful new ally in the scientific advancements we are discussing today. I see the swine industry's being a major beneficiary; and the 120,000 pork producers who are members of our Association are now in a most favored strategic position to significantly upgrade and improve their product.

Over the years, one major inhibiting factor in the broader acceptance of pork as a healthy, nutritious and attractive component of the American diet, has been the consumers' perception, clung to for many years, that pork, while tasty and appealing, has too much fat associated with the product.

This overhanging concern has been addressed by pork producers in the form of major investments we have made in past years and currently, to produce leaner hogs with the proportion of fat significantly declining. The payback from our long-standing research efforts has been a continuous program to improve our pork product for the consumers' benefit. No less than manufacturers of automobiles, home applicances, or electronic equipment, it has long been the strategy and commitment of the National Pork Producers Council to commit seed money to research and product development so that we can keep abreast of the consumers' changing preferences in meat and address the realities of what constitutes a healthy diet. We have worked closely with the medical community and nutritionist to bring pork into the family of foods which bear the blessing and the endorsement of physicians and institutions concerned with diet and health.

I am happy to say that pork has maintained its level of consumption at 55 to 65 lbs. per person for 60 years. This is in spite of diet-health

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Orville K. Sweet is the Executive Vice President of the National Pork Producers Council in Des Moines, Iowa.

issues and the dramatic decrease in the consumption of other red meats. Based on the latest research in using somatotropins, we now can look ahead to an era where pork can compete more directly. We foresee pork's winning over a larger share of the market from consumers who have found chicken and fish to be more appealing, based on the dietary disciplines physicians and nutritionists have urged them to accept. Our position has long been that pork is healthful and as nutritionally beneficial as any meat or fish which is a staple part of the American diet.

We believe it is in the American family's best interests that healthful diets be followed which combine variety, taste appeal, freshness and nutritional advantages on a balanced basis to meet the latest advice and counsel from medical research.

The new technology utilizing somatotropins can make it possible for pork to advance to a new level of wide-spread popularity in the American diet with the potential to be the preferred meat of the 21st century.

This research, fueled by the scientific advances now at hand, makes it possible for us to achieve further economics gains which are of far-reaching significance not only to agriculture but to the whole U.S. economy.

One dimension to the economic problem we have to content with is evident in a recent story that appeared in an Irish newspaper several months ago -- under the headline:

IRELAND INCREASES PORK EXPORTS TO THE UNITED STATES

> "A new 140 million pound expansion plan for the pig industry, involving the creation of 1,500 jobs, was today announced by the Minister of Agriculture, Mr. Michael O'Kennedy." And then, "A major breakthrough for the industry of pig meat processing facilities have been the awarding of licenses to export to the lucrative U.S. market."

These new threats of growing imports from the highly subsidized European Common Market countries further worsen the already threatening ratio of eight pounds of imported pork coming in to the U.S. compared to a single pound of U.S. pork being exported to world markets. These overseas imports are hurting the economic well-being of U.S. pork producers. Why is the U.S. such a "lucrative market" foroverseas producers who cannot compete with the U.S. on a level playing field of pure economic costs of production?

Recent research has shown unequivocably that no other country can produce pork as economically as do our American pork producers. If we are the low-cost producers, then why can we not compete on an international trade basis? If the Japanese can produce quality cars and sell them in the U.S. at lower prices than American-made vehicles, why shouldn't the American pork producers, as an industry with the least cost advantage, be able to sell their product in international markets at a favorable advantage based on the efficiencies of their production process?

As we all know, there are multiple factors influencing our potential to export, but only a few can be influenced by pork producers themselves. Among the economic factors with which we must contend are the changing value of the dollar, the inability of the U.S. government to negotiate fair trade practices, and the highly subsidized foreign-produced pork. Most of these are economic considerations which are beyond the control of our domestic pork producers. Concentrating on those factors over which our producers have some measure of control leads us to the quality and uniformity of our product.

Let's look at Denmark. Danish producers are responsible for about five percent of the world's pork, but they dominate 35 percent of the world's export market for pork. Denmark exports more than 70 percent of its production, while the United States exports only 1-1/2 percent of our home-produced pork!

Denmark is successful in its export program because of its highly motivated trade orientation. But another reason for their success in the world marketplace for pork products is their stringent emphasis on quality control. There is an interesting parallelism between the quality products of Japanese auto manufacturers and the quality of Danish pork products. Both are successful because the strategic planning in the management of the Japanese auto industry and in the management of the Danish pork industry have correctly stressed the economic gains to be achieved from quality control. The Danish have strict limitations for carcass weight and fat content. In Denmark live hogs and carcasses must fit within predetermined standards or heavy penalties are assessed against the producers. This managerial discipline from the top achieves lean, high yielding, uniform carcasses for the export market. In return, the producers are rewarded with premium prices.

PORK'S ECONOMIC DISPARITY

Now's let's look at the other side of the ledger and examine data from the USDA which shows that in 1986 American pork producers received 30 cents per hundredweight difference between No. 1 and No. 3 hogs. Packers, on the other hand, received $8 per hundredweight premium for their No. 1 carcasses!

Let me ask you a question: If packers can sort the No. 1 carcasses from the No. 3's, then why shouldn't it be feasible for producers to sort live hogs in the same manner, with the same incentive?

Our objective in the NPPC is to encourage our producers and the economic system under which we operate, to reward American hog producers for developing and producing a superior product with significantly improved quality, an improved lean-to-fat ratio, couple with uniformity of market hogs.

Any study of the long-range future of the pork industry shows that the two greatest challenges we face in achieving favorable economic goals for the future, involve improved quality control and enhanced market technology. How do we produce better hogs and then sell them in a competitive marketplace for what they are really worth based on the true value inherent in a superior product?

MOST EXCITING PERIOD IN HISTORY OF ANIMAL HUSBANDRY

In my judgement, we are living in the most exciting period in the history of animal husbandry. Since the day three decades ago when a controlled environment was introduced to the pork industry, the dynamics of our industry have been changing and the rate of change has been accelerating at a swift pace. Now for the first time in the history of pork production, we have found a means of certifying pork as being trichina-safe. With this guarantee safely in hand, our next objective will be to eradicate trichinosis in hogs forever in the United States.

Another major accomplishment is that for the first time in history, several very positive nutritional attributes of pork have been identified and proven which set it apart from other red meats. We have a product advantage.

We also have made great strides toward better feed efficiency resulting in still more economical production techniques. Again, like the auto manufacturers, we are presistently studying new ways to achieve low-cost economies in our "manufacturing" process, all the while we are concentrating on improving product quality.

Over the years, we have learned how to more effectively control various diseases afflicting hogs. Now, through pilot programs, we believe we can eradicate pseudorabies. All of these and still other successes have been the result of efforts in the field of animal science brought about by the application of new technologies achieved by the scientists and researchers who work in animal husbandry. In ever so many ways, these men and women who work in laboratories and research centers in the government, in universities, and in the private sector, are authentic heroes. We should acknowledge our debt to the many diverse benefits that flow from their laboratories to produce meat products which nourish the nation and keep us healthy and well-fed.

BIOTECHNOLOGY'S NEWEST ACCOMPLISHMENT

Porcine growth hormone, manufactured by recombinant DNA technology, is expected to reach the market in 1988. The advantages of its use will include leaner meat that maintains flavor and tenderness, while production costs are lowered. When the fat/body weight ratio in hogs is decreased, the conversion of grain into usable protein is more efficient. Our mission at NPPC is to provide farmers and the public with the best information possible so that the acceptance of porcine growth hormone, or porcine somatotropins (PST) is accomplished in an environment of accurate public understanding.

Biotechnology in its simplest form means the use of living organisms to make commercial products. Applying these principles in industry means that research will provide extraordinary potential benefits for the nation. We can expect products and processes contributing to a cleaner environment, improved diagnosis and treatment of disease, more vigorous food crops, and alternatives to petroleum as a source of energy. Biotechnology by itself is not new. Biotech principles have given us wine, beer, buttermilk, and made it possible for oil to be processed into gasoline.

The process by which living cells rearrange chemical elements to form new products is scientific progress from which we all have benefitted.

At a recent professional meeting of animal health care suppliers, it was stated: "The changes that somatotropins will bring on the pork industry will be more dramatic than those resulting in the introduction of antibiotics." And antibiotics have been a critical building block in the modern technology of meat production which has enabled us to advance to a high level of industry efficiency. So, we face an exciting future and porcine somatotropins are a major scientific accomplishment which promises measureable and profitable returns for producer, consumer, and the nation.

The documented proof of improved feed efficiency and lean-to-fat ratios by the use of PST are astounding. Their economic implications have far outstripped our most optimistic expectations. In research conducted at Pennsylvania State University, animals had improved feed efficiency of up to 25 percent, increased growth rate 19 percent and achieved a 70 percent reduction in carcass fat which a concurrent increase in muscle. The somatotropin revolution was begun with an $8,000 research grant of check-off funds by the National Pork Producers Council in the early 70's. We are proud of our working partnership with the scientific community which has achieved this progress.

FACING THE FUTURE

In the light of these impressive gains for the pork industry and the prospects for yet more impressive future benefits, there still are difficult questions to be answered. We are moving forward cautiously and are working to build in safeguards and assurances that will demonstrate to the public that we are conscientious in our obligation to test, re-test and endorse this new biotechnology process for the pork industry based on a consensus from all concerned that its adoption will be universally advantageous.

Here are some of the questions which must be answered:

1. Can this giant stride of progress be made genetically without the use of outside substances?

2. If so, can the industry wait for the slow time lapse required for natural genetic change?

3. How soon will the product be available?

4. What kind of delivery system will be workable?

5. What will it cost?

6. Will the public accept this perceived hormonal or artificial genetic change?

7. Will the biotechnical end procedure be acceptable to producers?

8. Will it replace the current seedstock producers?

9. Will it come on so rapidly as to shock the industry and put the smaller producers out of business?

10. Whose responsibility is to to educate producers?

11. Will it unfairly favor larger producers?

12. Is it moving ahead too quickly for the public to assimilate its advances into their existing educational, religious, and social belief systems.

Karen Rogers, a psychologist from Monsanto who is that company's Biotechnology Education Director, has posed that last question at a recent USDA Forum on the Biotechnology Challenge.

Indeed, the real world experience of Monsanto has demonstrated that despite having fostered a broad-based campaign to educate and prepare the public on biotechnology and the nature of change, using opinion research findings of studies done through Northern Illinois University and the Opinion Research Corporation, when Monsanto sought to field test a genetically engineered microorganism, they met failure. A skeptical press ignited vigorous local community opposition to the test and because of the gap between technological literacy and technological advance, Monsanto's astute management was thwarted from its test program. Psychologist Rogers warned the USDA Conference on Biotechnology in February of this year that "we are faced with a level of scientific illiteracy in this country that is truly frightening."

COOPERATION AND MUTUAL UNDERSTANDING NEEDED

Our approach to implementing the introduction of new products to the swine industry is to foster cooperation and mutual understanding in the entire pork industry. We believe that the answers to most of the questions I have cited can be resolved in business-like procedures of problem-solving. The real issue is one that lies in the public policy arena and is intertwined with public opinion acceptance of the impact of scientific change on industry - and the consuming public.

The national Pork Producers Council plans to develop pragmatic ways to deal with the impact of the shock that these substances and these new techniques might have on the pork industry. Independent researchers have provided sound data on the efficacy of the use of somatotropins and still more studies are underway. The powerful influence of competition for lower production costs may pressure farmers to accept this new treatment program.

Yet, we are alert and sensitive to one possible scenario wherein consumers might be mislead by emotion and biased distortions of scientific fact, so that they react negatively to PST-treated pork. This could lead to confusion in the consumer marketplace for pork so that <u>all</u> pork products -- treated and untreated -- are disparaged and pork sales in stores suffer a serious decline. If this unfortunate circumstance did occur, then farmers will be far more directly affected than any single food processor or any of the pharmaceutical companies.

Clearly, it is evident that there is an immediate need to design and conduct a broad-scale public educational and informational program to prepare the industry and the public to understand the true scientific basis by which porcine somatotropins work and the corollary evidence that demonstrates no adverse effects on human health. Only by this strategy of building consumer understanding leading to consumer confidence can we be successful as an industry in implementing the application of this major new scientific advancement for the pork industry that constitutes the inevitable but steady process of progress.

Farmers by themselves cannot fund or execute such a massive public educational effort. The National Pork Producers Council has as its mission the rendering of assistance to farmers to understand and decide on the merits of this new technology. Because of the complexity and the far-reaching ramifications of the application of biotechnology to the pork industry, this national educational program presents a major challenge to us all -- scientists, producers, pharmaceutical companies, foods processors, food distributors, as well as consumers.

It is our view, and in fact the policy of the NPPC, that our organization will provide leadership, the energy, talent and resources at our command to foster this all-industry cooperative educational and informational program -- but it must join forces with others to make it a true team undertaking. All who function in the pork industry must have a role to play -- and must help in the funding as well as the strategy formulation and the execution of the program. It truly must be a democratic undertaking.

Polypeptide growth hormones are new applications brought about by new technologies that can manufacture biological compounds identical to the natural ones. Porcine somatotropin is one of many biological compounds becoming available. Recombinant human somatrotropin is already being used to treat children with human growth hormone deficiencies.

But, the general public appears to lack the scientific understanding to differentiate a polypeptide growth hormone from a steroid hormone or an antibiotic, together with the implications and effect each has on health issues. Among all the biologically produced compounds under present development, only porcine somatotropin requires an urgent response because of the immediacy of its commercial availability and the immense magnitude of the population affected.

Farmers traditionally have made economic decisions on a cost basis that did not involve the risk of public acceptance -- or rejection -- of their product. Pork producers today know that antibiotic agents and antibiotics fed to animals to increase food production are under public scrutiny for their possible adverse effects on human health.

Farmers want impartial and totally objective government agencies to do their research to keep food safe and to control the industry in the public good. Public participation and discussion in open forums that affect policy-making and risk management before porcine growth hormone is finally approved will ensure that safe guidelines are accepted and consumers are intelligently and well informed. The educational/informational approach we suggest be taken for PST can establish a valuable precedent for other biological processes yet to come. The program we have decided

to use, educates people by interpreting scientific achievements and enables them to accept or reject a new technology based on its merits. It is designed to prevent selfish profit notices and hype or slick Madison Avenue mass marketing persuasion from overriding truthful health safety considerations which are in the best public and national interest.

We are NPPC can foresee the possibility that narrow vision activists could create an emotional issue that might adversely affect the orderly and well-balanced introduction of PST to the pork industry and thus injure the position of pork as an important component of the American menu plan. Our conviction is that the use of PST is one that should be discussed in open forum on the basis of proven scientific studies on health effects rather than permitting special interest groups to ignite opposition by duping an uninformed public.

ADOPTING A TASK FORCE APPROACH

Our approach is to form a representative Task Force which will be given the mandate to encourage open forum discussion based on the scientific evidence at hand. By this means, we intend to prevent the mobilization of emotional sentiment that can freeze public opinion in a posture of opposition to sound technological progress.

Recent consumer research has shown that randomly sampled consumers were supportive by a two-thirds margin of biotechnology and felt it would have a positive effect on an improved standard of living. This research also showed that the better educated and informed people were, the more likely they were to be positive about biotechnology's benefits. Other results of this consumer research study showed that among the positively-oriented respondents, their feelings were that "Science brings advance, not problems." Seventy-four percent of the respondents agreed with the creation of new animal life through genetic engineering, while 23 percent disagreed. It is relevant also to note that farmers' attitudes toward the use of bovine somatotropins (BST) in another opinion research study among dairy farmers showed that 38 percent said they would try BST on their herds, 28 percent said they may try it, and 23 percent refused to test it, and 11 percent didn't know what they might do.

It is noteworthy also to observe that the hard-core opposition tended to be older, economically depressed, and/or politically dissatisfied farmers or dairymen. The introduction of hybrid seed corn and environmentally controlled hog buildings were met with the same type of opposition. Fear of the unknown is the most dreadful. It is far less risky to talk openly about potential risk, than to risk suprising the public. We cannot risk the possibility of giving the activists a catastrophe to act upon. We have learned by bitter experience that when issues are overblown or exaggerated by the media, science does not do well. It is our view that we must deal honestly, directly, and openly with the whole subject of biotechnology in the pork industry. We must lay out all the facts. If there are risks we must talk about them truthfully and candidly.

In the final analysis, the democratic approach of full disclosure and open, two-way dialogue we believe will work in the best interest of the public, the industry, science and the nation.

To adhere to that principle, the National Prok Producers Council will soon issue a major position paper on biotechnology and the pork industry. It will state our policy and position quite clearly. Then, we will move forward within that policy to make the democratic process work for the benefit of all.

Management and Environment Considerations for Successful Use of Repartitioning Agents

Stanley E. Curtis

The new era in pork production that would be brought on by The Repartitioning Revolution would have potential challenges as well as potential benefits. There apparently would be advantages to being able to fashion pig growth so as to make pork products more acceptable to consumers and the pork business more profitable for producers and processors. However, in the production phase, although we do not yet know much about the effects and side-effects of these new technologies, what we do know already has provoked some questions regarding how swine management regimens might need to be changed in order for these new agents and procedures to be implemented in the swine industry. We would be wise to anticipate possible problems in integrating these technologies into existing systems of pork production. It would be best if we had adequate, scientifically sound answers at hand if and when the repartitioning era dawns instead of having to generate those answers in either a catch-up or a repair mode.

Moreover, nowadays, the complex systems of animal production, and each of their component technologies, must pass not only the test of economic feasibility but the test of societal acceptance, as well. Some in United States society already have wondered about the ethical dimensions of genetic engineering of agricultural animals and even of administering natural compounds exogenously at above-normal rates. Many of these concerns are based on suspicions that these techniques abuse animals by causing them to experience abnormal (here read "unnatural and therefore unethical") existences. Of course, abnormal does not necessarily mean either undesirable or unethical. But we will be better equipped to discuss these issues once we know something about how these new technologies affect certain facets of the animals' lives besides those production-related traits on which we have focused heretofore.

Two important aspects of the pig's life that might be affected by transgenic manipulation, beta adrenergic agonists, and porcine somatotropin are thermoregulation and certain behavioral patterns.

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THERMOREGULATION

The thermal status of a pig's body depends essentially on two factors: thermal insulation (which determines heat-loss rate) and heat-production rate. The repartitioning agents probably affect both of these factors. Therefore, the thermal-environmental and dietary-energy requirements of swine might be affected by these agents.

Thermal Insulation

The three thermal insulators between a pig's body core (the source of most metabolic heat) and the surroundings (the animal's heat sink) are (from inside out): tissue insulation, hair insulation, and boundary insulation (the very still layer of air that envelopes the body). Total insulation equals the sum of the three. The latter two layers of insulation are probably independent of any repartitioning agent, but the tissue insulation is not.

The contribution of hair insulation in a growing-finishing hog in a typical production setting averages around .2 C° m^2 hr/kcal, and that of boundary insulation, around .1; the two combined total around .3 C° m^2 hr/kcal.

Tissue insulation is due mainly to subcutaneous fat, which--when vasoconstricted--yields a maximal insulation value of around .1 C° m^2 hr/kcal per cm of thickness. Therefore, tissue insulation depends on the thickness of the subcutaneous fat, which may be determined in turn partly by a repartitioning agent. When the subcutaneous fat covering the entire body averages 1 cm thick, then maximal tissue insulation would be around .1 and maximal total insulation, around .4 C° m^2 hr/kcal. But when subcutaneous fat thickness is reduced by half to .5 cm, maximal total insulation would drop to around .35 C° m^2 hr/kcal.

A hog weighing 75 kg would have a body surface area of around 1.25 m^2. Therefore, it can be calculated, the slope of its environmental heat demand line would be around 3.1 kcal/hr per Celsius degree if it had 1 cm of subcutaneous fat and around 3.6 if it had .5 cm. In other words, for any given decrease in environmental temperature, heat-loss rate from the hog with less subcutaneous fat would increase some 16% faster than that from the fatter hog. This would make the leaner hog more vulnerable to cold environments, but less vulnerable to hot ones, provided all other factors remained equal. In particular, the somatotropin-treated hog's lower critical temperature (the effective environmental temperature below which the animal must raise metabolic rate above the thermoneutral level in order to maintain a normal body temperature) might be as much as 12 C° higher than that of an untreated hog. The treated hog's upper critical temperature also would be higher in the face of less insulation, but probably only a few degrees higher.

Alas, when some repartitioning agents are being used, critical factors probably do not remain equal. To gain overall understanding, heat-production rate must be taken into consideration.

Heat-Production Rate

The metabolic stimulation due to exogenous somatotropin increases the animal's heat-production rate. Present evidence indicates that this increase is associated mostly and perhaps entirely with increased productive rate in the lactating cow but at least partly with increased maintenance energy expenditure in growing pigs. One recent estimate is that maintenance energy expenditure is 24% higher in somatotropin-treated hogs; this would be reflected by a commensurate increase in heat-production rate. Regardless of how the extra heat production may be partitioned, it would affect the animal's thermal-environmental requirements; with respect to the extra metabolic heat, the somatotropin-treated animal would be less vulnerable to cold environments, but more vulnerable to hot ones, provided all other factors remained equal. In particular, under such conditions, the lower critical temperature of the somatotropin-treated 75-kg hog might be reduced by around 6 C°, and the upper critical also would drop by several degrees. However, as has been pointed out, under exogenous somatotropin administration, critical other factors probably do not remain equal.

The Bottom Line

How would the combined effects of exogenous porcine somatotropin on (a) subcutaneous fat thickness (and therefore tissue insulation value) and (b) heat-production rate affect a hog's thermal-environmental requirements? A hog's lower critical temperature is determined by the junction between the thermoneutral heat-production rate and the environmental heat demand line. The combined effects of these two consequences of somatotropin treatment would be, on the cool end of the scale, a 12 C° increase in lower critical temperature due to less thermal insulation offset partly by a 6 C° decrease due to higher heat-production rate; the net effect would be a 6 C° increase in the lower critical temperature of a 75-kg hog due to somatotropin treatment. In other words, the treated hog would be considerably more sensitive to cool or frankly cold environments.

As for the upper critical temperature (the point above which the hog starts to pant or to increase evaporative heat-loss rate by some behavioral means such as wallowing in mud), on balance it might be expected that this critical point would drop by a few degrees. That is, the effect of the somatotropin-induced increase in heat-production rate would be expected to override that of the decreased thermal insulation, resulting overall in the hog's being more vulnerable to high temperatures, also. Although it is not possible to be as precise quantitatively in estimating the effect of exogenous somatotropin on the hog at the warm end of the temperature scale, it is likely that in practical production settings over much of the United States the heightened sensitivity to warm or frankly hot surroundings would be of more economic consequence; high-temperature-induced reduction in appetite probably would be a greater problem in many operations.

HOG BEHAVIOR

Casual observation of the behavior of hogs being fed a beta adrenergic agonist have led to the conclusion that the treated hogs may be "more active" than normal: more alert, more excitable, and up more often and for longer periods. This tentative conclusion should be confirmed by measurements of the treated hogs' behavior patterns in a carefully controlled experiment. If the results of this research confirm the conclusion based on casual observation, then the hogs' environmental requirements would warrant investigation in several respects.

- Hogs use floor space for a variety of purposes. That space not being used for lying, eating, drinking, or dunging may be considered free space to be used for general motor activities, including playing and moving from one place to another within the pen. The socio-behavioral implications of this space and how it is used are potentially important. For example, if indeed treated hogs are up more often and longer each day, then on average the total free space in a given pen would increase, but the animal density in the free space would also increase. This would especially be so if the animals were more sensitive to a cool environment (as was suggested earlier) and thus chose to huddle more closely than normal. In any case, depending on the locations of critical resources such as the feeder and the waterer, this increase in animal density in the free space might become a limiting factor with respect to accesses to feed and water, especially for hogs in the lower ranks of the group's social dominance order.

- If hogs being treated with a repartitioning agent are indeed "more active", the ways they use various resources may be altered so as to change the animals' needs for optimal productivity. For example, if the hogs are up more often and longer each day, they may well have more eating bouts and spend more total time at the feeder. Also, if they are up more their chances of becoming embroiled in an agonistic encounter with a penmate would be increased--especially if they are in a more agitated state to begin with, and hence their use of the feeder as a place to hide their heads from agressors may increase markedly. As a consequence of either of these possibilities, the amount of feeder space provided in the pen might need to be increased above the normal. This possibility, too, would need to be investigated by means of a carefully designed, executed, and interpreted experiment.

REFERENCES

Bauman, D. E. 1987. Bovine somatotropin in lactation: impact on management and feeding. Proc. 1987 Cornell Nutrition Conference for Feed Manufacturers. Page 23. Cornell University, Ithaca, NY.

Curtis, S. E. 1983. Environmental Management in Animal Agriculture. Iowa State University Press, Ames, IA.

Curtis, S. E. 1985. Physiological responses and adaptations of swine: A--hot environments and B--cold environments. In: M. K. Yousek (ed.) Stress Physiology in Livestock, Vol. II--Ungulates. Pages 59 and 129. CRC Press, Inc., Boca Raton, FL.

Curtis, S. E. 1987. Animal well-being and animal care. In: E. O. Price (ed.) Farm Animal Behavior/Food Animal Practice/Veterinary Clinics of North America. Page 369. W. B. Saunders Co., Philadelphia, PA.

Steele, N. C., R. G. Campbell, and T. J. Caperna. 1987. Update of porcine growth hormone research: practical and biological implications. Proc. 1987 Cornell Nutrition Conference for Feed Manufacturers. Page 15. Cornell University, Ithaca, NY.

Changes in Carcass Characteristics and Implications for the Pork Processing Industry

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SUMMARY

Consumer demand has changed for meat and meat products in the past decade. The consumer aversion to dietary fat will likely continue and potentially become more pronounced. Improved breeding, feeding and management practices have done a great deal to enhance consumer acceptability of pork; however, these changes may not be rapid enough to keep up with changes in consumer demand.

Mechanisms which allow producers to produce lean, fast growing animals efficiently have been a goal of the animal industry for decades. The use of repartitioning agents in the swine industry may allow us to make progress toward this goal. The use of β adrenergic agonists and somatotropin have been shown to increase lean content and reduce fat content in pork carcasses while improving the rate and efficiency of growth.

The use of repartitioning agents will affect all aspects of livestock production and the meat industry. Dramatic reductions in fat content in pork carcass may affect the quality of bacon, increase the availability of low fat meat cuts, change fabrication and handling procedures, sausage formulations and alter the palatability of pork and pork products.

INTRODUCTION

The quantity and quality of lean tissue are major factors in determining the value of meat animals. A major objective of animal research involves increasing the quantity of lean produced in meat animals. Recently, the demand for lean meat has increased and this has stimulated the need to reduce fat and increase lean. The development and use of repartitioning agents has been evaluated as a mechanism of achieving this goal in the livestock industries.

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The effect of beta adrenergic agonists on carcass characteristics has been evaluated by numerous researchers (Jones et al., 1985; University of Illinois, 1986; Moser et al., 1986; Crenshaw et al., 1987; Prince et al., 1987; Hancock et al., 1987; Veenhuizen et al., 1987a; Veenhuizen et al., 1987b). These studies indicate that muscling increases and fat content decreases when pigs are fed this compound for approximately 7 weeks prior to slaughter.

The use of somatotropin as a repartitioning agent in swine has also been evaluated by numerous studies (Chung et al., 1985; Baile et al., 1983; Machlin, 1972; Boyd, 1987; Smith et al., 1987; Campbell et al., 1987; University of Illinois, 1987a; University of Illinois, 1987b). These studies involved pigs of various weights and ages and a wide range of levels of administration. The results of these studies are variable; however, very dramatic decreases in fat and increases in lean were observed in some studies.

Altering the composition of pork may have variable effects on the meat processing industry. Dramatic reductions in fat may require major changes in the way we fabricate pork carcasses. Existing fabrication techniques may not be well suited for very lean pigs. The processing techniques used for bacon may need to be altered since the thickness of the belly will be changed as well as lean to fat ratio.

Overall, the use of repartitioning agents will require changes to be adopted at both the producer level and packer level.

Application of Repartitioning Agents to the Swine Industry

A summary of the various methodologies employed in research involving repartitioning agents are presented in table 1. Repartitioning agents that will be discussed in the following tables include somatotropin and beta adrenergic agonists.

Somatotropin. Studies involving pork carcass composition changes in response to somatotropin (Chung et al., 1985; Baile et al., 1983; Machlin, 1972; Boyd, 1987; Etherton et al., 1987; Etherton et al., 1986; Bryan et al., 1987; Smith et al., 1987; Steele et al., 1987; University of Illinois, 1987a, 1987b) have evaluated at least three variables: 1) dosage levels; 2) various starting and ending weight ranges of pigs; and 3) various lengths of time of administration. The bulk of the studies reported utilized natural porcine somatotropin, but some studies have utilized recombinant somatotropin products. Few differences were observed when natural and recombinant somatotropin were evaluated in one study (University of Illinois, 1987); however, Smith et al. (1987) reported a variable response when recombinant bovine somatotropin was administered to swine.

The response of swine to somatotropin is quite variable for most traits evaluated. The variable response may be expected due to the wide range of weights, dose levels and/or the number of administration days employed in these studies. The starting weight of pigs treated with somatotropin ranged from 20 to 46 kg and off-test weights ranged from 55 to 100 kg. Dosage levels evaluated include a range from 0 to 1.1 mg/kg/day and

length of administration ranged from 30 to 70 days. Most of the studies evaluated barrows and/or gilts and few sex differences were observed. Steele et al. (1987) reported that somatotropin had a variable response relative to the growth and performance of pigs due to sex status. Boars did not respond to somatotropin treatment to the same extent that barrows and gilts did. Ultimately the use of somatotropin appeared to negate differences due to sex status.

Beta adrenergic agonists. Beta adrenergic agonists appear to have similar responses for most traits. The dosage level appears to be dependent on the specific compound being administered. Cimaterol (Jones et al., 1985; University of Illinois, 1986; Moser et al., 1986) was utilized at lower levels (less than 1 ppm) than phenethanolamine-Ractopamine (Crenshaw et al., 1987; Prince et al., 1987; Hancock et al., 1987; Veenhuizen et al., 1987a, b).

Muscle Characteristics

A summary of the response of pigs to repartitioning agents is described in table 2. All differences are reported as absolute values of the data reported.

Somatotropin. Somatotropin increased loin eye area in 7 of 10 comparisons; the increases were as high as 5 cm^2 (18% increase). Other indications of muscle that increased included percent lean (.5% increase), carcass soft tissue protein (up to 4% increase in protein), protein accretion rate (87/g/d higher) and muscle weights (up to 131 g increase or an 11.7% increase).

Variation in the response of muscle growth to somatotropin may change with the weight of pigs at slaughter (Chung et al., 1985; Bryan et al., 1987), administration dose (Chung et al., 1987; Bryan et al., 1987), type of somatotropin (Baile et al., 1983) number of administration days (Chung et al., 1985; Baile et al, 1987). Variable response may be associated with a host of factors including environment, genetics, nutrition. From the information available, a larger effect on muscle accretion may be achieved when animals are treated from 50-80 kg live weights and when levels of administration are near 3 mg/day. Studies treating animals from 50 to 100 kg in weight with the same relative dose achieve 10-15% increases in loin eye area. Typical studies inject animals until slaughter and little information is available regarding the effect of long term withdrawal from the compound. The point on the growth curve where optimal protein accretion is achieved has not been systematically evaluated.

Beta adrenergic agonists. Beta agonists increased loin eye area in all 8 comparisons with the maximum effect being 6.1 cm^2 (18.8% increase). Other factors evaluated included muscle weights (up to 157 g increases) and predicted carcass lean (up to 5.3% increase in predicted carcass lean). The effect of beta adrenergic agonists was consistent and the type of compound appeared to affect the magnitude of the response. Ractopamine consistently increased loin eye area (Crenshaw et al., 1987; Hancock et al., 1987; Veenhuizen et al., 1987); however, variable effects with cimaterol have been reported (Jones et al., 1985; University

of Illinois, 1986; Moser et al., 1986).

Lipid Characteristics

Repartitioning agents have a large effect on the lipid content of the carcass (Table 3). Backfat and/or tenth rib fat thickness decreased in 6 of 10 comparisons for somatotropin studies and 7 of 8 beta adrenergic agonists studies. Decreases in tenth rib back fat were as high as 1.59 cm for somatotropin treated animals and .45 cm for beta adrenergic agonists treated animals. Total carcass lipid was decreased as much as 12.7% (from 29.9% to 17.2%) with somatotropin and 5.1% (from 29.2% to 24.1%) with beta adrenergic agonists.

Somatotropin. Somatotropin had the largest effect in fat content/-backfat thickness with heavier pigs. This is conceptually what would be expected since pigs are depositing proportionately more fat at heavier weights. Somatotropin reduced total carcass fat from 29.9% to 17.2% (University of Illinois, 1987a). Several studies indicate that limited changes in lipid content are observed with the use of somatotropin may be associated with the use early in growth and development and/or low levels of administration (Etherton et al., 1986; Etherton et al., 1987).

Beta Adrenergic Agonists. Beta adrenergic agonists effectively reduce fat deposition but the magnitude of the reduction is lower than would be expected for somatotropin. Fat depots including leaf fat (14% decrease) and tenth rib fat (16.5% decrease) are reduced. Jones et al. (1985) reported some interesting results involving the withdrawal of cimaterol. A 7-day withdrawal of the compound stimulated a compensatory fat deposition. The fat measurements of the control animals approximately equalled the cimaterol treated animals after a 7 day withdrawal from the compound.

Dressing Percentage

Dressing percentage of the animals treated with repartitioning agents are presented in table 4. For market weight animals, somatotropin reduces dressing percentage up to 3.4% and beta adrenergic agonists consistently increases dressing percentage 1 to 1.5%. Although the data is limited, visceral components (liver, heart, kidneys) increase in weight dramatically with the use of somatotropin and the relative increase in visceral weight, decrease in fat content and increase in muscle tissue result in a net decrease in dressing percentage.

The increase in dressing percentage in pigs treated with beta adrenergic agonists may be expected because small differences are observed in visceral weights. The increase in muscle weight compensates for reductions in fat and a small increase in visceral weights resulting in an increase in dressing percentage.

The reduction in carcass dressing percentage observed with the use of somatotropin will be of concern to the slaughter industry; however, if productivity is evaluated on the basis of tons of lean or dollar value per man hour, the effect of somatotropin on dressing percentage will not be a stumbling block.

The effect of repartitioning agents on skeletal tissues appears to be variable. Jones et al. (1985) reported that the weight of the femur decreased slightly with the administration of beta adrenergic agonists and University of Illinois (1987a) data suggest that femur weights were increased with the use of somatotropin.

Carcass Cutting Yield

Carcass cutting yield and wholesale cut composition from our somatotropin studies are presented in Table 5. To our knowledge, no data has been published on carcass cutting yields in pigs treated with beta adrenergic agonists.

The weight of untrimmed wholesale cuts did not vary between control and somatotropin treated carcasses; however, when cuts were trimmed to .6 cm fat thickness, wholesale cuts were heavier for the somatotropin treatments except the picnic shoulder. Similar results were observed when boneless wholesale cuts were evaluated. Although the magnitude of the differences were small when the composition of the cuts was evaluated, these differences were accentuated. Fat content was reduced from 12.5 to 9.0; 27.2 to 14.1; 210 to 10 and 15.0 to 9.3%, respectively, for the boneless trimmed ham, loin, boston butt and picnic shoulder. Data indicate that the weight of boneless cuts are slightly increased but substantial reductions in fat content has occurred.

Industry Perspective

The meat industry will undoubtedly become involved in evaluating the use of repartitioning agents for meat production. The use of somatotropin may cause some concerns due to the dramatic changes in carcass composition. Concerns of the meat industry will include processing characteristics, cutting/ handling differences and belly thickness. The meat industry will also have concerns about merchandising, shelf life and consumer appeal of very lean pork.

Processing characteristics that need to be further evaluated include water holding capacity, salt-soluble protein extraction, myoglobin content and handling ease. A concern that potentially may develop is the acceptance of low-fat processed pork products. Often, low-fat products have altered texture, decreased juiciness and flavor characteristics.

As fat content is reduced, cutting and handling procedures are also altered. Fat is removed from the loin using a loin knife and it is more difficult to perform this operation on very lean pigs. In addition, the carcasses will be much softer or pliable during processing due to reduced fat, and therefore, processing temperature, equipment and procedures may need to be altered. Another potential concern regarding fat reductions will involve the suitability of the belly for bacon production. The belly is traditionally 50% or more fat; however, reducing the fat in the carcass will reduce its thickness and may require different or alternative processing procedures.

The meat industry will certainly find a host of advantages when they

evaluate the effects of repartitioning agents on the pork carcass. The increase in total lean and reduced trimming will be of great economic importance. Hopefully, existing technological advances will alleviate or minimize any potential problems that arise if the meat industry is to process leaner pigs.

The use of beta adrenergic agonists will be viewed positively by the meat processing industry. It will decrease fat, increase muscle and improve dressing percentage. Few, if any, changes in processing will be required in the transition to pigs treated with beta adrenergic agonists.

LITERATURE CITED

Anderson, D. B., Veenhuizen, E. L., Waitt, W. P., Paxton, R. E. and Mowrey, D. H. 1987. "Effect of Ractopamine on nitrogen retention, growth performance and carcass composition of finisher pigs." Journal of Animal Science. 65(Suppl. 1): 130 (Abstr.).

Baile, C. A., Della-Fera, M. A. and McLaughlin, C. L. 1983. "Performance and carcass quality of swine injected daily with bacterially-synthesized human growth hormone." Growth. 47:225-236.

Boyd, R. D. 1987. "Somatotropin and productive efficiency in swine." Animal Health & Nutrition. February. pp. 23-26.

Bryan, K. A., Carbaugh, D. E., Clark, A. M., Hagen, D. R. and Hammond, J. M. 1987. "Effect of porcine growth hormone (pGH) on growth and carcass composition of gilts." Journal of Animal Science. 65(Suppl. 1):244 (Abstr.).

Chung, C. S., Etherton, T. D. and Wiggins, J. P. 1985. "Stimulation of swine growth by porcine growth hormone." Journal of Animal Science. 60:118-130.

Crenshaw, J. D., Swantek, P. M., Marchello, M. J., Harrold, R. L., Zimprich, R. C. and Olson, R. D. 1987. "Effects of a phenethanolamine (Ractopamine) on swine carcass composition." Journal of Animal Science. 65(Suppl.1):308 (Abstr.).

Etherton, T. D., Wiggins, J. P., Evock, C. M., Chung, C. S., Rebhun, J. F., Walton, P. E. and Steele, N. C. 1987. "Stimulation of pig growth performance by porcine growth hormone: determination of the dose-response relationship." Journal of Animal Science. 64:433-443.

Etherton, T. D., Wiggins, J. P., Chung, C. S., Evock, C. M., Rebhun, J. F. and Walton, P. E. 1986. "Stimulation of pig growth performance by porcine growth hormone and growth hormone-releasing factor." Journal of Animal Science. 63:1389-1399.

Hancock, J. D., Peo, E. R., Jr. and Lewis, A. J. 1987. "Effects of dietary levels of Ractopamine (a phenethanolamine) on performance and carcass merit of finishing pigs." Journal of Animal Science. 65(Suppl. 1):309 (Abstr.).

Jones, R. W., Easter, R. A., McKeith, F. K., Dalrymple, R. H., Maddock, H. M. and Bechtel, P. J. 1985. "Effect of the β-adrenergic agonist cimaterol (Cl 263,780) on the growth and carcass characteristics of finishing swine." Journal of Animal Science. 61:905-913.

Machlin, L. J. 1972. "Effect of porcine growth hormone on growth and carcass composition of the pig." Journal of Animal Science. 35:794-800.

Moser, R. L., Dalrymple, R. H., Cornelius, S. G., Pettigrew, J. E. and Allen, C. E. 1986. "Effect of cimaterol (Cl 263,780) as a repartitioning agent in the diet for finishing pigs." Journal of Animal Science. 62:21-26.

Prince, T. J., Huffman, D. L. and Brown, P. M. 1987. "Effects of Ractopamine on growth and carcass composition of finishing swine." Journal of Animal Science. 65(Suppl. 1):309 (Abstr.).

Smith, V. G., Moseley, W. M., Kasson, C. W. and Kratzer, D. D. 1987. "Effect of exogenous recombinant bovine somatotropin (rbst) and porcine pituitary somatotropin (ppst) on growth performance of pigs." Journal of Animal Science. 65(Suppl. 1):243 (Abstr.).

Steele, N. C., Campbell, R. G. and Caperna, T. J. 1987. "Update of porcine growth hormone research: practical and biological implications." Cornell Nutrition Conference, October 27, 1987.

University of Illinois. 1986. Unpublished data.

University of Illinois. 1987a. Unpublished data.

University of Illinois. 1987b. Unpublished data.

Veenhuizen, E. L., Schmiegel, K. K., Waitt, W. P. and Anderson, D. B. 1987. "Lipolytic, growth, feed efficiency, and carcass responses to phenethanolamines in swine." Journal of Animal Science. 65(Suppl. 1):130 (Abstr.).

Table 1. Summary of Studies Utilizing Repartitioning Agents

Somatotropin		
Chung et al., 1985	Age/weight	32-60 kg
	Level of administration	.22 µg/kg/d
	Length of administration	30 days
Baile et al., 1983	Age/weight	23-90 kg
	Level of administration	0-.06 mg/kg/d
	Length of administration	70 days
Machlin, 1972	Age/weight	46-95 kg
	Level of administration	0-1.1 mg/d
	Length of administration	50-60 days
Boyd, 1987	Age/weight	45-100 kg
	Level of administration	0-200 µg/kg/d
	Length of administration	50 days
Etherton et al., 1986	Age/weight	49-80 kg
	Level of administration	30 µg/kg/d
	Length of administration	30 days
Etherton et al., 1987	Age/weight	40-75 kg
	Level of administration	0-70 µg/kg/d
	Length of administration	
Bryan et al., 1987	Age/weight	72 kg
	Level of administration	70 µg/kg/d
	Length of administration	30 days
Smith et al., 1987	Age/weight	40-80 kg
	Level of administration	0-250 µg/kg/d
	Length of administration	42 days
Steele et al., 1987	Age/weight	61 kg
	Level of administration	100 µg/kg/d
	Length of administration	31 days
Univ. of Illinois, 1987a	Age/weight	53-103 kg
	Level of administration	0-9 mg/day
	Length of administration	52-60 days
Univ. of Illinois, 1987b	Age/weight	64-99 kg
	Level of administration	0-3 mg/day
	Length of administration	39-70 days

Beta Adrenergic Agonists

Jones et al., 1985	Age/weight	65-105 kg
	Level of administration	0-1 ppm
	Length of administration	7 weeks
Univ. of Illinois, 1986	Age/weight	62-100 kg
	Level of administration	0-.5 ppm
	Length of administration	7 weeks
Moser et al., 1986	Age/weight	65-105 kg
	Level of administration	0-1 ppm
	Length of administration	7 weeks
Crenshaw et al., 1987	Age/weight	65-110 kg
	Level of administration	0-30 ppm
	Length of administration	50 days
Prince et al., 1987	Age/weight	65-100 kg
	Level of administration	0-30 ppm
	Length of administration	45-50 days
Hancock et al., 1987	Age/weight	65-?
	Level of administration	0-30 ppm
	Length of administration	--
Veenhuizen et al., 1987	Age/weight	?-104 kg
	Level of administration	20 ppm
	Length of administration	--
Anderson et al., 1987	Age/weight	61 kg
	Level of administration	0-20 ppm
	Length of administration	--

Table 2. Effect of Repartitioning Agents on the Muscle of Pork Carcasses[a]

Somatotropin		
Chung et al., 1985	Carcass weight	42-44 kg
	Loin eye area	NS
	Percent Lean	1.3 kg increase
Baile et al., 1983	Carcass weight	NS
	Loin eye area	NS
	Percent lean	NS
Machlin, 1972	Carcass weight	--
	Loin eye area	up to 4.2 cm^2 increase
	Ham protein (%)	1.5% increase
Boyd, 1987	Carcass weight	---
	Loin eye area	up to 4.1 cm^2 increase
	Semimembranosus wt.	up to 124 g increase
	Semitendinosus wt.	up to 60 g increase
Etherton et al., 1986	Carcass wt.	52.5 - 56.9 kg
	Loin eye area	4.4 cm^2 increase
	Soft tissue protein	1.2% increase
Etherton et al., 1987	Carcass wt.	---
	Loin eye area	up to 5 cm^2 increase
	Soft tissue protein	up to 1.9% increase
Bryan et al., 1987	Loin eye area	NS
Smith et al., 1987	Carcass wt.	43 - 83 kg
	Loin eye area	up to 4 cm^2 increase
Steele et al., 1987	Protein accretion (g/d)	increased 87 g/d
Univ. of Illinois, 1987a	Loin eye area	up to 3.81 cm^2 increase
	Biceps femoris wt.	up to 131 g increase
	Semitendinosus wt.	up to 68.1 g increase
	Carcass protein	up to 4% increase
Univ. of Illinois, 1987b	Loin eye area	up to 3.74 cm^2 increase
	Semitendinosus wt.	up to 49.1 g increase

Beta Adrenergic Agonists

Jones et al., 1985	Carcass wt.	NS
	Loin eye area	up to 2.24 cm^2 increase
	Biceps femoris wt.	up to 157 g increase
	Semitendinosus wt.	up to 57 g increase
Univ. of Illinois, 1986	Carcass wt.	3 kg increase
	Loin eye area	up to 3.13 cm^2 increase
	Biceps femoris wt.	up to 100 g increase
	Semitendinosus wt.	up to 37 g increase
Moser et al., 1986	Carcass wt.	NS
	Loin eye area	up to 3.2 cm^2 increase
	Predicted kg of muscle	up to 1.5 kg increase
Crenshaw et al., 1987	Loin eye area	up to 3.55 cm^2 increase
	Carcass lean percent	up to 5.3% increase
Hancock et al., 1987	Loin eye area	up to 6.1 cm^2 increase
	Carcass lean percent	up to 3.5% increase
Prince et al., 1987	Loin eye area	up to 4.8 cm^2 increase
	Carcass lean percent	up to 4.4% increase
Veenhuizen et al., 1987	Loin eye area	up to 5.2 cm^2 increase
	Predicted fat free lean	up to 4.2% increase
Anderson et al., 1987	Loin eye area	up to 5.2 cm^2 increase
	Predicted fat free lean	up to 3.2% increase

[a]All differences reported are absolute values (treated - control = difference) and percentage response was not calculated.

NS No significant difference between treated and control animals.

Table 3. Effect of Repartitioning Agents on the Lipid Characteristics of Pork Carcasses[a]

Somatotropin		
Chung et al., 1985	Tenth rib fat	NS
	Carcass fat	NS
Baile et al., 1983	Average backfat thickness	NS
	Specific gravity	NS
Machlin, 1972	Back fat thickness	up to .69 cm decrease
	Ham analysis (% fat)	up to 7.4% decrease
	Specific gravity of ham	up to .016 increase
Boyd, 1987	Average back fat thickness	up to .91 cm decrease
	Carcass lipid	up to 12.2 kg decrease
Etherton et al., 1986	Back fat thickness	NS
	Soft tissue fat (%)	up to 5.3% decrease
Etherton et al., 1987	Back fat thickness	NS
	Soft tissue fat (%)	up to 7.1% decrease
Bryan et al., 1987	Back fat thickness	up to 51% decrease
Smith et al., 1987	Back fat thickness	up to 1.1 cm decrease
Steele et al., 1987	Fat accretion (g/d)	up to 239 g/d decrease
Univ. of Illinois, 1987a	Leaf fat	up to 779 g decrease
	Tenth rib fat	up to 1.59 cm decrease
	Body wall thickness	up to 1.54 cm decrease
	Carcass lipid (%)	up to 12.28% decrease
Univ. of Illinois, 1987b	Leaf fat	up to 574.2 g decrease
	Tenth rib fat	up to .92 cm decrease

Beta Adrenergic Agonists

Jones et al., 1985	Leaf fat wt.	up to a 179 g decrease
	Tenth rib fat (cm)	up to a .45 cm decrease
	Semitendinosus fat (%)	up to a 1.04% decrease
Univ. of Illinois, 1986	Leaf fat wt.	NS
	Tenth rib fat (cm)	NS
	Semitendinosus fat (%)	up to a 61% decrease
Moser et al., 1986	Leaf fat wt.	NS
	Tenth rib fat	up to a .49 cm decrease
	Ham and loin fat (% carcass)	up to 7% decrease
Crenshaw et al., 1987	Tenth rib fat	up to .43 cm decrease
	Carcass fat (%)	up to 5.1% decrease
Hancock et al., 1987	Tenth rib fat	up to .3 cm decrease
Prince et al., 1987	Leaf fat wt.	up to 270 g decrease
	Carcass fat	up to 3.2% decrease
	Tenth rib fat	.35 cm decrease
Veenhuizen et al., 1987	Tenth rib fat	up to a .29 cm decrease
Anderson et al., 1987	Tenth rib fat	up to a .35 cm decrease

[a]All differences reported are absolute values (treated - control = difference) and percentage response was not calculated.

NS No significant difference between treated and control animals.

Table 4. The Effect of Repartitioning Agents on Dressing Percentage[a]

Somatotropin	
Chung et al., 1985	NS
Baile et al., 1983	≈ 1% decrease
Machlin, 1972	up to 3.4% decrease
Beta Agonists	
Jones et al., 1985	up to 1.5% increase
Moser et al., 1986	NS
Hancock et al., 1987	up to .9% increase
Veenhuizen et al., 1987	up to 1.5% increase
Anderson et al., 1987	up to a 1.1% increase

[a]All differences reported are absolute values (treated - control = difference) and percentage response was not calculated.

NS No significant difference between treated and control animals.

Table 5. Effect of Somatotropin on the Carcass Yield and Composition of Major Wholesale Cuts[a]

		Control	Somatotropin
Side wt. (kg)		34.56	33.79
Wholesale cuts (kg)	Ham	9.24	9.36
	Loin	8.49	8.28
	Belly	5.39	5.03
	Shoulder	7.36	7.22
Trimmed wholesale cuts (kg)	Ham	8.21	8.51
	Loin	6.75	7.07
	Picnic shoulder	3.85	3.84
	Boston butt	2.58	2.75
Boneless trimmed wholesale cuts	Ham (kg)	6.16	6.57
	Water (%)	68.34	71.67
	Lipid (%)	12.48	8.96
	Loin (kg)	4.50	4.99
	Water (%)	56.74	66.95
	Lipid (%)	27.18	14.05
	Boston butt (kg)	2.43	2.55
	Water (%)	62.70	70.92
	Lipid (%)	20.99	10.00
	Picnic Shoulder (kg)	2.94	2.89
	Water (%)	67.35	71.67
	Lipid (%)	15.00	9.34

[a]University of Illinois, 1987b.

Repartitioned Pork: Sensory Quality and Consumer Acceptance

Jan Novakofski

INTRODUCTION

Consumer acceptance is that somewhat elusive quality determining commercial success of a product. For the livestock industry, consumer acceptance of meat products depends on the perceived quality of meat cuts in the retail case and on the subsequent palatability of the cooked meat item. Additionally, acceptance depends on the perception of safety. For "repartitioning agents" to be successfully marketed, the meat from "repartitioned animals" must have an appealing appearance and taste good.

THE BASIS OF MEAT QUALITY

Retail Case Appeal

It is important to keep in mind that visual appearance of meat products affects only the perception of meat quality. Consumers don't eat or taste meat color, but the appearance of meat is mentally associated with characteristics of palatability such as flavor. For example, Savel et al. (1987) found that more closely trimmed meat "tasted better" to consumers. This mental association may be strong, but it is not necessarily fixed. It is determined by many factors: culture, background, experience and, of course, advertising, to name a few. The question of what determines retail case acceptability is a question of attitudes and perceptions.

The most important visual characteristic determining consumer attitudes about pork is leanness, specifically the quantity of subcutaneous and/or seam fat. Amount of fat in pork loin chops was a primary criteria for selection by 60% of consumers (Diamant et al., 1976). Unfortunately, excessive fat content is also the most commonly perceived "problem or

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weak point" of pork. This problem was cited by over one-third of consumers surveyed in a 1984 NPPC consumer tracking survey. In the1987 Consumer Climate for Meat study by Burke Marketing Research, fully two-thirds of individuals surveyed said they trimmed fat off the meat cuts they purchased, indicating a desire for reduced subcutaneous fat. Interestingly, this survey points out the contradictions frequently existing in consumer attitudes. Only 3% of respondents felt pork had too many calories, although 10 times as many felt it was too fat. Clearly, many consumers have concerns and perceptions not based on common sense or factual knowledge.

The effects of repartitioning agents in carcass fatness have been well described by Dr. McKeith (this volume) and need not be recounted here. Therefore, all else being equal, there is little doubt the decreased fatness of pork from repartitioned pigs will not only be acceptable to consumers, but that it will be sought after as well. This is not to say that similar results could not be obtained by adequate trimming of fat from conventional pigs. However, the number of consumers still further trimming the meat they buy indicates it has not been done.

The color and visual texture of meat are also generally considered to be important factors in perceived meat quality. These characteristics have been reported less important than fatness as consumer selection criteria in general (Diamant et al., 1976). However, meat at the extremes, such as pale, soft and exudative (PSE) pork, is clearly inferior in appearance regardless of its leanness.

The Question of PSE Pork

There is concern that repartitioning might result in PSE pork. The basis for this concern stems from the high incidence of porcine stress syndrome (PSS) among the very muscular, lean pigs of the 1960's and 1970's and the resulting PSE pork from these pigs. Specific concern with potential PSE problems in repartitioned pigs can be broken into two categories. First, will repartitioning by either somatotropin or adrenergic agonists cause PSS either directly or indirectly. Secondly, will repartitioning agents exaggerate the stress response in borderline PSS pigs present in the commercial swine population.

PSS pigs may be generally identified by a hyperthermic response to halothane (Malignant Hyperthermia or MH, a term synonymous with PSS). Affected animals are characterized by trembling, rapid breathing and elevated temperature. After slaughter, post-mortem glycolysis proceeds rapidly and PSS pigs enter rigor very early relative to normal pigs. The consequent combination of high muscle temperature (>30°C) and low pH (<6.0) causes atypical denaturation of proteins resulting in a pale muscle color and decreased water binding or exudation (Scopes, 1964).

PSS is a genetic lesion controlled primarily by a single locus (Carden et al., 1983) and associated with specific blood types (Rasmussen and Christian, 1976). The PSS lesion is believed similar to that of MH in humans. In MH humans, administration of halothane anesthetic results in fatal hyperthermia resulting from runaway metabolic futile cycling. Similarly, in PSS pigs, poor meat quality is a direct result of uncontrolled

muscle metabolism. The specific defect, although not clearly established, resides in the homeostatic control of muscle metabolism.

In normal muscle (white or red), lactate production is limited by ATP usage since the total adenine nucleotide content is limited. Therefore, excessive muscle pH drop in PSS pigs depends on ATP breakdown which is not properly regulated. This unregulated ATP breakdown results in hyperthermia and rapid breathing in live animals since muscle metabolism in these PSS pigs is initially aerobic (Hall et al., 1976). The PSS condition is normally apparent after a stressful stimulus. Like the MH human, which can't recover from the anesthetic effects, PSS pigs can't normally recover from a stress response.

One question then is whether exogenously increased muscle mass and decreased fatness would necessarily decrease an animals ability to normally resolve the physiological response to a stress stimulus. Despite frequent, but unsubstantiated, exclamations to the contrary, this is not necessarily so. If it were, athletes would suffer at unprecedented rates. Furthermore, since PSE is the result of abnormal metabolic regulation repartitioning agents would have to cause a regulatory defect in normal pigs to result in widespread PSE problems. Increases in muscle fiber size or changes in fiber type are not dependent on abnormal regulation nor does muscle hypertrophy directly cause abnormal metabolism.

Problems with PSE pork have not been apparent in reports regarding adrenergic agonists or somatotropin (Tables 1, 2 and 3). Furthermore, color and texture scores have been very similar in treated and control animals. Research animals tend to be handled somewhat more carefully than commercially slaughtered animals. Therefore, it is possible problems with borderline PSS animals in production herds may be more noticeable in production situations.

Since PSS is a response to stress and the stress response involves elevated circulating catecholamines, there is some basis for concern about β-adrenergic agents. Research to directly evaluate the role of catecholamines in PSS has employed adrenergic antagonists. Lister et al. (1976) reported that α-blockade prevented MH in halothane sensitive pigs while β-block with propranolol or carazolol did not. Severity of the halothane effect on meat color and texture could be reduced by α-block. The effects of β-blockade on color and texture were smaller than α-block, although carazolol, which did not affect MH, did tend to reduce the incidence of PSE pork. This would suggest that β-agonist repartitioning agents might increase the occurrence of PSE pork among potentially stress susceptible animals. If this does occur it is likely there would be agent specific differences in any stress response effects. However, unless the effects are pronounced, it is unlikely they would be a practical problem because commercial preslaughter practice will adequately stress most pigs predisposed to have PSE meat.

Actual Palatability

After purchasing a chop or roast, the satisfaction or enjoyment from eating is the final basis for consumer acceptance. Tenderness is

the major palatability attribute affecting acceptability with juiciness, flavor and aroma being distant seconds. Pork is a fairly mild flavored meat and much is consumed as a cured product so consumer's expectations are for a cured flavor, not necessarily a strong pork flavor. Fresh pork is also frequently overcooked so the expectation may be for a dry meal. Unfortunately, this cookery also exacerbates any tenderness problems.

Since repartitioning agents haven't been approved, there is relatively little data on actual acceptance. However, examining characteristics indicative of meat quality gives a basis for reasonable expectations.

For purposes of discussion, the basis for tenderness can be described as having three components: 1) an actomyosin component; 2) a connective tissue component, and 3) a bulk composition and density component.

The actomyosin component consists of the sarcomere length, indicating the relative relationship of the actin and myosin where the muscle goes into rigor. Shorter sarcomere with greater filament overlap result in tougher meat. Sarcomere length tends not to vary as much in pork as in beef and is not typically related to variation in pork tenderness (DeVol et al., 1987).

The connective tissue component of tenderness is determined by the amount of collagen and its relative amount of cross-linking. Tougher meat from an older animal has more numerous and more chemically stable collagen cross-links. As with sarcomere length, connective tissue characteristics are not highly correlated with palatability attributes of pork (DeVol et al., 1987). Repartitioning agents should reduce the connective tissue component if they have any effect, because animals are younger at slaughter.

The compositional component of tenderness is probably the most important for pork. The amount of lipid and water in meat affect tenderness by several means. Water fills the volume between fibrils and hydrates proteins. Reduced water content, therefore, increases toughness by increasing the relative amount of protein in a bite. Lipid in the form of marbling provides a lubricant between fibers and prevents desiccation during cooking. It is generally felt there is a threshold effect for fat. Too little is detrimental, but levels over the threshold amount (in the area of 2.5 - 3.0%) do not have a linear beneficial effect. Reduced subcutaneous fat will have no effect since lipid migration of melted fat into the lean during cooking is minimal (Novakofski and Park, 1988).

Warner Bratzler shear values (tables 1, 2 and 3) would indicate that tenderness is not a large problem in repartitioned pork. Actual taste panel evaluation (table 4) of tenderness shows a quadratic effect which does not coincide with the curve for fat content. Furthermore, none of the tenderness scores are in the unacceptable range.

CONCLUSION

Although the data base is limited, it seems clear that repartitioned

pork will have retail appeal to consumers. Similarly, there is a reasonable basis to expect acceptable palatability in cooked products. Further information is necessary to adequately address the concerns about PSE but widespread problems would not be expected based on physiology or existing experimental data.

REFERENCES

Burke Marketing Research. 1987. Consumer Climate for Meat. Prepared for National Live Stock and Meat Board and American Meat Institute.

Carden, A. E., W. G. Hill and A. J. Webb. 1983. The inheritance of halothane susceptibility in pigs. Genet. Sel. Evol. 15:65.

Diamant, R., B. M. Watts and R. L. Cliplef. 1976. Consumer criteria for pork related to sensory, physical and descriptive attributes. Can. Inst. Food Tech. J. 9(3):151.

DeVol, D. L., F. K. McKeith, P. J. Bechtel, J. Novakofski, R. D. Shanks and T. R. Carr. Variation in composition and palatability traits and relationships between muscle characteristics and palatability in a random sample of pork carcasses. 1988. J. Food Sci. In press.

Hall, G. M., J. R. Bendall, J. N. Lucke and D. Lister. 1976. Porcine malignant hyperthermia. II. Heat production. Brit. J. Anaesth. 48:305.

Jones, R. W., R. A. Easter, F. K. McKeith, R. H. Dalrymple, H. M. Maddock and P. J. Bechtel. 1985. Effect of the β-adrenergic agonist cimaterol (CL 263,780) on the growth and carcass characteristics of finishing swine. J. Anim. Sci. 61:905.

Lister, D., G. M. Hall and J. N. Lucke. 1976. Porcine malignant hyperthermia III. Adrenergic Blockade. Brit. J. Anaesth. 48:831.

Novakofski, J. and S. Park. 1987. The effect of removing subcutaneous fat before cooking on the composition of cooked pork chops. Report for the National Live Stock and Meat Board.

Rasmusen, B. A. and L. L. Christian. 1976. H blood types in pigs as predictors of stress susceptibility. Science 191:947.

Savell, J. W., H. R. Cross, J. J. Francis, D. Wilkes, J. W. Wise, D. S. Hale and G. C. Smith. 1987. National Consumer Retail Beef Study: Interaction of leanness, price and palatability on consumer acceptance of steaks and roasts of different grades and trimness levels. Report prepared for the National Live Stock and Meat Board.

Scopes, R. K. 1964. The influence of post mortem conditions on the solubilities of muscle proteins. Biochem. J. 91:201.

Wiese Research Associates. National Pork Producers Council Consumer Tracking Study. Wave IV. 1984. Prepared for the National Pork Producers Council.

Table 1. Effect of cimaterol on pork quality characteristics.

	Treatment (ppm cimaterol)				
Characteristic	0	.25	.50	1	S.E.
Loin eye area, cm^2	29.85	31.96	33.96	33.42	0.7
Marbling[1]	2.78	3.05	2.97	2.95	0.10
Color[1]	2.63	2.83	2.82	2.78	0.11
Firmness[1]	2.70	2.80	2.87	2.95	0.10
Semitendinosus (% fat)	7.40^{c}	7.20^{bc}	6.45^{a}	6.50^{ab}	0.25
Warner-Bratzler shear[2], kg	3.88^{b}	3.57^{b}	4.13^{a}	4.39^{a}	0.18

[1]Based on a 5 point scale where 1 = white, soft or no marbling.

[2]Shear of 1/2" cones from cooked longissimus muscle (modified from Jones et al., 1985).

Table 2. Pork quality characteristics following withdrawal of cimaterol.

	Treatment (ppm cimaterol/days withdrawal)				
Characteristic	0/0	0.25/1	.25/3	.25/5	S.E.
Loin eye area, cm^2	29.9^{b}	31.1^{ab}	32.6^{a}	31.7^{a}	.5
Color[1]	2.60^{ab}	2.84^{a}	2.48^{b}	2.62^{ab}	.10
Firmness[1]	2.53^{c}	2.89^{a}	2.61^{bc}	2.88^{a}	.08
Marbling[1]	2.67	2.80	2.68	2.93	.11
Semitendinosus, % fat	7.23	6.90	6.62	7.13	.12

[1]Based on a 5 point scale where 1 = white, soft or no marbling (C. Bellaner, R. W. Jones, R. A. Easter, P. J. Bechtel and F. K. McKeith, unpublished data).

Table 3. Least-squares means[1] for pork quality characteristics of somatotropin treated pigs.

		Dosage level (mg/day)				
Trait	Regression[2]	0	1.5	3.0	6.0	9.0
Loin eye area (LEA)(cm^2)	cubic	34.40	37.64	38.74	37.81	38.21
Color[3]	linear	2.85	2.78	2.71	2.57	2.43
Firmness[3]	cubic	2.95	3.00	2.89	2.61	2.86
Marbling[e]	quadratic	3.00	2.68	2.50	2.30	2.52

[1]Points on the least-square polynomial regression line.

[2]Degree of polynomial assumed.

[3]Based on a 5 point scale where 1 = white, soft or no marbling.

Table 4. Sensory properties[1] of the longissimus muscle from pigs receiving various levels of porcine somatotropin.

	Somatotropin level (mg/day)				
Trait	0	1.5	3.0	6.0	9.0
Juiciness	7.8	7.8	7.8	7.8	7.8
Tenderness	9.0	8.6	8.4	8.5	9.2
Off-flavor intensity	14.1	13.5	13.9	13.8	14.2
Pork flavor intensity	10.5	10.5	10.5	10.4	10.4
Overall acceptability	9.2	8.6	8.5	8.9	9.0
Warner-Bratzler shear, kg[2]	3.3	3.3	3.4	3.5	3.6
Longissimus fat (%)	3.5	2.9	2.5	2.1	2.3

[1]Based on a 14 cm unstructured scale where 14 is most juicy, most tender, no off-flavor, most flavor and most acceptable.

[2]Shear of 1/2" cones from cooked muscle.

The Impact of Repartitioning Agents on Genetic Improvement Programs

David McLaren

The U.S. pork industry stands poised on the brink of a technological -- or, more accurately, a biotechnological -- revolution. As evidence for this fact, Pitman-Moore, Inc. (a subsidiary of International Minerals and Chemical Corporation) broke ground in Terre Haute, IN, earlier this year for a $50 million bioengineering plant designed for production of recombinant porcine somatotropin (PST). Recombinant PST is manufactured by inserting the pig growth hormone gene into the genetically simple *E. Coli* bacteria. The bacteria multiply very rapidly, and recombinant PST is produced along with other bacterial gene products, from which it is subsequently purified. A cheap, stunningly effective method of producing many compounds that previously had to be extracted, at great expense, from animal tissues; or were simply not available. This same technology makes the harvesting of insulin (to treat human diabetics) from pig pancreases obsolete, and has also resulted in development of the first hoof-and-mouth vaccine.

With FDA approval, and a practical method of administration, recombinant PST will have a profound effect on U.S. pork production systems. Research conducted at the University of Illinois, injecting 120 finishing pigs daily (from 57 to 103 kg) with natural PST, revealed response in growth rate of up to 15%, with treated pigs reaching market weight as much as 12 days earlier than controls (McLaren et al., 1987). This accelerated rate of growth was associated with a 22% decrease in average daily feed consumption, and a 42% improvement in gain-to-feed ratio. Decreases of 42 to 59% in carcass fat measures, and 10 to 15% increases in muscle mass, were found in the carcasses of these pigs (Grebner et al., 1987).

Considering only the effects on growth rate and feed efficiency, PST could decrease the cost of producing a 104 kg market hog by $9.25. (This assumes total feed costs of $54.79 to produce the hog (Lattz et al., 1987), that 45% of this cost is for the finishing diet, and that PST decreases feed-to-gain ratio 29%; plus savings in non-feed costs of 17.5 cents per day for 12 days.) Naturally, costs associated with purchasing and administering the PST would have to be considered, as would the need to increase amino acid concentrations in the finishing diet (Easter, 1987). Nevertheless, given the improvement in carcass composition (to which no financial advantage has been assigned), as well as effects on growth, it is likely that PST will have a significant impact upon the efficiency of lean pork production.

The major U.S. drug manufacturers researching PST (American Cyanamid Co., Pitman-Moore, Inc., Monsanto Co. and SmithKline Beckman Corp.) reportedly hope to have a product on the market by 1990 (Fleming, 1987). While this might prove to be unrealistically optimistic, commercial use of

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repartitioning agents such as PST and beta-adrenergic agonists in pork production systems appears almost certain to become a reality in the not too distant future. The use of such agents will require careful reevaluation of all aspects of pork production, from mechanical and environmental to biological and economic considerations. Use of repartitioning agents poses a number of questions regarding genetic improvement programs for swine, and it is the objective of this paper to consider the genetic implications of this new technology.

IMPLICATIONS OF USE OF REPARTITIONING AGENTS IN COMMERCIAL PORK PRODUCTION FOR GENETIC IMPROVEMENT PROGRAMS IN SWINE

If we adopt the time scale of genetic improvement (i.e., a generation interval of one to two years), the impact of agents such as PST is imminent. It is therefore critical that research geneticists identify areas in which present knowledge is inadequate or lacking, decide upon the most immediate needs for experimental evaluation, and prioritize the implementation of experiments such that expected genetic progress under the anticipated future production environment is maximized.

The likely impacts of repartitioning agents appear to be so far-reaching, however, that there are many more questions than answers, and many of those questions are of a very fundamental nature. The following attempts to itemize some concerns likely to be raised by those involved in the business of producing breeding stock for the swine industry.

1. Should seedstock producers (whom we will assume are performance testing) test animals that are treated with repartitioning agents? How important a risk is the potential genotype x environment interaction that might arise from continuing to select breeding stock in a "traditional" (untreated) environment, when their commercial offspring will be metabolically manipulated with repartitioning agents?

2. If we decide that it would be desirable to test under the same conditions of metabolic manipulation, what effects do such agents have on the sexual development of the pig? How will repartitioning agents affect age at puberty and subsequent fertility? What withdrawal periods might be necessary? This question is relevant not only to breeders of seedstock, but also to the majority of commercial producers who practice rotational crossbreeding and select female replacements from the finishing pens.

3. If it is found to be impractical to treat performance tested seedstock, what are the genetic correlations between growth performance and carcass traits with and without treatment with repartitioning agents? What effects will repartitioning agents have upon heritabilities? Knowledge of these parameters will be critical for selection index construction and breeding value estimation where market hog populations are treated with such agents.

4. Given that it is practical to administer repartitioning agents to potential breeding stock, should all seedstock be tested this way, or will limited uptake of repartitioning agents in the commercial sector require the creation of specialist lines selected for PST modified production? Again, a knowledge of the effect of these agents upon genetic parameters is essential in order to answer these questions.

5. What effects will repartitioning agents have upon bioeconomic selection objectives for maternal and paternal lines ? The relative economic importance of genetic predisposition to deposit fat and lean may become of minor importance, for instance, while traits such as appetite might become of great economic importance.

6. Do genetic differences for response to repartitioning agents exist, both within and between major U.S. swine breeds? If they do, how might these differences be effectively exploited by selection programs and crossbreeding systems?

7. What "new" traits (appetite, response to administration of repartitioning agents, biochemical serum markers) might have value in accelerating genetic progress for aggregate genotype in lines of pigs treated with repartitioning agents?

8. As the industry moves towards production of a leaner, more muscular hog, will there be associated problems with stress susceptibility and deterioration of quality (i.e., organoleptic properties) of the meat produced? Should pork quality be included in the definition of net merit used in selection index formulation?

The above in no way purports to be a comprehensive listing of genetic considerations associated with commercial use of repartitioning agents. Rather, it (hopefully) serves to stimulate thought and to instill a sense of the wide-ranging implications of such agents for genetic improvement programs in swine. Genetic testing and selection programs in general must respond to changes in production techniques as they occur. If, for instance, pigs treated with PST are marketed at heavier live weights, it will probably be necessary to test breeding stock to heavier end points -- unless, of course, genetic correlations between untreated animals tested to approximately 100 kg, and their treated progeny finished at heavier weights, are more favorable than correlations where untreated breeding stock are tested to heavier weights.

The points listed above indicate the need to be aware of changes in genetic, phenotypic and economic parameters resulting from the use of repartitioning agents. This fact can be clearly demonstrated by considering some equations fundamental to quantitative genetics. If, for instance, it is impractical to treat potential breeding stock with an agent such as PST, breeders will be selecting for various traits in purebred herds in order to genetically improve similar (but not necessarily identical as regards genetic control) traits in crossbred populations. McLaren et al. (1985), among others, have demonstrated the genetic correlation between performance in purebred and crossbred populations to be less than 1.0 for postweaning performance of pigs reared under comparable conditions of management. Commercial use (only) of repartitioning agents will undoubtedly reduce this correlation even further, as well as affecting genetic and phenotypic variances. Seedstock herds would therefore be practicing indirect selection: i.e., selecting for performance in untreated purebreds to genetically improve performance of crossbred pigs treated with repartitioning agents. For the sake of clarity, consider selection for a single trait. The ratio (R) of the correlated response in a trait X resulting from direct selection on trait Y (CR_X) to the direct response expected from selection on trait X (R_X) is given by the formula:

$$R = \frac{CR_X}{R_X} = \frac{r_A \cdot i_Y \cdot h_Y \cdot \sigma_{A_X} \cdot L_X}{i_X \cdot h_X \cdot \sigma_{A_X} \cdot L_Y} = r_A \cdot \frac{i_Y}{i_X} \cdot \frac{h_Y}{h_X} \cdot \frac{L_X}{L_Y} \quad \text{(Falconer, 1981),}$$

where: r_A = the genetic correlation between X and Y;
i = intensity of selection;
h = square root of heritability, i.e., the ratio of the additive genetic to phenotypic standard deviations;
σ_A = additive genetic standard deviation;
L = generation interval;

and subscripts X and Y refer to the trait of direct interest and the trait used for indirect selection, respectively.

If we assume that the selection scheme does not affect either the intensity of selection or the generation interval, it can be readily seen that the correlated response in trait X will only exceed the direct response where $r_A h_Y > h_X$. Thus use of "novel" traits, such as selecting for decreased serum levels of lipogenic enzymes to decrease carcass fat (shown by Muller (1986) to be comparably effective to selecting for decreased probed backfat thickness in the pig), will only be successful at improving easily measured, non sex-limited traits where a high positive genetic correlation exists between the "novel" and "conventional" trait, and the heritability of the "novel" trait is considerably greater than that of the trait of interest. Conversely, selecting for performance in untreated purebreds to improve performance of crossbreds treated with repartitioning agents is likely to result in a decreased rate of genetic progress. Direct selection where purebreds cannot be treated would involve progeny testing, and a resultant increase in the generation interval and decrease in selection intensity. The relative merits of these alternative selection schemes can only be evaluated if the relevant genetic and phenotypic parameters, or estimates thereof, are known.

Selection in swine, of course, involves multiple traits, and various selection indexes are recommended for use in genetic improvement programs (Robison, 1981). In order to construct a selection index, it is first necessary to define the true merit, or aggregate genotype (T), of any individual (i), which can be written as:

$$T_i = \mathbf{a}'\mathbf{g}_i ,$$

where **a** is a vector of relative economic values weighting the elements of $\mathbf{g}_i$, a vector of unobservable values (typically breeding values, transmitting abilities or producing abilities) for all traits of economic importance. The selection index can be written:

$$I_i = \mathbf{b}'\mathbf{y}_i ,$$

where I_i is the index value for individual i, **b** the vector of selection index weights, and **y** the vector of phenotypic deviations for traits in the index (which may or may not be the same as the traits in **g**). The selection index weights maximize the correlation between T and I, and are calculated by solving a series of equations known as the Smith-Hazel equations, namely:

$$\mathbf{V}\,\mathbf{b} = \mathbf{C}\,\mathbf{a}$$

i.e.,

$$\mathbf{b} = \mathbf{V}^{-1}\,\mathbf{C}\,\mathbf{a} ,$$

where **V** is the variance of **y**, i.e., the phenotypic variance-covariance matrix of traits in the index; **C** the covariance between **y** and **g**, typically a genetic variance-covariance matrix; and **b** and **a** are as defined above.

It should be apparent, therefore, that knowledge of the effects of repartitioning agents on the relative economic values of traits included in the net merit function, and on the phenotypic and genetic variances and covariances among traits, will be essential in order to design meaningful swine improvement programs in the future should use of such agents becomes widespread in the pork production industry.

EXPERIMENTAL RESULTS

Virtually all of the experimental work, to date, with repartitioning agents has investigated efficacy and mode of action. It is only recently that scientists have begun to consider issues related to breeding and genetics, and consequently there is almost nothing by way of experimental data to help answer the questions posed above. Effects on genetic parameters and the importance of potential genotype x treatment interactions are unknown, although we might speculate that the metabolic effects of agents such as PST are so profound that it would be surprising if the nature of genetic and environmental variation within, and covariation between, traits was *not* changed. In reality, the data bases needed to study such effects are unlikely to become available until repartitioning agents are used extensively in commercial production.

Effects of Repartitioning Agents on Sexual Development of Swine

Probably the most pressing question to answer regarding the impact of repartitioning agents on swine improvement programs is not genetic, but concerns reproductive physiology. Namely: what effect do agents such as PST have upon puberty and subsequent fertility of treated pigs? The need to investigate the effects of repartitioning agents on sexual development of boars and gilts is clear, and as yet we can only speculate as to whether it will be possible to treat performance tested seedstock with such agents.

It is known that market weight animals that have been treated with PST have greatly reduced body fat, but it is necessary to investigate the effects of drug withdrawal on body composition and attainment of puberty in males and females. Data collected on reproductive tracts from market weight gilts, however, suggest that effects may be more complicated that a simple change in body composition. Figures 1 and 2 show a significant increase (as much as +52% and +68%) in weights of the ovaries and uteri, respectively, in treated animals. The functional effects of these morphological changes need to be investigated in order to determine whether or not it will be feasible to treat potential breeding stock with PST.

Genetic Differences For Response to Repartitioning Agents

While there are no published results for swine, two recent experiments with rodents have indicated differential responses for different genotypes treated with beta-agonists . Berne et al. (1985) reported the results of an experiment in which outbred (Sprague-Dawley and Wistar) and inbred (Osborne-Mendel and Wistar-Furth) rats were fed either a control diet or a diet containing 80 ppm clenbuterol. Clenbuterol decreased fat for all strains ($P<.05$), however the largest effect (a 48.2% decrease in combined epididymal, renal and white interscapular fat pad weights) was seen with the genetically obese Osborne-Mendel strain (J. Novakofski, personal communication). The other strains (Wistar-Furth, Sprague-Dawley and Wistar) had combined fat weight decreases of 34.7, 32.1 and 19.6%, respectively. Skeletal muscle mass (combined gastrocnemius, plantaris, soleus, psoas and extensor digitorum longus weights) was greater for treated versus control rats for all four strains, significantly so for two strains. Increases of 4.9% (Wistar-Furth), 8.5% (Osborne-Mendel), 11.1% (Sprague-Dawley, $P<.05$) and 12.9% (Wistar, $P<.05$) were recorded. Clenbuterol fed Sprague-Dawley rats exhibited increased psoas muscle mass while Wistar rats had increases in four of the five muscles weighed ($P<.05$). Differential response of genotypes to clenbuterol treatment was also found for feed efficiency. The interaction noted was consistent with that for muscle mass, with decreases in feed-to-gain ratio of 5.8% (Wistar-Furth), 12.4% (Osborne-Mendel), 17.3% (Sprague-Dawley, $P<.05$) and 17.4% (Wistar, $P<.05$). As with decreased adiposity, the greatest improvements in feed efficiency (17.3 and 17.4%) were associated with the least efficient strains (control feed-to-gain ratios of 3.46 and 3.39, respectively). Control Wistar-Furth and Osborne-Mendel rats averaged 3.13 and 3.14 g feed per g gain.

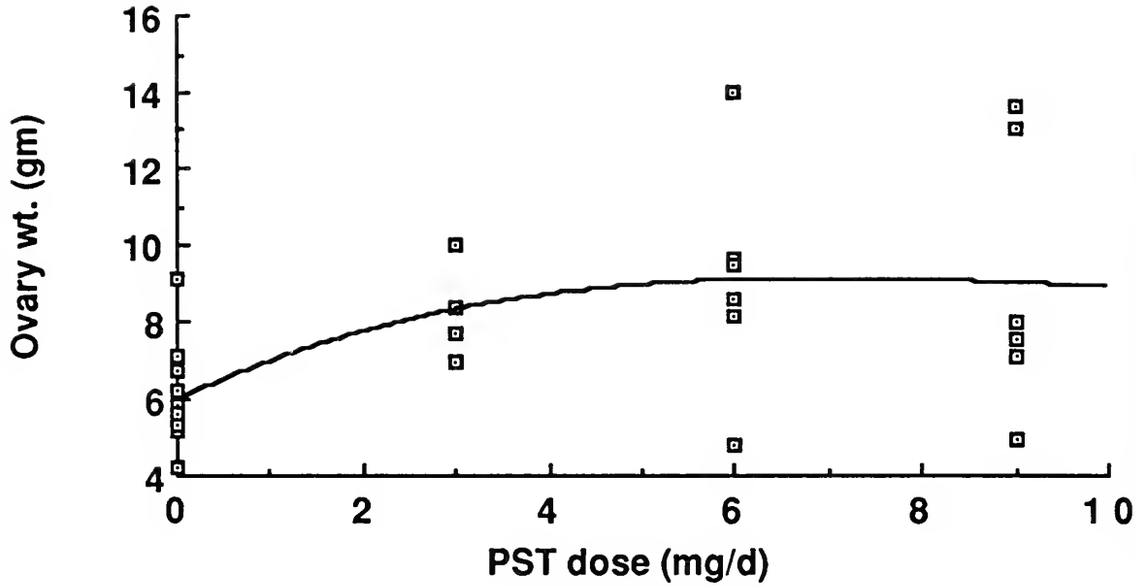

Figure 1. Ovary weights for 30 gilts treated with porcine somatotropin (PST) from 119 days of age (58 kg) until slaughter at 104 kg. Control gilt ovaries (n=12) averaged 6.01 ± .38 gm; gilts injected with 3 mg PST / day (n=6) averaged 8.24 ± .57 gm; gilts administered 6 mg PST / day (n=6) averaged 9.13 ± 1.22 gm; and gilts receiving 9 mg PST / day (n=6) averaged 9.03 ± 1.42 gm. The significant (P<.05) regression curve indicates a non-linear increase in uterus weight with increasing PST dosage (unpublished data).

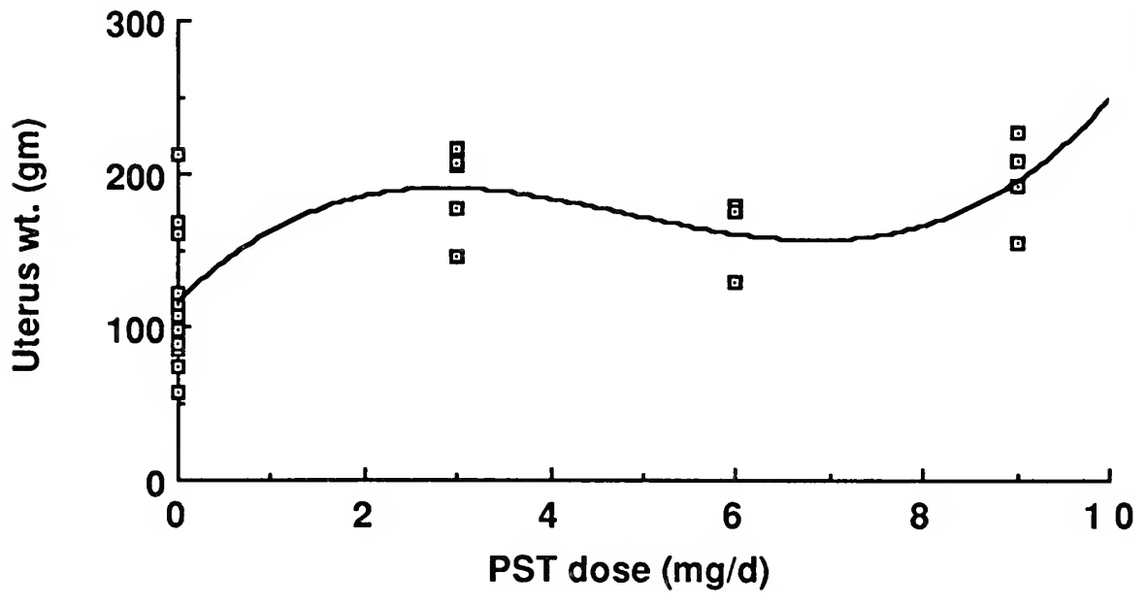

Figure 2. Uterus weights for 25 gilts treated with porcine somatotropin (PST) from 118 days of age (57 kg) until slaughter at 105 kg. Control gilt uteri (n=12) averaged 117 ±13 gm; gilts injected with 3 mg PST / day (n=5) averaged 191 ± 13 gm; gilts administered 6 mg PST / day (n=3) averaged 161 ± 16 gm; and gilts receiving 9 mg PST / day (n=5) averaged 196 ± 12 gm. The significant (P<.05) regression curve indicates a non-linear increase in uterus weight with increasing PST dosage (unpublished data).

Eisen et al. (1987) reported differential response to cimaterol in mice from a line selected for rapid three to six week (postweaning) gain versus control line animals. Male mice from the rapid gain and an unselected control line were fed a diet containing 0, 50 or 200 ppm cimaterol from four to either seven or ten weeks of age. Line x cimaterol interactions were found for growth rate, feed intake and feed efficiency ($P<.01$). In select line males, performance with 0 and 50 ppm cimaterol did not differ, and exceeded performance of mice fed 200 ppm cimaterol ($P<.05$). In the control line males, however, feed intake and growth rate were increased by addition of 50 ppm cimaterol to the diet (relative to 0 ppm), and feed intake (but not growth rate) further increased by adding 200 versus 50 ppm cimaterol ($P<.05$). Feed efficiency of control line males was not different for the 0 and 200 ppm treatments, but both were less efficient than the 50 ppm treatment ($P<.05$). A "lean body index" was calculated as empty body weight minus eight times epididymal fat pad weight, and line x treatment interactions were again evident, both for the index and for the ratio of fat pad to empty body weight. While select line mice treated with 0 and 200 ppm cimaterol did not differ for lean index, the 50 ppm treated males had a higher index ($P<.05$). Control line mice fed 50 or 200 ppm cimaterol had equivalent indexes, but both indexed significantly higher than mice fed 0 ppm cimaterol. Fat pad to empty body weight ratio decreased at a faster rate as the level of cimaterol increased in select line than it did in control line mice ($P<.01$). The ratio for mice fed 0 or 50 ppm cimaterol was greater for the select versus the control line mice, but no line differences were apparent at the 200 ppm treatment level. These results led the authors to speculate that genetic differences in metabolic regulation might exist between the two lines.

While genetic differences in performance clearly do exist between breeds of swine used in U.S. production systems (Johnson, 1981), it could be argued that the magnitude of these differences is not such as to suggest radical differences in growth metabolism between breeds. Differential response between Occidental breeds and crosses treated with repartitioning agents might not, therefore, be of great practical importance. If, on the other hand, we consider swine breeds worldwide, differential response between breeds may be significant. Certain highly prolific breeds of Chinese pig are currently of particular interest as potential candidates for importation to the U.S. These pigs are very different from Occidental breeds not only for reproduction, but also in terms of growth and body composition. Gianola et al. (1982) and Legault (1983) characterized these Chinese breeds as follows:

1. Exceptionally prolific (on average, 9 to 17 pigs born alive per litter).
2. Early maturing (reaching puberty between two and four months of age).
3. Hardy, long-lived and docile.
4. Adapted to production systems where forages are routinely fed.
5. Low growth rate (.25 to .80 kg/day).
6. Small mature size and poor feed efficiency.
7. Fat carcasses (20 to 50 mm backfat), but an excellent meat quality.
8. Poor conformation.

Although early maturing and prolific, it is generally recognized that the Chinese Taihu breeds (Meishan, Fengjing, Jiaxing-Black and Erhualian) produce fat carcasses that are not well-muscled. Research based upon a French importation of three Meishan and three Jiaxing pigs (one boar and two sows of each breed) in 1979 found that 1/2-Chinese females reached puberty at approximately three months of age, consumed less feed during lactation although weaning heavier litters than purebred European sows, and were able to wean 5 to 8 extra pigs per sow per year (table 1). A first farrowing at 260 days and a litter size of 13 pigs in F1 American x Chinese females appears to be realistic based upon literature reports. While the reproductive advantage of the Taihu breeds is clear, these pigs have poor lean tissue growth and carcass composition compared to Occidental breeds. Results obtained from mating 1/2-Chinese females to Belgian Landrace boars, producing 1/4-Chinese offspring, are presented in table 2. While 1/4-Chinese pigs grew at an acceptable rate, their feed efficiency, dressing percentage, carcass composition and carcass grade were not competitive with the 100% European three-way cross pigs. In fact, the cost savings associated with producing the 1/4-Chinese pig were cancelled out by poorer feed conversion and the penalty imposed under the EEC carcass grading system (Legault et al., 1985). Subsequent field evaluation of 1/2- and 1/4-Chinese sows in France

TABLE 1. Litter size and weight and lactation feed intake for purebred Chinese, 1/2-Chinese, 1/4-Chinese, and purebred European sows [a]

Genetic type of the dam[b]	No. of litters (sows)	Litter size			Litter weight (kg)		Lactation feed intake (kg)[c]
		Total born	Born alive	Weaned[c]	At birth	At 21 days	
MS	115 (35)	14.9 a	14.0 a	13.1 a	16.2 a	57.3 a	101.1 a
JX	86 (29)	11.6 b	10.8 b	10.0 b	9.5 b	38.5 b	85.4 b
LW, FL	42 (22)	10.7 b	10.2 b	9.2 b	14.7 a	56.8 a	161.8 c
1/2-MS	107 (42)	15.3 a	14.5 a	12.8 a	19.3 c	67.8 c	130.0 d
1/2-JX	68 (29)	15.2 a	14.7 a	13.2 a	15.8 a	64.5 c	130.7 d
1/4-Chinese	63 (24)	11.5 b	10.8 b	9.9 b	15.6 a	57.6 a	118.7 e

[a] *From Legault et al. (1984).*
[b] *MS = Meishan, JX = Jiaxing, LW = Large White, FL = French Landrace, 1/2-MS = MS x (LW and FL), 1/2-JX = JX x (LW and FL), 1/4-Chinese = LW x (1/2 MS or 1/2 JX).*
[c] *30 day lactation, concentrate feeding.*
Means within the same column with different letters differ significantly (P<.05).

TABLE 2. Growth and carcass traits in 1/4-Chinese and control European slaughter pigs [a]

Trait	Genetic type[b]		
	1/4-MS	1/4-JX	Control
No. animals (dams)	317 (31)	306 (20)	85 (22)
Average daily gain, 26-100 kg, kg/d	.790 a	.754 b	.818 c
Feed-to-gain ratio	3.63 a	3.74 a	3.40 b
Dressing percent	77.9 a	78.4 b	78.3 ab
Carcass length, cm	95.8 a	97.9 b	97.0 b
Backfat thickness, rump	31.1 a	29.9 b	27.8 c
mm back	27.3 a	25.6 b	25.1 b
Ham weight, kg	8.39 a	8.28 b	8.86 c
Loin weight, kg	10.47 a	10 54 a	11.20 b
Shoulder weight, kg	5.59 a	5.60 a	5.51 a
Belly weight, kg	4.32 a	4.39 b	4.23 a
Estimated percent lean	45.6 a	45.1 a	49.1 b
Meat quality index	86.3 a	86.1 a	85.8 a

[a] *From Legault et al. (1985).*
[b] *MS = Meishan, JX = Jiaxing, Control = Belgian Landrace x (French Landrace-Large White).*
Means within the same row with different letters differ significantly (P<.05).

found that 1/2-Chinese sows produced about three more pigs born, and two more pigs weaned, per litter than European contemporaries; while 1/4-Chinese sows had only a one pig born and one-half pig weaned advantage over European sows. Taken with data for production traits collected at central test stations (4.2% less lean tissue for the 1/4-Chinese pig and 2.5% less for the 1/8-Chinese pig), it was concluded that the increase in sow productivity was insufficient to compensate for the poorer carcass merit of the offspring (Gueblez et al, 1987).

Interest in exploiting the prolificacy of such breeds, despite the unacceptable carcass composition of market hogs produced by 1/2-Chinese dams, has prompted U.S.D.A. scientists at the Meat Animal Research Center (MARC) in Nebraska to initiate experiments treating genetically lean and obese swine with repartitioning agents. The lines were derived from Duroc and Yorkshire lines selected for high and low backfat thickness for 16 generations at Beltsville, Maryland (Hetzer and Harvey, 1967). After the selection experiment concluded in 1970, pigs were moved to MARC and composite lean and obese lines established. Results of an initial experiment treating pigs with cimaterol have yet to be released, but control pigs from the lean and obese lines averaged 29 versus 59 mm of backfat at the last rib, 31 versus 22 cm^2 loin eye areas and 56-day gain-to-feed ratios of .250 versus .209, respectively (J. T. Yen, personal communication).

Such lines clearly represent interesting genetic models with which to investigate possible genotype x repartitioning agent dose-response relationship interactions. Caution must be exercised, however, in extrapolating results obtained from such lines to breeds of Chinese pig. The fact that selected American pigs are also phenotypically fat does not necessarily imply equivalent genetic and metabolic regulation of growth, or equivalent expected response to repartitioning agents. A recent French experiment, for instance, found a 15.7 pig average litter size born for Meishan females, versus 12.1 pigs for Large White sows. This difference was the result of lower embryonic mortality in the Meishans, with little difference in ovulation rate between the two breeds (Bolet et al., 1986). Intensely selected 'hyperprolific' Large White sows in the same study averaged 13.1 pigs born per litter, but ovulated 30% more ova and had a 15% greater embryonic mortality than the "standard" Large Whites. Five thousand years of domestication, and perhaps 2,000 years of selection, has created the prolific Chinese pig. It would be unwise to suppose that less than two decades of single trait selection in two American lines could emulate the genetic makeup of these animals --and certainly this is not the contention of scientists at MARC.

Geneticists at Iowa State University are currently conducting an experiment to measure efficiency of lean tissue gain, and PST levels prior to slaughter, of stress positive and stress carrier (heterozygous) pigs under *ad libitum* and restricted feed intake. Porcine stress syndrome (PSS) is inherited as a simple autosomal recessive trait, although associated changes in growth and carcass composition appear to be codominant (Webb et al., 1982). In a cooperative project with Pitman-Moore, it is planned to treat three genotypes (stress positive, stress carrier and stress negative pigs, ideally groups of litter mates produced by mating heterozygous parents) under the two feeding regimes with PST and a control (L. L. Christian, personal communication). Porcine stress syndrome is associated with heavy muscling in pigs, and it will interesting to see if PST increases stress related problems in already susceptible animals, and whether there is a differential response to the repartitioning agent in lines that already differ for body composition characteristics.

Another study to investigate genetic differences for response to a repartitioning agent is currently in the planning stage at Purdue University. Scientists plan to feed pigs from five different genetic lines with two diets containing either 0 or 20 ppm of the beta-agonist ractopamine. Pigs will be taken to three end-points (100, 113 and 127 kg), and the effects of genotype, treatment, and the interaction of genotype and treatment on lean tissue growth rate and feed conversion efficiency assessed. Based upon a pilot study involving 32 market barrows, it is expected that pigs used in the study might show a range in percent lean of 44 to 60%, and of 7.5 to 12.5 for lean tissue feed conversion (A. P. Schinckel, personal communication).

Effects of Repartitioning Agents on Selection Objectives

The potential need to redefine selection objectives where market hogs are treated with repartitioning agents is apparent if we consider, for instance, the effects of PST on feed intake (McLaren et al., 1987). Appetite might be an important consideration in further improving production efficiency. Force-feeding studies are currently in progress with PST treated pigs at the University of Illinois to determine how limiting feed intake effects the biological potential for improvement in efficiency of lean tissue production with PST (R. A. Easter, personal communication).

A number of experiments selecting for rate of lean tissue gain in pigs have demonstrated undesirable correlated responses in meat quality traits (Glodek, 1982). Norwegian scientists (Froystein et al., 1979) reported significant line differences for pH, color and water-binding capacity of pork following eight generations of divergent selection based upon backfat thickness and rate of gain. Pigs in the high gain / low backfat line had a higher incidence of pale, soft and exudative (PSE) meat than those in the low gain / high backfat line. Results of 19 years of selection for lean growth in French Large White boars indicated a genetic trend towards paler meat, although no clear trends were observed for pH or water holding capacity (Ollivier, 1986).

Results of quantitative and subjective sensory panel measures of the quality of meat from PST treated pigs have indicated only small undesirable effects on pork quality, and suggested that treatment with PST will not result in a product that is unacceptable to the consumer (Novakofski, 1987). However, it may now be time to consider including meat quality in the definition of the aggregate genotype for selection index construction in order to prevent detrimental changes with continued selection. Some measure of pork quality (generally color) has for some time been included in the definition of the aggregate swine genotype in a number of European countries (Denmark, West Germany, The Netherlands, Switzerland; Lindhe et al., 1980); and Swatland (1982), addressing the challenges of improving Canadian meat quality, maintained it to be essential that meat quality become incorporated into swine breeding programs.

SUMMARY AND CONCLUSIONS

Extensive use of repartitioning agents such as porcine somatotropin and the beta-agonists appears almost certain to occur in U.S. pork production systems within the next decade. Implications of use of such agents for genetic improvement programs in swine include consideration of the optimal testing scheme and environment; the effects of repartitioning agents on phenotypic, genetic and economic parameters used to calculate selection indexes; and the need to consider traits such as appetite, response to repartitioning agents, and meat quality in true merit functions. The availability of recombinant PST is only an example of the probable future impact of biotechnology on livestock production. Rather than inserting the porcine growth hormone gene into bacteria, producing and harvesting the hormone, and then administering it to pigs; creation of transgenic animals by direct insertion of the growth hormone and other genes with major phenotypic effects into the genome of the pig, and successful incorporation, expression and segregation to subsequent generations, should become possible in the future (Pursel, 1987).

Repartitioning agents might have an impact not only on existing breeding programs, but also could make practical an increase in diversity of the gene pool upon which we base pork production in the U.S. The Taihu breeds of swine in China offer great promise for improvement of prolificacy, but present severe problems in the areas of carcass composition and economy of lean tissue gain. Selection for feed efficiency and lean content have proven effective in swine (Glodek, 1982), and it should therefore be possible to move Chinese (or some "composite" American x Chinese) populations to satisfactory levels for these traits in time using classical breeding techniques. However, it is also

possible that the impact of repartitioning agents on part-Chinese hogs may be such that the perceived disadvantages of the prolific breeds becomes largely irrelevant in an altered production environment.

It is critical that the effects of repartitioning agents on puberty and sexual function in the pig be investigated, and that appropriate genetic improvement programs be developed to meet the needs of commercial producers if significant uptake of the new technology seems likely. The promise offered by repartitioning agents for improving the efficiency of production of quality lean pork appears to be great, and probably represents only the initial impact of exciting developments taking place in the field of biotechnology -- developments that will pose exciting new challenges to swine breeders by the year 2000.

LITERATURE CITED

Berne, R. S., J. Novakofski and P. J. Bechtel. 1985. Effects of the β-agonist clenbuterol on body and tissue weights in four strains of rats. J. Anim. Sci. 61(Suppl.1) : 256 (Abstr.).

Bolet, G., F. Martinat-Botte, A. Locatelli, J. Gruand, M. Terqui and F. Berthelot. 1986. Components of prolificacy in hyperprolific Large White sows compared with the Meishan and Large White breeds. Genet. Sel. Evol. 18(3) : 333-342.

Easter, R. A. 1987. Nutritional requirements and repartitioning agents. Proc. Univ. of Illinois Pork Industry Conference, "The Repartitioning Revolution: Impact of Somatotropin and Beta Adrenergic Agonists on Future Pork Production", December 10-11.

Eisen, E. J., W. J. Croom and S. W. Helton.. 1987. Effects of cimaterol in mice selected for rapid postweaning gain. J. Anim. Sci. 65 (Suppl. 1) : 276-277 (Abstr.).

Falconer, D. S. 1981. Introduction to Quantitative Genetics. 2nd Ed. Longman, New York.

Fleming, B. 1987. Wonder drug promises super-lean hogs: Dream or reality? National Hog Farmer, August : 16-19.

Froystein, T., K. A. Schie and S. O. Nostvold. 1979. Halothane sensitivity, blood CPK-values and meat quality characteristics in pigs selected for rate of gain and backfat thickness. Acta Agric. Scand. Suppl. 21 : 432-440.

Grebner, G. L., F. K. McKeith, J. Novakofski, R. A. Easter, D. G. McLaren, K. Brenner, P. J. Bechtel, R. W. Jones and D. L. Ingle. 1987. Carcass characteristics of pigs injected with different levels of porcine somatotropin (PST) from 57 to 103 kg weight. J. Anim. Sci. 65 (Suppl. 1) : 245-246 (Abstr.).

Gianola, D., C. Legault and J. C. Caritez. 1982. Evaluation of Chinese swine germplasm in France: Preliminary results. Proc. Univ. Ill. Pork Industry Conf., Dec. 2-3, Urbana-Champaign. pp. 39-55.

Glodek, P. 1982. Selection responses in pigs: Results and implications. Proc. 2nd World Congr. on Genet. Appl. to Livest. Prod., Madrid, Spain. V : 568-577.

Gueblez, R., L. Bruel and C. Legault. 1987. Evaluation des croisements Sino-Europeens en conditions de terrain en France. Journees Rech. Porcine en France 19 : 25-32.

Hetzer, H. O. and W. R. Harvey. 1967. Selection for high and low fatness in swine. J. Anim Sci. 26 : 1244-1251.

Johnson, R. K. 1981. Crossbreeding in swine: Experimental results. J. Anim. Sci. 52 : 906-923.

Lattz, D. H., C. E. Cagley, R. P. Kesler and I. Chow. 1987. 1986 62nd annual Summary of Illinois Farm Business Records. Univ. Ill./Coll. Agric./Coop. Extn. Serv. Circular 1276.

Legault, C. 1983. Reproductive performance of Chinese pigs in pure and crossbreeding. Proc. Natnl. Swine Improvement Fedn. Meet., Dec. 4-6, St. Louis. pp. 45-50.

Legault, C., J. C. Caritez, J. Gruand et J. P. Bidanel. 1984. Le point de l'experimentation sure les races chinoises en France: Reproduction et production. Journees Rech. Porcine en France. 16 : 481-494.

Legault, C., P. Sellier, J. C. Caritez, P. Dando et J. Gruand. 1985. L'experimentation sure le porc chinois en France. II. Performances de production en croisement avec les races europeennes. Genet. Sel. Evol. 17 : 133-152.

Lindhe, B. G., E. W. Averdunk, E. W. Brascamp, H. Duniec, I. M. Gajic, C. Legault and D. E. Steane. 1980. Estimation of breeding values in pigs -- Report of a working group of the commission on animal genetics. Livest. Prod. Sci. 7 : 269-282.

McLaren, D. G., D. S. Buchanan and R. L. Hintz. 1985. Sire ranking based upon purebred versus crossbred progeny performance in swine. J. Anim. Sci. 60 : 902-912

McLaren, D. G., G. L. Grebner, P. J. Bechtel, F. K. McKeith, J. Novakofski, R. A. Easter, R. W. Jones and D. L. Ingle. 1987. Effect of graded levels of natural porcine somatotropin (PST) on growth performance of 57 to 103 kg pigs. J. Anim. Sci. 65 (Suppl. 1) : 245 (Abstr.).

Muller, E. 1986. Physiological and biochemical indicators of growth and composition. In: Smith, C., J. W. B. King and J. C. McKay (Eds). Exploiting New Technologies in Animal Breeding. Oxford University Press, U.K.

Novakofski, J. 1987. Sensory quality, consumer acceptance and safety concerns. Proc. Univ. of Illinois Pork Industry Conference, "The Repartitioning Revolution: Impact of Somatotropin and Beta Adrenergic Agonists on Future Pork Production", December 10-11.

Ollivier, L. 1986. Results of a long-term selection experiment for lean tissue growth in the pig. Proc. 3rd World Congr. Genet. Appl. Livest. Prod., Lincoln, NE. XII : 168-175.

Pursel, V. 1987. Potential for inserting the somatotropin gene into the swine DNA: long term goals and implications. Proc. Univ. of Illinois Pork Industry Conference, "The Repartitioning Revolution: Impact of Somatotropin and Beta Adrenergic Agonists on Future Pork Production", December 10-11.

Robison, O. W. (Ed). 1981. Guidelines for Uniform Swine Improvement Programs. NSIF / USDA / SEA Program aid 1157

Swatland, H. J. 1982. The challenges of improving meat quality. Can. J. Anim. Sci. 62 : 15-24.

Webb, A. J., A. E. Carden, C. Smith and P. Imlah. 1982. Porcine stress syndrome in pig breeding. Proc. 2nd World Congr. on Genet. Appl. to Livest. Prod., Madrid, Spain. V : 588-608.

The Role of Somatotropin in Swine Health and Disease[1]

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INTRODUCTION

Throughout the production cycle, swine are subjected to infections which result in losses due to mortality, reduced feed efficiency, prolonged growth period, downgrading of meat, and carcass condemnations. Producers must also pay the cost of disease prevention and treatment. Risk of infection may be enhanced by environmental and nutritional stressors which reduce natural defense mechanisms and thereby lower the resistance of animals to infection.

Keeping a herd healthy and disease free can mean the difference between a profitable and unprofitable operation. The U.S. Department of Agriculture has estimated that death losses from swine diseases exceed $1 billion annually (1985, NPPC Producer Report). Production losses associated with subclinical swine diseases would raise this figure considerably.

Although antibiotics are routinely used for disease control in animal production, one promising alternative currently under investigation is the use of immunomodulators to stimulate host resistance to infection. A number of well-characterized, poorly characterized, or as yet undiscovered soluble mediators (e.g. lymphokines, monokines, cytokines) exert stimulatory or suppressive effects on the various cells of the immune system. Such lymphoid

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[1]This research was supported in part by grants to KWK from NIH AG 06246-01, USDA 86-CRCR-1-2003, IMC Pitman-Moore Inc. Grant No. K-0503 and the Illinois Agriculture Experiment Station Project No. 20-0386.

cells, in turn, produce these mediators. These immunomodulators can be either synthetic compounds or natural proteins from the targeted animal. It is obvious that the isolation, characterization, and large-scale production of these factors, along with their application to immunoprophylaxis and/or immunotherapy in homologous or heterogous food-producing animals, would be great benefit to the agricultural industry.

The immune system is an intricate regulatory network that serves to defend animals against pathogenic substances, such as microorganisms like bacteria and viruses. The immune system has several host defense mechanisms which include several types of cells that work together to protect the body against infection (Figure 1).

Respiratory diseases of swine such as pneumonia are responsible for the greatest losses in revenue, causing an estimated annual loss of $400 million (1985 NPPC Producer Report). Reproductive diseases are responsible for another $200 million in lost revenue. Other diseases, and the losses they incur, include the following: (1) Enteric or gastrointestinal diseases of young pigs, $140 million, (2) Lameness (including arthritis), $25 million, (3) Enteric or gastrointestinal diseases of hogs, $96 million, (4) Milk production deficiencies, $100 million, (5) Pseudorabies, a herpes virus affecting the central nervous system, $5 million, and (6) External parasites, such as mites and lice, $5 million (1985 NPPC Producer report).

Endocrine regulation of immune events has added a new level of understanding of how lymphoid cells interact with one another, and represents a concept that was accurately predicted in 1977 by Besedovsky and Sorkin (Besedovsky and Sorkin, 1977). During the past several years, many laboratories, including ours, have been concerned with the role of hypothalamic-pituitary-adrenal axis on hormonal and cell-mediated immune functions of domestic animals (reviewed by Kelley, 1987). These findings have stimulated much current interest in state-of-the-art immunophysiology research.

A majority of research in immunophysiology has focused on the function of B and T lymphocytes after <u>in vitro</u> exposure to pituitary-derived endocrine hormones. However, since one of the most important lymphoid effector cells in host defense against microbial infection is the macrophage (Adams and Hamilton, 1984), current research in our laboratories have been concerned with the effect of somatotropin (ie. growth hormone) and prolactin on macrophages. Although there are limited published data in the area of macrophage activation and hormones, there are some recent studies which suggest that pituitary hormones play a substantial role in mononuclear cell function and activation (Bernton et al. 1987, Peck 1987, Sharp et al., 1985).

The possible neuroimmunomodulatory role of somatotropin in macrophage activation of mononuclear phagocytes in relationship to swine disease and health is addressed in this paper. Our findings to date suggest that somatotropin may have a fundamental role in

regulating cell-mediated macrophage immunity and also have important practical applications in improving host resistance of swine to bacterial, parasitic and viral diseases.

MAJOR SWINE RESPIRATORY DISEASES: *Haemophilus pneumonia*

Pleuropneumonia is a specific bacterial disease of pigs caused by *Haemophilus pleuropneumoniae*. The disease occurs in an acute and chronic form. During outbreaks of acute disease, the mortality can reach 100% among piglets and 25% among feeder pigs. The chronic form, characterized by pluritis and localized pulmonary necrosis, reduces growth rate and increases production costs. Financial losses due to this disease have been estimated at millions of dollars annually. In a recent review, Schultz (1986) offered an astounding statistic stating that for 1% of lungs showing lesions of pleuropneumonia, time taken to reach 90 kg live weight increased by 1.2 days. In a recent poll of swine producers, *Haemophilus* pneumonia was cited as a significant health problem by more than 15% of the responders, placing this infection the sixth most prevalent bacterial disease problem in swine today (1985 NPPC Producers report).

Etiology and Pathogenesis

The infectious agent of porcine pleuropneumonia was characterized for the first time in the early 1960s. Due to the fact that this small, gram negative coccobacillary bacterium requires nicotinamide adenine dinucleotide (NAD) for its growth and because of its pathogenic properties, Kilian *et al*. (1979) proposed to name it *Haemophilus pleuroneumoniae*. Recently, Pohl (1981) showed that *Haemophilus* species are closely related by DNA/DNA homology to members of the genus *Actinobacillus*. To facilitate communication between practitioners and research scientists, both names are often used (i.e. *Haemophilus (Actinobacillus) pleuropneumoniae)*.

Haemophilus (Actinobnacillus) pleuropneumoniae is reported from all countries where pig production is industrialized and of the 9 known serotypes, serovars 1, 3, 4, 5 and 7 cause disease in the United States. The disease is airborne, and is transmitted mainly by direct contact from pig to pig. All age categories are susceptible and mortality can vary from 10-100% (Shultz, 1986).

Haemophilus pneumonia is characterized by an acute hemorrhagic, necrotizing, fibrinous pneumonia involving all lobes of the lung. In contrast to other pneumonia-causing agents, the dorsal areas of all lobes are involved. Localized hemorrhagic lesions in the dorsal areas of the lung may be the only lesions present in pigs that have been euthanized. Generally, there is also a severe fibrinous pluritis and pericarditis, which adhesions and abscessed foci develop as the disease becomes chronic (Pijoan, 1986).

H. pleuropneumoniae is characterized by its high virulence owing to the presence of a capsule and the action of toxins. Experimental infections in specific-pathogen-free (SPF) pigs showed that as a few as 10^2 bacteria may induce a disease, and the lethal dose can vary from 10^4-10^6 (Pijoan, 1986). The severe pulmonary vascular damage in the peracute and acute stage of the disease strongly suggests an endotoxic shock (Pijoan, 1986).

ATROPHIC RHINITIS

Atrophic rhinitis (AR) is a widely prevalent contagious disease of swine that is characterized by sneezing, atrophy of the nasal turbinate bones, facial distortion, and nasal hemorrhage. The disease is of economic significance in swine production globally since disease symptoms are accompanied by large losses (5-20%) in feed efficiency and weight gain (Muirhead, 1979). In addition to reducing growth rates, AR can significantly increase the costs associated with clinical disease, such as medication costs, penalties at slaughter, or inability to sell breeding stock. Despite the availability of vaccines and antibiotics, a poll by the National Pork Council found that atrophic rhinitis was the second most common bacterial disease problem reported by producers (NPPC Report, 1985). Recent surveys by the American Veterinary Medical Association identified AR in 100% of growing-finishing swine operations (Straw, 1986).

Etiology and Pathogenesis

Atrophic rhinitis of pigs is a complex disease in which infectious agents, environment, management, and herd genotype all contribute to its prevalence and severity in individual herds. The pathological changes of atrophic rhinitis include varying degrees of atrophy of the nasal turbinates and shortening or distortion of the snout. Both Bordella bronchiseptica and Pasteurella multocida are involved in its etiology (Pijoan, 1986).

Infection with B. bronchiseptica alone or in combination with non-toxigenic P. multocida results in only mild to moderate lesions (Rutter and Rojas, 1982). However, combined infections with B. bronchiseptica and toxin-producing strains of P. multocida can result in the persistent form of the disease characterized by severe turbinate atrophy, nasal distortion, and reduced growth rate (Straw, 1986).

The prevalence of B. bronchiseptica infection often greatly exceeds that of clinical AR or marked turbinate atrophy at slaughter (Dominick and Rimler, 1986). These virulent microorganisms cause marked loss of cilia accompanied by morphological changes in the nasal mucosa after intranasal inoculation of gnotobiotic piglets (Rutter and Rojas, 1982). The lesions in B. bronchiseptica induced rhinitis are accompanied by a variable amount of turbinate hypoplasia in pigs up to

8 weeks of age. Ultrastructural observations indicate that defective osteogenesis rather than osteolysis is the basic mechanism of hypoplasia and infection has been shown to result in decreased synthesis of osteoid (Silveira et al., 1982).

In uncomplicated B. bronchiseptica infection, it is clear that the hypoplasia of turbinates produced by infection of young pigs is capable of regeneration (Rutter, 1981). This differs from the irreversible turbinate atrophy induced by toxigenic Pasteurella multocida which is characterized by osteolysis and bone resorption.

In summary, AR is transmitted from pig to pig by aerosol droplet infection, and from infected sows to suckling pigs. Experiments with different bacterial isolates have led several investigators (Straw, 1986; de Jong and Akkermans, 1986; Elling and Pederson, 1985) to describe the following model of AR pathogenesis:

The resistance of the nasal mucosa is weakened by noninfectious factors or by B. bronchiseptica infection. This permits toxigenic strains of P. multocida to become established on the nasal mucosa where they produce toxin. P. multocida toxin will enhance osteoclastic resorption and impair osteoblastic synthesis of the turbinate osseous core; irreversible changes can be produced within a few days. The epithelium and submucosa undergo secondary atrophy and turbinates may disappear almost completely within 10-14 days in growing-finishing swine. The lesions persist until slaughter, when the prevalence and severity of turbinate atrophy is established by a morphometric index (Done et al., 1984).

THE IMPORTANCE OF THE MACROPHAGE CELL IN SWINE HEALTH

The phagocytic and bactericidal capabilities of pulmonary alveolar macrophages are critically important in pulmonary clearance and defense mechanisms. The macrophage cell (M ϕ) is functionally and quantitatively the most important member of a series of cells identified collectively as the mononuclear phagocytic system. The concept of the mononuclear phagocytic system was first put forward by Langevoort in 1969 and was subsequently expanded by Van Furth (Van Furth, 1970). It is now generally accepted that this system is composed of a spectrum of cells that have a common origin in the bone marrow. Various constituents of the mononuclear phagocytic system differ markedly in morphology, functional characteristics, and metabolic patterns.

Mononuclear cells are ubiquitous in mammalian tissues, and they function critically in the ecology of the body. Phylogenetically, the mononuclear phagocyte is one of the most primitive cell types. Cells homologous to the mononuclear phagocyte are found in early life forms, and single-cell protozoa such as the amoeba have considerable similarity to the mammalian macrophage. Ontogenically, the macrophage has its origin in the yolk sac (Moore and Metcalf, 1970), but the cells

of the mononuclear phagocytic system in the adult derive from a common ancestor in the bone marrow (Cline et al., 1978). Figure 2 illustrates the lineage of the macrophage.

Mononuclear phagocytes serve five major functions:

1. They participate in host defense against microorganisms and are particularly effective in killing obligate intracellular microbes.

2. They are the principal cellular element involved in the removal of damaged, senescent, or dying cells, and they metabolize organic debris and sequester non-metabolizable inorganic materials.

3. Mononuclear phagocytes participate in bidirectional cellular interactions with T and B lymphocytes which are important in humoral and cellular functions.

4. Mononuclear phagocytes produce bioactive materials important in regulating other cellular functions. Such factors include colony-stimulating activity (CSF), plasminogen activator (t-PA), complement components, interleukin-1 (IL-1), tumor necrosis factor-alpha (TNF-α), prostaglandins (PG), thromboxanes (TX), and leukotrienes (LT).

5. Macrophages play a critical role in the cell-mediated response to cancer (Herberman, 1983).

Resting macrophages, when exposed to a variety of inflammatory agents, undergo a range of changes. Common agents that are used in vivo to induce macrophage activation include thioglycollate, lipopolysaccharide (LPS), muramyl dipeptide (MDP), casein, protease peptone broth, and glycogen (Adams and Hamilton, 1984). The morphological changes induced in peritoneal macrophages exposed to such irritant substances include increased size, increased rate and extent of spreading on a glass or plastic surface, increased ruffling of the membrane, prominence of pseudopodia, and increased cytoplasmic granules and vacuoles.

Functional activities of elicited macrophages include alterations in enzyme content and secretion, as well as enhanced phagocytic, bactericidal or tumoricidal effects. A variety of terms have been used to describe macrophages that express these changes, and these include stimulated, induced, armed, angry, and activated (Cohn, 1978).

The Respiratory Burst

One of the most important bacterial killing mechanisms of these cells is the release of highly reactive oxygen-intermediates such as Superoxide Anion (O_2^-) and Hydrogen Peroxide (H_2O_2) which have been

shown to kill ingested microbes both *in vitro* and *in vivo*. Upon recognition of a particulate or soluble stimulus, Mϕ experience a respiratory burst, that is characterized by a 2- to 20-fold increase in oxygen consumption and increased glucose metabolism via the hexose monophosphate shunt (Figure 3). In conjunction with the increase in oxygen consumption, phagocytes have been shown to secrete both O_2^- and H_2O_2 (Babior, 1978). Stimulating substances include phorbol myristate acetate (PMA), a nonspecific membrane soluble activator, opsonized zymosan (Op-Zym), and other live, heat-killed, or opsonized preparations of bacteria. Each of these substances has shown to induce secretion of nanomolar quantities of O_2^- and H_2O_2 after stimulation of 1-10 million cells *in vitro*.

In vitro studies have shown that greater than 90% of the oxygen consumed by phagocytes after initiation of the respiratory burst can be accounted for by the secretion of O_2^- (Root and Metcalf, 1977). These results suggest that most of the H_2O_2 released during phagocytosis is directly derived by dismutation of O_2^-. The classical mechanism for the generation of H_2O_2 from O_2^- has been described by Fridovich (1976). The classis Haber-Weiss reaction for production of hydroxl radial ($\cdot OH$) involves the direction reduction of H_2O_2 by O_2^- with the formation of $\cdot OH$-, O_2^- and OH:

$$O_2^- + H_2O_2 \text{ -------> } O_2 + OH^- + \cdot OH$$

The enzyme system responsible for the increased oxygen consumption and O_2^- generation has been identified as a membrane-associated nicotinamide adenine dinucleotide phosphate (NADP[H] oxidase). There has been great debate in recent years concerning the precise location and biochemical characterization of the O_2^- forming oxidase system in the phagocytic cells, there appear to be at least two identifiable components. The first is a substrate-binding moiety with specificity for reduced NADP[H]; the second component oxidizes NADP[H] in the presence of oxygen, generating a reduced form of molecular oxygen, O_2. Recent evidence (Hamilton and Adams, 1987) about the respiratory burst has linked together the arachidonic acid metabolism, direct activation of phopholipase A_2, and indirect activation of phopholipase C and diacylglycerol lipases.

Superoxide anion (O_2^-) may act as either an oxidant or a reductant, depending on the substrate with which it reacts. Reduction of ferricytochrome c or nitoblue tetrazolium (NBT) represents a sensitive assay for the detection of the production of O_2^- by phagocytic cells (Figure 4).

Where intracellular killing by macrophages has been clearly demonstrated *in vitro*, investigators of the mechanisms involved have almost implicated reactive products of oxygen such as O_2^- and H_2O_2. Studies conducted in mice using *E. coli* derived recombinant murine IFN-γ, both *in vitro* and *in vivo* systems, have shown relatively high amounts of O_2 release when macrophages are stimulated with PMA. Other *in vitro* activating agents, including lipopolysaccharide (LPS)(Hedegaard and Pabst, 1982), muramyldipeptide (MDP)(Hedegaard and Pabst, 1982), and substance P (Peck, 1987), have been shown to be comparable to IFN-γ in their ability to activate macrophages to release O_2^-.

Alterations in bacterial killing functions of macrophages may influence the efficiency of bacterial clearance and the overall retention of particles in the lung. For example, there is a reduction in bacterial clearance in lungs of pigs infected with P. multocida (Pijoan, 1986) and the disease persists in infected animals that are stressed (Schoneweis and Henry, 1982). Several investigators (Fuentes and Pijoan, 1986; Kume et al., 1986) have developed pneumonia models in pigs using pseudorabies virus and P. multocida. These investigations, as well as our own, should provide further insight into the role of pulmonary alveolar macrophages in combating swine respiratory diseases.

Enhancement of Respiratory Burst Metabolites by Somatotropin

During the course of our studies on somatotropin, we incubated porcine lever cells with somatotropin and noticed a marked change in the morphology of Kupffer cells which was phenotypically characteristic of activated macrophages. These changes included increased acid hydrolase content, phagocytic activity, and pseudopodia spreading on plastic (Cohn, 1978). Our major objective was then to extend this observation and to see whether or not the same held true for purified porcine lymphoid cells.

To test the hypothesis that somatotropin and prolactin can augment O_2^- production, porcine peripheral blood monocyte-derived macrophage cells were obtained using a plasma percoll isolation procedure and incubated in culture overnight with increasing concentrations (1 ng/ml -1000 ng/ml) of pituitary-derived somatotropin (npST), or recombinant porcine somatotropin (rpST). The results, shown in Figure 5, suggest that somatotropin and prolactin can activate porcine macrophages in vitro. Using a particulate stimulus (opsonized zymosan; Op-zym) to trigger the respiratory burst, cells treated with 500 ng/ml of either npST or rpST were able to produce large amounts of O_2^-. Normal, control mononuclear cells released only small amounts of O_2^- (36 nMol O_2-/mg protein/hr) when stimulated with Op-zym. Lipopolysaccharide (LPS), which is a potent inducer of O_2^- and which served as the positive control, caused the release of 228 nMol O_2/mg protein/hr when the cells were stimulated with Op-zym. This macrophage activating property of porcine somatotropin was not caused by contaminating hormones in the pituitary-derived preparation because rpST yielded a similar dose response curve.

We then determined whether the enhancing effect of these pituitary hormones on the production of O_2^- could be blocked with antibodies (Figure 6). In these experiments, adherent porcine blood-derived porcine mononuclear cells were incubated for 24 hr with 500 ng/ml of npST, rpST in the presence or absence of specific antibodies as outlined in Figure 4. A heat-inactivated (56 C, 1 hr) guinea pig antiserum specific for porcine somatotropin (Marple and Aberle, 1972) was added to culture media cocktail containing the hormones for 24 hr before addition of the cocktail to the macrophages. After overnight incubation with this cocktail mixture, O_2^- release was measured. As

shown in Figure 6, both npST and rpST caused an 15- to 18-fold increase in O_2^- production compared to control mononuclear phagocytes treated only with Op-zym. This enhancement was reduced by 90- to 100% when the specific guinea-pig antibody was used. This blocking of O_2^- secretion by specific antibodies strongly suggests that pST is the factor responsible for augmented O_2^- production rather than contaminating substances (e.g., endotoxin) in the hormones preparations. Normal heat-activated guinea pig serum, mouse serum, mouse acites fluid, or the antibody preparations alone did not affect the production of O_2^-.

Porcine alveolar macrophages (>98% α-naphthyl esterase positive) were then used to determine whether porcine somatotropin could substitute for IFN-γ in activating a purified population of macrophages. Significant enhancement of Op-zym induced O_2^- was caused by npST and rpST, and these levels were similar to the effects caused by the porcine MAF-containing supernatant (Table 1). Again, the macrophage enhancing effects of these pituitary hormones were totally blocked by specific antisera.

These data provided convincing evidence that somatotropin shares the macrophage-activating property of IFN-γ for augmented O_2^- production *in vitro*. We therefore asked whether somatotropin would also prime macrophages *in vivo*. To conduct these experiments we used hypophysectomized rats. Since the pituitary source of somatotropin was removed, confounding effects with endogenous somatotropin in plasma were avoided. An optimal concentration of recombinant rat IFN-γ (Edwards, et al., 1986), which is a potent inducer of O_2^- in both *in vitro* and *in vivo* systems (Nathan, 1986), was used as a positive control.

As expected, npST and rpST, as well as native, pituitary-derived rat somatotropin (nrST), caused a significant increase in growth rate, ranging from approximately 10% to 40% over the 10-day growth period (Table 2). Macrophages from the two control groups (hypophysectomized-untreated and hypophysectomized-vehicle-treated) released no superoxide dismutase-inhibitable O_2^-. In contrast, macrophages from hypophysectomized rats given recombinant rat IFN-γ released 417 nMole O_2/mg protein/hr when stimulated with Op-zym. Hypophysectomized rats injected daily with 12 ug of npST released 268 nMole O_2^-/mg protein/hr, and even higher amounts (438 nMole O_2^-/mg protein/hr) were released at 24 ug. Both of these values were greater than the two negative controls ($P<.01$) rpST at both 12 ug and 24 ug also induced significant increases ($P<.01$) in the production of O_2^-, and the magnitude of the increase was similar to that induced by similar concentrations of npST. Priming of rat peritoneal macrophages for augmented production of O_2^- by somatotropin was not simply caused by injection of exogenous foreign protein because nrST also caused a significant augmentation in the production of O_2^-.

CONCLUSION

In summary, we believe our progress made to date on the potential use of porcine somatotropin in the regulation of immune events is exciting. It is well-known that the macrophage is a key cell in regulating many of the responses of cells of the immune system. Before these studies were conducted, IFN-γ was the only substance that was unequivocally known to augment the production of O_2^- by macrophages in both *in vitro* and *in vivo* systems. While our present studies are far from being complete, results presented in this paper support the idea that porcine somatotropin exhibits macrophage-activating properties in both *in vitro* and *in vivo*.

Although our studies are limited, the results to date suggest that porcine somatotropin is not detrimental to lymphoid cells. We hypothesize that if negative effects of porcine somatotropin are found on porcine health and disease, the cause might be due to hyperreactivity of lymphocytes and macrophages. Although this interpretation is clearly sepculative, it is consistent with available data in a variety of species. It also provides a conceptual framework for studies designed to determine whether porcine somatotropin has a potential role in improving host resistance to pathogenic diseases of animals and to augment the efficiency of vaccination programs.

REFERENCES

Adams, D. O. and T. A. Hamilton. 1984. The cell biology of macrophage activation. Ann. Rev. Immunol. 2:283.

Babior, B. M. 1978. Oxygen-dependent microbial killing by phagocytes. N.Eng. J. Med. 298:659.

Bernton, E., D. Hartmann, M. Gilbreath, J. Holaday and M. S. Meltzer. 1986. Inhibition of Macrophage *in vivo* activation by pharmacolgic blockade of prolactin release. In: Leukocytes and Host Defense. Alan R. Ciss, Inc. pp. 213-219.

Besedovsky, H. and E. Sorkin. Network of immune-neuroendocrine interactions. Clin. Exp. Immunol. 27:1.

Cline, M. J., R. I. Lehrer, M. C. Territo and D. W. Golde. 1978. Monocytes and macrophages; functions and diseases. Ann. Intern. Med. 88:78.

Cohn, Z. 1978. The activation of mononuclear phagocytes: fact, fancy and future. J. Immunol. 121:813.

Dominick, M. A. and R. B. Rimler. 1986. Turbinate atrophy in gnotobiotic pigs intranasally inoculated with protein toxin isolated from type D *pasteurella multocida*. Am. J. Vet. Res. 47:1532.

Dominick, M. A. and R. B. Rimler. 1986. Turbinate atrophy in gnotobiotic pigs intranasally inoculated with protein toxin isolated from type D pasteurella multocida. Am. J. Vet. Res. 47:1532.

Done, J. T., D Upcott, D. Frewin and C. H. Hebert. 1984. Atrophic rhinitis: Snout morphometry for quantitative assessment of conchol atrophy. Vet Rec. 113:33.

Douglas, J. D. 1984. Cells involved in immune responses. In: Basic and clinical immunology. Lange Pub., H. H. Fudenberg ed. Los Altos, CA. pp. 78-95.

Edwards III, C. K., H. B. Hedegaard, A. Zlotnik, P. R. Gangadharam, R. B. Johnston, Jr. and M. J. Pabst. 1986. Chronic infection due to Mycobacterium intracellulare in mice: association with macrophage release of prostaglandin E_2 and reversal by injection of indomethacin, muramyl dipeptide, or interferon-γ. J. Immunol. 136:1820.

Fridovich, I. 1976. Oxygen radicals, hydrogen peroxide, and oxygen toxicity. In: Free Radicals in Biology. Vol. 1. Academic Press. New York. p. 239.

Golde, D. W. and W. G. Hocking. 1982. Monocyte and macrophage development. In: Advances in Host Defense Mechanisms, Vol. 1. Raven Press, J. I. Gallin and A. S. Fauci, eds. New York. pp. 13-30.

Hamilton, T. A. and D. O. Adams. 1987. Molecular mechanisms of signal transduction in macrophages. Immunol. Today. 8:151.

Hedegaard, H. B. and M. J. Pabst. 1982. Preservation of superoxide anion generating capacity in cultured human monocytes by treatment with muramyl dipeptide or lipopolysaccharide. In: Immunomodulation by Microbial Products and Related Synthetic Compounds. Elsevier, Y. Yamamura ed. Amsterdam. pp. 205-208.

Herberman, R. B. 1983. Lymphoid cells in immune surveillance against malignant transformation. In: Advances in Host Defense Mechanisms-Lymphoid Cells. Vol. 2. Raven, New York. p. 241.

deJong, M. F. and J. P. W. M. Akkermans. 1986. Investigation into the pathogenesis of atrophic rhinitis in pigs. I. Atrophic rhinitis caused by Bordetella bronchiseptica and Pasteurella multocida and the meaning of a thermolabile toxin of P. multocida. Vet Quarterly, 8:204.

Kelley, K. W. 1987. Cross-talk between the immune and endocrine systems. J. Anim. Sci. In Press.

Kilian, M., J. Metescky and R. Schronhenlohes. 1979. Pathogenic

species of the genus Haemophilus and Streptococcus pneumoniae produce immunoglobin A1 protease. Infect. Immun. 26:143.

Kume, K., T. Nakai and A. Sawata. 1986. Interaction between heat-stable hemolytic substance from Haemophilus pleuropneumoniae and porcine pulmonary macrophages in vitro. Inf. Imm. 51:563.

Marple, D. N. and E. D. Aberle. 1972. Porcine Plasma growth hormone levels: Radioimmunoassay Technique and its Application. J. Anim. Sci. 34:261.

Moore, M. A. S. and D. Metcalf. 1970. Ontogeny of the haemopoietic system: Yolk sac origin of in vivo and in vitro colony forming cells in the developing mouse embyro. Br. J. Haematol. 18:270.

Muirhead, M. R. 1979. Respiratory Diseases of Pigs. Brit. Vet. J. 135:497.

Nathan, C. F. 1986. Interferon-gamma and macrophage activation in cell mediated immunity. In: Mechanisms of host resistance to infectious agents, tumors, and allografts. Rockefeller Press, R. M. Steinman and R. J. North eds. New York, pp. 165-185.

Peck. R. 1987. Neuropeptides modulating macrophage function. In: Neuroimmune interactions: Proceedings of the Second International Workshop on Neuroimmunomodulation. Ann. N. Y. Academy Sciences. Vol. 496. pp. 264-270.

Pijoan, C. 1986. Respiratory System. In: Diseases of Swine. 6th ed, A. D. Lemon ed, Iowa State University Press, Ames. pp. 152-161.

Pijoan, C. 1986. Effect of Pasteurella multocida and Haemophilus pleuropneumoniae toxins on swine alveolar macrophages. Vet. Immunol. Immunopath. 13:141.

Pohl, S. 1981. DNA relatedness among members of Haemophilus, Pasteurella and Actinobacillus. In: Haemophilus, Pasteurella and Actinobacillus. Academic Press, London. p. 245.

Pork Report 4 (10), 1985. National Pork Producers Council.

Root, R. K. and J. A. Metcalf. 1977. H_2O_2 release from human granulocytes during phagocytosis: relationship to superoxide anion formation and cellular catabolism of H_2O_2: studies with normal cytochalasin B-treated cells. J. Clin. Invest. 60:1266.

Rutter, J. M. 1981. Quantitative observations on Bordetella bronchiseptica infection in atrophic rhinitis in pigs. Vet. Rec. 108:451.

Rutter, J. M. and X. Rojas. 1982. Atrophic rhinitis in Gnotobiotic piglets: Differences in the pathogenicity of Pasteurella multocida in combined infection with Bordetella bronchiseptica. Vet. Rec. 110:531.

Schoneweis, D. A. and S. C. Henry. 1982. Theory and practice of immunoprophylaxis in swine. J. Am. Vet. Med. Assn. 181:1154.

Sharp, B. M., W. F. Keane, H. J. Suh, G. Gekker, D. Tsukayama and P. K. Peterson. 1985. Opioid peptides rapidly stimulate superoxide production by human polymorphonuclear leukocytes and macrophages. Endocrin. 117:793.

Schultz, R. A. 1986. Swine pneumonia: Assessing the problem in individual herds. Vet. Med. 8:757.

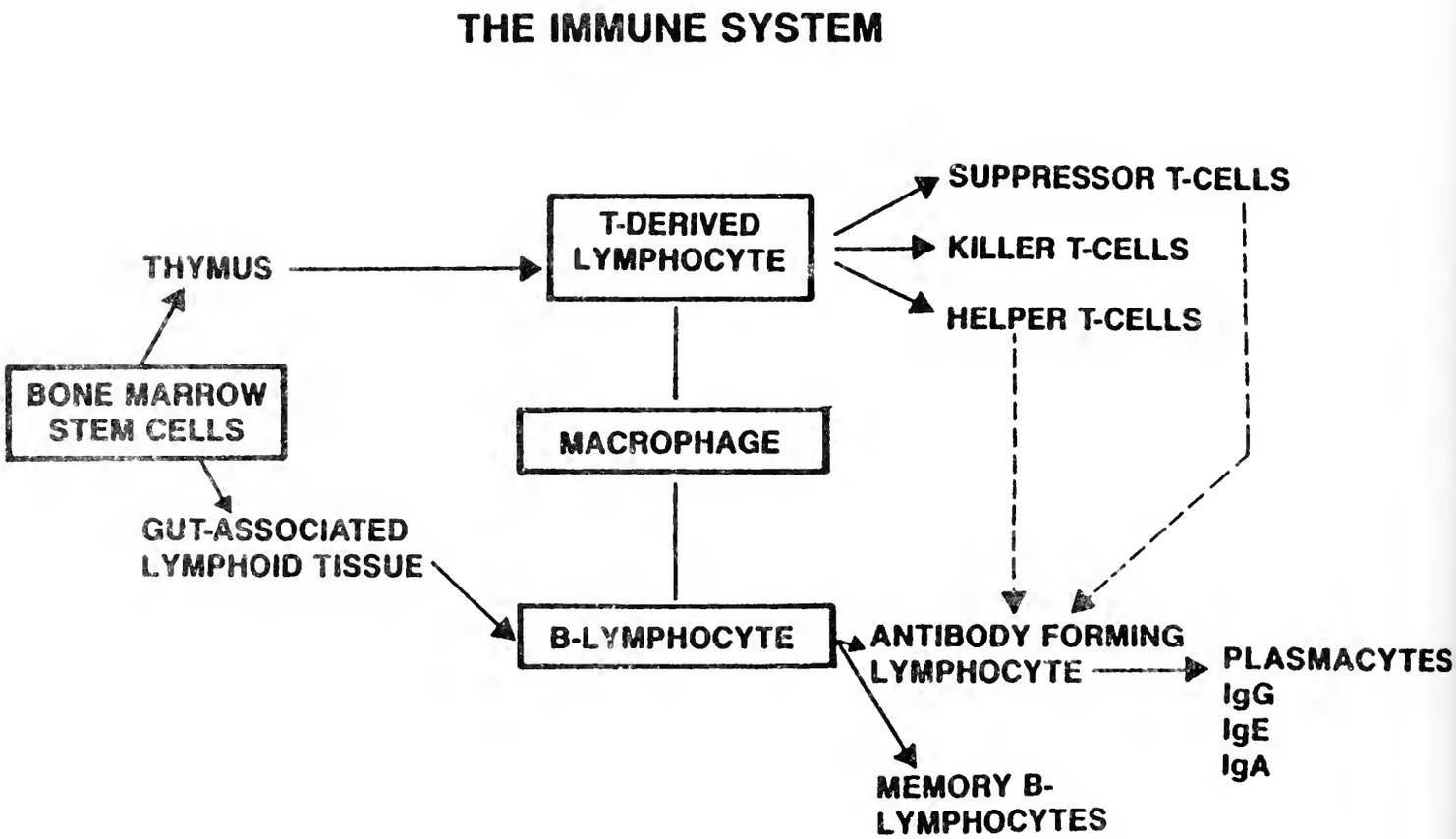
Silveira, D., N. Edington and I. M. Smith. 1982. Ultrastructural changes in the nasal turbinate bone of pigs in early infection with B. bronchipseptica. Res. Vet. Sci. 33:37.

Straw, B. 1986. A look at the factors that contribute to the development of swine pneumonia. Vet. Med. 8:747.

Tizard, I. 1986. Functions of macrophages. Vet. Med. 12:250.

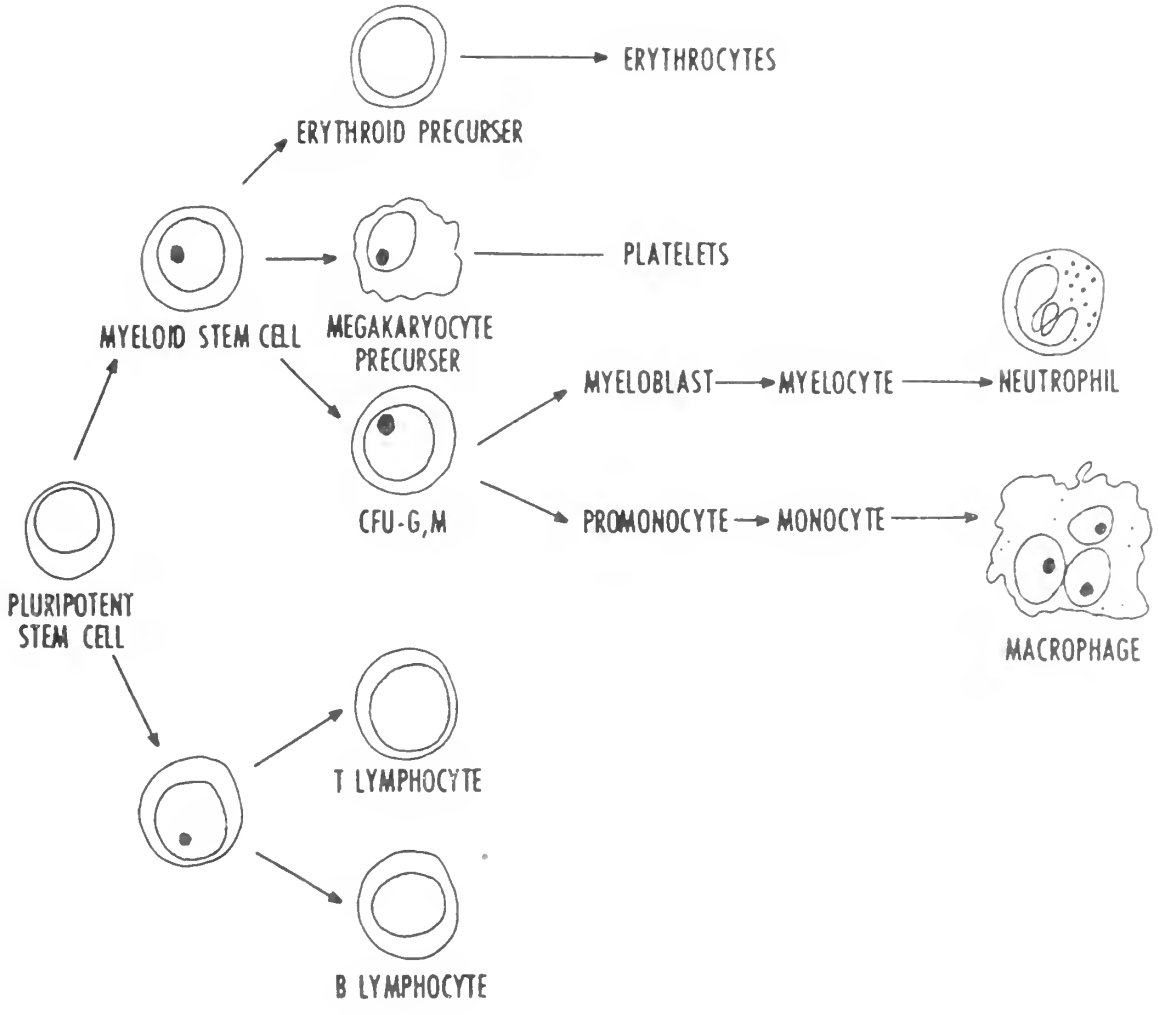
Van Furth, R. 1970. Origen and kinetics of mononuclear phagocytes. J. Exp. Med. 128:415.

FIGURE 1. An overview of cells of the immune system



Adapted from Douglas, 1984

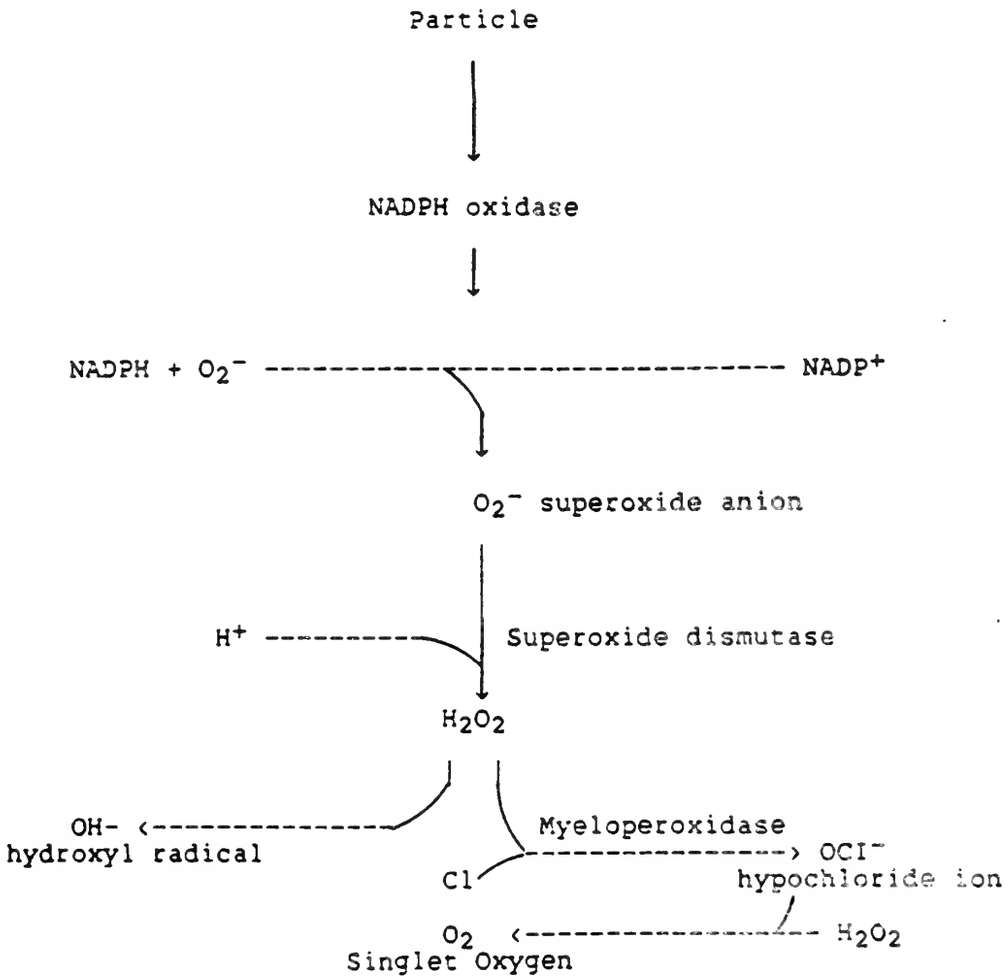
FIGURE 2. Origin of cells circulating in blood



Adapted from Golde and Hocking, 1982.

FIGURE 3.

The major pathways of the respiratory burst. In macrophages, myeloperoxidase is replaced by catalase. The bactericidal compounds include the hydroxyl radicals, hypochloride ions, and singlet oxygen.



Adapted from Tizard, 1986

FIGURE 4. Simplified Scheme for measuring superoxide anion secretion by macrophages.

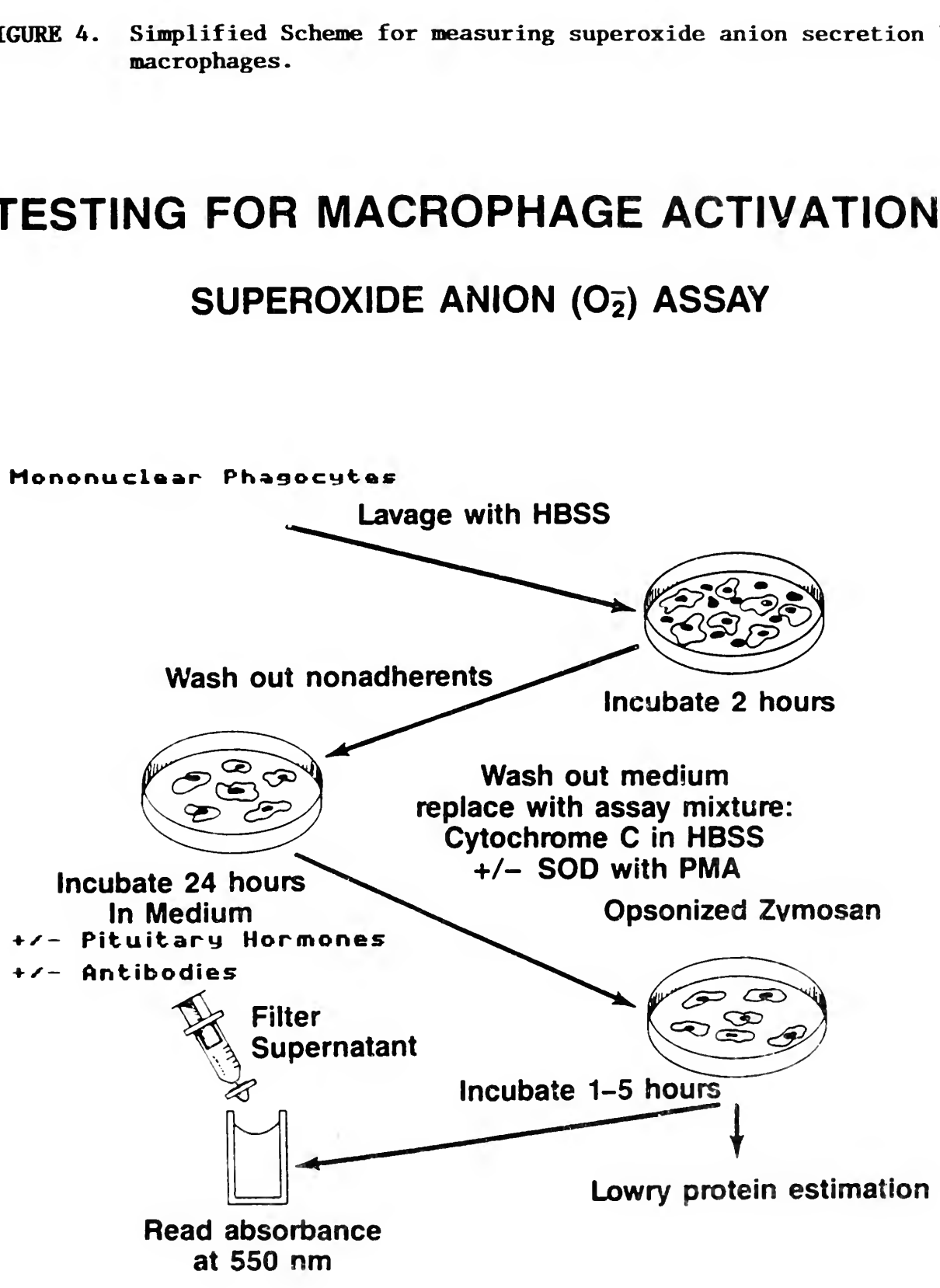


FIGURE 5. **Native porcine somatotropin (npST) and recombinant porcine somatotropin (rpST) augment the production of O_2^- by porcine monocytes.**

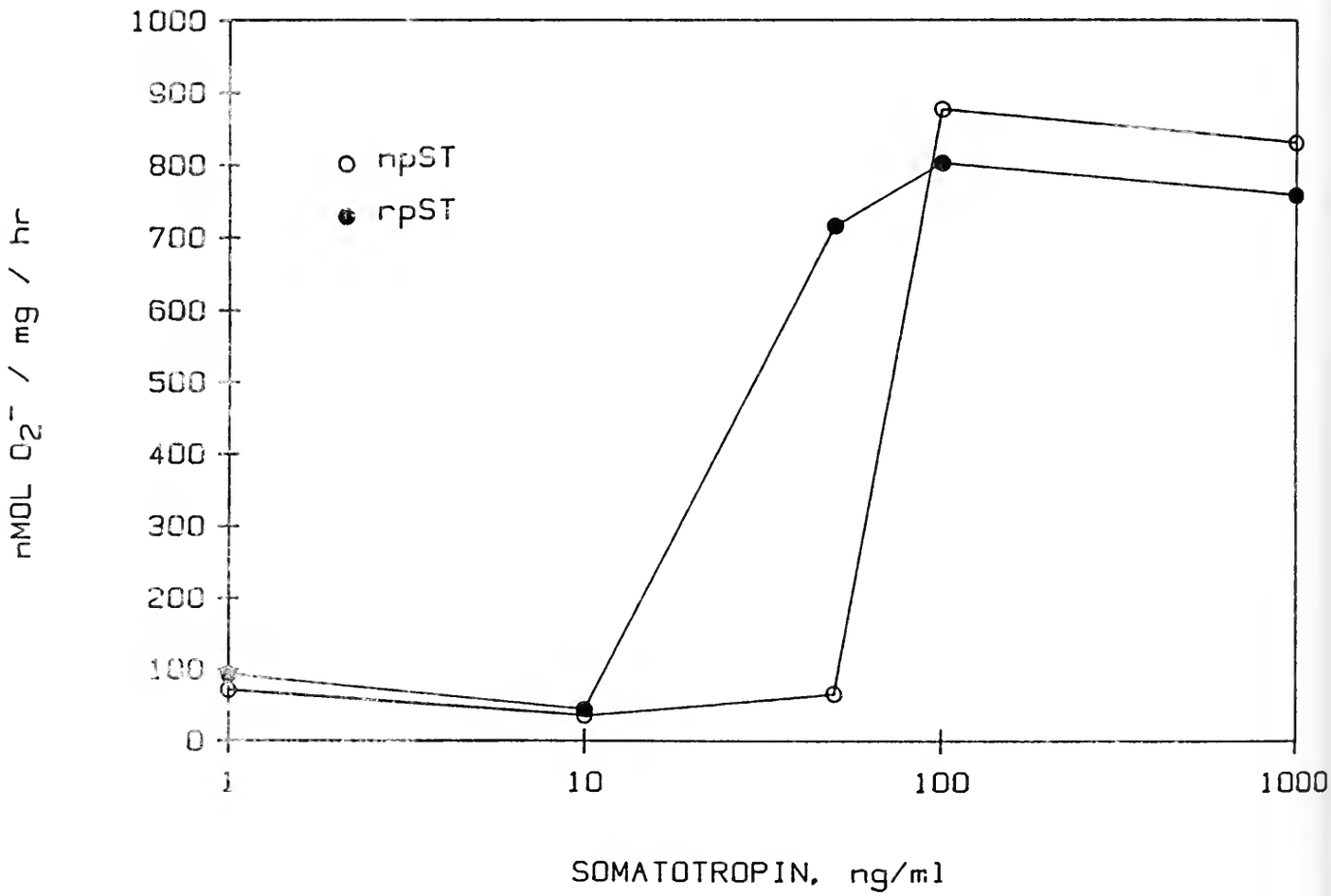


FIGURE 6. Specific antibodies to pST block the induction of O_2^- by both npST and rpST.

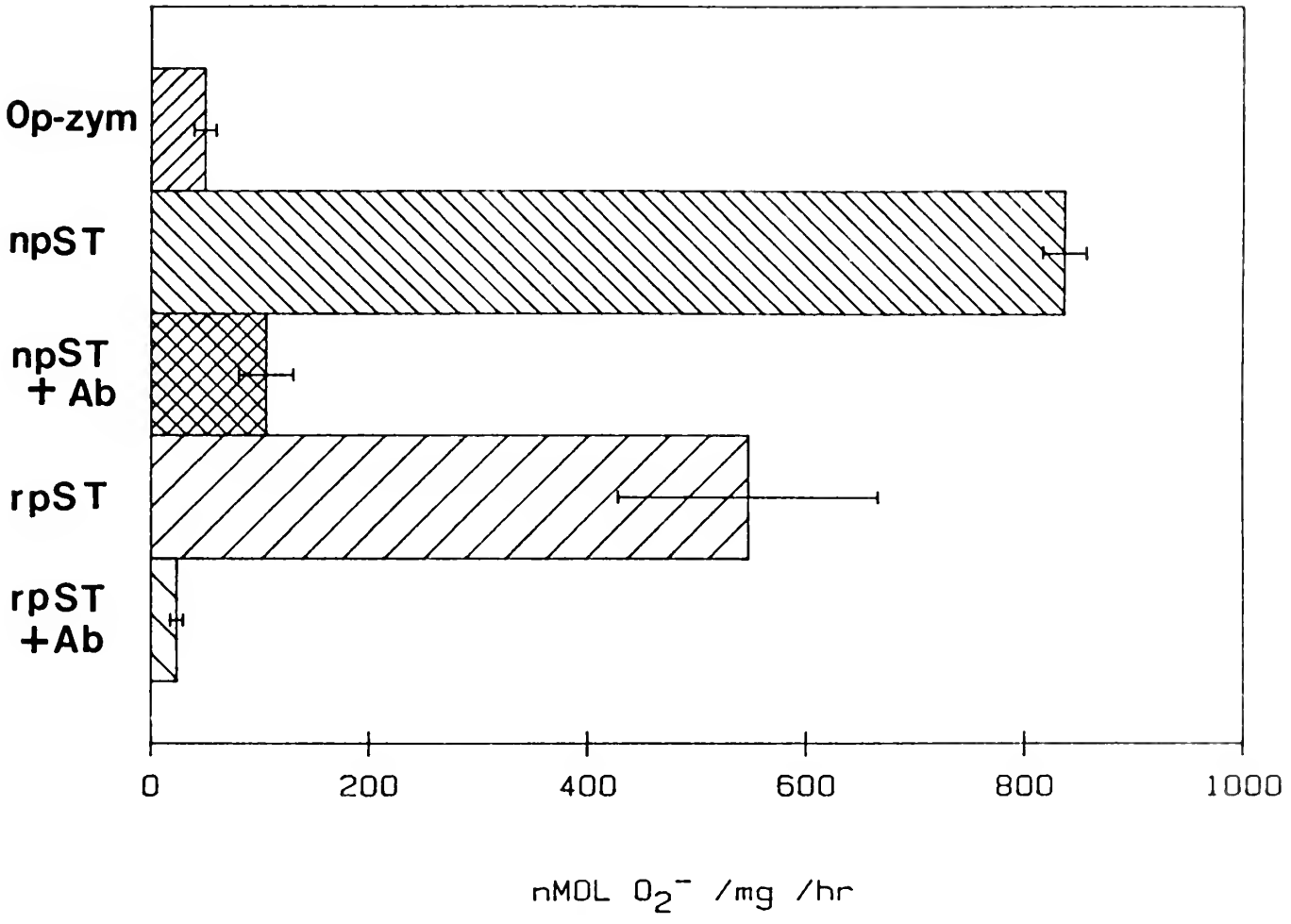


TABLE 1. Priming of porcine alveolar macrophages in vitro by npST and rpST.

Treatment	nMol O_2^-/mg protein/hr	SEM
Unstimulated	28[a]	14
Stimulated with Op-Zym	199[b]	48
Op-Zym + Superoxide Dismutase	28[a]	9
Op-Zym + Con A supernatant	418[c]	76
Op-Zym + npST (500 ng/ml)	430[c]	90
Op-Zym + rpST (500 ng/ml)	431[c]	81
Op-Zym + rpST + ST antibody	48[a]	20
Op-Zym + ST antibody	141[a,b]	30

Means with different superscripts are different at ($P < .05$).

TABLE 2. npST, rpST and native, pituitary-derived rat somatotropin (nrST) induce respiratory burst activity in rat peritoneal macrophages in vivo.

Item	Number	Dose/ Rat/Day	Growth (G/Day) Mean	SEM	nMol O_2^-/mg protein/hr Mean	SEM
Hypox Rats	6	-	0.392[a]	0.08	-40[a]	20
+ Vehicle	11	200 ul	0.282[a]	0.05	-17[a]	7
+ Rat IFN-γ	11	500 Units	0.252[a]	0.06	417[b]	79
+ npST	5	6 ug	1.322[c]	0.09	0[a]	19
	6	12 ug	1.633[d]	0.07	268[b]	71
	5	24 ug	2.060[e,f]	0.05	438[b]	70
+ rpST	6	6 ug	0.815[b]	0.03	-62[a]	14
	6	12 ug	1.067[b,c]	0.07	280[b]	59
	6	24 ug	1.297[c]	0.07	344[b]	42
+ nrST	5	12 ug	1.850[d,e]	0.11	3[a]	4
	5	24 ug	2.320[f]	0.10	267[b]	31
	5	48 ug	2.870[g]	0.10	247[b]	16
	5	96 ug	3.440[h]	0.10	309[b]	22

Means within a column with different superscripts are different ($P < .01$), as assessed by Duncan's New Multiple Range Test.

The Effect and Practical Implications of Recombinant Porcine Growth Hormone on Lactation Performance of Sows

Dean Boyd

Currently accepted standards for 21-day weights of nursing pigs are 12-14 pounds with an average littersize of 8.5-9.5 pigs or more. Some producers routinely achieve an average of 13-14 pounds, whereas, others consistently average 11-12. Differences in performance between herds can be accounted for, to a large extent, by factors which effect milk production. These factors include genetic merit of the sow, nutrition and level of feed intake, body condition prior to farrowing, parity and environmental temperature. To our knowledge, the upper limit on pig weight by 3-4 weeks of age is unknown, but appears to be much greater than we are presently achieving. Methods which enhance weaning weight, but do not compromise subsequent reproductive performance of the sow, could make an important contribution to adaptability and performance in the nursery, especially where pigs are weaned at younger ages (eg., 14-24 days).

It is commonly reported that the level of milk production (in a "good" milking sow) largely satisfies progeny needs through the initial 18-21 day period. To our knowledge, there is no substantive data which demonstrate that milk production, even in high producing sows ($\geq$ 24-26 pounds of milk/day), satisfies and maximizes growth potential of 3 week old pigs. In fact, several lines of evidence argue against this popular belief and for much greater 21-day weights. For example, pigs display signs of dissatisfaction for the quantity of milk available after 7-9 days of age even when the sow is producing at a high level. Artificial rearing studies, where pigs were weaned shortly after birth and fed a suitable complement of ingredients and level of solids, demonstrate that average 21-day weights of 16-19 pounds are routinely achievable(1,2). It is particularly noteworthy that these weights were attained on a program in which intake remained slightly restricted.

Several innovative approaches could be developed to assist producers in achieving heavier weaning weights. An approach which involves both a marked enhancement of lactation performance (interfaced with an effective strategy for sow feeding) and takes advantage of a sound creep-feeding program could result in weaning weights which more closely approximate the potential. Growth hormone has been shown to effect marked increases (25-40%) in milk production in high producing dairy cows without compromising body condition(3). It plays a coordinating role by chronically altering metabolism so that more nutrients are partitioned to "high priority" processes (eg., milk synthesis, growth)(4,5).

This paper summarizes research that we have conducted with recombinantly derived porcine growth hormone (r-pGH)(6). It demonstrates that we can simultaneously alter the pattern and level of milk production of sows; thus allowing us to increase the weaning weight of progeny through a sow-directed program. Also, some preliminary data is presented on the potential benefits of heavier weaning weights in "early" weaned pigs. This research is timely in that technological developments are such that metabolic hormones could become available commercially.

METHODS

Sixteen crossbred (Yorkshire X Duroc) multiparous sows were used to determine the effect of recombinant porcine growth hormone (r-pGH) on milk yield and composition, weaning weight of the pigs and plasma concentrations of GH and certain metabolites. Parturition was synchronously induced ($PGF_2\alpha$ and oxytocin) and litters standardized to 10 pigs each within 36 hours of farrowing. Subcutaneous injections of either placebo (excipient) or r-pGH (8.22 mg/sow/day, 1.35 IU/mg protein activity; donated by Amercian Cyanamid Co., Princeton, NJ) were given at 1000 hours daily beginning on day 12 through day 28 of lactation. Milk yield, milk composition and average pig weight was determined on days 16, 22, and 28 of lactation. Pre-treatment measurements were acquired on days 9 and 10 to provide a baseline and to be used in covariate analysis of the data. Milk yield was estimated using the weigh-suckle-weigh method described by Lewis(7), but modified to encompass a 6 hour time period(8). Sows were fed a corn-soybean meal diet (16% protein) ad libitum, and feed intakes were determined weekly. Sow weight and 10th rib backfat depth were determined at the beginning and end of the treatment period. Daily rectal temperatures were taken to monitor sow health and to detect any possible adverse reactions to r-pGH. Three of 8 sows/treatment were cannulated and hourly blood samples acquired at the beginning and end of the experiment to determine the average daily circulating concentration of GH and the effect on glucose, free fatty acids (FFA) and urea nitrogen.

LACTATION RESPONSES TO PORCINE GROWTH HORMONE

Data in figure 1 presents the pattern of milk production for sows throughout the 28-day lactation. Pretreatment milk yields (days 9 and 10) were similar for control (17.7 lbs/day) and r-pGH sows (17.2 lbs/day). Control sows maximized production at 3 weeks (19.6 lbs/day) and remained constant thereafter. Sows receiving r-pGH produced 10% more milk 4 days after the first injection (day 16 -- 20.0 *vs* 18.2 lbs/day) than their control counterparts. Milk production continued to increase throughout lactation in r-pGH sows which resulted in differences of 11% (21.8 *vs* 19.6) and 22% (24.2 *vs* 19.9) on days 22 and 28, respectively. Although the treatment X time effect was not significant, r-pGH appeared to alter the shape of the lactation curve so that more milk was available "early" and increased throughout commensurate with expanding needs of the progeny.

Relative increases in total yield of milk constituents (table 1, $P < .06$) were even more impressive than milk yield. Total yield of solids, fat and lactose were increased for sows receiving r-pGH by an average of 24%, 29% and 30%, respectively. Protein yield was not different (+7.5%). The increase in milk components were largely due to the effect of r-pGH on milk yield since percentages of milk components were not significantly different for treatment groups. The only exception was lactose, which, on day 28 which was greater ($P < .01$) for sows receiving r-pGH.

Progeny weight gain paralleled increases in milk production (table 2). Pigs from sows receiving r-pGH consistently weighed more than those in the control group with the 0.75 lbs/pig difference being significant ($P < .05$) on day 28 of lactation. Approximately forty-five percent of the variation in gain was accounted for by the quantity of milk and solids

produced. This compares well with estimates in the literature, for "early" weaned pigs, and emphasizes the importance of other factors such as health, environment and genetic ability to grow(9).

Both groups of sows consumed approximately the same amount of feed during the first two weeks of lactation (table 3). Sows receiving the placebo increased their feed-intake throughout lactation commensurate with increasing milk production. In contrast, those receiving r-pGH maintained approximately the same level of intake during weeks 3 and 4 as consumed during the second week, despite the marked increase in milk production. This resulted in an average difference between treatments of 22% ($P < .01$). As a consequence of the more negative energy balance, sows receiving r-pGH lost more ($P < .05$) weight (15.4 vs 29.9 lbs) and backfat (.12 vs .37cm). No adverse health effects were observed as a result of treatments.

The difference in feed intake makes it impossible to estimate the true potential of r-pGH to enhance milk production and weight gain of progeny. A study is being conducted to determine the cause of the feed intake response when r-pGH is given. A pair feeding component is included in order to determine the magnitude of response potentially available.

Daily injections of r-pGH increased the average daily circulating concentration of GH 2.3-2.7 fold above baseline values at the beginning and end of the treatment period, respectively. Circulating concentrations of plasma glucose and urea nitrogen were not effected by r-pGH treatment. In contrast to an earlier study by our group (pituitary derived pGH)(10), no acute changes in FFA concentration were observed in sows receiving r-pGH. Thus, mobilization of FFA coincident with 'normal' plasma glucose levels appear not to be the potential mechanism whereby feed intake is limited.

Associated with an r-pGH mediated increase in milk production, is a dramatic increase in nutrients required for milk synthesis. Growth hormone apparently altered nutrient availability (eg., mobilization of adipose lipid) so that plasma concentrations remained constant while the flux rate for milk synthesis was greater. Administration of growth hormone has been shown to chronically alter tissue responsiveness to a variety of homeostatic signals so that nutrients are repartitioned to support a highly productive state(11). In a recent long-term study, a coordinated increase in feed intake occurred in cows receiving bovine growth hormone so that energy balance was similar to that in the control group(12). However, the increase in feed intake did not occur until after several weeks of treatment. It remains to be determined whether a similar effect can be demonstrated in sows.

PRACTICAL IMPLICATIONS FOR PROGENY PERFORMANCE

Increasing 21-24 day weights by 2-2.5 lbs/pig would appear to be both of practical importance and achievable through enhanced milk production. This need not compromise subsequent reproductive performance of the sow if the "method" does not adversely affect feed intake and(or) if a good feeding strategy is in place. Results and behavioral observations from a recent study demonstrate that we have underestimated how rapidly an increase in milk production can be accommodated by the young pig

(7-8 vs 12-14 days as anticipated in this study) even if the sow is producing at a high level (eg., ≥ 24-26 lbs/day). It was determined that the maximum average hourly intake, for supplemented sow-reared pigs, ranged from 60-80 grams/hour/pig (10-12 days old, 18-20% solids) compared to the 35-40 grams achievable by better producing sows at that point in lactation (Ashley, Boyd and Cahen--unpublished data). This is equivalent to approximately 32-42 lbs of milk/day (10 pigs/litter) which is considerably higher than normally achieved, but is not surprising in view of the potential growth to 21 days of age.

Practical implications of increasing weaning weight where pigs are weaned at a younger age (eg., 14-24 days), potentially include easier adaptability to the nursery and, as a result, accelerated growth during the initial 10-14 days. The rationale is that pigs are potentially moved along the weight continuum so that proportionally fewer are small. This generally affords a considerable advantage in initial nursery performance which can impact greatly on marketable weight at the conclusion of the nursery phase. A second potential benefit is that total consumption of the preweaning diet may increase since pigs would be heavier prior to weaning. Both growth potential and appetite appear to exceed what could be satisfied with an increase in milk production alone.

Data is presented in table 4 which summarizes the effect of increasing weaning weights by approximately 3 lbs prior to weaning at 21-23 days of age (Welch, Boyd and Cahen--unpublished data). This difference was created by supplementing sow-reared pigs with a commercial milk replacer (20% solids) beginning on day 7. The difference was increased further to 5.4 lbs/pig after 14 days and slightly more (6.0 lbs) by the end of the 28 day period. From these studies, it is evident that earlier implementation of a method which markedly enhances milk production would potentiate greater differences in gains of nursery pigs.

We estimate that a 30% increase in milk production, during the 7-24 day period of lactation, would result in a 2.1 pound increase in weight per pig. This would be equivalent to approximately 18.9 pounds for an average littersize of 9 pigs. Since litters are sometimes weaned at an earlier age (eg., 16-18 days of age) and are at a distinct disadvantage, an increase in weight could confer a much needed advantage in nursery performance. An estimated difference of 1.3 pounds/pig could result if weaned at 18 days. This assumes an efficiency of .90 : 1 for conversion of milk solids to weight gain of progeny. Increasing milk production early in lactation could result in a greater gain response than predicted, however, since the efficiency of litter gain from milk has been reported to be considerably better (3.84, 7-14 days) than observed for 18-25 day old pigs (5.75).

The factorial method of estimating nutrient requirements was used to predict the effect of increasing milk production (+30%) on energy balance of the sow (table 5). This level of increase was selected since it's a realistic achievement and could produce a commercially important increase in weaning weight. Assumptions for the efficiency of energy transfer are given in the tabular footnotes. Increasing milk production by

this magnitude would require an increase in feed intake of 3.2 lbs/day in order to prevent lactation energy balance from becoming more negative.

REGULATION OF NUTRIENT USE - IMPLICATIONS FOR THE SOW

The concern that increased milk production will lead to greater fat depletion and further compromise subsequent reproductive performance fails to recognize the possibility that some agents which effect a repartitioning of nutrients and(or) alter tissue metabolism may also result in greater feed intake. There are genetically superior sows which produce greater quantities of milk early (eg., 7-10 days) and throughout lactation without suffering unusual health or reproductive problems provided they are properly fed in gestation and lactation. Also striking is the range in feed intake by sows and quantity eaten by many of the superior sows during lactation.

The biological controls which differ in these superior sows are not known, however, they must orchestrate physiological processes in an integrated manner to allow the sow to utilize a greater quantity of nutrients for milk synthesis. Components of this coordination would include greater utilization of body reserves in early lactation and increased feed consumption. This is best illustrated by examples with dairy cattle where genetically superior dairy cows such as Beecher Arlinda Ellen (25,300 kg/365 days) and Maplegrand Rockman Meadow (24,152 kg/360 days) achieved substantially greater levels of milk production than by the "average" cow without adverse effects on health and reproduction. The important issue is to understand the biology of the highly productive state and then determine management and environmental factors which best accommodate the level of production. The decision to undertake this area of research for commercial application must be based on the economic value of anticipated benefits.

Nutritionally related reproductive problems (eg., delayed return to estrus) are not evidence of reaching the physiological limit but rather that the feeding program isn't accommodating the productive state. They are largely a function of mismanagement during lactation and(or) gestation and represent a failure on the part of many to properly feed a higher producing sow. The first parity female may represent a special consideration which necessitates care in both litter size nursed and length of lactation. Again, the dairy cow is instructive in that periods of tissue loss occur to support a high level of production (eg., early lactation) without detriment, provided that tissue "reserves" are replenished during another part of the cycle. A sound feeding program, for the high producing sow, ensures full feeding during lactation and gives considerable emphasis to the gestation component (particularly where environmental conditions reduce lactation consumption). The gestation program must focus on meeting nutrient requirements of the conceptus, which become significant only in the latter part, and on re-building tissue used during the previous lactation.

Insufficient data exists to predict the energetic efficiency (and economics) of additional tissue gain during gestation if enhanced milk production results in a more negative energy balance. Assuming that feed energy is converted to body tissue with approximately similar efficiency as milk and that

inefficiency due to turnover would be minimal if replacement occurred in late gestation, additional inefficiency may largely be due to the subsequent conversion of tissue to milk [estimated at 85%(13) which is comparable to dairy cow(14)]. Tissue loss would likely be largely fat since protein concentration of the diet could be varied with level of milk production.

SUMMARY

In 1952, Hammond reviewed the physiological limits to animal production(15). He pointed out that, with each major improvement in productive efficiency (of dairy cattle), some have expressed the concern that these practices may be pushing the animal too far, thus compromising animal health and shortening productive life span. Hammond did not share that view and, indeed, substantial increases in productive efficiency of dairy cows have occurred during the past three decades.

We estimate that swine producers achieve approximately 60-66% of the potential weaning weight at 3-4 weeks of age. It is apparent, from our studies, that the level of milk production even in a high producing sow fails to fully accommodate progeny after 7-8 days of age and that lactation, thereafter, is a limit feeding program. Also, marked differences exist between sows in their ability to consume and partition energy in support of lactation. Striking differences were evident in the level of milk production achieved in early lactation (eg., 9-10 days) and, as a result, progeny weight at 14-16 days.

Our data with r-pGH demonstrates that an effective means of shifting the lactation curve and markedly increasing milk production is possible. This research emphasizes a "sow-directed" approach (since affinity of the pig for the sow is so strong) combined with a good creep feeding program. It could provide a much needed facet for an early weaning program (ie., 14-24 days). It is conceivable that average weaning weights could be increased by 1.5-2.5 pounds/pig from enhanced milk production alone, depending on when it is implemented and age at weaning. Further advantages are postulated to be derived by better adaptability and performance in the nursery during the early period (ie., 10-14 days) when performance is relatively poor. The net effect may be to shift some feed cost from the pig to the sow and to spread sow maintenance costs over greater production.

REFERENCES

1. Corley, J. and D. S. Pollmann. Central Soya Research, personal communication.
2. Jeppeson, A. E. App. Anim. Ethology 7:359. 1981.
3. Bauman, D.E., P. J. Eppard, M. J. DeGeeter and G. M. Lanza. J. Dairy Sci. 68:1352. 1985.
4. Bauman, D. E. and S. N. McCutcheon. Ch. 23 In Proc. VI Int. Symp. Ruminant Physiol. 1985.
5. Etherton, T. D. Research Report, Penn State Univ., p. 103, 1985.
6. Harkins, M., R. D. Boyd, D. E. Bauman and W. R. Butler. J. Anim. Sci. 61(Suppl. 1-Abstr):316. 1985
7. Lewis, A. J. and V. C. Speer. J. Anim. Sci. 37:104. 1973.
8. Speer, V. C. and D. F. Cox. J. Anim. Sci. 59:1281. 1984.
9. Lewis, A. J., V. C. Speer and D. G. Haught. J. Anim. Sci. 47:634. 1978.

10. Spence, C. A., R. D. Boyd, D. E. Bauman and W. R. Butler. J. Anim. Sci. 59(Suppl. 1):254. 1984.
11. Sechen, S. J., S. N. McCutcheon and D. E. Bauman. J. Dairy Sci. 68(Suppl. 1-Abstr.):146. 1985.
12. Verstegen, M. W. A., J. Mesu, G. J. M. vanKempen and C. Geerse. J. Anim. Sci. 60:731. 1985.
13. A.R.C. The Nutrient Requirements of Pigs. Commonwealth Agricultural Bureaux, Slough, England. 1981.
14. van Es, A. J. H. Livestock Prod. Sci. 1:21. 1974.
15. Hammond, J. Brit. Agr. Bull. 4:222. 1952.

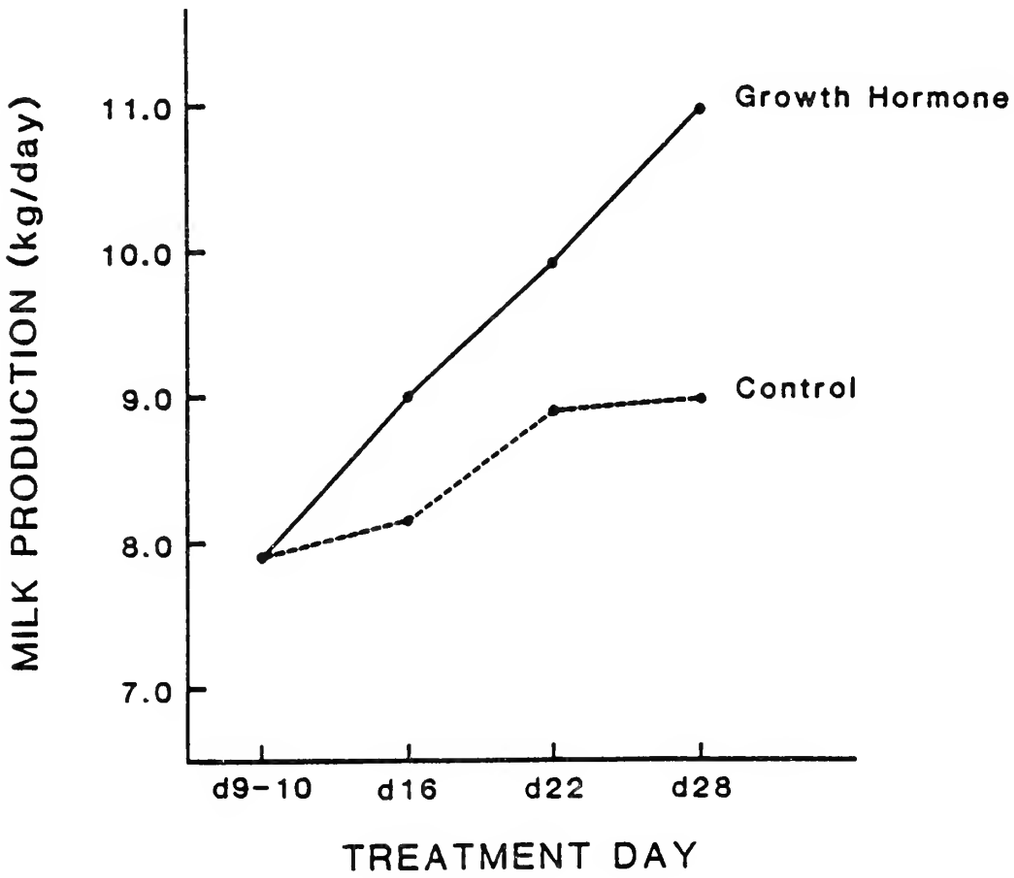


Figure 1. Pattern of milk production for sows receiving a placebo (excipient) or r-pGH from days 12-28 of lactation. Each point is the mean of 8 sows.

Table 1. Effect of r-pGH treatment on milk yields

Variable	16		22		28	
	Control	GH	Control	GH	Control	GH
Milk yield (lbs/da)[a]	18.2	20.0	19.6	21.8	19.9	24.2
Fat yield (lbs/da)[b]	1.19	1.50	1.12	1.36	1.08	1.50
Protein yield (lbs/da)	.95	1.01	1.12	1.12	1.14	1.32
Lactose yield (lbs/da)[c]	.79	.95	.90	1.10	.79	1.19
Solid yield (lbs/da)[c]	3.26	3.85	3.50	3.87	3.52	4.36

[a]Treatments commenced on day 12 and continued through day 28 of lactation (8 sows/treatment). Treatments differ (P=.08); Day 28 (P<.01).

[b]Treatments differ (P<.06).

[c]Treatments differ (P<.05).

Table 2. Effect of r-pGH on the average weight of nursing pigs.

Treatment	Day of Lactation				Total Gain[a]
	10	16	22	28	
Control (lbs/pig)	6.11	8.80	11.13	13.57 [b]	7.46
r-pGH (lbs/pig)	6.13	8.95	11.68	14.34 [b]	8.21

[a]r=.60 between milk yield and pig weight gain over the treatment period

[b]Treatments differ (P<.05)

Table 3. Effect of r-pGH on feed intake.

Treatment[a]	Lactation Days 0-7	8-14	15-21[b]	22-28[b]
Control (lbs/sow)	68.4	76.8	89.3	95.0
r-pGH (lbs/sow)	72.8	77.4	67.8	75.9
Difference (%)	-	-	24	20

[a]Eight sows/treatment. Treatment period: days 12-28 of lactation.

[b]Treatments differ (P<.01).

Table 4. Effect of weaning weight on nursery gain[a].

Day	Treatment-Weight, lbs[b] Control	Supplemented	Difference
Weaning			
22	12.5	15.3	2.8
Nursery			
0	13.2	16.3	3.1
14	22.0	27.4	5.4
28 (ADG-lbs/day)	39.1 (.92)	45.1 (1.03)	6.0

[a]Preliminary data. Summary of 2 litters/treatment-18 control, 10 supplemented pigs.

[b]Pigs randomized and placed on litters 3-5 days after birth. Supplemented pigs had access to 2 glands each and were trained to drink milk replacer. Supplementation began on day 7 of lactation.

Table 5. Factorial estimate of energy needs for enhanced milk synthesis[a]

Factor	Performance level, lbs/day	
	Control-18.3	+30%-23.8
Maintenance energy, kcal ME/day[b]	5310	5310
Requirement for milk production, kcal feed ME/day[c]	15071	19600
Energy balance, kcal ME/day	20381	24910
Feed, lbs[d]	14.0	17.2

[a]Production level derived from figure 1 assuming 7-21 day period.

[b]Assumed 160 kg sow and maintenance requirement of 118 kcal/kg$^{.75}$(12).

[c]Assumed 560 kcal ME/lb of milk(13) and partial efficiency of .68 for conversion of feed energy to milk(12).

[d]Corn-soybean meal diet, 1452 kcal/lb.

A European Approach to the Use of Growth Promoting Agents

G. E. Lamming

INTRODUCTION

The European Economic Community is similar to the United States in that it contains agricultural regions which differ markedly in their climate, topography and soil type and as a result their food animal production systems show equal diversity. However in the northern sector of the EEC there is a wide band where both extensive and intensive animal production systems are an important segment of the agricultural economy. This applies especially in Germany, Denmark, Holland, Belgium, Ireland and the UK. The EEC is steadily moving towards self-sufficiency in several areas of food animal production both by an expansion of the volume of production and an improvement in efficiency.

Currently there are Community-produced surpluses of several food commodities including milk for liquid consumption and beef. Under the present price support which is an integral part of the Common Agricultural Policy (CAP) of the EEC, surpluses are purchased and stored by applying a floor price support, but this procedure is expensive in terms of commodity purchase and the cost of storage. Currently it is stated that about 70% of total EEC spending is required to support the CAP and this is a matter of intensive debate and public concern with agriculture being generally regarded as a bottomless pit into which money in the form of CAP support is cast. Consequently policies which may appear to lead to an increase in the total production of animal products are actively opposed by politicians.

Therefore, in the light of these facts extra productivity in food animal enterprises is not seen by the public as a worthwhile current objective, although increased efficiency of production and improvement in public health through the use of healthy diets is well accepted. In addition there is active political lobby of well orchestrated consumer organizations which are generally opposed to techniques which they believe lead to an intensification of animal production systems. They have considerable political influence at both the level of the

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European parliament in Strasbourg and at the EEC level in Brussels. These consumer groups are opposed to the use of pharmacological agents to influence animal production and in the face of this opposition and criticism of the high costs of the CAP, the Communtity's Ministers of Agriculture, who form the executive body concerning the acceptance of Community directives, have been reluctant to support the use of the so-called 'growth promoting' agents.

In addition, in the late 1970s there was a major incident when stilbene hormone residues were detected in supplies of Italian babyfood. This incident occurred in a country which, along with others, had banned the use of all hormone-type growth promoters. It was widely reported in the press and led to widespread public fears about the dangers of drug residues. Generally those countries of the EEC which had licenced the use of the accepted hormone products had little incidence of drug abuse, whilst those countries which had maintained a ban, even against the use of the natural steroid sex hormones, had evidence of abuse.

Political opposition to the use of sex hormone type growth promoting agents culminated in an EEC directive which bans their use as implants for growth promotion. It is finally due to be in force throughout the EEC by December 1987. The UK and Ireland have already imposed a ban on their use from December 1986. Currently the debate is embracing the potential use of genetically engineered growth hormone products for increasing the efficiency of growth and milk production and at the present time the prospective progress for their acceptance is not good. So far there has been little public awareness in Europe about the efficacy of the repartitioning agents although an EEC sponsored scientific meeting on the use of the ß-agonists was held in Brussels in May 1987.

In supporting an informed public debate it is essential that full information concerning the lack of drug residues and therefore an absence of danger to human consumer health is presented as well as full information concerning quality, safety and target animal health and welfare before licensing authorities are requested to authorize products for licensing. It is more difficult to remove a ban than to have it imposed by lack of information on the safety of drug residues.

HISTORY

On 31st July 1981 the Council of the European Economic Community adopted Directive 81/602/EEC concerning the prohibition of use for the purposes of improving animal growth of certain substances having a hormonal action. While all member states agreed to the ban on the use of the stilbene-type compounds and their esters, and the thyrostatic materials, they were unable to agree about the use of five specific substances, oestradiol-17ß (E_2), testosterone (T) and progesterone (P_4), trenbolone and zeranol, which were licenced for use in some member states.

The Council allowed these five substances pro tem under existing national legislation and, in order to obtain further information, established a Scientific Working Group of European experts in endocrinology, drug residue analysis and drug toxicology with terms of reference to report whether the use of these substances for fattening purposes presented any harmful effect to health.

In 1983 the Working Group presented an interim report (EUR 8913) on E_2, P_4 and T which stated that their use under licence did not present any hazard. The Group requested additional information concerning zeranol and trenbolone, and this information was supplied between 1983 and 1986 by industrial and other research laboratories, and has been made available to interested organisations and individuals.

In December 1985 following an overwhelming vote in the European Parliament in favour of a ban (117 for, 10 against), the Council of the European Communities adopted Directive 85/649/EEC which 'prohibits the use in livestock farming of certain substances having a hormonal action'. The directive prohibits the use of anabolic agents (E_2, T, P_4, trenbolone and zeranol) which had previously been licenced under veterinary prescription in the UK within the 1968 Medicines Act. The UK Minister responsible voted against the 1985 ban and subsequently the British Government has contested the validity of the ban in the European Court. The decision of the European Court is still awaited.

Following the vote of the European Parliament in October 1985 the Commissioner responsible for establishing the EEC Working Group suspended the committee and declined to allow it to meet to complete the report on zeranol and trenbolone. However, the majority of the scientists participating decided they wished to complete the task and in 1987, working as a group of individuals, finalized the report for presentation at the 1987 Annual Meeting of the British Veterinary Association, held in Warwick.

In framing its conclusions the Working Group had to determine whether the two xenobiotics, trenbolone and zeranol, could act as direct carcinogens or whether they may be tumorogenic by virtue of their hormonal activity. The Working Group thoroughly reviewed the extensive information from both short- and long-term toxicity studies undertaken and concluded that both compounds act in a hormone-line manner and that cells of the appropriate target tissue of target species have receptors to which these anabolic agents attach and interact. In the case of both compounds, a full pharmacodynamic profile is available and the Working Group decided that in view of their clear specific effects on hormone-dependent tissues, a no-hormone effect level (NHEL) could be determined. The use of NHEL for hormones and hormone-like substances is a new concept and the Working Group had to develop a framework for considering new evidence in this area. Their detailed evaluation of the concept and the evidence for trenbolone and zeranol has been documented in a report published (The Veterinary Record, Oct. 24th, 1987).

The report contains a full review of the mutagenicity and long-term toxicity studies of the two compounds and justification for selecting a suitable safety factor (i.e. x100 or x1000) in the calculation of a tolerable daily intake, i.e. the levels of drug and drug metabolite residues below which it is concluded that no hazard would occur following the consumption of meat from treated animals.

The NHEL for 17ß TBOH (trenbolone) was set at 1µg/kg and that for 17α TBOH its major metabolite 10µg/kg. Similarly the NHEL for zeranol was set at 25µg/kg and for taleranol and zearalanone its major metabolites 1mg/kg. Using the commonly accepted formulae a tolerable daily intake of the compounds and their metabolites was calculated and published in the report.

The available information on the parent compounds (trenbolone and zeranol) and their major metabolites indicate that, following good husbandry practise, the level of residues in edible tissues does not create any hazards to human health. By inference these levels are substantially below the hormonally active doses which are effective in the sensitive animal test systems used to determine the NHEL.

The imposition of a complete ban on the use of hormonal-type growth promoters in Europe might be justifiable on political grounds, but it will be logical only if it can be effectively enforced. Enforcement of the ban is not possible at the present time in the case of illegal use of the natural steroid hormones where they are administered to cattle, other than in the case of veal calves. This is because the residues resulting from such treatments are within or below the normal range of endogenous steroid hormones present either in intact or gonadectomized males and in normal intact females. In the case of the xenobiotics trenbolone and zeranol, monitoring of residues with the precision required for legal enforcement will be difficult and costly. It is, therefore, anticipated that there will be widespread abuse where undesirable orally-active compounds are injected into edible parts of the animal rather than via implants of orally less active materials placed in an inedible part of the animal (the ear) the preferred site and mode of treatment for existing licensed products.

It required a tremendous volume of work to establish the safety of residues of anabolic steroids, but this procedure could form the pattern for the future licensing of new pharmacological compounds.

CLIMATE FOR ACCEPTANCE OF NEW TECHNIQUES
(ß-agonists and genetically engineered somatotrophins)

If the European experience concerning the ban on anabolic steroids is taken as precedent, then the prognosis for rapid acceptance of the newer pharmacological agents to manipulate the pattern of growth is not good, unless there is a concomitant change in public attitudes. It

will be essential for commercial organizations contemplating submissions to licensing authorities for product licences for their commercial use to obtain all the information required on efficacy and quality of the product and on the safety to those handling the product and to the target species. In the case of safety to the consumer it will be essential to show that the residues of the compounds and their metabolites present no hazard; and for this, evidence of a full pharmacodynamic profile in both target and test species will be required.

There is considerable current interest in Europe on the potential use of (a) genetically engineered somatotrophins to influence the pattern of growth and (b) the ß-agonists as repartitioning agents.

In the case of genetically engineered somatotrophin preparations, much of the current scientific debate revolves around its use to influence the efficiency of lactation in dairy cattle and there is little published evidence available so far on the use of porcine or bovine somatotrophins for influencing the pattern of growth.

In the case of the ß-agonists, some information on efficacy and on carcase composition has been published (see review by Lamming & Peters, 1987).

In the case of both groups of compounds there is little information available for wider public scrutiny concerning their mode of action, their pharmacodynamic profiles or the levels of residues present in edible tissue. It would appear that safety to the target species following the long-term use of genetically engineered somatotrophin preparations will be a matter of concern, while with ß-agonists, the short-term cardiovascular effects may be a more important factor.

REFERENCES

Commission of the European Communities (1984) Report of the Scientific Veterinary Committee, the Scientific Committee for Animal Nutrition and the Scientific Committee for Food on the basis of the report of the scientific group on anabolic agents in animal production. EUR 8913, ECSC-EEC-EAEC, Brussels.

Lamming, G.E. et al (1987) Scientific report on anabolic agents in animal Production. Vet. Record, Oct 24th, 1987.

Lamming, G.E. and Peters, A.R. (1987) Future developments in the manipulation of growth in farm animals. Vet. Record, May 23rd, 1987.

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Global Implications of the Use of Repartitioning Agents in the Swine Industry

Steven Schmidt

I. WHERE ARE WE? GLOBAL SITUATION AND SHORT TERM OUTLOOK

Stagnating demand and excessive supplies of most commodities are still a feature of world commodity markets. There has been a tendency towards over-production in most commodities partly as a result of policies to support domestic producers.

Grains. The world grain situation in 1986/87 and 1987/88 is one of large supplies and low prices on world markets. In 1987/88 world grain production is expected to decline 66 million tons to 1.61 billion tons, reversing the trend of the 3 previous years.[1] Utilization of grain is forecast to expand by less than 1 percent to 1.65 billion tons. All grains, wheat, coarse grain and rice contributed to the fall in 1987/88's total grain output.

The world wheat harvest in 1987/88 is forecast by the U.S. Department of Agriculture to decline 4 percent from last year to about 506 million tons, but this will still be the third-largest harvest in history. A weather-related drop in the Soviet wheat harvest will account for nearly 14 million tons of the global shortfall. U.S. wheat production is up from last year to about 58 million tons. World wheat consumption is expected to be down around 10 million tons from the 1986/87 record of 520 million tons, due to lower feed use. With large stockpiles left over from previous years, there will be enough wheat in world grainaries to ensure continued intense competition among exporters and low export prices.

World rice production in 1987/88 is projected at 305 million tons, milled basis, more than 3 percent below 1986/87 output. This year's world rice crop is expected to be the smallest since 1982/83. The decrease is due to the adverse effect of the late, weak monsoon on the autumn crops. Thailand, the United States' major competitor in world rice markets, is expected to see production drop another 8 percent from

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last year's poor crop. Reduced world supplies of long-grain rice have caused a sharp run-up in world prices.

Global coarse grain production in 1987/88 is expected to be about 804 million tons, a 4 percent drop from last season. While U.S. production declined nearly 35 million tons, foreign output rose 3 million tons to a record. World utilization of coarse grains is projected to exceed production and result in a 10 percent decline in stocks. World corn production of 448 million tons will be down 6 percent from last year. U.S. production is down by about 28 million tons, but foreign corn production will probably set a record of 267 million tons in 1987/88, up about a million tons from last year. China accounts for most of the increase.

Another record world oilseed crop is in prospect for 1987/88. World oilseed production for 1987/88 is estimated at a record 202 million tons, including records for soybeans, rapeseed and sunflowerseed. Brazil and Argentina, the major U.S. competitors are likely to increase combined soybean acreage by nearly a million hectares while U.S. acreage fell below the previous years.

World vegetable oil production in 1986/87 was a record 47 million tons reflecting higher production of soybean and rapeseed oils. Global consumption growth at 2 percent is well below year-earlier gains despite a 12 percent increase in rapeseed oil consumption. In developing countries, use continues to lag output, despite lower production and higher consumption.

Global vegetable oil output in 1987/88 is forecast up 4 percent due mainly to increase in palm and cottonseed oil production. Total consumption is also forecast up 4 percent led by increased uses of rape, cottonseed and palm oils.

Livestock. World meat production has shown little change in 1987 as beef and pork production contract slightly while poultry meat output registers strong growth (Table 1). Production of red meat is projected to expand about 2 percent in 1988 to 107.1 million tons thanks to a 3 percent growth in pork production. World beef and veal production is expected to decrease in 1987 from its 1986 level and to continue declining into 1988. World production of lamb, mutton and goat meat in 1988 is forecast to follow the slow expansionary trend, that for a slight decline in 1986, has continued for more than 10 years.

World hog numbers at the beginning of 1988 are projected down slightly as sizable herd increases in the United States, Canada and the EC are being offset by a large reduction in the Chinese herd (Appendix Table 1). Pork remained by far the most important type of meat at the world level with China being the leading producer (Table 2). World pork production was down slightly in 1987 primarily because of a drop of Chinese production due to sow culling in late 1986. Weak demand for pork and a temporary deterioration in feed supplies accompanied by high feed prices led to the heavier culling. Pork output contributed

Table 1. World Meat Production

	1986	1987[1]	1988[1]
	-------Million tons-------		
Beef and veal	44.0	43.9	43.8
Pork	56.2	55.9	57.7
Sheep and goat	5.4	5.5	5.6
Total red meat[2]	105.6	105.3	107.1
Poultry meat	27.3	29.0	30.1
Total meat[2]	132.9	134.3	137.2

[1] Forecast.
[2] Totals may not add due to rounding.

Source: U.S. Department of Agriculture, FAS, World Production and Trade Weekly Roundup. WR36-87, Washington, D.C., September 10, 1987, p. 2.

Table 2. Pork Production and Consumption, Selected Countries

	Production[1]				Consumption[1]			
	1983	1986	1987[2]	1988[3]	1983	1986	1987[2]	1988[3]
	-------Million tons-------							
United States	6.9	6.4	6.4	7.1	7.1	6.9	6.9	7.6
Canada	0.8	0.9	0.9	1.0	0.7	0.7	0.7	0.8
North America	8.9	8.2	8.3	9.1	8.9	8.5	8.5	9.2
Central America	-	-	-	-	-	-	-	-
South America	1.1	1.1	1.4	1.0	1.1	1.1	1.3	1.0
EC	11.0	11.5	11.7	11.8	10.8	11.4	11.5	11.5
Eastern Europe	6.6	6.8	6.8	6.8	6.1	6.2	6.2	6.2
USSR	5.8	5.9	5.8	5.8	5.9	6.1	6.1	6.1
China	13.2	18.0	16.9	18.1	12.9	17.7	16.6	17.9
Taiwan	0.5	0.9	0.9	0.9	0.5	0.7	0.8	0.8
Japan	1.4	1.5	1.6	1.6	1.7	1.9	1.9	2.0
Asia	15.9	21.3	20.3	21.7	16.1	21.5	20.5	21.8
Oceania	0.3	0.3	0.3	0.4	0.3	0.3	0.3	0.3
World	50.8	56.2	55.9	57.7	50.3	56.2	55.6	57.4

[1] Carcass weight equivalent.
[2] Preliminary.
[3] Forecast.
- Less than 100,000 tons.

Source: U.S. Department of Agriculture, FAS, World Livestock and Poultry Situation. FL&P1-87, Washington, D.C., September 1987, pp. 33-36.

roughly 94 percent of China's red meat output in the past few years. Demand for lean pork meat has been continuously growing, particularly in big cities. Improved breeds have provided more lean meat to the markets, but still cannot meet the demand.[2] In 1988, Chinese production is projected to recover to levels similar to 1986, because the government is expected to improve supplies of low-cost feed to producers.

The EC had an increase in hog numbers and pork production during 1986 and 1987 because feed costs have been cut by the low international prices of oilmeal and nongrain feeds. Additionally, the co-responsibility levy on marketed grain has created incentive for feeding grain on the farm.[3] Pork production growth is likely to be slowed in 1988 by falling hog prices which may make production unprofitable despite low feed prices (Table 2).

Soviet pork production declined slightly in 1987 and because of the lack of growth in pig numbers production is projected to stay at about the same level in 1988. Consumption requirements will have to be supplemented by above normal live hog imports. In Eastern Europe, inventories of hogs have been declining in 1986 reflecting the effect of drought in the southern countries and inadequate profit margins for producers. Hog inventories were expanded in 1987 and are forecast to remain at about the same level in 1988. Pork production was record large in 1987 but is projected to fall slightly in 1988 as a result of reduced Polish pork production.

Pork production in the United States, which is up slightly in 1987, is forecast to expand 11 percent in 1988 (Table 2). Encouraged by low feed prices, the removal of the domestic slaughter tax in April 1987 and reduced Japanese minimum import prices, Taiwan's pork production is projected to be up in 1987 to 919,000 tons. For 1988, production is projected to remain at 1987 levels because of producer uncertainty about new water pollution control regulations to be applied after February 1988.

Poultry meat production is expected to total 29 million tons in 1987 and is forecast to expand about 4 percent in 1988 (Table 3). Substantial increases in broiler and turkey meat production in the United States account for much of the increase in the world total. In the EC, the world's second largest poultry meat producer, production continued its upward trend in 1987 and is projected to increase further in 1988. Weak domestic and export demand, however, are slowing production growth. Poultry meat production in Eastern Europe has grown rapidly in the 1980's and further growth is forecast for 1988. In the USSR, 1987 poultry meat production is estimated to have grown 7 percent and similar rate of growth is anticipated for 1988.

Japan's poultry meat production has shown a steady advance in response to increases in domestic demand. Brazil's poultry meat production too has registered a strong expansion fueled by increased

Table 3. Poultry Meat Production

	1986[1]	1987[2]	1988[2]
	-------Million tons-------		
United States	8.26	9.07	9.53
Brazil	1.68	1.90	1.96
Canada	0.63	0.68	0.70
EC-12	5.41	5.55	5.64
Hungary	0.45	0.48	0.49
USSR	2.90	3.10	3.30
Japan	1.42	1.45	1.48
Total	20.75	22.23	23.10
Others	6.55	6.77	7.04
World Total[3]	27.30	29.00	30.14

[1] Preliminary. [2] Forecast. [3] Total includes 48 countries.

Source: U.S. Department of Agriculture, FAS, World Production and Trade Weekly Roundup. WR36-87, Washington, D.C., September 10, 1987, p. 5.

Table 4. Beef and Veal Production

	1986[1]	1987[2]	1988[2]
	-------Million tons-------		
Canada	1.04	0.99	0.98
United States	11.29	10.81	10.33
Argentina	2.85	2.65	2.55
Uruguay	0.36	0.29	0.31
EC-12	7.98	8.03	7.79
Eastern Europe	2.51	2.48	2.44
USSR	7.70	8.10	8.50
Australia	1.48	1.42	1.46
New Zealand	0.47	0.52	0.50
World total[3]	43.98	43.86	43.83

[1] Preliminary. [2] Forecast. [3] Total of 51 countries.

Source: U.S. Department of Agriculture, FAS, World Production and Trade Weekly Roundup. WR36-87, Washington, D.C., September 10, 1987, p. 3.

demand for poultry meat to compensate for domestic shortages of red meat. Poultry meat production in the United States, the leading producer in the world, has advanced rapidly in response to favorable growth in domestic demand. Heightened emphasis on reducing the amount of fat in diets and continued expansion into the fast food industry is increasing poultry meat's popularity in the United States as well as in other countries.

The United States is the world's leading beef and veal producer but production has been declining in line with the decline in cattle numbers (Table 4). Similar trend is apparent in the EC but with a slight interruption in 1987 due to increased slaughter of dairy cattle under tighter EC dairy controls. Argentine cattle numbers and beef production were on the downtrend in recent years which is forecast to extend into 1988. High taxes and interest rates and uncertainty about government policy on beef prices have discouraged beef production. Since 1980 Soviet cattle numbers have been increasing with slight reversals in 1986 and 1988 resulting from forage shortages. To reach the beef production target for 1988 the Soviets will have to import live cattle.

World production of sheep and goat meat in 1988 is forecast to follow the slow expansionary trend that, except for a slight decline in 1986, has continued for more than 10 years. The USSR is the world's largest sheep and goat meat producer followed by New Zealand and Australia (Table 5).

The United States and China are expected to have large (7 to 8 percent) production increases in 1988, while most other major producing countries will have only slight increases.[4] Exports are heavily dominated by Australia and New Zealand. The EC and Japan account for more than three-quarter of the lamb and sheep meat internationally traded.

New Policy Orientation. In recent years the policies of the developed exporting countries have generally aimed at restraining production. The United States has continued its policy of production cutbacks through voluntary area reduction programs, linked to eligibility for income support benefits. The EC has also adopted a series of policy measures aimed at reducing cereal production in the 1986/87 season. The main measures have been the reduction in intervention prices, the introduction of co-responsibility levy and the adoption of regulations to discourage sales by farmers to intervention agencies. Australia and Canada have also taken steps which would reduce support prices, and Sweden has introduced a land fallowing compensation scheme for farms with over 15 hectares for 1987. Yet, despite the introduction of production restraining policies, current policies still provide a level of incentive which results in production well in excess of demand.

Table 5. Sheep and Goat Meat Production

	1986[1]	1987[2]	1988[2]
	-------Million tons-------		
Australia	0.58	0.62	0.60
New Zealand	0.61	0.61	0.62
Soviet Union	0.87	0.88	0.88
Total[3]	5.43	5.52	5.62

[1] Preliminary.
[2] Forecast.
[3] 31 countries.

Source: U.S. Department of Agriculture, FAS, World Production and Trade Weekly Roundup. WR36-87, Washington, D.C., September 10, 1987, p. 6.

WORLD TRADE IN GRAINS AND PORK

The major factors which have influenced commodity markets in recent years are cyclical factors - in particular the level of economic activity in the major markets, variations in exchange rates, technological developments and protectionism. The use of the world market as an outlet for domestic surpluses and the promotion of these with export subsidies have major implications for production and trade of agricultural products of less developed countries.

World Grain Trade Situation. World grain trade improved slightly in 1986/87 from the previous season's depressed level reflecting increased purchases by China, the Middle East and North Africa.[5] Early indicators point to a continuation of a moderately upward trend in 1987/88 due largely to expanded imports by the USSR and China. Imports by other countries are expected to grow only moderately. Production gains in some key importing countries, together with continued financial difficulties will constrain expansion with import demand. World grain utilization rose by 4.5 percent in 1986/87 and is projected to continue to increase, though at a slower pace in 1987/88. With production lagging behind utilization, global carryover stocks are expected to be reduced from the 1986/87 level, reversing an upward trend of the past three years. Projections place world grain stocks at 22 percent of total annual utilization slightly above the level generally considered adequate for world food security.

World wheat trade is forecast to expand 6 million tons in 1987/88 and world coarse grain trade by 2.6 million tons. The anticipated growth in world trade is based on expanded Soviet and Chinese wheat import requirements. A continued important variable affecting world wheat trade in 1987/88 is the level of feed wheat imports; they are dependent on price competitiveness of feed wheat offers by exporting countries. The U.S. expected to capture most of the growth in world wheat trade in 1987/88.

World coarse grain trade though expanding is not expected to reach the high levels recorded in the first half of the 1980's. Increased purchases by China, Eastern Europe, Egypt and Saudi Arabia are expected to more than offset reduced purchases by the USSR.[6] With smaller exportable supplies in several key countries, demand for U.S. corn could be particularly strong pulling U.S. coarse grain exports above the previous years level.

World rice trade in 1987 was down approximately 700,000 tons from the 1986 level of 12.7 million tons and for 1988 world trade is forecast to drop by another 1 million tons. Growth in world trade in rice will be restrained by tightening supplies in the rice producing regions of South and Southeast Asia due to hot dry conditions. Reduced export availabilities in the surplus producing countries notably Thailand is expected to translate into increased opportunities for U.S. rice exports in 1988.

Because of ample supplies, and in view of limited volume of purchases by importing countries, international cereal prices have fallen substantially and are at their lowest level in real terms in the last few decades. The decline in prices has been accentuated by intensified competition, with all exporters trying to maintain or even expand their shares in world trade. There have been increased use of export aids, including subsidies, export rebates, export credits and cuts in export duties. There has also been increased use of barter arrangements and counter-trade by a number of developing countries which, because of prevailing highly competitive conditions, were unable to sell in the commercial market.

World oilseed trade has continued an upward trend in 1986/87 after a three-year downtrend that ended in 1984/85.[7] In 1987/88 world exports of oilseeds are forecast to reach 35.7 million tons reflecting primarily changes in world rapeseed trade. World soybean exports in 1987/88 are expected to fall 1.4 million tons below the previous years level. The bulk of decline will likely be accounted for by the U.S. due to greater anticipated competition from South America, larger EC soybean output and reduced U.S. supplies.

World trade in protein meals is expected to remain at 1986/87 level of 35.9 million tons reflecting stagnancy in soybean meal exports. The outlook for U.S. soybean meal exports in 1987/88 is less bright because of the good supply of meal now available from Southern Hemisphere producers. U.S. soybean meal sales in 1987/88 are seen to fall 17 percent short of the previous year's level to be largely offset by higher Argentine exports.

World Trade in Pork. World pork imports rose by around 5 percent to 1.55 million tons in 1986 resulting from large purchases by the USSR and Japan (Table 6). This was mainly due not only to higher production levels and higher consumption in these two countries, but also to a generally low level of prices on international pork markets, largely explained by the depressed level of feed prices. In 1987 world imports dropped by 2 percent to 1.52 million tons as a result of smaller imports by Brazil and the EC. The increase in U.S. and Japanese pork imports was insufficient to compensate for the cutback by the two former countries.

The United States is the world's third largest importer of pork after Germany and the U.K. United States pork imports are expected to rise to 530,000 tons in 1987, due to a moderate increase in consumption, before declining in 1988 to 510,000 tons (Table 6). Canada and Denmark are the major suppliers. Imports of pork from Canada have increased since the imposition of a countervailing duty on hogs in August 1985. Purchases from Denmark are being assisted by high EC export subsidies.

Imports by Japan have, since 1986, strengthened as a result of a steady increase in consumption stimulated by lowered minimum import prices and favorable exchange rate for imports. Taiwan held a 40

Table 6. Pork Trade, Selected Countries[1]

	1983		1986		1987[2]		1988[3]	
	Imports	Exports	Imports	Exports	Imports	Exports	Imports	Exports
	-----Thousand tons-----							
United States	317	99	509	39	533	45	510	54
Canada	20	158	14	215	12	235	12	250
North America	338	258	524	254	547	280	523	305
Central America	0	0	0	0	0	0	0	0
South America	0	7	70	5	0	15	0	20
EC[4]	122	296	107	317	98	302	96	299
Eastern Europe	39	569	44	612	40	690	31	703
USSR	100	0	261	6	260	6	260	6
China	0	248	0	238	0	247	0	266
Taiwan	0	47	0	123	0	155	0	155
Japan	238	0	297	0	310	0	340	0
Asia	458	296	535	366	560	408	595	427
Oceania	2	5	1	4	1	4	3	4
World	2,860	3,293	3,596	3,634	3,623	3,882	3,595	3,975
4	1,075	1,516	1,554	1,628	1,522	1,750	1,521	1,805

1 Carcass weight equivalent.
2 Preliminary.
3 Forecast.
4 Excluding intra-EC trade.

Source: U.S. Department of Agriculture, FAS, World Livestock and Poultry Situation. FL&P1-87, Washington, D.C., September 1987, pp. 34-35.

percent share of the market in 1986 while the U.S. share was a mere 7 percent. Taiwanese pork enjoys a competitive advantage in Japan because of the low costs of production, processing and transportation.[8] Japanese pork imports are forecast to set a new record of 340,000 tons in 1988.

On a regional basis, Eastern Europe is the world's leading pork exporter accounting for over 39 percent of global exports in 1987 (Table 6). Shipments from Eastern Europe, however, fluctuated widely in the 1980's due mainly to the variability of Hungarian and Polish exports. The GDR is the leading East European pork exporter followed by Hungary and Romania. The bulk of East European exports are destined to Russia and the EC. Rising pork production in Eastern Europe since 1985 has allowed lower import levels, a trend which is expected to carry over into 1988.

Asia is the second ranking pork exporting region with China and Taiwan being the major suppliers. China exports more than 3 million slaughter hogs each year, as well as an equivalent amount (between 238,000 and 266,000 tons) of pork. Much of these shipments go to Hong Kong. Virtually all of Taiwan's pork exports go to Japan. Recently Singapore was made an export promotion target for pork, but lower market prices prevailing in this market and its distance from Taiwan may limit export growth. Taiwanese export levels in 1987 and 1988 are projected to be 155,000 tons, 25 percent above 1986 and more than double the quantity exported in 1984.

The EC is the third ranking regional pork exporter. Excluding intra-EC transactions, its exports have accounted for about 17 percent of world pork trade in 1987-88 (Table 6). The region's share of global pork exports has trended slightly downward since the mid-1970's. The Netherlands is the major EC pork exporter and Denmark the largest exporter to third countries. Pork exports from these countries to the U.S. were adversely affected by the weak dollar exchange and their exports to Japan face strengthened competition from Taiwanese and U.S. pork exports.

Canada is steadily expanding pork exporter supplying 13.4 percent of global exports in 1987. Destinations for Canadian pork exports include the U.S., Japan and the EC. The U.S. Canada's major market for pork absorbing over 86 percent of total exports in 1985 and 1986.[9] Most of the remaining exports went to Japan.

The U.S. is a relatively small exporter of pork and shipments have been declining since 1976, the peak export year, partly because of strong European and Canadian competition. The U.S. share of world pork exports is expected to be a mere 1.4 percent, less than one-third as large as its record 1976 trade share. The major export markets for U.S. pork are Japan, Canada, Caribbean and Canada. Based on the recent strength of the yen against the U.S. dollar, U.S. pork exports to Japan are expected to increase in 1988. Overall, a weaker U.S. dollar and a moderately expanding world economy may help stabilize or slightly

increase foreign demand for U.S. pork in the late 1980's and early 1990's.

Trade Restrictions. It seems doubtful whether significant liberalization of trade in food and agricultural products will be forthcoming in the immediate future, even under the current GATT trade negotiations. This is particularly the case in respect of cereals, meat, sugar, cotton, tobacco and vegetable oils. Notwithstanding international efforts to liberalize trade, protection in the livestock sector has grown. Not only have import barriers been raised, but subsidization of exports has also grown considerably. In recent years, approximately a quarter of total world meat exports has been subsidized. The combined effect of slack demand, continued rapid technical progress in animal production and, in particular, protectionist policies has been a long depression of prices in international trade.

If the EC, the U.S. and other developed countries continue to escalate export subsidization in order to protect or augment market shares, prices of these commodities will decline further. While lower prices for commodities on world markets are benefiting importers in developing countries, such benefits are likely to be only a short-term nature since artificially low prices for imports act as a disincentive to otherwise economically viable domestic production.

Although a modest increase in the GNP growth rates of the major trading nations will have a positive impact on commodity demand and prices, the large stocks and surplus production capacities now existing for many commodities will override this influence.

II. U.S. AGRICULTURAL EXPORTS

Overall Trend. U.S. agricultural exports declined from a peak of $43.3 billion in 1981 to $26 billion in 1986. Several factors contributed to this decline including the strong value of the dollar, large supplies available from competing exporters at competitive prices, increased self-sufficiency of many importing nation, lack of purchasing power in food deficit countries and slower growth in world consumption of farm products than in the 1970's.

U.S. agricultural exports are expected to mark a turnaround in 1987 and rise above the 1986 level. Tentative projections place 1987 total U.S. agricultural exports at around $28 billion, 7.6 percent above a year earlier but 35 percent below the 1981 record (Appendix Table 2). The lowered loan rates for U.S. crops and hence lower U.S. market prices, aggressive export promotion programs and a less expensive dollar are boosting world demand for U.S. farm products. These measures have reduced prices for many U.S. agricultural exports, stimulating sales. Constraining growth of U.S. exports are lack of foreign exchange, continued foreign debt, political instability and protective agricultural and border policies.

Grains account for virtually all of 1987's expected rise in volume as lower U.S. prices, reduced competitor supplies, and the Export Enhancement Program (EEP) increase the U.S. share of world grain trade.[10] However, with lower prices offsetting virtually all the expected volume gains in grain, increased U.S. high-value products and cotton account for most of 1987's expected gain in value.

Composition of U.S. Agricultural Exports

Grains and feeds are the leading U.S. agricultural exports accounting for around one-third of the total in recent years (Appendix Table 2). Oilseeds and products are the second largest export category making up between one-fifth and one-fourth of all agricultural exports. Animals and animal products contribute about 15 percent to agricultural exports and thus form the third largest export category. Horticultural products rank fourth in importance followed by cotton and tobacco.

The export value of U.S. animals and animal products totaled $3.6 billion during calendar year 1986 and the projected value in 1987 is expected to rise about 14 percent to $4.1 billion (Table 7). Nearly all of the major commodity groups, such as beef, pork, variety meats and poultry products are expected to contribute to the increase. Live animals and pork variety meats are the major groups that are projected to show declines.

Exports of red meats (beef, veal and pork) constitute the largest animal and animal products group contributing around one-fourth of the projected total. Of the projected $985 million export value beef and veal are seen to account for $845 million and pork for $120 million. Beef and veal exports are seen to gain 33 percent in value over 1986 and pork 43 percent (Table 7). Japan is the No. 1 market for beef and pork, accounting for nearly 70 percent of the value of U.S. red meat exports.

Exports of beef, veal and pork variety meats are expected to total $350 million in 1987 of which beef and veal are seen to contribute $300 million. The value of pork variety meat exports are projected to fall to $25 million in 1987 compared to $31 million in 1986. Japan, France, the United Kingdom, Canada, Mexico and Egypt were the top individual markets, purchasing over 80 percent of the total by value. The EC accounted for approximately 37 percent of U.S. pork variety meat exports in 1986. Japan is the largest single country market for U.S. pork variety meats.

Live animal exports were valued at $335 million in 1986 but are expected to decline to $325 million in 1987. Breeding animals make up about 40 percent of total shipments. Dairy breeding cattle are the largest export category followed by beef breeding cattle and breeding swine. Breeding swine exports in 1986 totaled around 13,000 head, valued at $9.2 million; a decrease of 29 percent in volume and gain of

Table 7. U.S. Exports of Livestock and Livestock Products

	1985	1986	1987	1985	1986	1987
	----Thousand tons-----			---Million dollars----		
Animals & Animal Products	N.r.	N.r.	N.r.	3,327	3,645	4,075
Meat & Meat Products	428	491	530	900	1,072	1,335
Red Meats	158	219	260	563	740	985
Beef & Veal	110	184	225	467	634	845
Pork	41	27	30	76	84	120
Variety Meats	248	250	250	298	332	350
Beef & Veal	190	202	200	248	290	300
Pork	46	34	30	39	31	25
Animal Fats & Oils	1,327	1,245	1,200	602	400	415
Lard	47	48	45	26	23	25
Live Animals	N.r.	N.r.	N.r.	338	335	325
Breeding[1]	107	81	83	130	134	126
Beef	42	22	20	49	32	20
Dairy	45	44	50	53	58	60
Swine	18	13	7	8	9	6
Bull Semen	N.r.	N.r.	N.r.	23	28	35
Poultry, Eggs & Products	N.r.	N.r.	N.r.	384	504	600
Chicken Meat	198	263	350	202	276	370

[1] Head.

Source: U.S. Department of Agriculture, FAS, Dairy, Livestock, and Poultry: U.S. Trade and Prospects. FDLP5-87, Washington, D.C., September 1987, p. 23.

14 percent in value compared with the same 1985 period. Major export markets were: China, 3,291 head; Japan, 1,882 head; Indonesia, 1,772 head; Mexico, 1,623 head; and Korea, 794 head.[11] China led the field with a market share of 25 percent, followed by Indonesia and Japan each with 14 percent, and Mexico with 12 percent. Breeding swine exports are expected to decline in 1987 to about 7,000 head valued at $6 million.

Exports of poultry, eggs and products in 1986 totaled $504 million, a 31 percent increase over 1985. Chicken meats totaling $276 million accounted for 55 percent of the export value of the poultry group. The bulk of chicken meat exports consists of chicken parts. The leading chicken parts market, with market share percentages in parentheses, are Japan (34), Hong Kong (16), Singapore (11), Jamaica (6) and Canada (5). The export value of poultry eggs and products is forecast to rise to $600 million in 1987 of which chicken meat is to account for $370 million (Table 7).

U.S. export promotion efforts have and will play an important role in bolstering the sale of several agricultural commodities. EEP is currently supporting sale of six livestock and poultry commodities to 15 countries.[12] The commodities include dairy cattle, poultry meat and eggs. Dairy cattle EEP's cover 65,500 head targeted to 13 countries (in North Africa, the Middle East and Indonesia), all relatively new markets for U.S. dairy cattle. EEP's for poultry meat has been put into place for four countries totaling 140,000 tons. Outstanding EEP initiatives for table eggs total 258 million eggs to three countries.

Horticulture exports are forecast to rise because of greater U.S. competitiveness in the market and added import demand in response to the weakened dollar. In addition, the USDA Targeted Export Assistance (TEA) Program is helping horticultural exports.[13]

U.S. SHARES OF WORLD AGRICULTURAL TRADE

There are many different concepts and definitions of competitiveness. In the strictest sense, competitiveness refers to the ability to win or achieve some goal. In agriculture, this typically refers to being able to outsell another nation in either domestic or foreign markets. The extent of competitiveness under this definition is measured as market share. Under this definition, competitiveness can be achieved through the use of subsidies and various governement policy interventions.

Agricultural Exports. The EC, excluding Spain and Portugal, as a trading bloc, was the world's largest agricultural exporter in 1985, with a 33.7 percent share (Table 8). If trade within the EC (intra) is excluded, then the EC, with an 11.8 percent share, was the second largest agricultural exporting region in 1985. Although agricultural export value for the EC decreased between 1980 and 1985, its world market share increased by one percentage point.

Table 8. Major Agricultural Exporters and Shares of Total, 1980 and 1985

	Share of Total Exports	
	1980	1985
	--------Percent-------	
United States	18.1	14.8
EC	32.5	33.7
EC - extra trade	10.8	11.8
Canada	2.9	3.4
Australia	3.7	3.9
Argentina	2.5	2.7
Brazil	3.8	4.5
New Zealand	1.4	1.5
China	1.2	1.5

Source: U.S. Department of Agriculture, ERS, FATUS, Foreign Agricultural Trade of the United States, March/April 1987, p. 132.

Table 9. Major Agricultural Importers: Shares of Total and U.S. Shares, 1980 and 1985

	Share of World Imports	U.S. Share of Imports	
	1985	1980	1985
	--------Percent--------		
Japan	7.4	34.3	31.7
Canada	2.0	40.3	34.8
Mexico	0.9	78.6	67.3
South Korea	1.3	54.5	45.9
China	0.8	38.9	8.9
USSR	7.8	5.8	10.6
Germany	9.1	7.3	4.5
Netherlands	4.6	29.3	17.8
Egypt	1.6	32.8	24.7
Brazil	0.5	27.5	37.0

Source: U.S. Department of Agriculture, ERS, FATUS, Foreign Agricultural Trade of the United States, May/June 1987, p. 140.

The United States while retaining its position as the world's leading agricultural exporting country over the past 8 years, has lost some of its world market share to the EC and other major exporting countries. The United States market share of world agricultural exports fell from 18.1 percent in 1980 to 14.8 percent in 1985, the lowest market share since 1970-72, because of falling export volume and prices of grains and soybeans. The U.S. market share reached its highest point (about 19 percent) during the world food crisis in 1973-74 and in the rapid growth period for world agricultural trade in 1980-81 when agricultural trade reached its peak of $233 billion, more than four times the 1968-72 level of $53 billion.

Agricultural Imports. Historically, the developed countries have been the major markets for agricultural products as well as the major source of food supplies. Although diminishing in importance since the early 1970's the developed countries still continued to be the major markets for agricultural products. Major growth markets for agricultural imports during the period were the developing countries, primarily the Middle East. The U.S. market share of world agricultural imports was down at 13.2 percent in 1985, the lowest level since 1970. Between 1980 and 1985 the U.S. market share of the ten largest agricultural markets fell in eight markets, increasing only in the USSR and Brazil (Table 9). The U.S. had the largest market share in Mexico (67.3 percent), South Korea (45.9 percent), Brazil (37 percent) and Canada (34.8 percent).

Grain Trade. The five leading exporters of wheat and flour (in wheat equivalent) accounted for about 84 percent of wheat export volume in 1985 (Table 10). The United States share of world wheat exports reached a peak of approximately 50 percent in the early 1970's and has been in an irregular downtrend until 1985. The 1987 U.S. share is expected to be about 35 percent. Competition in wheat exports in the last few years has been expecially strong from EC, Canada, Australia and Argentina. However, export shares obtained by these countries decreased slightly in 1987.

Import markets for wheat are highly concentrated in a few countries. Six countries accounted for over 50 percent of the world import volume in 1980-85. In recent years, the USSR and China have accounted for a third of world imports. The decline in U.S. market share between 1981 and 1985 was due primarily to declines in U.S. market shares in the USSR (from 21 percent to 5 percent), in China (from 58 percent to 21.8 percent) and in the EC (from 21.9 percent to 6.4 percent) (Appendix Table 3).

The U.S. share of global coarse grain trade fell to 44 percent in 1985 from a high of 71 percent in 1980 and finally rising to about 56 percent in 1986. The United States has traditionally been the world's largest exporter of coarse grains. Seven major exporters, including the United States, account for over 90 percent of total coarse grain exports.

Table 10. Major Wheat Exporters and Shares of Total, 1979-80, 1981-82 and 1985

	1979-80	1981-82	1985
	-------Percent-------		
United States	39.6	41.3	24.8
Canada	16.6	17.0	16.5
France	11.7	13.1	18.1
Australia	12.2	10.3	15.0
Argentina	4.9	3.6	9.2

Source: U.S. Department of Agriculture, ERS, FATUS, Foreign Agricultural Trade of the United States, March/April 1987, p. 133.

Table 11. Major Corn Exporters and Shares of Total, 1979-80, 1981-82 and 1985

	1979-80	1981-82	1985
	-------Percent-------		
United States	78.2	69.4	63.4
Argentina	6.1	9.6	10.1
France	4.0	3.7	6.5
Thailand	2.7	3.5	4.0
South Africa	3.5	5.6	0.1

Source: U.S. Department of Agriculture, ERS, FATUS, Foreign Agricultural Trade of the United States, March/April 1987, p. 134.

Table 12. Major Soybean Exporters and Shares of Total, 1979-80, 1981-82 and 1985

	1979-80	1981-82	1985
	-------Percent-------		
United States	81.5	86.0	66.3
Argentina	10.5	7.4	11.8
Brazil	4.0	3.5	13.7
China	0.8	0.4	4.3

Source: U.S. Department of Agriculture, ERS, FATUS, Foreign Agricultural Trade of the United States, March/April 1987, p. 135.

Corn is by far the largest coarse grain representing over two-thirds of the groups total. The United States accounted for 78.2 percent of world exports in 1979-80 and 63.4 percent in 1985 (Table 11). The U.S. is expected to recapture part of its lost market share in 1987 with about 69 percent of total corn sales. Other major corn exporters include Argentina, France and Thailand, which increased their market shares during the early 1980's.

The major markets for coarse grains have traditionally been in Western Europe and Japan. These markets are rapidly being replaced by markets in less developed and centrally-planned countries. Major corn markets in 1981-85 were Japan, the EC, the USSR, South Korea and Taiwan (Appendix Table 4). The U.S. market share in Japan fell from 89.3 percent in 1981 to 77.1 percent in 1985 and in the EC from a high of 71.1 percent to 25.4 percent. Only in the USSR and Taiwan has the U.S. market share increased over the same period.

Soybean Trade. The United States is the world's leading soybean exporter but its share of global trade declined substantially since 1981-82 to 66.3 percent in 1985 (Table 12). The decline in U.S. share is the result of increased exports by Argentina, Brazil and China. Contributing factors behind the decline in U.S. soybean export share are the strong U.S. dollar in the 1981-86 period relative to South American currencies and high U.S. price supports which, at times, restricted the competitiveness of U.S. soybeans. Preliminary estimates place the 1986 U.S. share at 72 percent.

The major markets for soybeans are the countries of Western Europe, Japan, South Korea, Taiwan and Mexico. The U.S. market share of the Pacific Rim countries has changed little over the 1981-85 period but declined in the EC and the USSR (Appendix Table 5).

Meat Trade. The U.S. is a minor source of beef and veal exports supplying 7.2 percent of the world's total in 1987 (Table 13). The U.S. share of world beef and veal exports has increased slightly over the past 15 years when its trade share was only 0.7 percent. The low U.S. share is due to the predominance of lower quality (grass-fed) beef products rather than the high quality (grain-fed) beef produced in the United States. Major beef exporters in descending order of importance are the EC, Australia, New Zealand and Argentina.

The United States is the world's leading beef and veal importer absorbing around one-third of total imports (Table 13). The EC ranks second and the USSR third as beef and veal importers.

Eastern Europe is the world's largest pork exporting area accounting for nearly 40 percent of world exports in 1987 (Table 13). The EC's trade position deteriorated since the mid-1970's and its share of global exports was down at 17.3 percent in 1987. During the same period, Canadian, Chinese and East European export shares have increased. The U.S. pork export shares reached a peak in 1976 and have

Table 13. Major Beef and Veal, Pork and Poultry Meat Exporters and Importers and Shares of Totals, 1983, 1986 and 1987[1]

Exporters	1983	1986	1987[2]	Importers	1983	1986	1987[2]
	-------Percent-------				-------Percent-------		
Beef and Veal				Beef and Veal			
United States	3.7	6.0	7.2	United States	32.9	32.5	33.6
Australia	22.6	20.1	20.5	EC[3]	13.8	14.8	15.1
EC[3]	14.7	29.0	27.0	Brazil	1.0	14.1	3.0
New Zealand	10.9	8.5	10.7	USSR	19.9	11.0	11.6
Argentina	12.2	6.3	7.9	Japan	7.4	8.4	10.2
Pork				Pork			
United States	6.5	2.4	2.6	United States	29.5	32.7	35.0
Eastern Europe	37.5	37.6	39.4	Japan	22.1	19.2	20.4
EC[3]	19.5	19.5	17.3	USSR	9.3	16.8	17.1
China	16.4	14.6	14.1	Hong Kong	19.9	14.2	15.1
Canada	10.4	13.2	13.4	EC[3]	11.3	6.9	6.4
Poultry Meat				Poultry Meat			
United States	16.9	21.3	25.5	Japan	9.2	17.8	19.6
EC[3]	34.3	26.3	24.9	Hong Kong	6.9	12.2	12.1
Eastern Europe	22.3	24.7	25.0	USSR	18.0	17.3	14.6
Brazil	21.7	17.5	15.4	EC[3]	7.2	9.1	8.6

[1] Shares expressed in world totals excluding intra-EC trade.
[2] Preliminary.
[3] Excluding intra-EC trade.

Source: U.S. Department of Agriculture, FAS, World Livestock and Poultry Situation. FL&P1-87, Washington, D.C., September 1987, pp. 16-19.

been declining gradually since, partly because of strong European and Canadian competition.[14] The U.S. share of world pork exports is estimated at 2.6 percent in 1987. By contrast, the U.S. is the world's largest pork importer, taking some 35 percent of total imports in 1987. Japan purchases about one-fifth of total world imports and its purchases are forecast to expand further in 1988.

The United States together with Eastern Europe and the EC are the major poultry meat exporters (Table 13). Among the major exporters, the United States and Eastern Europe, notably Hungary, showed significant export gains in 1986 and 1987 while the EC and Brazil both showed declines. The U.S. poultry meat export share is expected to approximate 25.5 percent in 1987. Japan, the USSR and Hong Kong are the biggest import markets for poultry meat, although the USSR's import share dropped somewhat in 1987.

III. DIRECTION AND ADVANCES IN BIOTECHNOLOGIES

Biotechnologies in Animal Production. Research thrusts presently underway which promise to have a significant effect on animal agriculture are:

1. gene insertion into reproductive cells of livestock and poultry, also known as recombinant deoxyribonucleic acid (rDNA) technology;

2. monoclonal antibody (MAB's)[15];

3. embryo transfer procedures.

These technologies have the potential to fundamentally modify biological processes in animal production allowing livestock producers to achieve in a few weeks what would take many years by traditional breeding programs. Application of new technologies will increase the feed and reproductive efficiency of farm animals.

In livestock production, the major application of rDNA technology involves the utilization of genetically modified bacteria to synthesize pharmaceuticals. Pharmaceuticals useful in the production of livestock and poultry include somatotroprin, prolactin, numerous vaccines, and immunity enhancers such as interferons and interlukins.

Protein synthesis in animals can be enhanced by the administration of recombinantly-produced porcine somatotropin (pST) or growth hormone and by feeding several chemicals called beta-adrenergic agonists or repartioning agents. Studies have found that both somatotropins and the repartitioning agents enhance the transfer of feed nutrients to muscle protein build-up and limit fat deposition in swine, cattle, sheep and broilers.

Improvement in performance, especially feed efficiency results from the repartitioning effect. Feed efficiency improvement result from the energy saved when depositing muscle instead of fat.[16]. Advances in feed efficiency are expected to become most significant in the production of pork and, to a lesser extent, in poultry.

Gene insertion into reproductive cells of livestock and poultry provide opportunities to improve animal health and productivity. Genes for disease resistance or growth. Gene insertion allows future animals to be endowed permanently with selected traits.

Bovine Growth Hormone (bST). The first commercial application of biotechnology to enhance productivity in agriculture is expected to occur in the dairy industry by 1989. The increase in milk production and feed efficiency with somatotropin supplementation has been well documented.

The use of bST is expected to boost milk yield per cow far above the 2.6 percent annual growth rate of the past 20 years. In controlled experiments bST has increased milk production in dairy cows from 10 to 40 percent in the middle and last third of their lactating cycle. This translates into annual gains of 6 to 25 percent.[17] Somewhat lower yield increases (10 to 25 percent) are likely to be realized in commercial herds. The actual yield increase is strongly linked to such factors as resources, management skills and weather.

Competent nutritional management will play an important role in the realization of full benefits of bST treatment. Diets need to contain high concentrations of energy and protein as well as good quality forage so that cows adjust for the increased milk production. Kalter, et. al., estimated that bST wil increase requirements for concentrates by 30 to 110 percent.[18] Availability of forage may be a problem in arid areas lacking irrigation.

Greater feed use is one apparent cost of using bST. The commercial application of bST will depend on whether the expected increased production profits offset the cost of using the technology.[19] The economic benefits from using bST are promising. The increase in returns for the farmers from the use of bST comes from three main sources: reduction in feed use per kilogram of milk, the dilution of fixed costs (since fewer animals have to be housed and handled) and a reduced labor requirement. Because fewer cows are needed to produce the same amount of milk, feed efficiency improves; total feed consumption of a herd could decrease an estimated 5 to 10 percent.[20] A smaller portion of feed goes toward maintenance of the herd and a larger share to producing milk.[21] With bST a dairy farmer can produce as much milk with 20 cows as with 35. Or alternatively, the dairy farmer has the opportunity of increasing total output without changing the size of his herd.

Apart from greater feed efficiency and lower production costs an individual producer will also consider the initial milk price in

deciding whether to adopt bST technology. As a result of increased supplies the price of milk is expected to decline in the short time, until it reaches the support price. Kalter and Milligan show that the equilibrium price of milk in the U.S. would fall between 11 and 16 percent as a result of the new technology under low and high productivity scenario assumptions.[22] The costs of acquiring and applying the hormone must be deducted from the returns. Currently, the market price of the product is unknown. Overall, net returns of dairyman using bST are expected to be greater than with no usage.

Biotechnologies in Swine Production. Swine related biotechnology research is currently focusing on changing the carcass composition of finishing pigs through 1. application of porcine somatotropin (pST); 2. feeding of chemicals called beta-adrenergic agonints or repartitioning agents; and 3. creation of better hogs through genetics. Genetics, however, may take many years to make changes. PST and repartitioning agents can make big changes now.

Research results at the University of Illinois, Cornell University and Purdue University demonstrate that pST plays a key role in regulating metabolism and in partitioning of absorbed nutrients between muscle and adipose tissue. McLaren, et. al. have shown that pigs treated with natural pST recorded a near 40 percent increase in feed efficiency and a marked increase in average daily gain.[23] At a dosage of 6mg per pig per day treated pigs grew 15 percent faster and reached marketing weight two weeks earlier than control pigs. Average daily feed consumption decreases (21.4 and 22.4 percent) with increasing dosage of either natural or genetically engineered pST treatment.

Boyd has found that the net effect of the optimum dose on the rate and efficiency of gain response was to decrease the time required to gain 55 kilograms by 9 days and decrease the amount of feed by 47 kilograms.[24]

Grebner, et. al. have shown a dose dependent repartitioning effect with muscle mass increasing 14-18 percent and organ weights 40 percent as pST dose increased.[25] Also there was a 58 percent decrease in the amount of fat deposits and a slight loss in carcass weight. Boyd's reasearch has also shown that pST cuts fat in hog carcasses by 55 percent which is equivalent to reducing backfat by 0.3-0.4 inches. The technique produced an extra 16 to 18 lbs of lean carcass worth an extra $15 to $20.

An alternative way to production of lean, meaty carcasses is genetic selection of both gilts and boars for growth rate and backfat. The ideal combination, of course, would be breeding for more lean, plus using pST.

Interest has also increased in the use of beta-adrenergic agonists or repartitioning agents. Feeding trials by Jones, et. al., indicated that feeding cimaterol to finishing pigs has improved the gain-feed ratio to a small extent without a change in rate of growth.[26]

Carcasses of pigs fed cimaterol had approximately 10 percent less fat and 10 percent more muscle. The repartitioning response may be diminished or even reversed rather quickly after the drug is removed from the feed during the withdrawal period prior to marketing. Also, at very high levels, the agent has been shown to cause an increased incidence of lameness resulting from hoof lesions.

Published data by Cline and Forrest indicate that pigs fed the beta-adrenergic agonist, Ractopamine gained faster, were more efficient and produced leaner carcasses than control pigs.[27]

Trials in broilers with cimaterol indicated 4.5 percent improvements in weight gains and 3.6 percent in feed efficiency.[28]

Trend Toward Leaner Meat. The health concern inspired trend toward reduced fat intake has important implication for the U.S. livestock and meat processing industries. Completely trimmed meat may not be too far off in the future. Stores already offer lean or extra lean meats along with choice cuts. Two poulty producers have already responded to changing consumer preferences and are marketing lower fat chicken. The new Frank Perdue chicken contains an average of 21 percent less fat. Holly Farm's 14 percent.[29] These poultry products will often sell for 20 cents to 25 cents per pound more than the product offered by other companies.[30] To meet consumer concerns with additives and antibiotics, the meat industry is looking at products free of these. Grand Union Company recently became the first U.S. supermarket to offer "natural beef" -- free of synthetic or chemical additives. The natural beef appears to be a marketing success, showing steady sales growth despite its 40 cents to $2 per pound higher price tag.[31] These emerging consumer preference trends is likely to spur the development of leaner breeds of cattle. At the retail level this means that the leaner beef may take some from regular beef and attract consumer who previously consumed little beef.

Consumer demand for reduced fat intake puts high priority on cutting fat content of pork. The emergency biotechnologies will permit greater control over the quality characteristics of livestock products in general and that of pork products in particular. Treatment of hogs with pST and repartitioning agents will allow the production of leaner pork which will make pork a closer substitute for poultry meat. Consumer acceptance will also be influenced by the cooking qualities and palatability characteristics of treated pork products. Experiments show that increasing dosage of pST tends to make the meat tougher than the meat from untreated pigs. Can scientists overcome this undesirable side effects?

A major consideration will be the price of pork relative to the price of poultry meat. The price advantage of poultry over pork has, so far, been an important factor in shifting consumption away from pork to poultry. It remains to be seen whether pork produced with the emerging technologies will allow the competitive pricing of pork with other meats?

Constraints on the Application of Animal Biotechnology. Apart from expected profitability considerations several major hurdles must be passed before the farm level application of biotechnology is realized in swine production. First, acceptance by consumers of products from hogs treated with the output and processes of biotechnology must be assured. Second, the regulatory process is perhaps the most important constraint to transferring the results of research on biotechnology to on-farm application. Regulatory authorities scrutinize the potential health and environmental hazards associated with practical on-farm use.[32] Since pST is completely digestible by humans, it should pose no threat to the health of humans. However, there is still uncertainty, according to Easter whether pST treated gilts are reproductively sound and would thus be able to produce normal litters. Also, it is not known whether pST has an impact on animal health.

The timing of farm application, following expected FDA approval, will be affected by the availability of a convenient delivery system. At the present time, the form of the delivery system is still in the experimental stages which makes the evaluation of prospective farm application difficult.[33] A product requiring less frequent injections or implanting may well speed up farm application. It should be noted that the pST is only one of a "first generation" of technologies and research continues on developing new products with superior qualities.

Animal scientists expect that cimaterol and other repartitioning agents may have earlier on-farm application than pST. The attractive feature of these products is their metabolizability and thus cleared from the animal's body very rapidly. Another advantage of these products is that they can be administered as feed additives, a convenience that can be appreciated by feeders.

The full realization of productivity enhancing potentials offered by the biotechnology will require reliance on information technology. Farm managers may be able to integrate computers into their overall operations. Computers can be used to collect and analyze information and operate automated feeding systems tailored to individual nutrient requirements. Overall, application of information technology allows improvements in the speed and accuracy of management decisions and will increase management efficiency. It, however, is likely to significantly increase the use of capital, both physical and human. On farm application of pST and repartitioning technologies are expected to occur between three to five years.

Biotechnologies in Crop Production. Because cereals contribute 52 percent of world protein production and 50 percent of energy, the potential impact of biotechnology on the world food situation may be larger in the production of cereals than in livestock. Advances in research aimed at crop improvement can enhance the feed efficiency of animal biotrechnologies as well as permit the introduction of animal agriculture in areas where hitherto lack of feed crops were limiting

factors.

Biotechnological research in crop production focuses on: 1. improving the nutritional and processing quality characteristics; 2. endowing field crops with natural resistance to herbicides, insect pests and diseases; 3. improving nitrogen-fixation of certain legumes (i.e. soybeans) and developing nitrogen-fixing capabilities of non-leguminous field crops (i.e. grains); 4. enhanced photosynthesis capacity; and 5. increasing crop tolerances against heat, aridity, soil salinity, frost and other environmental stresses. Quality characteristics sought include: 1. higher protein content; 2. oil crops with less saturated edible oils; 3. wheat crops with better milling and baking qualities; 4. barley crops with better brewing qualities; and 5. feed crops with higher nutritional values and better digestive qualities.[34] Increased heat, pest and disease resistance are among the priorities of less developed countries. The ability to manipulate the generic material of microbial organisms to produce natural product chemicals which protect or assist crop development is further advanced then is the technology to directly engineer the DNA of higher plants and animal. Work is underway to develop nitrogen fixing bacteria that could live on the roots of non-leguminous field crops such as wheat or corn. Elmination of the need for nitrogen fertilizers could reduce corn production expenses by more than 25 cents per bushel.[35]

Scientists are hopeful that in the future it will be possible to introduce any gene into plant cells, and regenerate an improved crop from the genetically modified cells. There are, however, still many problems to be solved before an optimum use of the potential of cells and genes of higher plants. One of the main problems faced by the genetic engineer is the identification of useful genes which could aid in the improvement of crops.[36]

According expert opinions biotechnologies mentioned earlier are not likely to become available before the year 2,000.[37] By that time, technical advances will allow some major crops to be altered genetically for disease and insect resistance, higher production of protein and self-production of fertilizer and herbicide.

Genetic engineering for crops is seen to result in significantly higher yielding varieties and varieties requiring quite different cultivation pratices. Also greater homogeneity will be achieved which require less sorting and grading. Moreover, the development of hardier crops will extend the geographical range of crops and provide farmers with wider crop choices. While farmers are interested in yield potential and better environmental adaptation, the cost of applying many biotechnologies will be high. Adoption of technolgoies will then ultimately depend on whether they raise net farm income. And this, in turn depends on the availability of cost-reducing biotechnical inputs and government price suport policies. Increased supplies on the market will result in the adjustment of prices and redistribution of income, affecting the welfare of consumers and producers.

IV. WORLD PRODUCTION CONSUMPTION OF LIVESTOCK PRODUCTS TOWARD 2000

Prospects for Total Meat. The projections were derived from the extrapolation of FAO generated commodity projections to 1990 to the year 2000.[38] Figures were modified in cases where the 1990 projections seemed inconsistent with actual 1986 production and consumption levels. Overall, these projections are based on the assumption that past trends in livestock yields and consumption will continue through the 1990's and also take into account the latest measures to limit milk production in the EC. Current government policies in the rest of the world and current relative prices were largely assumed to be constant.

World demand for meat is expected to increase rather slowly, 1.6 percent a year, between 1986 and 2000 (Table 14). Most of the rise in demand is likely to occur in the developing countries and the centrally planned countries. Continued increases in incomes, urbanization and the associated changes in diets will contribute to higher demand for meat. Response to changes in incomes is and will remain high in all developing regions (Appendix Table 6). The income elasticity of demand for meat is believed to increase fairly quickly as per capita incomes grow and then decrease again at higher income levels. Most of the higher-income developing countries are now entering this stage of high income elasticity for meat consumption. For the 1986-2000 period, meat consumption in developing countries are projected to grow at 2.7 percent per annum. With economic growth projected to be most pronounced in East Asia, this region is likely to see the fastest exansion in meat consumption.

In the developed regions where per capita meat consumption levels are already high and dietary concerns are widespread, demand in these countries is projected to grow more slowly, 1.1 percent a year (Table 14). Income elasticities of demand in most of these countries are approaching zero (Appendix Table 6). Only in the USSR and in eastern and southern Europe and in particular Japan there seems to be scope for increased consumption. In both regions, income elasticities are still relatively high and prospects for economic growth favorable.

Prospects for Pork. Pork should remain the principal meat in 2000, although its production and consumption (1.5 percent per annum) will be rising less than that of poultry (2.3 percent annum) and sheep and goat meats (1.9 percent per annum). The share of pork in total meat consumption and meat production should stay approximately the same at 42.2 percent while that of poultry meat should rise from 20.5 to 23.1 percent (Table 14). Sheep and goat meats are expected to increase their share in global meat markets from 3.8 to 4.4 percent.

For the rest of the century, pork production and consumption are projected to rise at annual rates of 0.7 and 0.6 percent in the developed countries and by 2.7 and 2.8 percent in the developing countries respectively. As a result of these disparate growth rates, the developed countries share in world pork markets is expected to decline from about 61.6 percent in 1986 to 54.2 percent in 2000.

Table 14. Meats: Prospects For Production, Consumption and Trade

	1986				Net	1990			2000		
	Production	Consumption	Exports	Imports	Trade	Production	Consumption	Balance	Production	Consumption	Balance
					- million tons -						
Beef and Veal											
World	43.9	43.4	4.0	3.0	1.0	46.1	45.8	0.3	50.7	50.8	-0.1
Developing c.	10.3	10.3	1.0	0.9	0.1	11.2	11.6	-0.4	13.0	14.2	-1.2
Developed c.	33.6	33.1	3.0	2.1	0.9	34.9	34.2	0.7	37.7	36.6	1.1
Pork											
World	56.2	56.2	1.6	1.5	0.1	61.4	61.6	-0.2	70.7	70.9	-0.2
Developing c.	21.6	21.6	0.4	0.3	0.1	25.3	25.4	-0.1	32.4	32.8	-0.4
Developed c.	34.6	34.6	1.2	1.2	-0-	36.1	36.2	-0.1	38.3	38.1	0.2
Sheep & Goat											
World	5.5	5.0	0.9	0.4	0.5	6.2	6.1	0.1	7.3	7.4	-0.1
Developing c.	2.0	2.0	0.1	0.1	-0-	2.2	2.8	-0.6	2.7	3.4	-0.7
Developed c.	3.5	3.0	0.8	0.3	0.5	4.0	3.3	0.7	4.6	4.0	0.6
Poultry											
World	27.3	26.9	1.3	1.0	0.3	30.5	30.3	0.2	38.6	38.5	0.1
Developing c.	6.0	6.1	0.3	0.4	-0.1	7.1	7.2	-0.1	10.3	9.7	0.6
Developed c.	21.3	20.8	1.0	0.6	0.4	23.4	23.1	0.3	28.3	28.7	-0.5
Total Meat											
World	132.9	131.5	7.8	5.9	1.9	144.2	143.8	0.4	167.3	167.6	-0.3
Developing c.	39.9	40.0	1.8	1.7	0.1	45.8	47.0	-1.2	58.4	60.2	-1.8
Developed c.	93.0	91.5	6.0	4.2	1.8	98.4	96.8	1.6	108.9	107.4	1.5

Source: U.S. Department of Agriculture, FAS, World Livestock and Poultry Situation. FL&P1-87, Washington, D.C., September 1987.
FAO, FAO Agricultural Commodity Projections to 1990, FAO Economic and Social Development Paper 62, Rome, 1986, pp. 100-106.
Projections to 2000 were derived by the author.

Demand for pork will continue to increase mainly in East Asia. Income elasticities for pork are estimated to range from 0.45 in Taiwan to 0.71 in South Korea.[39]

Prospects for Beef and Veal. Beef and veal production, consumption and trade are heavily concentrated in the developed countries accounting for 76.5 percent of production and for 76.3 percent of consumption in 1986. Their projected shares in 2000 are 74.4 and 72 percent respectively. Although in many developed countries beef is still the preferred type of meat, the expected further decrease in the relative price of poultry meat, and to a lesser extent pork, will probably lead to a continuing shift in consumption towards these cheaper meats. In North America, consumption growth will depend on population growth as per capita consumption is likely to remain at current levels. In the U.S. per capita beef consumption may actually decline. Overall, here and elsewhere health considerations of consumers are expected to continue to contribute to the shift from beef to poultry consumption. As per capita incomes of the developing countries and the centrally planned economies increase, these countries should account for an increasing share of beef consumption as well as production. China and some countries in Africa and Latin America have the potential for increased pasture-based production of meat from ruminant animals. Eastern Europe and the USSR are emphasizing cattle production from pasture and other roughage.

Prospects for Poultry Meat. The pace of growth in poultry meat production and consumption in the developing countries is likely to exceed those in the developed countries. Thus the share of developing countries in world poultry meat market is expected to rise to over one-fourth of the total by 2000. Especially the higher-income and more urbanized developing countries will probably continue to expand intensive poultry meat production based heavily on imported feeds. With domestic meat demand expected to grow slowly and self-sufficiency levels in deficit countries increasing any further expansion in poultry meat production in the developed countries will result mainly from a further shift in meat demand away from beef and possibly from mutton. The income elasticities of demand for poultry meat are higher than for any other meat category, in both developed and developing countries (Appendix Table 6).

Prospects for Sheep and Goat Meat. The second highest share of developing countries in world meat markets is in sheep and goat meats. By 2000 developing countries are projected to produce 37 percent of the world's sheep and goat meat and consume about 46 percent of the total. In the Near East demand for sheep and goat meat is likely to remain particularly strong.

Trade Implications. The production and consumption prospects discussed in the precedings suggest that in the developing regions meat consumption is likely to increase faster than production requiring import supplementation. The developing countries will change from the current state of meat self-sufficiency to a meat deficiency

approximating 1.8 million tons in 2000 (Table 14). Given the difficulties in speeding up the development of local beef production, developing countries were expected to have their largest deficit, 1.2 million tons, in beef and veal and the second largest deficits, 0.7 million tons in sheep and goat meat. Consumption of pork is likely to exceed production by 0.4 million tons while the reverse developments are expected for poultry meat. By 2000 the developing regions could have a 0.6 million ton surplus in poultry meat. Surpluses generated by the developed countries are expected to cover the bulk of developing countries' deficits in beef, veal and pork and, in turn, can meet their own deficits in poultry meat from the surpluses produced in developing countries.

Prospects for Milk and Dairy Products. World milk production has increasingly exceeded effective demand over the past 15 years leading to an accumulation of large stocks of dairy products, high governmental expenditures on milk support and generally depressed prices in international markets. Excess productivity has been a problem primarily of developed market economy countries in North America and Western Europe. Milk production in the developing countries has been insufficient to meet growing demand necessitating large import supplementation (Table 15).

Barring major policy changes and continuation of past productivity increases existing imbalances in dairy markets are expected to persist through the remainder of the century. Thus, while production in developing countries is likely to continue to lag behind the growth in consumption, the developed countries despite the introduction of production constraining measures will continue to have growing exportable surpluses. The bulk of milk surpluses are expected to be produced in the EC while Asia will likely remain the biggest deficit region. In the developing countries an increase in incomes and continuing urbanization will contribute to higher demand for dairy products. Response to changes in incomes will remain high in all developing countries. Hence, these countries will continue to have sizable import requirements, some of which will be coming in as food aid. At the present, in most countries of Western Europe and in Canada milk marketings are governed by quota systems. The U.S. curbs milk production mainly by cutting the support price.

V. GLOBAL IMPLICATIONS OF BIOTECHNOLOGIES

General Considerations. The extent to which the new biotechnologies could and/or will change the projected production, consumption and trade patterns for meat and milk is highly speculative. Government price policies and related profitability of production along with government regulations will determine the timing of on-farm application. Adoption thus will vary greatly by county. As of now it is not certain whether the emerging technologies will be low-cost and scale-neutral -- increasing efficiency for small farms as well as large. They appear, however, to be less capital intensive than most

Table 15. Milk: Production, Consumption and Net Trade by Regions

	1980			1990			2000		
	Production	Consumption	Net Trade	Production	Consumption	Net Trade	Production	Consumption	Trade
	------Million tons------								
Developing countries	109.0	125.8	-16.5	142.5	163.3	-21.2	186.0	211.1	-25.1
Asia	67.0	75.7	-8.7	86.9	99.6	-12.7	112.4	131.3	-18.9
Near East	15.6	19.7	-4.1	19.6	26.0	-6.4	24.6	34.3	-9.7
Far East	43.3	47.2	-3.9	55.7	61.6	-5.9	71.3	80.4	-9.1
Asian CPE's	8.1	8.8	-0.7	11.6	12.0	-0.4	16.7	16.4	0.3
Africa	7.6	11.2	-3.6	9.6	14.8	-5.2	12.2	19.5	-7.3
Latin America	34.3	38.8	-4.5	45.7	48.7	-3.0	60.8	61.1	-0.3
World	469.7	470.0	2.7	531.6	531.1	0	599.1	598.5	0
Developed countries	360.7	344.2	19.2	389.1	367.8	21.2	421.4	398.3	23.1
North America	65.9	71.7	6.0	74.6	71.9	2.7	84.9	78.6	6.3
EC	95.2	102.2	-7.0	115.3	103.1	12.2	116.4	104.1	12.3
E. Europe & USSR	116.5	131.8	-15.3	144.2	144.5	-0.3	159.3	158.1	1.2
Oceania	13.6	6.0	7.6	13.2	6.5	6.7	14.6	7.0	7.6
Japan	4.5	7.8	-3.3	8.1	9.1	-1.0	10.0	12.8	-2.8

Source: FAO, FAO Agricultural Commodity Projections to 1990. FAO Economic and Social Development Paper 62, Rome, 1986, pp. 109-111. Projections to 2000 were derived by the author.

other technologies that enhance productivity. In both plant and animal production information technologies will have to be used to exploit the potential benefits of new biotechnologies. In developing countries management skill required for the operation of information technology will likely become the major limiting factor and hence slow the pace and spread of application.[40]

Implications for Meat Markets. It has been shown that the costs of producing meat and milk can be lowered further in the developed countries through new biotechnologies allowing a corresponding reduction in prices. Calculations made by Kalter and Milligan indicate potential cost and price reductions in the range of 5 to 8.6 percent for beef and between 5.7 to 12.8 percent for pork depending on the magnitude of productivity and feed efficiency improvements.[41] Because of the relative inflexibility of marketing charges, the actual decline in consumer prices will be less than the potential decline at the farm level. Given the low own price elasticities of demand for meats and dairy products increases in consumption of these products will not be proportionate with price declines (Appendix Table 6). The quantity of any type of meat consumed will also be affected by the level and change in the price of other meats. This relationship is referred to as the cross-elasticity of demand for a product. As the size of coefficients in Appendix Table 6 indicate the cross-elasticity of demand or the degree of substitutability among the four types of meat is also relatively small. That is, lowering the price of a particular meat will not cause of a proportionate decrease in the consumption of other meats. The degree of substitutability is biggest between poultry and red meats.

Since the potential decline in pork prices is greater than that foreseen for beef, the growth in demand for pork is expected to continue to outpace that for beef. Demand for pork will receive a further boost from the reduction of fat content in pork products. Dietary concerns over fat intake will continue to prod consumers to shift to leaner meats such as lean pork and poultry meat. Consequently, pork and poultry meat are likely to benefit more from future increases in meat demand than meat from ruminant animals. Per capita beef consumption in developed countries is likely to grow only marginally or to decline as pork and poultry meats continue to make inroads.

It is interesting to speculate on the indirect effects that may result from the use of bST. Most obviously there will be a reduction in the number of calves available for beef production. The decline in beef production will hinge on the extent to which beef is produced from calves from dairy herds. In the EC, for example, 80 percent of beef is presently produced from calves from dairy herds, and the reduction of dairy cows would accelerate a trend to a beef shortage in the 1990's. Alternatively, higher market prices for beef could encourage the build-up of specialized beef breeding and finishing.

Implications for Dairy Product Markets. Consumption of milk and dairy products too have small price responsiveness; the price elasticity of demand ranges from -0.05 to -0.35 percent for butter and from -0.05 to -0.41 percent for cheese (Appendix Table 6). Given the saturated state of developed country markets, lower prices will not boost consumption enough to absorb the prospective supplies coming to markets. And, in developed countries, income elasticities of demand for a number of dairy products have for some time been around zero or even negative.

In the EC attitudes to bST are split between those who see it as providing benefits to both producers and consumers and those who see it as yet another destablizing factor in a market burdened with oversupplies and who therefore prefer to prohibit its use.[42] Those favoring use of bST expressed concern that limiting such innovations on the basis of economic or social criteria, e.g. where there are milk surpluses, would diminish the competitiveness of European dairy industry. At the same time, production facilities for bST would be placed outside Europe and the innovation climate for all agricultural biotechnology would be damaged.

The EC Commission is currently studying both the health and economic effects of bST and similar products in order to determine what action may be necessary at Community level. The substance has already been under trial in five member countries -- France, the U.K., Italy, Germany and the Netherlands during the last three years and has shown its potential for consistently boosting dairy farm incomes.[43] The use of bST raises an important policy question. With productivity increasing, for example, there is no justification for continuously raising the milk target price, rather it can be lowered.

Other Implications. There seems to be an emerging interest in the development of a new type of carcass grading system that assesses lean muscle content and thus provides a basis for rewarding producers for extra lean meat. The technology exists (electromagnetic or ultrasonic devices) to make such a direct assessment of lean meat in hogs but is still in the pioneering stage.

Research with repartioning agents has shown that they are more effective with higher levels of protein in feed. This will likely decrease the use of corn in hog rations while increasing the demand for soybeans. Such change in feed use may impact on the grain market.

Biotechnologies may offer an opportunity for increasing the international competitiveness of countries that make first use of them. Enhancing U.S. competitiveness ranks high among national agricultural policy priorities and advances in emerging technologies will play a key role in the realization of this goal. Biotechnologies are also making significant advances in the creation of entirely new systems of production, yielding novel foods. Biosynthesized micro-proteins have the texture, taste and nutrient value of chicken or red meats, but contain no fats. New foods created with micro-proteins or plant-based

proteins will pose a growing challenge to foods based on animal products.

VI. U.S.-EC CONFRONTATION OVER HORMONES AND HYGIENE REGULATIONS

The Hormone Ban. The scare over hormones began in 1980 when consignments of meat-based infants' food in Italy were found to be laced with large amounts of hormone diethylstilbestrol, giving rise to rumors that babies were growing breasts and oversized genitals. This led to calls for a ban on five hormones (ternbolone acetate and zeranol, and the natural hormones testosterone, progesterone and oestradial 17-beta) used in the fattening of beef cattle and other animals. The hormone ban was approved by the EC Council of Ministers in December 1985. The majority of the 12 member states support the ban while the United Kingdom, backed by Denmark, has led the opposition. Although fundamentally opposed to the ban the United Kingdom has nevertheless implemented it already. The EC has determined that U.S. controls on the use of hormones and our system for monitoring residues is not acceptable. This regulation is of particular concern to the United States because our law does not permit the banning of such substances unless there is reason to question their safety. The EC directive also involves a very complex and expensive surveillance and residue testing program.

The United States has argued that the ban is politically motivated because there is no scientific evidence that the use of licensed hormone-based products pose risk to consumers' health. And, the U.S. position was confirmed in a report by members of the working group set up by the EC Commission to report on the safety of hormones. In an interim report published in 1982, the working group had said that residues of three naturally-occurring hormones, progeterone, testosterone and oestradiol, in meat were not harmful to the consumer. The group has worked out acceptable daily intakes for the above two groups. To consume these levels of hormones a person would need to eat at least 5-7 kilograms of treated meat per day, but even rates of consumption at 100 times these levels would not necessarily be dangerous, the group stressed. Members of the committee were critical of the EC ban, which they felt would lead to considerable abuse of illicit hormones. They also expressed worries over the cost of testing for hormone use in carcasses and live animals. These findings are expected to support the U.S. and the drug companies who are pressing for the ban to be repealed.

The United States has taken action through the General Agreement on Tariffs and Trade (GATT), which administers the world's trade rules, to cancel the implementation of this ban scheduled for January 1, 1988. The U.S. maintains that the requirement that meat be certified as coming from animals which have not been treated with hormones violates the GATT accord on technical barriers and would unjustifiably adversely affect U.S. trade interests. The United States has initiated a dispute settlement process under the GATT's Standards Code. The EC Commission

argues that the ban is non-discriminatory because it covers all meat sales within the Community and thus is not a legitimate target for action. If no compromise solution is found by January 1, 1988, the U.S. Administration has made it clear that it will take retaliatory action -- with effect January 2 -- against what it sees as a barrier to U.S. meat exports. At stake are U.S. sales to Europe of offal meat and other processed products worth $135 million a year. No specifics of what these repraisels might entail have yet been released by U.S. officials. The likely targets are farm products and selected industrial goods.

It is probably that the EC will postpone its scheduled January 1 imposition of a total ban on imports of U.S. beef products on condition that the U.S. agrees to limit the use of hormones on beef intended for export. Recently EC agriculture ministers appear to have agreed that hormone-treated meat will still be able to be sold in the Community after January 1 -- provided that it is from animals which were treated before the end of December. Bearing in mind the production period of beef cattle, this suggests a "transition period" to completely hormone-free beef in the EC of at least a year from January 1. National legislation to allow the ban to come into effect has still to be completed in France, Spain, Belgium and Portugal.

Hygiene and sanitation regulations. The new EC "Third Country Meat Directive" on standards for abattoires is another point of dispute with the United States. This legislation would also severely limit the U.S. meat into the Community, as only seven American slaughter plants out of the 400 reviewed by EC inspectors were certified.[44] Some 59 other slaughter plants are approved only until December 31, 1987, when they must be reinspected. The problem stems from EC unwillingness to accept the equivalency of U.S. inspection standards. The U.S. meat trade and producer organizations backed by government officials, has protested that the new standards are unnecessary and are designed purely as non-tariff restrictions to imports.[45] They have jointly filed a Section 301 petition against the Directive, July 14, 1987. If this issue cannot be resolved between the U.S. and the EC, the petition would be decided by panel of the GATT. The EC has always maintained that, as the slaughterhouse measure is nondiscriminatory since they apply equally to Community and third country meat producers, they do not contravene GATT rules.

Appendix Table 1. Hog Inventories, Selected Countries[1]

	1983	1986	1987[1]	1988[3]
	----------Million head----------			
United States	54.5	52.3	51.2	55.5
Canada	10.1	10.7	10.8	11.3
North America	81.1	76.1	74.3	78.2
Central America	1.1	1.1	1.1	1.1
South America	38.1	35.7	37.2	38.6
EC	94.4	100.9	104.0	105.9
Eastern Europe	70.6	73.1	74.9	74.9
USSR	76.7	77.8	79.4	78.0
China	300.8	331.4	336.9	329.3
Taiwan	5.2	6.7	7.0	7.1
Japan	10.3	11.1	11.3	11.6
Asia	326.4	359.3	365.6	359.2
Oceania	2.9	3.0	3.1	3.2
World	701.5	736.4	749.1	748.6

[1] January 1.
[2] Preliminary.
[3] Forecast.

Source: U.S. Department of Agriculture, FAS, World Livestock and Poultry Situation. FL & P1-87, Washington, D.C., September 1987, p. 31.

Appendix Table 2. U.S. Agricultural Exports of Selected Commodity Groups

	1985	1986	1987	1985	1986	1987
	----Thousand tons---			---Million dollars--		
Animals & Animal Products	N.r.	N.r.	N.r.	4,150	4,494	4,075
Dairy Products	466	471	N.r.	432	442	475
Fats & Oils	1,345	1,256	N.r.	619	411	N.r.
Hides & Skins	N.r.	N.r.	N.r.	1,295	1,304	1,350
Grains & Feeds	86,928	70,795	N.r.	11,882	8,622	9,500
Fruits & Preparations	1,416	1,600	N.a.	997	1,148	N.a.
Nuts & Preparations	473	460	N.a.	683	743	N.a.
Vegetables & Preparations	1,365	1,569	N.a.	930	1,081	N.a.
Pulses	410	520	N.a.	187	250	N.a.
Oilseeds & Products	23,672	29,285	N.a.	5,793	6,464	6,200
Tobacco	249	214	200	1,520	1,209	1,200
Cotton	1,097	657	1,500	1,633	773	1,700
Sugar & Tropical Products	716	940	N.a.	515	614	900
Total Agricultural Products	125,700	109,500	129,000	29,041	26,046	28,000

N.r.: Not relevant - unlike units
N.a.: Not available

Source: U.S. Department of Agriculture, ERS, FATUS Foreign Agricultural Trade of the United States Calendar Year 1986 Supplement. Washington D.C., May 1987.

U.S. Department of Agriculture, FAS, Dairy, Livestock, and Poultry: U.S. Trade and Prospects. FDLP5-87, Washington, D.C., September 1987.

Appendix Table 3. U.S. Share of Major Wheat Markets, 1980, 1981 and 1985

	World	Japan	South Korea	USSR	China	EC	Egypt	Brazil
				-------Percent-------				
1980	37.9	57.9	100.0	11.3	58.2	22.2	31.5	37.5
1981	43.3	60.7	95.0	20.9	58.0	21.9	44.1	77.3
1985	25.4	58.2	66.7	4.9	21.8	6.4	14.3	56.1

Source: U.S. Department of Agriculture, ERS, FATUS, Foreign Agricultural Trade of the United States, May/June 1987, p. 141.

Appendix Table 4. U.S. Share of Major Corn Markets, 1980, 1981 and 1985

	World	Japan	USSR	South Korea	Taiwan	Mexico	EC	Spain
				-------Percent-------				
1980	79.3	90.9	54.5	100.0	82.0	100.0	67.7	97.8
1981	68.3	89.3	39.2	96.1	58.6	100.0	71.1	94.2
1985	64.4	77.1	72.5	49.0	99.3	89.6	25.4	67.6

Source: U.S. Department of Agriculture, ERS, FATUS, Foreign Agricultural Trade of the United States, May/June 1987, p. 142.

Appendix Table 5. U.S. Share of Major Soybean Markets, 1980, 1981 and 1985

	World	EC	Japan	Taiwan	Spain	Mexico	South Korea	USSR
				-------Percent-------				
1980	80.5	79.9	96.1	100.0	66.3	92.3	100.0	29.3
1981	83.1	85.4	95.7	100.0	78.8	55.5	100.0	2.1
1985	66.7	57.6	88.6	95.2	59.2	76.2	100.0	0

Source: U.S. Department of Agriculture, ERS, FATUS, Foreign Agricultural Trade of the United States, May/June 1987, p. 143.

Appendix Table 6. Demand Elasticities With Respect to Own Price: Beef, Pork, Mutton and Lamb, Poultry Meat

	U.S.	Canada	EC	Japan	Australia N. Zealand	Eastern Europe	USSR	Mexico	Brazil	Argentina
					Beef					
Demand	-0.08	-0.19	-0.05	-0.20	-0.54	-0.12	-0.05	-0.09	-0.05	-0.43
Cr. with Pork	0.04	0.09	0.02	ml 0.10	0.05	0.06	0.01	0.01	-----	-----
Income	0.09	0.41	0.29	1.00	0.87	0.00	0.04	0.98	0.44	0.90
					Pork					
Demand	-0.56	-0.46	-0.09	-0.44	-----	-0.70	-0.05	-0.08	-0.05	-----
Cr. with Poultry	0.30	bf 0.16	-----	0.20	-----	0.30	-----	bf 0.04	-----	-----
Income	0.01	-0.01	0.35	1.00	-----	0.00	0.70	0.55	0.23	-----
					Mutton and Lamb					
Demand	-0.70	-1.00	-0.33	-0.29	-0.14	-0.28	-0.05	-----	-----	-0.70
Cr. with Beef	0.31	0.42	pk 0.10	pk 0.10	0.07	0.14	-----	-----	-----	pm 0.30
Income	0.01	-0.20	0.25	0.55	0.73	0.00	-0.20	-----	-----	-0.20
					Poultry Meat					
Demand	-0.30	-0.20	-0.53	-0.70	-----	-0.10	-0.70	-0.24	-0.38	-0.30
Cr. with Beef	0.03	0.09	pk 0.20	-----	-----	-----	0.19	0.11	0.15	0.20
Income	0.35	0.45	0.64	1.00	-----	0.00	1.00	1.00	0.83	1.00
					Butter					
Demand	-0.08	-----	-0.05	-----	-0.35	-0.12	-0.05	-----	-----	-----
Cr. with sb oil	-0.23	-----	-----	-----	ch 0.16	-----	-----	-----	-----	-----
Income	0.12	-----	0.14	-----	-0.20	1.00	1.00	-----	-----	-----
					Cheese					
Demand	-0.05	-0.28	-0.12	-0.29	-0.41	-0.06	-0.13	-0.15	-0.05	-0.05
Cr. with Beef	-0.07	0.10	dp 0.05	-----	bu 0.20	-----	-----	0.07	dp 0.01	-----
Income	1.00	1.00	0.55	1.00	0.50	0.10	1.00	1.00	0.51	0.89

Source: Karen Liu and V.O. Roningen, The World Grain-Oilseeds-Livestock (GOL) Model, A Simplified Version. U.S. Department of Agriculture, ERS Staff Report No. AGES850128, Washington, D.C., February 1985, pp. 38-62.

ENDNOTES

1. U.S. Department of Agriculture, FAS, World Crop Production. WCP-9-87, Washington, D.C., September 1987, p. 8.

2. U.S. Department of Agriculture, ERS, China Situation and Outlook Report. RS-87-8, Washington, D.C., July 1987, p. 25.

3. U.S. Department of Agriculture, ERS, Western Europe Situation and Outlook Report. RS-87-7, Washington, D.C., June 1987, p. 14.

4. U.S. Department of Agriculture, FAS, World Livestock and Poultry Situation. FL&P1-87, Washington, D.C., September 1987, p. 39.

5. U.S. Department of Agriculture, FAS, World Grain Situation and Outlook. FG-9-87, Washington, D.C., August 1987, pp. 7 and 28.

6. U.S. Department of Agriculture, FAS, World Grain Situation and Outlook. FG-9-87, op. cit., pp. 9 and 19.

7. U.S. Department of Agriculture, FAS, World Oilseed Situation and Market Highlights. FOP8-87, Washington, D.C., August 1987, pp. 10-15.

8. U.S. Department of Agriculture, FAS, World Livestock and Poultry Situation. FL&P1-87. op. cit., p. 7.

9. Agriculture Canada, Market Commentary March 1987. Ottawa, Canada, March 1987, p. 90.

10. The Food Security Act of 1985 established the Export Enhancement Program (EEP). The EEP enables exporters to sell specific commodities to targeted countries at prices available from other exporters. The Commodity Credit Corporation (CCC) awards bonuses to exporters in the form of generic certificates which can be exchanged for CCC Commodities t make up the difference between the price offered by other exporters and the unsubsidized U.S. price.

11. U.S. Department of Agriculture, FAS, Dairy, Livestock, and Poultry: U.S. Trade and Prospects. FDLP1-87, Washington, D.C., February 1987, pp. 16 and 27-28.

12. U.S. Department of Agriculture, FAS, Dairy, Livestock, and Poultry: U.S. Trade and Prospects. FDLP5-87, Washington, D.C., September 1987, p. 10.

13. The Targeted Export Assistance (TEA) was authorized by the Food Security Act of 1985 to assist export promotion programs of U.S. producers groups disadvantaged by unfair trade policies of competitor nations. Under the program, the CCC provides generic commodity certificates to the producer groups which may be redeemed for CCC commodities or sold to help finance market

promotion. At least $110 million a year will be spent during fiscal years 1986-88.

14. The World Food Institute, World Food Trade and U.S. Agriculture, 1960-1986. Iowa State University, Ames, Iowa, October 1987, p. 26.

15. MAB's are proteins recombinantly manufactured involving the fusion of cells. Use of MAB's includes immunization of calves and hogs against scours, detection of diseases, sexing of livestock embryos, and monitoring the level of hormonal drugs.

16. A unit of fat contains twice the calories of a unit of protein. Consequently, animals fed repartitioning agents are more efficient at cnverting feed to weight gain.

17. T.A. Stucker, R.F. Fallert and K.L. Lipton, "Bovine Growth Hormone Brings Progress to Dairy Farms." U.S. Department of Agriculture, National Food Review, NFR-35, Fall 1986, p. 13.

18. R. J. Kalter, et. al., A.E. Research 85-20. Cornell University, Ithaca, 1985.

19. Estimates by USDA analysts put the daily cost of the injections between 15 to 50 cents per cow depending on the cost of producing bST and the number of manufacturers competing in the market.

20. T.A. Stucker, R.F. Fallert and K.L. Lipton, "Bovine Growth Hormone Brings Progress to Dairy Farms," op. cit., p. 13.

21. Feed efficiency increases are at least as great as is explained by the reduction in maintenance requirements over the larger production. In such a situation a 25 percent increase in milk production from a cow producing 6500 kg of milk a year would result in a 8.7 percent increase in feed efficiency. See R.J. Kalter and R.A. Milligan, "Emerging Agricultural Technologies: Economic and Policy Implications for Animal Production." Paper presented at the symposium on Food Animal Research held in Lexington, KY, November 2-4, 1986, p. 5.

22. R.J. Kalter and R.A. Milligan, "Emerging Agricultural Technologies: Economic and Policy Implications," op. cit., p. 7.

23. "Effect of graded levels of natural porcine somatotropin (PSG) on growth performance of 57 to 103 kg pigs." Paper presented at the Annual Meeting of the American Society of Animal Science, Utah State University, July 28-31, 1987.

24. R.D. Boyd, "Somatotropin and productive efficiency in swine." Animal Health and Nutrition, February 1987, p. 24.

25. G.L. Grebner, et. al., "Carcass characteristics of pigs injected with different levels of porcine somatotropin (pST) from 57 to 103 kg weight." Paper presented at the Annual Meeting of the American Society of Animal Science, Utah State University, July 28-31, 1987.

26. R.W. Jones, R.A. Easter, F.K. McKeith, R.H. Dalrymple, H.M. Maddock and P.J. Bechtel, "Effect of the B -Adrenergic Agonist Cimaterol (CL 263,780) on the Growth and Carcass Characteristics of Finishing Swine." Journal of Animal Science, Vol. 61, No. 4, 1985, p. 912.

27. T.R. Cline and J.C. Forrest, "Repartitioning Agents and Somatotropin for Finishing Swine." Swine Day, Purdue University, September 3, 1987, p. 6.

28. R.H. Dalrymple, "Repartitioning Agents - An Update with Emphasis on Poultry Production." Proceedings, California Animal Nutrition Conference, Fresno, CA, March 11, 1987, p. 23.

29. "Toward Leaner Meat." National Food Review, NFR-35, Fall 1986, p. 11.

30. Pork '87, August, 1987, p. 46.

31. "Toward Leaner Meat," op. cit., p. 11.

32. The impact of regulatory processes on the timely release of biotechnologies for field tests are elaborated by Susan E. Offut and Fred Kuchler. "Issues in Developments in Biotechnology: What's An Economist To Do?"[2] U.S. Department of Agriculture, Agricultural Economics Research, Winter 1987, Vol. 39, No. 1, pp. 25-33.

33. The present daily injections required for the administration of pST makes it a cumbersome application procedure.

34. Howard Schneiderman, "Overview of Innovation in Agriculture." Paper presented at the Conference on Technology and Agricultural Policy, National Academy of Sciences, Washington, D.C., December 11-13, 1986, p. 12.

35. CRS Review, Washington, D.C., May 1987, p. 16.

36. At present there is a need for studies on the biochemistry and molecular biology of plant processes in order to identify the genes, which determine important agricultural traits.

37. W.J. Florkowski, "Economic Impact of Commercial Application of Biotechnology on Selected Crops." Unpublished University of Illinois, Ph.D. Dissertation, 1986, pp. 51-81.

38. FAO, FAO Agriculture Commodity Projections to 1990. FAO Economics and Social Development Paper 62, Rome 1986, pp. 100-104.

39. U.S. Department of Agriculture, ERS, East Asia and Oceania Situation and Outlook Report. RS-87-2, Washington, D.C., May 1987, p. 52.

40. The scale-dependence of new technologies and related issues were espoused in U.S. Congress, Office of Technology Assessment, Technology, Public Policy, and the Changing Structure of American Agriculture. OTA-F-285, Washington, D.C., March 1986.

41. R.J. Kalter and R.A. Milligan, "Emerging Agricultural Technologies: Economic and Policy Implications for Animal Production," op. cit., p. 8.

42. The Dutch minister of Agriculture Gerrit Brakes is widely quoted as saying of the hormone which can boost individual milk yields by up to 30 percent: "This is just the sort of thing we can do without." Agra Europe, September 18, 1987, p. E/1.

43. Trials at Wageningen Agricultural University in the Netherlands showed an approximate 20 percent increase in gross margin (gross output less cost of the bST itself. Agra Europe, September 18, 1987, p. E/1.

44. Only one pork packer (Lundy Packing of Clinton, NC), four horse slaughtering plants, one sheep plant and one veal slaughter plant were certified. National Hog Farmer, September 1987, p. 22.

45. Among the rules which U.S. firms object to are: separate rooms for various meat slaughter and processing operations; separate rooms for dry storage and box assembly; paved parking lots and prohibition of wooden pallets, knife handles, structural beams and fencing. National Hog Farmer, op. cit., p. 22.

Economic Implications of Repartitioning Agents in the U.S. Swine Industry

Marvin Hayenga and Brian Buhr

Animal agriculture seems poised for a potentially revolutionary change in efficiency and structure as a consequence of biotechnical innovations. While there are a variety of innovations emerging from biotechnology research and development (Greer), the class of products which we will call growth hormones, beta adrenergic agonists, and their effective substitutes* may have an especially dramatic impact.

The bovine growth hormone and its impact on the dairy industry has received the primary attention to date, and has also generated significant controversy and concern among U.S. dairy farmers (one group wants it to be prohibited). The EEC has gone even further and banned growth hormones. Some initial studies have shown that under ideal conditions, dairy cows have improved daily milk production by 40% and milk produced per unit of feed by 20% in the last two-thirds of the lactation period, with smaller improvements in the early stages of lactation (Beitz). Preliminary studies based on these optimistic data suggest that this could lead to 30% fewer cows and 51% fewer dairy farms by the year 2000 (Mix). While such dramatic performance changes aren't likely in commercial dairies, there still could be a significant effect on cow numbers and dairy farms, depending on the changes that would occur in government dairy programs which could also lead to significant changes in cropping patterns, feed demand, industry location and consumer price levels. Potentially dramatic changes might result from the repartitioning agents which are likely to be introduced commercially into the meat animal and poultry sector during the next 10 years. The situation is more complex than with dairy because there are dramatic changes likely in a) the characteristics of the end product (especially significant changes in fat composition), and b) the efficiency of converting feed to weight gain (also significant improvements).

*For convenience, these are collectively referred to as repartitioning agents in the remainder of the paper.

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In addition, there are possible differences in the timing of the FDA approval and commercial availability in the U.S. and abroad, with a corresponding impact on import competition. There is a substantial likelihood that the changes in production efficiency and animal composition resulting from these technological innovations will be quite different in the beef, pork, lamb, and poultry industries, resulting in shifts in demand within the meat complex and changes in relative prices at the farm and consumer levels. For example, it is quite conceivable that some red meats may become much more attractive to consumers compared to poultry, and also become much more price competitive, leading to substantial alterations in the market share trends in the last two decades.

Numerous questions need to be considered. For example, in the U.S. will the nature of the reduced fat content be apparent or hidden from the consumer? Will consumer attitudes toward pork or beef be appreciably changed, and for the better (hormones may be considered an additive, and have a negative connotation)? Will significant consumer education programs be necessary before producers could use these new technologies and still sell their products? Will product segregation or branding be necessary in the marketing process? Will the packer/processor/retailer pricing system fully reflect the value differences back to the farmer, or how will it have to change to avoid stifling these technological innovations?

Which species will experience the greatest rate of improvement from these technological breakthroughs, and what are the likely long term relative price and production volume consequences for each?

In the international market, will the regulatory approval process allow foreign suppliers to benefit sooner (or more) from the technology change, and affect the import supply, prices, and U.S. producers' share of the domestic market?

Obviously, the rate of adoption will be strongly influenced by the method and frequency of application of these repartitioning agents, (dairy is now daily injections) and the price of the product versus its perceived payoff for the producerusing it. Will the daily injections used in the early research be necessary, for growth hormones, or will two-week or once-a-month systems become feasible? How will they fare versus feed additives in the market place? Likely application methods pricing policies and competition among the manufacturers of various repartitioning agents need to be considered in determining likely market penetration levels. And will large farm operators be the primary adopters, putting smaller operators in a more tenuous competitive position, and changing the size distribution of pork producers?

These are not questions that are easy to answer, for the nature of the change being studied is clearly not marginal, but more in the nature of quantum leaps in the effectiveness of converting feed to lean meat in some species. Yet, the implications of such chages are so important that they ought to be considered in the strategic and investment planning for all segments of the livestock and meat industry, their suppliers, and by

policy makers here and abroad. Currently, some segments of the industry are not aware of these imminent changes, while others may react with alarm without a comprehensive appreciation for the implications of these potential changes.

At Iowa State University, a multidisciplinary research team is currently involved in a comprehensive study of these technological developments, but we don't have the answers to all these questions yet because our study is not finished. In addition, the final form of the products that will hit the commercial market two years from now still can change. As a consequence, we're gazing through a slightly cloudy crystal ball.

However, there are some fairly clear implications. I would like to focus on some of the clear and important consequences of these new technological developments, some that are not, and outline some challenges facing the pork industry.

In our book entitled, The U.S. Pork Sector, (Hayenga, et. al.) we identified one of the primary challenges facing the pork industry as the need to continually improve the efficiency by which feed is converted to lean pork. Both somatotropins and repartitioning agents do that, with somatotropins having a more dramatic effect. With muscle replacing fat, the lean yield per hog may increase approximately 15% while feed efficiency improves 25% as the hog grows from 100-240 lb. This could improve the feed cost per pound of lean by 13%, and make pork much more competitive with poultry. The primary cause of the market inroads made by poultry over the last 30 years has been its lower price; these new technologies should lead to lower pork prices, and cause the relative market shares of pork and poultry to stabilize, while pork could substitute for some beef.

The early studies of growth hormones and repartitioning agents suggest that the somatotropins cause a more dramatic performance improvement than the repartitioning agents, but the latter can be a very simple feed additive, a definite advantage over multiple injections or implants. Will the more dramatic performance effects be enough to outweigh the delivery system disadvantages? Which will be the best seller initially? I expect that feed additives would be more easily and universally adopted, but the larger or more sophisticated producers will get their calculator out and conclude that the benefits are worth the extra cost and hassle of periodic injections of somatotropins.

Beyond that, no studies have been published which tested these new technologies together, but it appears likely that they could be additive or at least synergistic. In that case, both the somatotropins and the repartitioning agents could sell very well and potentially lead to greater performance improvements.

Will the sharp reductions in fat content lead to increased consumer demand for pork? Over 60% of consumers indicate that fat and cholesterol are strong concerns affecting their purchases of red meats so it ought to

be a very positive influence on demand. However, there is also a concern about hormones which will have to be alleviated through consumer education, and a concern about additives that could have some negative influence. Intramuscular fat reductions, if large, could also affect taste, texture and palatability, so extensive consumer testing will be necessary to determine the net consumer impact.

Let's consider some possible concerns that may be misconceptions. When you hear about somatotropin implants or injections, possibly several times during the growth process, you naturally expect labor intensity to increase in market hog production. In one sense, labor use does increase when the hogs are handled several more times during the finishing phase; however, the faster growth rate trims off a week in the feedlot too, so the labor per hog may not change very much. But a fully synchronized production operation could simply adjust farrowing schedules or facilities, and lead to more labor required in the finishing operations where labor is currently being fully utilized.

When you hear about growth hormones, you tend to think of the hormone abuse by professional athletes, and the concern about residues in meat. The steroid hormones used by weight lifters and in cattle feeding are quite different than the amino acid chains of the somatotropins. The somatotropins are naturally occuring protein components in the body which would be broken down and denatured by your digestive system if there were elevated levels in the meat. Thus, there shouldn't be any residue problems with somatotropins, and it is likely that any beta agonists approved by the FDA would have a very short half life, leading to either a very short withdrawal period or no withdrawal required before slaughter. Research results suggest that compensating fat gains occur quickly after withdrawal of some beta agonists, so a long withdrawal period would reduce its effectiveness and increase the management problems for the swine enterprise.

Challenges that must be satisfactorily overcome relate to: a) possible consumer concerns about hormones, additives, and taste, b) determining what premium, if any, consumers will pay for less fat and more muscle in a pork product, c) accurate pricing systems for the potentially higher-value hogs and pork products.

Obviously, general and significant consumer concerns about pork produced with any new technology will simply keep it from being used--there's no sense producing something that can't be marketed. However, the Food and Drug Administration approval process should generally alleviate any general concerns, though significant consumer education may be necessary to alleviate some consumers' preconception regarding the risks associated with hormones or other growth stimulants used in meat production. And, the reduction in fat could have some negative impact on taste and texture of the meat to the extent to which there will be noticeable taste differences, and the tradeoffs with the lower fat and higher lean content from a consumer perspective is very important. The impact could range from a) consumers being willing to pay more per pound for a labeled 97% fat free pork product, to b) equal preferences with today's trimmed products, or c) a clear dislike by some consumers for the changes. Obviously, a clear dislike by many consumers would probably kill the technology's introduc-

tion, while slight negative changes in taste may be an acceptable tradeoff for lower prices and fat for many consumers, and little or no taste change with lower prices and fat might lead to significant improvements in consumer demand. Exactly where pork products produced using somatotropins and/or repartitioning agents will fall in this spectrum of possibilities will be important factors in the success or failure of these technologies. Both consumer tests of pork product acceptability, and the change in what consumers would be willing to pay for the changes in pork product characteristics need to be thoroughly studied to determine whether the product changes will warrant higher relative prices at the consumer level, which can be passed on to producers of those improved products. Processor yields of lean product will also increase. The combination of potential improved retail prices and processing yields will need to be accurately reflected back to producers of those products, or there may not be enough incentive to encourage them to use these technologies. Current carcass and live merit pricing systems would have to be revised, and those who sell solely on the basis of live weight might not get any benefits from the carcass improvements possible from these new technologies.

Will producers of the biotech and pharmaceutical companies get the primary benefits from these new technologies? Certainly the biotech and pharmaceutical companies which developed the new productivity-increasing from inputs will get a share of the benefits--perhaps a third of the benefits initially, less as more competitors and competitive products get into the market. The producers who first adopt the new technologies that clearly improve performance will get the biggest benefit initially in reduced production cost, increased carcass market premiums, or both. We estimate that likely cost reductions based on 1987 prices would be approximately $1.28 per cwt. for somatotropin users, and $.28 per cwt. for beta agonist users, assuming that the incremental costs for the new technologies and the related changes in labor, etc. are a third of the projected gross revenue changes. In addition, carcass value improvements of $.96 to $1.57 per cwt. are also likely, in which the biotech companies may share by pricing their products accordingly.

However, more profits stimulate producers to expand their production, and that will push prices down, and long term profitability for hog producers would not change appreciably in this competitively structured industry. Slow adopters will find their costs out of line, and be forced to adopt the cost-reducing technologies or accept lower profits or greater losses or leave the industry. The ultimate primary beneficiary will be the consumer, with lower priced, leaner pork.

Will these new technologies that clearly improve efficiency lead to king-sized hog factories and fewer hog farmers? You would think that any technology that increases the yield of end-product--whether it be milk in the dairy industry or lean meat in the pork industry--would mean fewer hogs would be needed to satisfy demand, and fewer hog farmers to produce them. In addition, many new farm technologies (e. g. mechanization) have facilitiated the growth of the larger, more sophisticated farm operations, to the detriment of their smaller competitors. However, there are some (e. g. hybrid corn) which have been adopted by all farm sizes on roughly an equal basis.

As we examine the likely impact of these new repartitioning agents in the pork industry, it seems clear that beta agonists used as feed additives are likely to be easily adopted by all size hog operations, unless some uneven withdrawal periods are required. The somatotropins, on the other hand, require injections of implants, involve more handling of hogs and more labor intensity and some shift in rations required; some smaller operators are not likely to be early adopters, putting them at a competitive disadvantage. With either new product, the typical hog producer will gradually increase in size, but it seems likely to be more rapid with the introduction of somatotropins.

But, will we end up with a substantial reduction in the number of hog farms due to these new technologies? It's certainly a cloudy area in my crystal ball, but the best answer that I can offer right now is not necessarily. It is going to depend on:

a. The extent to which reduced fat concerns and lower prices cause favorable shifts in the quality of pork demanded by consumers.

b. How much the yield of acceptable lean pork increases from each hog.

c. The shift that occurs in the most efficient size of hog production operation due to these technologies.

A strong favorable shift in consumer demand for pork could even lead to more hog producers. However, I would hazard a guess that these productivity increases are likely to be more quickly achieved and larger in magnitude than likely short term changes in demand, because it's difficult to change consumer habits and perceptions which were developed over a lifetime. As a consequence, the trends toward larger operations are likely to be continued, and possibly accentuated, if small to medium size producers aren't quick to adopt these potentially dramatic improvements in lean pork production efficiency.

What is the overall implication of these new technologies likely to be? Generally, the odds today seem in favor of both of these new technologies becoming commercially available to the pork industry in the next few years. While many uncertainties remain to be resolved, it seems likely that these new inputs into pork production can help produce leaner, more muscular pigs with less feed, and lead to pork being a much more viable competitor with the poultry industry. At the same time, these quantum leaps in new technology could force a significant revamping of the pork pricing and marketing system, especially with the onset of retail-ready packaging which is imminent in the red meat industries. But, suppose competitors do as much or more improving. The beef industry seems likely to make some significant improvements in the next decade, too, though probably not as quickly or as much as in the pork industry. The poultry industry, on the other hand, has done such a tremendous job of productivity improvement that these technologies do not appear to have much impact on poultry performance. Thus, it appears that the pork industry may be able to improve its competitive position in competing for the consumer meat dollar in the next five to ten years.

References

M. L. Hayenga, V. J. Rhodes, J. Brandt and R. Dieter. The U.S. Pork Sector; Changing Structure and Organization, Iowa State University Press, 1985.

Greer, James D., "Market Perspectives for Animal Biotechnology", Agricultural Chemicals Department, SRI International, 1985.

Beitz, Donald, "Futuristic Look at Iowa Dairying: Growth Hormone:, DYS-2864, Department of Animal Science, Iowa State University, December, 1985.

Mix, Lew S., "Potential Impact of the Growth Hormone and Other Technology in the U.S. Dairy Industry by the Year 2000", paper presented at the American Dairy Science Association Annual Meeting, June, 1985.

Nutritional Requirements and Repartitioning Agents

Robert A. Easter

In the introduction to his nutrition classic, Comparative Nutrition of Man and Domestic Animals, H. H. Mitchell (1962) stated with regard to the establishment of nutrient requirements, ". . . this purpose can best be realized most economically, not by studying all possible combinations of animal functions, but, in so far as possible, by studying animal functions each in turn with reference to the nutrient expenditures or storages involved -- in growth, activity reproduction, lactation, etc. -- and the conditions, within the animal or in the environment, that modify these methods of nutrient disposal." Repartitioning agents (Jones, et al. 1985, Anderson etal., 1987) and porcine somatotropin (McLaren et al., 1987; Grebner et al., 1987; Etherton et al., 1987)) cause marked alterations in basic physiological functions and, consequently, in the composition of growing-finishing pigs. It is the sum of these changes that will determine the dietary requirements of pigs treated with these agents.

The time-honored approach to the establishment of nutrient requirements has been based on feeding graded levels of a test nutrient and concurrently measuring an appropriate response criterion. The lowest dose providing the maximum response has generally been accepted as the requirement. This approach, albeit costly in both time and resources, has provided a valid basis for nutrient recommendations in existence today, cf., NRC (1979).

The successful introduction of repartitioning agents and porcine somatotropin will depend, to a significant extent, on the use of a diet capable of supporting a markedly increased rate of lean tissue accretion. It is unlikely that sufficient time will be available for a painstaking, nutrient by nutrient, evaluation of requirements. "Instant" predictions of specific nutrient requirements will be needed. It is arguable that a basis for estimating these requirements for pigs treated with the various growth-altering agents can be established by using Mitchell's concept of summing up the requirements for specific functions.

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Simply stated, this approach requires quantification of certain basic phenomena and prediction of requirements from this information. Consider dietary amino acids as an example. Growing pigs require indispensable amino acids to support protein accretion in muscle, organs and other tissues, for the production of enzymes, hormones and immune proteins associated with physiological functions and to offset obligatory losses, i.e., hair and skin. Estimates for many of these requirements are presently available. In that rate of protein accretion is the major variable among these factors, there should be a predictable relationship between daily deposition of protein and dietary amino acid needs.

The prediction of nutrient requirements from daily rates of lean tissue gain is not without precedent. The theoretical basis for this approach was established in a series of papers from the University of Edinburgh (Whittemore and Fawcett, 1974; Whittemore and Fawcett, 1976). This work culminated in the development of the "Edinburgh Model Pig" (Whittemore, 1981), a mathematical model designed to simulate the growth and growth composition responses that occur in pigs when provided different levels of nutrient intakes. The model has been adapted for computer execution and provides, in the present form, the opportunity for modification of the various assumptions that are made relative to environmental and genetic influences on growth. More recently, the concept has been extended to form the basis for the prediction of protein and energy requirements of pigs having different potentials for daily lean accretion (Whittemore, 1983; and Whittemore, 1987).

Australian scientists (Black et al., 1986) have used a similar approach to the simulation of energy and amino acid utilization by pigs. In this instance the model has been expanded to permit inclusion of nutrient digestibility estimates in the calculation.

The data provided in table 1 represent an attempt to predict a requirement for daily intake of crude protein, and dietary concentration of crude protein required to provide that intake, of pigs treated with 3 mg/day of porcine somatotropin. Half-carcasses were obtained on a representative sample of pigs slaughtered at 45 kg. These carcasses were ground and chemically analyzed for crude protein and fat. These values were then used to calculate the quantity of each of these components present in the half-carcass. Similar pigs were treated with porcine somatotropin from this initial weight to an approximate slaughter weight of 100 kg. After slaughter, the carcass-halves of these animals were also ground and analyzed. As the data in table 1 indicate, there was a net increase in carcass protein, i.e., nitrogen x 6.25, of 4.17 kg in the untreated pigs and 6.02 kg in the pigs receiving the somatotropin treatment. This represents a 44% increase in protein deposition.

In table 2 is an estimation of the increase in dietary protein concentration that would be required to support the 44% increase in protein accretion. Two variables were considered in this calculation, the reduction in feed intake that occurs when pigs are treated with porcine somatotropin and the increase in protein deposition. It would appear that 23% crude protein should be adequate.

Based on these calculations an experiment was conducted involving 96 finishing pigs treated with 3 mg/day of somatotropin. Diets were formulated, by varying the ratio of corn and soybean meal, to provide 14%, 17%, 20%, 23% or 26% crude protein. The pigs were given ad libitum access to feed during the experimental period. The growth-performance results are presented in table 3.

Table 1. EFFECT OF DAILY INJECTION OF PORCINE SOMATOTROPIN ON FAT AND PROTEIN GAIN IN THE CARCASSES OF FINISHING PIGS [1]

Item	Untreated controls	3 mg/day of porcine somatotropin
Initial protein content, kg[2]	7.42	7.42
Final protein content, kg[2]	11.59	13.44
Net gain in protein, kg	4.17	6.02
Increase due to treatment, %	---	44

[1] Unpublished University of Illinois data obtained from Dr. P. J. Bechtel, Meat Science Laboratory.

[2] Values are protein content of ground, half-carcasses of pigs slaughtered at an average weight of 45 kg (initial) or 100 kg (final).

Somewhat surprising is the absence of a dramatic gain response to the increased provision of dietary protein. It is of interest that feed intake was the greatest at the lowest level of crude protein and declined as protein level increased. Feed efficiency data show a clear response to dietary protein and suggest that the appropriate level is between 20% and 23%. Only limited carcass data were available at the time of writing. Liver, an organ rich in protein, increased in size with increasing level of dietary protein. Leaf fat, declined markedly as protein level increased. The changes in both liver and fat serve to indicate that other carcass changes may be more reflective of dietary protein intake than performance data.

Table 2. ESTIMATED "PROTEIN" REQUIREMENT OF PIGS TREATED WITH A DAILY 3 MG INJECTION OF PORCINE SOMATOTROPIN

Requirement of untreated animals:	
Daily feed intake, gm[1]	2,930
Protein intake, (14% crude protein diet), gm[2]	410
Requirement of pigs treated with 3 mg/day somatotropin:	
Protein needed, gm/day[3]	590
Daily feed intake, gm[1]	2,500
Calculated dietary protein concentration, %	23.6

[1] Daily feed intake estimates are based on the data of McLaren et al. (1987) from the University of Illinois.

[2] NRC (1979).

[3] Calculated on the basis of a 44% increase in protein deposition due to porcine somatotropin treatment.

Table 3. PERFORMANCE AND SELECTED CARCASS MEASUREMENTS OF PIGS FEED GRADED LEVELS OF CRUDE PROTEIN AND INJECTED EACH DAY WITH 3 MG OF PORCINE SOMATOTROPIN[1]

	Dietary crude protein, %				
Item	14	17	20	23	26
Daily gain, gm	.77	.80	.79	.77	.73
Daily feed, kg	2.29	2.24	2.18	2.13	2.07
Gain/feed	.32	.35	.37	.37	.35
Liver wt., kg	1.72	1.77	1.74	1.85	1.85
Leaf fat wt., kg	.99	.77	.82	.73	.66

[1] Unpublished University of Illinois data.

It is also true that these diets were formulated without attention to changes in amino acid profile. Until information is available on the relative changes in amino acid utilization for synthesis of various tissues, there is no basis for suggesting that the dietary

amino acid profile differs greatly from that now provided in conventional diets.

Undoubtedly, requirements for other nutrients change as well. In pigs treated with porcine somatotropin, there is an increase in bone mass (Grebner et al., 1987). This would suggest that requirements for minerals may be different. Other nutrients may also be affected. For example, dietary crude protein level has been shown to affect the requirement for vitamin B-6 (Russell, 1984).

One is not to assume that porcine somatotropin is alone in effects on nutrient requirements. One of the most interesting nutritional developments relative to the repartititioning agents is the demonstration by Anderson et al. (1987) that the extent of response to Ractopamine is affected by the concentration of crude protein in the diet. This point is illustrated by the results provided in table 4. As would be expected, the nutritional base must be provided in order for the repartitioning agent to exert its effect on performance and lean tissue accretion.

The introduction of porcine somatotropin and repartitioning agents will undoubtedly result in profound changes, not only in nutrient requirements, but also in the manner in which nutrient requirements are perceived and expressed. It may no longer be possible to have a single recommendation for a nutrient level for pigs during a specific phase of growth as presently exists in the NRC (1979) tables. That recommendation may be modified by assumptions related to potential for lean tissue growth, level of drug administered and other considerations. The best expression of a nutrient requirement is likely to be an equation that contains as elements the identifiable variables in the feeding environment. Specific values could then be calculated to estimate the nutrient requirement of a pig in a particular situation.

It is apparent that the rapid development of nutrient recommendations will require the nutritionist to integrate much of the basic information that has been developed over the past four decades. This includes data on efficiency of lean growth, energetic costs of protein synthesis and the effect of these changes on requirements for vitamins and minerals. It promises to be an exciting challenge.

Table 4. EFFECT OF RACTOPAMINE AND DIETARY PROTEIN IN FINISHER SWINE[1]

Ractopamine, ppm	Dietary protein, % 12	15	18	15+Lysine
		10th Rib Fat Depth, cm		
0	3.15	2.87	2.69	2.67
5	2.90	2.72	2.46	2.62
20	2.84	2.54	2.26	2.24
		10th rib loin eye area sq cm		
0	29.4	32.3	32.3	32.8
5	30.8	33.3	34.5	33.9
20	31.2	35.0	36.8	36.8
		Daily gain, kg		
0	.75	.78	.75	.77
5	.70	.81	.83	.82
20	.72	.79	.82	.80

[1] Anderson et al. (1987)

LITERATURE CITED

Anderson, D. B., E. L. Veenhuizen, W. P. Waitt, R. E. Paxton and S. S. Young. 1987. The effect of dietary protein on nitrogen metabolism, growth performance and carcass composition of finishing pigs fed ractopamine. Fed. Proc. 46:1021 (Abstr.).

Black, J. L., R. G. Campbell, I. H. Williams, K. J. James and G. T. Davies. 1986. Simulation of energy and amino acid utilisation in the pig. Res. and Dev. in Agric. 3:121.

Etherton, T. W., J. P. Wiggins, C. M. Evock, C. S. Chung, J. F. Rebhun, P. E. Walton and N. C. Steele. 1987. Stimulation of growth performance by porcine growth hormone: determination of the dose-response relationship. J. Anim. Sci. 64:433.

Grebner, G. L., F. K. McKeith, J. Novakofski, R. A. Easter, D. G. McLaren, K. Brenner, P. J. Bechtel, R. W. Jones and R. H. Dalrymple. 1987. Carcass characteristics of pigs injected with different levels of natural porcine somatotropin. J. Anim. Sci. 65(Suppl. 1)245.

Jones, R. W., R. A. Easter, F. K. McKeith, R. H. Dalrymple, H. M. Maddock and P. J. Bechtel. 1985. Effect of the B-adrenergic agonist cimaterol (CL 263,780) on the growth and carcass characteristics of finishing swine. J. Anim. Sci. 61:905.

McLaren, D. G., G. L. Grebner, P. J. Bechtel, F. K. McKeith, J. E. Novakofski, R. A. Easter, R. W. Jones and R. H. Dalrymple. 1987. Effect of graded levels of natural porcine somatotropin (PST) on growth performance of 57 to 105 kg pigs. J. Anim. Sci. 65(Suppl. 1)245.

Mitchell, H. H. 1962. Comparative Nutrition of Man and Domestic Animals Academic Press, New York.

NRC. 1979. Nutrient Requirements of Domestic Animals, No. 2. Nutrient Requirements of Swine. Eighth Revised Ed. National Academy of Science - National Research Council, Washington DC.

Russell, L. E. 1984. An investigation of factors affecting the pyridoxine requirement of postpubertal gilts. Ph.D. Thesis. University of Illinois. Urbana.

Whittemore, C. T. and R. H. Fawcett. 1974. Model responses of the growing pig to the dietary intake of energy and protein. Anim. Prod. 19:221.

Whittemore, C. T. and R. H. Fawcett. 1976. Theoretical aspects of a flexible model to simulate protein and lipid growth in pigs. Anim. Prod. 22:87.

Whittemore, C. T. 1981. Animal production response prediction. in (G. M. Hillyer, Ed.) Computers in Animal Production. British Soc. Anim. Prod., Thames Ditton, Surrey, England.

Whittemore, C. T. 1983. Development of recommended energy and protein allowances for growing pigs. Agric. Sys. 11:159.

Whittemore, C. T. 1987. Elements of Pig Science. Longman Scientific and Technical, Essex, UK.


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