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Inheritance of Resistance to Bacterial Infection in Animals

A Genetic Study of Pullorum Disease

By Elmer Roberts
and L. E. Card

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Inheritance of Resistance to Bacterial Infection in Animals

A Genetic Study of Pullorum Disease

By Elmer Roberts, Chief in Animal Genetics, and
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MAN HAS always been engaged in a struggle with the invisible forms of life, but not until after the time of Pasteur was fundamental information available enabling him to attack with any degree of success the problem of disease control. The accepted approach to this problem has been environmental; that is, thru the control of microorganisms or of environmental factors conducive to the health of the host. Some people have begun to ask what the role of the host, whether man, animal, or plant, may be in the phenomenon of disease. If an infectious disease is considered as the reaction between a parasite and its host, then the host as well as the parasite should receive consideration in disease control. A genetic study of such a disease is a study of the host in its relation to its resistance or susceptibility to infection.

Disease has been defined as a departure of the organism from normal functioning or constitution. On the basis of this definition diseases may be classified into two groups: those due primarily to abnormal structure and functioning of certain organs or tissues of the body and those due to living organisms of various kinds.

The hereditary nature of many diseases that are due to abnormal structure and functioning has been firmly established. The inheritance and, in many cases, the mode of inheritance, of such disorders as haemophilia, anhidrosis, absence of enamel of teeth, resistance and susceptibility to transplantable tumors, and of many defects of both hair and skin have been fairly well worked out during the past few years. Most of the diseases due to abnormal structure and functioning are relatively rare in comparison with the number of diseases caused by infecting organisms. It is a disease of the latter class with which this study is concerned.

*To L. C. Thomas, W. M. Dawson, and J. H. Quisenberry acknowledgment is due for valuable assistance at various times during the course of this investigation; also to J. A. Hunter of Tunghsien, China, for generous assistance in the study of Chinese fowls during 1929-30.

That resistance or immunity to certain diseases caused by infecting organisms varies with different genera of animals is a matter of common knowledge. One genus may be entirely resistant to a disease to which another genus is very susceptible. Furthermore, one species or race may be immune to a disease to which another species or race may be highly susceptible.[a] Facts such as these, drawn from general observation, are valuable for certain purposes, but fundamental information concerning the relation of heredity to disease resistance can be obtained only thru carefully conducted investigations under controlled conditions. Furthermore, it is upon the resistance or immunity of individuals that attention must be centered if facts are to be developed concerning the nature of disease resistance and its application to disease problems.

Those who work with animals have frequently noted that some individuals appear to be resistant to disease; that is, that they escape infection or give no evidence of having contracted infection when they have had ample opportunity to do so, or if infected the results are much less severe than in the case of other individuals. The investigation reported herein was designed to throw some light on the question whether such qualities in farm animals may be inherited. The immediate object of the investigation was to study chickens with respect to variability in their resistance to *Salmonella pullorum*, the causative organism of pullorum disease and, if a sufficiently wide difference was found among them, to attempt to ascertain the degree to which resistance or susceptibility to infection with this organism seemed to be inherited. Several preliminary reports of this work have been made.[4, 30-38*]

MATERIALS AND METHODS

Pullorum disease of fowls was chosen for this study for four reasons: (1) it is a well-defined disease of young chicks usually running its course in a few days; (2) the organism is easily cultured in the laboratory; (3) the biological tests for detecting the infection are considered reliable and are used in breeding flocks in many parts of the country for the elimination of infected individuals; (4) more

[a]The horse is immune to diphtheria, whereas man is susceptible. Brahma cattle are resistant to Texas fever whereas our native cattle, belonging to a different species, are very susceptible. It is reported that Negroes and Indians are more susceptible to tuberculosis than are Caucasians. The Chinese seem to be highly resistant to *B. tetanus*. Orientals in general are said to be much more resistant to syphilitic infection than are peoples of other races.

*These numbers refer to literature citations on pages 489 and 490.

individuals could be used in studying a disease of young chicks than could possibly have been used had adults been necessary.

Materials.—More than 29,000 birds of various breeds of the domestic fowl have been used in this investigation, which has extended, in its various phases, over a period of ten years.

The foundation breeding stock for the experiment was obtained from several hundred day-old chicks which were inoculated with *Salmonella pullorum.* The few that survived were used as breeders. Others obtained in like manner were added from time to time. At first only chickens that had survived inoculation were used in the selected stocks, but later the progeny test was used as a basis of selection, those parents whose offspring had shown the highest percentage of survival being selected for further use as breeders.

Procedure.—Chicks were inoculated the day after they were hatched, or the twenty-second day after the eggs had been placed in the incubator. The culture was administered orally thru a pipette.[a] The quantities administered ranged from 1/16 to 1/4 cc., the amount depending upon the virulence of the culture. Since no satisfactory method has yet been devised for standardizing the cultures, the amount to be used had to be arrived at by trial. Eggs from all the stocks to be tested were placed in the same incubator at the same time, thus insuring the same environmental conditions for all during the period of incubation. All chicks of a given hatch were inoculated from the same culture and were then put in the brooder for observation. Chicks of all the different kinds to be tested were placed in the same brooder. In this way environmental differences in temperature, time, space, feed, and differences in cultures were eliminated.

EXPERIMENTAL RESULTS

The investigation reported herein included three phases: (1) a study of the resistance of certain strains of chickens to inoculation with *Salmonella pullorum;* (2) a study of various crosses between selected and unselected strains with regard to the inheritance of resistance or susceptibility to inoculation; (3) a study of the possibility of the experimental birds having acquired resistance to the disease.

The term *resistance,* as used in reporting the data in these experiments, does not imply that the individual was necessarily free from the

[a]The Laboratory of Animal Pathology and Hygiene prepared the 24-hour broth cultures used for inoculation and also made the autopsies and biological tests of the birds.

infecting organism, but that it was able to survive exposure to the organism or to produce offspring able to survive exposure. Without question, varying degrees of resistance existed in the surviving stock, some chicks exhibiting definite symptoms of infection, while others were free from any visible signs.

Because of its simplicity and the accuracy with which it could be applied, *survival* was used thruout these tests as the basis for selecting resistant breeding stock and for judging the reaction of individuals to inoculation with the infecting organism. Incidentally some data are presented concerning the effect of inoculation on growth retardation and the relative susceptibility of the progeny of growth-retarded dams and the progeny of dams which, judged by their larger growth, had been less affected by inoculation.

Resistance of Selected Strains of Chickens to Inoculation

The results of inoculating 128 hatches of strains of chickens selected for resistance to infection with *S. pullorum* and 128 unselected controls are given in Table 16, Appendix. Table 1 gives a summary of all the hatches for Strain I, including those with fewer than fifteen chicks, which are not included in Table 16. The selected stock obviously showed a much greater resistance to disease than did the controls. The same results were obtained with two other strains (Table 2).

The mean survival of the chicks from the selected stock was 71.5 ± 1.08 and of the controls 27.44 ± 1.26. The difference was 44.06 ± 1.66, or 26.5 times the probable error. The probability that this difference occurred by chance is only $\frac{4.927}{10^{72}}$, which means that the difference between the control and selected stocks was genetic in nature and not due to chance.[a] In only one hatch of the 128 was the percentage of survival among the controls as great as or greater than the percentage of survival among selected stock. In that hatch the selected strain showed 70.6 percent survival and the control 77.4 percent. This high survival among the controls indicates that a relatively nonvirulent culture had been used.

While these results show that selection is effective in establishing resistant strains, the data do not show what progress can be made by continuous selection thru successive generations. A standardized, or

[a]Because of the high degree of statistical significance of the results in the different phases of this study, the mathematical constants are not given except for the data in Table 16 of the Appendix. Individual hatches were used in the statistical determinations. In all tables with the exception of Table 16 individual hatches are combined.

TABLE 1.—RESULTS OF INOCULATING CHICKS FROM STRAIN I SELECTED FOR RESISTANCE TO S. PULLORUM AND CHICKS FROM UNSELECTED STOCKS[a]

Year	Selected Strain I			Control		
	Number inoculated	Number surviving	Percent surviving	Number inoculated	Number surviving	Percent surviving
1925	336	160	47.6	446	115	25.8
1926	478	384	80.3	596	61	10.2
1927	1 615	1 142	70.7	820	384	46.8
1928	414	345	83.3	293	154	52.6
1929	188	96	51.1	559	118	21.1
1930	481	395	82.1	1 188	432	36.4
1931	374	231	61.8	764	46	6.0
1932	...	...	....	...	...	....
1933	252	186	73.8	1 030	290	28.2
Total	4 138	2 939	71.0	5 696	1 600	28.1

[a]Records of individual hatches are given in Table 16, Appendix.

TABLE 2.—RESULTS OF INOCULATING CHICKS FROM SELECTED STRAINS II AND III AND CONTROL CHICKS[a]

Year	Number inoculated	Number surviving	Percent surviving	Number inoculated	Number surviving	Percent surviving	Number inoculated	Number surviving	Percent surviving
	Selected Strain II			Selected Strain III			Control		
1930	408	330	80.9	301	255	84.7	1 188	432	36.4
1931	1 100	631	57.4	620	403	65.0	764	46	6.0
1932	297	241	81.1	216	186	86.1	594	322	54.2
1933	801	489	61.4	533	375	70.4	1 030	290	28.2
Total	2 606	1 691	64.9	1 670	1 219	73.0	3 576	1 090	30.5
				Strain II × Strain III			Control		
1934	.....	.....	....	540	470	87.0	123	25	20.3

[a]Data on individual hatches are given in Table 16, Appendix.

uniform, culture would be necessary for such work. The cultures used in this experiment were prepared in the same way during the entire time, yet variations in survival are so great that comparisons between results of two different years might be misleading. Not only does the possibility of a varying culture exist but there is also great probability of uncontrolled environmental factors which may vary from year to year and thus influence the host.

While a part of the death losses in these hatches may not have been due to inoculation with *S. pullorum*, the greatest part without doubt was the result of such infection.

Noninoculated chicks of selected strains, of unselected strains, and of various crosses between selected and unselected strains were

kept under observation at different times in order to determine their mortality compared with the mortality of inoculated chicks. When not inoculated, all these groups showed low mortality (Table 3).

While survival has been used in this study as the basis for judging the resistance of individuals to infection, other criteria of resistance could be used, such, for instance, as the effect of infection on the vitality of those that survived. One might expect that the rate of

TABLE 3.—SURVIVAL OF NONINOCULATED CHICKS FROM RESISTANT AND SUSCEPTIBLE PARENTS, AND FROM CROSSES BETWEEN THE TWO, WHEN SUBJECTED TO THE SAME ENVIRONMENT AS THE INOCULATED CHICKS EXCEPT FOR EXPOSURE TO S. PULLORUM

Mating	Number hatched	Number surviving	Percent surviving
1928			
Resistant ♂♂ × Resistant ♀♀	193	179	92.7
Resistant ♂♂ × Susceptible ♀♀	144	138	95.8
Susceptible ♂♂ × Resistant ♀♀	141	136	96.5
Susceptible ♂♂ × Susceptible ♀♀	89	81	90.5
Total	567	534	94.2
Hatch No. 1	22	22	100.0
Hatch No. 2	35	35	100.0
Hatch No. 3	37	36	97.3
Hatch No. 4	94	91	96.8
Hatch No. 5	84	77	91.7
Hatch No. 6	91	88	96.7
Hatch No. 7	85	82	96.5
Hatch No. 8	119	103	86.6
Total	567	534	94.2
1929			
F_1 ♂♂ × Susceptible ♀♀	38	33	86.8
F_1 ♂♂ × Resistant ♀♀	50	43	86.0
Susceptible ♂♂ × F_1 ♀♀	86	76	88.4
F_1 ♂♂ × F_1 ♀♀	154	141	91.6
Total	328	293	89.4

growth would be retarded in proportion to the severity of the infection which an individual had experienced. If this is so, the chickens that gained weight more rapidly would perhaps have a higher resistance to inoculation than would those which grew less rapidly. A test of the comparative resistance of the progeny of rapidly and slowly gaining dams that had survived inoculation would shed some light on the validity of this assumption. Weights of several hundred inoculated birds that had survived inoculation were therefore taken and the progeny of these dams inoculated. The progeny of the heavy dams showed a survival after inoculation of 75.4 percent and the progeny of the light dams a survival of 64.6 percent (Table 4).

Records were also kept of the growth of inoculated and noninoculated chicks to the age of 163 days. The average weights of the

TABLE 4.—SURVIVAL OF PROGENY OF HEAVY AND LIGHT DAMS THAT HAD SURVIVED INOCULATION

Pen	Sire	Heavy			Light		
		Number inoculated	Number surviving	Percent surviving	Number inoculated	Number surviving	Percent surviving
13	H–1907	177	140	79.1	152	97	63.8
14	J–3743	195	154	79.0	217	148	68.2
15	J–5649	234	163	69.7	...	...	....
16	J–7688	...	...	....	167	101	60.5
	Total	606	457	75.4	536	346	64.6

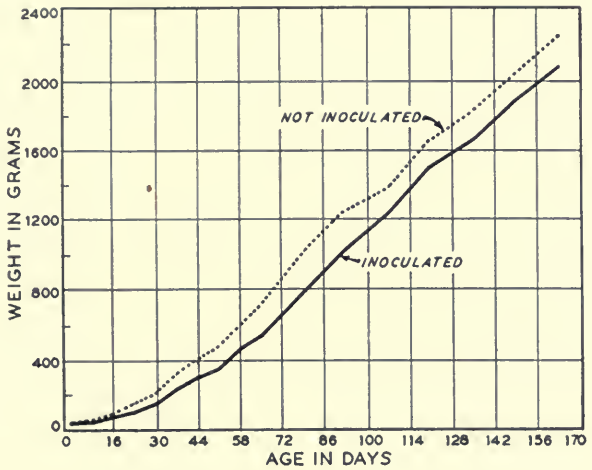

FIG. 1.—EFFECT OF INOCULATION ON GROWTH OF CHICKS

inoculated chicks that survived were consistently less thruout the period than were those of the noninoculated chicks (Fig. 1).

Evidently inoculation has a very definite effect in retarding the growth of individuals that survive infection.

Effect of Inbreeding on Resistance to Infection

A few comparative tests for resistance to infection with *Salmonella pullorum* of inbred and noninbred strains were made. An inbred strain of White Plymouth Rocks had a survival of 72.1 percent among 68 inoculated, while among 110 noninbreds only 3.6 percent survived. Another test with White Leghorns gave among 113 inbreds 52.2 percent survival and 31.6 percent among 462 noninbreds. This suggests that natural selection had occurred among the inbreds, eliminating to some extent the less resistant individuals.

The results of inbreeding tests with two strains (T and M) from Strain I (Table 1), which had been selected for two years for its resistance to *S. pullorum,* are shown in Table 5. Strain M had not been inbred until 1927, the year the data were taken. In the previous year the percentages of the progeny of Strains T and M surviving inoculation were practically the same. In 1927 there was no great difference in the survival of progeny as between brother-sister matings

TABLE 5.—RESULTS OF INBREEDING WITHIN RESISTANT STOCK, 1927

Mating	Strain T, highly inbred for several years[a]		Strain M, first inbred in 1927[a]	
	Number inoculated	Percent alive at 21 days	Number inoculated	Percent alive at 21 days
B × S	129	76.7	185	45.4
Other matings	100	74.0	1 105	72.4
Total	229	75.5	1 290	69.3

[a]These strains are branches of Strain I (Table 1); see pages 470-471.

and other matings in Strain T; whereas in Strain M, inbred for the first time, the progeny of brother-sister matings had a survival much lower than that of the progeny of other matings. These results are what would be expected if resistance and susceptibility are hereditary, provided no preventive measures for the protection of the birds had been used. In this case no such protection had been given Strain T.

Study of Chinese Birds for Resistance to Disease

If resistance and susceptibility are hereditary, one would expect, in an old country where fowls have long been domesticated and where there has been no artificial control of disease, to find the animals more resistant than those in a country where domestication is recent, where the disease had been present, and where disease-control measures have been in general use. Thru natural selection, in the older country, susceptible individuals would tend to be eliminated from the population, only the more resistant ones surviving. Under such circumstances it would be expected that strains would be developed showing greater resistance than the original population and presumably greater resistance than shown by animals under protection in a newer country.

In order to test this assumption a study of Chinese birds was

made by the senior author at Tunghsien, China, in the spring of 1930. Six different kinds of chickens were selected for the tests:

1. Chia Gi (small-type chicken) from the vicinity of Peiping, China
2. Cochins from the vicinity of Peiping
3. Shansi from Shansi Province
4. Langshans from Nantungchow, Central China
5. White Leghorns from stock imported from the United States about 1923
6. Rhode Island Reds imported from Canada specifically for the experiment

In addition some chicks from a local Chinese hatchery were tested. The same methods of incubation, inoculation, and brooding were used as described on page 469 except for the chicks from the local hatchery.

TABLE 6.—STUDY OF DISEASE RESISTANCE IN CHINESE CHICKENS: NUMBER INOCULATED WITH S. PULLORUM AND NUMBER AND PERCENTAGE SURVIVING AT THREE WEEKS OF AGE

Kind of chicken	Number inoculated	Number surviving	Percent surviving
All hatches			
Chinese			
Chia Gi (small type)	552	337	61.1
Cochin	397	152	38.3
Shansi	221	59	26.7
Langshan	409	142	34.7
Hatchery[a]	298	132	44.3
Imported			
White Leghorn[b]	306	208	68.0
Rhode Island Red[c]	442	259	58.6
Total	2 625	...	....
Excluding hatches with low mortality due to nonvirulent cultures[d]			
Chinese			
Chia Gi (small type)	370	185	50.0
Cochin	253	73	28.9
Shansi	158	25	15.8
Langshan	261	41	15.7
Hatchery[a]	298	132	44.3
Imported			
White Leghorn[b]	200	139	69.5
Rhode Island Red[c]	282	133	47.2
Total	1 822	...	....

[a]Parentage unknown. [b]Imported from the United States about six years before. [c]Imported from Canada for the experiment. [d]Judged by the low resulting mortality.

Judged by the number of chicks surviving inoculation with *S. pullorum*, the White Leghorns were much more resistant than the other kinds tested (Table 6). The small type of Chinese chicken, Chia Gi, was more than three times as resistant as the Shansi and the Langshan chickens and 1.8 times as resistant as the Cochin chickens,

and the Rhode Island Reds used were almost as resistant as the Chia Gi. The low mortality of the chickens in the first hatches indicates that the cultures used were probably less virulent than those used in later inoculations in which, with the same kinds of chickens, a higher mortality resulted. The mortality in the later hatches, however, showed about the same relative differences between the different kinds of chickens in their resistance to the disease as were obtained in the first hatches.

It is not known to what extent the Rhode Island Reds, the third most resistant of the kinds tested, may have been subjected to selection previous to this study. The disease had been present in the White Leghorn flock from which samples were taken for this study, according to the people from whom they were obtained, and the agglutination test showed several reacting individuals. The White Leghorns had also been inbred to some extent after their importation into China, some for as long as five generations. If resistance and susceptibility to disease are hereditary, this inbreeding would automatically produce some individuals tending to be pure for factors for resistance and others pure for factors for susceptibility; and the operation of natural selection would tend to eliminate the susceptible and retain the resistant. There would be no more rapid way of producing a highly resistant strain than to inbreed and at the same time subject the individuals to the disease in question.

Agglutination tests were made on the birds used in this experiment. Tests of commercial stock were made by Chen Ken of the University of Nanking, blood samples being obtained at the place of killing (Table 7).

TABLE 7.—RESULTS OF AGGLUTINATION TESTS FOR PULLORUM DISEASES IN STUDY OF CHINESE CHICKENS

Kind of chicken	Test negative	Test positive	Suspicious
Chinese			
Chia Gi (small type)	22	5	0
Cochin	24	2	0
Shansi	21	4	0
Langshan	28	0	0
Commercial stock	678	25	28
Imported			
Rhode Island Reds	27	0	1
White Leghorns	17	8	0

Positive reactors were found among all the Chinese chickens except the Langshans, indicating that the disease is present in China, but how long it has been present is not known. It may have been

brought in with imported stock or it may have been in existence in China for a very long time.

The Langshan chickens, which showed no reactors, were obtained from a region where no importation of foreign birds had been made. Mr. Yao, of the College of Agriculture, University of Nanking, who lived in Nantungchow for many years, said that no ailment similar to pullorum disease had been found, to his knowledge, among Langshan chickens.

Altho several reactors were found among the Shanshi chickens, the survival among these chickens, when inoculated with *S. pullorum*, was as low as among the Langshans. The possibility exists that pullorum disease had been of recent introduction into the Shanshi chickens; if so, results similar to those obtained with nonreacting Langshan chickens would be expected.

The small-type Chinese chicken, Chia Gi, which is very numerous in the vicinity of Peiping, under all conditions was more resistant to inoculation with *S. pullorum* than any of the other Chinese birds. The larger number of these chickens may be due partly to their greater resistance to pullorum disease.

The resistance of the Cochin, found in the same region as the Chia Gi but not in such large numbers, was not nearly so high as that of the Chia Gi, yet it was higher than the resistance of either the Shanshi or the Langshan chickens.

Stocks of Langshans and Chia Gi were shipped to the United States (Urbana, Illinois) for further work in connection with this investigation. In 1931 the percentage of Chia Gi chicks that survived inoculation was 50.8, of the Langshans 7.5, and of unselected Rhode Island Reds 6.2. The difference between the Chia Gi and the Langshan chickens exhibited under conditions in Urbana was practically the same as found in China in the spring of 1930, indicating that the differences were due to genetic and not to environmental factors.

The high resistance to pullorum disease shown by the Chia Gi chicken of North China and the low resistance of the Langshan of Central China suggest the operation of natural selection. In North China the birds had been exposed to pullorum disease; apparently the more susceptible had been eliminated and the more resistant had survived. In Central China the Langshan, so far as was known, had not been in contact with the disease, so that natural selection was impossible.

Resistance of Different Breeds to Salmonella Pullorum

The resistance to *Salmonella pullorum* exhibited by the different breeds tested in this study varied greatly. For example, Rhode Island

Reds were, in general, less resistant than the White Leghorns, White Plymouth Rocks, Barred Plymouth Rocks, and Chia Gi which were tested. The Langshans of Chinese origin were very susceptible (Table 8). The stock of Rhode Island Reds obtained in Canada for use in China was found to possess much more resistance than any other Rhode Island Red stock tested. Another group of Canadian Rhode Island Reds proved to be extremely susceptible to inoculation with *S. pullorum.*

These limited tests are not sufficient, however, to warrant the conclusion that all Rhode Island Reds are more susceptible than other

TABLE 8.—SURVIVAL OF INOCULATED CHICKS OF DIFFERENT BREEDS AND VARIETIES: UNSELECTED STOCKS[a]

Breed	Strain	Number of hatches	Number of chicks inoculated	Number surviving	Percent surviving
1926					
White Leghorn	B	5	76	39	51.3
White Leghorn	D	5	462	146	31.6
Rhode Island Red	E	5	508	51	10.0
1927					
White Leghorn	D	10	499	194	38.9
Rhode Island Red	E	10	516	190	36.8
Rhode Island Red	F	10	113	42	37.2
1930					
White Leghorn	A	7	301	255	84.7
White Leghorn	C	7	167	148	88.6
Rhode Island Red	E	7	647	177	27.4
1931					
White Leghorn	C	8	285	201	70.5
Rhode Island Red	E	8	495	42	8.5
Langshan	..	8	173	13	7.5
Chia Gi	H	8	193	98	50.8
1932					
White Leghorn	A	5	632	500	79.1
Rhode Island Red	E	5	594	322	54.2
Chia Gi	H	5	631	500	79.2
1933					
Rhode Island Red	E	7	712	226	31.7
Rhode Island Red	G	7	191	28	14.7

[a]This table includes only those tests in which there were five or more hatches in a given year, and each hatch contained chicks of the different breeds to be compared.

breeds. Extensive tests of samples of breeds from various parts of of the country would be necessary to provide conclusive information on breed differences. The degree of resistance would be greatly influenced by the extent to which pullorum disease had been present in the stock sampled and the birds consequently subjected to natural selection on the basis of their resistance to this disease. That resistance can be significantly increased by selection was demonstrated by the change produced in a strain of Rhode Island Reds used in one phase

of this study. In a strain selected for one generation the survival following inoculation was 32.8 percent among a population of 346 chicks, while among 764 controls the survival was only 6.0 percent.

RESULTS OF CROSSES

One of the important sources of evidence concerning the inheritance of characters is from crosses involving the characters to be studied. Analysis of the F_1, F_2, and back-cross generation provided information concerning the hereditary nature of resistance and susceptibility. A graphic presentation of the results of the various crosses is given in Fig. 2.

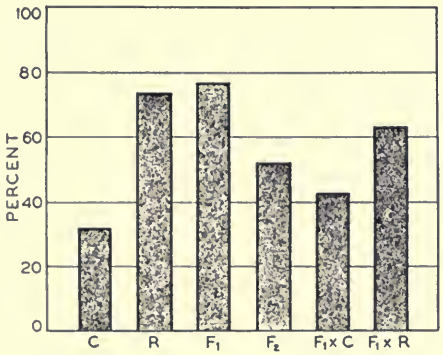

FIG. 2.—SURVIVAL OF CONTROLS, RESISTANT, F_1, F_2, AND BACK-CROSSES WHEN EXPOSED TO PULLORUM DISEASE BY INOCULATIONS WITH SALMONELLA PULLORUM

F_1 Generation.—The survival of the F_1 progeny was as high as that of the resistant parents, indicating dominance of resistance (Table 9). The characters found in wild animals have, in general, a survival value and are usually dominant in expression. It is interesting that in this case resistance to disease appears to be dominant.

Back-Crosses.—The back-crosses and $F_1 \times F_1$ matings gave further evidence of the hereditary nature of resistance and susceptibility to pullorum disease. From reciprocal crosses no evidence of sex-linked factors was found (Table 9).

Selection Among Back-Cross and F_2 Generations.—A selection for low and high resistance was made in back-cross and in F_2 generations. The basis of selection was the survival of the progeny. The selections were made in 1930. In this year 13.8 percent of the progeny in the stock selected for low resistance and 85.4 percent of the progeny

TABLE 9.—SURVIVAL OF CHICKS HATCHED FROM VARIOUS RESISTANT AND CONTROL STOCKS, WHEN INOCULATED WITH S. PULLORUM

Type of mating (R = resistant, C = control)	Number of chicks inoculated	Number alive at 3 weeks	Percent alive at 3 weeks
From purebreds and first crosses, 1928 and 1929			
R♂♂ × R♀♀	602	441	73.3
C♂♂ × C♀♀	852	272	31.9
R♂♂ × C♀♀	517	405	78.3
C♂♂ × R♀♀	467	346	74.1
From F_1 matings *inter se* and back-cross matings of resistant and control stock, 1929 only			
R♂♂ × F_1♀♀ ex R♂ × C♀	219	142	64.4
R♂♂ × F_1♀♀ ex C♂ × R♀	184	118	64.1
R♀♀ × F_1♂♂ ex R♂ × C♀	93	60	64.6
R♀♀ × F_1♂♂ ex C♂ × R♀	59	31	52.5
C♂♂ × F_1♀♀ ex R♂ × C♀	175	56	32.0
C♂♂ × F_1♀♀ ex C♂ × R♀	161	76	47.2
C♀♀ × F_1♂♂ ex R♂ × C♀	69	25	36.2
C♀♀ × F_1♂♂ ex C♂ × R♀	96	34	35.4
F_1♂♂ ex R♂ × C♀ × F_1♀♀ ex R♂ × C♀	193	106	54.9
F_1♂♂ ex R♂ × C♀ × F_1♀♀ ex C♂ × R♀	203	105	51.2
F_1♂♂ ex C♂ × R♀ × F_1♀♀ ex R♂ × C♀	194	95	48.9
F_1♂♂ ex C♂ × R♀ × F_1♀♀ ex C♂ × R♀	132	70	53.0
All matings	4 216	2 382	56.5

of those selected for high resistance survived exposure to the organism. The next year the survival was 8.2 percent and 69.2 percent, respectively in the two groups. The difference between the percentage survival of the two selections was 61.6 percent in 1930 and 61.0 percent in 1931 (Table 10).

The fact that successful selection can be made among individuals resulting from the original cross of susceptible and resistant stocks, and that these selections perform consistently in respect to susceptibility and resistance is evidence of the importance of heredity in relation to this disease.

Results of Matings of One Male to Both Resistant and Susceptible Females.—Further evidence of the hereditary character of resistance to disease was obtained from matings of susceptible and resistant females to the same male, either susceptible or resistant. In every mating with susceptible males the progeny from the resistant dam had a higher survival than did the progeny from the susceptible dam (Table 11). The average survival of the progeny of all susceptible dams mated with susceptible males was 45.7 percent and of all resistant dams so mated 73.8 percent. Since the males were the same for both

TABLE 10.—SURVIVAL OF PROGENY OF F_2 AND BACK-CROSS FEMALES SELECTED FOR HIGH AND LOW RESISTANCE TO S. PULLORUM: FEMALES MATED TO SUSCEPTIBLE MALES

(From the 15 females whose progeny had a survival of less than 20 percent in 1930, nine were selected for a similar test in 1931. From 20 females whose progeny had a survival of 70 percent or more the first year, ten were selected for the second year's test.)

Distribution of 1930 females on basis of progeny surviving inoculation		Females tested both years (Nos.)	1930 progeny from females tested both years			1931 progeny from females tested both years		
Percentage of progeny surviving	Number of females		Number of chicks inoculated	Number of chicks surviving	Percent surviving	Number of chicks inoculated	Number of chicks surviving	Percent surviving
			Records of progeny of females showing low resistance					
0–9.9.....	5	J 7747	25	2	8.0	36	7	19.4
		6705	31	3	9.7	3	1	33.3
10–19.9.....	10	6919	28	3	10.7	32	1	3.1
		6769	44	6	13.6	10	1	10.0
		5814	34	5	14.7	28	2	7.1
		6785	27	4	14.8	14	1	7.1
		6915	19	3	15.8	18	2	11.1
		7709	36	6	16.7	26	0	0
		6907	38	7	18.4	29	1	3.4
20–29.9.....	10	.....	..	..	....	..	..	....
30–39.9.....	13	.....	..	..	....	..	..	....
40–49.9.....	9	.....	..	..	....	..	..	....
Total.....	47	(9)	282	39	13.8	196	16	8.2
			Records of progeny of females showing high resistance					
50–59.9.....	17	.....	..	..	....	..	..	....
60–69.9.....	22	.....	..	..	....	..	..	....
70–79.9.....	12	J 5668	30	23	76.7	6	3	50.0
		5722	38	30	78.9	31	17	54.8
		6775	43	34	79.1	34	12	35.3
80–89.9.....	6	5690	27	22	81.5	22	14	63.6
		5678	27	23	85.2	19	17	89.5
		7529	40	35	87.5	32	27	84.4
		6686	17	15	88.2	28	21	75.0
		5710	45	40	88.4	21	20	95.2
90–99.9.....	2	5646	37	34	91.9	13	10	76.9
		5650	39	37	94.9	21	16	76.2
Total.....	59	(10)	343	293	85.4	227	157	69.2

sets of dams the results indicate a genetic difference between the dams. When resistant males were used, no significant difference was found between the survival of progeny from susceptible and resistant dams, the averages being 78.5 percent and 79.8 percent respectively.

Crosses of Chinese and American Strains.—To ascertain whether the susceptibility to pullorum disease found in a Chinese strain of chickens was produced by the same genetic factors as susceptibility in

TABLE 11.—SURVIVAL OF CHICKS FROM RESISTANT AND SUSCEPTIBLE DAMS MATED TO THE SAME SIRE

Sire No.	Chicks from susceptible dams			Chicks from resistant dams		
	Number inoculated	Number surviving	Percent surviving	Number inoculated	Number surviving	Percent surviving
Susceptible						
F1289	115	30	26.1	113	61	54.0
F1290	54	34	63.0	55	45	81.8
G2768	106	51	48.1	134	102	76.1
G2831	69	41	59.4	87	76	87.4
G8203	54	26	48.2	61	48	78.7
Total	398	182	45.7	450	332	73.8
Resistant						
70	28	17	60.7	61	50	82.0
578	39	28	71.8	15	12	80.0
1639	39	31	79.5	18	12	66.7
2133	67	55	82.1	24	21	87.5
2134	57	33	57.9	28	12	42.9
2537	99	86	86.9	34	32	94.1
5013	43	40	93.0	30	28	93.3
5546	47	44	93.6	69	52	75.4
5681	26	22	84.6	44	40	90.9
8163	67	46	68.7	34	26	76.5
Total	512	402	78.5	357	285	79.8

TABLE 12.—RESISTANCE OF CROSSES OF CHINESE AND AMERICAN STRAINS OF CHICKENS INOCULATED WITH S. PULLORUM

Cross	Number inoculated	Number surviving	Percent surviving
Susceptible × Langshan	31	2	6.5
Langshan × resistant	22	16	72.5
Resistant	884	643	72.7
Langshan	198	21	10.6
Canadian Rhode Island Red	191	28	14.7

an American strain, a susceptible American strain (G), which showed a survival of 14.7 percent was crossed with the Langshan, a susceptible strain from China which had shown a survival of 10.6 percent. The survival of the offspring from this cross when inoculated with *S. pullorum* was only 6.5 percent (Table 12). If these two strains had possessed different genetic factors for susceptibility, their progeny would probably have been more resistant than either one of the strains, rather than less resistant. Evidently they possessed the same genetic factors.

When the susceptible Langshan strain was crossed with a resistant Chinese strain, 72.5 percent of the progeny survived inoculation. The survival of the resistant Chinese strain was 72.7 percent. The genetic factor or factors for resistance would thus seem to be dominant.

The progeny from these parental stocks and crosses were obtained during the same season but not at the same time.

Nature of Resistance and Susceptibility

The results of the various crosses just described seem to indicate that resistance to inoculation with *Salmonella pullorum* is dominant to susceptibility. Of more importance, however, than the mere expression of resistance and susceptibility in the transmission of disease is the cause of such resistance or susceptibility. About this little is known. Pullorum disease is largely, so far as mortality is concerned, a disease of young chicks. Even in a susceptible strain, unless infection occurs within a very few days after hatching, the mortality is not high. According to Hanks and Rettger,[10*] the disease in young chicks appears to be a septicemia. Apparently some profound change occurs early in the life of the young chick causing it to become more resistant to the organism responsible for this disease. A preliminary note on blood studies of resistant and susceptible strains by Quisenberry, Roberts, and Card[29*] is of interest in this connection (Tables 13 and 14).

Blood samples from the progeny of susceptible Rhode Island Reds and resistant White Leghorns in three different hatches were studied 3, 6, 7, and 9 days after hatching. These birds had not been inoculated with *S. pullorum*. In six out of seven examinations the number of erythrocytes was greater for the resistant than it was for the susceptible strain (Table 13). In every hatch the leucocyte count was lower for the resistant than for the susceptible strain. Also the percentage of neutrophiles was lower in the resistant birds in six out of seven hatches. Hutt[12*] obtained the same general results except that he found a higher erythrocyte count in Rhode Island Reds than in White Leghorns.

In similar studies made on blood from chicks inoculated by mouth with 1/4 cc. of a 24-hour broth culture of *S. pullorum* (Table 14), the general relations among the red cells, white cells, and neutrophiles of the resistant and susceptible inoculated birds were the same as among the resistant and susceptible noninoculated chicks, but the degree of difference among the percentages of neutrophiles was changed. The neutrophilic percentage among the inoculated resistant birds was somewhat higher than among the noninoculated resistants of the same age, yet the difference was much less than that between the inoculated and noninoculated susceptible birds. The percentage of neutrophiles was high in the 3-day-old chicks and decreased with age, the 9-day-old chicks having the lowest percentage. This was true of the noninoculated susceptibles, noninoculated resistants, and inoculated resistants. Among the inoculated susceptibles the percentage of neu-

TABLE 13.—NUMBER OF ERYTHROCYTES AND LEUCOCYTES AND PERCENTAGE OF POLYMORPHONUCLEAR NEUTROPHILES IN THE BLOOD OF SUSCEPTIBLE AND RESISTANT CHICKS NOT INOCULATED WITH S. PULLORUM

Hatch No.	Breed	Number of chicks	Age in days	Erythrocytes per cubic millimeter	Leucocytes per cubic millimeter	Percent polymorphonuclear neutrophiles
1	Rhode Island Red	6	3	2 910 000	45 000	59.8
	White Leghorn	5	3	3 160 000	40 000	54.2
2	Rhode Island Red	6	3	2 835 000	63 000	60.7
	White Leghorn	6	3	2 945 000	37 000	53.3
3	Rhode Island Red	6	3	2 560 000	31 000	67.9
	White Leghorn	6	3	3 095 000	18 000	58.2
3	Rhode Island Red	6	6	2 630 000	29 000	37.6
	White Leghorn	6	6	3 035 000	16 000	35.8
1	Rhode Island Red	6	7	2 815 000	40 000	31.6
	White Leghorn	6	7	2 730 000	37 000	36.8
2	Rhode Island Red	6	7	2 630 000	29 000	43.9
	White Leghorn	6	7	2 885 000	19 000	38.6
3	Rhode Island Red	6	9	2 753 000	46 000	39.0
	White Leghorn	6	9	2 905 000	17 000	29.6

TABLE 14.—NUMBER OF ERYTHROCYTES AND LEUCOCYTES AND PERCENTAGE OF POLYMORPHONUCLEAR NEUTROPHILES IN THE BLOOD OF SUSCEPTIBLE AND RESISTANT CHICKS INOCULATED WITH S. PULLORUM

Hatch No.	Breed	Number of chicks	Age in days	Erythrocytes per cubic millimeter	Leucocytes per cubic millimeter	Percent polymorphonuclear neutrophiles
1	Rhode Island Red	6	3	2 845 000	41 000	62.2
	White Leghorn	5	3	3 035 000	47 000	53.4
2	Rhode Island Red	6	3	3 065 000	46 000	66.1
	White Leghorn	6	3	3 620 000	35 000	67.9
3	Rhode Island Red	6	3	2 630 000	24 000	68.3
	White Leghorn	6	3	2 830 000	23 000	76.8
3	Rhode Island Red	6	6	2 480 000	30 000	52.7
	White Leghorn	6	6	2 690 000	18 000	43.7
1	Rhode Island Red	6	7	2 565 000	75 000	62.5
	White Leghorn	6	7	2 410 000	49 000	39.7
2	Rhode Island Red	6	7	2 640 000	55 000	58.5
	White Leghorn	6	7	2 685 000	41 000	37.2
3	Rhode Island Red	6	9	2 300 000	51 000	55.8
	White Leghorns	6	9	2 490 000	22 000	30.7

trophiles was much higher at the ages of 6, 7, and 9 days than was the percentage among the inoculated resistants of the same ages.

This higher percentage of neutrophiles in the inoculated susceptibles compared with the inoculated resistants suggests that percentage of neutrophiles may be used as a measure of the relative resistance of different strains of chickens to *S. pullorum*.

Results of Agglutination Tests

In the fall of 1934, the last year of the experiments reported herein, all the birds in the strains selected for their resistance to *Salmonella pullorum* were tested by the whole-blood stained-antigen method for the presence of infection with this organism. Table 15 gives the results of these tests.

Of 271 birds tested, only 9 showed a positive reaction to the test. This may indicate a relatively high resistance in the selected birds.

TABLE 15.—REACTION OF TWO RESISTANT STRAINS OF CHICKENS TO AGGLUTINATION TESTS, 1934

	Sex	Age	Number	Reaction	
				Positive	Negative
Strain I.....	Female	− 1 year	39	3	36
	Male	− 1 year	5	0	5
	Female	+ 1 year	15	3	12
	Male	+ 1 year	2	0	2
Strain III.....	Female	− 1 year	136	0	136
	Male	− 1 year	16	0	16
	Female	+ 1 year	52	3	49
	Male	+ 1 year	6	0	6
Total.....	.....	.....	271	9	262

At no time during these tests was any attempt made to eliminate the reactors from the flocks, for the possibility of resistant individuals giving positive reactions to the test was recognized. However, in view of the results shown in Table 15 there is no reason to believe that resistant individuals, unless they are infected, give other than negative reactions to this test.

The fact that so few positives were found in the stocks selected for resistance, especially since these individuals came from ancestry that had survived inoculation, indicates that this stock did possess a high degree of resistance to pullorum disease.

Was Resistance Acquired or Natural

One usual explanation of resistance to a disease is that the individual at some time in its life cycle, either prenatally or postnatally, has been infected with the disease, unknown to the observer, and has thus established an immunity which may be mistaken for natural resistance.

In these experiments the chicks were inoculated so short a time

after hatching that it would have been impossible for them to acquire immunity between hatching and inoculation. If they did acquire immunity, they must have acquired it during embryonic development. This would mean that the pullorum organism was present in the egg.

It is well established that the organism of pullorum disease can be found in some eggs produced by infected hens. The resistant parental stock in this experiment was tested by means of the agglutination test. If the resistance shown by the progeny was acquired as the result of previous infection and was not a natural resistance, the offspring of reacting (infected) mothers would have shown a survival value higher than that found among offspring from nonreacting (noninfected) mothers. However, the survival among the progeny of reactors was no greater than among the progeny of nonreactors. In one of the resistant strains 67.6 percent of 142 progeny from 13 reactors survived inoculation. Among 1,341 progeny from 92 nonreactors 70.0 percent survived. Acquired immunity could not, therefore, have been involved in the results of these experiments.

BIOLOGICAL ASPECTS OF METHODS OF DISEASE CONTROL

If resistance and susceptibility to disease are hereditary, the genetic improvement of any animal population is retarded by any method of disease control which prevents natural selection. If hereditary factors are involved in resistance and susceptibility, then we must conclude that present methods of disease control[a] tend to prevent improvement in the hereditary constitutions of our animal populations, for under present methods susceptible individuals are saved and the percentage of resistant individuals in the population is not increased.[b]

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[a]No evidence is available to show that protection from disease which results from vaccination, sanitation, or the use of serums or bacterins improves in any way the quality of succeeding generations. So far as is known, acquired characters of this kind have no effect upon the germ plasm.

[b]The importance of hereditary constitution is likewise being recognized in the problem of human health. Faust[6]* (1934) says: "Without any disparagement of the splendid endeavors of men who have risked their lives to protect their fellows from disease it is time we pause to consider the final results of artificial protection alone without the fight which the body as a mechanism is prepared to wage against parasite invaders. It is obvious that natural and acquired resistance, and immunity are by far the more important to the human race. Let us recognize the interdependence and relative values of both types of defense and by rational as well as altruistic programs perpetuate the race as well as the individual. By so doing we can more surely guarantee a continuity on a higher plane of the contributions which are constantly being made to human knowledge and happiness."

Improvement in our animal populations made by increasing their resistance to disease would be very desirable from the standpoint of disease prevention. The necessity of utilizing present methods of disease control should not be minimized, but the importance of permanently improving the hereditary constitutions of our animals, and the necessity for so doing must be recognized as one of the urgent problems in the field of animal breeding.

When man displaces natural processes which are biologically beneficial, it is necessary for him to replace them by others of his own creation which are also biologically beneficial unless he is to suffer from the changes which he has instituted. The outcome of these biological situations depends largely upon the intelligence which man applies to them.

The three general methods of combating disease are prevention, cure, and genetic control. Which of these methods should be used under given circumstances is in part a question of which is most economical. Definite methods of cure or prevention that have already been worked out may, in some situations, be more economical than the genetic production of a resistant strain of animals. It must be remembered, however, that when methods of prevention and cure are relied upon, they must be in operation continuously unless the infecting organism can be entirely eliminated, which is unlikely with most diseases. The genetic method of control is more likely to be economically feasible with small animals which reproduce rapidly and have a low per-capita value than it is with the larger animals.

In all probability a combination of the genetic method and the preventive and curative methods now in use will produce better results than those produced by any one method alone. It should also be remembered that any progress toward hereditary resistance will mean easier and better functioning of the usual methods of disease control.

The processes by which hereditarily resistant strains of animals can be produced are so complicated and require so much time that the work of developing such animals cannot be done economically by the average animal producer. Some institution such as the agricultural experiment station must undertake it. After resistance and susceptibility to a given disease are established as hereditary, then the character resistance must be combined with other commercially desirable characters of the breed. And finally, such animals must be subjected to the conditions under which they would have to live on the farm, before being distributed to commercial breeders.

The genetic approach to disease control is of such recent origin that time has not yet permitted the formation of resistant strains of any farm animals for distribution as breeding stock.

SUMMARY AND CONCLUSIONS

From this experiment, ten years in duration and involving more than 29,000 birds, evidence has been obtained which clearly indicates that heredity is an important factor in resistance and susceptibility to infection with *Salmonella pullorum.* The existence of hereditary factors for resistance and susceptibility to pullorum disease is shown by the following results:

1. Selection was effective in producing strains of the domestic fowl more resistant than were unselected stocks in respect to infection by *Salmonella pullorum.*

2. The selected stocks were consistent in maintaining resistance thru successive generations.

3. The F_1 generation produced by crossing resistant and susceptible stock was as resistant as the resistant parents.

4. Progeny of the F_1 individuals mated to resistant were significantly more resistant than were the progeny of the back-cross to susceptible.

5. In the F_2 generation susceptible and resistant strains were recovered by selection.

6. A susceptible male mated to susceptible females produced progeny which were much less resistant than were progeny of the same male mated to resistant females.

7. No significant difference was found between the progeny of susceptible and resistant females mated to the same resistant male.

8. Acquired immunity was not present in the experimental birds, the progeny of infected hens exhibiting no greater resistance to disease than the progeny of noninfected hens, infection and freedom from infection being determined by the agglutination test.

9. Resistance is dominant to susceptibility, but probably more than one gene is involved.

10. In an examination of the blood of noninoculated young chicks the number of erythrocytes was found to be greater in the resistant (6 out of 7 cases) than in the susceptible strain. The number of leucocytes was greater in the susceptible strain. The percentage of neutrophiles was lower in the resistant individuals (6 out of 7 cases). In inoculated chicks, the percentage of neutrophiles was much higher among the susceptibles at 6, 7, and 9 days than among inoculated resistant individuals of the same ages.

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APPENDIX

TABLE 16.—SURVIVAL OF CHICKS FROM SELECTED AND CONTROL STOCKS AFTER INOCULATION WITH S. PULLORUM

(All hatches with 15 or more chicks in each of the selected and control groups are included)

Strain and date of hatch	Selected stock			Control stock		
	Number inoculated	Number surviving	Percent surviving	Number inoculated	Number surviving	Percent surviving
Strain I						
5-12-26	51	22	43.1	65	1	1.5
5-19	51	29	56.9	74	5	6.8
5-26	58	25	43.1	187	11	5.8
6-2	70	58	82.9	133	28	21.0
6-9	60	25	41.7	47	6	12.2
6-16	60	37	61.7	88	10	11.4
3-9-27	33	27	81.8	36	10	27.8
3-16	35	16	45.7	34	4	11.8
3-23	60	33	55.0	15	2	13.3
3-30	94	50	53.2	32	4	12.5
4-6	133	88	66.2	64	9	14.1
4-13	53	32	60.4	124	57	46.0
4-20	111	68	61.3	72	25	34.7
4-27	134	103	76.9	92	48	52.2
5-4	138	118	85.5	79	49	62.0
5-11	185	140	75.7	81	24	29.6
5-18	126	89	70.6	62	48	77.4
5-25	137	99	72.3	54	33	61.1
6-1	144	111	77.1	61	34	55.7
6-8	159	110	69.2	75	45	60.0
6-15	75	58	77.3	57	36	63.2
4-25-28	25	17	68.0	28	15	53.6
5-2	33	29	87.9	18	10	55.6
5-9	34	27	79.4	19	10	52.6
5-16	61	48	78.7	43	22	51.2
5-23	60	56	93.3	38	24	63.2
5-30	63	53	84.1	62	31	50.0
6-6	48	40	83.3	51	28	54.9
4-24-29	29	18	62.1	59	7	11.9
5-1	43	19	44.2	18	3	16.7
5-8	34	15	44.1	17	6	35.3
5-15	23	18	78.3	68	23	33.8
5-22	24	8	33.3	180	34	18.9
5-29	29	16	55.2	139	30	21.6
3-20-30	24	17	70.8	100	13	13.0
3-27	41	31	75.6	77	23	29.9
4-3	45	33	73.3	112	34	30.4
4-10	46	40	87.0	68	13	19.1
4-17	36	32	88.9	118	30	25.4
4-24	45	44	97.8	82	44	53.7
5-1	30	24	80.0	90	20	22.2
5-8	30	24	80.0	91	51	56.0
5-15	54	50	92.6	125	96	76.8
5-22	65	47	72.3	116	45	38.8
5-29	54	47	87.0	123	57	46.3
Strain II						
3-13-30	48	35	72.9	86	6	7.0
3-20	60	46	76.7	100	13	13.0
3-27	71	55	77.5	77	23	29.9
4-3	87	69	79.3	112	34	30.4
4-10	83	81	97.6	68	13	19.1
4-17	78	64	82.1	118	30	25.4
4-24	82	75	91.5	82	44	53.7
5-1	73	56	76.7	90	20	22.2

(Continued on page 492)

TABLE 16.—*Continued*

Strain and date of hatch	Selected stock			Control stock		
	Number inoculated	Number surviving	Percent surviving	Number inoculated	Number surviving	Percent surviving
Strain III						
3-20-30	50	42	84.0	100	13	13.0
3-27	44	38	86.4	77	23	29.9
4-3	45	37	82.2	112	34	30.4
4-10	39	35	89.7	68	13	19.1
4-17	48	45	93.7	118	30	25.4
4-24	35	31	88.6	82	44	53.7
5-1	40	27	67.5	90	20	22.2
Strain I						
4-22-31	33	19	57.6	61	1	1.6
4-29	30	7	23.3	54	3	5.6
5-6	39	14	35.9	77	2	2.6
5-13	48	29	60.4	57	2	3.5
5-20	53	32	62.3	54	1	1.9
5-27	42	34	81.0	49	4	8.2
6-3	34	27	79.4	66	10	15.2
6-10	26	20	76.9	73	5	6.8
6-17	40	38	95.0	65	15	23.1
Strain II						
4-1-31	46	20	43.5	61	2	3.3
4-8	37	11	29.7	65	0	0.0
4-15	57	9	15.8	74	2	2.7
4-22	72	26	36.1	61	1	1.6
4-29	78	40	51.3	54	3	5.6
5-6	86	20	23.3	77	2	2.6
5-13	118	48	40.7	57	2	3.5
5-20	114	77	67.5	54	1	1.9
5-27	96	62	64.6	49	4	8.2
6-3	94	68	72.3	66	10	15.2
6-10	91	69	75.8	73	5	6.8
6-17	84	77	91.7	65	15	23.1
Strain III						
4-1-31	39	15	38.5	61	2	3.3
4-8	31	21	67.7	65	0	0.0
4-15	41	16	39.0	74	2	2.7
4-22	47	24	51.0	61	1	1.6
4-29	48	26	54.2	54	3	5.6
5-6	48	16	33.3	77	2	2.6
5-13	68	43	63.2	57	2	3.5
5-20	68	49	72.1	54	1	1.9
5-27	49	39	79.6	49	4	8.2
6-3	43	36	83.7	66	10	15.2
6-10	36	32	88.9	73	5	6.8
6-17	57	49	86.0	65	15	23.1

TABLE 16.—*Concluded*

Strain and date of hatch	Selected stock			Control stock		
	Number inoculated	Number surviving	Percent surviving	Number inoculated	Number surviving	Percent surviving
Strain II						
5-18-32	41	41	100.0	143	98	68.5
5-25	63	40	63.5	116	25	21.6
6-1	70	57	81.4	117	42	35.9
6-8	67	56	83.6	114	83	72.8
6-15	56	47	83.9	104	74	71.2
Strain III						
5-18-32	44	42	95.5	143	98	68.5
5-25	50	40	80.0	116	25	21.6
6-1	44	33	75.0	117	42	35.9
6-8	48	45	93.8	114	83	72.8
6-15	30	26	86.7	104	74	71.2
Strain I						
5-3-33	47	30	63.8	81	2	2.5
5-10	41	31	75.6	95	34	35.8
5-17	41	27	65.9	129	39	30.2
5-24	34	27	79.4	104	36	34.6
5-31	35	32	91.4	99	39	39.4
6-7	34	23	67.6	125	33	26.4
6-14	26	16	80.0	79	43	54.4
Strain II						
5-3-33	98	61	62.2	81	2	2.5
5-10	113	66	58.4	95	34	35.8
5-17	113	75	66.4	129	39	30.2
5-24	101	80	79.2	104	36	34.6
5-31	79	73	92.4	99	39	39.4
6-7	68	56	82.4	125	33	26.4
6-14	48	43	89.6	79	43	54.4
Strain III						
5-3-33	81	59	72.8	81	2	2.5
5-10	63	47	74.0	95	34	35.8
5-17	74	59	79.7	129	39	30.2
5-24	57	47	82.5	104	36	34.6
5-31	57	51	89.5	99	39	39.4
6-7	39	30	76.9	125	33	26.4
6-14	22	20	90.9	79	43	54.4
Strain II × *Strain III*						
5-9-34	90	73	81.1	37	7	18.9
5-16	154	138	89.6	22	3	13.6
5-23	149	126	84.6	32	7	21.9
5-30	147	133	90.5	32	8	25.0

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