Overview of High Consequence Livestock Pathogens

For veterinarians

The USDA has identified some pathogens as being of high consequence and concern to livestock. This slide depicts the full list of diseases, and there is a handout of this list available for audiences if you choose to use it. The Center for Food Security and Public Health has designed specific PowerPoint presentations on many of these diseases that you will find on the Bioterrorism or the Agroterrorism CD-ROM.

Disease Awareness

- Veterinarians recognize animal diseases at the local level
- Prepare by knowing
  - Typical signs of diseases
  - Basic disease etiology
  - Economic and trade impact
  - How to report suspected cases
- Disseminate knowledge

Historically, infectious disease outbreaks are first recognized at the local level. As veterinarians, we should prepare ourselves by knowing: the clinical signs of high consequence livestock pathogens, the basics about the disease etiology, the economic and trade impact the diseases could have, and who to call if a disease is suspected. We should also be prepared to provide education to those most affected by the outbreak. This presentation will provide a brief disease overview of the High Consequence Livestock Pathogens.

Overview

- Importance of agriculture and livestock to U.S. economy
- High consequence livestock pathogens
  - CDC’s Category A, B, C Bioterrorism Agent List
  - Additional diseases
- Veterinarian’s responsibilities

In this presentation, we will address the importance of livestock and agriculture to the U.S. economy and cover the diseases on the USDA High Consequence Livestock Pathogen list some of which are also on the CDC’s Category A, B, and C Bioterrorism Agent List. Those diseases that overlap with the CDC’s list will discuss the agent, its use as a bioweapon, and what the U.S. response will be to its potential introduction. For each additional disease, we will discuss its etiology, geographic distribution, and review the impact the specific disease has had, or could have, on the U.S. economy. We will also discuss the response to the disease (i.e. treatment, vaccinations, and control). Finally, we will discuss the veterinarian’s role in preventing the spread of a high consequence livestock pathogen.

Importance of Agriculture & Livestock

This next section demonstrates how important agriculture and livestock are to our society and economy.
In 2003, the U.S. exported $56.2 billion in agricultural commodities; $12.2 billion came from animals and animal products. Some of the diseases on the USDA High Consequence Pathogens list are reportable to the OIE and could halt trade if an American animal is diagnosed with the disease. If we cannot export, our products lose value which has a negative effect on the economy, livestock and grain producers, and the unemployment rate could increase. (USDA Outlook Report, Nov 25, 2003 http://usda.mannlib.cornell.edu/reports/erssor/trade/aes-bb/2003/aes40.pdf)

From cattle to turkeys, livestock and poultry generate needed income for producers and the economy while supplying safe product to our tables. Listed is the economic importance of livestock and poultry in our nation and estimated numbers as of 2003. These are live animal values and do not consider the value of the products we harvest from these species. Interesting to note that in 2002, the U.S. was second only to China for egg production, and produced more eggs than the whole of the European Union combined.

Some of the agents discussed in this presentation are zoonotic. In some diseases, clinical signs may manifest in animals prior to humans. Pets can act as important sentinels because they are present in large numbers and often live in close contact with humans. It is estimated that pets are present in 59% of U.S. households. Livestock are also present in high numbers in certain areas of the country. Wildlife also play an important role in our communities because they could be important sources of infection for humans and animals, and could potentially contaminate large areas. Knowing what agents are zoonotic and protecting yourselves and your clients is essential in the event of a disease outbreak.

It is also important for us as veterinarians to set a good example for producers by following strict biosecurity regulations ourselves. We must be sure to disinfect our clothing, boots, and equipment between farms. We should avoid driving our vehicle through potentially contaminated materials. We must follow recommendations put forth by species-specific associations (for example, the American Association of Swine Veterinarians) regarding farm-to-farm biosecurity.

In this section, we first discuss how the CDC Category ABC disease/agent list was established and then overview the diseases.
In 1999 Congress requested that the national public health capabilities for response to acts of biological terrorism be upgraded. The CDC was designated as the lead agency for overall public health planning. In order to focus their preparedness efforts, the CDC needed to select and prioritize biological agents based on the threat they posed to public health. A group of national experts including infectious disease specialists, Department of Health and Human Services personnel, civilian and military intelligence experts, and law enforcement officials gathered to establish the list. The general criteria used for selection and prioritization were: 1) the public health impact based on illness and death; 2) the delivery potential to large populations based on stability and ability to mass produce and distribute a virulent agent; 3) potential for person to person transmission; 4) the public perception as related to public fear and potential civil disruption; 5) the special public health preparedness needs, stockpiles required, surveillance and diagnostic needs. Special attention was given to those agents that had previously been used or researched as a bioweapon. Based on these criteria, agents were scored and divided into A, B and C Categories. This is not a federally legislated list and is subject to change based on review of agents. Using this standardized system allows the CDC to add or remove agents. As veterinarians it is important to be aware of these agents and review these diseases as they relate to veterinary patients.

The agents/diseases in Category A are Anthrax, Botulism, and Tularemia (three bacteria).

For each Category ABC disease we discuss, we will briefly review the agent, highlighting transmission and clinical signs in humans and animals, discuss the agent as a bioweapon, and then how we can prevent and control disease. However, before discussing the diseases, it is important to understand weaponization of an agent. If an agent has been weaponized, characteristics of the pathogen may have been altered to make it a more effective weapon. For example, the transmission of a pathogen may be enhanced or the virulence increased; the organism may have been altered to make it resistant to antibiotics it would otherwise be susceptible to; weaponization of an organism may allow it to evade the normal protective immunity induced by vaccine, or it may even alter the clinical signs. It is difficult to know. However, reviewing the agents and what we currently know about them is still important for our enhanced awareness of these agents.

The disease coverage is brief. If you would like more information on a disease, refer to the fact sheet or to the disease specific presentation.
The agents/diseases in Category A are Anthrax, Botulism, and Tularemia (three bacteria).

Anthrax results from infection by Bacillus anthracis, a spore forming, Gram positive aerobic rod. Anthrax can be found as a spore in the soil worldwide; it is particularly common in parts of Africa, Asia and the Middle East. In the United States, foci of infection occur in South Dakota, Nebraska, Mississippi, Arkansas, Texas, Louisiana and California, with smaller areas in other states. Spores can remain viable for decades in the soil or animal products such as dried or processed hides and wool. Spores can also survive for 2 years in water, 10 years in milk, and up to 71 years on silk threads. However, the vegetative organisms are thought to be destroyed within a few days during the decomposition of unopened carcasses (exposure to oxygen induces spore formation). There are three forms of the disease in humans. 1) Cutaneous anthrax which develops after skin infections. This form is characterized by a papular skin lesion, which becomes surrounded by a ring of fluid-filled vesicles (as shown in picture). Most lesions (malignant carbuncle) are non-painful and resolve spontaneously, but disseminated, fatal infections occur in approximately 20% of cases. 2) Intestinal anthrax develops after eating contaminated meat. The initial symptoms may be mild malaise and gastrointestinal symptoms. Severe symptoms can develop and rapidly progress to shock, coma and death. 3) Pulmonary anthrax occurs after inhaling spores in contaminated dust. Natural infections are mainly seen among workers who handle infected hides, wool, and furs (wool sorter’s disease). Symptoms may include fever, tiredness, and malaise; a nonproductive cough and mild chest pain may be present. Then follows an acute onset of severe respiratory distress with fatal septicemia and shock within one to two days. Fatalities may be prevented if treated early, however, when symptoms are flu-like and non-specific, early treatment is not sought. In animals, sheep, cattle, and horses are very susceptible, while dogs, rats, and chickens are resistant to disease. In ruminants sudden death may be the only sign. However, the disease may manifest as flu-like symptoms; chronic infections often have edema. Photo: 7 mo. old child who visited a network news office in Oct. 2001 with his parent. Child developed cutaneous anthrax.

In the 1950’s and 1960’s, B. anthracis was part of the U.S. bioweapons research program. In 1979, there was an accidental release of aerosol anthrax from a military compound in the Soviet Union. The neighboring residents experienced high fevers, difficulty breathing, and a large number died. Fatality estimates ranged from 200-1,000. In 1992, Russian President Boris Yeltsin finally acknowledged that the release occurred from a large scale military research facility. In 1991, Iraq admitted it had done research on B. anthracis as a bioweapon. There are several characteristics of B. anthracis make it attractive as a bioweapon. It is widely available and relatively easy to produce. The spores are infective, resistant, and remain infective when aerosolized. The lethal dose for inhalation of spores is low and mortality is high; the case-fatality rate for inhalational anthrax could approach 100%. Untreated pulmonary and intestinal infections are almost always fatal, especially if recognized too late for effective treatment. Person-to-person transmission of anthrax is very rare and has been reported only in cases of cutaneous anthrax. Photo courtesy of D. Bickett-Weddle, DVM, ISU.
Anthrax: The Response

- **Vaccine**
  - Humans
  - Animals

- **Antibiotics**
  - Treatment
  - Prophylaxis

- **Disinfection**
  - Sporicidal agents, sterilization

Vaccines are available for humans who have a high risk of infection. Efficacy of the vaccine against inhalation of *B. anthracis* is unknown, and reactogenicity of the vaccine is mild to moderate. Vaccine is available for livestock. Natural strains of *B. anthracis* are usually susceptible to a variety of antibiotics, but effective treatment depends on early recognition of the symptoms. Treatment for cutaneous anthrax is usually effective, but pulmonary and intestinal forms are difficult to recognize and mortality rates are much higher. Prophylactic antibiotics are appropriate for all exposed humans. Anthrax spores are resistant to heat, sunlight, drying, and many disinfectants, but are susceptible to sporicidal agents or sterilization.

Botulism: The Agent

- **Clostridium botulinum** – Gram positive, spore-forming bacteria
- 7 different neurotoxins
  - Types A-G
- Clinical signs
  - Flaccid paralysis
  - Pigs, dogs, and cats fairly resistant

Botulism, or “limber neck” in waterfowl, is caused by toxins produced by *Clostridium botulinum*. It is a Gram positive, spore-forming, toxin-producing obligate anaerobic bacillus. The spores are ubiquitous in soil. Botulism was first discovered by a German physician, Justinius Kerner in 1793. He called the substance “wurstgift” and found it in spoiled sausages. During this period of time, sausage was made by filling a pig’s stomach with meat and blood, boiling it in water then storing it at room temperature, which were ideal conditions for clostridial spores to survive. Botulism gets it name from “botulus” which is Latin for sausage. United States federal regulations for food preservation resulted following several outbreaks of botulism in the U.S. Botulism spores germinate and release 7 different antigenic types of neurotoxins, classified as A through G. Different neurotoxin types affect different species. Only a few nanograms of the toxin can cause severe illness and all cause flaccid paralysis. Neurologic clinical signs, including generalized weakness, dizziness, dysphagia, and flaccid paralysis, are similar in all species affected. In humans, gastrointestinal symptoms may proceed the neurologic symptoms because the preformed toxin is ingested. In animals, many species of mammals and birds can be affected. Clinical disease is most often in wildfowl, poultry, mink, cattle, sheep and horses. Ruminants and horses will often drool while humans experience dry mouth. Paralysis of the respiratory muscles leading to death may occur in 24 hours in severe cases. Waterfowl are especially sensitive and pigs, dogs, and cats are fairly resistant. Canadian cooperative wildlife health centre http://wildlife.usask.ca/bookhtml/botulism/botulismc.htm

Botulism: The Bioweapon

- Used by Aum Shinrikyo cult in Japan
- Aerosolized
- Easy to produce and transport
- Potent and lethal
- Most poisonous substance known

Botulinum toxins are known to have been weaponized by several countries and terrorist groups in the past. It was part of the U.S. bioweapons program, Iraq has produced large volumes of this toxin, and the Aum Shinrikyo cult in Japan tried to use it unsuccessfully in 1990. The botulinum toxins are relatively easy to produce and transport. Botulinum toxin is extremely potent and lethal and is the single most poisonous substance known. Signs of a deliberate release of the toxin, either via aerosol, food, or water, is expected to cause clinical illness similar to foodborne illness. Additionally, uncommon toxin types, such as C, D, F, or G, may be the culprits and thus raise suspicion of an intentional release. The photo depicts a young child who had contracted botulism through a natural source. Note the limp appearance of the neck and arms. 75% of natural botulism cases occur in children under 1 year of age. California Department of Health Services http://www.dhs.ca.gov/dcd/InfantBot/toxfig2.htm
Botulism: The Response

- Toxoids for high risk people
- Antitoxin available
- Case-by-case basis
- Botulinum toxins are easily inactivated with many disinfectants and heat

In endemic areas, toxoids are typically used in horses, cattle, sheep, and goats, and investigational toxoids for high risk laboratory workers are available. However, these toxoids are not effective for post-exposure prophylaxis. Botulinum antitoxin (trivalent) is sometimes used in animals but response depends on the type of toxin causing the disease and the species of animal. In humans, if given early, the antitoxin may decrease the severity of disease and shorten the duration of symptoms. It has severe side effects and is only used on a case-by-case basis. The U.S. Army has an investigational heptavalent antitoxin. Antibiotics may be warranted if a wound is involved but immediate intensive care may be the only treatment. Botulinum toxins can be inactivated by sunlight in 1 to 3 hours as well as bleach, sodium hydroxide, or chlorinated water. The spores are very resistant in the environment but moist heat (120°C for at least 15 min) will destroy them. Israel Veterinary Medical Association http://www.isrvma.org/article/56_3_4.htm

Tularemia: The Agent

- Francisella tularensis
- Transmitted by ingestion, inhalation, vectors, direct contact through skin
- Six clinical forms in humans

Tularemia, or “rabbit fever”, is caused by Francisella tularensis, a Gram negative bacteria. The disease can be transmitted by ingestion of infected, undercooked meat (rabbit); bites from infected ticks, and less commonly deerflies; through direct contact with blood or tissues of infected animals (especially rabbits); and inhalation of contaminated dust. Initial symptoms are flu-like and they include fever, chills, headache, and myalgia. In humans there are six clinical forms of tularemia – glandular and ulceroglandular are the most common presentation of this disease. An ulcer may or may not be present at site of infection and local lymph nodes are enlarged. Oculoglandular occurs when conjunctiva become infected by rubbing eyes with contaminated fingers or by splashing contaminated materials in the eyes. The oropharyngeal presentation is caused by ingestion of organism in contaminated food (undercooked meat) or water. Typhoidal and pneumonic forms usually occur following inhalation, or hematogenous spread of the organism. Both of these forms tend to present as atypical pneumonia and most fatalities occur with these forms and can be as high as 30-60% if untreated. This photo is of the Dermacentor variabilis (American dog tick) which is an effective transmitter of tularemia. Image from: Iowa State University-Entomology Dept Image Gallery http://www.ent.iastate.edu/imagegal/ticks/aamer/aamerfanddvarf.html; Girl with ulcerating lymphadenitis due to tularemia, Kosovo, April 2000 Image from CDC website: http://www.cdc.gov/ncidod/eid/vol8no1/01-0131.htm; Ulcer caused by tularemia. Image from: CDC Photo Image Library (http://phil.cdc.gov/Phil/results.asp?page=1)

In animals the full spectrum of clinical signs is not known. Sheep, young pigs, horses, dogs, and cats are susceptible to tularemia. Signs of septicemia such as fever, lethargy, anorexia, and coughing are most commonly seen. In wildlife, clinical disease is not often seen and animals are found dead or moribund. However, when infected hares and cottontails are observed, they behave strangely in that they are easily captured because they run slowly, rub their noses and feet on the ground, experience muscle twitches, are anorectic, have diarrhea, and are dyspnic. These lagomorphs are an important reservoir for human infection. Older swine and bovine seem to be resistant to disease and are asymptomatic.
Tularemia: The Bioweapon

- Stable
- Aerosolized
- Low infective dose via inhalation
- Case fatality: 30-60% (untreated)
- WHO estimation: 1970
  - 50 kg agent: City population 5 million
    - 250,000 ill
    - 19,000 deaths

In the 1950-60’s, the United States military developed weapons which aerosolized *F. tularensis*, and it is suspected that other countries may have included this organism in their bioweapons research program as well. There are many characteristics that make *F. tularensis* a good agent for bioterrorism. It is stable, survives in mud, water, and dead animals for long periods of time, and has previously been stabilized as a bioweapon. Only a low dose is needed to cause inhalational disease. Case fatality rates of the typhoidal and pneumonic forms are reported to be 30-60% if untreated. In 1969, the World Health Organization (WHO) estimated that if 50kg of virulent *F. tularensis* particles were aerosolized over a city with 5 million people, the result would be 250,000 illnesses and 19,000 deaths. Recently, the CDC estimated the economic losses associated with an outbreak of tularemia to be $5.4 billion for every 100,000 people exposed.

Tularemia: The Response

- Person-to-person transmission not documented
- Antibiotics effective, if early or prophylactic
- Vaccine
  - For high risk individuals
  - Unknown efficacy against inhalational tularemia

Person-to-person transmission has not been documented with a tularemia infection, so secondary spread is of little concern. However, infectious organisms can be found in blood and other tissues so care must be taken when handling infected material. Antibiotics are generally effective if given early in the infectious process and as a prophylaxis. There is a live attenuated vaccine, given intradermally by scarification, that is available to individuals at high risk for exposure to the bacteria. The vaccines efficacy against high dose respiratory challenge is unknown. Disinfection of the bacteria is easily accomplished with many common disinfectants. However, the bacteria is stable at freezing temperatures for months to years. Image from: CDC PHIL: (http://phil.cdc.gov/phil/detail.asp?id=979)

CDC Category B

- Brucellosis
- Q Fever
- Glanders
- Toxins
- Melioidosis
- Viral Encephalitis

The diseases we just reviewed were the Category A agents, i.e. those that are given highest priority by the CDC. This next group is the Category B agents/diseases and have been given second priority by the CDC. The first three in this group are bacteria, the a rickettsial organism (Q Fever), then some select toxins, followed by one group of viruses (the viral encephalitides focusing on VEE).

Brucellosis: The Agent

- Gram-negative bacteria
- Ingestion, inhalation, or direct contact
- Clinical signs
  - Humans: cyclic fever and flu-like symptoms
  - Animals: reproductive signs

Brucellosis, or undulant fever, is caused by various species of *Brucella*, a Gram-negative, facultative intracellular rod. The organism can persist in the environment and indefinitely if frozen in aborted fetuses or placentas. Transmission occurs via ingestion of infected food or consuming infected unpasturized milk or dairy products, via inhalation of infectious aerosols (a means of infection in abattoirs), or through contact with infected tissues through a break in the skin or mucous membranes. Brucellosis can involve any organ or organ system and have a very insidious onset with varying clinical signs. The one common sign in all patients is an intermittent/irregular fever with variable duration, thus the term undulant fever. There are 3 forms of the disease in humans. In the acute form (<8 weeks from illness onset), symptomatic, nonspecific, and flu-like symptoms occur. The undulant form (< 1 yr. from illness onset and symptoms) include undulant fevers, and arthritis. In chronic form (>1 yr. from onset), symptoms may include chronic fatigue-like syndrome, and depressive episodes. Illness in people can be very protracted and painful and can result in an inability to work and loss of income. In animals, the clinical signs are mainly reproductive in nature, such as abortions, epididymitis, orchitis, and also fistulous withers in horses. Photo courtesy of D. Bickett-Weddle, DVM, ISU.
This table illustrates the many species of *Brucella* and their distinct natural hosts. However, many are also human pathogens with *B. melitensis* being the most pathogenic.

<table>
<thead>
<tr>
<th>Species</th>
<th>Natural Host</th>
<th>Human Pathogen</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>B. abortus</em></td>
<td>Cattle, bison, elk, horses</td>
<td>Yes</td>
</tr>
<tr>
<td><em>B. melitensis</em></td>
<td>Goats, sheep, cattle</td>
<td>Yes</td>
</tr>
<tr>
<td><em>B. suis</em></td>
<td>Swine, hares, reindeer, caribou, rodents</td>
<td>Yes</td>
</tr>
<tr>
<td><em>B. canis</em></td>
<td>Dogs, other canids</td>
<td>Yes</td>
</tr>
<tr>
<td><em>B. ovis</em></td>
<td>Sheep</td>
<td>No</td>
</tr>
</tbody>
</table>

In the 1950’s when the U.S. bioweapons research program was active, *Brucella suis* was the first agent weaponized. The World Health Organization prepared a bioterrorism scenario looking at aerosolized *B. melitensis* (which has more serious consequences for humans than *B. suis*) spread along a line with the prevailing winds with optimal meteorologic conditions. It was assumed that the infectious dose to infect 50 (ID₅₀) percent of the population would require inhalation of 1,000 vegetative cells. The case fatality rate was estimated to be 0.5% with 50% of the people being hospitalized and staying an average of seven days. It is highly infective and fairly stable in this form. Incubation period in humans is one week up to several months, which often complicates the diagnosis due to the latency of clinical signs. Person-to-person transmission is very rare.

Prolonged antibiotics are necessary to penetrate these facultative intracellular pathogens. Combination therapy has shown the best efficacy for treatment in humans. Vaccinating calves has helped eliminate infection in these animals, thus decreasing possible exposure to humans. Strict adherence to federal laws of identifying, segregating and/or culling infected animals is essential to success. Properly protect yourself to prevent exposure to tissues and body secretions of infected animals by wearing gloves, masks, goggles, and coveralls. Pasteurization or boiling milk and avoid eating unpasteurized dairy products will help decrease human exposure to brucellosis. The organism is susceptible to many disinfectants. Photo courtesy of D. Bickett- Weddle, DVM, ISU.

There are several common names associated with glanders and they include Equinia, Farcy and Malleus. Glanders is caused by a Gram negative bacteria, *Burkholderia mallei* (formerly *Pseudomonas mallei*). It is closely related to the next bacteria we will overview – *Burkholderia pseudomallei* that causes Meloidiosis (which we will review next). *B. mallei* is transmitted by ingestion or inhalation of infected tissues or fluids, and also through contact with broken skin or mucous membranes. Horses, mules and donkeys are the major host of this organism. Cats can be infected and may be particularly susceptible. Dogs, goats and camels can also be infected, but ruminants appear to be resistant. The clinical disease in horses and humans is similar. Transmission from animal to human appears to be inefficient. Infection by contact leads to ulceration of the skin, mucous membranes and soft tissues, as pictured on the slide. Infection by inhalation leads to acute glanders that results in pulmonary abscesses and nasal ulcers. Chronic glanders affects the joints and muscles forming ulcerated and purulent lesions. The photo is of a donkey with a ulcerative lesion on his lip. [www.vet.uga.edu/vpp/gray_book/Images/056.htm](http://www.vet.uga.edu/vpp/gray_book/Images/056.htm)
Glanders: The Bioweapon

- History
  - WWI Russian horses
  - WWII Chinese civilians, horses, POW's
- Easy to produce
- Aerosolized, highly infectious
- Mortality high in chronic form
  - 50-70%
- Person to person transmission: Rare

During World War I, glanders was believed to have been spread deliberately to infect large numbers of Russian horses and mules on the Eastern Front. This had an effect on troop and supply convoys, as well as on artillery movement, which were dependent on horses and mules. Human cases in Russia increased with these infections during and after WWI. During World War II the Japanese deliberately infected horses, civilians, and prisoners of war with *B. mallei* at the Pinfang (China) Institute. In 1943-44 the U.S. studied this agent as a possible biological weapon but did not weaponize it. After World War II the former Soviet Union is believed to have evaluated *B. mallei* as a potential bioweapon agent. In a single year in the 1980s, the Soviet Union produced more than 2,000 tons of dry agent for glanders. *B. mallei* can be aerosolized and infection via this route is almost always fatal if untreated. Even with treatment, the chronic form of the disease can develop and kill 50-70% of those infected despite hospitalization. Cases of human-to-human transmission have been reported, but are rare.

Glanders: The Response

- No vaccine
- Antibiotic therapy likely effective
- Destroyed by various chemicals

Currently, there is no available vaccine for humans or animals against glanders. *Burkholderia mallei* is usually sensitive to a variety of antibiotics but caution should be used in animals as it promotes the carrier state. The organism can be destroyed easily.

Melioidosis: The Agent

- *Burkholderia pseudomallei*:
  - Gram-negative
- Transmission: Contact, ingestion, inhalation
- Clinical signs: Humans, sheep, goats, and pigs
  - Asymptomatic to pneumonia, lung and wound abscesses

Melioidosis, a disease of rice farmers in Thailand, is caused by *Burkholderia pseudomallei*, an aerobic, Gram-negative motile bacillus found in certain soils and water. Disease is primarily located in Southeast Asia but isolated cases have occurred in Hawaii and Georgia. Transmission can occur when open skin wounds come in contact with contaminated soil or water, and also by ingestion of contaminated water. The most common route is inhalation of dust from contaminated soil. Most cases of melioidosis are usually asymptomatic but clinical cases commonly present as a pulmonary infection. This is demonstrated by a high fever and pneumonia with caseous lesions. In wound infections, focal melioidosis occurs with skin abscess formation. Infection can spread to other systems and infrequently CNS infection can occur. The animals most severely affected are sheep, goats and pigs and they present with pneumonia with caseous abscesses in the lungs. These animals may have nasal discharge or encephalitis. Additionally, joints can be affected and cause lameness. Thailand Rice Farmer Photo http://www.escati.com/photos/characters/rice_farmer.jpg

Melioidosis: The Bioweapon

- Easy to produce
- Available
- Aerosolization
- High mortality: 90%
- Person-to-person (rare)
- Animal-to-person (rare)

*Burkholderia pseudomallei* was studied by the U.S. as a bioweapon but it was never weaponized. There are reports that the former Soviet Union bioweapons program also researched this bacteria. The organism can be aerosolized and it is readily available in soil and water in southeast Asia and Iran. In natural infections, the mortality rate is usually less than 10%, but it is thought that bioweaponization would result in septicemia or severe pulmonary infections with mortality rates reaching 90% despite treatment. Person-to-person and animal-to-person transmission is rare but can occur via blood or contaminated body fluids such as urine, milk and nasal secretions.
Melioidosis: The Response

- Long-term, multiple antibiotics effective
- Vaccines available: not in U.S.
- Easily destroyed by disinfectants

B. pseudomallei is susceptible to various antibiotics, but relapses can occur once treatment is stopped. Long-term treatment may be necessary and multiple drugs may be needed. Vaccines are available in some countries, but not the U.S., and they are not effective against large challenge doses. In endemic areas, avoid contact with soil and water during the wet season. The organism can be destroyed by numerous disinfectants.

Toxins: The Agents

- Staphylococcal enterotoxin B (SEB)
- Ricin toxin from the castor plant
- Clostridium perfringens epsilon toxin

Next we will discuss three toxins that satisfy the criteria for Category B agents. They are Staphylococcal enterotoxin B, ricin toxin from the castor plant, and Clostridium perfringens epsilon toxin. Toxins are biological agents produced by living organisms such as bacteria, plants, or animals. Photo of soldiers eating-USARIEM http://www.usariem.army.mil/nbd/nutri.htm

SEB: The Agent

- Staphylococcal enterotoxin B (SEB)
- A common cause of food poisoning
- Clinical signs: Humans
  - Fever, chills, headache, myalgia
  - Non-productive cough if inhaled
  - GI signs if swallowed
- Animals: Likely similar to human

Bacterial toxins are more difficult to produce than plant toxins but are much more toxic. SEB produced by Staphylococcus aureus is an example of a bacterial toxin. SEB is one of the toxins responsible for staphylococcal food poisoning in humans. It is a superantigen and acts by stimulating cytokine release and an overwhelming immune response. Routes of exposure could include inhalation or ingestion. In humans, the clinical signs resulting from SEB exposure via ingestion would occur in approximately 4-10 hours. Following inhalation exposure, clinical signs would occur in approximately 3-12 hours. The general symptoms include sudden onset of high fever persisting for several days, chills, and myalgia. Following inhalation, a non-productive cough, chest pain, and dyspnea can occur. Following ingestion, nausea, vomiting, and diarrhea predominate. In animals, although S. aureus is associated with many diseases, there is little information about clinical signs resulting from SEB. Experimental exposure of mice and monkeys to aerosolized SEB resulted in diarrhea and vomiting, followed by depression, dyspnea, and shock; some animals died.

Ricin: The Agent

- Ricin toxin from bean of castor plant
- Available worldwide
- Clinical signs: Acute onset of fever, chest tightness, cough, dyspnea, nausea

Plant toxins are easy to produce at a low cost because raw materials are available worldwide such as the ricin toxin from the bean of the castor plant. Annually, of one million tons of castor beans are processed worldwide in the production of castor oil. The waste product is five percent ricin by weight. The toxin is stable and extremely toxic by several routes of exposure, including respiratory. Many species are affected and have an acute onset of fever, chest tightness, cough, dyspnea, and nausea. Castor plant - Cooperative extension, Purdue University http://www.vet.purdue.edu/depts/addl/toxic/plant11.htm
**Viral Encephalitis: The Agent**

- The Alphaviruses: EEE, WEE, and VEE
- Transmitted via mosquito
- Clinical signs
  - Humans, horses, donkeys, mules: Often asymptomatic to flu-like
  - Encephalitis in small proportions
- Birds are asymptomatic carriers, act as sentinels

**Epsilon Toxin: The Agent**

- *Clostridium perfringens* type B and D
- Increases intestinal and vascular permeability, liver and neurological damage
- Clinical signs
  - Calves: Diarrhea, abdominal pain, listlessness, neurologic
  - Sheep, goats: Watery to bloody diarrhea, neurologic
  - Humans: Little information

**Toxins: The Bioweapon**

- **History**
  - Aerosolized: SEB, ricin
  - Available worldwide
  - Easy to produce, stable
  - Many species affected
  - No person-to-person transmission

**Toxins: The Response**

- Supportive care
- No vaccines currently available for SEB or ricin
- Vaccines for animals for clostridial disease
- Toxins are inactivated with common disinfectants

Management includes supportive care. Currently there are no effective antitoxins or human vaccines available. Development, safety, and immunogenicity testing for a human vaccine is anticipated for the near future. Preliminary animal studies for a SEB vaccine have been encouraging. There is promising research in animals with vaccines for ricin and antiserum production. Currently there are animal vaccines available for clostridial disease, but none have been developed for humans. These toxins can be inactivated with common disinfectants and if exposure is suspected, decontamination of the area or exposed skin should be done with soap and water or with bleach.

**Toxins have been researched and used as bioweapons.** For example, the U.S. stockpiled and researched SEB during its bioweapons program before it was terminated in 1969. Ricin is thought to have been used in the assassination of Bulgarian defector Georgi Markov in London in 1978. It is said that a ricin pellet was injected into Markov by a specially engineered weapon disguised as an umbrella. Markov died three days after the incident. In Minnesota during 1991, four members of the Patriots Council, an antigovernment extremist group, were arrested for plotting to kill a US Marshall with ricin. The group planned to mix the homemade ricin with DMSO and then smear it on the door handles of the Marshall’s car. The plan was discovered and all four men were arrested. There are reports of ricin use in the Iran-Iraq war in the 1980’s and also reports that it was found in Al Qaeda caves in Afghanistan in 2002. In 2003 equipment for producing ricin along with traces of the toxin were found in a London apartment. Although aerosolized SEB would not likely produce significant mortality, it could render 80% or more of exposed people clinically ill and unable to perform their duties for 1-2 weeks. The demand on the medical and logistical systems could be overwhelming. If inhaled, ricin toxin may cause pathologic changes within 8 hours and severe respiratory symptoms and failure in 36-72 hours. All of these toxins are available worldwide, relatively easy to produce, stable, and affect many species. There is no concern about transmission of the toxin from an affected individual.

*C. perfringens* types B and D produce epsilon toxin. *C. perfringens* type B infection causes severe enteritis in young calves, foals, piglets, and lambs (lamb dysentery) and *C. perfringens* type D causes enterotoxemia (overeating disease, pulpy kidney disease) in sheep and goats, and rarely cattle. The clinical signs of enterotoxemia are primarily a result of epsilon toxin, which causes increased intestinal and vascular permeability, and liver damage. In calves, clinical signs include diarrhea, abdominal pain and listlessness. Sheep and goats have watery to bloody diarrhea. Neurologic signs can be common in all. There is little or no information about the effects of epsilon toxin on humans. Human disease is usually caused by *C. perfringens* type A and C.
Viral Encephalitis: The Bioweapon
- Easy to produce
- Aerosolization
- High rate of infection
- Person-to-person transmission possible

Viral Encephalitis: The Bioweapon
- VEE was tested in the U.S. bioweapons program in the 1950s and 1960s. It is thought that other countries have also weaponized VEE. All U.S. stocks of VEE were destroyed, along with the other agents that were part of the program. VEE can be produced in large amounts by unsophisticated and inexpensive systems. The virus can be aerosolized or spread by releasing infected mosquitoes. Humans are highly susceptible and approximately 90-100% of exposed individuals could become infected and have clinical signs, although most are mild. Equids would also be susceptible and disease would occur simultaneously with human disease. There is a low overall human case-fatality rate.

Viral Encephalitis: The Response
- Supportive care
- Vaccine
  - Equine
  - Human: High risk

Antibiotics are not effective for treatment and there are no effective antiviral drugs available. Treatment involves supportive care. There is a trivalent formalin inactivated vaccine available for horses for WEE, EEE, VEE in the United States, but the human vaccines are limited to those who are researchers and at a high risk of exposure. All of the virus types are unstable in the environment. Photo depicts a sentinel chicken flock used to monitor the presence of WEE and SLE (courtesy of D. Bickett-Weddle, DVM, ISU).

CDC Category C
Nipah Virus: The Agent
- Paramyxovirus
- Fruit bat reservoir
- Clinical signs
  - Humans: Encephalitis
  - Pigs: Respiratory, neurological
  - Dogs and cats: “Distemper”

Nipah virus was discovered Paramyxovirus in Malaysia in 1999, and causes a severe respiratory disease in pigs and severe encephalitis in humans. The reservoir for the virus is thought to be fruit bats, which are called flying foxes. Suspected transmission of the virus occurs from bats roosting in fruit trees close to pig confinements. The virus then spreads rapidly through the swine herd by direct contact or aerosolization (usually coughing). It can then be passed to humans, dogs, cats and other species. Transmission can also occur from direct contact with infected body fluids. To date, no person-to-person or bat-to-person transmission has been reported. In humans, the incubation period is 3-14 days. Initial symptoms include fever, headache, dizziness, drowsiness, disorientation and vomiting. Some cases show signs of respiratory illness. In severe cases, a rapidly progressive encephalitis can occur with a mortality rate of 40%. In swine, Nipah virus is highly contagious and easily spread. Many pigs are asymptomatic. Clinical signs include acute fever (>104 °F), tachypnea and dyspnea with open mouth breathing, and a loud, explosive barking cough may also be noted. Occasionally, neurological signs can occur. Clinical signs in pigs were noted 1-2 weeks before illness in humans making swine a sentinel for human disease. Disease in other animal species is poorly documented. Other species demonstrate respiratory and neurological signs. Photo of a Malayan flying fox.
Nipah virus is described as an emerging pathogen with potentially high morbidity and mortality as well as a major health impact. Currently, transmission of the disease involves close contact with pigs but aerosolization may be a possible bioterrorist method of dispersal. The potential for this virus to infect a wide range of hosts and produce significant mortality in humans makes this virus a public health concern. Photo from Dr. James Roth-ISU of hog confinement barns that were affected during the Nipah virus outbreak in Malaysia, 1999.

Nipah virus is a very dangerous pathogen and is classified as a Biolevel 4 agent. If you suspect an outbreak, contact your state veterinarian and state public health veterinarian IMMEDIATELY! Avoid all contact with potentially infected species (pigs, dogs, cats) until the proper authorities are consulted. Nipah virus can be readily inactivated by detergents. Routine cleaning and disinfection with sodium hypochlorite or several commercially available detergents is expected to be effective.

The next two diseases that will be covered include Rift Valley Fever and Hendra. These agents/diseases are not part of the CDC Category ABC list, but we at the Center for Food Security and Public Health have included them because they are important zoonotic diseases.

Rift Valley Fever (RVF) is an RNA virus caused by a Phlebovirus in the family Bunyaviridae. Rift Valley fever is a disease that is endemic throughout most of Africa. It can be transmitted by mosquitoes, inhalation of virus, or direct contact with the virus in infected body fluids and aborted fetuses. Mosquito eggs can be infected transovarially and lay dormant for many years in the dry soil of grassland areas. Following heavy rainfalls, the eggs hatch and these newly infected mosquitoes seek a feed source (human or animal). Once a ruminant or human is infected, they serve as an amplifying host with a viremia that infects other mosquitoes. Typically humans are asymptomatic or have self-limiting flu-like symptoms. In less than 1% of humans infected, severe disease can occur resulting in retinitis, hemorrhagic fever or encephalitis. Progression to shock, coma, and death occurs in about 50% of these patients. In sheep, cattle and goats, RVF causes a very high rate of abortion and death in neonates. Clinical signs most commonly seen include fever, mucopurulent nasal discharge and possibly vomiting. Mortality in adult animals, especially those that have aborted, can be 20-30%. Photo depicts a newborn lamb and a ewe with a retained placenta.
Rift Valley Fever: The Bioweapon

- WHO estimate: 1970
  - 50 kg of virus aerosolized
  - 35,000 incapacitated
  - 400 deaths (1% mortality)
- Stable at most temperatures
- Inactivated by various chemicals

The WHO prepared an estimate of casualties if RVF virus was aerosolized. The estimate suggests that if 50 kg of the agent were disseminated from an airplane, it would have a 1 km downwind reach with 35,000 humans incapacitated and 400 deaths (1% mortality). The virus is very stable and inactivated by various chemicals.

Rift Valley Fever: The Response

- Vaccinate ruminants in endemic areas
- Control mosquitoes
- Avoid contact with infected tissues & blood
  - Wear protective clothing
- No person-to-person transmission

Immunization of sheep, goats and cattle in endemic areas is the most effective method of controlling the disease. The current vaccine can be abortigenic and teratogenic but is usually less harmful than the effect of the disease. Current research is being conducted to develop a safer vaccine. Vaccines for humans are not commercially available. Avoid and control mosquito vectors and wear personal protective clothing when handling infected tissues. If RVF is suspected, the state or federal veterinarian should be contacted immediately and movement of animals should be restricted. To date, no person-to-person transmission has been documented. Photo depicts protective gloves and mask.

Hendra Virus: The Agent

- Newly discovered
- Australia
- Fruit bats
- Transmission: Urine, body fluids
- Humans
  - Flu-like illness, respiratory failure
- Horses, cats
  - Acute respiratory signs, nasal discharge, fever, encephalitis, sudden death

Hendra virus is one of three new Paramyxoviruses (Australian bat lyssavirus, Hendra virus and Nipah virus) recently discovered. It was first identified in Australia in 1994; twenty-one horses were affected with severe respiratory illness, of which 14 died or were euthanized. Three humans were also affected, two of which died. The reservoir for the virus has been found to be fruit bats (flying foxes). To date, natural infections have only been documented in horses and humans. Experimental infections have been reported in cats, horses and guinea pigs. Hendra virus does not appear to be highly contagious, but can be spread during close contact. Infected cats can transmit the infection to horses through their urine. Additionally, horses can be infected by eating feed contaminated with the virus. Infected animals can spread the virus to humans, but the method of transmission is unknown. It is thought to be through contact with body fluids (urine, blood, oral cavity) of the infected animal. Aerosol transmission appears to be inefficient. No person-to-person transmission has been reported to date. The incubation period is 6-18 days and initial symptoms in humans resemble viral flu-like signs. This rapidly progresses to respiratory failure or encephalitis, followed by death. In horses and experimentally infected cats, signs include acute respiratory dyspnea, nasal discharge (clear to serosanguinous), anorexia, depression and fever (up to 105.8 °F). Most horses become ataxic and head pressing may be occasionally seen. This is followed by sudden death 1-3 days after the onset of clinical signs.

Hendra Virus: The Response

- Little is known about disease
- Highest level of security to work with the agent
- Potentially serious consequences
  - High mortality rate
  - Lack of treatment

Currently, little is known about Hendra virus. Hendra virus is considered a biolevel 4 agent (highest–level security). Since there were 2 human deaths out of 3 human cases, mortality may be high in the event of an outbreak or attack. Currently there is no known treatment, although ribavirin may be useful.
As you have learned throughout this presentation, agriculture in the U.S. is driven by our ability to export. Changes in the disease status of one or more food animal species will compromise our ability to export with devastating results. Livestock, poultry, and wildlife can be affected by many devastating organisms and the impact will depend on the pathogen, dissemination and number of infected animals, rapidity of detection and response to control, trade restrictions enacted, and other factors. This next section will address additional USDA High Consequence Livestock Pathogens.

African horse sickness (AHS) is a serious viral infection that affects members of the Equidae family. The disease is spread by arthropod vectors (primarily Culicoides species—biting midges), with mortality in horses as high as 95%. Currently the disease is endemic in the central tropical region of Africa (sub-Saharan central and east Africa) but potential arthropod vectors for the disease exist in the United States. Since the disease has never occurred in the Western Hemisphere, our Equidae species are naive and highly susceptible to the virus. AHS is considered one of the most lethal horse diseases and is caused by a double-stranded RNA virus (Family Reoviridae: Genus Orbivirus). Outbreaks have been seen in the Near and Middle East (1959-63), Spain (1966, 1987-90) and Portugal (1989). The peak incidence of disease occurs in the late summer and early autumn. Its prevalence being directly influenced by climatic conditions which favor insect breeding.

The incubation period for AHS can range from 2-14 days. Clinical signs typically appear 5 to 7 days after infection. They are characterized by damage to the respiratory and circulatory system as a result of increased vascular permeability. There are nine serotypes of this RNA virus, which are viscerotropic (having a predilection for the abdominal and thoracic viscera). There are four different forms of the disease: the pulmonary or purpuric form where mortality can reach 100%, the cardiac or subacute edematous form where mortality ranges from 50-70%, the mixed or acute form (involving symptoms from the pulmonary and cardiac forms of AHS), and horsesickness fever. This last form is rarely fatal and has mild symptoms that wax and wane throughout the day. The horse in this photo is suffering from the pulmonary...
AHS: Impact & Response

- 1989: Portugal outbreak
  - Eradication cost $1.9 million
- U.S. Horse Industry
  - Inventory: 5.25 million horses
  - Value of sales: $1.75 billion
- Vaccine available in endemic areas
- No natural human infection
- Vector control imperative to disease control

Following a 1989 outbreak in Portugal on 104 farms, 206 equines died (14%) or were destroyed (16%) and an estimated 170,000 equines were vaccinated. One year after the eradication program Portugal was declared free of AHS, costing an estimated US $1.9 million. As of 1998, the U.S. equine industry had an inventory of 5.25 million horses and the value of sales was $1.75 billion (USDA, National Agricultural Statistics Service). In 1995, approximately seven million Americans were involved in the equine industry (i.e., horse owners, service providers, employees or volunteers). A vaccine is available in endemic areas only and horses must be permanently identified as vaccinated. There is no evidence of humans acquiring AHS naturally, but are cautioned when handling MLV vaccines. Epizootics of AHS outside the enzootic sub-Saharan zone have not been maintained for more than 2-3 consecutive years. Factors such as the absence of a long-term vertebrate reservoir, reduced prevalence and seasonal incidence of vectors and efficient control measures (vaccination and vector abatement) may play a role in preventing the disease from becoming endemic in these areas.

African swine fever (ASF) is a febrile, contagious, systemic viral disease affecting domestic and wild pigs, including the warthog, bush pig, and giant forest hog in Africa, and the feral pig in Europe. The primary method of spread of ASF between countries has been through the feeding of uncooked garbage containing ASFV-infected pork scraps. Once a pig becomes infected, ASFV spreads by direct contact (oronasal) with infected animals, contaminated people, equipment, vehicles, and feed. ASFV can be found in all tissues and body fluids, with particularly high levels in blood and some pigs may become carriers. ASFV also replicates in Ornithodoros sp. (soft ticks) and can be spread when the tick feeds on a pig. ASF is endemic in most of sub-Saharan Africa, with the highest area of incidence seen from the Equator to northern South Africa. This figure shows the current/recent distribution of countries affected by ASF since 1995; the most recently infected countries are colored red, the countries in which ASF is endemic are shown in white, and countries that have eradicated ASF but are still considered high risk are colored yellow. Photo courtesy of www.fao.org.

The incubation period is 48 to 72 hours following intranasal-oral exposure. ASF can be a peracute, acute, subacute, or chronic disease. Virulence of isolates varies from high (100% mortality in 7-10 days after exposure) to low virulence (only seroconversion occurs). Chronic infection can result in multi-focal erythema on the ears, abdomen, and inner thighs. The foci may become raised and necrotic. A low fever may be present. Other possible signs seen include pneumonia, painless swelling of the carpal and/or tarsal joints. Pigs may show emaciation and stunting and death can occur from these sequelae. More virulent isolates of ASFV cause a high fever, moderate anorexia, recumbancy, and erythema that is most apparent in white pigs (shown above). Cyanotic skin blotching on the ears, tail, lower legs, or hams may also develop. Diarrhea and abortions are sometimes seen, but most pigs infected with ASFV remain in good condition. With highly virulent isolates, progressive anorexia and
depression develop and are usually followed by death within 10 days.

In domestic pigs, morbidity approaches 100% in herds in which the pigs have contact with each other and have not been previously exposed to the virus. Mortality varies with the virulence of the isolate, and can range from 0%-100%. Factors that can increase mortality from virulent isolates include concurrent disease, a young age, and pregnancy. A confirmed case of ASF would lead to a ban on the export and import of pigs to and from many different countries, with obvious economic impact. For successful eradication to occur, isolation and slaughter are required because no treatment or vaccine currently exists for this disease. Fortunately, humans are not susceptible to ASF.

The Akabane ("A-ka-baine") virus is an arbovirus (transmitted by arthropods) in the family Bunyaviridae, genus Orthobunyavirus. The single-stranded RNA virus causes intrauterine infection of the fetus in pregnant cattle, sheep and goats resulting in abortions, stillbirths, premature births and deformities of the fetus or newborn while the dam remains asymptomatic during pregnancy. The vector for Akabane has not been proven, but the virus has been isolated from a number of mosquito species (Aedes, Culex, Anopheles) as well as Culicoides species (biting midges). Akabane is not transmitted by direct contact, infected tissues, exudates, body fluids or fomites. Ruminants do not appear to become long-term carriers of this virus. Akabane is common in the tropics and subtropics between latitude 35°N and 35°S (indicated by the red shaded box). It is endemic to northern Australia and occasional outbreaks have occurred in southern Australia, Asia, the Middle East and South Africa when conditions for virus transmission are favorable. Incidence for the disease and the potential for epizootics is correlated with climatic factors, a distinct seasonal pattern, the geographic distribution of competent vectors and the availability of susceptible ruminant populations.

Viremia usually occurs 1 to 6 days after Akabane infection and lasts for 1 to 9 days. Only during this limited time are viral titers sufficient for potential vectors. Adult ruminants with Akabane infection are typically asymptomatic and long term carriers are not thought to occur. Manifestation of the disease is not typically noticed until pregnant ruminants abort, have stillbirths, premature births or dystocia. Congenital abnormalities of the fetus or newborns are a hallmark of this disease and effect varies depending on the stage of gestation. When infected late in the first trimester, neonates are usually born bright and alert but are unable to stand, are ataxic, and may have one or more paralyzed limbs. Newborn animals who were infected during the second trimester have arthrogryposis at birth; most cannot stand. When infected late in pregnancy, neonates can usually stand and walk, but have behavioral abnormalities and skull deformities can be common. Most affected neonates die or must be euthanized soon after birth. Animals with mild symptoms may gradually become more mobile, but most die by 6 months. While thought to be a ruminant disease, antibodies to the virus have been found in horses, buffalo, deer, camels, dogs, monkeys and most recently in pigs.
In 2002, the calf-crop for the U.S. was reported at an estimated 38.2 million head while the lamb-crop was 4.36 million head. [No specific data on goats or kids in the U.S. could be found]. To date, Akabane has not occurred in the U.S making our livestock population highly susceptible to viral infection. Based on the severe mortality that can occur with Akabane disease in naïve populations, the economic impact of the disease can be great. Additionally, the potential vectors for the disease are found in the U.S. In endemic areas, most animals are immune to Akabane virus by the time they reach sexual maturity. There is no effective treatment for Akabane virus. Most affected neonates die or must be euthanized soon after being born. Subsequent pregnancies of the infected dam will not be affected. Human infections by Akabane virus have not been reported. Prevention of Akabane disease includes vector control measures (mosquitoes and midges) and vaccination. An inactivated and an attenuated vaccine have been developed and used in Japan. The vaccines must be used prior to exposure to the infected vectors. A vaccine is not currently available in the U.S.

Avian influenza affects domestic and wild birds (chickens, turkeys, pheasants, quail, duck, geese, guinea fowl) and results from infection by type A influenza viruses of the family Orthomyxoviridae. Influenza type B viruses also exist, but not in avian species. Numerous avian influenza viruses exist, but only viruses that meet specific virulence requirements in the laboratory are designated highly pathogenic avian influenza (HPAI). Two surface antigens, hemagglutinin (H) and neuroaminidase (N) are used to classify the viruses into serotypes. Most of the isolates in recent outbreaks have been H5 or H7 viruses. HPAI causes decreased egg production, depression, and often sudden death in affected birds. Migratory waterfowl are considered to be the reservoirs of avian influenza virus shedding it in their feces and respiratory secretions. Avian influenza virus can spread by aerosols and contaminated drinking water. Once a flock is infected, it should be considered a potential source of virus for life. Highly pathogenic AI viruses can be found worldwide. Recent outbreaks have occurred in the Netherlands, Australia, England, Ireland, Scotland, Pakistan, South Africa, Mexico, and the United States.
HPAI: The Disease

- Incubation period: 3-14 days
- Birds
  - Sudden death
  - Egg production drops
  - Neurological signs
- Humans
  - Conjunctivitis and respiratory signs
  - Death possible

Incubation period is from 3-14 days and is dependent on the dose of virus, the route of exposure, the species exposed. Some birds are found dead prior to observance of any clinical signs. There may be neurological signs and reduction in normal vocalizations. Depression is common as is a precipitous drop in egg production. Respiratory signs are less prominent but can include rales, sneezing and coughing. In mature chickens, the combs and wattles are often swollen and may be cyanotic. Conjunctivitis, edema of the head and neck, coughing, sneezing and nasal discharge may also be seen. Egg production in hens stops; the last eggs laid often have no shells. Death is common, but severely affected hens occasionally recover. The human risk for infection with AI exists but is very low because strains vary in their ability to transmit and infect. An outbreak in 1997 in Hong Kong resulted in hospitalization of 18 people and 6 deaths (due to severe bilateral pneumonia). An outbreak of AI (H7N7) in 2003 in the Netherlands resulted in 83 confirmed cases of avian influenza in humans. The most common clinical signs included conjunctivitis and/or mild influenza or respiratory signs. A 57 year old veterinarian who visited a poultry farm died from acute respiratory distress syndrome. Generally. In the 1997 outbreak in Hong Kong infection was widespread in people with high occupational risk, such as poultry workers. Most of the human infections in the 2003 outbreak resulted from non-compliance with personal bio-safety measures such as wearing gloves, gowns and masks. There was also evidence of transmission from poultry workers to family members.

HPAI: Impact and Response

- Direct losses
  - Depopulation and disposal
  - High morbidity and mortality
  - Quarantine and surveillance
  - Indemnities
- 2003: European outbreak (H7N7)
  - 30 million birds destroyed
  - Estimated at $338 million USD
- 2004: Asian outbreak (H5N1)

Economic losses from avian influenza vary depending on the strain of virus, species of bird infected, number of farms involved, control methods used and the speed of implementation of control or eradication strategies. Direct losses include depopulation and disposal costs, high morbidity and mortality losses (often 100%), quarantine and surveillance costs and indemnities paid for elimination of birds. The 2003 European outbreak of (H7N7) strain has resulted in the destruction of 30 million birds, the cost as of July 2003, is unknown. Since mid-December 2003, a growing number of Asian countries are experiencing an outbreak of HP AI (H5N1 subtype). The rapid spread with outbreaks occurring at the same time in several countries, is historically unprecedented and of great concern for human health as well as for agriculture. As of February 4, 2004, avian disease has been confirmed among poultry in Cambodia, China, Hong Kong, Indonesia, Japan, Laos, South Korea, Thailand, and Vietnam. Human cases and deaths have occurred in Vietnam and Thailand.

HPAI: Impact and Response

- Treatment
  - Poultry: none
  - Humans: antivirals
- Control outbreak through depopulation/disinfection
- Prompt response to MP AI outbreak
- Vaccine
  - Poultry: Expensive, no cross protection
  - Human: No cross protection

No practical, specific treatment exists for avian influenza virus infections in commercial poultry. Supportive care and antibiotic treatment have been used to reduce the effects of concurrent bacterial infections. Antivirals have been licensed for use in humans to treat influenza since 1966 and can be effective in reducing the severity of influenza Type A in humans. To control an outbreak of HPAI the birds must be destroyed, buried or burned, and the premises must be thoroughly cleaned and disinfected. One critical goal of prevention and control is the education of the poultry industry regarding how the virus is introduced, spread and how it can be prevented. HPAI can emerge from mildly pathogenic (MP) AI outbreaks so prompt response to MP AI outbreaks is important. Inactivated influenza virus vaccines, although fairly expensive, have been used and are effective in reducing mortality and preventing disease. The concern with vaccination is that there is no cross protection between the 15 known HA subtypes. An inactivated H5 vaccine and a recombinant vaccine are licensed in the United States for emergency use in future HP AI eradication efforts. The yearly influenza vaccine available for humans is serotype specific and not likely cross protective to the avian strain.
Bluetongue is a non-contagious, insect-borne viral disease of ruminants. Bluetongue virus (BTV) belongs to the family Reoviridae, genus Orbivirus. Bluetongue primarily affects sheep and wild ruminants, with asymptomatic infections occurring in cattle, goats, deer and carnivores. There are 24 serotypes identified worldwide, five of which have been isolated in the U.S. BTV is transmitted by biting midges in the genus Culicoides (shown above). Ticks or sheep keds can be mechanical vectors, but are of minor importance. BTV was first described in South Africa, and the virus has since been recognized in Africa, Europe, the Middle East, the South Pacific, North and South America, and parts of Asia. The distribution of the vector limits the spread of infection (southern and western US). From 1997 to 2002 there was a progressive spread of bluetongue within the Mediterranean region, as shown in red in the map above (www.fas.org).

**Bluetongue:**
- Viral disease
- Ruminants: Primarily sheep
- 24 serotypes worldwide
  - 5 isolated in the U.S.
- Vector-borne
  - Culicoides (biting midge)
- Worldwide distribution
  - Mediterranean outbreak, 1997-2002

The incubation period for bluetongue is 5-20 days. In sheep, the clinical signs may include excessive salivation (top photo), facial swelling, and discharge from the nose. The tongue is occasionally cyanotic (“blue-tongue”) (pictured), swollen and protrudes from the mouth. Erosions and ulcerations are often found in the mouth. Pregnant ewes infected during the first trimester may reabsorb the fetus, abort, or give birth to “dummy” lambs. The coronary bands on the hooves are often hyperemic, inflamed and the hooves painful; lameness is common and animals may slough their hooves if they are driven. In sheep, the severity of disease varies with the breed of sheep, strain of virus, and environmental stress. Morbidity can be up to 100% and mortality is usually 0-50%. Infections in cattle and goats are usually subclinical, and rarely, have mild hyperemia, vesicles or ulcers in the mouth, erosion and crusting around the nose, hyperemia around the coronary band. In pronghorn antelope and whitetail deer, the most common symptoms are hemorrhages and sudden death; morbidity rates as high as 100% and mortality usually reaching 80-90% in these two species.

Although BTV primarily affects sheep, it has a great economic impact on the cattle industry because cattle can become temporary asymptomatic carriers. This costs U.S. sheep and cattle producers $125 million per year in lost trade and in testing to certify that animals or animal products for export are free of bluetongue virus. There is no specific or efficient treatment that can be given for an acute case of bluetongue, only supportive therapy. Animals infected with bluetongue should be protected from the elements (e.g., out of the wind or sun), kept warm and dry, given fluids and electrolyte solutions if needed and antibiotics to prevent a secondary infection. Treatment procedures may also include vector control by insecticide, which will reduce transmission of the virus to non-infected animals. Control strategies for bluetongue include quarantine and movement controls, insect control, or slaughter depending upon the situation. Vaccines are available, but are serotype specific. There are also adverse effects to the use of vaccines: fetal malformations and the possibility that the vaccine strain may recombine with field strains to produce new strains of virus. Bluetongue is not a significant threat to human health. However, one human infection has been documented in a laboratory worker and reasonable precautions should be taken while working with the virus. BTV is not fatal in
Bovine spongiform encephalopathy (BSE) is thought to be caused by prions (short for proteinaceous infectious particles). These abnormal proteins cause a progressively fatal neurologic disease in cattle and in humans. The human disease is known as variant Creutzfeldt-Jakob disease (vCJD) and is thought to result after consuming BSE contaminated beef. The first cases of BSE appeared in the U.K. in 1986 and is thought to have occurred from feeding meat or bone meal from scrapie-infected sheep to cattle, or from spontaneous genetic mutation in a cow then fed to other cows. This map depicts the countries that have reported BSE in indigenous animals (red) and imported animals (yellow). The indigenous countries pictured include Austria, Belgium, Canada, Czech Republic, Denmark, Finland, France, Germany, Greece, Ireland, Israel, Italy, Japan, Lichtenstein, Luxembourg, Netherlands, Poland, Portugal, Slovakia, Slovenia, Spain, Switzerland, and the United Kingdom. Accessed at the OIE website on Jan 26, 2004 www.oie.int/Cartes/BSE/a_Monde_BSE

The incubation period for BSE in cattle is 2 to 8 years. The clinical signs are mainly neurological, such as apprehension, fear, being easily startled or depressed. During the final stages of disease, infected animal generally shows increased excitability, hypermetria, ataxia, muscle fasciculations, tremors and myoclonus. During the end phase of the disease most animals have decreased rumination, loss of body weight and condition despite a good appetite, bradycardia, and an altered heart rhythm. In humans with variant CreutzfeldtJakob Disease (vCJD), the incubation period for vCJD is unknown, but it is likely to be many years or decades. Clinical signs include depression and schizophrenia leading to ataxia and involuntary muscle movement. In contrast to classic CJD, the variant form (vCJD) in the UK predominantly affects young people with 28 years as the mean age at death. The mean duration of infection is 14.1 months for vCJD. Photo depicts a cow in the end stages of BSE struggling to rise. She has lost quite a bit of body condition. http://exn.ca/news/Images/19970428-cow.jpg

The United Kingdom has experienced the worst outbreaks of BSE, with the peak occurring in 1993. In April of 2000, their government estimated the crisis would cost £ 3.7 billion by the end of the 2001/2002 financial year. The economic impact of the 2003 Canadian case is still being realized, and the impact of the imported U.S. case is very preliminary at this writing. The trade implications following a BSE positive case are huge given the risk for human disease. Currently no effective treatment is available. The CDC has an active surveillance program in the U.S. for cases of vCJD and the USDA FSIS has been testing cattle older than 30 months of age at slaughter since 1990. Additionally, the Red Cross has restricted blood donors from the U.K. or persons who have lived for more than 6 months in an European country known to have BSE. Various restrictions on imports, animal feeding, animals accepted at slaughter, and mammalian products have been put in place to further protect the American public. Destruction of prions is extremely difficult since they are very resistant to heat, normal sterilization processes, and disinfectants.
Classical Swine Fever

- Viral infection
- Pigs and wild boars
  - Highly contagious reservoir
- Transmission
  - Oral (contaminated garbage), direct contact, aerosol, vertical, insects, fomites
- Worldwide distribution

Classical swine fever virus (CSFV) is an RNA virus in the family Flaviviridae, genus Pestivirus and it causes a highly contagious disease of swine that occurs in acute, subacute, chronic, or persistent form. While there are minor antigenic variants of CSFV, there is only one known serotype. The natural hosts of CSFV are the pig and the wild boar. Classical swine fever is often spread by the feeding of uncooked contaminated garbage (virus transmission is mainly oral). Blood, secretions and tissues contain infectious virus. Aerosol spread can sometimes be seen in confined spaces; however, the virus does not travel long distances in the air. Carrier sows may give birth to persistently infected pigs, and mechanical spread by fomites and insects can occur. CSF is distributed worldwide, with higher prevalence in East and Southeast Asia, the Indian subcontinent, China, and South and Central America. Most of Western Europe is free of classical swine fever, but foci of infection remain in Germany and some countries of Eastern Europe. The map above shows the areas in which CSF occurred in 2001 (red: East and Southeast Asia, India, China, South and Central America), where CSF has been eradicated (green: Canada, the U.S., Australia, New Zealand, Western Europe). Source: OIE.

Humans are not susceptible to CSF infection.

CSF: The Disease

- Incubation period: 2-14 days
- Variable clinical signs
  - Acute to asymptomatic
    - Fever, weakness, anorexia, purplish discoloration of ears/thighs
    - Chronic infection fatal
  - Strain of virus
  - Susceptibility of pigs
  - Indistinguishable from ASF

The incubation period ranges from 2 to 14 days. The clinical signs of CSF vary with the strain of the virus and the susceptibility of the pigs. More virulent strains cause acute disease, while less virulent strains can result in a high percentage of chronic, mild, or asymptomatic infections. In acute infections, common clinical signs include a high fever, dullness, weakness, drowsiness, tendency to huddle, anorexia, and constipation followed by diarrhea. Several days after the first symptoms appear, the abdomen, inner thighs and ears may develop a purplish discoloration. Convulsions may be seen in the terminal stages, and recovery is rare. Chronic disease symptoms include fever, anorexia, stunted growth, and alopecia; these symptoms may wax and wane for months. Chronic infections are almost always fatal. Reproductive symptoms may also be seen with any level of virulence. Clinical signs of CSF are clinically indistinguishable from those of African swine fever.

CSF: Impact and Response

- Mortality approaches 100% in acute/chronic infections
- Ban on import/export of pigs/products
  - Huge economic impact
- No treatment
- Control through quarantine, slaughter
- Vaccine in endemic countries
- Humans not susceptible to disease

Both morbidity and mortality are high in acute infections of classical swine fever. The mortality rate in acute cases can reach 90%, and most chronic infections are fatal also. A confirmed case of CSF would lead to a ban on the export and import of pigs and pork to and from many different countries, with a huge economic impact. For successful eradication to occur, isolation and slaughter are required because no treatment currently exists. CSFV is quite stable in a protein-rich environment, and is capable of surviving for months in refrigerated meat and for years in frozen meat and for as long as two weeks in contaminated pens or on fomites. Vaccines are available in endemic countries. While vaccination can protect animals from clinical disease, it does not eliminate infections and therefore may be inappropriate in countries with an eradication policy. In countries free of CSF, periodic serologic sampling is necessary to confirm freedom from infection. Fortunately, humans are not susceptible to CSF.
Coccidioidomycosis results from direct inhalation of the spores of the dimorphic fungus *Coccidioides immitis* from the soil or from dust in the air. *C. immitis* has both a saprophytic and parasitic phase in its life cycle. It grows as a mold in the soil, and when disturbed, the hyphae fragment forms a durable structure called arthroconidia (photo) which becomes airborne. When the arthroconidia are inhaled, they transform into thick-walled multinucleate spherules that then separate to produce thousands of endospores, which can then produce a new spherule and begin the cycle anew. Coccidioidomycosis occurs in dogs, cats, horses and humans. Pigs and ruminants tend to have asymptomatic infection.

*C. immitis* is endemic in the southwestern U.S., including parts of New Mexico, Texas (west of El Paso), the central valley of California, and Arizona, where the incidence in humans is particularly high. The endemic area extends into northern Mexico, and foci of infection are present in parts of Central America and Argentina. In endemic areas, 10-50% of the human population skin-test positive for coccidioidomycosis infection. Photo from Rippon JW. Medical Mycology, 3rd ed. WB Saunders, 1988, p436 – shaded areas indicate prevalence areas for Coccidioidomycosis.

The incubation period for coccidioidomycosis is 1 to 3 weeks. In animals, disease varies from asymptomatic to disseminated and fatal. Clinical signs vary depending on the severity of infection and the species affected. Dogs and humans are the most severely affected, but cats and horses also suffer disease. Signs of the primary form of coccidioidomycosis include fever (104-105°F), lethargy, loss of appetite, and a dry, harsh cough. The cough is pronounced and can be mistaken for kennel cough in dogs. While coccidioidomycosis is primarily a respiratory disease, the infection can disseminate to many tissues. Infection of the bones is most common, resulting in lameness. Other signs of disseminate infection include joint swelling, anorexia, chronic coughing, skin abscesses and draining skin lesions, fever, and intermittent diarrhea. Humans display many of the same signs but the pulmonary lesions should be distinguished from tuberculosis or other granulomatous infections, especially in HIV positive patients. Sixty percent of *Coccidioides immitis* infections are asymptomatic and are recognized by a positive skin test. The remaining 40% of cases can range from mild to severe symptoms and are usually pulmonary. About 90% of these cases will resolve their pulmonary infection without sequelae. Coccidioidomycosis is a reportable disease in humans in some endemic areas, such as California, New Mexico and Arizona. The state department of health should be notified if there is a human case in these areas.
Coccidioidomycosis can be a devastating disease, in terms of both direct medical costs, and time of productivity lost from work while the patient is ill. The outbreak of *C. immitis* infections in Kern County (located in the San Joaquin Valley of California) in the 1990’s cost more than $66 million dollars in medical care alone. Coccidioidomycosis also has an effect on the livestock industry and measures should be taken to decrease the animals’ exposure to dust. Should infection occur, it is usually widespread. The cost of diagnosis and treatment of afflicted animals can be significant. Coccidioidomycosis is not communicable from one person to another, or from animal to man.

Pharmaceutical treatment with antifungal agents has proven effective in both man and animals if initiated immediately following diagnosis. Long-term antifungal therapy is often necessary, and should continue for at least one year for disseminated infections. There is no effective vaccine available yet for man or animal to protect against coccidioidomycosis infections. Precautionary measures should be taken by persons and animals in endemic areas or who have significant exposure to dusty conditions.

*Mycoplasma mycoides mycoides* small colony type (SC type) bacteria are the causative agent of contagious bovine pleuropneumonia (CBPP). (CBPP is extremely infectious in cattle, and causes lung and occasionally joint disease. The genus *Bos* including bovine and zebu (a group of breeds of humped cattle found in India, East and West Africa, and Southeast Asia) are the main hosts for CBPP. European breeds seem to be more susceptible than African breeds, and animals less than three years old are also more susceptible. Bison and yak have been infected in zoos, and infections have been reported in water buffalo. Wild bovids and camels are resistant. Close contact is necessary for transmission, which occurs primarily through the inhalation of infected droplets from a coughing animal. The organism is also present in saliva, urine, fetal membranes, and uterine discharges. Transplacental infection has been known to occur.

Contagious bovine pleuropneumonia is endemic in Africa (shown in blue), and has a very high incidence in Zambia, Tanzania, and Botswana (red). It is less prevalent in Spain, Portugal, Italy, the Middle East, India, and China (yellow), and has been eradicated from the Western hemisphere, the UK and Australia (green).

The incubation period for contagious bovine pleuropneumonia can be as long as 20-123 days for this respiratory disease of cattle. Common clinical findings include coughing with an outstretched neck (top photo) and a broad stance with the front legs placed far apart (bottom photo). Animals with chronic infections have less obvious signs of pneumonia. They may cough with exercise, are thin and depressed, with recurrent mild fever. Infected calves commonly have polyarthritis with or without pneumonia. Chronic cases may appear to recover but 25% remain subclinical and serve as carriers. Morbidity and mortality rates vary greatly for CBPP. Breed susceptibility, general health and management systems all influence the severity of infection. Morbidity increases with close confinement, and can reach 100% in susceptible herds. Mortality ranges from 10-70% and can be affected by nutrition and parasitism. Photos courtesy of www.fao.org.
In countries which still have a high incidence of CBPP, such as Zambia, Tanzania and Botswana, the social and economic impact of the disease is substantial. Drought conditions have led to the increased movement of animals, resulting in rapid spread of the disease throughout Africa. Depending on the country, farmers may not be compensated for their lost livestock. Antibiotic treatment is generally not effective as it can result in extensive tissue damage and sequestration of the organism. As soon as an outbreak is suspected, slaughter and necropsy of a suspect animal is advisable. Immunization with attenuated vaccine (T1/44 strain) is helpful in disease eradication. However, many of the countries in which CBPP is a serious problem have desperate economic situations, and vaccination may not be possible. Humans are not susceptible to contagious bovine pleuropneumonia infection.

Two bacterial organisms have been reported as the causative agents for contagious caprine pleuropneumonia (CCPP). Mycoplasma capricolum subspecies capripneumoniae (biotype F38) is the most contagious and virulent. Mycoplasma mycoides capri (type strain PG-3) also appears to cause the disease in goats, although much less commonly and with somewhat different signs. Transmission of CCPP is by direct contact through inhalation of infected respiratory droplets. Mycoplasma F38 is much more contagious than M. mycoides capri, and carrier animals may shed more organisms after times of stress and sudden changes in climate. CCPP can be found in Africa, the Middle East, Eastern Europe, the former Soviet Union, and the Far East. Neither of the causative organisms has been found in North America.

The goat industry in the United States is not as large as it is in Africa and Asia, where goats are important sources of meat, milk and hides. In those countries, CCPP is a disease of major economic importance having both direct and indirect effects. The high mortality, reduced milk and meat production, and the costs of treatment, control, diagnosis and surveillance all have a direct effect on the goat industry. In addition to these, there are also indirect losses due to the implementation of trade restrictions. Antibiotics can be helpful in the treatment of CCPP, but their success depends on early intervention and treatment. In countries that are newly infected, trade and movement restrictions and the slaughter of infected animals is recommended. Vaccines are available in some countries, and have been reported to provide good to excellent protection. Humans have not been found to be susceptible to infection by either of these mycoplasma organisms.
Foot and mouth disease (FMD) is a highly contagious vesicular disease of cloven-hoofed animals caused by a *Picornavirus*. FMD is transmitted by direct contact, aerosol, and fomites. Direct contact with large infective droplets from the respiratory track of an infected animal, or contact with infective body fluids like saliva, feces or urine are potential modes of FMD transmission. Humans and animals that come in contact with an FMD infected animal may serve as a fomite (contaminated feed, coveralls, shoes, instruments, etc).

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It is important to understand that FMD has and is currently occurring in many countries around the world. This map is taken from the World Organisation for Animal Health (formally known as the OIE- Office of International Epizootics) website as of March 25, 2001, and while it is a little dated, gives an accurate assessment of the worldwide distribution of this disease.

The incubation period for FMD is 2 to 12 days with an average of 3 to 8 days. The virus is shed before clinical signs develop in infected animals. Initial clinical signs in cattle are fever, excessive salivation, depression, and anorexia caused by painful vesicles of the oral and nasal cavity and teats. Lameness is caused by hoof lesions in the area of the coronary band and interdigital space. The vesicles rupture, leaving large painful sores which may become secondarily infected. Cattle are the indicator host, and they are generally the first species to show signs. Their lesions are more severe and progress more rapidly than in other species. In pigs, sheep, and goats the clinical signs are similar to cattle but milder. Lameness tends to be the predominant sign. Sheep and goats are maintenance hosts because they have very mild clinical signs and diagnosis can be delayed. Pigs are considered the amplifying hosts. The photo depicts ruptured vesicles on this pig’s leg and coronary band due to FMD.

Estimates of the 2001 FMD outbreak in the U.K. put overall economic losses over $18 billion USD due to the total economic strain placed on the agriculture and food industry ($5.7 billion), compensation to farmers ($2.0 billion), tourism ($8.2 to $9.7 billion by 2005) and sports ($1.3 billion). Indeed, while it is known that 6 million animals were slaughtered in the U.K. to control this disease, resulting in them reaching FMD free status in less than one year, the true costs will likely never be known. The public witnessed something few had ever seen. Mass slaughter was called into question, as were animal welfare and animal rights. Pollution from pyres of burning carcasses was intense in some areas and also impacted public health.
Heartwater is a disease caused by *Cowdria ruminantium*, a rickettsial bacterium (family *Rickettsiaceae*). Heartwater causes severe disease in cattle, sheep, goats, and water buffalo; mild disease in some indigenous African breeds of sheep and goats; and inapparent disease in several species of antelope indigenous to Africa. The disease is spread by *Ixodid* ticks (primarily *Amblyomma variegatum* – tropical bont tick) and is endemic in Africa and the Caribbean islands. Potential arthropod vectors for the disease exist in the United States. These three-host ticks can become infected during larval or nymphal stages and transmit the organism to the subsequent life-cycle stage (transstadial transmission). In endemic areas, there has been evidence of transmission of heartwater from infected cows to their calves through colostrum. Some wild ruminants have been shown to harbor *C. ruminantium* subclinically for long periods and play a role as source of infection for ticks. Cattle egrets have become established in many regions with heartwater and have been implicated in the recent spread of the disease.

The incubation period for Heartwater ranges from 14-28 days, typically being shorter in sheep and goats than in cattle. Untreated non-native cattle, sheep, and goats often have morbidity rates approaching 100%. Death rates of 80% has been recorded in Merino sheep and Angora goats. Disease can be peracute (rare), acute (most common), subacute (rare), mild or subclinical (indigenous breeds) determined by various strains of the heartwater agent and animal susceptibility. **Peracute form:** sudden death in non-native breeds of cattle, sheep, goats, and heavily pregnant cows. **Acute form:** acute high fever, loss of appetite, depression and respiratory distress and tachypnea followed by nervous disorders. **Mild form:** transient fever mainly presenting in some indigenous breeds and antelope species. **Subacute form:** prolonged fever and coughing due to prolonged edema of the lungs. Hydropericardium is pictured.

Heartwater is a serious threat to the United States considering the presence of *Cowdria ruminantium* in Africa and the Caribbean. It has been estimated that between 40% and 100% mortality in the Caribbean and the proximity to the southern coast of the United States. The greatest components of economic loss were acarcide costs (76%), followed by milk loss (18%) and treatment cost (5%). Heartwater is a serious threat to the United States considering the presence of the disease in the Caribbean and the proximity to the southern coast of the United States. It has been estimated that between 40% and 100% mortality in cattle population will be recorded if heartwater enters the United States. Tetracycline antibiotics (especially oxytetracycline) are very effective in the treatment of heartwater, especially when used early in the course of the disease. The only commercial vaccine available is made of the blood of sheep infected with live *C. ruminantium* and it can be administered to cattle, sheep and goats.
Japanese Encephalitis (JE) is an enveloped single stranded RNA virus that affects humans and several species of animals. Mortality rates are very high in neonatal pigs. Known amplifying hosts include domestic pigs and wading bird species (i.e., egrets, herons). Studies have demonstrated that bats are susceptible to infection with JE and that their levels of viremia are also sufficient to infect mosquitoes, thereby serving as a reservoir as well. Several additional species including horses, donkeys, cattle, water buffalo, sheep, dogs, chickens and ducks can become infected with JE but are incidental hosts and do not achieve high enough viremias to cycle the virus in nature. Humans are also incidental hosts and often suffer from severe nervous sequela following infection. The most important vectors are *Culex* species, with *Culex tritaeniorhynchus* being the primary vector. The enzootic cycle involves mosquitoes and an amplifying (reservoir) host. JE virus infection occurs throughout the temperate and tropical regions of Asia. Currently, the disease occurs in China, India, Nepal, the Philippines, Sri Lanka and Northern Thailand. Occasionally sporadic cases of disease occur in Indonesia and northern Australia.

The incubation period in horses with JE disease is 8 to 10 days while humans range from 6-8 days. JE in animals is most commonly seen in late summer to early fall. Affected horses will show fever, impaired locomotion, stupor and teeth grinding. Blindness, coma and death are possible but mortality is typically low. Swine that are infected with JE commonly show few clinical signs except stillborn or mummified fetuses delivered full term. If infected piglets are born alive, they will have tremors, convulsions and die soon after birth. Human disease varies from a febrile headache to an acute and possibly fatal encephalitis. The majority of cases are asymptomatic or have mild clinical signs, such as fever and headache. Only one in 250 infections of JE results in symptomatic disease, but mortality rates can vary from 5-35% depending on treatment. Approximately 33-50% of the patients with symptomatic disease, who survive, have major neurologic sequelae at 1 year.

The mortality rate in piglets can be quite high from JE. This reduction in number of offspring can have an great economic impact for the swine market. Since humans are also quite susceptible to JE, the need for immunization of the population and treatment of affected persons can lead to an great economic demand to the public and the medical community. Additionally, vector control measures will be needed to aid and protect the population. There is no effective treatment for JE and supportive care is recommended. Prevention measures are very important for minimizing JE infection such as vector control. Equine and swine in affected areas should be vaccinated. The live attenuated vaccine is used in most JE endemic regions. For humans in endemic areas, vaccination should be implemented, as well as personal protective measures. A formalin inactivated vaccine (JE-VAX) is licensed in Canada and is recommended for those of increased risk such as laboratory workers and travelers spending more than one month in endemic/epidemic areas during the transmission season.
Lumpy skin disease (LSD) is caused by a virus in the family Poxviridae, genus *Capripoxvirus*. It is related antigenically to sheep and goat pox viruses. LSD is a disease of cattle, but the oryx, giraffe and impala are susceptible to experimental infection. Transmission of the LSD virus is primarily by biting insects, particularly mosquitoes (eg. *Culex mirificens* and *Aedes纳税ionius*) and flies (eg. *Stomoxys calcitrans* and *Biomyia fasciata*). Epidemics occur in the rainy seasons. Direct contact is also a minor source of infections. Virus can be present in cutaneous lesions, saliva, nasal discharge, milk, and semen. There is no carrier state, and the spread of the disease is often related to movement of cattle. LSD was first described in Zambia (then Northern Rhodesia) in 1929 and extended northwards through sub-Saharan West Africa through a series of epizootics through the 1960’s. During the 1990’s there was a resurgence of the disease in southern Africa.

The incubation period for LSD varies from 2 to 5 weeks. Clinical signs can range from unapparent to severe. Host susceptibility, dose and route of virus inoculation affect the severity of disease. Young calves often have more severe disease. In adults, animals often have a fever and decreased milk yield. Ten days later nodules appear on the skin, anywhere on the body. Skin nodules may be few to many hundred in number and may coalesce to form plaques. Cellulitis and sloughing of large areas of skin occur. Sterility in bulls and abortion in cows may occur, and the disease can affect almost all organs. The swollen skin nodules may separate from the healthy skin and dry and harden to form a “sitfast”. If the “sitfast” is shed, an ulcerative nodule remains. The morbidity rate in cattle can vary from 3 to 85% depending on the presence of insect vectors and host susceptibility. Mortality is low in most cases (1-2%) but can be as high as 20-85%.

Although the mortality rate is usually low, the disease is of major economic importance due to production losses resulting from severe emaciation, lowered milk production, abortion, secondary mastitis, loss of fertility, extensive damage to hides. Animals infected with LSD generally recover but it may take several months and be prolonged with secondary bacterial infection. Antibiotics to control secondary infection and good nursing care are recommended. If LSD occurs in an area usually free of the disease, quarantine, slaughter of infected and exposed cattle, cleaning and disinfection of premises, and ring vaccination are recommended. In endemic areas, vaccination against LSD has been successfully practiced. In the Union of South Africa, an attenuated LSD vaccine is used while in Kenya and Egypt, sheep and goat pox virus vaccine is used. There is no evidence that the lumpy skin disease virus affects humans.

Malignant catarrhal fever (MCF) is caused by one of two gamma herpesviruses. Wildebeest in Africa are the natural host species that carry the alcelaphine herpesvirus-1 (AHV-1). All varieties of domestic sheep in North America and throughout the world are carriers of ovine herpesvirus-2 (OHV-2). MCF in these natural hosts do not experience clinical disease. Other species, including cattle, bison, and other wild ruminants are susceptible to MCF and can develop an infection. Animal transmission varies depending on the serotype and species. Stressing animals can cause shedding in nasal secretions making spread to other susceptible animals (namely cattle) via aerosol possible or by contaminated feed and water. Cattle-to-cattle, bison-to-bison, or deer-to-deer transmission is rare and they considered are dead end hosts once infected with OHV-2 or AHV-1. All ages of sheep can be infectious to susceptible animals but spread to cattle most often occurs during lambing. Lambs become infected after birth up to 4 months of age.
Experimental infections with MCF have an incubation period of 9 to 77 days but it is unknown in natural infections. Some animals are subclinically infected and develop disease when they become stressed. Clinical signs initially include depression, diarrhea, disseminated intravascular coagulation (DIC), dyspnea, high fever of 105.8°F to 106.7°F, inappetence, and sudden death. Cattle have four clinical forms of MCF. First is the acute form where sudden death can occur, which is also common in deer. Second is the head and eye form which is the most common in cattle. It progresses through the early signs of fever, reddened mucosa and enlarged prescapular lymph node. Eventually the lesions become necrotic and death can occur. Third is the intestinal form which has the same early signs as the head and eye form but the animal dies of severe diarrhea before the lesions become necrotic. The fourth form is mild and only occurred in cattle that were experimentally inoculated with an attenuated virus and recovered. Deer and antelope may have minimal lesions or be less specific than cattle or bison, but many of the same signs occur.

Given the carrier status of this virus in the sheep, goat and wildebeest population, economic impact varies. Zoologic parks spend hundreds to thousands of dollars on some of their exotic species and could later lose them to infection with MCF. As this is not a reportable disease in all 50 states, tracking the true economic impact is difficult. It is a concern for bison breeders, as well as cattle producers, elk and deer farmers, but hard numbers were difficult to find to quantify the potential losses. Mortality in clinically ill animals is nearly 100% and survival in other exposed animals is rare. Supportive therapy (fluids) and antibiotics for secondary bacterial infections can be tried for valuable animals. If they recover they will remain virus carriers and could spread infection. Should an epidemic occur, clinical and carrier animals should be separated from susceptible species. As domestic sheep and goats are asymptomatic carriers, they should be kept separated from cattle at all costs, especially during parturition. There is no vaccine currently available, but experimental evidence in cattle has shown some protection from challenge inoculation. MCF has not been documented to cause disease in humans.

In 1997, a swine reproductive disease outbreak in Menangle, New South Wales, Australia, led to the discovery of a new virus, called Menangle. Menangle is one of several recently discovered RNA viruses (i.e., Nipah, Hendra and Tioman) in the family Paramyxoviridae. The virus is thought to infect pig fetuses by crossing the placenta. The virus does not appear to be highly contagious but tends to spread slowly throughout the population and appears unable to survive in the environment for any length of time. Currently, the exact mechanism of transmission of Menangle virus is not known; however a fecal-oral or urinary-oral route is suspected. The reservoir for the virus is considered to be native fruit bats. These bats have also been found to be the reservoir for other newly emerging Paramyxoviruses (i.e., Nipah and Hendra viruses). Photo: Grey-headed flying fox (Pteropus poliocephalus) Courtesy of G.B. Baker, School of Resources, Environment and Society – The Australian National University at http://sres.anu.edu.au/associated/batlas/grey.html.
Exotic Newcastle Disease (END) is a viral infection of poultry and is caused by a Paramyxovirus. There are nine avian paramyxovirus serotypes and Newcastle disease virus is designated as APMV-1. Newcastle disease virus strains are grouped into four different pathotypes based on their clinical signs and increasing virulence. Migratory birds have been implicated in the primary introduction of the virus, with outbreaks the result of secondary spread by humans. Feral pigeons have also been implicated in the contamination of poultry feed. Newcastle disease virus can survive for long periods in the environment, especially in feces. Some psittacine species can shed the virus intermittently for a year or more. People can become infected with vND and have signs of conjunctivitis which resolves quickly, with virus being shed in ocular discharges for 4-7 days. vND is endemic in many parts of the world including countries in Asia, the Middle East, Africa, and Central and South America. The United States and Canada have seen high mortality in wild cormorants caused by vND.

Although the incubation period for Menangle is not known, pigs seroconvert to the virus in 10-14 days. The virus causes disease in developing pig fetuses. Fetal mummification and stillbirths, some having deformities of the skeletal or nervous are the most commonly seen. Arthrogryposis, brachygnaethia and kyphosis can also be seen. Additionally, there will be a reduction in the farrowing rate as well as the number and size of litters. No clinical signs have been seen in postnatal pigs of any age. Serology on a variety of wild and domestic animals (cattle, sheep, birds, rodents, cats and dogs) in the vicinity of the affected piggery tested seronegative.

Menangle virus has the potential to cause a great economic impact when introduced into a naïve swine population. The loss of fetal and prenatal piglets, reduced piglet size and number per litter will greatly decrease the number of animals available for sale which will be economically damaging. Fortunately, all postnatal pigs that seroconvert to the virus within 10-14 days develop a strong immunity to the virus and reproductive failures no longer occur. Persistent infections in swine do not occur and once the infection is endemic in the herd no further reproductive failures occur. Farrowing percentages decreased 44% during the peak of the outbreak. Additionally the number of live piglets per litter declined from a mean of 9.7 to 8.1. Currently there is no vaccine for Menangle available. During the 1977 outbreak, two out of over 250 workers who had very close contact with infected pigs developed signs and were seropositive for the virus. Both individuals presented with a sudden onset of malaise, chills, drenching sweats, fever, severe headache and myalgia, but no coughing, vomiting or diarrhea were noted. Within 3-4 days both developed a spotty, red, non-pruritic rash. Both recovered after 10-14 days.

The incubation period varies from 2-15 days (average 5-6) depending on the severity of the strain and susceptibility of the population. Generally virus is shed during the incubation period and for a short time during recovery. Clinical signs in chicken flocks, include an initial drop in egg production followed by numerous deaths within 24-43 hours continuing for 7-10 days. Birds that survive may have permanent neurological damage including paralysis, and reproductive damage. There may be edema of the head especially around the eyes, and greenish-dark watery diarrhea, as well as respiratory and neurological signs. Clinical signs associated with the various strains can be different in species other than chickens. Morbidity and mortality rates can vary greatly depending on the virulence of the virus strain and susceptibility of the host. In chickens, morbidity can be up to 100% with 90% mortality. In other species such as finches and canaries, clinical signs may not be present. A carrier state may exist in psittacine and some other wild birds. Ducks and geese may be
infected and show few or no clinical signs, even with strains lethal for chickens. The photo depicts a chicken with respiratory signs and increased salivation due to vND.

The global economic impact of exotic Newcastle disease is enormous. No other poultry virus comes close and it may represent a bigger drain on the world’s economy than any other animal virus. Countries free of vND are faced with repeated testing to maintain that status for trade purposes. In October 2002, vND was confirmed in the State of California. Cases occurred in Nevada, Arizona, Texas and New Mexico. As of July 7, 2003, with the epidemic in the final phase of eradication, almost 4 million birds on 2,662 premises had been depopulated. Eradication efforts have cost taxpayers $160 million to date (July 2003). In developing countries with endemic vND this is an important limiting factor in development of commercial poultry and the establishment of trade links. Many developing countries rely on village chickens to supply dietary protein in the form of eggs and meat. Continued losses from vND affect the quantity and quality of the food of people on marginal diets. Vaccination is routine in poultry flocks. While vaccination will reduce the severity of clinical disease caused by vND it will not prevent infection and virus shedding. The economic impact of vND is not only measured in direct commercial losses, but in some countries in the effect on human health. Humans can acquire eye infections by direct contact that consists of unilateral or bilateral reddening, excessive tearing, edema of the eyelids, conjunctivitis and subconjunctival hemorrhage. Infections are usually transient, the cornea is not affected, and human-to-human spread has not been reported. Laboratory workers and vaccination crews are most at risk for ND infection, but poultry workers are rarely infected. No known infections have occurred from handling or consuming poultry products.

Peste des Petits Ruminants (PPR) is an acute or subacute viral disease of goats and sheep that is very similar to rinderpest virus. The name is French for “disasterous disease of small ruminants”. Goats are usually more severely affected than sheep. Transmission of PPR requires close contact. The virus is present in ocular, nasal, and oral secretions as well as feces. Most infections occur through inhalation of aerosols from sneezing and coughing animals. There is controversy over whether fomites can play a role in transmission of PPR. The morbidity and mortality rates from PPR can be up to 100% in severe outbreaks. Severity depends upon the susceptibility of the population. The incidence of PPR in an enzootic area is similar to that of rinderpest in that a low rate of infection exists continuously. When the susceptible population builds up, periodic epizootics occur with almost 100% mortality. Presently, PPR occurs in most African countries situated in a wide belt between the Sahara and the equator; the Middle East (Arabian Peninsula, Israel, Syria, Iraq, Jordan), and the Indian subcontinent.
The incubation period of Peste des Petits Ruminants is 3-10 days. Most cases of PPR are acute, with a sudden fever that may last for 5-8 days before the animal either dies or beings to recover and is characterized by fever, erosive stomatitis, conjunctivitis, gastroenteritis, and pneumonia. Young animals (4-8 months) have more severe disease. Poor nutritional status, stress of movement and concurrent parasitic and bacterial infections enhance the severity of clinical signs. The characteristic signs begin with a serious nasal discharge that becomes mucopurulent and may progress to a severe catarrhal exudates that blocks the nostrils causing respiratory distress. The nasal mucous membranes may develop small areas of necrosis and profuse catarrhal conjunctivitis with matted eyelids is often seen. Necrotic stomatitis is also common and can be severe. Concurrently, animals will most likely have profuse, non-hemorrhagic diarrhea resulting in severe dehydration, which may progress to emaciation and death within 5-10 days. Abortion may be seen in pregnant animals. The severity of the disease and outcome in the individual is correlated with the extent of the mouth lesions. Prognosis is good in cases where the lesions resolve within 2 to 3 days. It is poor with respiratory involvement or when extensive necrosis and secondary bacterial infections result in a fetid odor from the animals mouth.

The presence of Peste des Petits Ruminants can have a serious impact on the economics of a region. Economic losses are due to loss of production, death and abortion. The presence of disease can limit trade, export, import of new breeds and the development of intensive livestock production. PPR is a major constraint on the availability of protein for human consumption as well. There is no specific treatment for PPRV. However, drugs that control bacterial and parasitic complications, as well as supportive care, may decrease mortality. The tissue culture rinderpest vaccine protects goats for at least 12 months against PPR and is currently used in many African countries. The efficacy notwithstanding, its wide use hinders the ongoing Pan-African rinderpest campaign because it is impossible to determine if seropositive small ruminants have been vaccinated or are naturally infected. A homologous attenuated PPR vaccine is being tested and may soon be commercially available.

Rinderpest virus (RPV) is a single-stranded RNA virus in the family Paramyxoviridae. It is very similar to peste des petits ruminants virus, canine distemper virus, human measles virus, and marine mammal morbilliviruses. Rinderpest is highly contagious disease mainly of cattle and domestic buffalo, including water buffalo. Most wild and domestic cloven-footed animals can become infected including zebu, sheep and goats, pigs (Asian pigs appear to be more susceptible than African or European pigs) and wild ungulates. Rinderpest virus is mainly transmitted by direct or close contact with infected animals. Virus is shed in nasal and ocular secretions and in feces, urine, saliva and blood. To a lesser degree contaminated food or water can transmit RPV as well as fomites. As of the year 2000 only a small foci of rinderpest exists in East Africa and possibly Asia. The Global Rinderpest Eradication Program (GREP) is working to eradicate rinderpest by the year 2010. (Photo of calf: P.Roeder at fao.org; historical photo of RPV)
**Rinderpest: The Disease**

- Incubation period: 3-15 days
- Four forms:
  - Classical: Fever, diarrhea, nasal/ocular discharge, oral erosions
  - Peracute: Young animals, rapidly fatal
  - Subacute: Mild signs, low mortality
  - Atypical: Irregular fever, mild diarrhea

The incubation period as well as clinical disease varies with the strain of virus, dosage, and route of exposure. Following natural exposure, the incubation period ranges from 3 to 15 days but is usually 4 to 5 days. Clinically, RPV can occur in four different forms: classical, peracute, subacute, and atypical. The **classical** form of rinderpest virus is most common and consists of fever, constipation followed by watery hemorrhagic diarrhea; serous to mucopulent nasal and/or ocular discharge, necrotic oral erosions, enlarged lymph nodes, dehydration and death in 6-12 days. **Peracute** cases usually occur in young animals that show a high fever, congested mucous membranes resulting in death in 2-3 days. The **subacute** form of rinderpest shows mild clinical signs combined with low mortality rates. The **atypical** form is characterized by irregular pyrexia and mild or no diarrhea. Immunosuppression can lead to secondary infections and the emergence of latent infection.

**Rinderpest: Impact and Response**

- Africa: 1982-84 outbreak cost $500 million
- $100 million spent annually on vaccination world-wide
- Diagnosis usually means slaughter
- Vaccine offers life-long immunity
- Humans not susceptible to disease

Outbreaks of rinderpest virus can have devastating economic effects. Outbreaks can lead to famine in areas where cattle are depended upon for meat, milk and draft power. An epidemic in sub-Saharan Africa in the 1980s wiped out most of the cattle. A 1982-1984 outbreak in Africa caused an estimated $500 million as a result of livestock losses and control measures. It is estimated that $100 million is spent annually world-wide for vaccination. There is no known treatment for Rinderpest virus infection and combined with the high morbidity rates, accounts for the devastating nature of the disease. A diagnosis of RPV usually means slaughter of the affected animals and significant economic loss. In rare cases, supportive care and antibiotic therapy can help in the treatment of especially valuable animals. The most commonly used vaccine is safe for many species and produces life-long immunity in cattle. Humans are not susceptible to rinderpest infection.

**Screwworm Myiasis**

- Larvae of the Family Calliphoridae
- All warm-blooded animals
- Humans and animals infected when female fly deposits eggs into wound
- Morbidity variable, can reach 100%
- Tropical regions

Screwworm myiasis is caused by larvae of the Family Calliphoridae: *Chrysomya bezziana* (Old World Screwworm) and *Cochliomyia hominivorax* (New World Screwworm). Any warm-blooded animal, including humans, is susceptible to infestation; screwworm myiasis, however, is rarely seen in birds. Transmission occurs when a female fly deposits eggs in a superficial wound. One female can lay up to 400 eggs at a time. Morbidity varies between regions, but can near 100% in favorable environments. In some areas the navel of almost every newborn animal can be infested. Mortality is dependent on number of egg depositions and the treatment of such infestations. However, if wounds are left untreated and multiple fly oviposits occur, affected animals often die within 7-10 days as a result of secondary infection or toxicity. Screwworms have been eradicated from the US and much of northern Central America, however they are still present in portions of Central and South America and in the Caribbean Islands. They are also found in most of the remaining tropical and sub-tropical areas of the Eastern Hemisphere.
Screwworm Myiasis: The Disease

- Larvae
  - Emerge in 8-12 hours
  - Visible within 3 days
- Wounds
  - Bloody discharge
  - Foul odor
  - Secondary infection
- Depression, off feed, rubbing
- Signs similar in humans

Any wound can become infested by screwworms. Larvae emerge from eggs within 8-12 hours and begin feeding on living tissue (they do not feed on dead tissue). There may be hundreds of larvae within the wound. Within 3 days the larvae are usually visibly embedded in the wound, a bloody discharge develops and a distinct, foul odor can be detected. The affected animal usually exhibits signs of depression, goes off feed, and separates itself from the herd. Animals often rub against trees, lick the wounds, and stand in water in an attempt to relieve the discomfort. After several days the larvae drop to the ground to pupate. The adult screwworms emerge and are ready to mate within 3-5 days, beginning the cycle again. Infected wounds attract other female flies and multiple infestations often occur. Death can occur in untreated infestations. Lesions may extend into body cavities and lead to associated pleuritis, sinusitis or peritonitis. Death is usually the result of secondary infections and toxicity. Clinical signs in humans will be identical to those seen in animals but death is unlikely to develop. Photos: The top image depicts an infested calf navel. The navel of a newborn animal is a common site of screwworm infestation. Gray Book.

Screwworm Myiasis: Impact and Response

- Estimated losses if reintroduced
  - $540 million annually
  - $1.27 billion for eradication
- Treatment
  - Removal of larvae
  - Topical larvicide 2-3 days
- Sterile fly technique
  - U.S. free in 1966
  - Mexico free in 1991

If screwworm was not controlled in the US, livestock producers and consumers would be seriously affected. Reintroduction of screwworm would generate estimated losses of $540 million annually for production and lost meat supply. If screwworm had to be eradicated again, it is estimated that it could cost $1.27 billion. Treatment consists of careful removal of larvae from an infested wound and or topical application of larvicide directly into the wound for 2-3 successive days. The sterile fly technique has been used extensively throughout North America to aid in screwworm eradication. The technique takes advantage of the fly’s breeding habits. As females flies only breed once in a lifetime, the use of sterilized males will result in unsuccessful mating and eventual eradication of the larvae. The US was declared free of screwworm in 1966, however infection was still an issue due to recurrent cases from Mexico. Mexico was declared free of screwworm in 1991 and the eradication program was extended through Central America to create a permanent barrier to reinfestation. Screwworm is still present in Caribbean islands and portions of South America, necessitating strict control measures. This photo depicts a larvae that was removed from the abdomen of a human patient. www.epmonthly.com/SecondOpinion/SecOp1101B.gif

Sheep and Goat Pox

- Viral infection
  - Capripoxvirus
  - Contagious
- Most important pox disease of domestic animals
- Direct contact
  - Inhalation, insects?
- Parts of Africa, Asia, India, and the Middle East

Sheep and goat pox are contagious viral skin diseases classified with lumpy skin disease virus in the genus *Capripoxvirus* (Family: Poxviridae). Most isolates cause disease mainly in sheep or mainly in goats but some isolates can cause serious disease in both species. The causative viruses cannot be distinguished from each other with current techniques and only one serotype exists. Sheep and goat pox infection are the most important pox diseases of domestic animals, causing significant economic losses, especially among young animals, where the mortality is greatest. Sheep pox and goat pox viruses are usually transmitted by close contact through inhalation of aerosols and through abraded skin by fomites. Insect transmission is possible, but their role in transmission is not clear. Infectious virus is found in all secretions, excretions, and the scabs from skin lesions. Today sheep pox and goat pox are found in central and north Africa, central Asia, the Middle East and parts of the Indian subcontinent. A mild pox-like disease has been reported in California but was unlikely to be a capripox virus.
Sheep and Goat Pox: The Disease

- Incubation period: 4-13 days
- Clinical signs include:
  - Fever, conjunctivitis, dyspnea
  - Skin lesions take up to 6 weeks to heal
- Mortality
  - 50% in susceptible flock
  - 100% in young
- No chronic carriers

The incubation period for sheep and goat pox is 8 to 13 days in most natural infections, but may be as short as 4 days. All ages of sheep and goats can be affected, but it is more severe in the young. Systemic signs may include fever (104-107.6°F), conjunctivitis, rhinitis, lymphadenopathy, lung lesions can cause dyspnea and the mucous membranes can become necrotic. Skin lesions present as erythematous macules that eventually become hard papules. Dark, hard scabs eventually form and may take up to six weeks to heal. In animals with heavy wool, the lesions can be easier to find by palpation than visual inspection. Secondary bacterial infections are common and death can occur at any stage of the disease. Morbidity and mortality vary with the breed of the host and the strain of the virus. Mortality may be up to 50% in a fully susceptible flock and as high as 100% in young animals. Imported breeds of sheep and goats usually develop severe disease when they are moved into an endemic area. Infections have not been seen in wild ungulates and chronically infected carriers are not seen. (Photos: USDA).

Sheep and Goat Pox: Impact and Response

- Infection can limit trade of live animals and product
- Treat secondary infections
- Vaccination
- Endemic areas with attenuated virus
- Slaughter should be considered
- Humans not susceptible

Pox infections can limit trade, export, import of new breeds, and the development of intensive livestock production. Restrictions on the movement of animals and animal products (meat, hair, wool, and hides) are essential to prevent introduction of the disease. Wool, hair, and hides must be subjected to suitable decontamination procedures before entry into non-endemic areas. Treatment is directed at preventing or controlling secondary infection. The most effective means of controlling losses in an endemic area is vaccination, but consideration should be given to eliminating infected and exposed herds by slaughter. Killed vaccines have not proved to be practical under field conditions because they do not provide solid lasting immunity. There are numerous attenuated virus vaccines with immunity lasting up to 2 years. A carrier state has not been shown for SGPV but the virus may persist for many months on contaminated premises. In endemic areas, vaccination is an effective means of controlling losses from SGP. There is no conclusive evidence that sheep and goat pox viruses can infect humans. (Photo: USDA)

Swine Vesicular Disease

- Viral infection
  - Resistant to heat, pH, curing
  - Moderately contagious
- Swine and humans
- Ingestion or close contact
- Previously Europe and Hong Kong
  - Only in Italy as of 2002

Swine vesicular disease is caused by a very hardy virus which can survive for long periods in the environment, is very heat and pH resistant, and can survive up to 2 years in dried, salted, or smoked meat. SVDV is considered to be moderately contagious. Pigs are the only species that are naturally infected, although the virus may be present in sheep or cattle. Infection in humans has also occurred in workers who had contact with SVD-infected pigs and in the laboratory. Transmission can occur by ingestion of contaminated meat scraps and contact with infected animals or infected feces. SVD has been seen in many European countries as well as Hong Kong. Since the 1970s, this disease appears to have been eradicated from most countries. According to the OIE, as of 2002, only Italy was affected with the disease.

SVD: The Disease

- Incubation period:
  - Ingestion: 2-5 days
  - Direct contact: 2-7 days
- Clinically resembles FMD
- Fever, salivation, lameness
- Vesicles
  - Snout, mammary gland, coronary band
- Mortality low

The incubation period for SVD varies with the route of transmission. It can be as short as 2-5 days if the virus is ingested in contaminated meat or 2-7 days if it is acquired through contact with infected animals or fecal material. Clinical signs are very similar to foot-and-mouth disease, and include fever, salivation, and lameness. Vesicles and erosions can be seen on the snout, mammary glands, coronary band, and interdigital areas, but vesicles in the oral cavity are relatively rare. The infection may be subclinical, mild, or severe depending on the virulence of the strain. Recovery will usually occur within 2-3 weeks with little permanent damage. Mortality is not generally a concern with this disease, although it may reach 10% in piglets. No persistent infections have been reported, and all infected pigs have developed protective antibody for SVDV. 

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While it does not cause severe production losses, SVD is of major economic importance because it is difficult to distinguish from foot-and-mouth disease. Control measures and eradication of SVD are costly, and nations which are known to have SVD often face embargoes on the export of pigs and pork products. Treatment includes supportive care. Although there are inactivated vaccines against SVDV, none are commercially available, and vaccination of pigs has never been undertaken in the field. Only a small number of human cases have been documented in laboratory workers with contact with SVDV and SVD-infected pigs. The incubation period in humans varies from 1-2 weeks to up to 5 weeks. Clinical signs include mild influenza-like symptoms (fever, malaise) but vesicular lesions are not seen. All human cases have recovered without sequellae.

Vesicular stomatitis virus (VSV) causes lesions in the mouth and feet of a wide range of animals, but it primarily affects horses, donkeys, cattle, swine, and South American camelids, only in the Western hemisphere. Sheep and goats are resistant to VSV and rarely show clinical signs. Humans can also become infected producing influenza-like symptoms. Vectors, such as sand flies (Lutzomyia shannoni) and black flies (Simuliidae) will transmit the virus through injection and can pass it transovarially to their offspring. Seasonal patterns of transmission exist. Direct contact with infected animals’ saliva, exudate, epithelium of open vesicles or contaminated objects is also effective between animals and to humans. Finally, aerosol transmission in a laboratory setting has led to infection. Morbidity can be up to 90% but does vary with conditions and species. Often infection is sporadic in the exposed group and only 5-10% of the animals in affected herds show clinical signs of VSV. Mortality rate is low. Outbreaks of VSV tend to occur in the warmer regions around riverways and valleys but occasionally occur in more temperate regions. In the United States, the southwest has experienced outbreaks during the warmer months. The top photo is of a sand fly accessed at edis.ifas.ufl.edu/pdffiles/IG/IG08100.pdf

The incubation period for VSV in animals is 3-5 days. Clinically, all vesicular diseases produce a fever with vesicles that progress to erosions in the mouth, nares, muzzle, teats and feet. Lesions in the oral cavity and interdigital region/coronary band can lead to salivation and lameness. These vesicles seem to isolate to one area of the body unlike other vesicular diseases. VSV has clinical signs almost identical to Foot and Mouth Disease. Unlike FMD, horses are affected and very severely. Recovery is within 2 weeks if there is no secondary infection. Vesicular diseases are clinically indistinguishable from one-another, especially in swine and diagnosis can only be made through virus isolation. Following an incubation of 1 to 6 days, humans may display influenza-like symptoms. These include headache, fever, retrobulbar pain when
moving eyes, malaise, nausea, limb and back pain, and rarely, oral vesicles. The
disease is self-limiting and treatment consists of supportive care. Recovery can
be prolonged but death is rare. Clinical diagnosis is difficult as many patients
only exhibit flu-like symptoms and never seek treatment. Recovery occurs
within 4-7 days if not secondarily infected.

Epizootic waves of VSV tend to occur approximately every 10 years in the
United States. There was a major outbreak in 1982 in the western U.S. and
dollars lost per cow varied from $97 to $202. During a 1995 outbreak in the
western U.S., beef cattle owners put the cost per head at $53 for each case of
VS. Losses were attributed to increased culling, reduced milk production,
increased mortality, labor, medicine, and veterinary costs. The most recent large
outbreak in the U.S. outbreak started in a horse in New Mexico in May of 1998
and spread to other horses in Colorado and Texas, and ended in January 1999.
In all, 130 were positive and VSV was isolated out of 27 horses. As with most
viruses, there is no treatment available except supportive care. If secondary
infection is present, antibiotics should be used. Prognosis is good for VSV
infection but production losses can be permanent if the udder of cattle is
affected. There are inactivated and attenuated vaccines that may be made
available during an outbreak but efficacy data is unknown. The photo depicts
vesicles on the teats of a dairy cow with VSV.

The Veterinarian’s Responsibility

As veterinarians, it is your responsibility to be a guardian of animal and public
health, as stated in the oath we took upon graduation. It is important to sharpen
your awareness of disease agents and alert officials if you see signs in animals
or people that suggest either accidental or intentional introduction of a
biological agent. Integrate into emergency response plans at all levels.
Veterinarians deal daily with infectious diseases, many of which are zoonotic,
and have a good understanding of biosecurity issues. Provide leadership and
sound scientific input to your clients and the community because you are an
expert in this area.
A handout with phone numbers of state veterinarians, state public health veterinarians, and APHIS area veterinarian in charge is provided. It is important to keep this list close at hand. If in doubt it is better to call and let the officials decide if your situation needs further investigation. The faster an outbreak can be identified, the faster it can be contained and controlled. For your convenience, there is a handout on your CD ROM that lists the state veterinarian, state public health veterinarian, and the AVICs and their contact phone numbers. If you are presenting this to colleagues in your area, you may want to include the specific name and phone number on this slide for better reference.

We have discussed several main points. We addressed the importance of livestock and agriculture to the U.S. economy. We discussed specific diseases on the USDA High Consequence Livestock Pathogen and list, noting the zoonotic potential. We also discussed the veterinarian’s role in preventing the spread of a high consequence livestock pathogen, pointing out that awareness education is an important component of preparedness and protection.

"The best prescription, is knowledge."

Dr. C. Everett Koop
Former U.S. Surgeon General

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