African horse sickness (AHS) is a serious, often fatal, disease of equines (e.g., horses, mules, donkeys). AHS is considered one of the most lethal horse diseases. The disease is spread by insect vectors (primarily Culicoides species—biting midges), with mortality in horses as high as 95%. The disease primarily occurs in Africa (see map) but outbreaks have occurred in parts of the Middle East, Egypt, Spain, Portugal, Morocco, Pakistan, and India. The disease is not found in the U.S. but potential insect vectors for the disease exist in the United States. Since the disease has never occurred in the Americas, our Equidae species are naïve and highly susceptible to the virus. The disease most commonly occurs in the late summer and early autumn and is related to climatic conditions that favor insect breeding (periods of drought followed by heavy rains). No natural human cases of AHS have been reported. [Photos: (top) Distribution of most AHS cases from Government of United Arab Emirates at http://www.uae.gov.ae/uaeagricent/livestock/img/horse_Sickness1.jpg; (bottom) A biting midge from the USDA]

The impact of AHS in loss of animals as well as control measures is highlighted by the outbreak of AHS in Portugal in 1989. The disease was found on 104 farms; over 200 equines died or were destroyed and an estimated 170,000 equines were vaccinated. It took over one year, for Portugal to successfully eradicate the disease and be declared free of AHS, at an estimated cost of US $1.9 million. In the U.S., the equine industry (in 1998) has an estimated 5.25 million horses (sales value of $1.75 billion) and employs over 7 million Americans (i.e., horse owners, service providers, employees or volunteers) (USDA, National Agricultural Statistics Service). Current efforts to prevent the introduction of AHS into the U.S. include import restrictions or mandatory 60-day (minimum) quarantine of any equids from endemic countries in an insect-proof facility at the point of entry. Additional prevention and control measures (that you can do) for AHS include vector control measures (e.g., destroying biting midge habitat, pesticides) and stable horses in insect-proof housing (especially during dusk to dawn when insect vectors are most active). Closely monitor animals for fever and contact your

Viral infection
- Early autumn
- Monitor animals for fever
- Horses, mules, donkeys
- Stabling in insect-proof facility at point of entry
- Import restrictions and quarantines
- Vector control
- Stabling in insect-proof housing
- Monitor animals for fever
- Vaccine available in endemic areas
- Droughts followed by heavy rains
- Animals (i.e., horse owners, service providers, employees or volunteers)

Impact
- 1989: Portugal outbreak
  - Eradication cost: $1.9 million
  - Sale: $1.75 billion
- Prevention and Response
  - Import restrictions and quarantines
  - Vector control
  - Stabling in insect-proof housing
  - Monitor animals for fever
  - Vaccine available in endemic areas

Incubation period
- 2–14 days
- Clinical signs in 5–7 days
- Respiratory and cardiac disease
  - Fever
  - Difficulty breathing, foaming from nostrils, swelling of head and neck

AHS: Impact & Response

AHS: The Disease

AHS: The Disease

AHS: The Disease

AHS: The Disease

Animal Disease Emergencies

Diseases of Concern

African Horse Sickness

- Viral infection
- Spread by insects
  - Death rate up to 95%
- Spread by insects
  - Biting midges (Culicoides)
- Occurs in Africa
  - Outbreaks in other countries
  - Not found in U.S.
- Late summer – early autumn
- Droughts followed by heavy rains
- Does not affect humans
African swine fever (ASF) is a highly contagious viral disease affecting domestic and wild pigs; the disease is usually fatal. The virus (ASFV) is spread by direct contact (oronasal) with infected animals, ingestion of contaminated animal by-products, indirectly by contaminated equipment, vehicles, footwear, feed or clothing. The virus can also be spread by certain ticks \(\text{(Ornithodoros sp. (soft ticks))}\) and possibly by biting flies. ASFV can be found in all tissues and body fluids of infected swine, with particularly high levels in blood, which may lead to environmental contamination; the virus can persist for up to a month in contaminated pig pens and in some pork products for over 4-1/2 months. ASF has primarily spread between countries through the feeding of uncooked garbage containing ASFV-infected pork scraps. ASF is endemic in most of sub-Saharan Africa, including the island of Madagascar, with the highest area of incidence seen from the Equator to northern South Africa. Outbreaks have also occurred in Europe, South America, and the Caribbean. ASF has been eradicated from the Western Hemisphere, and has never been found in the U.S., however increasing globalization increases the risk of introducing ASF into North America. There is no known risk to humans. [Photo (top) shows endemic countries (red) and those with sporadic outbreaks and infected wild pigs (orange); yellow indicates areas where ASF has been eradicated following incursion (from Institute for Animal Health at http://www.iah.bbsrc.ac.uk/ASF_Georgia_12jun07_copy(1).htm). Photo (bottom): Ornithodoros spp. (soft tick) from http://www.nhc.ed.ac.uk/images/collections/ticks/soft/image019.jpg]
<table>
<thead>
<tr>
<th>Slide 7</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ASF: Impact and Response</strong></td>
</tr>
</tbody>
</table>
| • Huge economic impact  
  – Import/export ban  
  – Movement restrictions  
  – Depopulation  
  – Disinfection  
• No treatment or vaccine  
• Virus killed by high temperatures  
• Many disinfectants ineffective  
• Humans not affected |

<table>
<thead>
<tr>
<th>Slide 8</th>
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<tbody>
<tr>
<td><strong>ASF: Prevention</strong></td>
</tr>
</tbody>
</table>
| • Do not feed uncooked garbage  
• Biosecurity  
  – Isolate animals before introduction into herd  
  – Restrict and monitor visitors  
  – Cleaning and disinfection protocols  
* • Vehicles, trailers, equipment, footwear  
• Tick and fly control  
• Prevent contact between domesticated and feral swine |

<table>
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<th>Slide 9</th>
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<tbody>
<tr>
<td><strong>Anthrax: The Agent</strong></td>
</tr>
</tbody>
</table>
| • Gram positive, spore-forming bacteria  
  – Bacillus anthracis  
• Forms spores  
• Human disease  
  – Skin  
  – Intestinal  
  – Inhalational  
• Animal disease  
  – Septicemia and rapid death |

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 the Dakotas, northwest Minnesota, Texas, and Nevada, with smaller areas in other states. Spores can remain viable for decades in 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) **Gastrointestinal anthrax** develops after eating contaminated meat. The initial signs may be mild malaise and gastrointestinal symptoms. Severe symptoms can develop and rapidly progress to shock, coma, and death. 3) **Inhalational 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 usually sought. In animals, sheep, cattle, and horses are very susceptible, while dogs, rats, and chickens are more 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. (Top photo: Eschars and edema of anthrax lesions on upper left arm; Bottom photo: A
healing cutaneous anthrax lesion on the neck. [Images from CDC: http://www.bt.cdc.gov/agent/anthrax/anthrax-images/ cutaneous.asp]

In the 1950s and 1960s, *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* that 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 gastrointestinal 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.)

Modified live vaccines are available for livestock, and should be used annually to protect animals in endemic areas. 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 gastrointestinal 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 (5% formaldehyde, 2% glutaraldehyde, 10% sodium hydroxide) or sterilization (chlorine dioxide, formaldehyde gas, heating to 121°C for at least 30 minutes).

Aujeszky’s disease, also known as pseudorabies or mad itch, is a highly contagious viral disease of swine that causes reproductive and severe neurological disease in affected animals; death is common. Pigs are the natural host for Aujeszky’s disease virus and the only animals to become latent carriers. The virus can infect nearly all domesticated and wild mammals, including cattle, sheep, goats, cats, and dogs. It does not affect humans and infections in horses are rare. The virus is somewhat persistent in the environment and may survive for several days in contaminated bedding and water. Aujeszky’s disease still occurs in parts of Europe, Southeast Asia, and Central and South America, including Mexico, and has also been reported in Cuba, Samoa, and Rwanda. Successful eradication of the disease has occurred in several countries of Europe, Canada and New Zealand. Additional countries are conducting eradication programs. Until recently, Aujeszky’s disease was endemic in the United States; however, a successful eradication campaign has eliminated the virus from domesticated swine as of December 2004. The virus remains present in feral pigs in the U.S.; this remains a concern due to the potential for transmission to domesticated herds. A surveillance program continues to monitor domestic herds for the disease. As of Feb 2008, all U.S. states were classified as status 5 (free of pseudorabies). Disease from Aujeszky’s disease has not been seen in humans. [Photo: Distribution of Aujeszky's disease from Jan-June 2007. Red indicates...
The Aujeszky’s disease virus is spread between pigs by direct contact, reproductive routes, fomites, aerosol or oral routes as well as by sexual transmission (considered a principal route of transmission between feral swine) or from infected sows to their fetuses. Under suitable conditions (relative humidity at least 55%), the aerosolized virus may travel up to two kilometers or remain infectious for up to seven hours. Fomites (contaminated objects such as equipment, vehicles, footwear) or infected carcasses can serve to spread the virus. Other animal species usually become infected following direct contact with infected pigs or following ingestion of contaminated raw meat. Disease occurs 2-6 days after exposure. Piglets less than a week old will have fever, listlessness, and anorexia, followed quickly by nervous system signs (e.g., tremors, paddling, seizures, hind leg paralysis). Death within 24-36 hours is common. In older piglets, the death rate is lower; vomiting and respiratory signs are seen. Adult pigs usually have mild or inapparent infections; respiratory signs are the most common manifestation, but neurologic signs can occur. Pregnant sows may resorb infected fetuses, abort, or give birth to weak neonates; a litter can contain a mixture of normal piglets, stillborn piglets, and weak piglets. Feral swine usually show no signs of disease. Other animal species (e.g., cattle, sheep, goats, dogs, cats) are severely affected and typically die within a few days. Affected animals will have intense itching concentrated in a patch of skin and is manifested as severe licking, rubbing, or gnawing. Self-mutilation, convulsions, bellowing, teeth grinding, cardiac irregularities, and rapid, shallow breathing are common. In dogs and cats, neurological signs, such as pharyngeal paralysis and profuse salivation may resemble rabies. [Photos: (top) Sow and piglets from USDA ARS; (bottom) Calf with Aujeszky’s disease licking “itchy” area from www.vetmed.uni-muenchen.de/med2/skripten/b8-5.html].

Aujeszky’s disease is a reportable disease (in the U.S. and internationally). Occurrence of the disease can result in trade restrictions and consequential economic impacts for the country. Eradication programs are underway or have been successful in many countries. Treatment is usually not recommended due to the possible development of latent infections. In the U.S., following identification of a confirmed case, all movement of swine within a five-mile radius of the case and from exposed herds must be stopped until testing is completed. Disease eradication strategies include depopulation and repopulation, test and removal, use of a marker vaccine and companion diagnostic test, and offspring segregation strategies. The premises are cleaned, disinfected, and left empty of pigs for 30 days. Vaccines are available in some countries. [Photo: Feral pigs from USDA Wildlife Services].
Avian Influenza, Highly Pathogenic (HPAI)

- Type A Influenza virus – H5 or H7 surface antigens
- Domestic and wild birds
- Humans
- Reservoir: Migratory waterfowl
- Aerosols, contaminated drinking water
- Influenza-like infection for humans exists but is very low because strains are not in avian species. Numerous avian influenza viruses exist, but only those with surface antigens designated as H5 and H7, are considered highly pathogenic. Low pathogenic avian influenza viruses also exist and can in some situations mutate to highly pathogenic forms. Highly pathogenic avian influenza (HPAI) causes decreased egg production, depression, and often sudden death in affected birds. Migratory waterfowl are considered reservoirs of avian influenza virus, and shed the virus in their feces and respiratory secretions; the virus can also spread by aerosols, contaminated water and fomites (contaminated objects). Once a flock is infected, it should be considered a potential source of virus for life. Outbreaks of HPAI have occurred worldwide, but have been eradicated from many countries.

Incubation period is from 3-14 days and is dependent on the dose of virus, the route of exposure, the species exposed. Some birds have sudden death, drops in egg production and vocalization; neurological signs can also occur. Affected birds are often depressed. In mature chickens, the combs and wattles are often swollen and may be cyanotic (blue-purple coloration). Swollen, reddened eyelids and swelling of the head and neck can occur. Respiratory signs are less frequent but can include rales, sneezing and coughing; nasal discharge may be seen. Death is common, but birds, even severely affected ones, occasionally recover. The risk of avian influenza infection for humans exists but is very low because strains vary in their ability to transmit and infect. Disease in humans was first reported from an outbreak in Hong Kong in 1997 (18 people were hospitalized and 6 died). Since then other human cases have been reported in association with outbreaks in poultry. Most human cases occurred following close contact in infected birds. The current (2003-2008) H5N1 outbreak, which began in poultry in Southeast Asia and has since spread to parts of Europe, the Pacific, the middle East and Africa, has resulted in over 380 human infections and 241 deaths (as of April 2008).

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. The current H5N1 outbreaks occurring at the same time in several countries, is historically unprecedented and of great concern for human health as well as for agriculture and wildlife.
Bluetongue

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

Bluetongue is a non-contagious, insect-borne, viral disease of ruminants. Bluetongue virus (BTV) belongs to the genus *Orbivirus* in the family Reoviridae. Bluetongue primarily affects sheep and wild ruminants, with asymptomatic infections occurring in cattle, goats, deer, and carnivores. There are 24 serotypes identified worldwide, six of which have been isolated in the U.S. BTV is transmitted by biting midges in the genus *Culicoides*. Ticks or sheep keds can be mechanical vectors, but are of minor importance. While bluetongue is not a contagious disease, the virus can be transmitted to the fetus *in utero* or spread mechanically on surgical equipment and needles. Although BTV can be found in semen, venereal spread does not appear to be a major route of infection. 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 to the southern and western states. 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). In 2006, a serotype 8 virus, which may have come from Africa, caused outbreaks in Germany, Belgium, and the Netherlands. Due to the adaptability of its vector, *Culicoides dewulfi*, to European weather conditions, the virus has the potential to expand geographically into northern Europe.

- Incubation period: 5-10 days
- Sheep
  - Salivation, facial swelling, cyanotic (blue) tongue
  - Reproductive disorders
- Cattle, goats
  - Subclinical; possible mild hyperemia
- Wildlife
  - Hemorrhages, sudden death

The incubation period for bluetongue is 5-20 days. In sheep, the clinical signs may include excessive salivation, 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 resorb the fetus, abort, or give birth to “dummy” lambs. The coronary bands on the hooves are often hyperemic and inflamed, and the hooves are 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 stresses. Morbidity can be as high as 100%; mortality is usually 0-30%, but may reach as high as 70% in susceptible sheep. Infections in cattle and goats are usually subclinical, but may rarely cause mild hyperemia, vesicles or ulcers in the mouth, erosions and crusting around the nose, and hyperemia around the coronary band. In pronghorn antelope and whitetail deer, the most common symptoms are hemorrhages and sudden death. Morbidity rates can be as high as 100%, and mortality usually reaches 80-90% in these two species.

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 low pathogenic avian influenza (LPAI) outbreaks, so prompt detection and response is important. Vaccines for poultry, although fairly expensive, have been used and may be effective for reducing deaths and preventing the disease. The concern with this practice (vaccination) is that protection there is no cross protection between the 15 known HA sub-types. An inactivated H5 vaccine and a recombinant vaccine are licensed in the United States for emergency use in future HPAI eradication efforts. The yearly influenza vaccine available for humans is serotype specific and not likely cross protective to the avian strain.

- Treatment
  - Poultry: none
  - Humans: antivirals
- Control
  - Depopulation
  - Cleaning and disinfection
- Vaccine
  - Poultry: Expensive, no cross protection
  - Human: No cross protection

HPAI: Impact and Response

The yearly influenza vaccine available for humans is serotype specific and not likely cross protective to the avian strain.
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 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 are thought to have occurred from feeding meat or bone meal from scrapie-infected sheep to cattle, or from spontaneous genetic mutation in a cow that was then fed to other cows. This map depicts the countries that have reported BSE from 1989 to June 2006. The countries shaded pink have had BSE in indigenous animals. They include Austria, Belgium, Canada, Czech Republic, Denmark, Finland, France, Germany, Greece, Ireland, Israel, Italy, Japan, Lichtenstein, Luxembourg, Netherlands, Poland, Portugal, Slovakia, Slovenia, Spain, Sweden, Switzerland, United Kingdom, and the United States. (Map accessed at the OIE website on July 10, 2007 http://www.oie.int/eng/info/en_esbcarte.htm)

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 from 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., the wind or sun), kept warm and dry, and given fluids and electrolyte solutions if needed, as well as antibiotics to prevent a secondary infection. Treatment procedures may also include vector control by insecticides, 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, so reasonable precautions should be taken while working with the virus. BTV is not fatal in humans; treatment includes supportive care.

BSE: The Disease

- **Cattle (BSE)**
  - Incubation period: 2-8 yrs
  - Initial signs subtle
  - Final stages
    - Excitable, hypermetria, ataxic, tremors, loss of condition, death
  - Humans (vCJD)
    - Incubation unknown
    - Neurological signs progressing to death
    - 26 years old (mean age of onset)

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 depression. During the final stages of disease, infected animals generally show 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 Creutzfeldt Jakob Disease (vCJD), the incubation period is unknown, but it is likely to be many years or decades. Clinical signs include depression and schizophrenia-like symptoms leading to ataxia and involuntary muscle movement. In contrast to classic CJD, the variant form (vCJD) in the U.K. predominantly affects young people with 26 years as the mean age at the onset of symptoms. The mean duration of infection from the onset of clinical signs 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 estimate of the impact of the first occurrence of BSE in Canada was initially estimated to cost the country and its producers upwards of $2.5 billion dollars, depending on the length of any trade bans. A May 2005 Kansas State University report estimated the economic impact of the first case of BSE in the U.S. In 2003, U.S. beef exports were valued at $3.95 billion and accounted for 9.6% of U.S. beef production. In response to the late December 2003 news that a cow in the U.S. had tested positive for BSE, 53 countries banned imports of U.S. cattle and beef products. These bans included such major markets as Japan, Mexico, South Korea, and Canada. These top four markets accounted for 88% of the value of U.S. beef exports during 2003. These import bans have caused U.S. beef exports to drop; quantities for 2004 declined 82% below the 2003 level. While some important markets, including Mexico and Canada, reopened in 2004, the U.S. did not regain access to the Japanese and South Korean beef export markets in 2004, which were the second and third largest markets for U.S. beef in 2003. If the U.S. had regained access to these two key markets and 2004 exports would have been similar to those in 2003, wholesale revenue per head would have increased between $45 and $66 for every cow slaughtered in the U.S. The KSU economists reported minimal impact on domestic markets from the initial U.S. case and, as of July 2005, it remains to be seen how the first case in an indigenous animal will affect foreign and domestic markets. 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.

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 unpasteurized milk or dairy products, inhalation of infectious aerosols (a means of infection in abattoirs and laboratories), or 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 human patients is an intermittent/irregular fever of 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) includes undulant fevers and arthritis. In the 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, and orchitis. Disease manifests as fistulous withers or poll evil in horses. (Photo courtesy of D. Bickett-Weddle, DVM, ISU.)

Last Modified: May 2008

Animal Disease Emergencies – Local Preparedness

Animal Diseases of Concern
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>
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<tbody>
<tr>
<td>B. abortus</td>
<td>Cattle, bison, elk, horses</td>
<td>Yes</td>
</tr>
<tr>
<td>B. melitensis</td>
<td>Goats, sheep, cattle</td>
<td>Yes</td>
</tr>
<tr>
<td>B. suis</td>
<td>Swine, hares, reindeer, caribou, rodents</td>
<td>Yes</td>
</tr>
<tr>
<td>B. canis</td>
<td>Dogs, other canids</td>
<td>Yes</td>
</tr>
<tr>
<td>B. ovis</td>
<td>Sheep</td>
<td>No</td>
</tr>
</tbody>
</table>

In the 1950s 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 5 days up to three 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 avoidance of 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.)

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. Classical swine fever is found in much of Asia, some Caribbean islands and African countries and much of South and Central America. The disease has been reported in parts of Mexico. The disease has been eradicated from the United States, Canada, New Zealand, Australia and most of western and central Europe. Photo of CSF outbreaks occurring during January through June 2006. From the OIE (World Organization of Animal Health)- World Animal Health Information Database (WAHID) for Jun-Dec 2007. The red, pink and purple areas indicate areas where disease was reported. The green areas indicate areas where CSF was not reported. Humans are not susceptible to CSF infection.
Incubation period: 2
Humans not susceptible to disease
IHSEMD, IDALS, CFSPH
Cattle (European breeds, zebu)
Transmission
Fever, weakness, anorexia,
Control by quarantine, slaughter
Aerosol (close contact)
No treatment
Direct contact
Chronic infections
Transplacental
Can cause death
Morbidity ~100%
Eradicated in Western Hemisphere, UK, Australia
Mortality up to 100%
Bacteria
Buffalo, bison, yak, water buffalo
Vaccine in endemic countries

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.

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.

Mycoplasma mycoides mycoides small colony type (SC type) bacteria is the causative agent of contagious bovine pleuropneumonia (CBPP). CBPP is extremely infectious in cattle, and causes lung and occasionally joint disease. Cattle of the genus Bos, including European breeds 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, and have 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)
CBPP: Impact and Response
- High economic and social impact
  - Zambia, Tanzania, Botswana
  - Drought leads to migration to spread of disease
- Treatment not always effective
- Vaccine available in endemic areas
  - Not always economically feasible
- Humans not susceptible

Contagious Caprine Pleuropneumonia (CCPP)
- Bacterial respiratory disease of goats
  - Mycoplasma capripneumoniae (F38)
  - Mycoplasma mycoides capri
- Transmission
  - Direct contact, inhalation
- Africa, Middle East, Eastern Europe, Soviet Union, Far East
- Not in North America

CCPP: The Disease
- Incubation period: 6-28 days
- Mycoplasma F38 strain
  - Respiratory symptoms
    - Coughing, labored respiration, nasal discharge,
    - Chronic cases: Carriers
  - M. mycoides capri
    - Septicemia, reproductive, intestinal, and respiratory
    - Morbidity 100%; Mortality 60-100%

CCPP: Impact and Response
- Africa and Asia
  - Goats essential to economics
    - Meat, milk, hides
  - Treatment with antibiotics early
    - Newly infected countries
      - Slaughter recommended
    - Vaccine available in some countries
    - Humans not susceptible

Equine Encephalitis Viruses: The Agent
- Eastern (EEE), Western (WEE), Venezuelan (VEE)
  - Viruses transmitted by mosquitoes
- Clinical signs
  - Humans and Equids (horses, donkeys, mules)
    - No to mild signs to flu-like illness
    - Asymptomatic carriers, act as sentinels
  - Birds
    - Asymptomatic carriers, act as sentinels

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 an 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 capripneumoniae (biotype F38) is the most contagious and virulent. Mycoplasma mycoides capri (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 incubation period is often 6-10 days, though it is sometimes as long as 3-4 weeks. Clinical signs of CCPP caused by Mycoplasma F38 strain are distinctly respiratory, and include coughing, labored respiration, frothy nasal discharge (top photo), a very high fever (106°F/41°C), lethargy, and anorexia (bottom photo). Acute cases generally die within 7-10 days. Chronic cases occur when animals have some resistance through previous exposure; these animals are more likely to survive and become carriers. M. mycoides capri infection is often more generalized with septicemia, and the reproductive, gastrointestinal, and respiratory systems are commonly affected. Morbidity is often 100% and mortality ranges from 60-100%. Close confinement increases the spread of disease. Morbidity and mortality are higher with Mycoplasma F38 infection than with M. mycoides capri. (Photos courtesy of www.ivis.org and www.usda.gov)

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.

This is the only viral group in the list of Category B agents. This group of equine encephalitis viruses are RNA viruses in the Alphavirus genus. Eastern, Western, and Venezuelan Equine Encephalitis viruses are transmitted by mosquitoes. The female mosquito takes a bloodmeal from a viremic host, generally birds for EEE and WEE, and birds and horses for VEE. The virus replicates in the salivary glands of the mosquito and is transmitted back to birds or to dead end hosts, such as humans and horses, where overt disease occurs. In humans, infections can be asymptomatic or cause flu-like illness. In a small proportion of cases viral encephalitis can occur and lead to permanent neurological damage or death. Horses, donkeys and mules have similar clinical signs as humans. The disease in these animals often precede human cases by several weeks. EEE and VEE have
mortality rates of 40-90%; WEE has a lower mortality rate ranging from 20-30%. Birds are asymptomatic carriers. The detection of viremia in sentinel birds is detected via ELISA.

### Equine Encephalitis Viruses: The Bioweapon
- Easy to produce
- Aerosolization
- High rate of infection
- Person-to-person transmission possible

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.

### Equine Encephalitis Viruses: The Response
- Supportive care
- Vaccine
  - Equine
  - Human: High risk
- Virus unstable in environment

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).

### Exotic Newcastle Disease
- Virus affecting poultry
- Four disease types
- vND endemic in Asia, Middle East, Africa, Central/ South America
- Outbreaks continue due to illegal importation of exotic birds and poultry

Newcastle disease affects 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. These include: asymptomatic enteric, which is generally subclinical; lentogenic, which has mild or subclinical respiratory signs; mesogenic, which has respiratory and occasional neurologic signs; and velogenic, which is the most virulent pathotype with high mortality rates. Velogenic Newcastle Disease (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. 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 causing decreased egg production. The photo depicts a chicken with respiratory signs and increased salivation due to vND.
Animal Disease Emergencies – Local Preparedness

**END: The Disease**
- Incubation period: 2-15 days
- Drop in egg production, neurological damage
- GI signs, respiratory distress
- Numerous deaths
- Deaths continue for 7-10 days
- Morbidity 100%, mortality 90%

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 psittacines 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.

**END: Impact and Response**
- Most costly poultry disease worldwide
  - 2002-2003: California outbreak
  - $160 million impact
  - Developing countries
  - Affects quality and quantity of dietary protein
  - Vaccine available
  - Human’s can acquire eye infections from contact with virus

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.

**Foot and Mouth Disease**
- Highly contagious virus
- Considered the most important livestock disease in the world
- Not in U.S. since 1929
- Vesicular disease of cloven-hoofed animals
- Spread by aerosol & fomites

An example of an agroterrorism agent that would have severe repercussions is foot and mouth disease (FMD) virus. FMD has not occurred in the U.S. since 1929 and would have great impact on our livestock sector if it did. This picornavirus is probably the most important infection in livestock in the world today. FMD is a highly contagious vesicular disease of cloven-hoofed animals that causes fever and the formation of vesicles in the mouth, on the tongue, muzzle, feet, teats, and vulva. Production losses can be great and death usually only occurs in the young. Sheep and goats often have very mild signs and cases may be missed if not examined closely. FMD can be transmitted by saliva, respiratory aerosol, direct contact, and vehicles (contaminated feed, coveralls, shoes, instruments, etc). It has also been shown that humans can harbor FMD virus in their respiratory tracts for up to two days, posing a theoretical risk for transmitting this agent to uninfected animals. The photo depicts ruptured vesicles on this pig’s leg and coronary band due to FMD. Any case of FMD discovered in the U.S. would need to be reported to the World Organisation for Animal Health (formerly the Office International des Épizooties (OIE) created in 1924) within 24 hours.
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). FMD has not occurred in the U.S. since 1929 and would have great impact on our livestock sector if it did. Photo: Pig foot showing sloughing of the claws. Dr. D. Gregg, Noah’s Arkive, Plum Island Animal Disease Center, CFSPH.

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. Photo: Elongated erosion (ulceration) on cow oral skin – Plum Island Animal Disease Center.

Foot and Mouth disease virus (FMD) only infects cloven-hoofed animals: cattle, pigs, sheep, goats, buffalo, and various wildlife such as deer and elk. FMD could affect approximately 60 species of wildlife and zoo animals and could have an enormous impact on our food animal production. Listed are the animals at risk in the U.S. according to 2001 data. There is also a risk of it spilling over into wildlife and creating a permanent enzootic presence. It is important to note that FMD rarely affects humans. The fear of the economic impact and its affect on trade is the biggest scare associated with FMD. Countries around the world would refuse our exports of beef, pork, mutton, cattle, pigs, sheep, and dairy products. This means that the $3.1 billion in beef exports and the $1.3 billion in pork exports each year would vanish unless we control this disease very quickly. There have been many estimates as to the impact of a FMD outbreak in the U.S. Paarlberg, et al., in their recent analysis of a FMD outbreak in the U.S., estimated that $14 billion would be lost in farm income. This cow is salivating due to the painful vesicular lesions in her mouth making it difficult to swallow.

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.
Glanders is by far the most economically devastating livestock disease virus in the world. This is largely due to the fact that it is highly transmissible, results in economic losses in animal production, and depopulation is the most effective means of control. The USDA has upgraded the safeguarding measures in place to prevent introduction of FMD into the U.S. If FMD is introduced, a response and recovery plan is initiated including a confirmatory diagnosis, quarantine, depopulation, and disinfection. Use of vaccine in an outbreak is a complex decision because it would affect exportation and could potentially cost livestock producers billions of dollars. A decision to vaccinate during an outbreak would be made by collaboration of USDA, state, and local officials. The inactivated virus vaccine is serotype specific and does not prevent infection. It would be costly and possibly ineffective due to the many subtypes circulating worldwide. Under current policy, depopulation of vaccinated animals would still be required before our export markets could be reopened. It is important to note that FMD rarely affects humans and would only cause mild symptoms should it occur.

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

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.
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.

The estimated total annual losses to control heartwater in Zimbabwe were U.S.$56 million. Losses in commercial systems may be 25 times greater than losses in the communal system. The greatest components of economic loss were acaricide 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. Human infections with Heartwater have not been reported. Preventative measures for heartwater include implementation of an effective tick control program, as well as regular inspection of animals and pastures for ticks. Elimination of the vector can be achieved through the use of acaricides; however, acaricide resistance may develop.
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. Occasional outbreaks continue to occur in Australia. Four of the seven human cases of Hendra have 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.

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.

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.
Lumpy Skin Disease

- Viral infection
- Cattle
- Arthropod vector
  - Mosquitoes and biting flies
- Endemic in sub-Saharan Africa
- Peak: Rainy season

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%.

JE: The Disease

- Incubation period: 6-10 days
- Horses
  - Fever and neurologic signs
- Swine
  - Stillbirths
- Humans
  - Fever, headache
  - Fatal encephalitis possible

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.

JE: Impact and Response

- High financial loss in pigs
- No effective treatment
- Vector control measures
- Vaccine
  - Horses and swine
  - Humans

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: Impact and Response

- Severe economic losses
  - Decreased production
  - Secondary infections
- Attenuated vaccine
  - South Africa
- Sheep and goat pox vaccine
  - Kenya, Egypt

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

- Viral infection
- Wildebeest-Africa
- Sheep/goats- N. America
- Susceptible species: Cattle, bison, other wild ruminants
  - Dead-end hosts
- Aerosol or mechanical transmission

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.

MCF: The Disease

- Incubation period: 9-77 days
- Four clinical forms
  - Acute
    - Sudden death
  - Head and eye
  - Fever, necrotic lesions
  - Intestinal
  - Severe diarrhea
  - Mild

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.

MCF: Impact and Response

- High economic losses in exotics
- Mortality near 100% in clinically ill animals
- No effective treatment
- No current vaccine
- Human disease not documented

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.
Melioidosis: The Agent
- *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 https://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)

*Melioidosis* 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.

Nipah Virus: The Agent
- Emerging viral disease in Southeast Asia
  - Fruit bat reservoir
- Malaysia, Singapore
- Bangladesh
- Clinical signs
  - Humans: Encephalitis
  - Pigs: Respiratory, neurological
  - Dogs and cats: "Distemper"

*Nipah virus* infection is an emerging disease endemic in Southeast Asia. Nipah virus was first discovered in during widespread outbreaks in Malaysia in 1998-1999 and caused severe respiratory disease in pigs and severe encephalitis in humans in close contact with infected animals. Some other animal species (dogs, cats) were also affected. The reservoir for the virus is thought to be fruit bats (e.g., 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.

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.

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 serous nasal discharge that becomes mucopurulent and may progress to a severe catarhal exudates that blocks the nostrils causing respiratory distress. The nasal mucous membranes may develop small areas of necrosis and profuse catarhal 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. (Photo:USDA/APHIS)
Q Fever: The Agent
- Bacteria: Coxiella burnetii
- Transmission:
  - Aerosol, direct contact, ingestion, ticks
- Sheep, cattle and goats
  - Can be asymptomatic, abortions possible
- Humans
  - Acute: Flu-like + pneumonia & hepatitis
  - Chronic: Endocarditis, osteomyelitis

Q Fever: The Bioweapon
- History
- Easily accessible
- Environmentally resistant
- Highly infectious
- Aerosolization
  - Travel ½ mile by wind
- Low mortality- chronic morbidity

Peste des Petits Ruminants: Impact and Response
- Economic losses
  - Loss of production, death, abortion
  - Limit trade, export
  - Constraints on availability human consumption
- No specific treatment
- Rinderpest vaccine
  - Protects for 12 months
  - Hinders rinderpest campaign in Africa

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.

Q Fever (“query” or “puzzling” fever) is caused by Coxiella burnetii, an obligate intracellular parasite, which is currently considered a rickettsial agent (new studies may change its family). The disease has been found worldwide, except in New Zealand. Transmission occurs by inhalation or direct contact of infectious organism; it also occurs following ingestion of the of the organism, and ticks spread the infection among ruminants and sometimes people. The organism is shed in high numbers in placental tissue and body fluids, and is highly infectious (one organism can cause disease). There was a report of a case where a cat infected with Q fever had kittens in the same room where a child’s birthday party was being held. Several of the children developed Q fever. Coxiella burnetii forms an unusual spore-like structure and can survive 7-10 days on wool at room temperature, 1 month on fresh meat in cold storage, and more than 40 months in skim milk. However, it is killed by pasteurization. People usually get Q fever by breathing in contaminated barnyard dust. Occasionally people can get Q fever by drinking contaminated milk or from tick bites. Symptoms of Q fever include fever, chills, headache, fatigue and chest pains. Pneumonia (lung infection) and hepatitis (inflammation of the liver) can occur in serious cases. In pregnant women, infections can cause premature delivery, abortion and infection of the placenta. In people with pre-existing heart valve disease, endocarditis (inflammation of the heart valves) may occur. Two clinical forms of disease occur in humans, acute (less than 6 months duration) and chronic (greater than 6 months). Symptoms of acute disease vary in severity and duration and usually manifest as self-limited febrile or flu-like illness, but pneumonia or hepatitis may also occur. Chronic disease occurs in 1-5% of those infected and the most common complication is heart related (endocarditis). Farm animals, including sheep, cattle, and goats, are the most important reservoirs of disease and are usually asymptomatic. Abortions, stillbirths, mastitis in dairy cattle, and complicated deliveries have been reported in these animals. Dogs, cats, rabbits, horses and many other animals can harbor the organism, but is usually asymptomatic.

This agent was part of the U.S. bioweapons research in the 1950’s and 1960’s. Some reports suggest that a portion of the information about the human infectivity of this organism (i.e. one organism can cause disease) was gained during experiments at the bioweapon research facility. This agent could be used as a bioweapon because it is easily accessible, very resistant, highly infectious, and is stable when aerosolized. Coxiella burnetii organisms can be carried up to ½ mile or more by the wind. Mortality is low with this disease. The picture is of a crop duster, and contrary to popular belief, experts believe that wide dissemination could be done with any type of plane, not something that requires intensive training to operate. Image: USDA website.
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.

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.
Animal Disease Emergencies – Local Preparedness

Animal Diseases of Concern

Rinderpest

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

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 and irregular pyrexia and mild or no diarrhea. Immunosuppression can lead to secondary infections and the emergence of latent infection.

Rinderpest: Impact and Response

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.

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.
**Slide 8**

**Screwworm Myiasis**
- Exotic fly larvae
- 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.

**Slide 9**

**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.

**Slide 90**

**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 reinfection. 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

**Slide 91**

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

Sheep pox and goat pox are contagious viral skin diseases classified with lumpy skin disease virus in the genus *Capripoxivirus* (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.

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).

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 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
- Resembles FMD
- Fever, salivation, lameness
- Blisters
  - Snout, mammary gland,
- 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 upon recovery. Virus can also be shed in the feces for up to three months following infection. Photos: Top-Multiple large ulcers on feet of pig (Source: Plum Island Animal Disease Center); bottom-deep ulcer on the snout of a pig (Source: Iowa State University, College of Veterinary Medicine). From www.usda.aphis.gov.

SVD: Impact and Response

- Control measures costly
- Export restrictions
- Supportive care
- Vaccine not commercially available
- Human infection not common
  - Incubation period: 1-5 weeks
  - Mild influenza-like symptoms
  - Vesicular lesions not seen

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.

Tularemia: The Agent

- Sheep, young pigs, horses, dogs, cats
  - Sudden fever, lethargy, stiffness, prostration, and death
- Wildlife
  - Usually find dead
  - Rabbits behave strangely
- Cattle, older pigs resistant

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 Agent

- Francisella tularensis
- Transmission
  - 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. Ulceroglandular occurs when conjunctiva become infected by rubbing eyes with contaminated fingers or by splashing contaminated materials in the eyes. The ophthalmoglandular presentation is caused by ingestion of organism in contaminated food (undercooked meat) or water. Typhoidal and pneumatic 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; Image from CDC website: http://www.cdc.gov/ncidod/eid/vol8no1/01-0131.htm; Ulcer caused by tularemia. (http://phil.cdc.gov/Phil/results.asp?page=1)
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 50 kg 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 given early or before exposure
- 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)

Vesicular Stomatitis

- Viral infection
- Horses, donkeys, cattle, swine, South American camelids
- Arthropod-borne, direct contact, aerosol
- Morbidity 90%, mortality low
- Southwest United States

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

VSV: The Disease

- Animals
  - Oral/mammary/coronary band lesions, salivation, lameness
    - Resembles FMD
  - Recovery in 2 weeks
- Humans
  - Incubation period 1-6 days
  - Influenza-like symptoms
  - Oral lesions rare
  - Self-limiting

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. [http://www.aphis.usda.gov/vs/ep/fad_training/VESVOL7/page105_7.htm](http://www.aphis.usda.gov/vs/ep/fad_training/VESVOL7/page105_7.htm) This concludes the review of the additional High Consequence Livestock Pathogens.

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