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Animal and Plant Health Inspection Service

Foreign Animal Disease Report

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We are initiating a plan to reconstruct the FAD report completely. Therefore, your questions, comments, and suggestions would be very much appreciated. Please send them to

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Contents

Emergency Programs Activities

The primary responsibilities of the U.S. Department of Agriculture's (USDA) Animal and Plant Health Inspection Service (APHIS) are to protect American agriculture and its livestock from the ravages of disease, to prevent the quality of our food supply from being degraded, and to minimize the economic impact of disease and disasters on American agriculture. The principal responsibilities of the Emergency Programs unit in APHIS' Veterinary Services (VS) are to ensure prompt evaluation of suspect outbreaks, and, when a foreign animal disease is diagnosed, to coordinate a prompt and thorough eradication of an outbreak.

The mission of Emergency Programs is to prevent destructive and harmful effects on the health of animals and human populations in the United States from epizootics of foreign and emerging animal diseases and from natural and technological disasters, such as hurricanes, floods, nuclear fallout and chemical fallout. To accomplish this, VS has developed and maintained a high level of expertise and preparedness and leads and coordinates rapid response efforts to safeguard the well-being of our animal populations and enhance public health.

Field Investigations—1996

During fiscal year (FY) 1996 (October 1, 1995–September 30, 1996), veterinary medical officers from USDA, APHIS, VS, and State departments of agriculture conducted 420 investigations of suspicious foreign animal diseases in the United States to explore the possibility that an exotic disease might have been introduced. Seventy-four investigations were conducted in the Central Region, 100 in the Northern Region, 50 in the Southeastern Region, and 196 in the Western Region. These diseases or conditions included abortion, avian diseases, acute death, central nervous system (CNS) disorders, myiasis and ascariasis, mucosal disorders, respiratory disorders, septicemia, and vesicular conditions. VS personnel also performed surveillance activities in regard to bovine spongiform encephalopathy (BSE). These figures do not include the field investigations done for vesicular stomatitis in FY 1996.

Field Investigations—1997

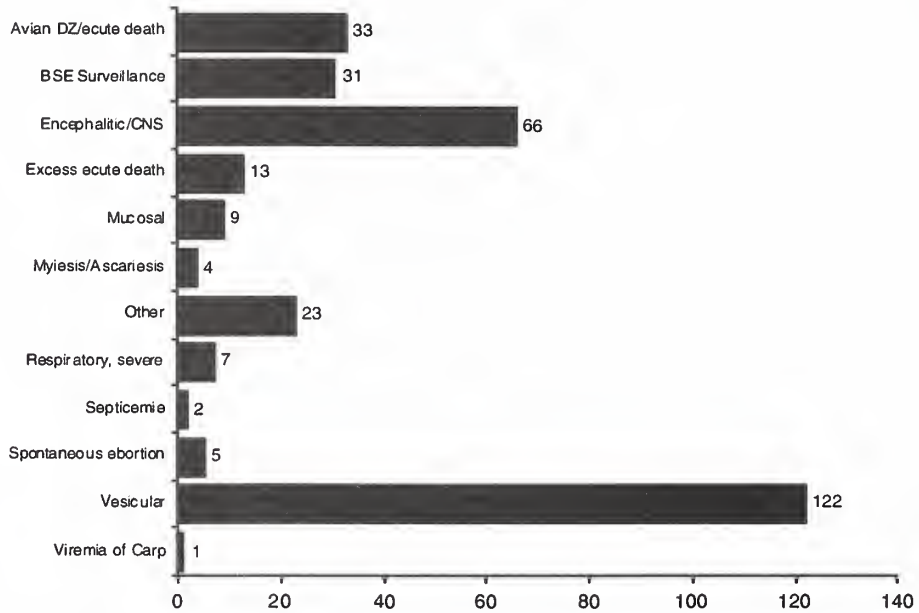
During FY 1997, 718 foreign animal disease (FAD) investigations were conducted (316 routine FAD investigations and 402 investigations for vesicular stomatitis). Chart 1 illustrates the number of investigations for FY 1997 by the condition investigated. These values do not include vesicular investigations from May 18 to September 30, 1997. That notwithstanding, the majority of investigations were for vesicular conditions (122) with encephalitic and CNS conditions (66) next most frequent.

Chart 2 shows the number of routine investigations by species for FY 1997. The largest number of investigations for suspected FAD's was 129 for bovines, and equines came in second with a total of 88 investigations. Other species investigations were as shown.

Chart 1

Routine FAD Investigations*—By Condition Investigated

October 1, 1996, to September 30, 1997



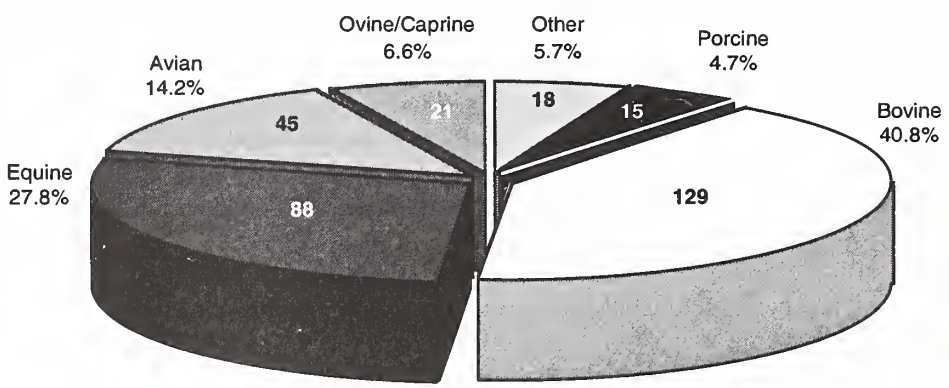
*Does not include Vesicular investigations from May 18, 1997, to September 30, 1997.

Chart 2

Surveillance for FY 1997

Routine FAD Investigations*—By Species

October 1, 1996, to September 30, 1997

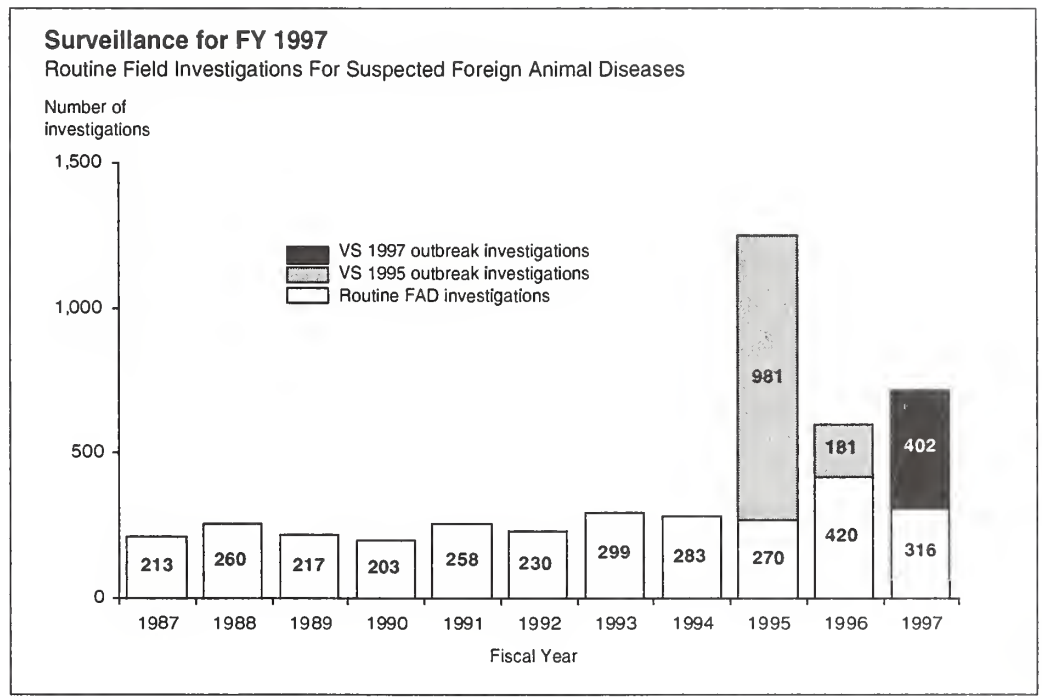


*Does not include vesicular investigations from May 18, 1997, to September 30, 1997.

Total: 316

Chart 3 presents the numbers of routine field investigations for suspected FAD's that have occurred over the past 10 years, reported by fiscal year. Total investigations for 1995 increased by over 300 percent with a total of 981 investigations for vesicular stomatitis virus (VSV) alone. The number of routine FAD investigations increased in 1996, during which 181 investigations for VSV took place. There was a decrease in routine FAD investigations in 1997 and an overall increase in the total number of investigations as the result of the VSV outbreak during the year.

Chart 3



Regional Emergency Animal Disease Eradication Organization (READEO) Update

Reorganization—As part of the USDA's overall efforts to colocate, restructure, and "right-size," APHIS is restructuring VS from four regions to two. In anticipation of this restructuring, VS has decided to take a proactive posture and immediately begin the restructuring of the READEO from four organizations to two. This will enable VS to have a stable emergency response posture, should an exotic or new animal or poultry disease emerge during the VS restructuring. The agency will benefit significantly through cost reductions by maintaining only two organizations rather than the previous four and will better be able to allocate reduced training dollars.

Dr. Tom Holt, Assistant Regional Director for VS, Northern Region, has been named director for the eastern READEO, and Dr. Jan Huber, Assistant Regional Director for VS, Central Region, has been named director for the Western READEO. VS has also taken this opportunity to enhance the previous READEO structure. Each READEO will have a new position of assistant READEO director. The previous structure of field operations and technical and administrative support has been maintained, although some of the subunits have been shifted among the three units. Another addition to the READEO is the early response team (ERT)—an FAD diagnostician, an epidemiologist, and a pathologist from the National Veterinary Services Laboratories (NVSL). This team will be called upon to assist the Regional Director, Area Veterinarian-in-Charge, and the State official when an emergency animal disease situation is being evaluated with the possibility of activating the READEO.

FAD Update

This update consolidates information from the Office of International Epizootics (OIE) bulletins dated **January through April 1997.** Countries reporting disease outbreaks are listed below the appropriate disease heading followed the by month and year of the outbreaks and the total number of outbreaks for that period. The notation "+" indicates that the presence of disease was reported without information on the number of outbreaks. A number followed by "+" indicates that specific outbreaks were reported during some months, whereas the distribution and occurrence of outbreaks were unknown during other months.

Foot-and-mouth disease (FMD)

Virus not typed

Bangladesh	10–12/96	+
Bhutan	3,4,9/96	2
Burkina Faso	8–12/96	8+
Cambodia	12/96–2/97	3
Georgia	8/96, 1–2/97	12
Ghana	7–12/96	19+
Hong Kong	11/96	1+
India	10/96	124
Kirghizstan	2/97	4
Laos	12/96–2/97	6
Mali	11,12/96–1/97	3+
Myanmar	11/96, 1–2/97	7
Pakistan	1–7/96,1–2/97	24+
Philippines	12/96	3
Qatar	1,3/97	12
Saudi Arabia	11/96	+
Senegal	12/96–1/97	6
Uganda	10,11/96	+
United Arab Emirates	3,4,6,11/96	4

Virus O

Afghanistan	6/96	+
Bhutan	1,2,7,10,11/96	6
Bolivia	12/96–1/97	3
Brazil	2/97	1+
Colombia	12/96-1/97	7+
Ecuador	1/97	1+
Georgia	7,9–12/96	19
Hong Kong	12/96, 2/97	1+
India	9,11,12/96–1/97	696
Iran	6/96–2/97	132+
Kuwait	3/97	+
Malaysia (peninsular)	4,12/96–3/97	14
Nepal	10–12/96	55+
Oman	11,12/96	11+
Pakistan	8–12/96, 3/97	8+
Palestinian Territories	1/97	3
Peru	3/97	1
Philippines	1–3/97	117+
Sri Lanka	1/97	24
Taipei China	3/97	1,300
Tanzania	7/96	+
Thailand	12/96–3/97	9
Turkey	12/96–3/97	9
Uganda	4–7,9,12/96	+
Vietnam	10/96, 2–3/97	8

Virus A

Albania	5/96	10
Bhutan	7/96	1
Bolivia	1/97	2+
Colombia	12/96–2/97	6+
Ecuador	2/97	1+
India	9,11,12/96–1/97	696
Iran	6/96–3/97	132+
Kenya	2/96	+
Pakistan	8–12/96	8+

Virus C

India	9,11,12/96–1/97	526
Kenya	2/96	+
Pakistan	10–12/96	3+

Virus SAT 2

Kenya	1–4/96	+
Uganda	4–9,12/96	+

Virus Asia 1

India	9,11/96, 1/97	514
Malaysia (peninsular)	4,5/96,12/96–2/97	15
Nepal	10–12/96	55+
Pakistan	8–12/96	8+

Vesicular Stomatitis

Virus not typed		
Costa Rica	12/96–1/97	10
Honduras	2,4–5,7–11/96	15
Peru	12/96, 2–3/97	7
Virus Indiana		
Colombia	12/96–2/97	90
Mexico	1/97	2
Panama	1–2/97	4
Peru	11/96, 1/97	10
Venezuela	1,2/97	5
Virus New Jersey		
Colombia	12/96–2/97	90
Costa Rica	2/97	1
Ecuador	12/96	3
Honduras	6/96, 1–2/97	12
Mexico	1–12/96	33
Panama	3/97	1
Peru	1/97	8
Venezuela	10,11/96, 1–3/97	12

Swine Vesicular Disease

Italy	1,2/97	3

Rinderpest

Kenya	12/96	3
Pakistan	1–12/96—2/97	+
Saudi Arabia	11,12/96	+
Tanzania	1/97	+

Peste des Petits Ruminants

Bangladesh	12/96	+
Burkina Faso	10/96	1
Central African Republic	3/96	1+
Cote d'Ivoire	1,2/97	+
Ghana	7–12/96	45
Guinea	10/96,12/96–3/97	+
India	4,10/96,12/96–1/97	20
Nepal	10–12/96	3+
Oman	11,12/96	14
Palestinian Territories	1/97	1
Saudi Arabia	9,11,12/96	+
Senegal	3/97	1
United Arab Emirates	4,5,7/96	3

Contagious Bovine Pleuropneumonia

Angola	10/96	4
Bangladesh	10–12/96	+
Burkina Faso	8–12/96	17
Cote d'Ivoire	1,2/97	+
Ghana	9/96	2
Guinea	10/96,12/96–3/97	9+
Mali	11/96–3/97	7
Namibia	12/96–2/97	2+
Pakistan	4–12/96	+
Qatar	3/97	2
Uganda	4–12/96	+

Lumpy Skin Disease

Ghana	7–9/96	7
Guinea	10/96,12/96–3/97	+
Madagascar	10,11/96	12
Senegal	1/97	1
South Africa	1–3/97	112
Swaziland	12/96–1/97	+
Uganda	5–12/96	+
Zimbabwe	11,12/96	6

Rift Valley Fever

Mozambique	12/96–2/97	+
South Africa	3/97	1
Uganda	7/96	+

Bluetongue

Guinea	10/96,12/96–3/97	+
India	9–12/96—1/97	487
Mozambique	12/96–2/97	+
Pakistan	1–12/96	+
Saudi Arabia	9,12/96	+
South Africa	1–3/97	17
United States	12/96–3/97	+

Sheep Pox and Goat Pox

Algeria	1–3/97	11
Bangladesh	10–12/96	+
China	11/96–1/97	5
Georgia	2/97	1
Greece	12/96–1/97	19
India	10–12/96—1/97	101
Iran	6/96—1/97	71
Kazakhstan	10/96	1
Kirghizstan	8,9/96,12/96–2/97	8
Libya	11,12/96	+
Mozambique	12/96	+
Oman	1/97	5
Pakistan	12/96–2/97	4+
Palestinian Territories	1/97	4
Qatar	3/97	3
Russia	11/96	1
Saudi Arabia	11,12/96	+
Tunisia	11/96–1/97	9
Turkey	12/96–3/97	12
United Arab Emirates	1,9,11/96	3

African Horse Sickness

Mozambique	12/96–2/97	+
Senegal	1/97	1
South Africa	1/97	2
Zimbabwe	1–5/96	8

African Swine Fever

Cote d'Ivoire	1,2/97	+
Italy	2–4/97	4
Mozambique	12/96–2/97	+
Senegal	2/97	1
South Africa	4/97	2
Uganda	4,5,8,10–12/96	+

Classical Swine Fever (Hog Cholera)		
China	11/96–1/97	28+
Colombia	2/97	1
Cuba	1/97	5
Czech Republic	2/97	1
Federal Republic of Yugoslavia (Serbia and Montenegro)	11/96–2/97	13
Germany	1–4/97	35
Honduras	6/96,9/96–1/97	4
Hong Kong	10/96	1
India	9/96–1/97	57
Indonesia	10–12/96	+
Italy	1–4/97	13
Korea	12/96–2/97	9
Laos	10–12/96	+
Madagascar	10,11/96	2
Malaysia (peninsular)	12/96–4/97	2
Mexico	1–3/96,6/96–1/97	27
Myanmar	12/96	+
Netherlands	2–4/97	162
Peru	11/96–2/97	2
Philippines	7–9/96	+
Russia	11/96	4
Slovakia	1–3/97	15
Spain	4/97	10
Sri Lanka	12/96	1
Taipei China	1,2/97	15
Venezuela	11/96	1
Vietnam	10–12/96	+

Highly Pathogenic Avian Influenza		
Laos	10–12/96	+
Myanmar	11,12/96	+
Pakistan	1–12/96	+

Newcastle Disease		
Virus not characterized		
Algeria	4/97	10
Bangladesh	10–12/96	+
Central African Republic	10/96	1
Colombia	2/97	2
Cote d' Ivoire	1,2/97	+
Fedral Republic of Yugoslavia (Serbia and Montenegro)	11,12/96	2
Ghana	7–12/96	129
Great Britain	12/96–4/97	9
Guinea	10/96,12/96–3/97	+
Honduras	3,5,6/96	3
Hong Kong	10/96	1+
India	9/96–1/97	222
Indonesia	10–12/96	+
Iran	6–12/96—1/97	287
Jordan	10–12/96—3/97	+
Korea	1–3/97	13
Laos	10–12/96	+
Madagascar	10,11/96	13
Malaysia (peninsular)	12/96, 2/97	3
Myanmar	10–12/96	+
Namibia	1,3/97	3
Nepal	10–12/96	11+
Northern Ireland	2,3/97	21
Norway	12/96	1
Pakistan	1/96–2/97	4+
Philippines	7–9/96	+
Russia	12/96	1
Saudi Arabia	9–12/96	+
South Africa	1,3/97	3
South America	1/97	2
Sri Lanka	12/96–1/97	10
Swaziland	1,2/97	5
Turkey	1,2/97	3
Uganda	5–9,11/96	+
Unites Arab Emirates	1–3,6/96	4
Vietnam	10–12/96	+
Zimbabwe	11,12/96	7
Velogenic Virus		
Denmark	9/96	1
Great Britain	1/97	5
Kenya	11,12/96	2
Korea	12/96	2
Mexico	2,3,5,6,8/96	13
South Africa	2/97	4
Uganda	10,12/96	+
Mesogenic virus		
Mexico	1,2,9/96	7
Israel	4/97	1
Lentogenic virus		
Mexico	8,9,11/96	5

(Compiled by Ms. Christine Allen, secretary, and Dr. Richard Pacer, epidemiologist, USDA, APHIS, International Services (IS), Riverdale, MD, (301) 734–8892)

Foreign Animal Disease Review: Bovine Spongiform Encephalopathy

Bovine spongiform encephalopathy (BSE), widely known as "mad cow disease," is a chronic, degenerative disease affecting the CNS of cattle. It is one in a family of diseases known as the transmissible spongiform encephalopathies (TSE's). Worldwide, there have been more than 170,000 cases of BSE since it was first diagnosed in 1986 in Great Britain. BSE has had a substantial impact on the livestock industry in the United Kingdom. The disease has also been confirmed in native-born cattle in Belgium, France, Ireland, Luxembourg, the Netherlands, Northern Ireland, Portugal, and Switzerland. However, more than 98 percent of all BSE cases have occurred in Great Britain. BSE is not known to exist in the United States.

Owing to the nature of the disease, it is difficult to develop all-inclusive guidelines that would establish BSE-free status for a country. Currently, OIE has criteria to assist countries in evaluating the status of trading partners. This code was adopted in May 1997.

The lack of a preclinical screening test that definitively detects all BSE infection adds to the complexity of verifying the absence of the disease. Hence, an active and aggressive surveillance program is essential in any country wishing to claim freedom from BSE. Because many outside factors influence reporting, it is rarely possible to claim freedom from BSE on the basis of passive surveillance alone. In addition, the OIE guidelines take into account certain risk factors that may be present in countries. These risk factors include the importation of infected cattle feed contaminated with the BSE agent (Schreuder et al. 1997), the presence of other animal TSE's, and feeding practices, among others. For the same reason, it is virtually impossible to obtain a true picture of disease prevalence in a country where BSE has been detected.

TSE's are caused by a transmissible agent that is yet to be fully characterized. They share the following common characteristics:

- A prolonged incubation period of months or years;
- A progressive debilitating neurological illness that is always fatal;
- The presence of scrapie-associated fibrils (SAF's) when detergent-treated extracts of brain tissue from animals or humans affected by these diseases is examined by electron microscope;
- Pathological changes, such as vacuolation and astrocytosis, that appear to be confined to the CNS; and
- The absence of any detectable specific immune response in the host.

The lack of antibody formation in BSE-infected animals has inhibited the development of a live animal diagnostic test. Listed below are other TSE's.

Creutzfeldt–Jakob disease

Creutzfeldt–Jakob Disease (CJD) is a sporadic presenile dementia that affects humans. It occurs at an annual incidence of about one in a million of the population. About 90 percent of the cases are sporadic and have no known source of exposure. Five to 10 percent of the cases are familial and are associated with certain inherited gene mutations. An extremely small number of cases result from iatrogenic transmission through such mechanisms as corneal transplants, contaminated pituitary growth hormone injections, or the use of poorly disinfected brain electrodes (Brown 1988a, b).

New-variant Creutzfeldt–Jakob disease (nvCJD)

Twenty-four cases of nvCJD have been identified in the United Kingdom and France between 1994 and 1996. Unlike sporadic CJD, the cases were unique because the patients were younger than usual, the clinical manifestation was different, and the patients displayed a new neuropatho- logical profile. The Spongiform Encephalopathy Advisory Committee (SEAC) in the United Kingdom concluded that, although there was no direct scientific evidence of a link between BSE and CJD, on the basis of current data and in the absence of any credible alternative, the most likely explanation is that nvCJD is linked to exposure to BSE before the introduction of the specified bovine offal ban in 1989 (Will et al. 1996).

Fatal familial insomnia

An extremely rare human disorder characterized by trouble sleeping and disturbances of the autonomic nervous system. An inherited mutation in the PrP gene has been identified as the precipitating factor of this disease (Tateishi et al. 1995).

Gerstmann–Sträussler–Scheinker syndrome

A rare familial syndrome of humans caused by an inherited mutation in the PrP gene affecting about 50 extended families. The syndrome is characterized by loss of coordination and dementia (Prusiner 1995).

Kuru

A disease of the Fore tribe in the eastern highlands of Papua New Guinea. The disease is characterized by a loss of coordination followed by dementia. The infection was thought to spread through the practice of cannibalism and exposure to high-risk tissues such as brain. Since cannibalism was discontinued, kuru has essentially disappeared.

Chronic wasting disease (CWD)

CWD was first discovered in 1967 and reported by Williams and Young (1980) and originally was confined to mule deer and Rocky Mountain elk held in captivity in Colorado and Wyoming (Williams and Young 1982). CWD has more recently been confirmed in free-ranging cervids in Colorado and Wyoming, such as mule deer, white- and black-tailed deer, and elk. CWD has also been diagnosed in ranch-raised elk in South Dakota. The disease is characterized by emaciation, changes in behavior, and excessive salivation.

Feline spongiform encephalopathy

A naturally occurring TSE first reported in domestic cats in 1990 (Wyatt et al. 1990). More than 70 cases of FSE have been diagnosed in the United Kingdom since 1990. In addition, one case of has been identified in Norway (Bratberg et al. 1995) and one in Liechtenstein. Cats with the disease display locomotor disturbances, behavioral changes, and hypersensitivity to sudden movements or noises (Wyatt et al. 1991). Current evidence suggests that the disease in cats is most probably linked to BSE.

Scrapie

An insidious degenerative disease affecting the CNS of sheep and goats. The disease is also called la tremblante (French: trembling), Traberkrankheit (German: trotting disease), or Rida (Icelandic: ataxia or tremor). Scrapie was first recognized as a disease of sheep in Great Britain and other countries of Western Europe more than 250 years ago. Scrapie has been reported worldwide and affects most sheep-producing regions. Australia and New Zealand are commonly accepted to be scrapie free. The disease has been recognized for over two centuries in England, Wales, and Germany (Parry 1983). The first U.S. case of scrapie was diagnosed in 1947.

Transmissible mink encephalopathy (TME)

An uncommon disease of ranch-raised mink first reported in the United States in 1947 and described by Hartsough and Burger (1965). TME has been reported in Canada, Finland, Germany, Russia, and the United States. The last U.S. case was in Wisconsin in 1985 (Marsh and Hadlow 1992). It has been suggested that TME is a result of feeding either scrapie-infected sheep or infected cattle to mink.

Other cases of spongiform encephalopathy have been reported in kudus, elands, nyalas, gemsboks, and a few exotic cats. These occurrences are also thought to be linked to contaminated feed.

Etiology

The clinical, pathological, and molecular genetic features of BSE, as well as other TSE's, have led to speculation on the nature of the etiologic agent and the pathogenetic mechanisms of the disease. There are three main theories on the nature of the scrapie agent.

The Virus Theory—In this theory, the virus would have to have unusual biochemical and biophysical characteristics that would help explain its remarkable physicochemical properties (Rohwer 1984, Czub et al. 1988, Manuelidis et al. 1988).

The Prion Theory—Here the agent is believed to be composed exclusively of the host-coded protein (PrP^{c}), which becomes partially protease resistant (PrP^{BSE})—most likely through a posttranslational conformation change after infection. In this theory, there are no nonhost components of the agent. That is, a specific informational molecule (nucleic acid such as RNA or DNA) is not present (Prusiner 1982, Bolton and Bendheim 1988).

The Virino Theory—This theory states that the BSE agent consists of a host-derived protein coat (PrP is one of the candidates for this protective protein) and a small noncoding regulatory nucleic acid (Dickinson and Outram 1979, Kimberlin 1982).

Comparing the Theories—All three proposed theories have some degree of validity. Proponents of the virus and virino theories conclude that the existence of different scrapie strains unequivocally proves the existence of a nucleic acid component of the infectious agent, which, as in conventional viruses, may undergo mutations responsible for phenotypic variations. The problem with these two theories is that no agent-specific nucleic acid has been convincingly identified to copurify with infectivity (Manuelidis and Manuelidis 1981, Duguid et al. 1988, Oesch et al. 1988, Meyer et al. 1991, Sklaviadis et al. 1993). Moreover, chemical, enzymatic, or physical treatments that usually inactivate or degrade nucleic acids have no effect on the transmissible properties of the BSE infectious agent (McKinley et al. 1983; Bellinger Kawahara et al. 1987a,b; Neary et al. 1991). Possible reasons for this lack of effect are that the amount of nucleic acid of the putative agent is too small to be detected with available techniques and that its tight bond to the protein protects it from chemical or physical inactivation. Weakening the virus and virino theories are also the inability to identify any virus particles under the electron microscope (Bots et al. 1971, Cho and Greig 1975) and the failure of an infected host to generate an immune response. [It should be noted that small particles resembling virus structures have recently been observed by electron microscopy (Ozel and Diringer 1994).]

The prion model involves propagation of a protein-only agent (PrP^{BSE}) whereby PrP can assume various tertiary structures caused by a combination of host genetics and the introduction of altered (infectious) PrP. Hence, the structure of the infecting PrP^{BSE} imprints upon the normal cellular precursor (PrP^{c}), which results in a change of shape to the protease-resistant form. Researchers suspect that mutations in the PrP gene may render resulting proteins susceptible to "flipping," and the shape changes account for what is commonly referred to as "strain" differences. Several explanations for scrapie strain genetics in the context of the prion theory have been suggested, but not one has been proven (Prusiner 1991, Weissman 1991). A recent publication suggests that passaging the agent through different hosts causes the conformational change, possibly limiting prion diversity (Scott et al. 1997). However, this study could not eliminate the existence of a mixture of prion strains.

It should be pointed out that the prion theory fails to explain (1) how the PrP of the infecting agent originally assumed the aberrant structure associated with infectivity and (2) how the different structures originated as a function of the different strains. Although numerous scrapie strains can be differentiated in a single host (i.e., sheep), the PrP associated with these strains has not shown any biochemical and molecular differences. BSE seems to be caused by a single strain type. This BSE strain is different from historical or contemporary isolates from sheep or goats with natural scrapie as determined by study of incubation periods and brain "lesion profiles" in mice.

Regardless of whether the prion (PrP^{BSE}) is or is not the agent, the partially protease-resistant form is a marker of infection. Currently several of tests are available to detect the presence of the PrP^{BSE}.

Epizootiology

Different scientific hypotheses concerning the origins of BSE have been advanced. The epidemiologic data suggest that BSE in Great Britain is an extended, common-source epidemic involving feed containing TSE-contaminated meat and bone meal as a protein source. The causative agent is suspected to be from either scrapie-affected sheep or from cattle with a previously unidentified TSE.

Changes in rendering operations in the early 1980's—particularly the removal of a solvent-extraction process that included a steam-heat treatment—may have played a part in the appearance of the disease and the subsequent amplification of the agent in the food chain. A ban in Great Britain on feeding animal protein of ruminant origin to ruminants was enacted in July 1988 (Wilesmith et al. 1992).

In Great Britain, the epidemic peaked in 1992–93, when about, 1,000 cases were reported each week. In 1997, BSE remained on the decline, with approximately 100 cases reported weekly. Cases that have been detected in other countries appear to be a result of importations of live cattle or, more significantly, contaminated feed from the United Kingdom.

There is no evidence that BSE spreads horizontally, that is, by contact between unrelated adult cattle or from cattle to other species. Results of recent British research show very low levels of transmission of BSE from affected cows to their offspring. These results demonstrate about a 9-percent increase in the occurrence of BSE in offspring of BSE-affected dams as compared with calves born to dams that did not later demonstrate BSE infection. The study did not ascertain if this was the result of genetic factors or true transmission. The research did, however, point out that at this level, if maternal transmission does occur, it will not sustain the epidemic (Wilesmith et al. 1997).

A TSE has been diagnosed in eight species of captive wild ruminants as well as exotic and domestic cats. There have been more than 80 cases of feline spongiform encephalopathy (FSE) in Great Britain, 1 in a domestic cat in Norway, 1 in Northern Ireland, and 1 in Liechtenstein. The agent isolated from several of these cases using strain typing in mice is indistinguishable from BSE in cattle, suggesting that FSE is actually BSE in exotic and domestic cats. This also appears to be true for the other ruminants. Epidemiologic evidence suggests protein-enhanced feed from ISSE-infected carcasses to be the primary source of infection in these species (MAFF Progress Report, June 1997).

Some authorities have suggested that 24 cases (as of January 31, 1997) of a variant form of CJD (nvCJD) in Great Britain (U.K. Department of Health, March 1998) and France may be linked to exposure to BSE before the introduction of a specified bovine offal (SBO) ban at slaughter in 1989. The SBO ban excluded from human consumption brain, spinal cord, and other tissues with potential BSE infectivity.

BSE has been experimentally transmitted to the following species via intracerebral inoculation: cattle, sheep, goats (Foster et al. 1993), mink (Robinson et al. 1994), pigs (Dawson et al. 1990), marmosets (Baker et al. 1993), macaques (Lasmézas et al. 1996), and mice (Fraser et al. 1988). Intracranial transmission was attempted in hamsters but was not successful. BSE has been successfully transmitted via the oral route to cattle, sheep, goats (Foster et al. 1993), mice (Barlow and Middleton 1990), and mink (Robinson et al. 1994). Oral transmission has been attempted in swine. The inoculated swine were euthanized after 84 months of age and had not exhibited any signs of a TSE. Parenteral and oral transmission have also been attempted in chickens with no evidence of disease thus far, even 12 months into a second passage.

BSE and CJD—Human Health Concerns

On March 20, 1996, the SEAC announced the identification of 10 cases of nvCJD. All of the patients developed symptoms of disease in 1994 or 1995. The following points describe how these 10 cases differed from other routinely diagnosed cases of CJD:

- The affected individuals were much younger than the sporadic CJD patient. Typically, CJD patients are over 63 years old. The average patient age for the variant form of CJD is 27.5 (range of 16 to 42) years. (The term "sporadic" is used here, after Dr. John Collinge, to indicate classical CJD.)
- The course of the disease in the nvCJD averaged 13 months. Sporadic CJD cases average a 6-month duration.
- In the 10 victims, electroencephalographic activity was not typical of CJD.
- Although brain pathology was recognizable as CJD, the pattern was different from sporadic CJD, as manifested by large aggregates of prion protein plaques.

Epidemiologic and case studies have not revealed exposure to a common risk factor. According to the SEAC, all victims were reported to have eaten beef or beef products in the last 10 years, but none had knowingly eaten brain material. One of the affected individuals had been a vegetarian since 1991 (Will et al. 1996).

The SEAC concluded that, although there was no direct scientific evidence of a link between BSE and nvCJD, on the basis of current data and in the absence of any credible alternative, the most likely explanation was that the cases resulted from exposure to BSE before the introduction of control measures—in particular, the SBO ban in 1989.

Research reported in later 1996 and 1997 has found further evidence to link nvCJD to BSE. Two significant studies published in the October 2, 1997, edition of *Nature* led the SEAC to conclude that BSE agent is highly likely to be the cause of nvCJD. Dr. Moira Bruce and colleagues at the Institute for Animal Health in Edinburgh, Scotland, inoculated three panels of inbred mice and one panel of crossbred mice with BSE, nvCJD, and CJD. Mice inoculated with BSE showed the same pattern of incubation time, clinical signs, and brain lesions as mice inoculated with tissues from patients with nvCJD. This finding provides evidence that BSE and nvCJD have the same signature or are the same "strain." In addition, sporadic CJD and known scrapie strains were found not to be similar to nvCJD or BSE (Bruce et al. 1997).

The second set of results, published by Dr. John Collinge and colleagues of Imperial College School of Medicine, London, strongly supported Bruce's results. Collinge's paper reported findings of BSE transmission to transgenic mice expressing only human PrP (Hill et al. 1997).

Another paper by Collinge et al. in the October 24, 1996, edition of *Nature* discussed a molecular analysis of prion strain variation in relation to the etiology of nvCJD. Collinge and colleagues demonstrated that two of the three forms of CJD (sporadic and iatrogenic) can be distinguished from one another, after treatment by proteolytic cleavage, by differing band sizes using Western blot analysis. Types 1 and 2 are associated with different clinicopathological phenotypes of sporadic CJD, and type 3 is seen in iatrogenic CJD cases where there is a peripheral route of inoculation. Iatrogenic cases of CJD with a direct CNS exposure resemble the pattern manifested by sporadic CJD. The nvCJD, although resembling type 3 to some degree, can be differentiated from each of the three types by a specific pattern of band intensities.

Furthermore, glycoform patterns of the nvCJD were "closely similar to" patterns from wild-type mice inoculated with BSE and "closely resembled" those from FSE as well as patterns from macaques inoculated with BSE. However, natural BSE was not detected through Western blot analysis using the antibodies in the aforementioned studies (3F4 monoclonal or R073 polyclonal). The use of rabbit antibody to synthetic human PrP peptide (95–108) with BSE from its natural host, the cow, did generate a signal and glycoforms closely similar to transmitted BSE and nvCJD (Collinge et al. 1996).

The Health and Safety Executive in the United Kingdom now advises that BSE must be considered a biological agent (human pathogen) within the meaning of the Control of Substances Hazardous to Health Regulations 1994 (HSE Press Release, October 1997).

Actions Taken in the United Kingdom in Response to nvCJD

As a result of the identification of the variant form of CJD, the following measures have been put into place in the United Kingdom (MAFF Progress Report, June 1997):

- In November 1989, entry into the human food supply of any protein derived from bovine brain, spinal cord, tonsil, thymus, spleen, or intestine was prohibited. These tissues were referred to as SBO and now are referred to as specified bovine material (SBM).
- The spinal column and heads of bovine animals over 6 months old, except for the tongue, are to be treated as SBM. Such materials and their derivatives may not be used in cosmetic, pharmaceutical, or medicinal products.
- Mammalian meat and bonemeal are not to be incorporated into any feed for any farmed animals, including fish or horses, or into fertilizer likely to be used on land to which ruminants have access.
- Meat from animals over 30 months old is prohibited from entering the human or animal food supply.

- On July 24, 1996, MAFF announce its intention to introduce controls requiring the removal of sheep heads from the food chain following a study showing evidence that BSE could be isolated from spleens of sheep experimentally infected with BSE.
- In September 1996, the Heads of Sheep and Goats Order was put into effect.
- In January 1997, selective cull began. The selective cull targets cattle born on a farm around the same time as BSE cases born on that farm between July 1989 and June 1993 that would have been exposed to the same risk of infection by the BSE agent in feed.

Infectivity of Tissues, Products, and Body Fluids

In the naturally infected animals, the BSE agent has been identified in brain, spinal cord, and retina. Agent identification was by mouse bioassay. The route of inoculation into the mice was intracranial. The naturally infected animals were adult cattle exhibiting clinical signs of disease (Fraser et al. 1988).

Experiments with mice that were fed milk, mammary glands, placentas, lymph nodes, or spleens have failed to transmit the disease within the natural lifespan of the mice or to establish subclinical infection of the lymphoreticular system (Middleton and Barlow 1993).

Another study is ongoing to examine the pathogenesis of BSE by quantifying the replication (tissue distribution) of the agent during the incubation period. This study has identified the agent via mouse bioassay in the distal ileum of experimentally infected calves. It is thought that the agent may be associated with the lymphoid tissue of the intestines. The calves were 4 months of age at the time of oral dosing. First isolation of the agent in the distal ileum was made at 6 months after the challenge. Subsequent isolations from the distal ileum were made at 10, 14, and 18 months after dosing (Wells et al. 1994). Recently, this study has also disclosed infectivity in bone marrow and trigeminal and dorsal root gangli (UK MAFF Website).

More than 40 tissues have been examined from cattle using the mouse bioassay. It appears as if the distribution of the BSE agent is not as diverse as that of the scrapie agent in sheep. Another consideration that must be noted is the species barrier using the mouse bioassay. There is a possibility that the agent is present but is found in such very low levels that the bioassay is not sensitive enough to detect it (MAFF Progress Report, June 1997).

Clinical Signs

Cattle afflicted with BSE develop a progressive degeneration of the nervous system. Affected animals may display changes in temperament, abnormalities of posture and movement, and changes in sensation. More specifically, the signs include apprehension, nervousness or aggression, incoordination (especially hind-limb ataxia, tremor, and difficulty in rising), and hyperesthesia to sound and touch. In addition, many animals have decreased milk production and/or loss of body condition despite continued appetite. There is no treatment, and affected cattle die.

The incubation period ranges from 2 to 8 years. Following the onset of clinical signs, the animal's condition gradually deteriorates until the animal becomes recumbent, dies, or is destroyed. This usually takes from 2 weeks to 6 months. Most cases in Great Britain have occurred in dairy cows (Frisians) between 3 and 6 years of age (Wilesmith et al. 1992).

Diagnosis

The diagnosis of BSE is based on the occurrence of clinical signs of the disease and currently must be confirmed by postmortem laboratory testing. Histopathologic examination of brain tissue collected after the animal dies or is euthanized is the initial step in the diagnostic process. Bilaterally symmetrical degenerative changes are usually seen in the gray matter of the brain stem. These changes are characterized by vacuolation or microcavitation of nerve cells in the brain-stem nuclei. The neural perikarya and axons of certain brain-stem nuclei contain intracytoplasmic vacuoles of various sizes, giving the impression of a spongy brain. Hypertrophy of astrocytes often accompanies the vacuolation (Wells et al. 1987). A diagnosis may also be made by the detection of scrapie-associated fibrils using electron microscopy.

Supplemental tests are available to enhance the diagnostic capabilities for BSE. Research has shown that the partially protease-resistant form of the prion protein (PrP^{BSE}) is found in the brain of BSE-infected cattle. Two tests may currently be used to detect the PrP^{BSE}. These are immunohistochemistry and the Western blot technique. In the past, if the brain tissue was not harvested shortly after the animal's death autolysis made it very difficult to confirm a diagnosis. Both of these tests allow for the possibility of confirming a diagnosis of BSE even if the brain has been frozen or has autolyzed.

The potential live-animal tests, including some in development, are as follows:

- Tests specific for the partially protease-resistant form of the prion protein:
  (1) A capillary electrophoresis test, and
  (2) A Western blot test with increased sensitivity.

- Tests that identify unique substances of infected animals or humans:
  (1) A cyclic voltametric method that describes unique substances in urine, and
  (2) An immunoblot test describing unique substances in cerebrospinal fluid.

Tests for PrP^{BSE} in the preclinical cow may not be as successful as they are appearing to be in scrapie diagnosis because PrP^{BSE} has not yet been detected in peripheral tissues.

Differential diagnoses for BSE include rabies, listeriosis, nervous ketosis, milk fever, grass tetany, lead poisoning, and other toxicities or etiological agents that affect the nervous or musculoskeletal systems of adult cattle.

Treatment, Prevention, and Control

There is no known treatment for BSE or any of the TSE's, and there is no vaccine to prevent these diseases. BSE from foreign sources may be prevented by the implementation of import regulations prohibiting live ruminants and ruminant products (especially meat, bone meal, and offal). Because the origin of BSE remains unknown, preventing a domestic epidemic of BSE would involve, at a minimum, prohibiting the feeding of ruminant proteins to ruminants. The prevention program of any country should also include active surveillance for the early detection of BSE. Most countries have prohibited the importation of cattle and bovine products from countries known to have BSE. In addition, many countries have taken steps to enact regulations prohibiting the feeding of ruminant proteins to ruminants. This is true even in countries such as Australia and New Zealand with no known animal TSE's.

Agricultural officials in countries known to have BSE have taken a series of actions to control and, it is hoped, eradicate BSE. These include making BSE a notifiable disease, prohibiting the inclusion of certain animal proteins in ruminant rations (the feed bans vary depending on the amount of BSE detected), and the depopulation of certain groups of cattle thought to be of higher risk owing to epidemiologic findings.

To prevent human exposure to the BSE agent, some countries have established prohibitions on the inclusion of high-risk material in foods, pharmaceuticals, and cosmetics.

European Union (EU) Actions

Several EU member countries have had cases of BSE in native cattle or only in imports from the United Kingdom. Other countries have reported BSE in both imports and native cattle, and some member states have not reported BSE.

In June 1994, the EU enacted regulations that prohibit feeding mammalian protein to ruminants. On March 27, 1996, the European Commission prohibited the export of most bovine products, including live animals, embryos, meat, meat meal, meat-and-bone meal, and other products from the United Kingdom. These restricted products were those most liable to enter the animal or human food chain or be used in medicinal, pharmaceutical, or cosmetic products.

In July 1997, the EU adopted legislation restricting the use of "specified risk material" (SRM). SRM's include skulls, brains, eyes, tonsils, and spinal cords from all bovines, caprines, and ovines 12 months of age or older. The category also includes spleens from ovines and caprines of any age. The SRM's may not be used in any food, feed, medicinal products, pharmaceuticals, or cosmetics. This legislation is scheduled to be effective as of January 1999.

U.S. Actions

An active surveillance program in place for 8 years has not disclosed the presence of BSE in the United States. USDA, FDA, and industry groups actively work to maintain this status. The measures APHIS has taken in this regard include prohibitions or restrictions on certain animal and product imports, ongoing surveillance for signs of the disease in the United States, preparation of an emergency response plan in the event that the disease does occur, and ongoing educational efforts. APHIS actively shares information and coordinates closely with other Federal agencies as well as with the States, livestock and affiliated industries, veterinary and research communities, and consumer groups to ensure that the United States has a uniform approach to TSE's that is based on sound scientific information.

APHIS has a comprehensive surveillance program in place in the United States to ensure timely detection and swift response in the event that BSE were to be discovered here. This surveillance program incorporates both the location of imports from the United Kingdom and targeted active and passive surveillance for either BSE or any other TSE in cattle.

APHIS has conducted a traceback effort to locate each of the 496 British cattle that were imported into this country between January 1, 1981, and July 1989. In July 1989, the United States prohibited the importation of ruminants from countries affected with BSE. Only 17 of these animals are known to be alive in the United States, and these animals are being carefully monitored by APHIS personnel on an ongoing basis. In addition, in cooperation with the States and industry, APHIS has placed five head of cattle imported from Belgium in 1996 under quarantine, and APHIS makes ongoing attempts to purchase these animals for diagnostic purposes. No evidence of BSE has been found in any of these imported animals.

The United States has had an aggressive and active surveillance program for BSE in place since May 1990. BSE is a notifiable disease, and there are more than 250 Federal and State regulatory veterinarians specially trained to diagnose foreign animals diseases, including BSE. Several agencies are involved in the surveillance program, including the USDA's Food Safety and Inspection Service (FSIS) and the Health and Human Services Department's Centers for Disease Control and Prevention. APHIS leads this interagency effort. The surveillance samples include field cases of cattle exhibiting signs of neurologic disease, cattle condemned at slaughter for neurologic reasons, rabies-negative cattle submitted to public health laboratories, neurologic cases submitted to veterinary diagnostic laboratories and teaching hospitals, and random sampling of cattle that are nonambulatory at slaughter. As of February 21, 1998, more than 6,600 brains had been examined for BSE or any other form of TSE in cattle. No evidence of either condition has been detected by histopathology or immunohistochemistry.

As of December 12, 1997, APHIS has prohibited the importation of live ruminants and most ruminant products from all of Europe until a thorough assessment of the risks can be made. The new restrictions apply to Albania, Austria, Bosnia–Herzegovina, Bulgaria, Croatia, the Czech Republic, Denmark, Federal Republic of Yugoslavia, Finland, Germany, Greece, Hungary, Italy, the former Yugoslavian Republic of Macedonia, Norway, Poland, Romania, the Slovak Republic, Slovenia, Spain, and Sweden.

This action was taken because, in the past year, the Netherlands, Belgium, and Luxembourg have reported their first cases of BSE in native-born cattle. There is evidence that European countries may have had high BSE risk factors for several years and less than adequate surveillance. Additionally, Belgium reported that the cow diagnosed with BSE was processed into the animal food chain.

The U.S. Food and Drug Administration (FDA) has recently established regulations that prohibit the feeding of most mammalian proteins to ruminants. The effective date of this regulation was August 4, 1997.

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Vesicular Stomatitis Virus Outbreak—Update 1997

Vesicular stomatitis is an acute and contagious viral disease that affects horses, cattle, swine, goats, and humans. Many New World species of wildlife are also susceptible but rarely affected. Some of these animals include deer, antelope, bighorn sheep, raccoons, monkeys, sloths, rodents, and bats. This virus has caused sporadic outbreaks of disease in the United States, Mexico, and Central and South America. The characteristics of this disease are vesicles in the mouth, on the teats, and on interdigital areas of some animals. Humans can become infected when handling affected animals. In people, vesicular stomatitis is a flulike disease; symptoms include fever and muscle aches and self-limiting blisters that may appear on the hands and in the mouth.

The causative agent of vesicular stomatitis is a rhabdovirus of the genus *Vesiculovirus*. There are two antigenically distinct types of vesicular stomatitis, New Jersey and Indiana. The Indiana type is further categorized into three subtypes: Indiana 1, Indiana 2, and Indiana 3. The most virulent and most common of the two types is the New Jersey strain.

The incubation period for this disease is usually 9 days but can range from 3 to 14 days. After the incubation period, infected animals may have increased temperature and oral lesions. The muzzle may appear hyperemic, and a serous fluid may drain from the nose. The animals may also project their tongues from their mouths and make a smacking noise. The lesions may rupture to form large painful ulcerations, leading to dysphagia and reluctance to eat. Frothing at the mouth, drooling, agalactia, weight loss, mastitis, and lameness are observed in some species. Other clinical signs include depression, local soreness, and general discomfort. Affected animals often become emaciated.

Vesicular stomatitis can affect all breeds, sexes, and ages of susceptible animals. There is increasing evidence of biological and mechanical transmission via insect vectors. Several potential vectors include phlebotomine flies, mosquitoes, and *Culicoides* midges. The Indiana strain of vesicular stomatitis has been shown to be transmitted by phlebotomine sandflies. Vesicular stomatitis usually occurs during the warmer months or at the end of a rainy season. Most of the previous outbreaks have occurred in summer months and ceased abruptly with the onset of frosts, except the outbreaks of 1982–83, which continued during the winter. The seasonality of outbreaks supports the theory of transmission via an insect vector. Another major route of infection is believed to be through abraded oral mucosa or skin resulting from grass seeds or awns. Nonbiting flies that feed on ruptured vesicles may also play a role in mechanical spread of infection. The virus can remain viable in the environment for several days, leading to infection. Fomites such as contaminated milking equipment may also be involved in spreading infection.

Vesicular stomatitis does not generally cause severe morbidity or mortality. But it can still have a great economic impact on the livestock industry due to import and export restrictions placed by vesicular-stomatitis-free countries and various States within the United States that establish movement restrictions to help prevent any exposure to infected or affected animals. The restrictions vary between the individual States and countries and are based on their specific interests and needs.

Current Situation

The index case of the 1997 vesicular stomatitis virus (VSV) outbreak, an equine in Cornville, AZ, was confirmed by NVSL on June 5. This outbreak affected Arizona, Colorado, New Mexico, and Utah. The 1997 epizootic is more geographically confined than, but similar in location to, the 1985 epizootic. The 1995 outbreak included Texas and Wyoming as well as the four States currently involved, whereas the 1985 outbreak included only Arizona, Colorado, and New Mexico.

The Disease Reporting Unit was activated in Fort Collins, CO, on June 20, 1997. Owing to the progression of the outbreak, the Disease Reporting Unit was relocated to Englewood, CO, on July 14, 1997.

As of January 16, 1998, 703 premises had been investigated for vesicular stomatitis with 380 case-positive premises identified: 2 in Arizona, 273 in Colorado, 67 in New Mexico, and 38 in Utah. Table 1 is a summary of investigations outlining the number of closed investigations with negative and positive results, the number of open investigations pending and positive (premises still under quarantine), the total number of investigations and the total positive results and the number of investigations where the agent was isolated by State. This table is accurate up to January 16, 1998.

Table 1—VSV investigations in the United States as of January 16, 1998, by number of premises

State	Closed		Open		Total investigations	Total positive	Agent isolated
	Negative	Positive	Pending	Positive			
Alabama	3	0	0	0	3	0	0
Arizona	2	0	0	0	2	0	0
Arkansas	29	2	0	0	31	2	1
California	5	0	0	0	5	0	0
Colorado	99	273	0	0	372	273	44
Connecticut	2	0	0	0	2	0	0
Florida	1	0	0	0	1	0	0
Georgia	5	0	0	0	5	0	0
Idaho	7	0	0	0	7	0	0
Illinois	5	0	0	0	5	0	0
Indiana	2	0	0	0	2	0	0
Iowa	2	0	0	0	2	0	0
Kansas	8	0	0	0	8	0	0
Kentucky	3	0	0	0	3	0	0
Louisiana	7	0	0	0	7	0	0
Massachusetts	2	0	0	0	2	0	0
Michigan	2	0	0	0	2	0	0
Minnesota	1	0	0	0	1	0	0
Mississippi	1	0	0	0	1	0	0
Missouri	6	0	0	0	6	0	0
Montana	2	0	0	0	2	0	0
North Carolina	2	0	0	0	2	0	0
North Dakota	1	0	0	0	1	0	0
Nebraska	5	0	0	0	5	0	0
New Jersey	3	0	0	0	3	0	0
New Mexico	37	66	0	1	104	67	8
Nevada	1	0	0	0	1	0	0
New York	2	0	0	0	2	0	0
Ohio	2	0	0	0	2	0	0
Oklahoma	17	0	0	0	17	0	0
Oregon	4	0	0	0	4	0	0
Pennsylvania	2	0	0	0	2	0	0
South Carolina	1	0	0	0	1	0	0
South Dakota	1	0	0	0	1	0	0
Tennessee	3	0	0	0	3	0	0
Texas	15	0	0	0	15	0	0
Utah	15	38	0	0	53	38	5
Virginia	6	0	0	0	6	0	0
Washington	3	0	0	0	3	0	0
Wisconsin	3	0	0	0	3	0	0
Wyoming	6	0	0	0	6	0	0
Totals	323	379	0	1	703	380	58

Chart 1 is an epidemiologic curve of all affected States beginning the week of May 18, 1997. It illustrates the number of positive premises per week through the week of January 11, 1998. There was a dramatic reduction in positive cases beginning the week of October 19 and decline to zero by the week of November 16.

Chart 2 demonstrates the number of case-positive premises released from quarantine covering the timeframe from the beginning of the outbreak. The first premises was released the week of July 6, and the majority of the premises were released between November 9 and 30.

Chart 1

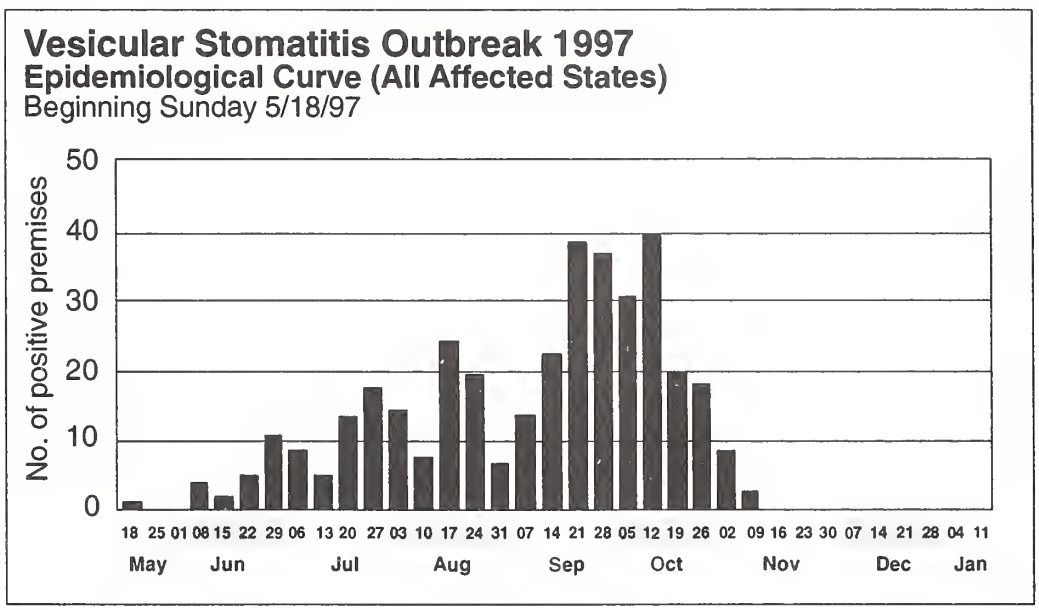
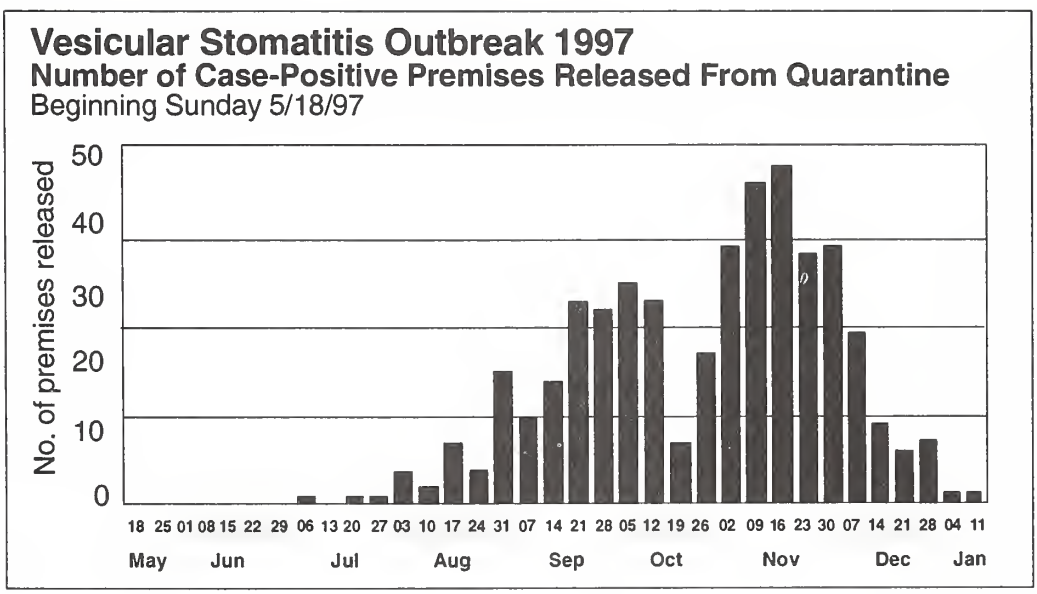


Chart 2



Quarantines and Restrictions—Officials of affected States have placed positive premises under quarantine and instituted various animal movement restrictions. Quarantines remained in place for 30 days after healing of the last clinical lesion on a premises. The 10- to 20-mile restricted circle requirements used in the 1995 vesicular stomatitis outbreak were found scientifically unwarranted and are no longer used. Immediately following a positive diagnosis of VSV, an epidemiologic investigation was conducted.

Most States have implemented temporary restrictions regarding importation of livestock from States affected by vesicular stomatitis. The basic and most common requirement is a statement on the health certificate similar to the following:

> I have examined the animals listed on this certificate and have found no clinical signs of vesicular stomatitis. To the best of my knowledge, these animals have not been exposed to vesicular stomatitis, nor have they been vaccinated with vesicular stomatitis vaccine within the previous 30 days.

Other restrictions implemented by some States include requirements of permits for entry, postentry quarantines, negative VSV tests, bans on livestock from affected premises, and prohibition of VSV-vaccinated animals.

International Restrictions—Some international trade restrictions were implemented in response to the diagnosis of vesicular stomatitis in the United States. These restrictions vary from requiring health certificates with statements that the equidae have not been in any State in which vesicular stomatitis has been diagnosed in the previous 6 months to declarations that the equidae have not been in contact with livestock that have been, during the last 30 days, in any State in which vesicular stomatitis has been diagnosed in the previous 6 months.

Reporting Communications—The USDA–APHIS–VS Emergency Programs staff provides a weekly report containing information on new cases, quarantines released, cases closed, virus isolations, and other demographic data. Emergency Programs also continues to report relevant data on VS to the Office International Des Epizooties (OIE). Various informative materials have also been widely distributed, including factsheets on vesicular stomatitis in dairy, beef, equine, and swine, vesicular stomatitis vaccine and a vesicular stomatitis *v.* foot-and-mouth disease color card.

Since the outbreak onset, a weekly conference call has been held with APHIS, State, and Federal officials and industry to discuss issues such as updates, State restrictions, quarantines, laboratory results, and other related concerns. Industry groups that have participated include the American Horse Council, Livestock Marketing Association, National Animal Health Association, National Milk Producers Federation, National Pork Producers Council, and the National Cattlemen's Beef Association.

No new cases have been reported since the week of November 9, 1997. The Disease Reporting Unit for vesicular stomatitis was closed on December 12, 1997. The last premises under quarantine was released on January 20, 1998.

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Risk Analysis of Potential Control Options for the 1997 Nonpathogenic Avian Influenza Outbreak in Pennsylvania

Background

Pennsylvania is currently dealing with an outbreak of nonpathogenic (H7N2) avian influenza (AI) in commercial egg-layer flocks. At this point, Pennsylvania has identified more than a dozen infected flocks in the Lancaster County area. In July 1997, the VS management and the Pennsylvania Department of Agriculture (PDA) requested an analysis of potential control options.

The objective of this report is to provide decisionmakers with information regarding the relative consequences and costs of various control options. This analysis may be useful in framing the problem and for suggesting areas for further exploration. In the short timeframe for this analysis, we (the APHIS personnel performing this analysis) consulted with the poultry industry, poultry researchers, and USDA's Economic Research Service to obtain best available data. The results in this report apply to the situation as of August 1997.

Methods

We used a state-transition disease model and economic welfare analysis to simulate the results of four different control options. These options are (1) voluntary controls or "do nothing,"(2) depopulation–repopulation, (3) quarantine and containment, and (4) vaccination with quarantine. Also, we modeled several regionalization and export-restriction scenarios. Brief descriptions of the models are presented below.

Disease-Spread Model—The disease-spread model is an application of state-transition analysis. State-transition analysis involves defining mutually exclusive categories ("states") into which animals, herds, flocks, and plants can be divided. We used a basic three-state infection model: susceptible, infectious, recovered, or immune. In this analysis the flock was the basic unit of concern. For purposes of the simulation, flocks in the susceptible state were defined as normal, healthy, and available to be infected with AI. Flocks in the infectious state have AI virus circulating among birds and are capable of infecting other flocks. After a flock is infected, it will be immune to reinfection or removed by depopulation, depending on the scenario being modeled. If repopulated, these flocks will return to the susceptible state.

Pathways ("transitions") between the states must also be defined. Pathways are represented mathematically as the probability of moving from one state to another. States and pathways serve as the basis for a series of simulations. These simulations project the spread of a disease or pest over time in hypothetical epidemics and can be used to compare the relative value of different scenarios.

Information generated from the disease-spread model is combined with physical impact data such as death or production losses. The resulting output gives an estimate of the biological consequences associated with the disease spread. The economic model then uses the calculated biological consequences as input in estimating the monetary impact on producers and consumers.

For this analysis, the area of risk was defined as all Pennsylvania counties affected by the final Federal quarantine in 1984. According to the 1982 census, 468 layer premises (>3,200 birds) were in this area. The model used this number, 468, as the starting number of susceptible flocks. Each hypothetical flock consisted of 70,000 egg-laying chickens. Each egg layer was assumed to produce 270 eggs per year (Pennsylvania Agriculture State Statistics 1996).

Flocks move from susceptible to infected to immune and back to susceptible status based on incidence rates, proportion of flocks becoming immune, proportion of susceptible flocks vaccinated, and depopulation to repopulation rates. These proportions were derived by estimating the average waiting time a flock or premises would spend in the infectious or immune states under various control options. Time in the infectious state is the time that a flock would be infected with the virus before it was diagnosed and an action was taken to isolate the flock. Time in immune state is the time a premises is either vacant or the birds on the premises are no longer susceptible to infection through vaccination or natural infection. The original rates of transition were set to approximate the outbreak of 1983–84.

Economic Model—The economic model is an application of economic welfare analysis. Economic welfare analysis evaluates how market prices and quantities adjust to changes in disease control measures. It also measures the effects of disease spread and how consumers and producers are affected by the adjustments in market prices and quantities. The effects on consumers and producers are measured in terms of changes in the difference between what consumers are willing to pay and what they actually pay for products (consumer surplus) plus returns to producers fixed factors of production (producer surplus).

For the purpose of this analysis, impact on domestic production was measured in terms of the number of eggs lost owing to disease or depopulation. The analysis examines economic impacts under high and nonpathogenic scenarios and the different control options. Output from the disease-spread model was used to help estimate the number of eggs lost.

Export losses were measured in terms of a single aggregate poultry meat. Therefore, the estimated consumer benefit may be somewhat overstated because of differences in consumer preferences for products consumed domestically versus those intended for export. Modeling the differences in consumer preferences is beyond the scope of this analysis.

Estimates of regionalized export losses were based on the proportion of total broiler production in each State. This proportion was applied to estimates of the U.S. impact if poultry meat exports were eliminated entirely. If a regionalization strategy is implemented, multi-State poultry companies may be able to alter their distribution channels, minimizing the adverse effects of export restrictions in a given region. The level to which these adverse effects can be minimized is unknown at this time. If an acceptable regionalization strategy cannot be established, the full impact of any export bans would be felt.

Disease Control Scenarios—Several scenarios were developed to examine the costs and benefits of alternative control options. The scenarios were evaluated using computer simulation models described above. The scenarios were modeled over a 21-week period starting with one infected flock. The following is a description of each scenario and assumptions.

1. Voluntary Control or "Do Nothing" Approach—After consulting with the PDA, we considered the do-nothing approach an infeasible option and we did not model its impacts. The main reason was that, if, in 1997, PDA had not imposed quarantines and restricted poultry and poultry product movement, spread of infection could have been substantial. Other reasons are discussed below.

- This scenario would assume that neither State nor Federal authorities will take any steps to contain or control the situation. Because of the traffic in feed, feed ingredients, service personnel, maintenance personnel, vaccination, placement, and catch crews, infection among flocks could be expected to spread rapidly within the State.

- Without local control, AI would likely be transported out of the State into surrounding areas. Large poultry-producing States nearest Pennsylvania—specifically the Delaware, Maryland, Virginia (Delmarva) region—would likely be the first areas affected. We understand that some of the layer flocks in Maryland share common feed company sources and vaccination crews with the currently affected area in Pennsylvania. Feed and feed ingredient trucks serving flocks within the current quarantine area also serve broiler flocks in the Delmarva region. The estimated value of the broiler and support industries on the Delmarva is $1.5 billion. Pennsylvania's poultry industry is valued at $563.5 million. Virginia produces $773 million worth of poultry and poultry products yearly, and many of their producers have direct ties and traffic with North Carolina.

- If Pennsylvania did not quarantine the affected area, neighboring States could impose embargoes on poultry and poultry products moving from Pennsylvania. Placement of embargoes might send an unintended message to our international trading partners, potentially resulting in trade restrictions.

- Producers lack the authority to impose and enforce quarantine zones and movement controls. When nonpathogenic AI was identified in commercial layer flocks in Lancaster County, the industry requested that a quarantine be placed around an area approximating a 5-mile radius from the first positive flock. In 1983–84 and during the current situation, the PDA was able to enlist the Pennsylvania State Police to enforce the quarantine zone. Establishment of this quarantine zone provides reassurance to surrounding States and industry.

2. Depopulation Scenario—For this scenario, flocks were moved from the infectious to the depopulated or removed state after 1 week. This assumption was based on experience in the 1983–84 outbreak, when active surveillance was devised to sample flocks on a weekly basis. In 1983, flock owners were instructed to place recently dead birds in containers near the road. State or Federal personnel collected tracheal and cloacal swabs from these birds. The swabs were tested for evidence of virus. Collection personnel established a daily route allowing them to cover a large area while sampling many flocks. The routine was repeated the following week, providing a weekly test record for each flock in the quarantine zone (referred to as "dead bird pickup"). Upon detection of disease in a flock, a quarantine was implemented immediately to prevent movement of the virus off the premises. Thus, from the time a flock entered the infectious state until quarantine was generally not more than 1 week.

In 1983–84, the offer of indemnity payment also encouraged owners to report illness as early as possible. Producers were paid only for the live birds on the premises at the time of field diagnosis and quarantine. With the highly pathogenic AI virus it would not be uncommon for an owner to experience several thousand bird deaths in a single day. The owner could expect no compensation for losses that occurred before the quarantine and diagnosis. This setup encouraged early reporting because doing so would provide maximum compensation from the authorities. If the flock owner did not contact authorities, dead-bird pickup would likely identify the infected flock within a 7-day window. We assumed that placement of the quarantine effectively removes the flock from infected status and places it into the immune status, where it no longer poses a risk for other susceptible flocks.

In 1983–84, an infected flock was quarantined at the time of field diagnosis. Soon after that, birds were destroyed. After depopulation there was an obligatory downtime during which the premises were cleaned and disinfected. Flock houses were sampled and must have tested negative on two tests, a minimum of 30 days apart, before restocking. The final laboratory report releasing the premises from quarantine took a minimum of 14 days after the last sampling. At this time the premises were eligible for restocking with new, susceptible egg-laying chickens.

Considering the preceding discussion, we assumed it would require a minimum of 45 days to complete the necessary testing to reach the point of repopulation. Therefore, theoretical repopulation of flocks occurred 7 weeks after diagnosis. Also, because of the imperfections of the laboratory tests and the necessary downtime for removing manure, repairing the caging and electrical systems within a house, and so on, we further assumed that only 10 percent of the potentially eligible houses would be repopulated after 7 weeks. Beginning in the eighth week, 10 percent of the houses found infected in Week 1 moved to the susceptible category. In the ninth week, 10 percent of the flocks diagnosed in weeks 1 and 2 were repopulated and moved into the susceptible category.

3. Quarantine and Containment of Infected Flocks—We assumed that positive commercial flocks are detected fairly quickly with the dead-bird pick up procedure discussed above. The average time the birds were infected and remained infectious was again assumed to be 1 week. In this scenario, some flocks would become naturally infected and remain immune for life. The time in immune state was set at 52 weeks, meaning flocks do not reenter the susceptible state through the duration of the simulated outbreak.

Flocks infected with highly pathogenic AI in 1983–84 lost their table-egg market because the eggs were destroyed along with the birds during the depopulation. Flocks infected in the current nonpathogenic AI outbreak may similarly experience production (morbidity and mortality) losses. Under the plan currently in place in Pennsylvania, eggs from infected flocks are diverted to an instate pasteurization plant. Infected flocks were released from quarantine when the flock no longer exhibited clinical signs and was negative on two tests (30 days apart) for AI. Flock owners are then permitted to reenter the table-egg market; therefore, infected flocks lose their table-egg market only for a short period. However, if this pasteurization market

becomes saturated, flock owners may be unable to recoup any salvage value for their eggs, thus significantly increasing their cost.

4. Vaccination with Quarantine—Research has shown that clinical signs and production losses can be mitigated by use of vaccine. However, research also shows that vaccinated birds can become infected with field strain virus. Vaccinated birds or flocks can be expected to shed less virus, to develop less severe clinical signs, and to experience fewer production losses than unvaccinated birds or flocks. Some of the literature notes a 1–2 log-factor decrease in the amount of virus shed by experimentally challenged vaccinated birds.

The vaccination scenario was simulated by vaccinating 60 flocks (assuming 70,000 birds per flock) over the initial 4-week period. This is approximately the number of flocks capable of being vaccinated once with the 4 million doses of vaccine ordered by PDA.

The key parameter for vaccine use is its efficacy in reducing flock-to-flock transmission of field virus. We are not aware of any research or data on this topic. In the absence of this information, and for example purposes, we simulated four levels of vaccine efficacy: 0 percent, 25 percent, 50 percent, and 75 percent. These levels were implemented in the model by starting with varying numbers of flocks in the immune state, assuming that 1 million birds per week will be vaccinated. For example, the 75 percent effective scenario starts with 45 (60×0.75) flocks in the immune state during the first 4 weeks of the simulation.

In Pennsylvania, there is a current proposal to place unvaccinated sentinel chickens in vaccinated flocks. These sentinel birds will be tested regularly to determine if field strain virus has entered the flock. If there is evidence of infection, the flock will be treated as an infected flock. When a vaccinated flock is found infected with field strain virus, it will be managed as any other infected flock. Eggs will be diverted to pasteurization if there is market available. Therefore, we modeled the economic impacts with and without total loss of table and pasteurization egg markets.

Vaccinated flocks or naturally infected were assumed to be immune for the remaining flock life. We assumed quarantine was effective in reducing flock spread and set the model for only 1 week's waiting time in the infectious state to match the other scenarios.

Results—The simulated number of infected flocks and economic impact of "quarantine only" compared with "quarantine with depopulation" are shown in table 1 for highly pathogenic and nonpathogenic AI. Figure 1 shows the course of the simulated epidemics compared with the actual for 1983–84. The morbidity and mortality (M&M) impacts for the nonpathogenic scenarios are based on data provided by PDA for the current outbreak. For nonpathogenic AI, the economic losses are shown with and without total loss of table and pasteurization egg markets. For the quarantine and vaccination scenarios, any morbidity and mortality occurring during the time of flock quarantine were included in the market loss calculation.

Table 1—Simulated number of AI-infected flocks and economic impact of quarantine and depopulation control options in Pennsylvania layer flocks

(*N* = 468 at-risk flocks [>3,200 birds] in the 1983–84 quarantine zone)[a]

	Quarantine only	Depopulation and quarantine
Time in infectious state	1 week [b,c]	1 week[b,c]
Time in immune state	52 weeks	7 weeks, 10% exit each week after
Number of infected flocks	262	281
Losses for highly pathogenic AI ($ million)		
Producer loss	$17.2 plus export impacts[d]	$27.2 plus export impacts[d]
Consumer loss	$55.6 plus export impacts[d]	$89.6 plus export impacts[d]
Net loss	$72.8[e] plus export impacts[d]	$117.4[e] plus export impacts[d]
Losses for nonpathogenic AI ($ million) M&M[f] / M&M plus Market Loss[g]		
Producer loss	$4.0 / $17.2	$27.2[h]
Consumer loss	$13.1 / $55.6	$89.6
Net loss	$17.1 / $72.8[e]	$117.4[e]

[a]Under the depopulation option, incidence rate and number of flocks infected were based on the 1983–84 Pennsylvania outbreak.

[b]Assumes early detection procedures.

[c]Assumes 100-percent effectiveness of flock quarantine in reducing virus spread.

[d]Expected export losses result from conversion to highly pathogenic AI. See impacts in table 3 for various regionalization scenarios.

[e]Government costs were not included because they are similar for both scenarios with the exception of indemnity, which is a transfer cost from Government to industry. Detailed data are available from the authors.

[f]M&M = morbidity and mortality, which are based on mortality and egg losses for the 1997 virus reported by the Pennsylvania Department of Agriculture.

[g]Market loss due to infection with field virus and loss of table and pasteurization egg market if pasteurization market is saturated. Under current conditions, it is estimated that approximately $3.4 million of the market losses shown could be salvaged or regained in the pasteurization market.

[h]Figures in this column do not include export-related impacts because nonpathogenic AI does not usually influence poultry exports.

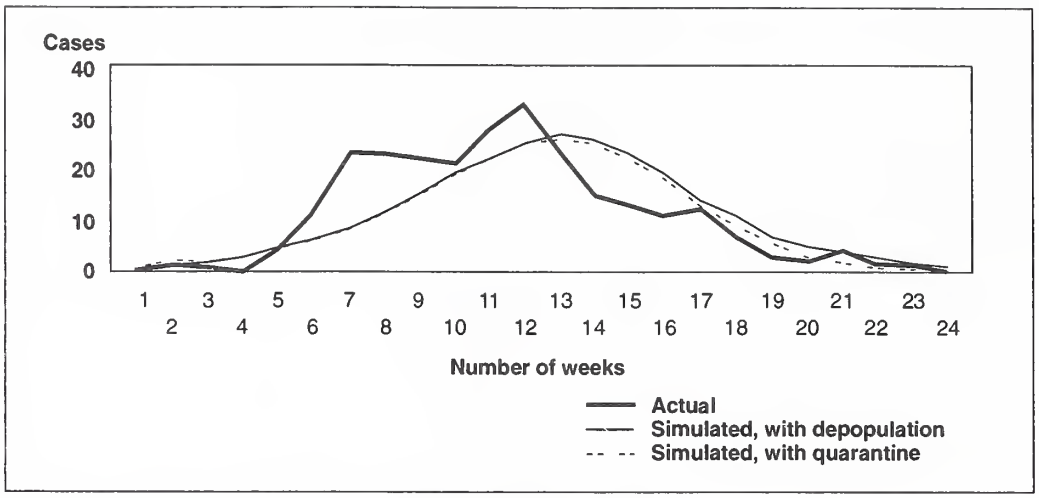

Figure 1—Avian influenza. Simulated epidemic curves for quarantine and depopulation and 1983–84 outbreak.

The simulated number of infected flocks and economic impact of vaccine for hypothetical efficacy is shown in table 2. Economic losses are reported with and without total loss of all egg markets. Table 3 shows the projected annual export losses

Table 2—Simulated number of infected flocks and economic impact (excluding export losses) of vaccine use with different assumed efficacies for reducing flock-to-flock spread[a]

Vaccine efficacy[b]	Zero	25%	50%	75%
Infected flocks	262	236	210	184
Losses ($ millions)		**M&M[c] / M&M including market loss[d]**		
Producer loss[e]	$1.4/$17.2	$1.3/$15.7	$1.3/$13.5	$1.3/$12.0
Consumer loss	$1.2/$55.6	$1.1/$50.8	$1.0/$43.6	$0.8/$38.7
Net loss	$2.6/$72.8	$2.4/$66.5	$2.3/$57.0	$2.1/$50.7

[a]Export losses could result from use of vaccine and must be added to all scenarios. See impacts in table 3 for various regionalization scenarios.

[b]Assumes 60 flocks (70,000 birds/flock) started in immune state during first 4 weeks of outbreak. Efficacy refers to reducing flock-to-flock spread, not clinical signs.

[c]M&M = morbidity and mortality, which are based on mortality and egg losses for the 1997 virus reported by the Pennsylvania Department of Agriculture.

[d]Market loss due to infection with field virus and loss of table and pasteurization egg market if pasteurization market becomes saturated. Under current condtions, it is estimated that approximately $3.4 million of the market losses shown could be salvaged or regained in the pasteurization market.

[e]Includes production losses due to handling of birds and costs of vaccine administration (4 million birds vaccinated twice at $0.12 each = $1 million) plus about 2 days of low-level production losses under the nonpathogenic scenario.

if Pennsylvania vaccinates for AI or the virus converts to highly pathogenic form. Four different potential regionalization scenarios are shown. For the purpose of this evaluation, the estimated impacts of these scenarios were based on the total impact to the United States due to a complete loss of poultry meat exports weighted by the proportion of broiler production occurring in each region. Note that these losses are on an annual basis, and export restrictions are expected to last more than 1 year.

Table 3—Projected annual[a] export losses if Pennsylvania (PA) uses AI vaccination or the virus converts to Highly Pathogenic under four potential regionalization scenarios (in millions of dollars)

	PA only	PA + Delmarva	PA + Delmarva + S.E. U.S.A.	Rest of U.S.A.
Producer loss[b]	$21.5	$166.5	$863.5	$1,300.0
Consumer gain[c]	$19.4	$154.0	$799.0	$1,200.0
Net loss	$ 1.6	$ 12.4	$ 64.4	$ 96.7

[a]Annual export losses shown above should be multiplied by the number of years trade sanctions would remain in place (probably between 2 and 3 years).

[b]These losses were based on proportion of all poultry meat production that comes from respective regions. It is likely that, should Pennsylvania be embargoed, production from other States can be shifted to fill that market, decreasing the loss.

[c]Restrictions on exports of poultry in a highly pathogenic AI situation yield consumer gains because the supply of domestic poultry in the marketplace goes up drastically, causing retail prices for chicken to fall.

Discussion

This analysis focused on the disease production impacts in commercial layer operation, as well as potential impacts in the export markets for U.S. poultry meat. We looked for benefit of vaccine use in three areas: (1) reducing producer losses, (2) decreasing the likelihood of conversion to highly pathogenic virus, and (3) decreasing the number of number of infected flocks. Compared with the high cost of potential export market loss, the benefits appear minimal.

The efficacy of vaccine at reducing flock-to-flock spread is the key variable affecting the outcome of this analysis. No data were available on this topic. However, there is agreement in the literature and expert opinion that vaccination will not prevent the circulation of field virus among flocks. Vaccination will reduce the amount of virus shed from field-strain-infected birds. The impact of this reduction on the probability of flock-to-flock spread is expected to be slight because of the large amount of virus excreted by 70,000 vaccinated birds. We had planned to model the likelihood of the virus converting from nonpathogenic to highly pathogenic. Instead, we estimated

economic impacts for high and nonpathogenic scenarios without assigning probabilities. We could find no data applicable to the probability of conversion for individual birds or flocks. If vaccination decreases the number of birds infected, it may decrease the number of opportunities for random molecular changes in the virus. The mechanism that triggers conversion is currently unknown. Also, experts generally feel that the H7N2 virus in Pennsylvania is stable and unlikely to convert to a highly pathogenic strain. This opinion suggests that vaccine use would have little impact on the emergence of highly pathogenic AI.

The time a flock spends in the infectious state is a critical variable because it affects the number of flocks that can spread disease at any one time. According to field experience and expert opinion, flocks stay infectious for an average of 4 weeks. However, the time flocks spend in the infectious state was set to 1 week for all scenarios. This assumption allows us to examine the impact of depopulation and vaccine efficacy without an added variable.

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Index Case

On March 14, 1997, a hog farmer near the port city of Hsinchu in the Prefecture of Hsinchu notified his local livestock disease control center that he had a sow in a farrowing crate that had vesicular lesions on the feet and snout. Claws had also sloughed. His was a midsized farm with about 2,000 hogs, and that sow was in the middle of the farrowing house. The farm was operated by the farmer and his wife, and no animals had been introduced onto the farm in the last month.

No people other than the farmer and his wife had entered the farrowing house in the past several weeks. The management was considered to be good but sparrows did fly in and out of the animal buildings. Morbidity in the subsequently infected herd was very high, and the mortality in young stock neared 100 percent.

Clinical Disease

The incubation period was short and appeared to be approximately 1 to 4 days. Only swine were affected. The disease was characterized by vesicles on the feet, snout, teats and tongue. Vesicles were commonly observed on the snout, and it was not uncommon to examine several hundred pigs from several pens and observe fluid-filled vesicles on their snouts. The vesicular fluid was clear to slightly cloudy. Vesicles were commonly observed on the teats.

Many of the vesicles on the feet had ruptured, leaving raw, hemorrhagic, ulcerated lesions around the coronary band, between the claws, and on the soles. Sloughed claws, were very common, as were abortions. Adults very quickly became emaciated because their feet were so sore they would not move to eat or drink.

Morbidity often reached 100 percent in adults and piglets. Neonatal mortality was commonly associated with acute myocarditis and malnutrition and related to other undiagnosed unthrifty conditions in neonates.

Laboratory

Specimens were collected from the index farm on March 14, 1997, and sent to the Taiwan Provincial Research Institute for Animal Health in Tanshui, Taiwan. Laboratory workup was completed over the weekend, and all other swine diseases were excluded. The foot-and-mouth disease (FMD) diagnostic kit was opened, and on March 19 a diagnosis of FMD was tentatively confirmed as types O1 and Asia 1.

On March 20, 1997, OIE was notified of the outbreak. Samples from Taiwan were sent to the OIE/World Reference Laboratory for FMD, Institute for Animal Health, Pirbright, UK, for further evaluation. Employing indirect enzyme-linked immunosorbent assay (ELISA) and cell-culture techniques, the Laboratory identified FMD type O.

To determine if the Taiwan strain was naturally adapted to swine, various experiments were initiated at the Laboratory. Experimental pigs inoculated with the Taiwan FMD isolate became ill with generalized FMD. Four normal pigs and four normal cattle were placed in contact with the FMD pigs for 2 hours. The contact-exposed pigs developed generalized FMD. The contact-exposed cattle remained normal. Further animal studies at the Laboratory indicated that the Taiwan FMD isolates are naturally adapted to pigs and are considered porcinophilic strains of FMD.

Epidemiology

FMD disease was previously diagnosed in Taiwan in 1913–14 and again in 1924–29. The exact mode of entry remains unknown for the 1997 outbreak. This information may become available after exhaustive epidemiologic studies. The disease may have entered Taiwan via (1) smuggling of live pigs, (2) smuggling of pig meat products, (3) legal importation of live pigs, (4) legal importing of pig meat products, (5) smuggling of animal biologics, (6) legal importing of animal biologics, (7) legal and illegal moving of people, or (8) intentionally introducing the disease agent.

Although the laboratory diagnosis was being made on the index farm in Hsinchu Prefecture, clinical cases of an FMD-like disease were being found on hog farms in Tainan and Pingtung Prefectures, about 200–300 km south of the index farm. This fact suggests that FMD was present in Taiwan long before the official date of March 20, 1997. Currently, however, there is no scientific information available to support that hypothesis.

The index farm was also located near a port city that was well known for the smuggling of pigs, especially the black-skinned pigs, which are highly valued by many local markets. The index farm was also situated near legal and illegal slaughterhouses.

On March 17, FMD was clinically diagnosed on a second hog farm in the Hsinchu Prefecture. On March 18, FMD was also diagnosed on a hog farm in the Prefecture of Taoyuan, bringing the number of affected farms to three. Unfounded comments have suggested that when FMD was diagnosed on the index farm, the disease was also present on 25–30 other hog farms.

During March 1997 and the preceding several months, Taiwan had initiated a control and eradication program for hog cholera that included the collection of at least 100,000 blood samples from 17 livestock disease control centers around the island. In a recent preliminary study to determine any prior serologic evidence of FMD, approximately 550 samples were tested in April and May 1997, and all were negative for FMD.

Japan and South Korea set February 21, 1997, as the official date of FMD infection in Taiwan for import reasons. Comments and news articles suggest that FMD was present on the island as early as October 1996 or in the late summer of 1996. There is currently no scientific evidence to support this conclusion.

Swine Vesicular Disease (SVD)

SVD is well recognized in Taiwan. It is characterized by vesicular lesions on the snout, teats, and feet, including the interdigital space. *SVD is clinically indistinguishable from other vesicular diseases, especially FMD.*

Reports from Taiwan suggest that SVD can easily be easily recognized based on clinical lesions and that laboratories were not routinely employed to diagnose SVD. It is very possible, on the basis of this reliable information, that early cases of FMD may have been misdiagnosed as SVD. Serologic evidence of SVD was detected at the World Reference Laboratory.

FMD moved very rapidly throughout Taiwan, as the data indicate. For example, on March 20, 70 farms were officially infected. Within 8 days, 1,175 farms were infected. The disease appeared to infect an additional 200–300 farms per day. At the moment, no explanation can be given to account for the lightninglike speed at which this disease moved throughout the island.

FMD infection in Taiwan was confined to swine despite observations of cattle and other small ruminants in very close proximity to infected hogs. No infection was ever reported in other species, including zoo animals.

Therefore, contributing factors to the introduction and very rapid spread of FMD in Taiwan have been associated with (1) very high swine density (Taiwan has been reported to have the world's highest density of hog farms), (2) garbage feeding, (3) hog farms located in close proximity to slaughterhouses, (4) the absence of a vaccination program (although the disease has been absent for almost 70 years), (5) frequent social farm visits, and (6) lack of complete laboratory confirmation of vesicular diseases.

Figures available as of June 17, 1997, indicated that 6,144 farms were affected, involving 1,011,421 FMD-infected pigs, 184,231 dead pigs, and 3,850,536 pigs killed.

Depopulation and Disposal—Immediate steps to be taken on an FMD-infected farm include the following:

- Stop shipping pigs immediately.
- Kill all infected pigs.
- Spread lime around infected areas and hallways.
- Spread alkali flakes in sewage around pens and sterilize pigpens with a strong acid or alkali solution.
- Strictly control infected areas.
- Consider adding organic acid to the hog drinking water.
- Vaccinate as soon as possible.

The proper and rapid depopulation and disposal of millions of hogs can present grave economic, manpower, animal care, and environmental challenges. The speed and efficiency of depopulation are directly related to the speed and efficiency of disposal. Very high water tables and other environmental concerns in Taiwan complicated these procedures.

Electrocution was the means of choice for depopulation. The disposal methods were dependent upon local and regional conditions, including EPA regulations. Once EPA officials formulated their burial, incineration, and rendering procedures, depopulation and disposal continued at a smooth pace.

Because of the immensity of the task, there was an initial lack of supplies, equipment, and manpower that caused a significant delay in the prompt and timely depopulation and disposal of infected pigs. The military made a substantial contribution toward manpower needs in the area of depopulation and disposal. The military recorded

50,732 military person-days and the use of 3,277 military vehicles. Many stress-related problems were evidenced in the military recruits working on this detail. Some military conscripts employed in depopulation activities reported problems associated with psychological distress and anxiety requiring medical leave. This is most understandable, if one considers the magnitude of the depopulation and disposal effort. When military recruits completed their tour of duty, they returned to the barracks for a 10-day quarantine period. A peak depopulation capacity of 200,000 hogs per day was achieved.

The delay in depopulation posed a serious problem because the longer an infected hog farm remained intact, the greater the opportunity for spread of the FMD virus through the air, fomites, and animal and people traffic became. This must also be evaluated in view of the very high density of hog farms in Taiwan, where one can stand on a main road or backroad in Pingtung Prefecture (a very high-density Prefecture) and identify numerous pig farms (10) located about 200–300 m apart along a several-km stretch of road.

Indemnity—Much of the information provided to APHIS requires translation which was not available at this writing. Farmers could apply to their livestock disease control center or local agriculture office for indemnity payment for FMD-infected hogs. The indemnity price was based on production costs set at New Taiwan Dollars (NTD)$2,400 for a 100-kg pig and NTD$350 for pigs less than 25 kg. For registered breeding stock, the indemnity price was set at NTD$4,800. Initially, this indemnity price was very high and significantly more than that for a healthy pig, which caused some farmers to collect and introduce FMD-infected pigs to their uninfected farms. This would enable them to collect the much higher indemnity price for the FMD-infected pigs. This disparity was quickly rectified.

Disposal plans called for the burial in large municipal landfills of approximately 80 percent of the hogs. Fifteen percent were rendered, and percent were burned. Burning consisted of open burn and incineration. A capacity of approximately 200,000 pig carcasses per day was achieved. In water resource protection areas, only incineration or open burning was used.

On April 11, 11 industrial portable kerosene incinerators arrived in Taiwan. The capacity of the incinerators was 2.5 or 40 metric tons (t) per day. A day represented 24 hours of operation. One site visited in Pingtung Prefecture contained several 2.5- and 40-t incinerators surrounded by telephone poles with floodlights for around-the-clock operation. It would require several 24-hour days of operation to complete disposal on most of the farms visited.

Vaccination—Animal disease emergency planning procedures in place in Taiwan for many years included the maintenance of an FMD vaccine bank. The emergency planning procedures also involved the completion of several FMD test exercises over the past several years.

At the outset of the epidemic, 40,000 doses of trivalent FMD vaccine (O1, A24, Asia 1) were in the repository. This vaccine was immediately dispersed to the east side of the

island and elsewhere to vaccinate all susceptible zoo animals and valuable hog-breeding stock.

Because of a preliminary FMD diagnosis of both O and Asia, an order for a bivalent vaccine was placed. The bivalent vaccine (O1 and Asia 1) of approximately 526,000 doses arrived on March 26 and 27. This vaccine was immediately disbursed free of charge for islandwide use.

In mid-March, officials purchased 3 million doses of a bivalent vaccine, O and Asia, to be used on the eastern side of the island in valuable zoo animals and hog-breeding farms. A significant portion of the vaccine was given free to small farmers. To prevent the spread of the disease by vaccination crews, trained, experienced farmers under veterinary supervision were permitted to administer the vaccine. An order for 13 million doses of a monovalent FMD vaccine O was received on May 3, 1997, and distributed free of charge to farmers.

Initially, all FMD-infected hogs were destroyed. When enough vaccine became available, all pigs (including those with the disease) were vaccinated. About 17–21 million doses of vaccine were administered. Additional material on vaccination is in the process of translation.

Cleaning and Disinfection—The cleaning and disinfection process is one of the most critical and essential aspects of recovery after an FMD epidemic. Various protocols for these procedures were supplied by the local livestock disease control Centers, other government agencies, livestock journals, and private industry. These procedures included the standard textbook sterilization disinfectants used against the FMD virus. Onfarm procedures included scrubbing and spraying with disinfectant twice a week. Large tonnages of New Formula Farm Fluid, Farm Fluid S, and Virkon S were airlifted to Taiwan as needed. The Japanese Pig Producers also donated disinfectant. Abundant material is available on this subject matter but requires translation.

Restocking—A five-step procedure was initiated to restock hog farms infected with FMD. Step I, ii stipulates that farms applying for reintroduction must have no reported cases of FMD in a month within 6 km of their surroundings. Farms with only partial slaughter are required to have two doses of FMD vaccinations and no reported cases of FMD in 2 weeks.

Other pertinent points include part III, number 3, which states that applicants must hire inhouse or contract a veterinarian to be in charge of health management and disease reporting for the farm. Part III, number 4 states that reintroduced pigs must come from FMD-free farms, must weigh more than 25 kg, and must be effectively vaccinated against FMD and hog cholera. Part III, Environmental Protection Regulations, is quite specific and pertains mostly to wastewater pollution control and waste disposal. Part V addresses violations and includes fines up to NTD$150,000 for reintroducing pigs without permission. In addition, no compensation will be given if farms are reinfected with FMD or any other serious disease. Increased surveillance of actions likely to pollute rivers and the environment was included.

The Agriculture Sector in Taiwan

Taiwan's major agricultural commodities are hogs, rice, poultry, shrimp, eels, squid, tuna, sugar, and bananas. In 1996, agricultural exports were worth approximately U.S.$5.48 billion and represented nearly 5 percent of Taiwan's total exports. Exports of live animals and poultry, frozen meat, and pork chops comprised 29 percent of total agricultural exports (U.S.$1.61 billion). Japan has been Taiwan's principal market for agricultural exports (57 percent of total agricultural exports), followed by Hong Kong (20 percent) and the United States (7 percent).

The livestock sector in Taiwan has seen sharp growth in production in the last several decades. Meat output increased tenfold from 1952 to the early 1990's. By the mid-1990's, livestock accounted for over a third of the total value of Taiwan's agricultural production.

The two leading livestock sectors in Taiwan are the hog and poultry sectors. Taiwan is self-sufficient in poultry production and exceeds domestic needs in hog production. Taiwan has small cattle and dairy industries (over 90 percent of the country's beef needs are met through imports).

Taiwan's Swine Industry Prior to the Outbreak

Before the FMD outbreak, hog production was the leading agricultural product in Taiwan, and was worth U.S.$2.6 million in 1994, which was well above the value of the rice (U.S.$1.5 million) and poultry (U.S.$1.1 million) industries. More than 14 million swine were slaughtered in both 1995 and 1996, yielding more than 1 million t of product each year.

Pork, the preferred meat in Taiwan, accounts for about 60 percent of total meat consumption. Per-capita consumption averaged 37 kg in 1991. Because Taiwanese consumers prefer fresh pork, hogs for domestic consumption are typically slaughtered at night and sold in wet markets the following day. Before to the outbreak, about 20 processing plants provided meat for the domestic market. Hogs move from the farm to slaughter via auction markets. In the auction markets, hogs are sold individually. The auctions run on a daily basis with a relatively small daily volume of sales. A small but growing percentage of hogs (22 percent in 1991) are raised under contract and go directly from the farm to the packing plant.

Exports have been important to the Taiwanese swine industry. Prior to the outbreak, nearly 40 percent of the hogs destined in Taiwan were raised for the export market, and exports were worth U.S.$1.5 billion. (The USDA'S National Agricultural Statistics Service reports that only 3 percent of total U.S. pork production is exported.) Meat products constitute the majority of Taiwanese exports and totaled nearly 270,000 t in 1995 and 1996. Most of these exports were shipped to Japan, where Taiwan captured 44 percent of the market in 1996.

Exports of fresh, chilled pork had been increasing, reaching nearly 40 percent of total exports by the mid-1990's. Before the FMD outbreak there were approximately 20 processing plants in Taiwan of export quality (i.e., they met the standards and requirements set by Japan). In addition to meat products, several thousand live swine were exported annually before the outbreak.

Taiwan was among the top 15 producers of pork and pork products worldwide in 1996. Approximately 270,000 t of pork products were exported from Taiwan in 1995 and 1996, leading to Taiwan's status as the third largest exporter of pork products worldwide. Most of these exports were sent to Japan.

Hog production in Taiwan developed during the last 3 decades from a sideline farm activity to a major enterprise. By 1995, the number of hog farms with more than 1,000 head jumped to 9 percent of all hog farms, whereas those farms with fewer than 200 head dropped to 65 percent of the total.

Hog density in Taiwan is extremely high. Eighty-three percent of the hog population is concentrated in the southwestern portion of the country. Swine density is approximately 6,500 hogs/mi^2 there. By comparison, in Sampson County, NC (one of the top swine-producing counties in the United States), swine density is approximately 1,800 hogs/mi^2.

Because of the high concentration of hogs in Taiwan, hog waste and environmental pollution have been a problem. Water quality regulations have been adopted in recent years, and efforts have been made to implement onfarm manure treatment. By 1993, 86 percent of farms had implemented manure treatment regimens. A controversial plan prepared in 1991 called for the end of pork exports to reduce the size of the hog herd to better match environmental carrying capacity. This plan was not enacted.

To support the development of the livestock sector, the Taiwanese Government operates an integrated disease-prevention system, including an inspection system for imported meat. In addition, the government oversees the production and use of veterinary medication. Routine monitoring of the livestock population is also carried out.

In the 1990 "Council of Agriculture Yearbook," Taiwan considered itself free of FMD, rinderpest, and African swine fever. Prior to the 1997 FMD outbreak, hog cholera and pseudorabies were the major diseases of concern to hog producers, and Taiwan had been working toward the eradication of hog cholera.

Economic Impacts of the Outbreak

The most significant economic impacts of the FMD outbreak will be those related to the loss of export markets, particularly the fresh pork market in Japan, possibly for as many as 4 to 5 years. Because nearly 40 percent of the hog population was raised to meet export demand, significant structural impacts will take place to adjust production to a new market carrying capacity. It is too early to tell what these adjustments will be. Indications are, however, that prices will be more unstable and that farms and processors will leave the market.

Within 1 week of the outbreak, hog prices had dropped 60 percent, falling from NTD$4,500 per 100 kg (about U.S.$167) to NTD$1,700 per 100 kg (about U.S.$63). These price decreases resulted from the immediate loss of export markets and an initial sharp drop in domestic consumption. With hog production costs estimated at NTD$4,100 per 100 kg (about U.S.$152), even farms without FMD infection were affected.

Hog prices rebounded, regaining preoutbreak levels by mid-May. To encourage pig prices to recover, the Taiwanese Government provided low-interest loans and storage subsidies. A survey taken in Taiwan in July showed higher-than-expected hog numbers, which resulted in another drop in hog prices in late August and early September. At that time, prices fell again to NTD$3,000 per 100 kg (approximately U.S.$111).

The Taiwanese Government conducted a survey in July showing a total of 21,891 swine farms nationwide, which represented a decrease of 14 percent from the 25,357 reported in November 1996. In addition, the survey indicated a total of 8,533,476 pigs on these farms, which was down 20 percent from November 1996 (10,698,366 pigs). A standing herd of 7 million pigs is seen as sufficient to meet the needs of the domestic market. Numbers in excess of 7 million are likely to put downward pressure on prices. Production estimates for 1997 suggest an annual hog slaughter of 11.7 million head, which is a decline of 20 percent from the 1996 level of 14.6 million head. Taiwan's processing capacity has fallen. After the outbreak, seven of the meat packers exporting to Japan closed or downsized, including a plant run by Cargill. Early estimates released in March suggested that as many as 50,000 persons might become unemployed as a result of the outbreak. These same early estimates projected the impact on swine-related industries at U.S.$6.9 billion.

The Taiwanese Government's goal is to have no additional FMD outbreaks from July 1997 through June 1998. Taiwan then hopes to reach FMD-free-with-immunization status by June 2000. From July 2000 to June 2001, Taiwan intends to eradicate FMD and reach FMD-free-without-immunization status.

To meet these goals, regulations controlling repopulation efforts were issued and farmers were allowed to begin restocking in July. Under these regulations, farmers will need to apply for a permit to be allowed to restock. Conditions for the permit include ensuring that the farmer has disinfected his or her premises, that the farm has a contract with a veterinarian, and that the farm is equipped with wastewater and waste-disposal devices. Owing to new regulations regarding watershed areas, permits may not be issued for farms located in fragile watershed districts. Indications are that farmers began restocking herds prior to July in response to good hog prices.

Taiwan's export market will be affected for many years. Japanese import requirements prohibit unprocessed pork being imported from an area infected with FMD unless the disease has been eradicated and vaccines have not been used for at least 2 years. These requirements may exclude Taiwan from the Japanese fresh pork market for at least 5 years.

Taiwanese officials are negotiating with Japan regarding the possibility of Japan accepting processed pork such as hams, sausages, and similar products. It was unlikely that such exports would occur in 1997. The United States, Denmark, and Canada have replaced Taiwan in the Japanese market. Exports of pork from the United States to Japan are projected to total 185,000 t in 1997 compared with 142,000 t in 1996. About 93,000 t of U.S. exports are projected to be chilled pork, which is increase from 77,000 t in 1996.

Exports from Denmark are projected at 135,000 t in 1997, which is up from 119,000 t in 1996, whereas exports from Canada are projected to increase from 39,000 t in 1996 to 55,000 t in 1997. The U.S. share of the Japanese market is expected to grow from 22 to 40 percent by 1998.

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Chronology of Hog Cholera Outbreak in the Dominican Republic

Introduction

Hog cholera (HC), or classical swine fever (CSF), was first reported on the island of Hispaniola in Haiti in the fall of 1996. The disease spread rapidly with high mortality. A vaccination program was developed using both the Minnesota and Chinese strains of the virus. High mortality was associated with the Minnesota strain because no neutralizing sera were used with the vaccine. Vaccine with the Chinese strain was donated by a private French group. Approximately 50 percent of the swine population may have been vaccinated in Haiti. The first positive serology on the other end of Hispaniola, in the Dominican Republic, was reported in March 1997. Viral antigen was detected in June 1997. These early detections were found near the border with Haiti.

Chronology

In November 1996, the Dominican Republic received official notification that several HC outbreaks had occurred in Haiti. This prompted the Dominican Ministry of Agriculture to initiate an HC epidemiologic surveillance program.

In March 1997, 8 of 29 samples submitted to the Central Veterinary Laboratory were seropositive for HC. The seropositive samples were from Hondo Valle, Elías Piña Province, and originated from two separate locations. The positive samples were sent to NVSL for (BVD) differentiation.

On May 7, 1997, one of nine samples from the Elías Piña Province was tissue positive for HC. This sample came from a dying animal, which was one of the eight positive pigs found in March.

In June 1997, 6 of 115 samples submitted were seropositive. Five of the six cases came from the northwestern part of the country. Tissues were collected from two of these cases and were positive on ABC–AP for HC antigens. Ministry records indicate that at least three of these five animals were found to be seropositive on June 12, 1996, in the community of Mariano Cestero, Municipio de Restauración, Province of Dajabón. The sixth seropositive animal came from Las Matas de Farfan, San Juan Province.

In July 1997, 19 of 69 cases were seropositive. Two of the 19 samples came from a kill plant in the National District Province. Traceback on these two cases was not conclusive. The other 17 seropositive cases were from the Province of Elías Piña (34 submitted). In mid-July 1997, the entire pig population of the Elías Piña Province was depopulated. A 20-km hog-free zone was thus formed along a portion of the Haitian border. The depopulation operation was carried out the last 2 weeks of July, but stragglers continued to be sacrificed through mid-October. A total of 6,412 pigs were sacrificed from the Elías Piña Province.

In August 1997, 13 of 761 samples were seropositive. Four seropositive samples came from San Cristobal, San Cristobal Province. Five seropositive samples came from Neiba, Bahoruco Province (86 submitted), and four seropositive samples came from various areas in the Province of Valverde (257 submitted).

In September 1997, 18 samples of 1,031 submitted to the central laboratory were seropositive. Eight of 11 tissue samples submitted were positive for viral antigens. In the National District Province, seven samples were seropositive (77 submitted), and two tissue samples were positive (4 submitted). In the Northwest Region: one seropositive case was submitted from Mato Nuevo, Mao, Province of Valverde (70 submitted), and one seropositive sample from the Dajabón area (32 submitted). The southern region of the country had nine seropositive samples. Six seropositive samples were from Neiba, Province of Bahoruco (95 submitted). One seropositive sample was submitted from Polo, Barahona Province (18 submitted). Finally, two seropositive samples were submitted from Duvergé, Independencia province (20 submitted). From August 14 to September 23, a total of 77 were sacrificed, burned, and buried in Neiba to try and stop the spread of the disease.

In October 1997 (exclusive of October 31 data), 47 samples of 1,131 samples submitted to the central laboratory were seropositive. Fourteen of 42 tissue samples submitted were positive (the total number of tissues submitted is not accurate because "single-owner lot samples" from slaughterhouses were counted and processed as one tissue sample. A more accurate estimate of total tissue samples submitted would be 80). See tables 1 and 2.

Approximately 300 pigs were depopulated from Bavaro, La Altagracia Province, by mid-October. Ninety-nine pigs were sacrificed from the Villa Mella Barrio, National District Province on October 24 and 25, 1997. Tables 1 and 2 summarize the HC-positive tissue (14) and serum samples (47) and their sources throughout the Dominican Republic in the first 30 days of October 1997.

Table 1—HC-positive tissue samples (14) from the Dominican Republic, October 1–30, 1997

Positive samples	Total samples	Source location	Premises positive	Date
3	6	Vavaro, La Altagracia Province	1	10/2
2	4	Barahona, Barahona Province	2	10/7
3	20	Villa Mella, National District Province	3	10/8, 10/14
4	4	San Ysidro, National District Province	1	10/10
1	1	Haina, San Cristobal Province	–	10/8
1	2	Cesda, San Cristobal Province	1	10/16

Table 2—HC-positive serum samples (47) from the Dominican Republic, October 1–30, 1997[1]

Positive samples	Source location	Premises positive	Date
9	Bauaro, La Altagracia Province	4	10/2, 8, 16, 24
4	Los Rios, Villa Jaragua, Bahoruco Province	2	10/2
9	Vicente Noble, Barajona Province	2	10/7, 30
5	Galvan and Barahona, Barahona Province	unk.	10/21
4	Los Matas de Farfan, San Juan Province	2	10/8
2	Villa Mella, National District Province	2	10/14, 27
1	San Isidro, National District Province	2	10/16
1	Los Tres Brasos, Santo Domingo, National District Province	1	10/21
1	La Bomba, Dajabón Province	1	10/30
1	Dajabón Province	1	10/24
1	Los Ingenitos, Santiago Rodriguez Province	1	10/24
2	Mao, Valverde Province	1	10/24, 30
1	Haina, San Cristobal Province	1	10/10
2	Boco de Nigua, San Cristobal Province	1	10/27
3	Independencia Province	1	10/30
1	Moca, Espillat Province[2]	1	10/30

[1]The total number of samples submitted from each province is not known because a power outage occurred at the lab on October 31.

[2]In the herd of 38 animals, only 1 pig tested positive for HC.

USDA Intervention

The Dominican Republic contacted USDA on August 27, 1997, for technical assistance and equipment. Since the first serologic evidence of this outbreak was found in March 1997, APHIS had several other representatives in the Dominican Republic before this action team. We have examined the methods of pig farming and marketing and the activities of the Ministry of Agriculture in its campaign to control and eradicate HC. In addition, assistance has continually been provided by various means.

Several APHIS personnel have been to the Dominican Republic, on permanent assignment or detailed as consultants, to provide technical support and expertise:

Permanent Assignment
Osvaldo Perez, International Services, Dominican Republic

Consultants
Jose Diez—VS, Area Veterinarian-in-Charge, Puerto Rico
Douglas Gregg—VS, veterinary medical officer, Foreign Animal Disease
Timothy Deveau—VS, veterinary medical officer, Wisconsin
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Action Plan Team
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Mark Schoenbaum—VS, Regional Epidemiologist, Central Region
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Edward Rossy—VS, veterinary medical officer, Puerto Rico
Eloisa Jones—International Services, veterinary medical officer, Riverdale, MD
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Epidemiology Study : The Oklahoma City Bombing—Working Dogs Who Responded and Their Associated Injuries and Illnesses

An epidemiologic study entitled "Injuries and Illnesses in Working Dogs Used During the Disaster Response After the Bombing in Oklahoma City" was conducted by USDA, APHIS, VS, EP personnel and VS employees from the Centers for Epidemiology and Animal Health, Animal Disease Information and Analysis unit, working in conjunction with the Injury Prevention Service, Oklahoma State Department of Health. The study was published in vol. 212, issue 8 (April 15, 1998) of the Journal of the American Veterinary Medical Association. See pages 1202–1207 under the Disaster Medicine section heading.

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The Acute Porcine Reproductive and Respiratory Syndrome (PRRS) Investigative Study—an Update

During 1996, swine producers began reporting outbreaks of high sow abortions and preweaning mortality. The severity of the syndrome and the current clinical picture mirror that of the original porcine reproductive and respiratory syndrome (PRRS) when it first hit the United States in 1989. For this reason, the syndrome was named acute PRRS. If this disease continues to spread, the pork industry will experience significant economic losses. Should acute PRRS reach the same level of herd infection as original PRRS, producer surplus would decrease by $583 million per year. Consumer surplus would decrease by $121 million per year. In the United States, direct losses due to conventional PRRS (cPRRS) have ranged from $50 to $250 per sow in the herd. Data from the National Animal Health Management Study's Swine '95 study showed that 68.5 percent of hog operations had cPRRS virus on the premises in 1995. Also, the hog and pork export market is severely threatened by the existence and spread of any undefined pathogen.

The pork industry and the U.S. Government have responded aggressively to this new emerging disease problem. NVSL and the National Animal Disease Center (NADC) in Ames, IA, and other veterinary diagnostic laboratories have begun diagnostic and research efforts directed toward understanding acute PRRS. In December 1996, the National Pork Producers Council and the American Association of Swine Practitioners called upon VS to help investigate the epidemiology of acute PRRS.

During the week of December 16–20, 1996, APHIS–VS sent an early response team to southeastern Iowa to conduct an investigation of acute PRRS. The team investigated operations where swine were experiencing acute PRRS and operations that had not reported acute PRRS. The team's analysis suggests that clinical signs and laboratory findings in affected herds were consistent with conventional PRRS as initially defined in the United States in 1989 and 1990. The PRRS virus was isolated from some premises. The team did not identify risk factors for increased abortions and deaths in the area.

On December 24, 1996, the American Association of Swine Practitioners surveyed its members to estimate the number of acute PRRS-affected swine herds in the United States and Canada over the previous 15 months. Affected herds were defined as those with (1) acute onset, (2) clinical signs over a 2- to 4-week period, (3) high mortality (>5 percent) in sows and boars, and (4) high rates of abortion (>10 percent) occurring in all stages of parity and gestation (Zimmerman et al. 1997). Results of the survey show that at least 138 herds may have been affected with acute PRRS.

Veterinary Services, the National Pork Producers Council, the American Association of Swine Practitioners, and other members of the swine industry are now implementing an investigative study of acute PRRS epidemiology. This is the first collaborative effort by these groups in response to an emerging animal health issue and may be the new model of cooperative studies for the future. Planning began in January 1997. Data collection began in May. The goals of the study are (1) better characterization of the role of the agent involved and (2) identification of management factors that put producers at risk for acute PRRS. This report describes the study and provides an update on data collected to date.

There are three current theories on the causes of acute PRRS, including (1) a change in management or herd immunity that alters the clinical expression of conventional PRRS virus, (2) a change in the conventional PRRS virus, or (3) emergence of a previously unidentified risk factor.

To address these hypotheses, this study will have three parts: (1) a prospective case-control study, (2) a retrospective case-control study, and (3) multiple add-on or followup studies. The prospective part will investigate current outbreaks (starting May 1997) of acute PRRS using case herds selected from submissions to designated State veterinary diagnostic laboratories. The retrospective study will collect data from case herds that have previously reported outbreaks through the American Association of Swine Practitioners' survey.

Followup projects will be implemented by various universities. The followup studies will assess the longer range production impacts, provide a better characterization of the virus, or perform other detailed investigations. Producer and practitioner participation in these studies is voluntary and completely confidential.

The case-control study design offers significant benefits for studying a disease such as acute PRRS. The approach works well for a disease whose occurrence is relatively rare. Sample size requirements are relatively low, and the design can be used to go back and retrospectively study outbreaks that have already occurred (Cole 1979).

Prospective Phase—For the prospective part, producers from any State can submit samples for consideration through their veterinarian. However, samples must be submitted to the seven participating veterinary diagnostic laboratories (VDL's). The laboratories are located in Nebraska, Iowa, South Dakota, Minnesota, North Carolina, Indiana, and Illinois. These States' laboratories were selected because the States account for most of the U.S. hog population (table 1). In addition, these States were concerned about acute PRRS and were willing to cooperate in finding cases of the disease.

The herd selection methods will be different for case and control premises but should represent the same population of potentially affected producers. Case herds will be identified when an operation with high abortion or preweaning mortality submits samples through its veterinarian to one of the seven participating VDL's. The receiving diagnostician will proceed with the laboratories' normal abortion workup. If the diagnostician suspects that a herd qualifies for the study, the diagnostician will notify the designated VDL contact. The VDL contact will conduct a screening survey over the phone by talking with the submitting veterinary practitioner. The survey will be completed on an APHIS site on the World Wide Web. Each laboratory has been issued a unique access code to prevent unauthorized data entry. No data will be accessible via the World Wide Web. Using Internet technology will allow immediate feedback to the diagnostician and veterinarian as to whether the herd in question qualifies as a case. If the herd qualifies, the project coordinators in Fort Collins, CO, are immediately notified by e-mail to begin additional data collection.

Table 1—Hog populations and number of operations in States with diagnostic laboratories participating in the acute PRRS investigative study[1]

State	Breeding inventory	Total sows	Number of operations			
			Total	With < 100 sows	With > 100 sows	Total operations
	Thousand hogs	*Percent*	*Thousand*			*Percent*
Iowa	1,250	28.6	21.0	4.6	16.4	38.8
North Carolina	1,000	22.9	6.0	4.0	2.0	4.7
Minnesota	540	12.3	11.0	5.0	6.0	14.2
Illinois	520	11.9	8.8	2.9	5.9	13.9
Indiana	460	10.5	8.5	4.2	4.3	10.2
South Dakota	155	3.5	3.5	1.2	2.3	5.4
Nebraska	45	10.3	8.0	2.6	5.4	12.8
Total	4,375	100	66.8	24.5	42.3	100
U.S. total	6,663	—	157.45	96	61.5	—
Percent	—	65.7	—	—	—	68.8

[1]Based on the Hogs and Pigs Report, Dec. 27, 1996, published by USDA's National Agricultural Statistics Service.

Case herds are currently defined as those with abortion rates greater than 3 percent if less than 7 days into the outbreak, 8 percent if 7 to 10 days, or greater than 12 percent if the outbreak lasts more than 10 days. A case herd may also have preweaning mortality greater than 25 percent within last 28 days. Also, if the diagnostician considers this herd to have acute PRRS, an investigation will be performed. To qualify for the prospective phase, case herds must still be experiencing these production problems. Control herds will need to meet the same criteria as a case herd without reporting an abortion episode with rates greater than 5 percent for any 1 month within the last 15 months or preweaning mortality greater than 25 percent. Case and control herds must also have computerized production records they are willing to share and have more than 50 sows on March 1, 1997.

Control herd selection will begin after case identification. The APHIS, VS, veterinary medical officer (VMO) assigned to the district involving a case herd will be notified. The VMO will visit the veterinarian. He or she will generate a list of all swine clients in the practice. From this list, the VMO will randomly select client names using a random numbers table. The VMO will conduct a quick screening survey and select another name if the herd chosen does not qualify. The practitioner will contact the producer asking permission to arrange a farm visit.

The farm visit will involve a management survey, conducted by the VMO, and blood and tissue collection by the practitioner. Data collection will be the same on case and control farms. Additionally, on case farms, fresh tissues (lung, spleen, and kidney) and thoracic fluid or serum will be collected from recently aborted piglets. Serum samples will be collected from 30 randomly selected sows or gilts and 15 neonatal (< 24 hours old) piglets (maximum of 3 piglets/litter). Sows will be rebled 3 to 4 weeks later to evaluate changes in serum antibody levels. All samples will be sent to NVSL. Serum samples will be tested for the presence of PRRS virus and stored for later use, as needed. Neonatal piglet serum and fetal tissue samples will undergo virus isolation procedures to isolate PRRS or any other potential viral agent.

Data on farrowing and weaned pig production will be collected for the baseline time period of January through February 1997 and the time period of the outbreak. This production data was used to reclassify herds as cases or controls for analysis. Data entry and analysis will be conducted at the Centers for Epidemiology and Animal Health (CEAH) in Fort Collins, CO.

Retrospective Phase—For the retrospective part of the study, members of the American Association of Swine Practitioners responding to the Association's survey will be telephoned. They will be asked to provide names of producers they believe have experienced acute PRRS in 1996 or 1997. Practitioners reporting they had not seen acute PRRS will be asked to provide names of two producers to serve as controls. All producers will be given a short telephone interview. The questions asked will be similar to those asked in the on farm management survey of the prospective phase. Analysis will be conducted at CEAH in the same manner as the prospective phase of the study.

To aid in the prevention and control of acute PRRS, results will be disseminated to producers and practitioners through as many venues as possible.

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***Salmonella typhimurium* DT104**

A multiple-antibiotic-resistant strain of *Salmonella* serotype *typhimurium* known as Definitive Type (phage type pattern) DT104 is being recognized as an emerging pathogen in the veterinary and human health world on both sides of the Atlantic.

In Humans—*S. typhimurium* DT104 was first reported in the United Kingdom in 1984 and has been reported in the United States, Canada, and many other European countries. This organism is now the second most prevalent strain of *Salmonella* isolated from humans in England and Wales (Threlfall et al. 1992, DT104 Workshop Minutes 1997). The number of reported isolates of DT104 in England and Wales has increased from 259 in 1990 to 3,837 in 1995 (Thelfall et al. 1992). Of particular importance in this increase has been the epidemic spread of DT104 with a multiple antimicrobial resistance pattern (R-type) to ampicillin, chloramphenicol, streptomycin, sulfonamides, and tetracycline (ACSSuT). In England and Wales, the proportion of DT104 that was R-type ACSSuT increased from 27 percent in 1990 to 79 percent in 1993 and decreased to 54 percent in 1995 (Threlfall et al. 1992). Molecular studies have demonstrated that in this strain, resistance genes are chromosomally encoded. This is of concern because removal of the selective pressure, as in plasmid-mediated resistance, is expected to have no effect on reversion to susceptibility. Of additional concern is the increasing additional resistance of DT104 to trimethoprim and ciprofloxacin (a fluoroquinolone).

In the United States, the proportion of *S. typhimurium* isolates from the Centers for Disease Control and Prevention (CDC)-selected county studies that were R-type ACSSuT increased from 2 percent in 1980 to 4 percent in 1985, to 9 percent in 1990, and to 12 percent in 1995 (DT104 Workshop Minutes 1997). Studies are under way to determine how many of these isolates are DT104. National CDC studies in 1995 (all 50 States) and 1996 (14 State and local health departments) found that 28 and 33 percent, respectively, of *S. typhimurium* isolates were R-type ACSSuT (DT104 Workshop Minutes 1997). In both studies, approximately 85 percent of isolates phagetyped were DT104. To date, no additional resistance to trimethoprim or fluoroquinolones has been reported among DT104 isolates in the United States.

The clinical features associated with infection with DT104 R-type ACSSuT suggest that these infections may be more severe than other nontyphoid *Salmonella* infections. These outcomes include increased hospitalization and mortality rates. Epidemiologic studies in the United Kingdom suggest DT104 infections are associated with foodborne exposure, including processed and unprocessed foods of animal origin, or domestic and farm animal contact (particularly animals with diarrhea such as cattle and cats) (DT104 Workshop Minutes 1997).

In Animals—*S. typhimurium* DT104 has been isolated from a wide range of wild, companion, and food animals (DT104 Workshop Minutes 1997).

In the United Kingdom, from 1993 to 1995, the prevalence of reported DT104 R-type ACSSuT isolations increased in cattle, poultry, and sheep to become the most common *Salmonella* serotype and phage type (DT104 Workshop Minutes 1997). The proportions of *Salmonella* DT104 isolates from cattle received at the national veterinary diagnostic laboratory were 44 percent in adult cattle and 45 percent in calves during 1993; this increased to 56 percent and 68 percent, respectively, in 1995 (DT104 Workshop Minutes 1997). Of the 110 *Salmonella* isolates from cats in the United Kingdom received by the Laboratory of Enteric Pathogens during 1991–95, 78 (71 percent) were serotype *typhimurium,* and 40 (51 percent) of those were DT104 R-type ACSSuT (Wall et al. 1996).

In the United States, retrospective examination by the diagnostic laboratory at Washington State University of *S. typhimurium* isolates obtained from cattle in the Pacific Northwest showed none submitted in 1983–86 were R-type ACSSuT compared with 12 percent of isolates obtained between 1987 and 1990, and over 60 percent in 1991–96. Selected isolates have been phagetyped as DT104 (DT104 Workshop Minutes 1997). Although differences in sample source, species, and animal clinical status limit interpretation and comparison of prevalence data, the proportion of *S. typhimurium* isolates from national USDA 1995 and 1996 studies that were R-type ACSSuT increased from 10 percent in 1995 to 15 percent in 1996 (DT104 Workshop Minutes 1997). Studies are under way to determine how many of these isolates are DT104. Of 549 *S. typhimurium* isolates submitted to USDA from October 1996 to mid-February 1997 from various species nationwide, 143 (26 percent) were R-type ACSSuT and 90 (16 percent) of these were DT104 (DT104 Workshop Minutes 1997).

The clinical features associated with infection with DT104 R-type ACSSuT suggest that these infections may be more severe (higher morbidity and mortality rates) than other nontyphoid *Salmonella* infections. Animals may also serve as asymptomatic carriers of DT104. Long-term carriage has been observed in multiple species, particularly in cats and cattle.

A single case-control study of risk factors for DT104, R-type ACSSuT infection in cattle in Great Britain identified the following factors with an increased risk of infection: introduction of new additions to a herd; purchase of cattle from dealers, close confinement, inadequate or nonexistent isolation facilities for sick cattle, a high population density of feral cats, and access of birds to feed storage facilities (Evans

and Davis 1996). Possible modes of cat acquisition of this infection are through eating contaminated human food or by preying on rodents infected with DT104. Cats may then serve as a source of the organism for humans through contact or for cattle though feed contamination.

Prevention and Control of DT104 Infections—To address this emerging public and veterinary health problem, representatives from the United States (CDC, USDA, Food and Drug Administration, and various academic institutions), Canada, the United Kingdom, and the Netherlands attended a workshop held in Atlanta, GA, in May 1997. The workshop's goals were to review the available data on DT104, and to identify research (laboratory and epidemiology) needs, available resources, and potential areas for collaboration. The workshop recommended the appointment of an interagency project team of public and animal health agency representatives to coordinate activities involving DT104. USDA's Food Safety and Inspection Service is leading a working group in assessing the situation with DT104 in the United States and developing objectives to be addressed by the interagency team.

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FAD Training and Education Emerging Disease Course Summary

A training course on emerging diseases was held from April 28 to May 1, 1996, in Atlanta, GA. The course, jointly sponsored by Emory University, the Armed Forces Institute of Pathology and CDC, was entitled, "Emerging Infections: Clinical and Pathologic Update" and presented numerous topics of current interest from both clinical and pathological perspectives. Topics covered were diverse and included plague, ehrlichiosis, borreliosis, microsporidiosis, Hantavirus, viral hepatitis, *Ebola* virus, streptococcal disease, bartonellosis, cryptosporidiosis, amebiasis, and *Helicobacter pylori*. A comparative medicine theme was a resounding refrain, with physicians and veterinarians as participating speakers. Presentations on the following topics included information on approaches to the pathologic diagnosis of infectious diseases with emphasis on morphology and molecular biology:

- Human ehrlichiosis in the United States
- Pathology of *Ehrlichia* and other rickettsial infections
- Lyme disease
- Pathology of human *Borrelia* infection
- Hantavirus pulmonary syndrome
- *Ebola* virus hemorrhagic fever
- *Ebola* virus infection in nonhuman primate models
- Zoonoses and emerging pathogens: the role of veterinary medicine
- Emerging fungal infections: cryptococcosis, fusariotoxicosis, and *Penicilliosis marneffei*
- Laboratory diagnosis of cryptococcosis, fusariotoxicosis, and *P. marneffei*
- Pathology of infection with cryptococcosis, fusariotoxicosis, and *P. marneffei*
- Epidemiologic and clinical features of cryptosporidiosis: What have we learned since Milwaukee?
- Pathology and diagnosis of cryptosporidium infections

(For more information please contact Dr. Corrie C. Brown, Department of Pathology, College of Veterinary Medicine, University of Georgia, Athens, GA, (706) 542–5842 voice, (706) 542–5828 fax)

Veterinary Services and Emergency Programs

The primary mission of VS is to protect U.S. agriculture and the livestock industry by preventing the introduction of foreign animal diseases. To that end, VS is committed to ensuring the highest level of competence of foreign animal disease diagnosticians, veterinary medical officers, and accredited veterinarians in their ability to diagnose foreign animal diseases. Formal and informal training courses are being scheduled as time and funding permit. The following courses and seminars were conducted in 1996, 1997, and the first half of 1998.

- Animal Identification Coordinators Workshop—July 16–18, 1996, Richmond, VA. Four lectures on FAD's were presented.

- Wildlife seminar on FAD preparedness, August 13–15, 1996, College of Veterinary Medicine and Southeastern Cooperative Wildlife Disease Unit, University of Georgia, Athens. The course was designed primarily for trained FAD diagnosticians.

- FAD's satellite TV seminar, September 5, 1996, Washington, DC, with numerous downlink sites throughout the United States. This course was also primarily designed for trained FAD diagnosticians.

- Military FAD course at the Foreign Animal Disease Diagnostic Laboratory (FADDL), September 16–26, 1996, Plum Island, NY. This course was contracted by the U.S. Army.

- FAD awareness training (a continuing education course) for the Virginia Academy of Food Animal Practitioners, November 9, 1996, Harrisonburg, VA.

- The second annual FAD seminar via satellite was held on April 17, 1997. In cooperation with Organizational and Professional Development, Emergency Programs, APHIS, USDA, and Home Team Sports of Bethesda, MD, the seminar was an excellent continuing education program. The program was beamed to about 1,000 people at 68 downlink sites (3 outside the United States) and included USDA regional participation, 12 colleges of veterinary medicine, 5 veterinary science departments, and industry colleagues. The program included 6 hours of instruction. The objectives of the seminar were (1) to incorporate revised procedures for investigating a suspected FAD into day-to-day work; (2) to describe the current world situation on FMD, HC and BSE; (3) to discuss on-the-farm investigations in the United Kingdom and the impact of BSE on the British and American farmer; and (4) to visualize the impact of emerging diseases on the state of American agriculture. Several program topics were presented by a wide range of experts. The afternoon session was followed by a panel discussion.

- A 2-week FAD training course for Federal veterinarians was held from April 28 to May 9, 1997. The first week of the course was conducted at the NVSL, Ames, IA, and the second week was held at Plum Island, NY. Twenty-four Federal veterinarians, six from each region, were participants in the course. Two trained, experienced FAD diagnosticians, Drs. T. Schiefer and T. Vardy, assisted with the course. Speakers were drawn from various sources to include universities and Federal veterinarians. The course objectives were to be able (1) to conduct and report a suspected FAD investigation properly; (2) to recognize clinical signs and lesions, identify the species affected, and draw upon knowledge of the causative agent of the foreign animal diseases discussed; (3) to provide a differential diagnosis to include relevant domestic diseases; (4) to collect a probang specimen and take a tonsillary biopsy from a pig properly; (5) to conduct a systematic postmortem of the avian, porcine, and bovine species; (6) to collect correct diagnostic specimens, properly preserve and package the specimens, and prepare them for shipment to the laboratory; (7) to prepare essential paperwork properly; and (8) to recognize the importance of vectors in FAD's. The course was very well received, and we would like to thank all the speakers and support staff at NVSL and Plum Island for their support, especially the animal caretakers.

  Special thanks are extended to Drs. Schiefer and Vardy.

- Wildlife seminar on FAD preparedness, June 1997, College of Veterinary Medicine and Southeastern Cooperative Wildlife Disease Unit, University of Georgia, Athens. The course was designed primarily for trained FAD diagnosticians.

- Military FAD course at the FADDL, September 1997 Plum Island, NY. This was a course contracted by the U.S. Army.

- FAD's Satellite TV Seminar, February 11, 1998, from 10 a.m. to 5 p.m. with numerous downlink sites throughout the United States. This course was also primarily designed for trained FAD diagnosticians.

- Spanish language FAD training course at FADDL on Plum Island, NY, February 9 to 13, 1998.

- State VMO FAD training course at FADDL, March 23 to April 3, 1998.

- Federal VMO FAD training course at FADDL, June 8 to 19, 1998.

Planned Training for 1998

- Military Veterinary Corps, VMO FAD training course at FADDL, August 17 to 28, 1998.

We would like to hear your comments and suggestions for future topics and speakers. We are anxious to include a cross section of VS and APHIS personnel as speakers as well as field VMO's.

(For more information on FAD training, please contact Dr. Terrance Wilson, USDA, APHIS, VS, Emergency Programs, Riverdale, MD, (301) 734–4917 voice, (301) 734–7817 fax, e-mail : twilson@aphis.usda.gov)

Emergency Programs Staff and Current Major Projects

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- **Ms. Ida Ceesay,** veterinary program assistant *(program support for training, FAD kit correlation and distribution, office automation)*
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- **Dr. Karen James,** senior staff veterinarian *(Assistant Chief, avian influenza—Hong Kong H5N1 coordinator, contagious equine metritis coordinator)*
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