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Descriptions of Recent Incursions of Exotic Animal Diseases

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Descriptions of Recent Incursions of Exotic Animal Diseases

**Abstract**
This overview describes recent incursion of exotic animal diseases. Important lessons can be learned from these examples. U.S. agriculture is very vulnerable to the introduction of a foreign animal disease. Outbreaks can occur when a pathogen is inadvertently introduced in contaminated material carried by an international traveler, or in imported animals or animal products. Foreign animal diseases could enter the U.S. vectored by wild animals, insects, or migratory birds or they could be intentionally introduced to cause severe economic problems or to target human health. Descriptions of recent outbreaks of foreign animal disease in various countries and the impact they had are presented here to raise understanding of the importance of these diseases and their detection, prevention, and control.

**Disciplines**
Veterinary Microbiology and Immunobiology | Veterinary Preventive Medicine, Epidemiology, and Public Health

**Comments**
U.S. agriculture is very vulnerable to the introduction of a foreign animal disease. Important lessons can be learned from these examples.
sive surveillance program and has a high awareness of avian influenza, is considered to be a sentinel for new viral reassortants. From 1997 to 2002, Hong Kong experienced repeated outbreaks with various H5N1 influenza viruses. These outbreaks raised the suspicion that new virulent reassortants of H5N1 were becoming established in the region. After an epidemic in 1997, during which the first serious zoonotic infections were reported, Hong Kong was diligent in maintaining surveillance for new viruses and rapidly stamping out each outbreak. The poultry population of Hong Kong was partially or completely depopulated three times in five years. In contrast, an avian influenza H5N1 virus that appeared in several Asian countries in 2003 was not immediately stamped out everywhere. As a result, this virus seems to have become endemic in Asian birds, has spread to birds in Europe, and has triggered fears of a human pandemic.

Avian influenza, a disease seen mainly in poultry, is caused by viruses in the genus influenzavirus A, family Orthomyxoviridae. The avian influenza viruses are usually spread by the fecal-oral route; they can be transmitted directly, or indirectly on fomites and mechanical vectors such as flies. They may also be transmitted in respiratory secretions when birds are in close contact. In addition, these viruses are found inside eggs; although these eggs are unlikely to hatch, they may spread the virus if they break. There are two forms of avian influenza in domestic poultry. The more common form, called low pathogenic avian influenza (LPAI), is usually a subclinical or mild infection. Typically, an infected flock has subtle signs such as decreased egg production or a somewhat increased mortality rate; serious symptoms occur only if there are concurrent diseases or stressors. In contrast, highly pathogenic avian influenza (HPAI) or ‘fowl plague’ is a severe disease with morbidity and mortality rates as high as 90-100%. HPAI can affect domestic poultry, game birds, and ratites; however, a HPAI virus does not necessarily affect all species equally. For instance, a virus might cause severe disease in chickens and turkeys, but minimal symptoms in ducks and quail. Typical HPAI symptoms include depression, inappetence, and respiratory signs such as coughing, nasal and ocular discharge, a swollen face, and cyanosis of the comb and wattles. The birds may also have diarrhea or neurologic signs such as paralysis. In some cases, sudden death can occur with few clinical signs. Any surviving birds are usually in poor condition.

The avian influenza viruses, which are highly variable, can be classified into subtypes based on two proteins, the hemagglutinin (‘H’) and neuraminidase (‘N’). There are at least 16 different hemagglutinin antigens (H1 to H16) and 9 neuraminidase antigens (N1 to N9). Influenza viruses can change very quickly. Due to their poor proofreading during gene replication, they have a very high mutation rate. They also have a segmented genome, which facilitates reassortment. Reassortment can take place whenever two different influenza viruses infect the same cell;
when the new viruses (the 'progeny') are assembled, they may contain some genes from one parent virus and some genes from the other. Because the influenza viruses are so variable, viruses that share a subtype are not necessarily closely related and may differ greatly in their virulence, host specificity, or other factors. An avian influenza virus is classified as a LPAI or HPAI virus based on its genetic features and its virulence in poultry. HPAI viruses, which have been eradicated from poultry flocks in most developed nations, are usually H5 or H7 viruses. However, not all H5 and H7 viruses are highly virulent; many H5 and H7 LPAI viruses also exist. These LPAI viruses are also of concern, because some of them mutate and become highly pathogenic after circulating in poultry flocks for a time.

Where Do Highly Pathogenic Avian Influenza Viruses Come From?

Although avian influenza is a disease seen mainly in domestic poultry, avian influenza viruses are carried asymptotically by a wide variety of birds. Waterfowl, which carry all of the subtypes, are considered to be the reservoir hosts. These birds shed viruses in the feces, sometimes resulting in the emergence of a virus into domestic poultry (or, rarely, mammals). Thus, outbreaks of HPAI can occur even in countries that have eradicated these viruses from their commercial poultry flocks. Many new isolates come from southern China, an area some scientists call an “epicenter” for the avian influenza viruses. Farmers there often raise different types of poultry, including domestic waterfowl, alongside each other on high-density small farms - creating a perfect breeding ground for new influenza strains. These poultry are often reared under low biosecurity conditions, and may be exposed to wild birds or water where these birds have been swimming. In addition, poultry may be raised in close contact with both pigs and people, increasing the likelihood of virus recombination and virus transmission between birds and mammals as well.

The island territory of Hong Kong, which imports large quantities of poultry from China, is considered to be a sentinel for these new viruses. China supplies over 70% of the 100,000 fresh chickens eaten in Hong Kong every day and is the territory’s leading source for geese, ducks, quail, and pheasants. Unlike Mainland China, Hong Kong also has a comprehensive influenza surveillance system that allows it to quickly detect new viruses, and an established response system that allows it to respond effectively and rapidly to disease outbreaks. From 1997 to 2002, Hong Kong experienced several disquieting outbreaks with new strains of H5N1 viruses.

Avian Influenza in Hong Kong, 1997 – A Deadly Strain in Humans

The first disturbing outbreak occurred in Hong Kong poultry flocks in 1997. Before this epidemic, which began in late March, the avian influenza viruses were not thought to cause serious disease in humans. Then, in May, a H5N1 avian influenza virus was isolated from a fatal case of acute
pneumonia and respiratory distress syndrome in an otherwise healthy 3-year old boy. This virus, which seemed to be transmitted by contact with sick birds, eventually killed five more Hong Kong residents and caused serious illness in 12 others. The discovery that the H5N1/97 virus was pathogenic for humans added urgency to the eradication efforts. Approximately 1.5 million chickens in live bird markets and farms were eventually slaughtered in a successful bid to stop the epidemic. Some people think this rapid response may have averted an incipient human influenza pandemic. Since 1997, sporadic human infections have been reported with other avian influenza viruses, including other isolates of H5N1 as well as H7N2, H7N3, H7N7, and H9N2 viruses. To date, human infections with the highly pathogenic viruses, particularly H5N1, seem to be more severe. Most zoonotic infections with the non-H5N1 viruses have been limited to conjunctivitis or relatively mild respiratory infections, but severe, fatal, infections have also been seen. Human cases generally seem to result from direct contact with infected poultry or fomites, although rare instances of limited person-to-person transmission have also been documented.

The H5N1/97 virus may hold clues to help researchers determine which avian influenza viruses will infect humans. When this virus was analyzed, it was found to be a reassortant that contained genes from several different species of birds. One of the ‘parent’ viruses was a H5N1 virus similar to one first isolated from geese in China’s Guangdong province in 1996. Such viruses are known as the Goose/Guangdong/1/96 (H5N1 Gs/Gd)-like viruses. Another segment was contributed by influenza viruses found mainly in quail. The reassortment between these viruses was probably facilitated by the mixing of bird species in Hong Kong’s many retail live bird markets. These markets, where poultry is bought live, and killed and plucked in front of the customer, are very popular among Hong Kong consumers. In 1997, the live bird markets contained waterfowl such as ducks and geese, and terrestrial birds such as chickens, quail, and guinea fowl. In addition to allowing new reassortant viruses to arise, these markets facilitate their spread; birds not sold at the market and returned to the farm may carry new infections with them.

To prevent the reemergence of another H5N1/97-like virus, Hong Kong established a central slaughterhouse for ducks and geese in 1998. This measure was intended to keep the influenza viruses found in these aquatic poultry separate from other parent viruses found mainly in quail. Government representatives also called for a central slaughterhouse for chickens in Hong Kong, but the industry feared that this would undermine the livelihood of its 20,000 chicken sellers. Terrestrial poultry—chickens, quail, pigeons, pheasants, and guinea fowl—continued to be sold and slaughtered in the retail live bird markets. After 1997, Hong Kong also established an elaborate system of blood tests, inspections,
and quarantine rules to screen imported birds, as well as a surveillance system to test birds at the central slaughterhouse and in the live bird markets. However, Hong Kong can only control avian influenza viruses in its own domestic poultry. Hong Kong officials have no authority to monitor or regulate health and environmental conditions on Mainland China farms, nor are they able to control the avian influenza viruses in the millions of migratory wild birds in the area. Although the H5N1/97 virus was successfully eradicated from poultry, individual viruses containing its gene segments continued to circulate in birds in the region.

**Avian Influenza in Hong Kong, 2001**

For a time, Hong Kong's separation of terrestrial and aquatic poultry seemed to work. From 1999 to 2001, H5N1 viruses were intermittently isolated from geese and ducks at the central slaughterhouse, but no H5N1 viruses were found during routine surveillance in the live terrestrial poultry markets. Then, in April 2001, H5N1 viruses were found in three of eight retail markets; these viruses were isolated in fecal swabs from several apparently healthy chickens, silky chickens, pigeons, quail, and pheasants. This finding led to more intense scrutiny of the markets, and additional H5N1 isolates were found in cloacal swabs from dead chickens in 30 live bird markets. There were no symptoms of HPAI until mid-May, when three markets reported that the mortality rates in their poultry had increased greatly. On May 17, 2001, the Government of Hong Kong Special Administrative Region of China (SAR) reported an outbreak of highly pathogenic avian influenza type A (H5N1) virus to the OIE. The three affected live-bird markets were closed and all birds were destroyed. The following day all wholesale and retail markets selling chickens in Hong Kong were closed and the birds were culled. Beginning May 21 Hong Kong authorities depopulated approximately 1.2 million live birds as a precautionary measure. The cull covered 208 farms raising chickens, pigeons, and quail. Importation of live birds from Mainland China was stopped and retail markets for live poultry remained closed for four weeks. This outbreak is estimated to have cost $3.86 million including compensation to poultry vendors.

Although the cost of eradication was high, the surveillance system and quick response allowed Hong Kong to eliminate the H5N1/2001 viruses before illness became widespread in poultry. These viruses were found to be reassortants that contained gene segments from various influenza viruses of waterfowl. One of the parental viruses was, once again, a H5N1 Gs/Gd-like virus. This time, waterfowl viruses had been able to infect terrestrial poultry by reassorting with other waterfowl viruses. At least five different H5N1 genotypes were isolated during the 2001 outbreak. Although all five were highly pathogenic for chickens after experimental infection, only one was associated with the increased mortality rates
in the three markets. This genotype had a mutation in the neuraminidase gene that increased its ability to spread in terrestrial poultry. The genetic analysis also suggested that the quick response to the outbreak might have prevented human disease. None of the H5N1/2001 viruses had the same genotype as the H5N1/97 virus and no human cases were reported in 2001. However, some of the viruses that had contributed gene segments to the H5N1/97 virus were still circulating in quail in the live bird markets. Given time, these viruses might have reassorted with the H5N1/2001 virus and produced another genotype that could infect humans. After the 2001 outbreak, Hong Kong authorities prohibited selling live quail where other poultry were sold in live bird markets.

The exclusion of waterfowl from Hong Kong's retail live bird markets, together with the screening of imported poultry, were successful in keeping H5N1/97-like viruses out of terrestrial poultry from 1998 until 2001. But the reassortant viruses found in 2001 seemed to be more difficult to exclude, perhaps due to their wider host range and/or more efficient transmission among birds. To interrupt the amplification of any viruses that might enter the retail markets, a once-a-month “rest-day” was introduced. On these rest days, the live bird markets are completely emptied of poultry, any remaining poultry are slaughtered to be sold as chilled carcasses, and the markets are thoroughly cleaned before being restocked the following day.

Avian Influenza in Hong Kong, 2002—An Unusual Outbreak in Wild Waterfowl

Late in 2002, Hong Kong once again had an outbreak with H5N1 viruses—this time in wild birds. The first episode occurred in Penfold Park, a small nature park that contained a number of resident waterfowl including geese, ducks, and swans, as well as captive psittacine and passerine birds, free-ranging white pigeons, and feral egrets. Neurologic disease and unusual deaths were first reported in early December. Thirty-one waterfowl died, and the remaining ducks and geese were culled on December 10. The second outbreak occurred at Kowloon Park, located 12 km away. This park had an aviary with 35 species of captive free-flying birds, and a bird lake that housed 26 species of captive pinioned waterfowl and flamingos. In addition, wild herons were seen at the waterfowl ponds and five species of feral birds visited regularly to scavenge grain from the feeding troughs. The first unusual deaths occurred at this park from December 14 to 17, and the first confirmed case of H5N1 avian influenza was reported on December 17.
Kowloon Park, like Penfold Park, was closed, drained, and disinfected. All of the remaining resident waterfowl were quarantined. Many of the birds from the open ponds, including geese, ducks, swans, and flamingos died during December; however, the terrestrial and feral birds at these parks seemed to be unaffected. At the same time, H5N1 viruses were found in dead chickens in live bird markets and on a local chicken farm. H5N1 viruses were also isolated from dead little egrets, gray herons and other wild migratory birds that overwinter in Hong Kong. More than one virus seemed to be responsible for these outbreaks; at least three different H5N1 viruses isolated in late 2002 were able to cause severe disease and death in experimentally infected ducks.

Epidemics of avian influenza are very unusual in wild birds. Before this outbreak, researchers had seen little or no evolutionary change in the avian influenza viruses isolated from wild waterfowl over the last 60 years. Therefore, they believed that these viruses were stable in their normal reservoir hosts. Earlier viruses found in Hong Kong, including the H5N1/97 virus, did not replicate well in ducks and were asymptomatic in this species. The repeated outbreaks of H5N1 viruses in Hong Kong in 1997, 2001, and 2002 suggested that H5N1 viruses had become widespread in the region, and that new pandemic or panzootic strains could emerge through reassortment. There were also other concerns about these viruses. H5N1 viruses isolated in the region in 2001 and 2002 were much more variable than the H5 viruses isolated in Hong Kong between 1979 and 1997. In addition, it was worrisome that some of the new H5N1 isolates could infect the brain as well as the respiratory tract in both birds and mammals. During this time, there were also hints that viruses pathogenic to humans might be circulating in Mainland China. In 2003, a H5N1 virus infected two members of a Hong Kong family who had traveled to China. The 5-year old son recovered, but his 33-year old father died. Another family member died of a respiratory illness while in China, but no testing was done there.

**Avian Influenza in Southeast Asia and Europe, 2003-2005**

In 2003, a new epidemic broke out. This time, it was widespread. From late 2003 to March 2004, HPAI (H5N1) viruses were reported among poultry, particularly chickens, in Cambodia, China, Indonesia, Japan, Laos, South Korea, Thailand, and Vietnam. More than 100 million birds died or were culled in an effort to stop the outbreak. In rare instances, this virus was able to infect humans; 35 cases were confirmed in Thailand and Vietnam, most apparently the result of direct contact with birds. Twenty-four of the human infections were fatal. In many parts of Southeast Asia, humans live in close contact with their animals, including poultry. This facilitates the spread of influenza viruses between species, and may have contributed to the human infections. At first, culling and other measures appeared to control this virus. By March 2004, the outbreak seemed
to be contained in poultry and human infections were no longer being reported in most nations. However, beginning in June 2004, a number of countries once again began seeing the disease. This time, infected poultry were reported in Cambodia, China, Indonesia, Malaysia, Thailand, Vietnam, and the Democratic People’s Republic of Korea (North Korea). Human infections were seen in Indonesia, Vietnam, Thailand, Cambodia, and China. In addition, fatal H5N1 infections were reported in zoo and domestic cats fed infected poultry, and H5N1 virus transmission to domestic cats was confirmed in laboratory studies. In some of the infected tigers, which lived in a zoo in Thailand, the virus seems to have spread horizontally. There were also reports of infected zoo birds in Indonesia and pigs in China. In April 2005, a H5N1 virus killed more than 6,000 migratory birds at isolated Qinghai Lake in central China. Mongolia also reported the death of 89 migratory birds at two lakes in August 2005. These various reports were worrisome, as they suggested that the H5N1 viruses were adapting to multiple mammalian and avian hosts. As of November 2005, more than 150 million poultry had been culled or died in this outbreak, which was not yet under control. The U.S. Centers for Disease Control and Prevention (CDC) warned that the H5N1 virus now seemed to have become endemic among birds in Asia.

In 2005, H5N1 viruses spread beyond Southeast Asia. They were first reported in Russia and Kazakhstan. Authorities in these countries hoped to confine the virus to Asia by intensive eradication efforts. But within a few weeks, a H5N1 virus passed the Ural mountains, the boundary between Europe and Asia, and appeared in Turkey and Romania, prompting mass culls and fears of a worldwide panzootic. In Croatia, a H5 virus was found in dead wild swans at a fish pond. As a result, Croatia began to cull poultry in villages near the pond, and stopped all bird and poultry exports. In Germany, up to 25 wild geese and ducks were found dead at a pond in October 2005, and avian influenza virus was isolated from some of these birds. Cases of H5N1 HPAI were also reported in the Aegean Sea islands in Greece. How the H5N1 virus entered Europe is unknown, but there are suspicions that migrating wild birds might be carrying the virus into new regions. Some nations in the European Union mandated or recommended that poultry flocks, particularly those located near wetlands, be kept indoors to reduce the possibility of virus transmission from this source. In October 2005, the United Kingdom reported that the H5N1 virus had been found in mesias (a type of bird) that died in quarantine during the import process. Partly as a result of this finding, the European Commission banned the importation of wild birds into the E.U., with some exceptions allowed under special circumstances or quarantine conditions.

The specific origins and parent viruses of the currently circulating H5N1 viruses remain to be determined, but circumstantial evidence sug-
gests that southern China may be the reservoir. Although they have not been fully characterized yet, these viruses seem to be related to H5N1 viruses found in Hong Kong from 1997 to 2002. Isolates from South Korea resemble the virus found in Penfold Park in 2002. The current H5N1 viruses also share some characteristics with one of the genotypes found during the 2001 outbreak. There is also some evidence that the H5N1 viruses may be evolving as the outbreak continues. Genetic differences have been reported between the South Korean viruses, which did not infect humans, and viruses that infected humans in Vietnam and Thailand. Isolates from South Korea, which acted very quickly to contain its outbreak, are genetically homogeneous; the H5N1 viruses found in some Southeast Asian countries with prolonged outbreaks are heterogeneous.

Fears of a Human Pandemic

One of the major concerns in the Southeast Asian outbreak has been the ability of the virus to infect humans. Between December 2003 and November 29, 2005, 133 human cases and 68 deaths were confirmed in Thailand, Vietnam, Cambodia, Indonesia, and China. Although a few of these cases may have been due to limited person-to-person transmission, the vast majority seemed to be caused by direct contact with poultry. However, authorities fear that the current viruses could recombine with a human influenza virus and produce an isolate that is more easily transmitted from person to person. An avian virus could also adapt to humans without reassortment, if it developed certain mutations. Fears of a new human pandemic are fueled, in part, by the recent discovery that the H1N1 virus responsible for the deadly 1918 pandemic was probably an avian influenza virus that became adapted to humans. The H5N1 viruses isolated from humans in the current epidemic share certain genetic features with the H1N1/1918 virus. In addition, the human population does not have immunity to H5N1; the currently circulating human influenza viruses are H1N1, H1N2 and H3N2.

As a result of this epidemic, countries are establishing plans to protect their populations in the event of an avian influenza pandemic in humans. Some countries have stockpiled antiviral drugs. A human H5N1 vaccine is also in development. Two events - reports of sustained human-to-human transmission, or genetic reassortment with human influenza viruses - may signal that the H5N1 virus is adapting to humans. As of November 2005, neither event had been reported.

Highly Pathogenic Avian Influenza and the U.S.

Highly pathogenic avian influenza is a foreign animal disease in the U.S. Although outbreaks of LPAI are relatively common, only three or four outbreaks of HPAI have been recorded in the U.S. since 1900. The first, in 1924-1925, was associated with live bird markets. This virus, which seemed to be disseminated mainly through the movement of poult-
try, spread to nine eastern states before being eradicated. HPAI was seen again in 1929; it may have been caused by the same virus or it may have been a new introduction. This disease was not reported again until the autumn of 1983, when a H5N2 virus caused an extensive epidemic in Pennsylvania and surrounding states. This virus, which was highly virulent in chickens, turkeys, and guinea fowl, was very similar to a LPAI virus that had been circulating in the area for 6 months. As a result, some birds had immunity to the HPAI virus, which complicated diagnosis and probably helped the virus spread further. Control and eradication of this epidemic, which was not completed until 1984, cost over $63 million in federal funds and an additional $350 million in increased consumer costs. Over 17 million birds died or were slaughtered. There was no evidence of transmission to humans. HPAI was not reported again until February 2004, when a virus was isolated from a south-central Texas broiler chicken flock. This virus, which also had the subtype H5N2, caused symptoms consistent with LPAI in the Texas flock, and was not virulent for experimentally infected chickens. However, some of its genetic characteristics suggested that it was a HPAI virus. Researchers also found that further changes in its hemagglutinin gene could increase the mortality rate. As a result, this virus was designated as a HPAI virus, and the USDA and state of Texas culled the approximately 6,600 birds in the flock. Surveillance on all flocks within 10 miles of the affected farm revealed no additional cases. No zoonotic infections were reported.

Like other countries, the U.S. is concerned about the possibility that the H5N1 strains from Southeast Asia could enter domestic poultry flocks or wild birds. APHIS has re-examined its HPAI prevention and eradication plans in light of that epidemic. All imported birds, including pet birds of U.S. origin, must now be quarantined and tested for the avian influenza virus before they enter the country. In addition, APHIS has placed trade restrictions on the importation of poultry or poultry products from countries that have reported cases of HPAI. Poultry or poultry products from East and Southeast Asia must be processed or cooked before importation to destroy any influenza viruses. APHIS has also alerted the U.S. Department of Homeland Security to be particularly vigilant in its agricultural inspections of passengers and cargo from Asia, and has increased its surveillance of domestic markets for illegally imported poultry products. In addition, the USDA is working with the World Organization for Animal Health (OIE), the United Nations' Food and Agriculture Organization (FAO), and World Health Organization (WHO) to help affected countries and their neighbors with disease prevention, management, and eradication to reduce the global threat from this virus.
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**Bovine Spongiform Encephalopathy in the United Kingdom, 1986-2005**

An epidemic of bovine spongiform encephalopathy (BSE) was first recognized in the United Kingdom in 1986. From 1986 to February 2003, over 179,900 cattle on more than 35,740 U.K. farms were affected. The epidemic peaked in January 1993 at almost 1,000 new cases per week. BSE also spread to other European countries that had imported cattle from the U.K., and eventually a small number of cases were recognized in North America. The BSE outbreak may have resulted from the feeding of scrapie-containing sheep meat-and-bone meal to cattle or it may have arisen as a rare spontaneous formation of a spongiform encephalopathy in a cow that then spread to other cattle through contaminated meat and bone meal. There is strong evidence and general agreement that the outbreak was amplified by feeding rendered bovine meat-and-bone meal to young calves. Most countries have now banned this practice and, as a result, the BSE epidemic has been waning for the last decade. A parallel outbreak of new-variant Creutzfeldt-Jakob disease (vCJD) in humans is most likely a result of the consumption of beef products contaminated by central nervous system tissue from cattle with BSE. As of June 2005, 177 cases of vCJD had been reported, mainly in the United Kingdom. Deaths due to vCJD peaked in 2000 and have since been declining.

BSE developed into an epidemic as a consequence of an intensive farming practice—the recycling of animal protein in ruminant feed. The question of how to handle the BSE agent, a known hazard to cattle and potential hazard to humans, is key to the BSE story. The government took measures to address both hazards, but they were not always timely or adequately implemented and enforced because the basic biology of BSE was unknown and it was believed that BSE was not a threat to human life.
What are BSE and vCJD?

BSE is a progressive neurological disorder of cattle that results from infection by an unconventional transmissible agent. The causative agent of BSE and other transmissible spongiform encephalopathies (TSEs) is yet to be fully characterized. The BSE agent is smaller than most viral particles and is highly resistant to heat, ultraviolet light, ionizing radiation, and common disinfectants that normally inactivate viruses or bacteria. It causes no detectable immune or inflammatory response in the host and has not been observed microscopically. The incubation period for BSE ranges from two to eight years and clinical disease usually occurs in older animals. Most cases in the United Kingdom were seen in dairy cows between three and six years of age. Affected animals may display changes in temperament such as nervousness or aggression, abnormal posture, incoordination and difficulty in rising, decreased milk production, or loss of body condition despite continued appetite. Following the onset of clinical signs, the animal’s condition deteriorates until it dies or is destroyed. This usually takes from two weeks to six months. There is no treatment.

Creutzfeldt-Jakob disease (CJD) is a rare and fatal human neurodegenerative disease of unknown cause. Patients with the conventional form are usually between 50 and 75 years of age. The new variant form (vCJD) in the United Kingdom mainly affects younger people; the median age at death is 28 years.

The first cases of BSE

Individual cattle were probably first infected by BSE in the 1970s. If they lived long enough to develop signs of disease, these were not reported to or investigated by the Central Veterinary Laboratory (CVL) of the State Veterinary Service (SVS). The first clinical cases were reported in 1984, although it was two years before the nature of the disease was actually recognized.

On December 22, 1984, Peter Stent of Pitsham Farm in Sussex called Dr. David Bee, a private veterinarian, to examine Cow 133. The cow had an arched back and had lost weight. Dr. Bee visited the farm several times over the following months, and continued to see animals showing unusual symptoms. Cow 133 developed a head tremor and incoordination before dying on February 11, 1985. By the end of April, five more cows on the farm had died. Dr. Bee requested assistance from Dr. J. M. Watkin-Jones, a veterinarian at the Winchester Veterinary Investigation Center (VIC) of the Veterinary Investigation Service. A number of samples of body tissue were submitted to the CVL for pathological analysis. Various possible ailments were identified, but despite a wide range of tests there was no definite diagnosis. The CVL suggested that Mr. Stent submit a live affected cow for an epidemic of BSE was first recognized in the United Kingdom in 1986. The epidemic peaked in January 1993 at almost 1,000 cases per week. A parallel outbreak of new-variant Creutzfeldt-Jakob disease (vCJD) in humans is most likely a result of the consumption of beef products contaminated by central nervous system from cattle with BSE.
Normal bovine brain tissue

The spongiform lesions of BSE
Source: USDA APHIS

slaughter and post-mortem. Cow 142 was sent live to the CVL in September for euthanasia and a post-mortem examination. The pathologist on duty examined the tissues and concluded that the problem was associated with fungal contamination of feed and mycotoxin production. An April 1985 laboratory report stated that a fungal toxin called citrinin had been found in the feed at the farm. New cases ceased to develop on the farm and the veterinarians assumed that the problem had run its course (from www.bseinquiry.gov.uk/report/volume3/chapterd.htm).

The mysterious disease soon reappeared on other farms. At the end of 1986, the Pathology Department of the CVL considered four more cases of unusual neurologic disease in cattle from farms in Kent and Bristol. They identified these cases as a probable transmissible spongiform encephalopathy in cattle and named the new disease bovine spongiform encephalopathy. By the end of 1987, the CVL Epidemiology Department concluded that the cause of the reported cases of BSE was the consumption of meat-and-bone meal (MBM), which was made from animal carcasses and incorporated into cattle feed. At first it was thought that cattle were becoming infected from scrapie-contaminated sheep tissues and that the MBM had become infectious because rendering methods that had previously inactivated the conventional scrapie agent were changed. However, the cases of BSE identified between 1986 and 1988 were not index cases, and they were not the result of the transmission of scrapie. They were the consequences of recycling BSE-infected cattle into MBM. In addition, the theory that BSE resulted from changes in rendering methods is probably not correct because rendering methods have never been capable of completely inactivating TSEs. Although the origin of the disease will probably never be known, BSE probably originated from a novel source early in the 1970s, possibly a cow or other animal that developed the disease spontaneously. The disease did not become apparent until the agent had been disseminated to large numbers of cattle via MBM and, after a long incubation period, these cattle began developing clinical signs.

**Precautions taken**

In June 1988, the Southwood Working Party, set up to provide advice on the implications of BSE, recommended that cattle showing signs of BSE be destroyed and that compensation be paid to farmers. In February 1989, the Southwood Working Party submitted a report to the government that concluded that the risk of transmission of BSE to humans appeared remote and that ‘it was most unlikely that BSE would have any implications for human health.’ This assessment of risk was made assuming that BSE was probably derived from scrapie and could be expected to behave like scrapie. The Southwood Report never underwent a scientific review by experts in the field. Precautionary measures were put in place that went...
beyond those recommended by the Working Party and an expert committee was set up to advise BSE research.

Once MBM was identified as the probable vector of BSE in 1988, the government implemented a ban on incorporating ruminant protein in ruminant feed. This ban reduced the escalating rate of infection. After BSE was experimentally transmitted to a pig in 1990, new measures to protect pigs and poultry from BSE were introduced. However, the measures were unenforceable and widely disregarded. It was later discovered that a cow could become infected with the BSE agent by eating an amount of infectious tissue as small as a peppercorn. Cross-contamination in feedmills caused thousands of cattle to become infected, but because of the long incubation period this was not apparent until later. In 1994, because of the continuing infection, regulations were revised and a rigorous enforcement campaign was initiated. After March 1996, the incorporation of all animal protein in animal feed was banned. The BSE epidemic in the United Kingdom peaked in 1993 and, as a result of these control measures, has subsided.

Recognition of the potential risks to humans

In June 1989, specified bovine offal (SBO) was banned from use in human food as a precaution. Specified bovine offal includes the brain, spinal cord, spleen, thymus, tonsils, and intestines of cattle. At the time of the ban, some questioned whether all of the spinal cord could be removed during the abattoir process. Questions were also raised about the process of mechanical recovery of scraps left attached to the vertebral column for use in human food (mechanically recovered meat). Instances of failure to remove all of the spinal cord from the carcass were discovered and in December 1995, the extraction of mechanically recovered meat from the spinal column of cattle was banned. Mechanically recovered meat can include dorsal root ganglia that have been demonstrated to be infectious in the late stages of incubation.

In May 1990, a domestic cat was diagnosed as suffering from a ‘scrapie-like’ spongiform encephalopathy. This generated widespread public and media concern that BSE had been transmitted to the cat and might also be transmissible to humans. As time passed, the increasing knowledge about BSE made the theory that it would behave like scrapie less and less viable. The public was not informed of any change in the perceived likelihood that BSE might be transmissible to humans and in fact was repeatedly reassured that it was safe to eat beef. There was, nevertheless, some recognition that the pathways by which bovine products or by-products might come into contact with humans or other animals needed to be examined. Known or suspected pathways included

As of 2005, there is no diagnostic test to detect the BSE agent in living animals.
Source: Travis Engelhaupt, ISU
meat, vaccines, cosmetics, surgical instruments, bovine or human tissues, agricultural fertilizer, and agricultural waste. However, no coordinated consideration was implemented until March 1996.

**The first human cases**

Scientists suspected that if BSE were to spread to humans it would resemble Creutzfeldt-Jakob disease. In 1991, surveillance for atypical cases or changing patterns of CJD was put in place. Three dairy farmers who had had BSE in their herds were diagnosed with CJD in August 1992, July 1993, and December 1994. The fourth annual report of the CJD Surveillance Unit (CJDSU), issued in August 1995, noted the apparently high incidence of CJD in farmers. The Spongiform Encephalopathy Advisory Committee (SEAC) released a press release about suspected CJD in a cattle farmer in October 1995.

In May 1995, Stephen Churchill, age 18, died. He was later confirmed as the first known victim of vCJD. His was one of three vCJD deaths in 1995. The CJDSU identified its second suspect vCJD death in a remarkably young patient in August 1995. A third individual died in November 1995. Both cases were later confirmed as vCJD. The CJDSU announced the emergence of vCJD and on March 16, 1996, the SEAC announced that the most likely explanation for the cases of a new variant of CJD in young people was exposure to BSE. This has since been compellingly supported by scientific evidence. A policy of banning consumption of cattle over 30 months of age was introduced. However, the incubation period for transmissible spongiform encephalopathies is long and, not surprisingly, cases of vCJD continue to be diagnosed. Annual vCJD-related deaths in the U.K. rose gradually to a high of 28 in 2000, and tapered to nine in 2004. Three deaths were reported from January 1, 2005 to October 8, 2005, bringing the total number of deaths in the U.K. to 151. As of June 2005, 177 cases of vCJD had been reported worldwide, including one from the U.S. in 2002. Nearly all of these people had lived in the U.K. during multiple years between 1980 and 1996 and had been exposed to BSE there.

**BSE outside the United Kingdom**

When the BSE epidemic became evident, the European Union prohibited the export from the U.K. of live bovine animals, their semen and embryos, mammalian-derived MBM, or the meat of bovine animals slaughtered in the U.K. that is liable to enter the animal feed or human food chain. The export of materials destined for use in medicinal products, cosmetics, or pharmaceuticals was also banned. Despite these measures, BSE spread to countries outside the U.K. Eighteen European countries have reported at least one case of BSE in indigenous cattle. Portugal had the highest incidence rate; in 2001, it reported more than 100 indigenous cases per million cattle aged over 24 months. Significant numbers of cases
As of October 2005, the intensified surveillance has found one case of BSE in an indigenous animal. A 12 year old Brahma cross cow from Texas was sold in a livestock sale and transported to a packing plant. The animal, which was dead on arrival, was sent on to a pet food plant, where it was sampled for BSE. The carcass was incinerated and was not used in pet food. The test results on this animal were initially conflicting, but in June 2005, a sample sent to a BSE reference laboratory in the U.K. was determined to be positive. APHIS, the FDA, the Texas Animal Health Commission and the Texas Feed and Fertilizer Control Service conducted a joint investigation to determine the source of the infection and test the infected cow’s cohorts and offspring. The infected animal was traced to the ranch in Texas where it had been born and raised. All adult animals that left this farm after 1990, and the two calves born to the infected cow within 2 years of its death were traced. Most of these animals had been slaughtered, died or were presumed to be dead, although a few animals were untraceable. One surviving animal was tested and found to be negative, and another was determined to be of no interest due to its age. As the BSE-infected cow had been born before the USDA implemented the ban on MBM in ruminant feeds in 1997, it appears to have been infected from that source.

As of October 8, 2005, the surveillance program has tested more than 484,000 cattle, with no additional positive animals found. However, because of these two BSE cases, a number of countries have banned the importation of a variety of ruminant products or live animals from the U.S. These bans vary greatly. While some nations now prohibit the importation of all U.S. beef, others have placed only temporary or limited bans, such as a ban on beef products from Texas and Washington states. Some countries allow certain products from animals of a specified age or under specific conditions.
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Emerging & Exotic Diseases of Animals
Canine Influenza, 2004-2005

Viruses rarely jump from one species into another. When they do, the outbreak tends to be brief; typically, the virus is poorly adapted to the new host and cannot be transmitted efficiently in the new species. Two characteristics of influenza viruses — their high mutation rate and their ability to recombine with each other — help them adapt to new species. Although most cross-species infections by these viruses are self-limited, there are records of a few permanent jumps. One is currently occurring in dogs, which have acquired an equine influenza virus from horses.

In January 2004, 22 racing greyhounds at a Florida racetrack became ill with an unknown respiratory disease. Fourteen of the dogs developed a fever, followed by a persistent cough that lasted for 10 to 14 days. These dogs recovered. Eight other dogs died suddenly with evidence of hemorrhages in the respiratory tract. At necropsy, the fatal cases had signs of severe hemorrhagic pneumonia. Using lung tissue samples from the dead dogs, researchers discovered an influenza A virus that was very similar to a H3N8 equine influenza virus circulating in horses. All of the genes in the canine virus were of equine origin, suggesting that the virus had been transmitted whole from horses into dogs. Serology using paired acute and convalescent sera demonstrated that the recovered dogs had also been infected by this virus. Some asymptomatic dogs that had been exposed during the outbreak also seroconverted. In addition, it became apparent that the canine influenza virus had been circulating among greyhounds in Florida for several years. Some Florida racetracks had experienced outbreaks of an unknown respiratory disease from 1999 to 2003; seropositive dogs were found at these tracks. Antibodies to the virus were not found in canine serum samples from 1996 to 1998. The same virus, or a very similar one, was also found in preserved lung tissues from a greyhound that died of an unknown respiratory disease in 2003. At first, the canine influenza virus did not seem to be a threat to other breeds of dogs. Experimentally infected beagles developed a fever, but no respiratory signs.

In 2004 and 2005, canine influenza continued to be reported in greyhounds. Between June and August 2004, outbreaks of respiratory disease were seen at 14 greyhound racetracks in Alabama, Arkansas, Florida, Kansas, Texas, and West Virginia. Some of these outbreaks were linked to the canine influenza virus and others were not investigated. From January to May 2005, more episodes were seen. The disease affected greyhounds at 20 tracks in Arkansas, Arizona, Colorado, Florida, Iowa, Kansas, Massachusetts, Rhode Island, Texas, and West Virginia. The evidence that the new virus was circulating widely in racing greyhounds made researchers suspect that it might become a threat to other breeds of dogs. Serology on dogs with respiratory disease in shelters and veterinary clinics in Florida and New York revealed evidence of the virus in pets and prompted state offi-
cials to issue a warning to the public. Recently, cases of canine influenza were identified in pets in other states including California and Washington. It now appears that this virus may be an unrecognized cause of respiratory disease in dogs throughout the U.S.

**What are influenza viruses and how do they jump from species to species?**

Influenza is caused by influenza viruses, members of the Orthomyxovirus family: There are three genera of these viruses: influenza A, influenza B and influenza C. The influenza B and C viruses mainly infect humans, while the influenza A viruses also infect other mammals and birds. Influenza A viruses are classified into subtypes based on two proteins, the hemagglutinin (‘H’) and neuraminidase (‘N’). There are at least 16 different hemagglutinin antigens (H1 to H16) and 9 neuraminidase antigens (N1 to N9). Antibodies to the hemagglutinin and neuraminidase proteins are important in immunity to these viruses. The H and N proteins also help the virus attach to cells and aid in releasing newly formed viruses from a cell; therefore, these proteins help determine the species specificity of each influenza virus. However, these proteins are not the only ones important in adapting the virus to a species; internal virus proteins must also have a good ‘fit’ with the cell if the virus is to efficiently replicate and spread. Wild birds, particularly waterfowl, are the reservoirs for the influenza A viruses; these birds carry all of the subtypes but rarely become ill themselves. The avian influenza viruses are sometimes transmitted to poultry, which may cause outbreaks of avian influenza in these birds. Some influenza viruses have become adapted to mammals. They include the equine influenza viruses, swine influenza viruses, and human influenza viruses. The equine, swine, and human viruses are now well adapted to their host species and are not easily transmitted to other species of mammals or birds.

Although jumps from one species to another are rare, they are aided by the influenza virus’ tendency to change. Influenza viruses quickly accumulate small mutations, a process called ‘antigenic drift.’ In addition, they can exchange proteins with other influenza viruses, an ability facilitated by their segmented genome. If two influenza viruses infect a cell simultaneously, the segments may mix when new virus particles are assembled. An influenza virus can ‘reassort’ with any other influenza virus, regardless of its origin. For example, if a cell is infected by a swine and a human influenza virus, the new viruses budding from that cell might contain some pieces from the swine influenza virus and other pieces from the human influenza virus—a process that could make the swine virus better able to infect human cells. Reassortment is particularly common in pigs, which have receptors for the human, swine and avian influenza viruses. Sometimes, an influenza virus can also jump ‘whole’
from one species to another. Such jumps have been seen when avian influenza viruses infected people, cats, mink, seals, horses and other animals, and swine influenza viruses infected humans and turkeys. Usually, the virus is poorly adapted to the new species, can’t be transmitted efficiently, and quickly dies out. Occasionally, a virus is able to replicate and spread well in the new hosts, and a permanent jump is made. Such permanent jumps were seen in 1918, when pigs acquired their first influenza viruses, and in China in 1989, when horses were infected by a new kind of equine influenza virus. Until recently, no influenza viruses circulated in dogs or cats.

Where did the canine influenza virus come from?

An analysis of the canine influenza virus isolated from greyhound lung tissue has demonstrated that this virus is most closely related the H3N8 “Florida lineage” equine influenza virus that emerged in the early 1990s. There are four amino acid differences between the hemagglutinin proteins in the equine and canine viruses; these changes were probably important in adapting the virus to dogs. Although it’s remotely possible that this virus was repeatedly introduced into dogs from some other species, the evidence suggests that a single virus was transmitted whole from horses to dogs, as a one-time event. Transmission between dogs probably occurs via aerosols, similarly to other influenza viruses. However, canine influenza viruses have, so far, proven impossible to isolate from naturally infected, live animals, possibly because virus isolation has always been attempted relatively late in the infection, after the symptoms have appeared.

What can we expect from the canine influenza virus?

Canine influenza is an emerging disease in dogs. Dogs are not expected to have any naturally-acquired or vaccine-induced immunity to this virus, and some experts warn that the canine population may be facing a pandemic similar to the influenza pandemics that swept through humans in 1918, 1957 and 1968, or swine in 1918. Although early reports suggested that canine influenza was limited to greyhounds, all dogs regardless of breed or age are now considered to be susceptible. In kennels, the infection rate may reach 100% and symptoms may be seen in 75% of the dogs infected. Most dogs are expected to develop the less severe form of the disease, and recover. In this form, the major symptom is a cough that may persist for up to 3 weeks, in spite of treatment. Occasionally, the cough is accompanied by a fever and/or a nasal discharge that responds to antibiotics. A few dogs develop a more severe form with pneumonia and possibly pulmonary hemorrhages. In dogs with severe disease, the overall mortality rate is thought to be 1-5%. However, as experience with this virus grows, these numbers may be adjusted up or down; some
sources suggest that the mortality rate may be as high as 10%, while others expect a high prevalence of mild, self-limiting or asymptomatic infections and suggest that the overall mortality rate in pets will be less than 1%. A vaccine for dogs, based on the equine influenza vaccine, is in development and may be available soon.

Once some dogs develop either naturally-acquired or vaccine-induced immunity to this virus, any epidemic or pandemic will probably subside. However, all influenza viruses constantly acquire small changes and periodic outbreaks may continue, similar to the yearly flu epidemics in humans or outbreaks of equine influenza in horses.

Are there any public health concerns about this virus?

There is currently no evidence that any other species, including humans, can be infected by the canine influenza virus. However, some experts are concerned that, due to their close associations with humans, dogs might become a source of novel influenza virus transmission to humans. As a precaution, physicians, veterinarians and others have been asked to report any cases of human influenza that seem to be linked to exposure to canine influenza.

Sources of Information


Classical Swine Fever in Great Britain, 2000

This is an example of how the British veterinary infrastructure was able to trace, control, and eradicate an outbreak of classical swine fever before the disease became widespread. Classical swine fever is an exotic disease that has been eradicated from a number of developed countries; however, it still exists in some parts of the world and could be re-introduced at any time in infected animals or animal products.

On August 4, 2000, a suspected case of classical swine fever (CSF) in a pig herd was reported to the British Ministry of Agriculture, Fisheries, and Food (MAFF) Animal Health Divisional Office at Bury St Edmunds, Suffolk. The herd consisted of 3,500 weaned pigs in seven houses. The pigs had been ill since July 11, when weaned pigs had been introduced from a breeding/multiplier unit. The infection had spread to four houses and as of August 4, a total of 1,110 pigs were ill and about 200 had died. A MAFF veterinary officer visited the premises the same day and, after examining the pigs on site, placed the holding under official movement restrictions and took blood samples to test the pigs for classical and African swine fever. On August 7, two cases of suspected classical swine fever were reported on other farms. One case was in a herd of rearing pigs. The second was in a breeding herd that had supplied weaned pigs to

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the other two infected farms. Both herds were immediately placed under quarantine and blood samples were sent for laboratory examination.

An outbreak of classical swine fever was declared on August 8, 2000. National and local crisis centers were established to deal with the outbreak. Three-kilometer protection and 10-kilometer surveillance zones were established around the infected premises and the movement of all pigs within the zones was prohibited. The remaining 3,300 pigs on the first identified farm were killed on August 10 and their carcasses destroyed by rendering. The premises were cleaned and disinfected on August 11. The other two farms were also depopulated. The movements of pigs, feedstuffs, vehicles, and people onto and off the premises were traced to identify possible sources of the virus and limit the spread of infection.

During the next few months, classical swine fever was found on several more farms. Before the first farm had been placed under quarantine, it had sent infected pigs to four other premises. The disease also spread to two contiguous outdoor pig farms. From one of those, classical swine fever spread to another contiguous holding and then, through the movement of pigs, to two additional premises. Two more outbreaks occurred in pig units owned by haulage operators. A total of 16 infected sites were confirmed in Great Britain between August 4 and November 3. However, by December the outbreak had been contained. All controls relating to the 16 infected premises were lifted on December 30, 2000.

**What is classical swine fever?**

Classical swine fever, also known as hog cholera, is a contagious febrile disease of pigs. This disease is caused by infection with the classical swine fever virus, a member of the Pestivirus genus of the Flaviviridae family of RNA viruses. Pigs can become infected by ingestion, inhalation, genital (semen) infection, or wound contamination. Classical swine fever is most easily spread by contact with infected pigs or the feeding of inadequately cooked garbage (swill). Spread of the virus by fomites or by biting insects is also possible. The clinical signs include lethargy, yellow diarrhea, conjunctivitis, incoordination, fever, and excessive thirst. Additional signs include skin lesions ranging from cyanotic patches on the ears and abdomen to raised, scabby lesions mainly on the legs. Classical swine fever strongly resembles African swine fever and must be distinguished from it by laboratory tests.

**Tracing the virus' footsteps**

The source of the outbreak appears to have been the breeding farm identified on August 7. The epidemiological inquiry found that the CSF virus probably entered the breeding unit on May 1 then spread to the index farm and herd of rearing pigs by the movement of infected pigs.
Classical swine fever is most easily spread by contact with infected pigs or the feeding of inadequately cooked garbage (will). Spread of the virus by lice or by vectors (e.g., biting insects is also possible.

These three farms were all owned by or contracted to the U.K.’s largest outdoor pig rearing company. The company’s pigs were born on breeding units and remained there for approximately three to four weeks before being moved to rearing premises where they remained for a further six to eight weeks. From the rearing units, the pigs moved to finishing units where they remained for 10 weeks before being slaughtered. This method of swine production was designed to reduce the transmission of enzootic diseases by early weaning of pigs from the breeding farm to a series of remote locations. Disease transmission from older finishing pigs to young growing pigs is avoided by having a series of separate finishing farms.

All rearing and finishing premises that had received pigs born after May 1 at the breeding unit were traced, tested for classical swine fever, and placed under official movement restrictions. All the pigs on premises that had received pigs born after June 1 were treated as “dangerous contacts” and were destroyed. The other 47 breeding herds owned by or contracted to the production company were traced, placed under quarantine, clinically inspected by a MAFF veterinary officer, and sampled for evidence of CSF. The government traced the movements of the transporter who took weaned pigs from the breeding premises. All the premises that the transporter visited were tested and placed under official movement restrictions.

The origin of the virus and its route of introduction were not established with complete certainty. However, the evidence strongly suggests that infection did not come through the introduction of infected pigs, contact with feral pigs, contaminated vehicles or personnel, discharges of effluent, or contaminated vaccines or biological products. It appears more likely that the infection was introduced in contaminated pig meat in food discarded by people; a public footpath runs adjacent to the outdoor paddocks containing dry sows on the breeding farm. Genetic typing showed that the outbreak was caused by a virus strain that is not currently present in Europe. This strain is in the same genetic group that was isolated during a classical swine fever outbreak in Belgium, Italy, the Netherlands, and Spain in 1997–98.

Sources of Information


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*defunct link as of 2005
Foot and Mouth Disease in the United Kingdom, 2001

This is example of how foot and mouth disease can spread in a country with a veterinary infrastructure similar to that of the United States and how the international community reacted to an outbreak in the U.K. Outbreaks of foot and mouth disease cause major economic and trading difficulties for infected countries. Because the disease can spread on fomites as well as between animals, it can sweep through a country rapidly in spite of control measures.

How it began

On February 19, 2001, a veterinary inspector from the State Veterinary Service of the Ministry of Agriculture, Fisheries and Food (MAFF), undertaking routine inspections at an abattoir at Little Warley near Brentwood, Essex, saw vesicular lesions on 27 sows and one boar. Vesicles (skin blisters) are a characteristic symptom of foot and mouth disease (FMD). Vesicles caused by FMD are clinically indistinguishable from those caused by other vesicular diseases such as vesicular stomatitis, swine vesicular disease, or vesicular exanthema of swine. In this case, laboratory tests confirmed the disease to be foot and mouth disease. On February 20, MAFF announced an immediate "stop movement" of all susceptible livestock in the United Kingdom, including the movement of animals to abattoirs, sale markets, and pastures.

Efforts to trace the disease back to the infected farm and suppress the outbreak began immediately. The infected pigs had arrived at the abattoir on February 16 from farms in Buckinghamshire and the Isle of Wight. The pigs were traced back to a farm at Heddon-on-the-wall, Northumberland. By the time the outbreak was discovered, foot and mouth disease had spread to a cluster of holdings in the County of Essex through the movement of pigs and people and local airborne spread. Infected sheep from the farm at Heddon-on-the-wall had also been moved to the Longtown market near Carlisle. These sheep infected thousands of additional sheep and cattle holdings in other parts of Great Britain, initially through the movement of sheep through markets and subsequently by local spread around infected holdings.

What is foot and mouth disease?

Foot and mouth disease is a highly infectious viral disease that can affect all cloven-hoofed animals including cattle, swine, deer, goats, and sheep. More rarely, it affects hedgehogs, rats, elephants, giraffes, and antelopes. The FMD virus is spread in aerosols and on fomites such as manure-contaminated tires, boots, and clothing. The disease is characterized by fever and vesicles, which progress to erosions in the mouth, nares, muzzle, feet, or teats. In cattle, oral lesions are common, with vesicles on the tongue, dental pad, gums, soft palate, nostrils, or
muzzle. Hoof lesions can be found in the area of the coronary band and interdigital space. The erosions are quite painful and affected animals are lame, refuse to eat, and may lose weight. The mouth lesions can cause profuse salivation. Sheep and goats show very mild, if any, signs. Animals generally recover in about two weeks but secondary infections may lead to a longer recovery time.

**How it entered the U.K.**

Seven immunologically different serotypes of the FMD virus are known to exist. The virus in the U.K. was identified as serotype “O” Pan-Asian. This strain was first recognized in India in 1990 and has since spread to a number of countries around the world. It is identical to the virus found in recent outbreaks in Africa, including one in South Africa where the virus was traced to pig swill—waste food from human tables—sold illegally from an Asian boat.

The source of the 2001 epizootic in the U.K. is also thought to have been pig swill. The feeding of pig swill is a practice that has been going on for generations. Today, pig swill comes from restaurants, schools, and anywhere humans eat and waste food on a large scale. In recent years, the feeding of pig swill has declined because it is thought to be inefficient and outmoded. In 1998, a government panel of agricultural experts advised that it be banned; however, the advice was rejected by ministers who did not want to impose new costs on hard-pressed farmers. Only about one percent of producers in the U.K. were using pig swill at the time of the outbreak. Farmers are supposed to treat the swill by heating it to 100 degrees centigrade to kill potential pathogens. MAFF officials suspect that the infectious swill originated as waste food from a ship or international restaurant that was not properly heat-treated.

**The spread**

By March 2, foot and mouth disease had spread to 40 locations, with many linked to infected markets. A total of 25,000 animals had been destroyed and incinerated on-farm. An outbreak was also confirmed in County Armagh in Northern Ireland. (The term “outbreak” used here refers to infections at a farm or abattoir that was previously uninfected.) On March 9, there were cases in 127 locations. The MAFF sent information to farmers and veterinarians on how to avoid spreading FMD and how to report suspected outbreaks. It also publicized the details of the clinical signs of FMD in sheep, as the symptoms in this species can be subtle.

At the start of the outbreak, MAFF veterinarians who had been on infected premises were required not to have contact with uninfected, susceptible animals for five days. A shortage of “clean” unexposed veterinarians quickly developed. Private practice veterinarians and foreign government veterinarians were enlisted to help with the outbreak. The U.S.

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sent the first group of 20 veterinarians the week of March 5. One month after the start of the outbreak, MAFF decreased the time required to become “clean” from five to three days to enable more veterinarians to investigate potentially infected premises. Eventually the time required to become “clean” decreased to 24 hours to visit a highly suspect farm. Veterinary teams for infected premises, surveillance, and trace back from sale markets were established to better utilize personnel.

The control measures implemented by MAFF resulted in a number of difficulties. Because of the restriction of animal movement, cows could not cross roads for milking or be moved to fresh grazing pastures. Pregnant ewes were prevented from moving to shelter for lambing. There was a public outcry to allow some animal movement for welfare reasons. To reduce the transmission of virus by humans, footpaths in the countryside were closed and the public was strongly discouraged from going anywhere near livestock farms. Carcass disposal also became a problem. The MAFF initially planned to render the carcasses of destroyed livestock rather than incinerate them on-farm. However, the large number of carcasses resulted in a lack of sealed trucks for hauling carcasses to rendering plants, delays in burial, and shortages of material for incineration. The National Farmers Union (NFU) protested the delay in destroying infected animals and burning carcasses. About one month after the start of the outbreak the military became involved to coordinate the disposal of carcasses.

In spite of control measures, the epidemic continued to spread and cases began to appear outside the U.K. On March 13, FMD was confirmed at La Baroche-Gondouin in northwestern France. The infected farm was already in a movement control zone, put in place around a sheep farm that had imported sheep from the U.K. two weeks earlier. The sheep had been preventively slaughtered at that time. Six cattle on the farm showed symptoms of FMD, and the entire herd of 114 cattle was destroyed. On March 15, the MAFF made the decision to “ring depopulate” in the U.K. A ring was defined as three kilometers around an infected premises. A total of 251 farms were infected on March 15 and about a million healthy animals were scheduled to be killed. The media called it “the mass cull.” On March 20, FMD was found in the Republic of Ireland; typical FMD lesions were detected in sheep on a farm only four miles away from the single outbreak which occurred in Northern Ireland. The farm was within the surveillance zone established after this earlier incident. The source of the Republic of Ireland outbreak was believed to be sheep imported via N. Ireland from mainland U.K.

On March 21, FMD was confirmed in four cows on a farm in the Netherlands. Temporary restrictions were imposed throughout the country on the movement of cattle, poultry, transport vehicles for cattle and

Animals with foot and mouth disease develop vesicles and erosions on the mouth, muzzle, feet, or teats. These lesions are indistinguishable from the lesions found in other vesicular diseases such as vesicular stomatitis, or vesicular exanthema of swine. Source: USDA

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poultry, and the semen, ova, and embryos of ungulates. All animals on the affected farm were immediately culled. The animals on the six farms within a one-kilometer radius of the infected holding were also destroyed. Animals were either buried on the farm or burned. All farms within a radius of three kilometers of the affected farm were inspected for signs of FMD. The FMD virus is believed to have reached the Netherlands via a shipment of veal calves from the Republic of Ireland. The calves were rested in an animal holding near Barouche Gondouin, France for 12 hours, where they were apparently infected by sheep coming from the U.K. On March 23, to fight the spread of FMD, European Union veterinarians in Brussels agreed to limited emergency vaccination in the Netherlands around infected farms and animals awaiting slaughter. This overturned the 15-year E.U. policy of prohibiting vaccination for FMD.

On March 30, there were 60 new outbreaks in the U.K., the highest daily total of the epidemic so far, with a total of 839 outbreaks to that date. The farming and tourism industries had by this point been devastated, and even politics was affected. On April 2, Prime Minister Tony Blair announced that the general election scheduled for May 3 would be delayed until June 7 because of the FMD crisis. However, the severe control measures eventually succeeded in controlling the epidemic. By June 12 the spread had slowed; only four new locations were affected that day, bringing the total number of new outbreaks to 1,736. By this time, over 3,281,000 animals had been slaughtered, and 8,334 premises had been affected.

**The international reaction**

The U.K. was required to notify the World Organization for Animal Health (OIE) of the outbreak within 24 hours of the first case. On the following day, February 21, the European Commission banned the export of live animals, germplasm, fresh meat, meat products, milk and milk products, hides, and skins of FMD susceptible species from all of the U.K.

**The U.S./USDA response**

Immediately after FMD was confirmed in the U.K., the USDA stepped up its efforts to guard against FMD. The importation of swine, ruminants, any fresh swine or ruminant meat (chilled or frozen), and other products of swine and ruminant origin from the European Union was temporarily prohibited. Travelers were prevented from carrying into the U.S. any agricultural products, particularly animal products from the European Union that could spread the disease. Security was tightened at ports of entry and airports to ensure that passengers, luggage, and cargo were checked as appropriate. The USDA also heightened the alert and coordination with state agriculture officials and other USDA officials stationed around the globe to monitor the situation, and developed a public education campaign that included additional signs in airports, public

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service announcements, an information hotline, website, and other tools to inform the public about the issue. In addition, the U.S. sent a team of experts to the European Union to monitor, evaluate, and assist in containment efforts.

As the FMD outbreak grew in the U.K., the USDA also established an emergency operations center to coordinate communication, answer technical questions, and provide consumer and traveler information about FMD and other related issues. In addition, the USDA reviewed its current Animal and Plant Health Inspection Service programs and staffing to ensure appropriate resources were available to prevent the entry of FMD into the United States, both short and long-term. Federal and state emergency operations plans were also reviewed to ensure that appropriate response mechanisms were in place to act quickly if FMD were ever to enter the United States.

**Final statistics**

The last case of this outbreak was reported on September 30, 2001, bringing the total number of confirmed cases in the U.K. to 2,030. Many more animals had been killed to prevent the spread of the disease. According to official U.K. government figures, 4,068,000 animals were culled between the first case on February 20, 2001 and the last case on September 30, 2001. Unofficial figures from the Meat and Livestock Commission put the number of animals slaughtered at more than 10 million. Those figures include animals slaughtered for welfare reasons such as dwindling feed and space, animals killed because there was no market for them, and animals killed with their mothers and only counted as one animal. On January 22, 2002, the OIE declared that the U.K. had regained its previously recognized FMD-free status without vaccination, clearing the way for international export trade in animals and animal products.

**Sources of Information**


Dr. Larry Ludemann, USDA APHIS, VS, Center for Veterinary Biologics, and a veterinarian assigned to assist with the outbreak in the U.K. in spring 2001.

*defunct link as of 2004
Foot and Mouth Disease in Uruguay, 2001

Foot and mouth disease (FMD) also occurred in Uruguay at the same time as the epidemic in the U.K. Although these two countries are approximately the same size, their livestock composition is quite different. Uruguay has nearly seven times as many cattle as the U.K. (10.6 million compared to 1.6 million) but fewer sheep and pigs. Faced with a similar number of FMD-infected farms, the two countries’ approach to this disease was drastically different. The U.K. used a stamping-out policy with no vaccination, while Uruguay culled few animals and concentrated its efforts on a massive vaccination campaign. In the U.K., more than 6 million animals were killed. In Uruguay, a little over 6,900 animals were killed, and over 24 million doses of vaccine were used. The two outbreaks lasted about the same time, but the overall cost to control the epidemic was far less in Uruguay. The FMD outbreak in the U.K. is estimated to have cost approximately $5 billion to agriculture and the food chain and an additional $5 billion from loss of tourism. The cost of the outbreak in Uruguay was $243.6 million, with much of this due to the loss of export markets. This outbreak illustrates how vaccination can be effective in controlling and eradicating FMD.

In 1987, the countries of South America established the Hemispheric Plan for the Eradication of Foot and Mouth Disease (PHEFA). Under this plan, comprehensive vaccination with modern, improved vaccines is the backbone of eradication efforts, but depopulation is also conducted if the disease threatens a disease-free region. The adoption of PHEFA led to a decrease in the number of FMD outbreaks reported in South America from 955 in 1990 to 130 in 1999. The adoption of PHEFA also strengthened veterinary systems overall and promoted private sector cooperation in control and eradication activities, resulting in an overall improvement in national animal health programs and services in nearly all countries.

Livestock breeding is the major agricultural activity in Uruguay, and a significant contributor to its economy. In 2001, Uruguay had 10.6 million cattle, 12.1 million sheep, 480,000 horses, and 270,000 pigs. Livestock production represents more than 65 percent of all Uruguayan exports. The presence of FMD, however, places significant restrictions on trade. In the 1990s, the European Union decided to stop general vaccination for FMD, prompting South American meat-exporting countries to discontinue vaccination if possible and acquire a more favorable trade status. In 1994, Uruguay was recognized by the OIE as “FMD free where vaccination is practiced.” In the same year, it discontinued vaccination, in the hope of obtaining the status of “FMD free without vaccination,” a goal it achieved in 1996. Also hoping to achieve the coveted “FMD free without vaccination” status, Argentina and Paraguay stopped vaccinating in 1999, as did portions of Brazil in 2000. However, Ecuador, Peru, Bolivia, Colom-

Chapter 5—Descriptions of Incursions of EADs
bia, Venezuela and parts of Brazil continued to report FMD outbreaks through the 1990s and continued to vaccinate.

As a result of discontinuing vaccination, Uruguay, Argentina, Paraguay and parts of Brazil were at great risk for FMD. Their increased susceptibility was due to the progressive loss of immunity in large cattle populations over a short period of time, the continual danger of the spread of FMD from the remaining endemic areas, and the movement of large numbers of now-susceptible young livestock to fattening areas. Because they were free of FMD, these countries devoted fewer people and resources to the eradication project. Some people believe that the decreased resources contributed to the failure of surveillance and communication systems between countries. Education and training of public and private individuals also decreased, and political and commercial interests became more important than sanitary requirements. In a few years, the entire veterinary infrastructure promoted by PHEFA was weakened, and FMD invaded the southern region of South America, including Uruguay.

The re-introduction of FMD into the region

In 2000, Argentina, Brazil and Uruguay reported outbreaks of foot and mouth disease with both the type O and the type A viruses. FMD types A and type O were reported in Argentina, where 124 premises were eventually involved. Twenty-two facilities were affected in Brazil, with 12 confirmed as type O, and three farms were infected in an adjacent part of Uruguay. All three countries conducted depopulation ("stamping-out") campaigns and, by the end of 2000, believed the viruses to be eradicated. However, in February 2001, Argentina reported the first cases of a massive FMD outbreak that would eventually affect all three countries. This virus, a type A, spread rapidly and explosively through the central and eastern part of Argentina, although a special control region prevented the epidemic from extending into the south. Despite extensive depopulation efforts, the disease had affected over 2,000 premises by the end of 2001 and was still out of control in Argentina. Brazil reported its first outbreak with this virus in May 2001, in the state of Rio Grande do Sul. The affected herd and contact animals were immediately culled, and a vaccination campaign was initiated that prevented the virus from spreading outside this state. Ultimately, Brazil would report 37 outbreaks of FMD in Rio Grande do Sul in 2001.

On April 23, 2001, FMD type A appeared in Uruguay, from Argentina. The first infected farm was reported in Palmitas, Soriano Department (state). Palmitas is approximately 70 km from Uruguay’s border with Argentina, the Uruguay River. Thirty-nine of the 430 cattle on the affected farm had signs of FMD. Lesions were not seen on the farm’s 640 sheep. The affected and exposed animals were killed the following day. On April 26, FMD was found on a neighboring farm, which had a...
mixed population of cattle, sheep and pigs. At the same time, several FMD outbreaks occurred in the adjacent Colonia Department, 25 km from the Uruguay River and 40 km from the first cases. The zone where the outbreak first occurred is economically integrated with the adjacent region of Argentina, which was experiencing FMD outbreaks, and the virus is assumed to have spread from this region via fomites or people.

Quarantines were immediately placed on both affected departments and, the following day, the remaining affected and exposed animals were destroyed and buried. In total, 5,093 cattle, 1,511 sheep, and 333 pigs were culled. Three days later, the government was forced to suspend the stamping-out procedure because of strong resistance by local farmers and the discovery that the disease had spread to other areas of the country. Authorities learned that, a few days before the first cases were recognized, cattle had been sold at auction and delivered to other parts of Uruguay. The movement of people, agricultural equipment and machinery, and milk and beef trucks are also thought to have contributed to the spread of the virus. On April 26, Uruguay began ring vaccination of cattle within a 10 km radius of the affected farms. Beginning on April 27, all movement and trade of animals were prohibited throughout the country. On April 30, vaccination was extended to form a protective barrier to prevent the virus from entering uninfected states or neighboring countries.

**The vaccination program**

On May 5, Uruguayan authorities initiated a massive vaccination program for all cattle. The Uruguayan veterinary services established a vaccination timetable, scheduling routes, dates, and times. The vaccine was provided to farmers free of charge, and the farmers were responsible for vaccinating their animals within a given time period. Animals in areas adjacent to the state of Rio Grande do Sul were vaccinated first in order to protect Brazilian livestock. Vaccination proceeded from north to south and from east to west, and was completed on June 7; movement and transit restrictions were then relaxed. In total, nearly 11 million cattle were vaccinated. Government-administered serological tests at the completion of the vaccination program suggested that compliance had been 99 percent. Uruguay’s 12 million sheep, which share pastures with the cattle, were not vaccinated; however, this did not seem to hamper the eradication of the virus. The approximately 270,000 pigs were also left unvaccinated, as the vaccine used was not thought to be effective in this species. At the height of the epidemic, 40-60 new infected farms were being found each day; however, by the end of the first round of vaccination, there were fewer than 10 new foci per day.

From June 15 to July 22, Uruguay conducted a re-vaccination program. A total of 24 million doses of FMD oil-adjuvanted vaccines were distributed during these two vaccination rounds. In November 2001, an additional 4.5
million young cattle that had been born since 2000 were vaccinated or re-vaccinated, and each animal was identified by an ear-tag tracking system. This revaccination effort boosted immunity in the cattle population to the optimum levels, and decreased the risk that vaccinated animals might become carriers. A few days after the completion of the second round of vaccination, only a few sporadic cases were being found. The last case of FMD was found on a dairy farm on August 21. By October, Uruguay was again classified as “free of FMD, with vaccination.” Re-vaccination of all cattle was carried out again in February 2002 and May 2002.

There have been some concerns about the use of vaccines in eradication efforts. Some FMD outbreaks in the past were linked to incompletely inactivated, older vaccines. Although newer vaccines use better inactivation methods, there are still fears that this could occur. It may also be possible for animals to become FMD carriers, even when vaccinated. In this South American epidemic, there were no documented cases of vaccinated animals causing new outbreaks.

**The cost of the outbreak**

From April 23 to August 21, 2001, a total of 2,057 farms or facilities in Uruguay were affected by FMD, a number similar to the farms affected by the epidemic in the U.K. However, Uruguay was able to eradicate its extensive outbreak solely by restrictions on livestock movement and the vaccination of cattle, in spite of having a large and fully susceptible sheep population in close contact with the cattle. The total direct cost of eradication was estimated at $13.6 million. Vaccine purchases accounted for $7.5 million, with the remainder used for compensation payments to farmers, cleaning and disinfection, and operating expenses. The $13.6 million does not include some expenses incurred by the Army, which collaborated by controlling illegal livestock movements in border areas and providing other support. Argentina and Brazil also managed to control their epidemics, in part by vaccination.

The loss of export markets and a pronounced decrease in livestock prices associated with the epidemic were costly for Uruguay. The estimated losses as a result of the closing of external markets to Uruguayan farmers exceeded $200 million. Financial losses to meat and dairy producers, in particular, had a significant negative impact on the national economy. In addition, movement restrictions on the entire livestock sector affected many workers and associated industries such as packing plants. Losses associated with closed packing plants, as well as the return of 380 containers of meat that were in transatlantic transit at the time of the outbreak, added approximately $30 million in costs. In total, the epidemic cost Uruguay approximately $243.6 million, a much smaller figure than the approximately $10 billion in losses to agriculture, the food chain and tourism during the outbreak in the U.K. In addition,
Uruguay was able to eradicate the extensive outbreak solely by restrictions on livestock movement and the vaccination of cattle, in spite of having a large and equally susceptible sheep population in close contact with the cattle.

approximately 6,900 animals were culled in Uruguay compared with the more than 6 million animals killed in the U.K.

Sources of Information


Correa Melo E, Saraiva V, Astudillo V. Review of the status of foot and mouth disease in countries of South America and approaches to control and eradication. Rev Sci Tech. 2002; 21(3):429-436


Monkeypox in the United States, 2003

Veterinarians work to prevent, diagnose and treat disease in a wide variety of species, but they also take on the role of gatekeeper to decrease the incidence of disease transmission between humans and animals. Veterinarians need to have a knowledge of zoonotic diseases, and should question animal owners about illness if a zoonosis is suspected in animals or people. The 2003 monkeypox outbreak demonstrates the importance of close cooperation between the medical, public health, and veterinary communities in addressing zoonotic diseases.

On June 7, 2003, public health officials from the Centers for Disease Control and Prevention (CDC) and the states of Wisconsin, Illinois, and Indiana reported the first outbreak of human monkeypox in the Western Hemisphere. Monkeypox is a rare, zoonotic viral disease that occurs primarily in the rain forest countries of Central and West Africa. The monkeypox virus is a member of the orthopox family of viruses. Other orthopoxviruses that can infect humans include variola (smallpox), vaccinia (the attenuated virus used in the smallpox vaccine), cowpox virus, buffalopox virus, and the newly described Cantagalo virus in Brazil. In humans, infection with the monkeypox virus results in a rash illness similar to, but less infectious than, smallpox. The incubation period is approximately 12 days. Monkeypox in humans is not usually fatal; depending on the outbreak, deaths typically occur in 1-10% of all cases.

Chapter 5—Descriptions of Incursions of EADs
Animal species known to be susceptible to monkeypox include non-human primates, rabbits, and some rodents.

**How did monkeypox get to the United States?**

Traceback investigations found that the source of the infection was a shipment of animals from Ghana imported into Texas on April 9. The shipment contained approximately 800 small mammals of nine different species, including six African rodents. The rodents included rope squirrels, tree squirrels, Gambian giant rats, brush-tailed porcupines, dormice, and striped mice. Some of these animals had reportedly become ill and died suddenly, soon after their arrival in the U.S. The CDC tested some of the surviving animals by PCR and virus isolation, and found that one Gambian giant rat, three dormice, and two rope squirrels were infected with the monkeypox virus.

Before the outbreak was detected, the monkeypox virus had spread into several states in these animals and in prairie dogs. Some of the imported animals were shipped from Texas to an Iowa distributor and then to a distributor in Illinois. In Illinois, the Gambian rats and dormice were kept in close proximity to prairie dogs, which became infected. The prairie dogs were then sold to other dealers and individuals in several states, including a Milwaukee animal distributor who purchased prairie dogs and a Gambian giant rat that was ill at the time. In May, some of these prairie dogs were sold to two pet shops in the Milwaukee area. Others were sold or traded during a pet “swap meet” (pets for sale or exchange) in northern Wisconsin. All of the exposed prairie dogs could not be traced during the investigation.

The first human case of monkeypox occurred in a child who had been bitten by an infected pet prairie dog. The child’s mother and father also became infected through contact with this animal. Scientists at the Marshfield Clinic in Marshfield, Wisconsin recovered the first viral isolates from one of the patients and a prairie dog. Using electron microscopy, they found a poxvirus in the skin of the human patient and the lymph node of the prairie dog. The CDC conducted further laboratory testing including PCR, serology, immunohistochemistry, and gene sequencing that confirmed these results and demonstrated that the poxvirus was the monkeypox virus. The CDC advised physicians, veterinarians, and the public to report instances of rash illness associated with exposure to prairie dogs, Gambian rats, or other animals to local and state public health authorities. In total, 37 laboratory-confirmed and 35 suspected human cases were reported in Illinois, Indiana, Kansas, Missouri, Wisconsin and Ohio. In most patients, the disease took the form of fever and vesicular skin eruptions. Two patients, both
Before the outbreak was detected, the monkeypox virus had spread into several states. All of the human patients reported direct or close contact with sick prairie dogs. Cases of monkeypox were also reported in animals. In prairie dogs, the illness included fever, cough, conjunctivitis, and lymphadenopathy, followed by a nodular rash. Some prairie dogs died and others apparently recovered. Preliminary information suggests that the Gambian giant rat under investigation experienced a much milder illness with no respiratory signs and possibly limited dermatologic involvement. Veterinarians who suspected monkeypox in an animal were asked to contact the state health department for information on specimen submission, and not to perform necropsies or biopsies because of the risk of infection. The CDC recommended that all animals with suspected monkeypox be humanely euthanized to prevent further spread of the disease and the carcass be incinerated. If the animal was associated with a human case, it was to be tested to confirm the disease. In addition, the CDC recommended that all rodents from the April 9 shipment, and any prairie dogs on the premises at the same time as these African rodents, be euthanized. Other mammals that had been in contact with these animals were placed under quarantine for 6 weeks.

On June 25, 2003, the CDC issued updated interim guidelines on the use of the smallpox vaccine, the antiviral drug cidofovir and vaccine immune globulin. The CDC recommended that people who had close or intimate contact with a confirmed case be vaccinated with the smallpox vaccine. These people could be vaccinated up to 14 days after exposure. Seven people including three veterinarians, two laboratory workers, and two health-care workers received pre-exposure prophylaxis. Another 23 people were vaccinated after exposure. The CDC also issued recommendations to medical workers on preventing transmission. Limited person-to-person transmission has been reported in monkeypox outbreaks in Africa, and health care personnel attending hospitalized patients were advised to follow standard precautions for guarding against airborne or contact illness. No cases of human-to-human transmission were confirmed in the outbreak in the U.S. Veterinarians examining or treating sick rodents, rabbits, and exotic pets like prairie dogs and Gambian rats were advised to use personal protective equipment such as gloves, surgical masks or N-95 respirators, and gowns.
Monkeypox: Report of Cases in the United States

Data reported to CDC as of July 30, 2003. This is the final report.

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On June 11, 2003, the CDC and the FDA issued a joint order announcing an immediate embargo on the importation of all rodents from Africa, due to the potential for these rodents to spread the monkeypox virus to other animal species and to humans. The joint order also banned within the U.S. any sale, offering for distribution, transport, or release into the environment, of prairie dogs and six genera of African rodents implicated in the monkeypox outbreak. On November 3, 2003, the joint order was replaced by an interim final rule in which the CDC restricts importation of these animals and the FDA restricts domestic interstate and intrastate movement, with exemption procedures to accommodate special circumstances. The last human case of monkeypox in the U.S. was acquired on June 20, 2003.

**Sources of Information**


Emerging & Exotic Diseases of Animals


**Nipah Virus in Malaysia, 1999-2000, and Bangladesh, 2001-2004**

The Nipah virus is an example of an emerging viral pathogen. This virus is a previously unknown member of the family Paramyxoviridae that has been identified primarily in humans, pigs, and fruit bats. It was first recognized during an outbreak of respiratory and neurologic disease in pigs. More than 200 people who had contact with infected pigs developed encephalitis, which was often fatal. The virus later reappeared briefly in Malaysia, and several times in Bangladesh. Two elements that can be significant in the emergence of a viral epidemic or epizootic are the agent’s pathogenicity to the host and its capacity to establish itself in new hosts. The Nipah virus, which appears to exist naturally in fruit bats, became established in pigs in Malaysia and was lethal to humans. Enormous numbers of pigs contracted the disease, which became so widespread that for public health protection, half of Malaysia’s commercial pig population had to be destroyed. In Bangladesh, where pigs are uncommon, clusters of encephalitis were seen periodically in humans between 2001 and 2004, but these outbreaks were self-limiting and unrelated to exposure to livestock.

From late 1998 through the first half of 1999, a new pig disease characterized by pronounced respiratory and neurologic signs, sometimes with sudden death in sows and boars, began to spread among pig farms in Malaysia. It was not initially identified as a new syndrome because the morbidity and mortality rates were not high and the symptoms were not markedly different from other known diseases including Japanese encephalitis, a mosquito-borne disease prevalent in most countries in Asia. But when measures to control Japanese encephalitis did not prevent an increased incidence of viral encephalitis in pig farm workers, attention was again focused on the mysterious pig disease. In March 1999, Malaysian researchers isolated an unknown virus, which was identified by the U.S. Centers for Disease Control and Prevention (CDC) as a previously unknown paramyxovirus. The virus was termed the Nipah virus and the syndrome in pigs became known as the Porcine Respiratory and Encephalitis Syndrome, Porcine Respiratory and Neurologic Syndrome, or simply Barking Pig Syndrome after the loud cough seen in infected pigs.

By the time the virus was identified, pigs on many farms in peninsular Malaysia were already showing signs of the disease. Transmission between farms was attributed to the movement of pigs, as well as the sharing of boar semen and possibly the movement of dogs and cats. The Nipah virus spread rapidly among pigs on the infected farms, probably by...
direct contact with infected pigs’ excretions and secretions such as urine, saliva, or pharyngeal and bronchial secretions. Pigs in Malaysia are typically kept in close confinement, which can encourage the spread of pathogens between animals.

**What is the Nipah virus?**

The Nipah virus is a previously unrecognized paramyxovirus that appears to be related to another emerging virus, the Hendra virus in Australia. The Nipah virus can infect pigs, humans, dogs, and goats. Antibodies to the virus have also been reported in cats and horses, and viral antigens were found in one case of meningitis in a horse. Sheep may also be affected. In pigs, the Nipah virus causes rapid and labored breathing, an explosive and non-productive cough, neurologic changes including lethargy or aggressive behavior, and sudden death. The Nipah virus spreads readily from infected swine to other species. In pigs, the virus is found in high concentrations in the epithelial cells of the airways, facilitating its airborne spread.

In Malaysia, most human cases seemed to occur after close direct contact with the excretions or secretions from an infected pig. No cases of human-to-human transmission were documented during this epidemic; however, researchers suspect that person-to-person transmission occurred during more recent outbreaks in Bangladesh. In humans, the Nipah virus localizes in the brain after it circulates in the blood. The most common signs of infection are fever, severe headache, myalgia, encephalitis, or meningitis. Approximately half of all human cases seen to date have been fatal.

**Where did the virus come from?**

Fruit bats (flying foxes) are thought to be the natural hosts for the Nipah virus. Environmental circumstances could have led to the emergence of the virus from this species into pigs. There is greater contact between humans and their domestic animals and bats as intensive farming practices encroach into previously undisturbed natural habitats. The concentration of pigs and fruit trees on the same farms can lead to increased contact between fruit bats and pigs. Biologists have also noted that flying foxes are increasingly seen in urban areas. When a virus exists in a ubiquitous wild animal reservoir, such as bats, its emergence into humans and domestic animals can be difficult to prevent. Recently, researchers have found that at least two major strains of the Nipah virus circulated in pigs during the 1998 epidemic. A strain isolated from the initial outbreak in the northern regions differs significantly from a strain isolated four months later in the south. These results suggest that the Nipah epidemic in Malaysia was not due to a single transmission of the virus from fruit bats into pigs. Instead,
it now seems that the virus may have emerged at least twice over the course of this outbreak.

Advances in microbiological techniques can also contribute to the recognition of emerging diseases. The discovery of the Nipah virus was facilitated by increased technical abilities, as well as by the discovery of a related virus, the Hendra virus, in Australia in 1994. Like the Nipah virus, the Hendra virus appears to be found in fruit bats in nature and was only discovered when it emerged into other species and caused disease.

Control measures

The Nipah virus is a biosafety level 4 agent because it causes death in people and there is no treatment or vaccine. At the time of the outbreak, there were no biosafety level 4 laboratories in Malaysia. Researchers from both the U.S. CDC and the Australian Commonwealth Scientific and Industrial Research Organization (CSIRO) helped the Malaysian government to isolate the virus, develop diagnostic tests, conduct transmission studies, and implement an eradication program.

The primary measure used to control the Nipah virus outbreak was the culling of pigs. Between the end of February and the end of April 1999, over 900,000 pigs from almost 900 farms were destroyed. The depopulation of infected pigs successfully controlled the human epidemic. The culling program was stopped after all known and suspected infected herds were destroyed. An ELISA test was developed to identify infected farms and a national swine testing and surveillance program was begun at the end of April 1999. The program required that each farm be sampled twice, with a minimum interval of three weeks between sampling. In the following three months, 889 farms were tested and 50 farms were found to be positive. The positive farms were considered infected and 172,750 pigs were destroyed. The government then developed a control program to provide continued monitoring of all pigs prior to slaughter. An educational program for farmers was also implemented.

Before the outbreak, pigs were second only to poultry in the Malaysian livestock industry. The Nipah outbreak resulted in the reduction of the Malaysian swine population from 2.4 million to 1.32 million pigs. The total number of farms decreased from 1,885 to 829. The outbreak also caused dramatic changes in the pig farming industry. In one state, Negeri Sembilan, pig farming is completely prohibited. In other areas, pig farming is now only allowed in an identified Pig Farming Area. The restocking of farms that had been depopulated is subject to government approval. After the outbreak, farmers were encouraged to undertake other agriculture and livestock activities.
Continued threats from the virus in Malaysia, Bangladesh and other countries

The presence of the Nipah virus in native wildlife populations poses a continuing threat to the pig and human populations in Malaysia. The Nipah virus may have reemerged in June and July 2000 when neutralizing antibodies were found in pigs on some farms in Peninsular Malaysia. A total of 1,700 pigs were destroyed in two farms in the state of Perak. In July 2000, IgG antibodies to the Nipah virus were also found in pigs on some farms in Sarawak. Four pig workers in Sarawak also had antibodies. Approximately 6,000 pigs were destroyed to control this outbreak.

More recently, a series of Nipah virus outbreaks unrelated to pigs have been reported in Bangladesh. In April and May 2001, several people in Chandpur village, Meherpur District developed an unknown neurologic disease with fever. The first case occurred in a 33 year old farmer who became ill on April 20 and died 6 days later. The farmer's wife, son, brother, and sister also developed the disease. In total, 13 cases and 9 deaths occurred in eight households. Japanese encephalitis, dengue fever, and malaria were ruled out. No samples were taken from the nine patients who died, but a later investigation revealed Nipah virus antibodies in the four surviving patients. From January 11 to 28, 2003, a cluster of similar cases appeared in villages in Naogaon District, approximately 150 km from the first outbreak. Twelve people in eight households were affected. Eight patients died, usually within a few days of the disease onset; no diagnostic samples were available from these cases. The four survivors of this outbreak also had Nipah virus antibodies.

No obvious zoonotic source was found in either of these two outbreaks. No clusters of illness were seen in pigs, which are uncommon in Bangladesh, or in any other species. Serum samples collected from 2 pigs and 31 bats in Meherpur were all negative for Nipah virus antibodies. In Naogaon, 50 animals including 10 birds, 4 pigs, 4 dogs, 2 shrews, 5 rodents, and 25 bats were tested by serology. Antibodies to the Nipah virus were found only in two flying foxes. Although case control studies did suggest that patients were more likely than controls to have been in contact with a sick cow, no cows were available for testing and this association may be due to chance. It also appears that the virus might have been spread, in part, by person-to-person transmission. There were several clusters of cases within households, with family members becoming ill over a short period. In addition, people in Meherpur were more likely to be infected if they lived with or cared for patients, particularly if they had contact with secretions such as saliva or urine.

From January through April 2004, two new clusters of fatal encephalitis were seen in Bangladesh. The first occurred in Manikganj, Rajbari Jaipurhat, Naogaon and Faridpur provinces from early January through Fruit bats are thought to be natural hosts for both the virus in Malaysia and the virus in Australia.
Source: Ministry of Agriculture Malaysia
February. As of February 26, 22 cases and 17 deaths were attributed to the Nipah virus, with an additional 51 cases still under investigation. Another cluster of cases was reported in April in Faridpur district. As of April 20, 30 cases and 18 deaths had been seen. In both outbreaks, CDC laboratories confirmed that the Nipah virus was the cause of disease in a number of the cases.

Health officials continue to be on the lookout for the Nipah virus or similar viruses, which could re-emerge in Bangladesh, Malaysia, or other countries. Although henipavirus (Nipah and Hendra) outbreaks have been reported only in Australia, Malaysia, and Bangladesh, their actual range could be considerably broader. Flying foxes, the natural hosts for these viruses, can be found from the east coast of Africa across south and Southeast Asia, east to the Philippines and Pacific islands, and south to Australia. Recently, the Nipah virus was isolated from Lyle’s bats in Cambodia.

Sources of Information


Originally reviewed by:

Jasbir Singh, Department of Veterinary Sciences, Malaysian government and former ISU graduate student

*Screwworm—New World

(Cochliomyia hominivorax) in the United States, 2000

This is an example of how prompt actions taken by veterinary practitioners prevented the introduction and spread of screwworm myiasis, a devastating disease. Screwworms were once endemic throughout the southern United States, but were eradicated by a program that involved the release of sterile male flies. The New World screwworm still exists in parts of the Caribbean and South America and could be re-introduced to the U.S. at any time.
On February 27, 2000, a shipment of 17 horses from Argentina arrived at a quarantine facility in Miami, Florida. Two days later, 16 of the 17 horses were released from quarantine. On March 1, the one remaining horse, a four-year-old chestnut thoroughbred gelding, was also released. The next day, a private practitioner performed a physical examination on this horse and found minor discharge from the prepuce, no swelling, and a bad odor—and, on closer examination, a number of insect larvae in the penis. The practitioner collected 50-100 larvae from the distal penis of the horse and contacted federal authorities. On March 3, a USDA APHIS foreign animal disease diagnostician (FADD) submitted samples of larvae from the horse to the USDA National Veterinary Services Laboratories (NVSL) in Ames, Iowa, and appropriately treated the horse and premises. On March 4, the NVSL confirmed that the samples from the horse were screwworm larvae in the third instar stage. The horse received a second treatment on March 6 and remained in quarantine until its wound was completely healed. It was released from quarantine on March 15, after being examined by a federal veterinarian. The other 16 horses in the February 27 shipment were traced and each horse was examined twice by a FADD, at three to five day intervals. No evidence of disease was found in any of these horses. APHIS Veterinary Services began intensive screwworm surveillance in Florida and sentinel animals were placed in the West Palm Beach area from March 10 to April 17. Screwworms were not found.

Another screwworm incident occurred in December 2000 in Dade County, Florida, in a pet cat that had traveled with a U.S. military employee from the Guantanamo Bay military base in Cuba to the U.S. In Cuba, a veterinarian had treated an abscess on the foot of the cat with ivermectin for five consecutive days before departure. Throughout the treatment, the veterinarian removed several dead larvae from the wound, which was healing over. When the owner arrived in Florida, he took the cat to a private practitioner, who removed one larva from the partially healed abscess. The practitioner shipped the larva to the NVSL where the diagnostician identified a mature Cochliomyia hominivorax larva in the third instar stage. The cat was treated and the disease did not spread.

What are screwworms?

Screwworm myiasis is a devastating parasitic disease that has long been a leading cause of livestock losses in tropical areas of the Western Hemisphere. The larvae of the New World screwworm fly, Cochliomyia hominivorax, feed on the open wounds of warm-blooded animals, including humans. Unlike ordinary maggots that subsist on debris and dead tissue, screwworm larvae attack living flesh, causing debilitation and sometimes even death. Wounds prone to screwworm infestation include those caused by feeding ticks, the bites of vampire bats, castra-
tion, dehorning, branding, shearing, wire cuts, sore mouth in sheep, and shedding of the velvet in deer. The navels of newborn mammals are also common sites of infestation.

New World screwworms were once found throughout the tropical and subtropical regions of North, Central, and South America, but have been eradicated from many countries by a series of cooperative programs involving the release of sterile male flies. This approach, conducted and sustained by the USDA APHIS, has systematically eliminated screwworms from the U.S., Mexico and most of Central America over the last five decades. In late 1998, the USDA and Panama began the final phase of the Screwworm Eradication Program in that southernmost Central American country. Because there is no screwworm control plan for South America, sterile fly releases across eastern Panama will continue to be necessary even after Panama becomes screwworm-free. The sterile flies will create and maintain a biological barrier in Panama’s Darien Gap, halting the pest’s northward migration at the Panama-Colombia border. In addition to South America, screwworm is endemic on a few islands in the Caribbean, including Hispanola, Cuba and Jamaica.

**Sources of Information**


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*defunct link as of 2004

**West Nile Virus in the United States, 1999–2005**

The outbreak of West Nile fever in New York in 1999 illustrates how a mosquito-borne disease that affects both humans and animals can spread. The West Nile virus had never before been reported in this hemisphere. The outbreak provides lessons about detecting and responding to a new disease, including the importance of local disease surveillance and response systems, communication among public health agencies, and links between public and animal health agencies. Veterinarians played an important role in the initial diagnosis of this outbreak. West Nile is now considered an endemic disease in the U.S.

In early August 1999, Tracy McNamara, DVM, head of the department of pathology at the Bronx Zoo, became concerned when she heard
that a large number of crows had been dying around the zoo. By late August, 40 crows had died. Then birds at the zoo began to die. Over the Labor Day weekend, the zoo lost a Guanay Cormorant, three Chilean flamingos, a pheasant, and a bald eagle. Because these deaths followed those of the crows, experts strongly doubted that the disease originated in the zoo. Necropsies of the birds revealed streaking in the heart and brain hemorrhages. Eastern equine encephalitis was suspected but McNamara was skeptical because the emus in her care, which are very susceptible to eastern equine encephalitis virus, were thriving. "It was becoming more and more suggestive that this was not a regular bird disease," McNamara said. When two more flamingos died on September 9, she sent samples to the USDA's National Veterinary Services Laboratories (NVSL) in Ames, Iowa. The NVSL ruled out avian influenza and Newcastle disease viruses. The Centers for Disease Control and Prevention (CDC) was also sent samples, as were doctors at an Army laboratory in Fort Detrick, Maryland.

Meanwhile, on August 23, 1999, an infectious disease physician from a hospital in northern Queens contacted the New York City Department of Health (NYCDOH) to report two patients with encephalitis. On investigation, NYCDOH initially identified a cluster of six patients with encephalitis, five of whom had profound muscle weakness. Testing of these initial cases was positive for St. Louis encephalitis virus on September 3 at the CDC. Eight of the earliest case-patients were residents of a 2 x 2-mile area in northern Queens. On the basis of these findings, aerial and ground applications of mosquito adulticides and larvacides were instituted in northern Queens and South Bronx on September 3.

**What happened next?**

In Ames, Iowa, the NVSL isolated a virus from the birds' tissues and, after ruling out several viral agents that cause encephalitis in birds, performed an electron microscopy examination. Forty nanometer virus particles with the morphology of togaviruses or flaviviruses were observed. On September 20, the NVSL forwarded the virus cultures to the CDC for identification and characterization. Testing at the CDC on September 23 indicated that the isolate was closely related to the West Nile virus (WNV), which had never been isolated in the western hemisphere. CDC experts also detected flavivirus antigens in one of the human autopsy specimens by immunohistochemistry and found a West Nile-like virus genomic sequence in a human brain specimen from an encephalitis case; this sequence was identical to that derived from the bird tissues. Concurrently, specimens of brain tissue from three human encephalitis cases, forwarded by the New York State Department of Health to the University of California, Irvine, were reported as positive for the West Nile-like virus sequence by genomic analysis.
The outbreak of West Nile fever in New York in 1999 illustrates how a mosquito-borne disease affects both humans and animals. The four deaths occurred among persons over 68 years of age. The onset dates ranged from August 5 to September 16. The median age of the patients was 71 years (range 15-87 years), with the most severe clinical cases and all fatalities occurring among older persons. In October 1999, the NVSL first isolated the West Nile virus from the brain tissue of a Long Island horse that had clinical encephalitis. WNV was also isolated at NVSL from two additional encephalitic horses in 1999 and WNV antibodies were identified in ill horses in Suffolk and Essex counties, New York. Retrospective classification of likely West Nile cases occurring prior to October resulted in a total of 25 equine cases.

**What is the West Nile virus?**

The West Nile virus is a flavivirus belonging taxonomically to the Japanese encephalitis subgroup. This subgroup also includes the St. Louis encephalitis virus, Kunjin virus, Murray Valley encephalitis virus, and others. The West Nile virus was first isolated in the West Nile province of Uganda in 1937. It is a mosquito-transmitted virus that, in endemic regions, cycles between birds and mosquitoes. Many infected birds are asymptomatic, but high mortality rates have been seen in some species—particularly crows, ravens, and jays. When environmental conditions favor high viral amplification, mosquitoes can also spread the virus to mammals. In the northern United States, the West Nile virus has been most closely associated with *Culex pipiens*, a mosquito species that breeds in standing water, especially water polluted with organic matter. It has been thought that these mosquitoes “prefer” to bite birds, but if breeding sites are available near homes and domestic animal enclosures, *Culex pipiens* may bite people and domestic animals. *Culex pipiens* is most active at dawn and dusk. Another hypothesis suggests that other species of mosquitoes, not *Culex pipiens*, act as a “bridge,” biting both birds and mammals. Some recent evidence indicates that *Culex salinarius* is responsible for WNV transmission to people. *C. salinarius* is found in fresh and saltwater marshes, lakes, ponds, and seepage areas, as well as in the many types of artificial containers found around human residences and businesses. This species is active from sunset to sunrise. Like the St. Louis encephalitis virus, the West Nile virus is not transmitted from person to person or from birds to people.

Among mammals, symptomatic infections mainly seem to occur in humans and horses. In humans, many cases of West Nile fever are mild and flu-like; however, in more severe cases, there may be signs of encephalitis, meningoencephalitis or meningitis. Horses develop symptoms of encephalitis, often without a fever. The first recorded epidemics of West Nile fever occurred in Israel during 1950-1954 and in 1957. The largest recorded epidemic occurred in South Africa in 1974. Epidemics were also...
reported in Europe in the Rhone delta of France in 1962 and in Romania in 1996. The West Nile virus had never been recognized in the United States or any other area of the Western Hemisphere prior to 1999.

**The response to the outbreak**

Vector control measures had been initiated in northern Queens and the South Bronx on September 3. These measures were followed by a citywide pesticide application, after a laboratory confirmed a case of West Nile encephalitis in a Brooklyn resident with no travel history to Queens and two additional cases in the South Bronx. Surveillance of wild birds and sentinel chickens was instituted to assess WNV distribution in the region. Emergency telephone hotlines were established in New York City on September 3 and in Westchester County on September 21 to address public inquiries about the encephalitis outbreak and pesticide application. Approximately 300,000 cans of DEET-based mosquito repellent were distributed citywide through local firehouses, and 750,000 public health leaflets were distributed with information about personal protection against mosquito bites. Recurring public messages were announced on radio, television, web sites, and in newspapers, urging personal protection against mosquito bites. Recommended actions included limiting outdoor activity during the peak hours of mosquito activity, wearing long-sleeved shirts and long pants, using DEET-based insect repellents, and eliminating any potential mosquito breeding niches. Spraying schedules were also publicized and people were advised to remain indoors during spraying to reduce pesticide exposure.

By the end of 1999, the West Nile virus had been identified in a limited area of the northeastern United States in wild birds, mosquitoes, humans, and horses. Naturally occurring virus had been found in birds and mosquitoes in parts of Connecticut, New York, New Jersey, and in one county in Maryland. Clinical illness in humans and horses occurred during a period from early August through late October and was limited to New York. WNV activity ended for the season because of various factors, including climate and vector control activities. In all, 62 human cases, with seven deaths, were recognized in 1999. Twenty-five cases of West Nile encephalitis were also identified in horses, all in Suffolk and Nassau Counties on Long Island, New York. Because horses are known not to play a role in the transmission of WNV, quarantines were never
placed on any non-clinically ill horses in the outbreak area. However, the movement of horses was restricted, particularly the export of horses from affected areas to the European Union and the shipment of any horses to the E.U. via Kennedy airport.

In genetic sequencing studies, the West Nile virus isolates from the New York outbreak showed strong similarities to isolates from Israel, suggesting that this region may have been the origin of the virus. How the West Nile virus was introduced into the United States is unknown, but speculation has centered on infected humans, mosquitoes, or birds being transported by aircraft. Several other speculated routes of entry also exist.

**The continuing spread of the West Nile virus**

In 2000, 21 human cases of West Nile encephalitis were reported. Two elderly patients, an 82-year-old man in New Jersey and an 87-year-old woman in New York, died of the disease. Sixty confirmed equine cases were confirmed in seven states; 37 horses survived and 23 (38%) died or were euthanized. Six wild mammals were classified as WNV-positive and 4,323 infected birds were documented in 12 states plus the District of Columbia. A total of 143 counties in 12 eastern states and the District of Columbia had confirmed findings of WNV in a mosquito, bird, or mammal.

In 2001, the virus spread through bird migration south to Florida, west to Iowa, and north to Canada. There were 66 human cases in 10 states, including nine deaths. As before, most of the cases occurred in older patients; the median age was 68 and the range was 9 to 90 years. A total of 733 equine cases were reported from 127 counties in 19 states, a 12-fold increase from 2000. More than 7,000 WNV-positive wild birds were found in 328 counties in 27 states and the District of Columbia. In 66 percent of the counties, dead crows were the first indication of West Nile virus activity. Positive birds were collected from April to December 2001. West Nile virus did not affect any commercial poultry.

From 2002 to 2005, the virus continued to spread across North and South America. By the end of the 2002 mosquito season, the West Nile virus was found throughout the Midwest and was spreading into the western states. In 2003, this virus was first reported from some countries in South America. By 2005, it was found in all U.S. states except Alaska and Hawaii. The first West Nile virus vaccine for horses was licensed in 2002 and a second vaccine was licensed in 2003. As a result, the number of West Nile virus cases in horses decreased from more than 15,000 in 2002, to approximately 1400 in 2004. Reported human infections also declined from over 9800 cases and 264 deaths in 2003, to approximately 2500 cases and 100 deaths in 2004. As of Sept 13, 1299 cases and 29 fatalities had been reported in 2005. The reasons for the declining case rate in humans
are unknown but may include weather patterns, mosquito control programs, public education, or other factors.

**Sources of Information**


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