Reportable and Foreign Animal Diseases

OBJECTIVES

To demonstrate mastery of Module Subject the trainee will:

1. From a list of animal diseases, be able to select those which are reportable.

2. Be able to recognize clinical signs and/or lesions suspicious of a reportable or foreign animal disease.

3. Know the appropriate procedures to follow when a reportable or foreign animal disease is suspected in an animal presented for slaughter.

4. Be able to properly identify and submit possible lesions of bovine tuberculosis for identification.

5. Be able to follow appropriate procedures when TB reactors, suspects, or exposed animals are presented for slaughter.

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I. Introduction:

As a FSIS Public Health Veterinarian (PHV) in a slaughter facility, you have the responsibility of conducting ante mortem and postmortem inspection on up to thousands of animals each day. For this reason, you play a valuable role in detecting reportable and foreign animal diseases. This module will focus on the significance of reportable and foreign animal diseases, clinical and pathological diagnosis of significant disease conditions, and procedures to report suspected reportable and foreign animal diseases. As a FSIS PHV, you can play a valuable role in detecting and assisting in the control and eradication of reportable and foreign animal diseases.

FSIS Field Operations (OFO) cooperates with Veterinary Services in their various activities and plays an important role in the disease eradication program that Veterinary Services administers. The intent is not to make you an expert at recognizing by name the various reportable diseases when seen, but rather to make you aware of your responsibility to report abnormal symptoms and lesions Veterinary Services (VS).

Your work in the packing plant is very important to the animal disease eradication effort because you work at a place in the food animal chain where often you are the first to encounter a disease process in an animal.

Remember that you are the first line of defense in bringing to the attention of your Public Health Veterinarian any symptoms seen on ante mortem or lesions seen on postmortem that could be part of a disease entity that should be reported.

Veterinary Services (VS) and OFO are both in the U.S. Department of Agriculture. VS, however, is a discipline of the Animal and Plant Health Inspection Service (APHIS), while FO is a discipline of the Food Safety and Inspection Service (FSIS). The overall mission of VS as a regulatory agency is to administer an important part of the animal health program of our nation. Primarily this means controlling or eradicating specified animal diseases already in this country. Since VS has so few personnel compared to OFO, it becomes very important that OFO food inspectors at the packing plant serve as vigilantes in discovering unusual symptoms or lesions.

Reportable diseases are those that are designated by World Animal Health Organization (Office International des Epizooties or OIE). When suspected, either on ante mortem or postmortem, they must be reported to your Public Health Veterinarian. The list of reportable diseases include anthrax, bluetongue, bovine spongiform encephalopathy (BSE), cysticercosis, scabies, tuberculosis, contagious ecthyma, myiasis (screwworm), scrapie, and vesicular diseases. Of these diseases anthrax, cysticercosis, tuberculosis, and contagious ecthyma are transmissible to humans.

Emergency diseases are defined as those foreign animal diseases that are not currently found in this country. They are classed also as *reportable* diseases, but reportable diseases of

especially profound significance. The list of emergency diseases includes foot and mouth disease, rinderpest, African swine fever, hog cholera (classical swine fever), contagious bovine pleuropneumonia, and Teschen's disease.

II. Reportable and Foreign Animal Diseases:

A. Notifiable Diseases

The United States Department of Agriculture (USDA) Animal and Plant Health Inspection Service (APHIS) follows standards and rules in concert with the World Organization for Animal Health (Office of International Epizooties or "OIE"). As a member country, the United States monitors animal diseases from a list of "Notifiable diseases" that is generated by OIE.

The OIE is an intergovernmental organization that was created by the International Agreement of 25 January 1924. This agreement was signed by 28 countries. Over the years OIE has grown considerably and in May 2004, the OIE totaled 167 member countries.

OIE member countries report animal diseases that are detected on its territory to OIE, and the OIE then disseminates the information to other countries. This dissemination of information allows neighboring countries to take the necessary preventive actions to minimize the chances of the disease entering into their country. This information also includes diseases transmissible to humans and intentional introduction of pathogens. Information is sent out immediately or periodically depending on the seriousness of the disease. This objective applies to diseases or natural origin as well as those that have been deliberately introduced. Dissemination is via the OIE Web site, e-mail and the following periodicals: *Disease Information*, published weekly and the annual compilation *World Animal Health*.

The current list of diseases that are reportable to the OIE are included at the end of this module. The list may also be found at the following weblink:
http://www.oie.int/eng/maladies/en_classification.htm

B. Critical Foreign Animal Disease Issues for the 21st Century

Animal health officials define an exotic or foreign animal disease (FAD) as an important transmissible livestock or poultry disease believed to be absent from the United States and its territories that has a potential significant health or economic impact. The USDA - APHIS is working vigilantly with State animal health officials and veterinary professionals to identify, control, and eradicate these animal diseases and diminish their impact. As a preface to the updated disease information, this introductory article will provide an overview of the ways in which FAD's may impact U.S. consumers and producers. It will also highlight the new challenges facing those involved in prevention, management, and recovery from FAD threats to the United States.

IMPACTS OF FAD's ON THE U.S. ECONOMY

Foreign animal diseases are considered a threat to the United States when they significantly affect human health or animal production and when there is an appreciable cost associated with disease control and eradication efforts. Diseases such as classical swine fever (hog cholera), foot-and- mouth disease (FMD), and highly pathogenic avian influenza (HPAI) can cause high

death rates or severe illness and production losses. This loss of productivity can increase the cost of food products obtained from those animal sources. For example, during the 1983-84 outbreak of HPAI, the average cost of one dozen eggs increased by 5 percent (1). McCauley et al. predicted that the price of beef would increase by $0.19 per pound because of an outbreak of FMD (2). Other diseases such as tuberculosis (TB) and brucellosis affect human and animal health. These two diseases, although very prevalent in other countries, will soon be eradicated from U.S. domestic livestock and will thus become exotic.

To protect the long-term health and profitability of U.S. animal agriculture, incursions of a FAD must be rapidly controlled. In the United States, control usually means disease eradication. These eradication efforts can present significant short-term costs to industry and government. For example, in 1983-84 the control and eradication of a highly pathogenic avian influenza outbreak cost the USDA $60 million. In the final stages of hog cholera eradication (1971-1977), the U.S. government spent $79 million (3).

In addition to control costs, one of the most immediate and severe consequences of a FAD occurrence in the United States will be the loss of export markets. U.S. animal agriculture is becoming more dependent on exports. The long-term strategic plans of these industries call for increasing the amount of goods sold abroad. As the percentage of total production destined for export grows, the impact of a domestic FAD outbreak also grows. Other countries will not allow the importation of animals or animal products that pose a risk to their industry. In 1997, the total value of exported U.S. animals and animal products exceeded $7 billion: $2.3 billion in poultry, $1 billion in pork, and $2.6 billion in cattle and cattle products. Theoretically, the long-term trade impacts of a FAD occurrence can be reduced by applying regionalization concepts. A country could, during a FAD outbreak, recognize specified regions of the United States as affected with the disease. The remaining unaffected areas might be free to continue exporting. However, it would take considerable time to have these regions identified and other regions certified as disease-free. In the meantime, all trade in that commodity would be stopped.

NEW CHALLENGES FOR THE MANAGEMENT OF FAD's

As we move into the 21st century, many new issues and factors are affecting FAD prevention, control, management, and recovery. These factors include free trade agreements, free trade blocks, regionalization, increased international passenger travel, intensification of animal production, the constant evolution of infectious agents, and the uncertain impact of biotechnology and bioterrorism.

Evidence is accumulating that these factors are having an impact. For example, hog producers in Taiwan recently experienced a devastating outbreak of FMD for the first time since 1929. Over four million animals were destroyed. Virtually all export markets were lost. The Netherlands recently sustained an outbreak of hog cholera that resulted in major export losses of 65 percent of their production. Other countries in the European Union struggle to eradicate hog cholera. As this book goes to press, hog cholera is active in the Dominican Republic, which is situated only 150 miles from the continental United States.

The world is moving toward more open market access. Free trade agreements such as GATT (General Agreement on Tariffs and Trade) and NAFTA (North American Free Trade Agreement) stipulate that trade in animals and animal products should only be restricted if there is a valid human or animal health risk to the importing country. To stop trade, the importing country must show, with a scientifically valid analysis, that a risk exists. This policy will increase responsibility

for the United States to evaluate risks carefully. It also will probably increase the flow of animals and animal products into the United States.

A related element of free trade agreements is the concept of regionalization. As an importing country, the United States is required to evaluate geographic regions of potential importers. More effort and information will be required for the United States to evaluate the risk of a disease from a region that may be smaller than or larger than an area defined by political borders. The United States must have some methods to evaluate the security of the region's boundaries. The acceptance of regionalization puts increased pressure on the United States to remain vigilant for the presence of disease at home and in various countries exporting or hoping to export, to our shores. Examples of regionalization include recognizing the northern U.S. states as Bluetongue free, northern Spain as free from African horse sickness, and portions of Argentina as FMD free.

Around the world countries are joining into free trade blocks. They hope these alliances will give them a competitive advantage against other trading blocks such as the European Union and the NAFTA countries. Problems arise as livestock or animal products are allowed to move freely within these blocks because we may not always know the origin of the products we import.

The volume of international passenger travel is steadily increasing. In 1980, 20 million passengers arrived in the United States on international flights. In 1995, this number rose 131 percent to 47 million (4). The airline industry expects this trend to continue. International travelers may unknowingly bring contaminated animal products from FAD infected countries. Contaminated foodstuffs have often served as a source of a FAD in the United States and other countries (5).

As the world population grows and animal production intensifies, the risks and impacts of FAD incursions increase. Today, infection at one premises can affect 300,000 laying hens, 100,000 hogs, or 100,000 feedlot cattle. When one company owns a large number of animals, frequent and rapid interstate movement occurs. This movement can spread infection across many states before clinical signs are manifest in the source herd.

Lastly, the infectious disease agents and vectors are changing. For example, as the importation of reptilian pets increases, potential disease-transmitting vectors such as Amblyomma ticks are finding new routes of entry. Also, natural selection pressures predict that the FAD of the next decade will be different from the last. Recent examples include the swine-specific FMD virus in Taiwan, *Salmonella DT104*, and *Salmonella enteritidis*. Actions and information that accurately prevented disease or predicted risk in the past may not be effective in the future. Around the world, new agents never before a threat to U.S. agriculture have become an important human health or economic concern. Examples include bovine spongiform encephalopathy and porcine reproductive and respiratory syndrome. Today's new emerging disease may be tomorrow's significant exotic disease.

U.S. RESPONSES TO CHANGING EXOTIC ANIMAL DISEASE THREATS

The Animal and Plant Health Inspection Service has taken the lead in publishing a rule on regionalization expectations. This rule will contribute to international negotiations on animal trade. To define optimal methodologies for conducting risk analyses, APHIS is working with universities, consultants, and the Economic Research Service (ERS). Also, APHIS is beginning

to educate animal health officials, the animal agricultural industry, and our trading partners about the concepts and impacts of regionalization.

Disease surveillance data are a critical element for early FAD detection and for accurate risk analyses. Consequently, APHIS is constantly exploring different methodologies for monitoring the health of the U.S. livestock and poultry population. As traditional program diseases such as tuberculosis and brucellosis are eradicated and funding decreases, new surveillance systems will be needed. The U.S. animal health surveillance systems are therefore being reviewed by APHIS to achieve the highest efficiency and breadth without compromising disease detection abilities. Also, APHIS is working with our Latin American trading partners to design feasible surveillance systems for the region. In protecting American agriculture, APHIS is playing a key role in collaborating with international health organizations such as OIE (Office of International Epizootics), IICA (Inter-American Institute for Cooperation on Agriculture), FAO (Food and Agriculture Organization), and others to harmonize trading regulations, risk analysis methods, disease surveillance, and diagnostic methods.

The USDA, state animal health officials, universities, and the animal agricultural industry are taking many steps in response to these changing threats and risks. The diagnostic laboratory system is constantly improving and applying state of the art technology for FAD diagnosis and differentiation. International contacts are used to maintain awareness of disease occurrence. Consolidating the Agricultural Research Service (ARS) and APHIS and remodeling laboratory facilities at Plum Island will strengthen the opportunities for collaboration on FAD research and diagnostic programs.

The emergency management plan is being revised with greater involvement of partners to ensure rapid detection and response. These efforts are discussed in Part III, Protecting Livestock and Poultry Industries from Foreign Animal Diseases, in “Foreign Animal Diseases” published by the United States Animal Health Association, Richmond Virginia, 2004. Veterinary Services (VS) has downsized just like other U.S. government agencies. In that process, VS has gone from four regional emergency response teams to two. However, in doing this, VS has also created small Rapid Response Teams that can quickly be deployed to investigate possible FAD outbreaks. Additionally, VS is working more with State departments of agriculture, private veterinary practitioners, and other veterinary specialty groups to formulate better responses to these new threats. Moreover, VS has been examining the distribution of specially trained diagnosticians to determine any needed changes to improve the availability of these individuals. Key diagnosticians to be sent to outbreaks in other countries have also been identified by VS. This adds to our current knowledge base of the disease outside the laboratory and of the real-life problems involved in control and eradication.

Finally, VS has made efforts to create a manageable data base to collect information on all potential FAD investigations. This begins by having the diagnostician corps enter the most accurate and inclusive data into a computer data base. The future goal is to be able to look at trends and give values back to the reporting producer and veterinary practitioner. The trends may help VS to distribute and train its corps of diagnosticians better. It is hoped that the returned added value will stimulate more reporting by the private sector.

CONCLUSION

Exotic or emerging animal diseases continue to threaten the health and productivity of U.S. livestock and poultry. All of those with the potential of being affected are working to manage these threats by responding to these new challenges.

By Joan M. Arnoldi, D.V.M., M.S in "Foreign Animal Diseases", published by United States Animal Heath Association, Richmond Virginia, 2004

GUIDE TO THE LITERATURE

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2. McCAULEY, E. H., AULAQI, N.A., NEW, J.C., SUNDQUIST, W.B., and MILLER, W. M. 1979. A Study of the Potential Economic Impact of Foot-and-Mouth Disease in the United States. University of Minnesota, United States Department of Agriculture, Washington, D.C.: U.S. Government Printing Office.

3. WISE, G. H. 1981. Hog Cholera and Its Eradication: A review of U.S. Experience. United States Department of Agriculture, Animal and Plant Health Inspection Service. Washington, D.C.: U.S. Government Printing Office.

4. National Transportation Statistics (NTS). 1997. http://www.bts.gov/btsprod/nts/acp.html. United States Department of Transportation.

5. Risk Assessment of the Practice of Feeding Recycled Commodities to Domesticated Swine in the U.S. 1995. United States Department of Agriculture, Animal and Plant Health Inspection Service. Washington, D.C.: U.S. Government Printing Office.

C. Protecting Livestock and Poultry Industries from Foreign Animal Diseases

Protecting the livestock and poultry industries of the United States from foreign animal diseases (FAD's) involves four basic principles or phases of emergency management. They are prevention, preparedness, response, and recovery. To be effective, these principles require the support and cooperation of persons, groups, and organizations at the local, State, regional, and national levels. Livestock and poultry owners, veterinarians in private clinical practice, industry groups, the Federal government, State government, State universities, veterinary diagnostic laboratories, and the traveling public must all be included.

PREVENTING THE INTRODUCTION OF FOREIGN ANIMAL DISEASES

The responsibility for preventing the introduction of FAD' into the United States has been assigned to several Government agencies. The U.S. Department of Agriculture, Animal and Plant Health Inspection Service (APHIS) (Fig. 1) has the primary responsibility for preventing the introduction of FAD's through importation regulations governing animals, poultry, and animal and poultry products. To accomplish this objective, APHIS cooperates with other Federal agencies, including the Department of Homeland Security, the U.S. Customs Service, the U.S. Fish and Wildlife Service, the U.S. Department of Agriculture's Food Safety and Inspection Service, and State Animal Health Agencies.

The port inspectors of Plant Protection and Quarantine (PPQ), originally part of APHIS, but now a part of the Department of Homeland Security (DHS), Customs and Border Protection (CBP) is responsible for inspecting ships and planes and their cargo, private and commercial vehicles, and passengers and their luggage arriving from foreign countries. Working closely with their fellow inspectors originally from U.S. Customs Service, this unit intercepts animals, poultry, animal and poultry products, and disease vectors at U. S. ports of entry.

Veterinary Services (VS) within APHIS administers laws and regulations pertaining to the importation of animals, poultry, pet birds, semen, embryos, hatching eggs, and other animal products to ensure that those imported from foreign countries are free from certain disease agents.

International Services (IS) within APHIS cooperates with its counterparts in foreign countries to reduce the international spread of animal and poultry diseases. The focus is to protect U.S. livestock and poultry by reducing the disease risk through participation in disease-management strategies before animals and poultry are imported into the United States.

State Animal Health Agencies, especially those located along the international borders with Canada and Mexico, cooperate with all regulatory entities at the land ports-of-entry. Among their activities are stray animal control, providing inspection services when imported animals are re-assembled after importation, and assistance with notification of livestock movement to receiving states.

PROTECTING THE LIVESTOCK AND POULTRY INDUSTRIES FROM DISEASE INCURSIONS

The responsibility for rapidly detecting and effectively responding to incursions of FAD's is primarily that of the livestock and poultry owners, veterinarians in private clinical practice, personnel from the State Animal Health Agency , APHIS, and the local community where the outbreak is occurring. The State Animal Health Official, usually the State Veterinarian, and the USDA, APHIS, VS Area Veterinarian in Charge (AVIC) and their staff routinely conduct surveillance activities to detect any FAD outbreaks quickly. These activities require the support of state veterinary diagnostic laboratories, the Cooperative Extension Service of the USDA, State and Federal meat and poultry inspection services, animal scientists, livestock market operators, and again, livestock and poultry producers and their private veterinarians.

To detect FAD outbreaks early, suspicious signs of a FAD must be promptly reported to the State Veterinarian, the AVIC , or both. Private veterinarians in clinical practice are conversant with the occurrences of domestic animal diseases in their area and are likely to be the first to suspect the presence of a FAD. Prompt reporting of suspicious signs will enable responsible agencies to conduct an investigation, obtain a diagnosis, and contain a FAD outbreak before it spreads.

When suspicious FAD cases are reported, an investigation of the affected herd or flock is immediately conducted by a specially trained FAD diagnostician (FADD). There are over 500 State and Federal FADD's throughout the United States, strategically located so that a suspicious case anywhere in the U.S. can be investigated within 8 hours of notification. On the basis of history, signs, lesions, and species involved, specimens are collected and submitted to the National Veterinary Services Laboratories (NVSL), Ames, IA, or to the Foreign Animal

Disease Diagnostic Laboratory (FADDL), Plum Island, NY, to confirm the presence or absence of a FAD.

On the basis of initial FAD investigation findings, often before the laboratory has completed testing of the samples, State and Federal officials in the affected state(s) will take action to quarantine suspect animals or poultry, increase area surveillance, restrict movement of animals and initiate steps to characterize and control the outbreak. An Early Response Team (ERT) composed of a senior FAD diagnostician, a senior laboratory pathologist from NVSL, and a senior epidemiologist can be called upon to provide greater technical assistance in the investigation, further assessment of the situation, and assistance in identifying needs of local officials to combat the problem.

LEADERSHIP, PARTNERSHIP, AND MEMORANDUMS OF UNDERSTANDING

Veterinary Services has the critical leadership role for the rapid detection of and the effective response to incursions of potentially devastating FAD's. However, in the event of an international introduction of an FAD, the Department of Homeland Security will assume the lead role to manage law enforcement aspect of the disease but will cooperate with VS and State Animal Health Officials to control and mitigate the outbreak. Veterinary Services is also responsible for providing FAD training. In conjunction with State Animal Health Officials, they help maintain an awareness of FAD threats and conduct test exercises. State Animal Health Agencies implement their state emergency response plan and initiate the response. VS Emergency Management oversees submission of samples for FAD's and coordinates further Federal; response activitgi4es to the outbreak.

Veterinary Services has established Memorandums of Understanding (MOU's) to obtain resources and cooperation from State animal health and wildlife agencies and the Department of Defense. Wildlife specialists from all 50 States and the Commonwealth of Puerto Rico have agreed to assist in FAD's involving wildlife. In addition, MOU's have been signed with State veterinary diagnostic laboratories to provide for FAD surveillance and laboratory support in the event of an outbreak.

The National Animal Health Laboratory Network supports diagnostic activities at NVSL and FADDL and is designed to provide additional surveillance capacity, and in the event of an outbreak of an FAD, additional surge capacity. This network incorporates a number of state diagnostic laboratories that have capabilities for rapid nucleic acid-based testing for some of the more devasting FAD's.

EMERGENCY RESPONSE TO A FAD OUTBREAK

When field investigations and laboratory tests confirm that a FAD is present, VS will immediately activate a response. If the FAD is on the OIE disease list for immediate notification, USDA is obligated to notify OIE within 24 hours. The OIE then transmits the news to all member nations.

USDA, APHIS, VS is in the process of transitioning a state based, nationally coordinated Animal Emergency Response Organization (AERO). AERO's will be central in the emergency management of an outbreak.

The emergency response follows guidelines set out in the National Incident Management System (NIMS) The NIMS was created as a result of Homeland Security Presidential Directive 5 and is designed to address all hazards, including FAD's. It formalizes the Incident Command

System (ICS) developed by the Forest Service in the 1970's to mobilize resources and people in the management of forest fires. ICS is composed of five major sections and is highly flexible with sections growing or shrinking depending on the extent of the outbreak and its complexity. It can involve individuals from a number of different agencies and is designed to streamline activities, maximize resources, and clarify chains of command. For an FAD outbreak, the incident command structure might include veterinarians, technicians, disease specialists and support personnel drawn from the military, universities, industry, private practice, as well as state and federal governments.

The five majors sections of Incident Command Management are: Command, Finance, Logistics, Operations and Planning. Duties and responsibilities of the sections are outlined below.

The Command Section is led by a single Incident Commander and this section controls all personnel and equipment, maintains accountability for task accomplishment, and serves as a liaison with outside agencies. In the event of an ICS for an FAD outbreak, the Incident Commander position would be occupied, at least initially, by the State Veterinarian and the AVIC.

The Planning Section is responsible for creating the Incident Action plan. The incident Action plan defines the response activities and use of all resources. During an FAD outbreak response, the Planning section will also be concerned with animal welfare issues, formulatio9n of a vaccination plan, epidemiology, wildlife interaction, laboratory coordination, Geographic Information System (GIS), development of surveillance plan for the control area, and reporting of the disease outbreak situation.

The Operations Section carries out the operational aspects of the response primarily on the affected premises. The response activities and duties here include quarantine, vector control, euthanasia, disposal of carcasses, and further disease detection activities as called for in the surveillance plan.

The Logistics Section is responsible for providing facilities, materials, and services. Functions of this section are geared toward addressing the needs of the responders themselves.

The Finance Section manages the expenditures required by all sections and participants to respond to a disaster.

From: "Foreign Animal Diseases" published by the United States Animal Heath Association, Richmond Virginia, 2004

USDA - APHIS RESPONSIBILITIES

The primary agency within the USDA that will address animal disease and plant disease events is the Animal Plant Health Inspection Service (APHIS). Events occurring on the farm or at land, sea or air port are a USDA APHIS responsibility. APHIS supplies border inspections, animal import testing, and quarantine, as well as training of veterinarians for Foreign Animal Disease (FAD) detection. Each year, APHIS oversees over 850 FAD investigations, with usually only 1-2 responses.-

- **Veterinary Services (VS)** has the primary responsibility to protect, detect, contain, and eliminate foreign animal diseases and promote animal health in the United States. VS has 45 state offices in the United States and Puerto Rico.

    - Federal Veterinarians. Most states have an area veterinarian in charge (AVIC) and a variable number of veterinary medical officers (VMO) to investigate possible disease events. The VS personnel work closely with the State Veterinarian to protect animal health. The AVIC for your area can be found at http://www.aphis.usda.gov/vs/area_offices.htm. An updated list of AVIC's can also found at the end of this module.

    - Foreign Animal Disease Diagnosticians (FADD) are either State Department of Agriculture or USDA APHIS VS veterinarians who have received specialized training in the detection of FADs. Upon notification of a suspicious disease, an FADD will be sent to the site within 8-16 hours.

    - The USDA APHIS VS operates the National Veterinary Services Laboratory (NVSL) in Ames, Iowa and the other is the Foreign Animal Disease Diagnostic Laboratory (FADDL) at Plum Island, New York. These diagnostic laboratories perform laboratory testing to diagnose and confirm all FAD suspect samples.

    - AERO (Animal Emergency Response Organization),are state-level organizations that involve cooperative agreements between states and the USDA APHIS VS as a response system using ICS that will enhance disease surveillance and emergency preparedness capabilities. Personnel are trained animal health emergency managers and can be mobilized to support and fight an outbreak. They may include veterinarians, technicians, disease specialists, and administrative or clerical personnel.

Local

The local veterinarian, producers, FSIS public heath veterinarians are usually the first line of defense in the event of an FAD. Prompt detection of unusual animal behavior or clinical signs by the producer is essential.

State and Federal

If an FAD or suspicious event is suspected, the local veterinarian or FSIS public health veterinarian should immediately contact the AVIC. The AVIC notifies the State Veterinarian. The federal and state veterinarians will operate in tandem on all diagnostic and control issues. A Foreign Animal Disease Diagnostician (FADD) will then be sent to the site to assess the situation and collect samples for disease diagnosis. Based on their findings and laboratory results, a designation will be placed for the animal.
Case classifications include:

- Suspect – Animal with clinical signs, which may be consistent with an FAD.

- Presumptive Positive Case – Animal with clinical signs consistent with FAD and positive lab results and epidemiology are indicative of the FAD.

- Confirmed Positive – Animal with clinical signs consistent with FAD and from which the FAD agent was isolated and identified in a USDA lab or other laboratory designated by the Secretary of Agriculture.

Based on the case classification selected, appropriate control measures will be taken. Actions will depend on several factors such as the FAD suspected or confirmed, the animal species affected, and the economic impacts and results from risk analyses.
Once final laboratory results are obtained, additional actions will be taken at local, state and federal levels. A positive identification of an FAD (confirmed positive) may lead to additional animal quarantines of contiguous premises, animal movement restriction orders, and euthanasia actions.

Depending on the disease agent, scale of the event and level of spread of the disease, additional federal agencies may become involved. The U.S. Secretary of Agriculture has authority to declare an emergency, which provides federal resources and funding for the situation. The National Response Plan may be initiated depending on the level of response needed.

Additionally, within 24 hours of confirmation of an FAD, OIE is notified by the chief veterinary officer in APHIS.

D. FSIS Responsibilities

FSIS Directive 6000.1 provides instruction to FSIS Public Health Veterinarians when they suspect that animals may have a foreign animal disease (FAD)., or when PHVs observe symptoms of FADs or other reportable diseases.

If a reportable or FAD is suspected, inspection program personnel should find out as much history as possible. This may be gathered from animal records, ante-mortem pen cards, verbal information from driver, or any other source of information or materials. Much of this information may not be available; however, if information of this type is available, it needs to be accurately passed on to the District Office (DO).

Signs of FADs

If program personnel observe the following signs or findings, or come across the following information relative to animals presented for slaughter, FAD may be suspected:

- high morbidity;
- high mortality;
- severe abortion storms of unknown etiology;
- avian disease with acute deaths or central nervous system (CNS) signs;
- history of foreign travel; foreign visitors; foreign mail or gifts; or importation of animals, embryos, or semen.

Ante-mortem signs:

- that do not fit with the typical conditions for a specific disease
- vesicular lesions;
- severe respiratory conditions;

- pox or lumpy skin conditions;
- CNS diseases (or undiagnosed encephalitic conditions);
- mucosal diseases;
- larvae in wounds;
- unusual myiasis or ascariasis; or
- unusual or unexplained illness or symptoms.

Postmortem conditions such as:

- hemorrhagic septicemia;
- suspicious or unusual post-mortem (necropsy) findings;
- findings that do not fit with the typical conditions for a specific domestic disease.
- Lesions such as necrotic foci on tonsils, enlarged spleen, or hydro pericardium, which may be seen in some domestic diseases, if coupled with suspicious information (antemortem findings, records, etc.) should warrant further investigation.

PHV Responsibilities

If PHVs observe animals exhibiting the signs described above, the animals are to be considered "U.S. Suspects" under 9 CFR 309.2. PHVs are to notify the DO, as soon as possible, when they suspect that any undiagnosed or unusual disease condition is reportable, foreign, or both. This module will cover the conditions and symptoms associated with FADs. The APHIS Foreign Animal Disease CDs should be reviewed for pictures and movie clips of these conditions. Extra copies of the CDs may be acquired from the Center for Learning.

PHVs are to gather, and provide to the DO, as much of the following information as possible:

- producer's name, address, county, and phone number;
- any clinical history, including any treatments given and responses noted from the certification accompanying the animal;
- number and species of animals affected that were presented for slaughter;
- what diseases or conditions are suspected to be present;
- any gross lesions seen;
- the PHVs contact information, including name, address, and relevant phone numbers.

The DO will notify the APHIS Area Veterinarian-in-Charge (AVIC), the State Animal Health Official (SAHO) or both, and provide the information gathered by the PHV.

In most cases, APHIS or the State Animal Health Official will want the animal held so they can examine it. The veterinarian will have the animal placed in a separate pen identified with a pen card. The establishment employees will be notified that the animal is not to be removed from the pen for any reason without the permission of the veterinarian or some other animal health official.

The State Animal Health Official or APHIS AVIC will determine how the case is to be handled and give the DO specific instructions at that time. If it is determined that an investigation is warranted, a Foreign Animal Disease Diagnostician from APHIS or the State will be assigned.

There are two types of animals specially identified before being sent to slaughter that you need to be familiar with: TB reactors (tuberculin reactors) and brucellosis reactors. These animals may show no abnormal signs; however, they still require your special attention. Details on how

to handle TB reactors and brucellosis reactors will be covered under subsequent section of this training.

Lists of the current AVIC and State Veterinarians are provided at the end of this module. They may also be located at the following web links:

For State animal health offices see: http://www.aphis.usda.gov/vs/sregs/official.html
For APHIS AVIC offices see: http://www.aphis.usda.gov/vs/area_offices.htm

E. Bovine Tuberculosis

Incidence and Etiology

Tuberculosis is a reportable disease, which has not been eradicated from the United States. It is still seen in imported animals, wildlife, and some livestock herds in California, New Mexico, Texas, and Michigan. The TB eradication program depends heavily on the efforts of meat inspection, and all granulomas of unknown origin should be submitted for analysis. Your submission of positive tuberculosis lesions assists APHIS Veterinary Services in eradicating Tuberculosis from American U.S. cattle herds.

Bovine tuberculosis is caused by *Mycobacterium bovis*, and in some cases, *Mycobacterium avium*. *Mycobacterium tuberculosis* is the species most often implicated in human cases of tuberculosis, although *M. bovis* can cause human disease. *M. avium* can cause disease in cattle and in swine.

Mycobacterium species cause granulomas. Most *M. bovis* granulomas are found in the thorax and in lymph nodes, but can also be found in the liver, spleen, and mesentery. Tuberculosis granulomas can be granular to pyogenous in nature. Not all pyogenous granulomas are “acti”. Complete incision of all lymph nodes is essential for identification, because some granulomas can be very small.

Tuberculosis is an ancient disease, as evidence of bovine tuberculosis has been found in Egyptian mummies. The eradication program was started in the U.S. in 1917 when 5% of the nation's cattle were said to be TB-infected. As a matter of fact, 50,000 cattle carcasses were condemned for the disease that year alone.

Today, bovine TB is more prevalent in beef cattle than in dairy cattle, probably due to the early emphasis on eradication in the dairy breeds. The bovine TB eradication effort is becoming more dependent on efforts of food inspectors, since routine testing of cattle for TB has been de-emphasized.

Granulomas in Regular Kill Animals

Now, let's explore the methods by which you as an FSIS Public Health Veterinarian cooperate with VS in the TB eradication effort. Let's suppose you are performing postmortem inspection on cattle viscera and you find a lung lesion that *could* possibly be TB. What would be your action?

Your first action would be to retain the carcass and all its parts, including the lesions. As a part of this step you would want to collect and coordinate any identification information pertaining to the animal such as backtags, eartags, sales tags, etc. M-branded Mexican cattle will have a blue metal ear tag. Plant personnel are required to collect all man made identifications from such animals and attach them to the carcass. Mexican cattle have a higher incidence of TB granulomas than do U.S. origin cattle.

Subsequently, lesions should be sent to the National Veterinary Services Laboratory (NVSL) in Ames, Iowa, for confirmation or non-confirmation of suspicions. Granulomas must be divided into the two bottles provided: one is for histopathology and the other for bacteriology. The VS Form 6-35, "*Report of Tuberculous Lesions or Thoracic Granulomas in Regular Kill Animals*" would be utilized if these specimens were submitted to the laboratory.

If specimens are found by the laboratory to be positive for TB, then VS, with the aid of identifying information OFO has given them, can accomplish traceback to the herd of origin. This is and will continue to be the "backbone" of the TB eradication program. It is by far the most economical method of locating infected cattle herds. In other words, because of the high cost of routine "down the road" testing of cattle for TB and the low possibility of finding infection, Veterinary Services must rely more and more on the submission of suspicious lesions from slaughtered animals by OFO personnel utilizing the VS 6-35 Forms. Your role as an FSIS Public Health Veterinarian is to facilitate the traceback testing effort, thereby greatly enhancing the TB eradication effort.

Veterinary Services is quite optimistic about the chances of complete eradication of bovine tuberculosis and sees several factors that would tend to *favor* its complete eradication in the near future. Those factors are as follows:

1. Better procedures for testing high risk herds and areas for tuberculosis.

2. Decline in the prevalence of *Mycobacterium Bovis,* the causative agent of tuberculosis in cattle.

3. Cattle are generally slaughtered younger now, with less chance of infection spread.

4. Increased slaughter inspection coverage through laws requiring inspection.

5. Improved animal identification systems.

6. Increased federal indemnities (payments to producers for their losses), thereby enhancing the use of depopulation (total slaughter) of infected herds as a method of *eradicating* the disease rather than merely *controlling* it.

However, Veterinary Services sees certain factors that could hamper the eradication effort. These are as follows:

1. Development, from time to time, of other crises that divert funds and manpower from the TB surveillance program.

2. *Failure* of inspectors to detect TB lesions on postmortem or to submit those that are suspicious to the VS laboratory.

3. *Failure* to collect and submit identification devices with laboratory specimens to aid in possible traceback procedures.

4. Inadequate animal identification and record-keeping at feedlots and markets, as many of the unsuccessful tracebacks dead end at feedlots or livestock markets.

In order to more fully recognize the importance of the food inspector and the Public Health Veterinarian in the bovine TB eradication effort, Veterinary Services has implemented an incentive awards program, known as the APHIS Bovine Tuberculosis Eradication Performance Awards Program. Under this program, food inspectors and Public Health Veterinarians will be considered for cash awards as follows:

1. A cash award of $100 for steers and $500 for adult animals to be shared equally each time Mycobacteriosis is reported on histopathology by the National Veterinary Services Laboratories (NVSL).
   - If the specimen is positive for *Mycobacterium tuberculosis (complex)* on Polymerase Chain Reaction (PCR) test, or *M. bovis* is isolated, the cash award will be increased to a total of $200 for steers and fed heifers and $1,000 for adult animals.
   - Tissues submitted only to FSIS field service laboratories or to other approved, diagnostic laboratories that are indicative of tuberculosis shall be forwarded to NVSL for reconfirmation in order to qualify for an award.

2. A second cash award of $6,000 to be shared equally when an infected herd located in the United States is initially found as a result of the information provided to Veterinary Services (VS) regarding the identification of the lesioned animal.

Each award is shared with the Food Inspector(s) responsible for retaining the affected carcass and the PHV initiating the VS 6-35 report. In the event of multiple cases in the same slaughter lot, awards will be granted for as much as three cases from such a lot. Specimens from animals sent to slaughter under permit because of tuberculosis (reactors, suspects, animals from quarantined herds, exposed animals being depopulated, and exposed animals traced to new herds) will not qualify as a basis for an award.

3. A team award of **$**300 per team member will be awarded annually to high submitting FSIS slaughter inspection groups irrespective of histopathology results. High submitting establishments will qualify, at the end of each 12-month period (Fiscal Year), when the plant is credited with one or more suspicious tuberculosis lesions or thoracic granulomas submitted per 1,000 cattle killed.

To be considered for an award, the food inspector must recognize the possibility of lesions of TB in a *regular* kill animal, collect and coordinate identification of the animal, and immediately report the facts to the PHV. The PHV is then responsible for retaining the carcass and submitting the samples for analysis. Two or more cases from the same source will be considered one submission. Specimens from animals slaughtered under permit because of TB, such as reactors, suspects, animals from quarantined herds, and exposed animals being depopulated will not qualify for an award.

TB Reactors and Suspects

A TB reactor is an animal that has reacted to an official test for tuberculosis and the reaction is such that the animal is determined to be a reactor. When an animal is identified as a TB reactor, it may be branded with a "T" brand on the left hip, near the tailhead and a TB reactor tag is placed in its left ear before being sent to a packing plant for slaughter.

When a TB reactor arrives at the plant, it is handled differently during ante mortem inspection. The plant must place the animal in the suspect pen and notify the FSIS Public Health Veterinarian. All TB reactors must be examined for signs of TB. If you condemn a TB reactor on ante mortem, you must have the animal removed to an inedible department where an expanded postmortem examination is performed. FSIS is required to do this for live TB reactors condemned on ante mortem, as well as those reactors that have died; either en route to the plant or in the pens. FSIS needs to ensure that all permitted animals are actually slaughtered, and collect samples or assist Veterinary Services employees in collecting samples for submission to NVSL.

A TB reactor is further identified by a form (VS Form 1-27 Permit for Movement of Restricted Animals) that serves as a permit for the movement of the animal. A copy of the form is mailed in advance to the veterinarian at the plant where the animal is to be slaughtered and a copy of the form accompanies the animal during shipment. Plant management must segregate the animals, notify the FSIS PHV of their presence, and give a copy of VS Form 1-27 to the FSIS PHV.

TB reactors and suspects will have an approved blue or silver metal eartag bearing a serial number and inscription "U.S. Reactor" or a similar State reactor tag attached to the left ear.and a “T” brand for reactor, or a “S” brand for suspect on the left hip near the tailhead. If animals are unbranded, they must meet the following provisions in order to be moved to slaughter: they may be shipped under permit but they must bear the letters "TB" for reactor or "S" for suspect on the left hip applied with yellow paint; and they must be shipped under seal or accompanied directly by a State or Federal animal health official.

FSIS PHVs should note any discrepancies on the VS Form 1-27. if any animals are presented without proper identification, VS Form 1-68 must be executed. PHVs should complete VS Form 1-27 after they have verified that the animals have been slaughtered.

TB reactors are handled as U.S. Suspects, and the reactor number is used in place of the suspect tag number. The time of slaughter is determined by the FSIS PHV. You need to perform a complete ante mortem physical examination of these animals. If they are DOA or DIP, you will perform postmortem examination using expanded procedures.

TB suspects or exposed animals are handled differently from TB reactors. They must be segregated and identified by the plant to the PHV, but require no special handling unless they are showing clinical signs. If they are dead, they will receive expanded postmortem inspection procedures.

On postmortem, TB reactors must have all identification devices kept with the carcass, and you must perform expanded postmortem procedures. You do not routinely collect samples unless requested by Veterinary Services. Once you have conducted your postmortem examination, you must document your findings on FSIS Form 6200-14.

On postmortem, TB suspects are handled differently. If no suspicious lesions are found, you perform routine postmortem procedures only, and collect sections of apparently healthy lymph nodes from the head, neck, and thorax for NVSL. If suspicious lesions are found, you must perform the expanded procedure and send samples to NVSL. Your postmortem findings must be recorded on FSIS Form 6200-14.

On postmortem, TB exposed animals must have the modified expanded procedure performed. If lesions are found, the expanded procedure must be performed and samples must be submitted to NVSL. If no lesions are found, submit sections of apparently healthy lymph nodes to NVSL. Your postmortem findings must be recorded on FSIS Form 6200-14.

For TB reactors, suspects and exposed animals, APHIS Veterinary Services personnel usually accompany the animals. If not present, FSIS PHVs are instructed to assume that the animals are of critical diagnostic value and collect samples if lesions are present or collect healthy lymph nodes if no lesions are present.

APHIS Veterinary Services Bovine Tuberculosis Fact Sheet

Tuberculosis (TB) is a contagious disease of both animals and humans. It is caused by three specific types of bacteria that are part of the Mycobacterium group: *Mycobacterium bovis, M. avium,* and *M. tuberculosis.*

Bovine TB, caused by *M. bovis*, can be transmitted from livestock to humans and other animals. No other TB organism has as great a host range as bovine TB, which can infect all warmblooded vertebrates. *M. avium* can affect all species of birds, as well as hogs and cattle. *M. tuberculosis* primarily affects humans but can also be transmitted to hogs, cattle, and dogs.

Bovine TB has affected animal and human health since antiquity. Once the most prevalent infectious disease of cattle and swine in the United States, bovine TB caused more losses among U.S. farm animals in the early part of this century than all other infectious diseases combined. Begun in 1917, the Cooperative State–Federal Tuberculosis Eradication Program, which is administered by the U.S. Department of Agriculture's (USDA) Animal and Plant Health Inspection Service (APHIS), State animal health agencies, and U.S. livestock producers, has nearly eradicated bovine TB from the Nation's livestock population. This disease's presence in humans has been reduced as a result of the eradication program, advances in sanitation and hygiene, the discovery of effective drugs, and pasteurization of milk.

The Disease

In general, disease-causing mycobacteria live only a few weeks outside a host's body because they cannot tolerate prolonged exposure to heat, direct sunlight, or dry conditions. Under cold, dark, and moist conditions, the organisms can survive longer.

Mycobacteria do not grow outside of a host except in cultured media, where they multiply approximately once every 20 hours. Because of this relatively slow rate of growth, the disease usually takes many months to develop. In some instances, the organisms lie dormant within the host's body for its lifetime, both in animals and in humans, without causing progressive disease.

Bovine TB is a chronic disease, seldom becoming apparent until it has reached an advanced stage in cattle, captive cervids, and swine. Some infected livestock seem to be in prime condition, showing no evidence of infection until they are slaughtered, yet they may be found so seriously infected during slaughter inspection that their carcasses must be condemned.

TB Transmission

Bovine TB can be transmitted from animals to humans and vice versa. Although young animals and humans can contract the disease by drinking raw milk from infected dams, the most common means of transmission is through respiration. Invisible droplets (aerosols) containing TB bacteria may be exhaled or coughed out by infected animals and then inhaled by susceptible animals or humans. The risk of exposure is greatest in enclosed areas, such as barns. Inhalation of aerosols is the most common route of infection for farm and ranch workers and veterinarians who work with diseased livestock. Livestock also are more likely to infect each other when they share a common watering place contaminated with saliva and other discharges from infected animals. Calves, hogs, and humans can contract bovine TB when they drink unpasteurized milk from infected cows.

Diagnosis

TB lesions may be found in any organ or body cavity of diseased animals. In early stages of the disease, these lesions are difficult to find, even during post mortem examination. But in later stages, the nodules or lumps caused by bovine TB become very evident in the lungs and associated lymph nodes and in the lymph nodes of the head and intestinal tract. Lesions may also appear in the abdominal organs, reproductive organs, nervous system, superficial body lymph nodes, and bones.

Humans and animals with TB develop an immune response, which can be detected by the tuberculin skin test. Tuberculin is a sterile laboratory product made by growing TB bacteria, killing them with heat, removing them from the substance on which they were grown, and properly diluting and preserving the remaining mixture. About 72 hours after tuberculin is injected into animals affected with TB, a characteristic swelling reaction appears at the point of injection. This reaction is a positive test result, indicating exposure to one type of mycobacteria.

Further diagnostic methods are necessary to confirm the presence of bovine TB. In humans, these tests include chest x rays and sputum cultures. For animals, the comparative cervical tuberculin test, serological tests, post mortem examinations, and other laboratory procedures are used.

The course of treatment for humans with bovine TB takes 6 to 9 months, and the success rate following treatment is more than 95 percent. In livestock, bovine TB can be controlled within an affected herd through regular testing and slaughter of any single animal that tests positive until the entire herd tests negative for this disease. However, because there is no method available to ensure that bovine TB has been eliminated from an affected herd, APHIS recommends herd depopulation.

Control and Eradication

The most effective way of handling the problem of bovine TB in humans is to eradicate it in livestock. At the start of the cooperative eradication program at the beginning of this century, all cattle herds were systematically tested, and all reactors were sent to slaughter. Federal and State agencies shared in the payment of indemnities. Premises were cleaned and disinfected after infected cattle were removed. As a result of this program, the reactor rate in cattle was reduced from about 5 percent to currently less than 0.02 percent. Consequently, the incidence of human TB caused by M. bovis also decreased significantly. The resurgence of human TB in recent years is attributable to *M. tuberculosis.*

Today, with a very low rate of bovine TB, the most efficient way of finding the disease is through a nationwide surveillance program in slaughter plants. State or Federal meat inspectors check the glands and organs of cattle for signs of TB. If these inspectors find lesions indicative of TB infection, tissue samples are sent to APHIS' National Veterinary Services Laboratories in Ames, IA, for confirmation. If the laboratory confirms that the lesions are the result of bovine TB, an exhaustive attempt is made to trace the infected livestock back through market channels to the originating herd, which is then tuberculin tested.

If the herd of origin is diagnosed with M. bovis, every effort is made to eliminate all animals in the herd. Indemnities, as available, are paid to help compensate owners for their losses. If the herd cannot be depopulated, it is held under quarantine and tested repeatedly until all evidence of infection is eliminated.

Veterinary epidemiologists also attempt to determine the date the herd was probably infected. They then undertake a concerted effort to trace all cattle that moved into or out of the affected herd to try to find out where the disease came from and where it might have gone.

Area Accreditation

For a State to be accredited free of bovine TB, there must have been no confirmed cases of the disease for at least 5 years, and the State must have a set of stringent laws and regulations governing livestock dealers. The State must also maintain surveillance of cattle in marketing channels and require that records be kept that would allow animal health officials to trace infected animals back to their source.

Herd Accreditation

Livestock owners may achieve accredited TB status for their individual herds by following the "Accredited Herd Plan." Details can be found in the publications, "Bovine Tuberculosis Eradication, Uniform Methods and Rules" (UM&R) and "Tuberculosis Eradication in Cervidae, UM&R."

For a herd to qualify as accredited, a negative finding on two annual TB tests must be attained for all cattle over 24 months of age and cattle of any age that are not natural additions to the herd. Deer and elk herds must test negative for 3 consecutive years. To qualify and continue as an accredited herd, livestock must be tested annually within 10 to 14 months of the anniversary of the original test. Livestock from any herd in an accredited free State may be added to an accredited herd without a qualifying test.

What You Can Do

As a livestock producer—dairy, beef, deer, or elk—there are certain things you can do to protect your animals from TB.

The first and most important is to be aware that TB is not gone! Too many farmers falsely believe that TB in cattle has already been eradicated. Remember that this chronic disease will continue to be a threat to animal health until the last infected animal has been eliminated.

So, to be safe, have your livestock tested for TB by an accredited veterinarian to make sure the disease isn't present in your herd. Other tips for preventing TB infection are as follows:

• One of the best ways to avoid TB—and other diseases, too—is to keep a closed herd. Doing so involves raising your own replacement stock. If this system isn't practical for you, demand that sellers give you historical health information about the herd of origin. It's best to buy from accredited TB-free herds.

• If you cannot obtain health histories, make sure any prospective livestock are tested before purchase. Isolate these animals and have them retested 60 days later by an accredited veterinarian.

• Maintain fences in good repair to keep your animals from mingling with neighboring animals.

• And, finally, cooperate with State and Federal animal health officials who are carrying out traceback investigations.

F. Brucellosis

Handling of Brucellosis Reactors

Brucellosis is a reportable disease. Brucellosis (Bangs) reactors are identified by APHIS Veterinary Services with reactor tags and permit VS Form 1-27. Brucellosis is zoonotic: it causes undulant fever in humans. This disease has been largely eradicated from the United States, but is still present in Texas. Brucellosis reactors will come to slaughter accompanied by appropriate Veterinary Services documentation (VS Form 1-27).

Now let's talk about the brucellosis eradication program and how you as an FSIS PHV assist in this program. You will need to verify the reactor status by examining brands and documentation. You need to work cooperatively with APHIS Veterinary Services employees to collect and submit blood and tissue samples. Disposition of reactor carcasses is the same as for regular slaughter animals, and should be based on FSIS disposition guidelines. Remember that when you handle brucellosis reactor carcasses to take care: brucellosis is zoonotic.

The Brucellosis Eradication Program

The accelerated brucellosis eradication program began in 1954 and has gone through many changes in the past 45 plus years. OFO has had an increasingly important role in this program in that we are responsible for collecting blood samples at federally inspected plants from *all*

mature cattle. The market cattle testing (MCT) guidelines in Section 21.6 of the MPI Manual define mature cattle as those bulls and cows 2 years of age or over and cows that are giving or about to give birth, or those that have given birth and are *less* than 2 years old. Samples should be taken from those animals branded as reactors.

The blood samples can be taken at any adequate site, but the heart at the time of postmortem inspection is the preferred site of collection.

The blood tubes should be filled to about one-half to three-fourth's capacity for laboratory handling. Each blood sample should be placed in a plastic bag with all identifying devices (including reactor tags, if any) and sent to the appropriate laboratory. Proper care and handling of the samples is very important. Assuring that the samples are protected from freezing, moisture, and contamination cannot be overemphasized. Refrigeration at 35-40 degrees F after serum separation is important. When possible, blood samples should be mailed daily or at least every other day. Franked labels addressed to the proper laboratory are provided, as well as blood sample tubes, mailing boxes, and record forms. In plants where F0 personnel are unable to collect samples, it is usually arranged through VS for a plant employee or contract technician to collect the samples under OFO supervision.

The brucellosis eradication program depends very heavily on you as a food inspector and how efficiently you submit the blood samples to the laboratory for analysis. This is especially important since the number of blood samples taken at places other than packing plants is on the decline.

Before we leave our discussion of brucellosis, we should mention a few points about its transmissibility to man. The potential for inspectors contracting brucellosis from cattle or swine is great and you should take all possible precautions to decrease the likelihood of becoming a victim of the disease. In recent years, most of the reported human brucellosis cases have been of swine origin, probably due to the concentrated bovine eradication effort of former years. When performing routine postmortem you should practice sound hygienic principles to include frequent washing of the hands, and avoiding as much as possible open cuts in the hands through which the bacteria could gain entry. Also, you should strive not to be splattered in such areas as the eyes and mouth with blood and reproductive tract fluids. You should not place your contaminated hands around your mouth at any time. Although you cannot totally eliminate this hazard of your profession, you should always be aware of the things you can do to decrease chances of infecting yourself.

The other type of specially identified animal mentioned above is a brucellosis reactor. Brucellosis (Bang's disease) is another disease that we have been attempting to eradicate from this country for a long time. The identification of these animals is similar to tuberculosis reactors. Animals that react to a brucellosis or Bang's test must be identified and sent to slaughter. A Bang's reactor tag is placed in the animal's left ear and a "B" is branded on the left hip. A shipping permit form is completed and sent along with the Bang's reactor to the slaughter plant.

APHIS Brucellosis Fact Sheet

1. What is brucellosis?

It is a contagious, costly disease of ruminant animals that also affects humans. Although brucellosis can attack other animals, its main threat is to cattle, bison, and swine. The disease is also known as contagious abortion or Bang's disease. In humans, it's known as undulant fever because of the severe intermittent fever accompanying human infection or Malta fever because it was first recognized as a human disease on the island of Malta.

2. How serious is brucellosis?

Considering the damage done by the infection in animals-decreased milk production, weight loss in animals, loss of young, infertility, and lameness, it is one of the most serious diseases of livestock. The rapidity with which it spreads and the fact that it is transmissible to humans makes it all the more serious.

3. What disease agents cause brucellosis?

The disease is caused by a group of bacteria known scientifically as the genus *Brucella*. Three species of *Brucella* cause the most concern: *B. abortus*, principally affecting cattle and bison; *B. suis*, principally affecting swine and reindeer but also cattle and bison; and *B. melitensis*, principally affecting goats but not present in the United States. In cattle and bison, the disease currently localizes in the reproductive organs and/or the udder. Bacteria are shed in milk or via the aborted fetus, afterbirth, or other reproductive tract discharges.

4. What are the signs of brucellosis?

There is no effective way to detect infected animals by their appearance. The most obvious signs in pregnant animals are abortion or birth of weak calves. Milk production may be reduced from changes in the normal lactation period caused by abortions and delayed conceptions. Not all infected cows abort, but those that do usually abort between the fifth and seventh month of pregnancy. Infected cows usually abort once, but a percentage will abort during additional pregnancies, and calves born from later pregnancies may be weak and unhealthy. Even though their calves may appear healthy, infected cows continue to harbor and discharge infectious organisms and should be regarded as dangerous sources of the disease. Other signs of brucellosis include an apparent lowering of fertility with poor conception rates, retained afterbirths with resulting uterine infections, and (occasionally) enlarged, arthritic joints.

5. How is brucellosis spread?

Brucellosis is commonly transmitted to susceptible animals by direct contact with infected animals or with an environment that has been contaminated with discharges from infected animals. Aborted fetuses, placental membranes or fluids, and other vaginal discharges present after an infected animal has aborted or calved are all highly contaminated with infectious *Brucella* organisms. Cows may lick those materials or the genital area of other cows or ingest the disease-causing organisms with contaminated food or water. Despite occasional exceptions, the general rule is that brucellosis is carried from one herd to another by an infected or exposed animal. This mode of transmission occurs when a herd owner buys replacement cattle or bison that are infected or have been exposed to infection prior to purchase. The

disease may also be spread when wild animals or animals from an affected herd mingle with brucellosis-free herds.

6. What is being done to fight brucellosis?

Before 1934, control of brucellosis was limited mainly to individual herds. Today, there is a Cooperative State Federal Brucellosis Eradication Program to eliminate the disease from the country. Like other animal disease-eradication efforts, success of the program depends on the support and participation of livestock producers. The program's Uniform Methods and Rules set forth the minimum standards for States to achieve eradication. States are designated brucellosis free when none of their cattle or bison are found to be infected for 12 consecutive months under an active surveillance program. As of June 30, 2000, 44 States, plus Puerto Rico and the U.S. Virgin Islands, were free of brucellosis. Six States currently have a herd infection rate of less than 0.25 percent and are considered to be in Class A status. There are no States in Class B (herd infection rate between 0.26 percent and 1.5 percent) or Class C status (herd infection rate greater than 1.5 percent).

7. What about free-ranging bison herds?

The presence of brucellosis in free-ranging bison in Yellowstone National Park and Grand Teton National Park threatens the brucellosis status of the surrounding States and the health of their livestock herds, which are free of the disease. Reintroduction of the disease into a brucellosis-free State could have a serious economic impact on domestic livestock markets and potentially threaten export markets. The U.S. Department of Agriculture's (USDA) Animal and Plant Health Inspection Service (APHIS) is working cooperatively with other State and Federal agencies toward containing the spread of brucellosis from bison to domestic livestock and eliminating the disease from the Yellowstone and Teton herds while maintaining viable free-roaming bison herds in the Parks.

8. How do epidemiologists help fight brucellosis?

Epidemiologists are specially trained veterinarians who investigate disease sources and the means of eliminating infection in affected herds and areas. Epidemiologists are concerned with disease in a group or population of animals and evaluate circumstances connected with the occurrence of disease. These veterinarians help eliminate brucellosis by identifying factors essential to its control and prevention.

9. How costly is brucellosis to the livestock industry?

The livestock and dairy industries and the American consumer have realized great financial savings from the success of the Cooperative State Federal Brucellosis Eradication Program. Annual losses from lowered milk production, aborted calves and pigs, and reduced breeding efficiency have decreased from more than $400 million in 1952 to less than $1 million today. Studies have shown that, if brucellosis eradication program efforts were stopped, the costs of producing beef and milk would increase by an estimated $80 million annually in less than 10 years.

10. How effective is the Brucellosis Eradication Program?

At the beginning of the program, brucellosis was widespread throughout U.S. livestock, but eradication efforts have had dramatic results. In 1956, there were 124,000 affected herds found

by testing in the United States. By 1992, this number had dropped to 700 herds, and as of June 30, 2000, there were only 6 known affected herds remaining in the entire United States. USDA, APHIS expects the Cooperative State Federal Program to achieve the goal of nationwide eradication of brucellosis from domestic cattle and bison in the very near future.

11. What is the basic approach to eradication?

The basic approach has always been to test cattle for infection and send infected animals to slaughter. Identification of market animals for tracing, surveillance to find infected animals, investigation of affected herds, and vaccination of replacement calves in high-risk areas are important features of the current program.

12. How is infection found in cattle?

Two primary surveillance procedures are used to locate infection without having to test each animal in every herd. Milk from dairy herds is checked two to four times a year by testing a small sample obtained from creameries or farm milk tank for evidence of brucellosis. Bison herds and cattle herds that do not produce milk for sale are routinely checked for brucellosis by blood-testing animals sold from these herds at livestock markets or at slaughter. In addition, some States require adult cattle and bison to be subjected to blood tests for brucellosis upon change of ownership even if sold directly from one farm to another. The cattle and bison remaining in the herds from which such animals originated are not tested unless evidence of brucellosis is disclosed among the market animals.

13. What happens when evidence of disease is found by surveillance testing?

Once an infected herd is located, the infection is contained by quarantining all infected and exposed cattle and bison and limiting their movement to slaughter only, until the disease can be eliminated from the herd. Diagnostic tests are used to find all infected cattle and bison. Also, Federal and State animal health officials check neighboring herds and others that may have received animals from the infected herd. All possible leads to additional infection are traced.

14. How does the brucellosis ring test (BRT) surveillance work?

The BRT procedure makes it possible to do surveillance on whole dairy herds quickly and economically. Milk or cream from each cow in the herd is pooled, and a sample is taken for testing. A suspension of stained, killed *Brucella* organisms is added to a small quantity of milk. If the milk from one or more infected animals is present in the sample, a bluish ring forms at the cream line as the cream rises.

15. How does market cattle identification (MCI) work?

Numbered tags, called backtags, are placed on the shoulders of adult breeding animals being marketed from beef, dairy, and bison herds. Blood samples are collected from the animals at livestock markets or slaughtering plants and tested for brucellosis. If a sample reacts to a diagnostic test, it is traced by the backtag number to the herd of origin. The herd owner is contacted by a State or Federal animal health official to arrange for testing of his or her herd. Once the animals have been gathered, all of the eligible animals in the herd are tested at no cost to the owner.

16. Which animals are eligible for MCI testing?

At slaughter, all cattle and bison 2 years of age or older are tested, except steers and spayed heifers. At market, all beef cattle and bison over 24 months of age and all dairy cattle over 20 months of age are tested except steers and spayed heifers. Pregnant or postparturient heifers are also eligible for testing regardless of their age. Herd tests must include all cattle and bison over 6 months of age except steers and spayed heifers.

17. Why is identification of market cattle important?

The key to the MCI program is proper identification of all animals so they can be traced to their herds of origin. Most livestock markets identify cattle and bison with numbered USDA-approved backtags. Backtags, as well as eartags and other identification devices, are collected and sent to the diagnostic laboratory along with the matching blood samples to aid in identifying ownership of test-positive animals.

18. What are the advantages of MCI?

MCI provides a means of determining the brucellosis status of animals marketed from a large area and eliminates the need to round up cattle and bison in all herds for routine testing. MCI, along with other preliminary testing procedures, is effective in locating infection so control measures can be taken to contain the disease and eliminate it.

19. What is a blood agglutination test?

It is an effective method of diagnosing brucellosis. To pinpoint infection within a herd, a blood sample is taken from each animal and tested in the field or at a laboratory. The blood serum is mixed with a test fluid or antigen containing dead *Brucella* organisms. When the organisms in the test fluid clump together in a reaction known as agglutination, the test is positive.

20. What is the brucellosis card test?

It is a rapid, sensitive, and reliable procedure for diagnosing brucellosis infection. It is similar to the blood agglutination test but employs disposable materials contained in compact kits. Brucella antigen is added to the blood serum on a white card. Results of the test are read 4 minutes after the blood serum and antigen are mixed.

21. Are there any other tests for brucellosis?

There are a number of supplemental tests based on various characteristics of antibodies found in the blood and milk of infected animals. These tests are especially useful in identifying infected animals in problem herds in which chronic brucellosis infection exists and from which infection is difficult to eliminate. Another diagnostic method involves culturing Brucella organisms from infected tissues, milk, or other body fluids, from aborted calves or fetal fluids and membranes.

22. What animals are eligible for testing?

With certain exceptions, herd tests must include all cattle and bison over 6 months of age except steers and spayed heifers.

23. What is the incubation period of brucellosis?

An incubation period is the interval of time between exposure to an infectious dose of organism and the first appearance of disease signs. The incubation period of brucellosis in cattle, bison, and other animals is quite variable ranging from about 2 weeks to 1 year and even longer in certain instances. When abortion is the first sign observed, the minimum incubation period is about 30 days. Some animals abort before developing a positive reaction to the diagnostic test. Other infected animals may never abort. Generally, infected animals that do not abort develop a positive reaction to the diagnostic test within 30 to 60 days after infection, although some may not develop a positive reaction for several months to over a year.

24. Can brucellosis in animals be cured?

No. Repeated attempts to develop a cure for brucellosis in animals have failed. Occasionally, animals may recover after a period of time. More commonly, however, only the signs disappear and the animals remain diseased. Such animals are dangerous sources of infection for other animals with which they associate.

25. Can brucellosis be prevented?

The disease may be avoided by employing good sanitation and management practices. Replacement animals should be tested when purchased and retested after a 30- to 60-day isolation period during which they are kept separate from the remainder of the herd. These practices will allow detection of animals that were in the incubation period of the disease when acquired.

26. What about vaccination?

For cattle and bison in heavily infected areas or replacement animals added to such herds, officials recommend vaccinating heifers with an approved *Brucella* vaccine. The vaccine is a live product and must be administered only by an accredited veterinarian or State or Federal animal health official. For best results, female calves should be vaccinated when they are 4 to 6 months old. At the time of vaccination, a tattoo is applied in the ear; that tattoo identifies the animal as an "official vaccinate." The tattoo identifies the year in which vaccination took place.

27. How does the vaccine work?

Brucella abortus vaccine produces a bodily response that increases the animal's resistance to the disease. However, vaccination is not 100-percent effective in preventing brucellosis; it typically protects about 65 percent of the vaccinated cattle from becoming infected by an average exposure to *Brucella*.

28. Is Strain 19 the only approved *Brucella* vaccine?

No. USDA recently licensed a new *Brucella* vaccine, called Strain RB51, for use in cattle. Strain RB51 is as efficacious as Strain 19 vaccine but virtually eliminates adverse post vaccination reactions in cattle, such as abortions and localized inflammation at the vaccine injection site. Most importantly, unlike Strain 19, Strain RB51 does not stimulate the same type of antibodies that can be confused on standard diagnostic tests with those antibodies produced by actual infection.

29. Is Strain RB51 vaccine approved for use in bison?

As of June 2000, *B. abortus* Strain RB51 had not yet been approved for use in bison. Preliminary studies indicate that RB51 is safe and efficacious in bison calves. However, in order for RB51 to be conditionally licensed in bison, additional safety and efficacy trials must be completed.

30. Where or when is calf-hood vaccination most important?

Owners whose herds are located in areas of relatively heavy infection or who ship replacement cattle or bison to, or receive animals from, such areas should carry out a vigorous calf-hood vaccination program. Every cattle or bison owner, regardless of location, should discuss the advantages and disadvantages of vaccination with his or her veterinarian. Some States do not allow cattle and bison to be imported for breeding if they are not official vaccinates and they are beyond the age at which they should have been vaccinated.

31. Where is vaccination less important?

In many areas of the country, low herd infection rates coupled with improvement in the detection of early infection through BRT, MCI, and other surveillance systems have lessened the need to continue calf-hood vaccination. Vaccination should be reduced in such areas, provided that adequate regulatory measures are in effect to prevent reintroduction of the disease.

32. How does brucellosis affect humans?

People infected with the brucellosis organism usually develop symptoms similar to a severe influenza, but this disease, called undulant fever, persists for several weeks or months and may get progressively worse. Farmers, ranchers, veterinarians, and packing plant workers are infected most frequently because they come into direct contact with infected animals. The initial symptoms are fatigue and headaches, followed by high fever, chills, drenching sweats, joint pains, backache, and loss of weight and appetite. Undulant fever does not often kill its victims, but the disease is too serious to be dealt with lightly.

33. What are the main sources of human infection?

In years past, prior to pasteurization, raw milk was considered the prime source of brucellosis in humans. Today, most humans contract the disease by coming in direct contact with aborted fetuses, afterbirth, and uterine discharges of diseased animals or with infected carcasses at slaughter. However, one 1994 study suggests that human brucellosis in California is most likely to be a food-borne illness (unpasteurized milk or cheese products) acquired in Mexico or from Mexican products consumed in California. Rarely, if ever, does a human contract the disease from another human.

34. How common is human brucellosis in this country?

Fortunately, the combination of pasteurization of milk and progress in the eradication of the disease in livestock has resulted in substantially fewer human cases than in the past. Ninety eight cases of human brucellosis were reported in 1997, a fraction of the 6,400 cases reported in 1947. Sixty two (62) cases of brucellosis in humans have been reported to the Centers for Disease Control and Prevention for 1998 (provisional data).

35. Can people get brucellosis by eating meat?

There is no danger from eating cooked meat products because the disease-causing bacteria are not normally found in muscle tissue and they are killed by normal cooking temperatures. The disease may be transmitted to humans when slaughtering infected animals or when processing contaminated organs from freshly killed animals.

36. How can people be protected from brucellosis?

Ranchers, farmers, or animal managers should clean and disinfect calving areas and other places likely to become contaminated with infective material. All individuals should wear sturdy rubber or plastic gloves when assisting calving or aborting animals, and scrub well with soap and water afterward. Precautions against drinking raw milk or eating unpasteurized milk byproducts are also important. Ultimately, the best prevention is to eliminate brucellosis from all animals in the area.

G. Vesicular Stomatitis

Definition

Vesicular stomatitis (VS) is a viral disease characterized by fever, vesicles, and subsequent erosions in the mouth and epithelium on the teats and feet. Horses, cattle, and pigs are naturally susceptible; sheep and goats are rarely affected.

Etiology

The vesicular stomatitis virus is a *Vesiculovirus* in the family *Rhaboviridae*. The virion is a large bullet-shaped (65-185 nm) RNA virus. There are two serotypes of VSV: New Jersey and Indiana- 1. In the serotype Indiana 1, there are two subtypes: Indiana 2 (Cocal) and Indiana 3 (Alagoas). In addition to these two serotypes of VSV, there are other viruses within the genus *Vesiculovirus* that can experimentally cause vesicular lesions in domestic animals and infect humans; these are as follows:

Piry — first isolated from an opossum in Brazil.
Chandipura — first isolated from a person in India.
Isfahan — isolated from sandflies and humans in Iran.

Effective disinfectants are 2 percent, sodium carbonate - 4 percent, sodium hydroxide - 2 percent, iodophore disinfectants and chlorine dioxide disinfectants.

Host Range

The host range in decreasing order of severity of infection are horses, donkeys, mules, cattle, swine, and man. South American cameilids develop clinical infection. Sheep and goats are quite resistant and rarely become affected. Vesicular stomatitis virus has also been shown experimentally to infect a wide host range, including deer, raccoons, bobcats, and monkeys.

Geographic Distribution

Classical VS occurs only in North and Central America and the northern part of South America. Serotypes New Jersey and Indiana I occur in the United States and Central America. Serotypes New Jersey and Indiana 1, 2, and 3 occur in South America.

Transmission

The vesicular stomatitis virus has been shown to be transmitted by the sand fly (*Lutzomyia shannoni*) and the black fly (*Simuliidae*). Transovarial transmission has been shown to occur in both flies. The VS-NJ serotype was isolated from a variety of field-collected hematophagous insects such as *Culicoides* (biting midges), *Simuliidae* (black flies), *Aedes* (mosquitoes) and nonbiting insects such as *Chloropidae* (eye gnats), *Anthomyiidae*, and *Musca* (house flies) during the 1982 epizootic in the southwestern United States (1). Except for *Lutzomyia* and *Simulidae*, the role of these other insects in the transmission of VSV is unknown. Before the 1982 outbreak in the United States, people, on the basis of past experience, expected an outbreak to stop about 2 weeks after a killing frost. In the 1982 outbreak, cases and spread occurred through the winter. The winter spread of the disease is believed to have resulted from movement of infected animals and the resulting exposure of uninfected animals to contaminated waterers and feed bunks as well as contact with infected animals. It is known that VSV can be spread by a contaminated milking machine. Overwintering did not occur in the 1995 outbreak in the United States.

Humans may be infected by contact and by aerosol.

Epidemiology

The disease occurs throughout the year in subtropical and tropical areas of the Americas. The disease occurs sporadically during the warm months in southern and western United States. Epidemics have occurred irregularly at 10 to 15 year intervals. The virus is spread by insect vectors, movement of infected animals, and contaminated objects. Researchers have shown transovarial transmission in the sand fly and black fly; this may be a way the virus can overwinter.

Incubation Period

A vesicle appears in about 24 hours after intradermal lingual inoculation of VSV. This similar to the incubation period for foot-and-mouth disease. In humans, the incubation period is 24 to 48 hours.

Clinical Signs

Animals develop a fever ranging to 104-106^{o} F (40-41^{o} C).

Horse

Vesicles in the mouth may cause the animal to chomp its jaws, drool, and rub its mouth on the manger or other objects. Lesions on the coronary band can cause lameness.

Cattle and pigs

See the clinical signs section in the FMD chapter. The signs are very similar.

Humans

In humans, VSV causes an influenza-like illness; there is fever, headache, muscular aches, and blisters in the mouth similar to those caused by herpes virus. The disease course is 4 to 7 days.

Morbidity and Mortality

Interesting data on the economic effect of VS in cattle were collected by Alderink during the 1982 outbreak of VS in Colorado. In 13 of the dairy herds studied, there were 2,404 cows and 378 cases of VS. Lesion distribution in these 378 was as follows:

Oral lesions only	263 animals (69.3%)
Teat lesions only	87 animals (23%)
Oral and teat lesions	22 animals (5.8%)
Foot lesions only	7 animals (1.9%)

Herds experiencing primarily oral lesions had an attack rate of 19.8 percent The attack rate in two of four herds with teat lesions was 55.8 percent and in the other two herds 1.6 percent. The clinical course in cases with oral lesions was 23.8 days. Mastitis complicated 72% of the cases with teat lesions.

The total cost to the 13 dairymen was $95,752, which came to an average cost of $253 per case. The approximate cost of a case with only oral lesions was $174 in contrast to an average cost of $568 for cases with teat lesions. Of the total $95,752 loss, 46 percent was for cows culled; 30 percent was for decreased production; 11 percent for deaths; and 11 percent for drugs, labor, weight loss, and veterinary charges.

Differences Between VS and FMD

The characteristics of VS are as follows:

Horses affected.

Sporadic incidence in the herd (see preceding section).

Distribution of lesions in an animal (small percentage of animals have lesions at more than one site of predilection; see preceding section).

No rumen lesions observed at necropsy.

No heart lesions observed at necropsy.

Vesicular stomatitis is less severe in young animals.

Stabled animals usually not affected.

In spite of these differences, do not attempt to make a final differential diagnosis in the field; get laboratory confirmation of the diagnosis.

Diagnosis

See FMD chapter.

Differential Diagnosis

Differential diagnosis for VS in cattle should include foot-and-mouth disease, foot rot, and chemical and thermal burns. In cattle, oral lesions caused by rinderpest, infectious bovine rhinopneumonitis, bovine virus diarrhea, malignant catarrhal fever, and bluetongue can be similar to the later lesions in FMD. In pigs, the differential diagnosis for VS should include foot-and-mouth disease, swine vesicular disease, vesicular exanthema of swine, foot rot, and chemical and thermal burns. In sheep, the differential diagnosis for VS lesions should include bluetongue, contagious ecthyma, lip and leg ulceration, and foot-rot.

Control and Eradication

Control movement of animals — no movement from an infected premise, except for slaughter, for 30 days after last lesion has healed.

Separate infected and healthy animals.

Stable animals if possible.

Disinfect milking machines between cows.

Milk infected cows last.

Control insects.

Commercial vaccines are available, but efficacy has not been field tested.

Public Health

Vesicular stomatitis (New Jersey and Indiana) infection frequently occurs in man and causes influenza-like symptoms but rarely results in vesicles. Other vesicular stomatitis viruses (Piry, Isfahan, and Chandipura) are much more infectious for man.

GUIDE TO THE LITERATURE

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APHIS Veterinary Services FAQ: Vesicular Stomatitis: Questions and Answers

Vesicular stomatitis (VS) is a viral disease that primarily affects horses, cattle, and swine and occasionally sheep, goats, llamas, and alpacas. Humans can also become infected with VS when handling affected animals, but this is a rare event. VS has been confirmed only in the Western Hemisphere. It is known to be an endemic disease in the warmer regions of North, Central, and South America, but outbreaks of the disease in temperate geographic parts of the Hemisphere occur sporadically.

Outbreaks in the Southwestern United States usually occur during warm months, often along waterways and in valleys. The Southwest experienced a VS outbreak from May 2004 through January 2005. Animals in Texas, New Mexico, and Colorado were involved. A total of 294 premises in 43 counties were affected in these three States. There could be another outbreak in 2005, and it is essential that veterinarians and livestock owners be on the alert for animals displaying clinical signs characteristic of VS.

Q. What are the clinical signs of VS?

A. In affected livestock, VS causes blister-like lesions to form in the mouth and on the dental pad, tongue, lips, nostrils, hooves, and teats. These blisters swell and break, leaving raw tissue that is so painful that infected animals generally refuse to eat or drink. If the hooves are affected, the animal may show signs of lameness. Severe weight loss usually follows, and in dairy cows, a severe drop in milk production commonly occurs. Some affected dairy cattle can appear to be clinically normal and will continue to eat about half of their normal feed intake. Lesions in horses may also be expressed as crusting scabs on the muzzle, lips, or ventral abdomen.

Q. How is the disease spread?

A. How VS spreads is not fully known; insect vectors, mechanical transmission, and movement of animals are all factors. Once VS is introduced into a herd, the disease may move from animal to animal by contact or exposure to saliva or fluid from ruptured lesions.

Q. Can humans contract VS?

A. Humans rarely contract VS when handling affected animals but can become infected. To avoid exposure to this disease, use protective measures when handling affected animals. In people, VS causes an acute influenza like illness with symptoms such as fever, muscle aches, headache, and malaise.

Q. Why is it so important to recognize animals with VS promptly?

A. VS is recognized internationally as a reportable disease. What this means is that there are serious economic and regulatory repercussions associated with the diagnosis, and once the disease is detected in the United States, many countries take action to block international trade of U.S. animals. Interstate movement of animals is also impacted. Premises containing affected animals are quarantined until 21 days after the lesions in the last affected animal have healed. These quarantine periods may be quite long.

While VS can cause economic losses to livestock producers, it is a particularly significant disease because its outward signs are similar to (although generally less severe than) those of foot–and–mouth disease, a foreign animal disease of cloven– hoofed animals that was eradicated from the United States in 1929. The clinical signs of VS are also similar to those of swine vesicular disease, another foreign animal disease. The only way to tell these diseases apart is through laboratory testing.

Q. What are clinical signs of VS? How does the disease progress in affected animals?

A. In affected livestock, the incubation period for VS ranges from 2 to 8 days. Often, excessive salivation is the first sign that an animal is affected. Body temperature may rise immediately before or at the same time lesions first appear. Initially, close examination of the mouth reveals blanched and raised vesicles. If there are no complications such as secondary infections, affected animals recover in about 2 weeks. VS does not generally cause animals to die.

Clinical signs of VS and other vesicular diseases include:

- Excessive salivation
- Swollen lips
- Blanched skin and raised or broken vesicles of various sizes around and in the mouth

Horses: Upper surface of the tongue, surface of the lips and around nostrils, corners of the mouth, and the gums.
Cattle: Tongue, lips, gums, hard palate, and sometimes the muzzle and the area around the nostrils.
Pigs: Snout.
• Lesions involving feet of horses and cattle are not commonly seen in the Southwestern United States. However, if lesions do occur, lameness may be noted as a clinical sign.
• Foot lesions and lameness are frequent in pigs.

• Teat lesions may occur in dairy herds. Loss of production and mastitis in dairy herds due to secondary infections may be a secondary complication.

Q. If there is another VS outbreak this year, what do I need to do if I plan to travel with my horse to another State or country?

A. During a VS outbreak, States and countries may put regulations into place restricting the movement of animals and requiring testing of animals prior to movement. Each State and country has different requirements for movement testing. It is important that the animal owner become familiar with the regulations and testing requirements associated with moving an animal.

Not all laboratories run the same antibody tests, so it is important to check with your laboratory to find out if they run the test you need to comply with regulations or movement restrictions. It is also important to contact the laboratory in advance to determine the samples needed submission procedures, and testing cost.

Not all VS tests are conducted on a daily basis, and during an outbreak of VS, there is an increased demand on laboratories for movement testing of animals. Therefore, it is important that a person submitting a sample to a lab for movement testing plan well in advance and have the sample at the lab at the earliest possible time based on the movement regulations.

For more information on testing, please visit our Web site at http://www.aphis.usda.gov/vs/nvsl.

Q. What can we do to protect our animals?

A. There is no specific treatment or cure for VS. Owners can protect their animals from this disease by keeping their animals from congregating in the area where VS has occurred. Mild antiseptic mouthwashes may bring comfort and more rapid recovery to an affected animal. Good sanitation and quarantine practices on affected farms usually contain the infection until it dies out of its own accord. If you suspect that you have a horse with VS, do not allow it to come in contact with your other horses.

When a definite diagnosis of VS is made on a farm, the following procedures are recommended:

- Separate animals with lesions from healthy animals, preferably by stabling. Animals on pastures are at an increased risk of disease.
- As a precautionary measure, do not move animals from premises affected by VS, unless they are going directly to slaughter, for at last 21 days after the last lesion found has healed.
- Implement on-farm insect control programs that include the elimination or reduction of insect breeding areas and the use of approved insecticide sprays or insecticide-treated eartags on animals.

Additional Information

To see pictures of what lesions might look like, please visit the following Web site: http://www.aphis.usda.gov/vs/ep/fad_training/VESVOL7/page02_7.htm.

Additional information can be obtained from the following Web pages:

- http://www.quarterh.com/health3.htm
- http://www.oie.int/eng/maladies/fiches/A_A020.HTM
- http://www.vetmed.wisc.edu/pbs/zoonoses/vsv/vsvindex.html
- http://www.aphis.usda.gov/vs/ep/fad_training/VESVOL7/vesindex.htm

H. Foot and Mouth Disease

(Afta epizootica, Bek-en-klouseer, Fiebra aftosa, Fievre aphteuse, Maul-und-Klauenseuche**)**

Definition

Foot-and-mouth disease (FMD) is a highly contagious viral infection primarily of cloven-hoofed domestic animals (cattle, pigs, sheep, goats, and water buffalo) and cloven-hoofed wild animals. The disease is characterized by fever and vesicles with subsequent erosions in the mouth, nares, muzzle, feet, or teats.

Etiology

The FMD virus (FMDV) is a member of the genus *Aphthovirus* in the family Picornaviridae. There are seven serotypes of FMDV: A, O, C, Asia 1, and Southern African Territories (SAT) 1, 2 and 3. Within these serotypes, over 60 subtypes have been described, and new subtypes occasionally arise spontaneously. However, at a specific time, there are only a few subtypes causing disease throughout FMD endemic areas. The importance of subtypes is that a vaccine may have to be tailored to the subtype present in the area in which the vaccine is being used.

The FMD virus is pH sensitive; the virion is inactivated when exposed to pH below 6.5 or above 11. However, in milk and milk products, the virion is protected and can survive at 70^{o} C for 15 seconds and pH 4.6. Between pH 6.7 and 9, stability increases with decreasing temperature; the virus in cell culture medium will remain viable for a year at 4^{o} C. The virus in serum or other organic material will survive drying and can be carried on inanimate objects. In meat, the virus can survive for long periods in chilled or frozen bone marrow and lymph nodes.

Host Range

Cloven-footed domestic and wild animals are primarily affected. Examples of other susceptible species are hedgehogs, armadillos, nutrias, elephants, capybaras, rats, and mice.

Geographic Distribution

Foot-and-mouth disease, after World War II, was widely distributed throughout the world. In 1996, endemic areas were Asia, Africa, and parts of South America. In South America, Chile is free, and Uruguay and Argentina have not had an outbreak since April 1994. Most European countries have been recognized as free. Countries belonging to the European Union have stopped FMD vaccination. North and Central America, Australia, New Zealand, Japan, and the British Isles have been free of FMD for many years.

Geographic Serotype Prevalence of FMD

It is interesting how certain serotypes tend to be restricted to certain areas of the world.

Some examples are as follows:

Europe (historically) Asia	A (5) O (1) C (1)
Near East	A (22) O (1)
Middle East	A (22) O (1) C Asia (1)
Far East	A O (1) C Asia (1)
Africa	
Central East to West	A O
Northeast Central and South	SAT 1 and 2
South	SAT 3
Serotype C is uncommon in Africa	
South America	A (24), (27) O (1) C (3)

Transmission

The FMD virus can be introduced into a free area by the following means:

1. Direct or indirect contact with infected animals.

2. Spread of aerosol from infected animals (requires proper humidity and temperature). Aerosol from bulk milk trucks spread FMD in England. A person in contact with infected animals can have sufficient FMDV in his or her respiratory tract for 24 hours to serve as a source of infection for susceptible animals.

3. Feeding contaminated garbage (meat, milk, blood, glands, bones, cheese, etc.)

4. Contact with contaminated objects (hands, footwear, clothing).

5. Artificial insemination.

6. Contaminated biologicals such as hormones (extraction procedure may not inactivate the virus).

After an animal becomes infected by any means, the primary mode of spread is then via respiratory aerosols. Other important means of spread are direct and indirect contact. In an outbreak of FMD, the roles of the three primary hosts in transmission are as follows:

- Sheep act as maintenance hosts,
- Pigs act as amplifiers,
- Cattle act as indicators.

When sheep or goats become infected with FMDV, the disease may not be diagnosed for a considerable time because signs and lesions can be very mild. However, during this time, the

animals will be producing infectious aerosols, contaminating fomites, and spreading the virus by contact.

Foot-and-mouth disease in pigs spreads very rapidly, for they produce 30 to 100 times more virus in aerosols than sheep or cattle. An infected pig can produce a hundred million infectious doses per day.

When cattle are infected with FMDV, signs and lesions usually develop more rapidly and are more severe than in pigs, sheep, or goats. If cattle, sheep, and pigs are exposed together, cattle will usually get sick first. This may result from increased exposure due to a greater pulmonary tidal volume.

Some animals can be carriers of FMDV. Most ruminant species can harbor the virus in their pharyngeal tissues for a long period. Recovered cattle or vaccinated cattle exposed to diseased animals can become healthy carriers for 6-24 months. Sheep can be carriers for 4-6 months. Although under experimental conditions it has been difficult to demonstrate transmission of FMD from carriers to susceptible livestock, there is strong circumstantial field evidence that carriers may have been the occasional cause of outbreaks. Also it has been shown that the virus was maintained for many years in a relatively small, isolated group of African buffaloes without the appearance of clinical signs.

Some strains of FMDV seem to have a predilection for certain species. There have been strains that affect pigs but not cattle. In South America, mature cattle have had clinical signs of FMD, when sheep in an adjacent pasture were normal.

Incubation Period

After experimental exposure, signs may develop as early as 12 hours. The usual interval is 24 to 48 hours.

When susceptible animals are in contact with clinically infected animals (peak time of transmission is generally when vesicles rupture), clinical signs usually develop in 3 to 5 days.

Pigs fed infected garbage usually develop signs in 1 to 3 days. Intact oral epithelium is resistant to infection, but during the process of ingesting food there may be injury, and the virus may also enter through the tonsils.

Clinical Signs

Cattle

Initial signs are fever of 103-105^{o} F (39.4-40.6^{o} C), dullness, anorexia, and fall in milk production. These signs are followed by excessive salivation; drooling, serous nasal discharge; shaking, kicking of the feet or lameness; and vesicle (blister) formation. Sites of predilection for vesicles are the tongue, dental pad, gums, soft palate, nostrils, muzzle, interdigital space, coronary band, and teats. Vesicles may be difficult to see. The animal may need to be tranquilized to facilitate a thorough examination.

After vesicle formation, drooling may be more marked, and nasal discharge, lameness or both may increase. Pregnant cows may abort, and young calves may die without developing any vesicle.

The course of an FMD infection is 2 to 3 weeks. Secondary infection may delay recovery. A lactating animal may not recover to preinfection production because of damage to the secretory tissue.

Sequelae to FMD in Cattle

Secondary infection — mouth, nose, feet
Hoof deformation
Low milk production
Mastitis
Unthriftiness — failure to gain weight
Breeding problems
Panting — associated with pituitary gland damage
Diabetes mellitus

Swine

Initial signs are fever of 104-105^{o} F (40-40.6^{o} C), anorexia, reluctance to move, and squeal when forced to move. These signs are followed by vesicles on the coronary band, vesicles on the heals, vesicles in the interdigital space (foot involvement is usually severe), and vesicles on the snout. Mouth lesions are not too common and when they occur are smaller and of shorter duration than in cattle and tend to be a "dry"-type lesion. There is no drooling. Sows may abort. Piglets may die without showing any clinical sign.

Sheep and Goats

Clinical signs, if they occur, tend to be very mild, and may include dullness; fever; and small vesicles or erosions on the dental pad, lips, gums, and tongue. Mild lameness may be the only sign. In lame animals there may be vesicles or erosion on the coronary band or in the interdigital space. Infected animals may abort. Nursing lambs may die without showing any clinical sign.

Gross Lesions

Cattle

The diagnostic lesions are single or multiple vesicles ranging from 2 mm to 10 cm. These can occur at all sites of predilection. Gross lesions on the tongue usually progress in the following manner:

1. A small blanched whitish area develops in the epithelium.

2. Fluid fills the area, and a vesicle (blister) is formed.

3. Vesicle enlarges and may coalesce with adjacent ones.

4. Vesicle ruptures.

5. Vesicular covering sloughs leaving an eroded (red) area.

6. Gray fibrinous coating forms over the eroded area.

7. Coating becomes yellow, brown or green.

8. Epithelium is restored, but line of demarcation remains; line then gradually fades.

Occasionally "dry" FMD lesions develop. Instead of forming a vesicle, the fluid is apparently lost as it forms and the upper layers of the epithelium become necrotic and discolored. The lesion therefore appears necrotic rather than vesicular.

Gross Lesions on the Feet:

The vesicle in the interdigital space is usually large because of the stress on the epithelium caused by movement and weight. The lesion at the coronary band at first appears blanched; then there is separation of the skin and horn. When healing occurs, new horn is formed, but a line resulting from the coronitis is seen on the wall of the hoof.

Gross Cardiac and Skeletal Lesions:

Animals that die may have grayish or yellowish streaking in the myocardium - degeneration and necrosis. These findings are known as "tiger heart". Skeletal muscle lesions occur but are rare.

Swine

Vesicles on the snout can be large and filled with clear or bloody fluid. Mouth lesions are usually the "dry" type and appear as necrotic epithelium. Feet lesions are usually severe, and the hoof can become detached. Animals that die may have grayish or yellowish streaking in the myocardium with degeneration and necrosis ("tiger heart").

Sheep

Lesions in the mouth and vesicles on the coronary band may be few, small, and difficult to find. Animals that die may have grayish or yellowish streaking in the myocardium with degeneration and necrosis ("tiger heart").

Morbidity and Mortality

The morbidity rate is essentially 100 percent in a susceptible population of domestic animals. Mortality is usually less than 1 percent, but in young animals and with certain isolates mortality can be high. In an FMD outbreak in Israel, there was a high mortality (at least 50 percent) in wild mountain gazelles. The same virus caused typical low mortality in cattle. In the gazelles, there was a severe viral pancreatitis that accounted for the high mortality.

Diagnosis

Field Diagnosis

In cattle, FMD should be considered whenever salivation and lameness occur simultaneously and a vesicular lesion is seen or suspected. Fever often precedes other clinical signs; therefore, febrile animals should be carefully examined. Early diagnostic lesions may be found before animals start to salivate, have a nasal discharge, or become lame. To avoid missing a diagnosis, examine the mouth of a lame animal and the feet of any animal with signs or lesions involving the mouth or nostrils. Typically, FMD spreads rapidly and there is a high clinical attack rate; however, this cannot be counted upon, for a relatively avirulent strain could appear, or more resistant animals (sheep) could be affected.

In pigs, sheep, and goats, FMD should be considered when animals have sore feet, vesicular lesion is suspected, or both.

Specimens for Laboratory Diagnosis

Because the various vesicular diseases have similar clinical signs, a laboratory diagnosis is mandatory. Oral, nasal, foot, or mammary lesions are good sources of specimens. The following should be collected from each of two or three animals:

1. Vesicular fluid (as much as possible).

2. Epithelium covering a vesicle.

3. Flaps of epithelial tissue still attached.

(For 2 and 3 above, try to collect about 0.5 gm.)

Old necrotic or fibrinous material that is difficult to remove is undesirable and often is highly contaminated with bacteria.

4. About 5 ml of blood with anticoagulant (viremia ends about 5 days after the onset of disease).

5. Esophageal—pharyngeal (OP) fluid from convalescent cattle, sheep, or goats.

This should immediately be diluted with an equal volume of cell culture fluid (e.g., Hanks balanced salt solution with lactalbumin hydolysate) and shaken vigorously for about 1 minute. If the solution turns yellow, the pH is low and the virus could be inactivated; discard and collect another sample.

6. Blood for serum (10 ml of serum).

7. From dead animals, collect samples of epithelial lesions, lymph nodes, thyroid, adrenal gland, kidney, and heart (about 10 gm).

8. Full set of tissues in formalin.

If the specimens can be delivered to a laboratory within 24 hours, they should be placed on ice. If delivery will take longer, quickfreeze the specimens, and do not allow them to thaw during transit. If dry ice is used, be sure that the vials are tightly sealed with stopper and tape so that

no carbon dioxide enters the vial. The carbon dioxide will lower the pH and inactivate FMDV. Epithelium can also be placed in buffered glycerin and kept at 39° F (4° C) or -4° F (-20° C). Ratio of epithelium to glycerin should not exceed 1:10.

Laboratory Diagnosis

To confirm the initial case of FMD, the virus has to be isolated and identified. After confirmation of the initial case, diagnosis can be made by antigen or nucleic acid detection, or both.

Serological tests are available to detect antibody and differentiate infected and vaccinated animals.

Differential Diagnosis

Differential diagnosis for FMD should include vesicular stomatitis, swine vesicular disease, vesicular exanthema of swine, foot rot, and chemical and thermal burns. In cattle, oral lesions caused by rinderpest, infectious bovine rhinopneumonitis, bovine virus diarrhea, malignant catarrhal fever, and bluetongue can be similar to the later lesions in FMD. In sheep, lesions caused by bluetongue, contagious ecthyma, and lip and leg ulceration can be similar to the later lesions of FMD.

Vaccination

Starting about 1951, FMD vaccine was produced by the Frenkel method. Normal tongue epithelium was removed, minced, placed in a nutrient broth, and inoculated with FMDV. After replication of

FMDV, the virus was inactivated with formalin, and aluminum hydroxide was added as an adjuvant. This method as well as virus propagation in cell culture is being used today to produce FMD vaccine.

Outbreaks of FMD have been traced to use of formalin-inactivated vaccine. Apparently, in some cases, vaccine contained viable virus. Today (1996) the classical FMD vaccines are prepared using binary-ethyleneimine (BEI) inactivated virus and aluminum hydroxide-saponin or oil as an adjuvant. Double emulsion oil vaccines have been shown to produce an immunity of longer duration than aluminum hydroxide-saponin vaccine.

To date, molecular-engineered vaccines have not been as effective or as economical as the cell culture vaccines.

When vaccinating animals, it is important that the vaccine contain the same subtype of virus as is in the area. This necessitates frequent checking of the serotype and subtype during an outbreak because FMD virus frequently changes during natural passage through various species.

Protection induced by a good aluminum hydroxide vaccine decreases rapidly in 4-6 months. A double emulsion oil vaccine can protect for up to 1 year.

Vaccinated animals that are not completely protected can be a source of infection. The virus may replicate and be shed, but the animals may not show any clinical sign of infection.

Control and Eradication

The official attitude of a country regarding control of a disease depends on how seriously the disease affects the country, the financial and technical ability of the country, and what its neighbors are doing. The degree of control of FMD varies as follows:

1. Virtually no control in some Asian and African countries where FMD is enzootic.

2. Protection of valuable or accessible animals or vaccination along a border to provide a buffer zone. (May vaccinate cattle because of severity of the disease, but not sheep and goats.)

3. Large-scale vaccination and quarantine with or without slaughter of infected animals.

4. Regulatory measures to prevent entry of FMD virus and quarantine and implementation of an eradication program.

A country where FMD is endemic should be as concerned about introduction of FMD virus as a country that is free of FMD because the introduced virus may be a serotype to which the native animals have no immunity.

The following are the essential features of a control and eradication program:

1. Stop movement of animals and animal products in the area affected.

2. Slaughter infected animals (and known contact animals).

3. Destroy carcasses.

4. Disinfect vehicles leaving the infected area.

5. Perform vaccination.

If eradication by slaughter fails, vaccination may be used to control the outbreak. There are experimental results indicating that potent vaccine may induce significant immunity in 4 days to protect exposed cattle to FMD.

6. Inform and educate the community.

Most developed countries have detailed plans to deal with an outbreak of FMD.

Public Health

In a review of the zoonotic aspects of FMD by K. Bauer in 1997, he reported that, since 1921, FMD virus has been isolated and typed from slightly over 40 human cases (4). The cases occurred on three continents: Europe, Africa, and South America. Type O predominated, followed by C, and rarely A. Because infection is uncommon, FMD is not considered to be a public health problem.

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I. Swine Vesicular Disease

Definition

Swine vesicular disease (SVD) is an acute, contagious viral disease of swine caused by an enterovirus and characterized by fever and vesicles with subsequent erosions in the mouth and on the snout, feet, and teats.

Etiology

Swine vesicular disease virus is in the enterovirus group of picornaviruses and is closely related to the human enterovirus Coxsackie B-5 and unrelated to known porcine enteroviruses. Some researchers believe this is a case where a human pathogen transferred to pigs through the eating of human feces. The virion is a roughly spherical 28 nm single-stranded RNA virus. This pathogen is resistant over a wide pH range (2.5-12), relatively resistant to heat (inactivated at 157^{o} F [69^{o} C]), and persists for a long time (up to 2 years) in salted, dried, and smoked meat products.

Host Range

Pigs are the only natural host. Baby mice can be experimentally infected, and there has been accidental laboratory infection of humans.

Geographic Distribution

Swine vesicular disease first occurred in Italy and was subsequently recognized in Hong Kong, England, Scotland, Wales, Japan, Malta, Austria, Belgium, France, the Netherlands, Germany, Poland, Switzerland, Greece, and Spain. Outbreaks in the 1990's were reported in Italy, Spain, and Portugal.

Transmission

The disease can be introduced into a herd by feeding garbage containing infected meat scraps, by introducing infected animals, or by contacting infected feces (e.g., an improperly cleaned truck).

Recent outbreaks in Europe appeared after the introduction of animals that had no clinical sign of SVD, which indicates that there is a subclinical form of the disease. After the initial infection, the disease spreads through contact of susceptible pigs with infected pigs and infected feces.

Incubation Period

Signs of SVD develop in 2 to 3 days after eating contaminated feed and in 2 to 7 days after contact with infected pigs.

Clinical Signs

Clinical signs are very similar to those of foot-and-mouth disease and other vesicular diseases. There is a fever, vesicles in the mouth and on the snout and feet, and lameness, all of which are grossly indistinguishable from FMD. More suggestive of SVD is an unsteady gait, shivering, and chorea — (jerking) — type leg movements due to an encephalitis.

Gross Lesions

Vesicles are indistinguishable from those of foot-and-mouth disease, vesicular stomatitis, and vesicular exanthema of swine. See the foot-and-mouth disease chapter.

Morbidity and Mortality

Morbidity in SVD is lower, and lesions are less severe, than in foot-and-mouth disease. There is essentially no mortality in SVD.

Diagnosis

See chapter on foot-and-mouth disease.

Serology is complicated by cross reactions with other undefined porcine enteroviruses.

Differential Diagnosis

Differential diagnosis for SVD should include foot-and-mouth disease, vesicular stomatitis, vesicular exanthema of swine, and chemical and thermal burns.

Vaccination

There is no vaccine.

Control and Eradication

Prevention measures are similar to those for FMD: control of animals imported from infected areas, and sanitary disposal of garbage from international aircraft and ships

Eradication measures consist of quarantining infected farms and areas, slaughtering and disposing of infected and contact pigs, and cleaning and disinfecting infected premises.

Public Health

Human infection has been reported in laboratory personnel working with the virus. Caution should be taken when working with infected material.

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J. African Swine Fever

(Peste porcine Africaine, fiebre porcina Africana, maladie de Montgomery)

Definition

African swine fever (ASF) is a tickborne and contagious, febrile, systemic viral disease of swine
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Etiology

The ASF virus is a large (about 200 nm) lipoprotein-enveloped, icosahedral, double- stranded DNA virus. For many years the agent was classified as an iridovirus (3), but in recent years it was found to have many characteristics of poxvirus; thus, researchers have suggested establishment of a new family for ASF virus (ASFV) (19).

This virus is quite stable and will survive over a wide range of pH. In serum-free medium, ASFV is inactivated at pH 3.9 or lower and at pH 11.5 or higher. In the presence of 25 percent serum, ASFV will remain viable for 7 days at pH 13.4 (17). The virus will survive for 15 weeks in putrefied blood, 3 hours at 50^{o} C, 70 days in blood on wooden boards, 11 days in feces held at room temperature, 18 months in pig blood held at 4^{o} C, 150 days in boned meat held at 39^{o} F, and 140 days in salted dried hams (8A).

Over the years, ASFV isolates with lower virulence have emerged — particularly in the Iberian peninsula. Virulence of isolates varies from highly virulent (essentially 10 percent mortality in 7-10 days after exposure), to moderately virulent (acute illness in which a high percentage of the pigs survive) , to low virulence (only seroconversion occurs).

Host Range

Initially, domestic and wild pigs (Africa: warthog, bush pig, and giant forest hog; Europe: feral pig) were thought to be the only hosts of ASFV (1,16). In 1963, Spanish workers isolated ASFV from the soft tick *Ornithodoros erraticus* collected from ASF-infected farms (13). Subsequently, researchers showed that ASFV replicates in the tick and that there is transstadial, transovarial, and sexual transmission in *Ornithodoros* ticks. *O. moubata* collected from warthog burrows in Africa were shown to be infected with ASFV (5). African swine fever in wild pigs in Africa is now believed to cycle between soft ticks living in warthog burrows and newborn warthogs (18). *Ornithodoros* ticks collected from Haiti, the Dominican Republic, and southern California have been shown to be capable vectors of ASFV (4,5), but in contrast to the African ticks, many of the ticks from California died after being infected with ASFV. Many researchers believe that ASFV is really a tick virus and the pig is an accidental host (11).

Because ASFV-infected ticks can infect pigs, ASFV is the only DNA virus that can qualify as an arbovirus.

Geographic Distribution

African swine fever is present in several African countries and on the island of Sardinia.

Transmission

Even though the soft tick has been shown to be a vector (and in Africa probably the reservoir of ASFV), the primary method of spread from country to country has been through the feeding of uncooked garbage containing ASFV-infected pork scraps to pigs. Once a pig becomes infected, ASFV spreads by direct contact, and contaminated people, equipment, vehicles, and feed. The role of carrier pigs has been difficult to prove experimentally, but circumstantial evidence from the field incriminates carrier pigs. An outbreak of ASF in a contained swine operation in Africa was traced to workers feeding the entrails of guinea fowl to pigs. It was shown that the guinea fowl feed on soft ticks; thus, ASFV was present in the guinea fowl intestines fed to the pigs.

The amount of ASFV needed to infect a pig depends on the route of exposure. Experimentally, a pig can be infected by intramuscular or intravenous inoculation with a 0.13 hemadsorbing dose (HAD_{50}); intranasal-oral inoculation required 18,200 HAD_{50}.

In an ASF endemic area where there are soft ticks, ticks can be the source of infection. However, in these areas in Africa, pigs can be very successfully raised in confinement with double fencing, proper isolation, and sanitary procedures. In Africa, the production system with the highest risk of ASF is the village pig, for these pigs roam. The owners do not practice isolation procedures when the pigs are confined.

In other areas, the disease has to be introduced by infected live pigs or by feeding uncooked garbage containing ASFV-infected pork. Once the disease is introduced into a herd, it spreads by direct and indirect contact with secretions and excretions from infected pigs. Aerosol transmission is not important in the spread of ASF. Because ASFV does not replicate in epithelial cells, the amount of virus shed by an ASF-infected pig is much less than the amount of virus shed by a hog-cholera-infected pig. The blood of a recently infected pig contains a very high ASFV titer: $10^{5.3}$ to $10^{9.3}$ HAD_{50} per milliliter (7). Therefore, if pigs fight, an infected pig develops bloody diarrhea, or an infected pig is necropsied, blood is shed, and there is massive environmental contamination.

Piglets born of ASF-convalescent dams are free of ASFV and ASF antibody at birth but seroconvert after ingesting colostrum (14,15). When piglets from noninfected (control) and ASF-convalescent dams were challenge-inoculated when 7 weeks old, the control piglets developed an average viremia of $10^{7.6}$ and died, whereas the piglets from convalescent gilts developed an average viremia of $10^{4.9}$ and survived. However, because of persistent infection by ASFV, reestablishing a herd using pigs from convalescent animals will not result in an ASFV- free herd. When farmers in Cameroon repopulated their herds using ASF-convalescent animals, the herds experienced recurring periods of high mortality due to ASF.

Incubation Period

After intranasal-oral exposure, pigs usually develop fever and leukopenia in 48 to 72 hours.

Clinical Signs

Highly and Moderately Virulent ASF Isolates

The clinical signs of ASF are influenced by the virulence of the virus and the physiological state (age and pregnancy) of the pig. After inoculation of feeder pigs with either a highly virulent or moderately virulent isolate, the clinical course for both isolates is similar for the first 4-6 days post infection. About 2 DPI, the pigs will develop a fever of 105-107^{o} F (40.5-41.7^{o} C) and white pigs will have a reddened skin, moderate anorexia, and leukopenia. When disturbed the pigs will get up and move about but if left alone will after a short time lie down.

After 4-6 DPI, a difference between the pigs inoculated with the different isolates will become apparent.

Highly Virulent Isolate

The pigs become progressively sicker (eat and move less), and most die between 7 and 10 DPI. It is not unusual to see a pig walking and a short time later to find it dead.

Moderately Virulent Isolate

Pigs infected by moderately virulent ASFV usually have a high fever for 10-12 DPI. Some mortality usually occurs at this time. After 12-14 DPI, temperatures and leukocyte counts start to return to normal levels. It is not unusual to have one or more pigs die as early as 7-8 DPI, but when these pigs are necropsied, the cause of death is frequently hemorrhage into the stomach; the underlying mechanism of death was that ASFV infection caused a thrombocytopenia, resulting in a prolonged bleeding time and hemorrhage from a preexisting gastric ulcer (2). Very young pigs may have a high mortality and have lesions similar to infection by highly virulent virus.

Pigs affected with either isolate, in addition to the reddened skin, may develop dark red to purple discoloration of the skin on the ears, tail, extremities of the legs, or skin on the hams. This is a nonspecific sign also seen in other diseases. Some groups of pigs will develop diarrhea; this is probably due to disturbed gut physiology and flora rather than a direct effect of the virus because the virus does not replicate in epithelium. In contrast to Classical Swine Fever (hog cholera), ASFV-infected pigs do not develop a conjunctivitis or encephalitis, and, despite the high fever, the ASFV- infected pigs stay in good condition, whereas classical swine fever - infected pigs quickly lose much weight.

Pregnant animals infected with a high-, moderate-, or low-virulence ASF isolate abort.

Low Virulence Isolates

Non-pregnant animals infected by certain low-virulence ASFV may only seroconvert; pregnant animals will abort.

Other low-virulence ASFV isolates will cause a low fever for 2-3 weeks and then reddened areas 1 cm^2 to many centimeters in size may develop in the skin. These areas then become raised and necrotic. These pigs may also have painless enlargements of joints—particularly the carpal and tarsal joints. This form is referred to as chronic ASF (10). Many of these pigs will have recurring episodes of a more acute disease and eventually die during an acute episode.

Gross Lesions

Highly Virulent ASFV Infection

Pigs that die peracutely from an infection with a highly virulent ASFV may have poorly developed lesions. Animals that die 7 or more DPI have more classic lesions. Three lesions most consistently found and highly suggestive of ASF infection are as follows:

- Greatly enlarged dark red to black friable spleen
- Very enlarged hemorrhagic gastrohepatic lymph nodes
- Very enlarged hemorrhagic renal lymph nodes.

Other lesions described for ASF are more variable and are as follows:

- Dark red to purple areas of skin on ears, feet, and tail
- Petechial hemorrhages on serosal surfaces
- Petechial to ecchymotic hemorrhages in the renal cortex
- Perirenal edema
- Edema of the gall bladder
- Swollen liver
- Edema of the lung.

In pigs infected orally, the submandibular lymph node may be enlarged and have some hemorrhage. Other peripheral lymph nodes may have only edema.

Moderately Virulent Virus

The gross lesions 8-12 DPI in pigs infected with a moderately virulent ASFV are similar to those infected by a highly virulent ASFV. The main difference in the lesions between these two types of isolates is that in infections by a moderately virulent ASFV, the spleen although enlarged, has a more normal color and is not friable.

Low Virulent Virus

The most common lesions in chronic ASF are necrotic skin lesions, consolidated lobules in the lung, generalized lymphadenopathy, swollen joints, and pericarditis.

Aborted fetuses may be anasarcous, and there may be petechial hemorrhages in the placenta, skin, and myocardium, and a mottled liver.

Morbidity and Mortality

The warthog and bush pig develop a viremia but have a very mild or subclinical disease, whereas ASF infection in domestic pigs and European feral pigs can cause a high mortality.

Morbidity in a previously unexposed herd will usually be 100 percent in pigs that have contact with each other. Mortality varies with the virulence of the isolate. Highly virulent isolates will cause about a 100 percent mortality. Infection by lesser virulent isolates can cause mortality that varies from a low percentage to 60-70 percent. Factors that can increase mortality in infections by the lesser virulent isolates are concurrent disease, a young age, and pregnancy.

Diagnosis

Field Diagnosis

The highly virulent form of ASF will be easiest to diagnose because essentially 100 percent of the pigs will die. African swine fever caused by the lesser virulent isolates will be more difficult to diagnose but should always be suspected when there are febrile pigs and necropsy findings include the following:

Greatly enlarged dark red to black spleen
Very enlarged hemorrhagic gastrohepatic lymph nodes
Very enlarged hemorrhagic renal lymph nodes.

African swine fever has frequently been misdiagnosed as classical swine fever . In contrast to classical swine fever , ASFV-infected pigs do not develop a conjunctivitis or encephalitis, and despite the high fever, the ASFV-infected pigs stay in good condition. In contrast, classical swine fever -infected pigs are severely depressed and quickly lose much weight; moreover, they usually have a foul smelling diarrhea.

Specimens for Laboratory

The ASFV is present in the blood starting about 2 DPI. In infections by lesser virulent isolates, ASFV can usually be isolated from the blood for 25 or more DPI. Specimens for laboratory diagnosis are as follows:

- Heparinized blood
- Clotted blood or serum
- Submandibular lymph node
- Inguinal lymph node
- Tonsil
- Spleen
- Gastrohepatic lymph node
- Lung
- Liver
- Kidney

Bone marrow should be submitted if there are considerable postmortem changes.

The specimens should be shipped refrigerated or frozen. Pieces of the tissues listed above, the brain, and any other gross lesion should be submitted in 10 percent buffered formalin.

Aborted fetuses are usually free of virus; therefore, it is necessary to submit a blood sample from the dam.

Laboratory Diagnosis

The initial diagnosis of ASF in a free area requires isolation and identification of the virus. After the initial diagnosis, confirmation of a diagnosis can be made by demonstrating ASF antigen in tissue or ASF antibody.

Differential Diagnosis

Differential diagnoses for ASF should include classical swine fever, erysipelas, salmonellosis, and eperythrozoonoisis.

Vaccination

There is no vaccine.

Control and Eradication

Prevention

Introduction of the disease into free areas can be prevented by cooking all garbage fed to pigs (this applies to commercial and backyard pigs and pets [potbellied pigs]) and importing only ASF-disease free pigs.

Eradication

Control and eradication of ASF in developed countries can be accomplished by slaughter and disposal of all acutely infected pigs, widespread testing and elimination of all seropostive animals, and good herd isolation and sanitary practices.

Today (1996), ASF is not as great a threat to the United States as it was several years ago. The major pork-exporting countries have eradicated the disease in domestic pigs.

Public Health

Human beings are not susceptible to ASFV infection.

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K. Classical Swine Fever (Hog Cholera)

(Note: The preferred term for this disease is now classical swine fever.)

(Classical swine fever, peste du porc, colera porcina, Virusschweinepest)

Definition

Classical Swine Fever (CSF), formerly known as Hog cholera (HC) is a highly contagious viral disease of swine that occurs in an acute, a subacute, a chronic, or a persistent form. In the acute form, the disease is characterized by high fever, severe depression, multiple superficial and internal hemorrhages, and high morbidity and mortality. In the chronic form, the signs of depression, anorexia, and fever are less severe than in the acute form, and recovery is occasionally seen in mature animals. Transplacental infection with viral strains of low virulence often results in persistently infected piglets, which constitute a major cause of virus dissemination to noninfected farms.

Etiology

Although minor antigenic variants of CSF virus have been reported, there is only one serotype. CSF virus is a lipid-enveloped pathogen belonging to the family Flaviviridae, genus *Pestivirus*. The organism has a close antigenic relationship with the bovine viral diarrhea virus (BVDV) and the border disease virus (BDV), as demonstrated in the immunodiffusion and immunofluorescence tests. The serum neutralization test can, however, differentiate between HCV and BVDV. In a protein-rich environment, HCV is very stable and can survive for months in refrigerated meat and for years in frozen meat. The virus is sensitive to drying (desiccation) and is rapidly inactivated by a pH of less than 3 and greater than 11.

Host Range

The hosts of HCV are the pig and wild boar.

Geographic Distribution

According to the FAO—WHO—OIE Animal Health Yearbook 1989, HC (CSF) is recognized in 36 countries and is suspected of being present in another 2. The disease has been eradicated in Australia, Canada, and the United States. Constant progress toward eradication has been made in the countries of the European Economic Community since the guidelines for CSF control in individual member states were accepted in 1980.

Transmission

The pig is the only natural reservoir of CSF virus. Blood, tissues, secretions and excretions from an infected animal contain CSF virus. Transmission occurs mostly by the oral route, though infection can occur through the conjunctiva, mucous membrane, skin abrasion, insemination, and percutaneous blood transfer (e.g., common needle, contaminated instruments). Airborne transmission is not thought to be important in the epizootiology of CSF, but such transmission could occur between mechanically ventilated units within close proximity to each other.

Introduction of infected pigs is the principal source of infection in CSF-free herds. Farming activities such as auction sales, livestock shows, visits by feed dealers, and rendering trucks are also potential sources of contagion. Feeding of raw or insufficiently cooked garbage is a potent source of CSF virus. During the warm season, CSF virus may be carried mechanically by insect vectors that are common to the farm environment. There is no evidence, however, that CSF virus replicates in invertebrate vectors. Husbandry methods also play an important role in CSF transmission. Large breeding units (100 sows) have a higher risk of recycling infection than small herds. In large breeding units where continuous farrowing is practiced, strains of low virulence may be perpetuated indefinitely until the cycle is interrupted by stamping-out procedures and a thorough cleaning and disinfection are carried out.

Incubation Period

The incubation period is usually 3 to 4 days but can range from 2 to 14 days.

Clinical Signs

The clinical signs of CSF are determined by the virulence of the strain and the susceptibility of the host pigs. Virulent strains cause the acute form of the disease, whereas strains of low virulence induce a relatively high proportion of chronic infections that may be inapparent or atypical. These strains are also responsible for the "carrier-sow" syndrome from which persistently infected piglets are produced.

Acute Classical Swine Fever

In acute CSF, the pigs look and act sick. Their disease progresses to death within 10 to 15 days, and remissions are rare. In an affected herd, some pigs will become drowsy and inactive and will stand with arched backs. Other pigs will stand with drooping heads and straight tails. Some pigs may vomit a yellow fluid containing bile. The sick pigs will huddle and pile up on each other in the warmest corner of the enclosure and will rise only if prompted vigorously. Anorexia and constipation will accompany a high fever that may reach 108° F (42.2° C) with an average of 106° F (41.1° C). Pigs may continue to drink and may have diarrhea toward the end of the disease process. Conjunctivitis is frequent and is manifested by encrustation of the eyelids and the presence of dirty streaks below the eyes caused by the accumulation of dust and feed particles. Sick pigs become gaunt and have a weak, staggering gait related to posterior weakness. In terminal stages, pigs will become recumbent, and convulsions may occur shortly before death. In the terminal stage, a purplish discoloration of the skin may be seen; if present, the lesions are most numerous on the abdomen and the inner aspects of the thighs.

Chronic Classical Swine Fever

Chronic CSF is characterized by prolonged and intermittent disease periods with anorexia, fever, alternating diarrhea and constipation, and alopecia. A chronically infected pig may have a disproportionately large head relative to the small trunk. These runt pigs may stand with arched backs and their hind legs placed under the body. Eventually, all chronically infected pigs will die.

Congenital Classical Swine Fever

Congenital CSF virus infection by virulent strains will likely result in abortions or in the birth of diseased pigs that will die shortly after birth. Transplacental transmission with low-virulence strains may result in mummification, stillbirth, or the birth of weak and "shaker" pigs. Malformation of the visceral organs and of the central nervous system occurs frequently. Some pigs may be born virtually healthy but persistently infected with CSF virus. Such infection usually follows exposure of fetuses to CSF virus of low virulence in the first trimester of fetal life. Pigs thus infected do not produce neutralizing antibodies to CSF and have a lifelong viremia. The pigs may be virtually free of disease for several months before developing mild anorexia, depression, conjunctivitis, dermatitis, diarrhea, runting, and locomotive disturbance leading to paresis and death. In breeding herds affected with low virulence strains of CSF virus, poor reproductive performance may be the only sign of disease.

Gross Lesions

Acute Classical Swine Fever

The most common lesion observed in pigs dying of acute CSF is hemorrhage. Externally, a purplish discoloration of the skin is the first observation. There may be necrotic foci in the tonsils. Internally, the submandibular and pharyngeal lymph nodes are the first to be affected and become swollen owing to edema and hemorrhage. Because of the structure of the pig lymph node, hemorrhages are located at the periphery of the node. As the disease progresses, the hemorrhage and edema will spread to other lymph nodes. The surface of the spleen, and particularly the edge of the organ, may have raised, dark wedge-shaped areas. These are

called splenic infarcts. Infarcts are frequently observed in pigs infected experimentally with older strains of CSF virus but are less commonly seen with the contemporary strains.

Pinpoint to ecchymotic hemorrhages on the surface of the kidney are very common in CSF. Such lesions are easier to see in the decapsulated kidney. Hemorrhages are also found on the surface of the small and large intestine, the larynx, the heart, the epiglottis, and the fascialata of the back muscles. All serous and mucosal surfaces may have petechial or ecchymotic hemorrhages.

Accumulation of straw-colored fluids in the peritoneal and thoracic cavities and in the pericardial sac may be present.

The lungs are congested and hemorrhagic and have zones of bronchopneumonia.

Chronic Classical Swine Fever

In chronic CSF, the lesions are less severe and are often complicated by secondary bacterial infections. In the large intestine, button ulcers are an expression of such a secondary bacterial infection. In growing pigs surviving for more than 30 days, lesions may be seen at the costo-chondral junction of the ribs and at the growth plates of long bones.

Congenital Classical Swine Fever

In pigs infected transplacentally with CSF virus strains of low virulence, the most commonly seen lesions are hypoplasia of the cerebellum, thymus atrophy, ascites, and deformities of the head and of the limbs. Edema and petechial hemorrhages of the skin and of the internal organs are seen at the terminal stage of the disease.

Morbidity and Mortality

In acute CSF, the morbidity and mortality are high.

Diagnosis

Field Diagnosis

Septicemic conditions in which pigs have high fever should be investigated carefully. A thorough history from the herd owner should be obtained to determine if raw garbage was fed, if unusual biological products were used, or if recent additions were made to the herd. Careful observation of the clinical signs and of the necropsy lesions should be recorded. In acute CSF, it is helpful to necropsy four or five pigs to increase the probability of observing the representative lesions.

A marked leukopenia is detectable at the time of initial rise in body temperature and persists throughout the course of the acute and chronic disease. This feature was once widely used in the field diagnosis of CSF. Nowadays, with the development of more specific laboratory diagnostic methods, which are aimed at demonstrating the virus or its structural antigens in tissues or at detecting specific antibodies in the serum, the white blood count is not as widely used. In endemic areas it could be helpful.

Specimens for Laboratory

For virus isolation and antigen detection, the tonsils are considered essential. In addition, submandibular and mesenteric lymph nodes, spleen, kidneys, and the distal part of the ileum should be collected. In live pigs, tonsil biopsies and whole blood collected with anticoagulants are useful to diagnose CSF. Sample collection should be targeted to pigs having fever or showing other signs of the disease. Each sample of tissue should be placed in a separate plastic bag and identified. The samples should not be frozen (interference with fluorescent antibody tissue section test) but kept at refrigeration temperature. The material should be transported and stored in leak-proof containers in accordance with national regulations for transportation of diagnostic biologic samples.

Serum samples for antibody detection should be collected from animals that have recovered from suspected infection or from sows known to have been in contact with infected or suspected cases. A sufficient number of samples should be collected to ensure a high probability of detecting infection.

A complete set of tissues, including the whole brain, should be submitted in 10 percent buffered formalin.

Laboratory Diagnosis

Any clinical diagnosis of CSF must be confirmed by the submission of specimens to a specialized diagnostic laboratory that should also have the capability to distinguish between CSF and African swine fever.

The laboratory diagnostic procedures for CSF have evolved in parallel with the emergence of new technologies. Until the 1960's, laboratory diagnosis was restricted to recognition of gross lesions and confirmation by histopathology. Inoculation of susceptible pigs was often used as final confirmatory test and to determine the virulence of the viruses. Numerous laboratory techniques have been described to diagnose CSF, but only a few have gained international acceptance and have been integrated into national CSF control programs. Only these will be discussed in this presentation.

In the fluorescent antibody tissue section test (FATST), direct fluorescent antibody technique is applied to detect CSF viral antigens in frozen tissues of organs from dead pigs, in biopsy material, or in impression smears. Theoretically, a diagnosis can be confirmed within hours from the reception of the specimen. In countries where the disease has been eradicated, the diagnosis of the "index case" by the FATST alone may be difficult, and confirmation in cell culture may be needed. The FATST may not differentiate CSF from BVDV infection; an accurate distinction between the two viruses has to be made before releasing a final diagnosis. Differentiation between CSFV and BVDV can readily be made with the immunoperoxidase test using monoclonal antibodies or the serum neutralization test.

The isolation of CSFV in cell culture and the identification using fluorescein-labeled hog cholera antibody (fluorescent antibody cell culture test) can provide confirmation in cases where the results of investigation of frozen tissue sections are inconclusive.

As control measures for CSF are implemented in a country, virulent strains of CSFV will be reduced, and there will be a relative increase of low-virulence strains. As the proportion of subclinical cases in a national herd increases, it will become increasingly difficult to recognize

the disease. The antigen detection systems previously described become less effective; thus, serological tests are essential for a successful control and eventual eradication program.

Approximately 75 percent of pigs infected with acute CSF have microscopic lesions of an encephalitis characterized by perivascular cuffing, endothelial proliferation, and microgliosis. This feature is easily recognized in a nonspecialized diagnostic laboratory and may constitute the most important single factor that will cause the pathologist to suspect CSF.

Differential Diagnosis

Differential diagnosis of CSF should include African swine fever, erysipelas, salmonellosis, eperythrozoonosis, and salt poisoning.

Vaccination

Over the years, numerous regimens of vaccination have been advocated with a variable degree of success. In the past two decades, modified live vaccines (MLV) with no residual virulence for pigs have become available. The lapinized Chinese (C) strain, the Japanese guinea pig cell culture-adapted strain, and the French Thiverval strain have been widely used. All three strains are considered innocuous for pregnant sows and piglets over 2 weeks old.

Control and Eradication

In countries where CSF is enzootic, a systematic vaccination program is effective in preventing losses. Experience in the United States and in some countries of the European Union has proven that a strict regimen of vaccination will reduce the number of outbreaks to a level at which complete eradication by sanitary measure alone will be feasible. At that point, vaccination must be stopped. A successful eradication program requires a massive input of funds from a central government and cooperation from the government, the swine industry, and the veterinary profession. Eradication measures will be assisted by strictly enforcing the garbage cooking laws, having an effective swine identification system, and using serological surveys targeted primarily to breeding sows to detect subclinical infections.

In countries where CSF has been eradicated and in which the threat of reintroduction is significant, it is essential to initiate an effective serological monitoring system. Sampling may be limited to strategic locations such as the border of an infected neighbor country or be intensified to target populations such as the garbage-fed herds. Such a system has been in effect in the United States since successful eradication in 1976; several thousand samples have been accessed annually.

Public Health

Human beings are not susceptible to CSFV infection.

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L. Bovine Spongiform Encephalopathy

Definition

Bovine spongiform encephalopathy (BSE), widely known as "mad cow disease," is a chronic, afebrile, degenerative disease affecting the central nervous system (CNS) of cattle.

Bovine spongiform encephalopathy belongs to the family of diseases known as the transmissible spongiform encephalopathies (TSE's). These diseases are caused by a transmissible agent that is yet to be fully characterized. They share the following common characteristics:

a. A prolonged incubation period of months or years;

b. A progressive debilitating neurological illness that is always fatal;

c. When examined by electron microscopy, detergent-treated extracts of brain tissue from animals or humans affected by these diseases reveal the presence of scrapie-associated fibrils (SAF's);

d. Pathological changes appear to be confined to the CNS and include vacuolation and astrocytosis;

e. The transmissible agent elicits no detectable specific immune response in the host.

Specific types of TSE's include scrapie, which affects sheep and goats; transmissible mink encephalopathy; feline spongiform encephalopathy; chronic wasting disease of deer and elk; and five rare diseases in humans: kuru, Creutzfeldt-Jakob disease (CJD), Gerstmann-Sträussler-Scheinker syndrome, fatal familial insomnia (FFI), and new variant Creutzfeldt-Jakob disease (nvCJD).

Etiology

The clinical, pathological, and molecular genetic features of BSE, as well as other transmissible spongiform encephalopathies, have led to speculation on the nature of the etiologic agent and the pathogenic mechanisms of the disease. There are three main theories on the nature of the scrapie agent:

1. The virus theory, in which the virus would have to have unusual biochemical and biophysical characteristics that would help explain the remarkable physicochemical properties (12, 24, 39, 40).

2. The prion theory, in which the agent is conceived of being composed exclusively of a host-coded normal cellular protein (PrP^{c}) that becomes partially protease resistant (PrP^{BSE}) — most likely through a post-translational conformation change after infection. In this theory there is no nonhost component of the agent. That is, a specific informational molecule (nucleic acid e.g., RNA or DNA) is not present (5, 36).

3. The virino theory, which states that the agent consists of a host-derived protein coat, (PrP being one of the candidates for this protective protein) and a small noncoding regulatory nucleic acid (14, 21).

All of the proposed theories have some degree of validity. Proponents of the virus and virino theories have concluded that the existence of different scrapie strains unequivocally proves the presence of a nucleic acid component of the infectious agent which, as in conventional viruses, may undergo mutations responsible for phenotypic variations. The problem with these theories is that no agent-specific nucleic acid has been convincingly identified to copurify with infectivity (15, 25, 28, 32, 42). Moreover, chemical, enzymatic, or physical treatments that usually inactivate or degrade nucleic acids have no effect on the transmissible properties of the infectious agent (3, 4, 27, 31). Possible reasons for this are that the amount of nucleic acid of the putative agent is too small to be detected with available techniques and that its tight bond to the protein protects it from chemical or physical inactivation. Also weakening the virus and virino theories is the inability to identify any virus particles under the electron microscope (6, 10), and the failure of an infected host to generate an immune response. Recently small particles resembling virus structures have been observed by electron microscopy (33).

The prion model involves propagation of a protein-only agent (PrP^{BSE}) whereby PrP^{c} can assume various tertiary structures caused by a combination of host genetics and the introduction of altered (infectious) PrP (PrP^{BSE}). More simply stated, the structure of the infecting PrP^{BSE} imprints upon the normal cellular precursor (PrP^{c}) and results in a conformation change to the protease-resistant form. It is suspected that "strain" differences result from mutations in the PrP gene that may cause proteins "flip" and change shape. Several explanations for scrapie strain genetics in the context of the prion theory have been suggested but none have been proven (35, 41, 46).

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

Regardless of whether the prion (PrP^{BSE}) is or is not the etiologic agent, the partially protease-resistant form of the prion protein is a marker of infection.

Host Range

Bovine spongiform encephalopathy has been experimentally transmitted to the following species via intracerebral (IC) inoculation: cattle, sheep, and goats (17), mink (38), pigs (13), marmosets (1), macaques (22), and mice (16). Intracerebral transmission was attempted in hamsters but was not successful. Via the oral route, BSE has been successfully transmitted to cattle, sheep, and goats (17); mice (2); and mink (38). Oral transmission has not been successful in swine. Parenteral and oral transmission has also been attempted in chickens with no evidence of disease thus far.

A transmissible spongiform encephalopathy has been diagnosed in eight species of captive wild ruminants as well as exotic (cheetahs, pumas, a tiger, and an ocelot) and domestic cats. There have been about 81 domestic cat cases of feline spongiform encephalopathy (FSE) in Great Britain and in 1 domestic cat each in Norway, Northern Ireland, and Liechtenstein. The agent isolated from several of these cases using strain typing in mice is indistinguishable from BSE in cattle, which suggests that FSE is actually BSE in exotic and domestic cats. This also appears to be true for the other ruminants. Epidemiological evidence suggests BSE-contaminated feed to be the primary source of infection in these species (30).

Other cases of spongiform encephalopathy have been reported in kudu, eland, nyala, gemsbok, and a few exotic cats. These too are thought to be linked to contaminated feed.

It has also been suggested that 23 cases (as of January 31, 1998) of a variant form of CJD (nvCJD) (a human disease) in Great Britain (U.K. Department of Health, March 2, 1998) and 1 case in France may be linked to exposure to BSE before the introduction of a specified bovine offal (SBO) ban at slaughter in 1989. The SBO ban excludes from human consumption brain, spinal cord, and other tissues with potential BSE infectivity.

Geographic Distribution

Worldwide there have been more than 170,000 cases since the disease was first diagnosed in 1986 in Great Britain. Over 95 percent of these cases have occurred in the United Kingdom. The disease has also been confirmed in native-born cattle in Belgium, France, Ireland, Luxembourg, the Netherlands, Northern Ireland, Portugal, and Switzerland. One case has been reported in the United States (Washington state, December 2003).

Transmission

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

Changes in rendering operations in the early 1980's — particularly the removal of a solvent-extraction process that included a steam-heat treatment — may have played a part in the appearance of the disease and the subsequent amplification of the agent in the food chain. A

ban on feeding animal protein of ruminant origin to ruminants was enacted in Great Britain in July 1988(50).

In Great Britain the epidemic peaked in 1992-93, when approximately 1,000 cases were being reported per week. In 1998 it remains on the decline with approximately 100 cases reported per week. Cases that have been detected in other countries appear be a result of importations of live cattle or, more significantly, contaminated feed from Great Britain.

There is no evidence that BSE spreads horizontally; that is, by contact between unrelated adult cattle or from cattle to other species.

New evidence suggests that maternal transmission may occur at an extremely low level. Results of British research show low levels of transmission of BSE from affected cows to their offspring. These results demonstrated that there is approximately a 9 percent increase in the occurrence of BSE in offspring of BSE-affected dams as compared with calves born to dams where BSE was not detected. The study did not ascertain if this was the result of genetic factors or true transmission. The research did, however, point out that, at this level, if maternal transmission does occur, it alone will not sustain the epidemic (51).

In the naturally infected animals, the agent has been identified by mouse bioassay in the brain, spinal cord, and retina. The route of inoculation into the mice was intracranial. The naturally infected animals were adult cattle exhibiting clinical signs of disease (16).

Mice fed milk, mammary gland, placenta, lymph nodes, or spleen have failed to develop the disease or to establish subclinical infection of the lymphoreticular system within their natural lifespan (29).

Another study was conducted to examine the pathogenesis of BSE in cattle; that is the replication (tissue distribution) of the agent during the incubation period. This study, which has not yet been completed, has identified the agent via mouse bioassay in the distal ileum of the experimentally infected calves. It is thought that the agent may be associated with the lymphoid tissue of the intestines. The calves were 4 months of age at the time of oral dosing. First isolation of the agent in the distal ileum was made at 6 months after oral dosing. Subsequent isolations from the distal ileum were made at 10, 14, and 18 months after dosing (47). Recently this study has also identified infectivity in bone marrow, trigeminal ganglion, dorsal root ganglion, brain, and spinal cord (48).

No infectivity has been found by parenteral or oral challenge, or both, in over 40 other tissues from clinically ill cattle using the mouse bioassay. It appears as if the distribution of the BSE agent is not as diverse as the scrapie agent in sheep. However, there is a possibility that the agent is present but is at such low levels that the bioassay is not sensitive enough to detect it (30).

Incubation Period

The incubation period usually ranges from 2 to 8 years. Following the onset of clinical signs, the animal's condition gradually deteriorates until the animal becomes recumbent, dies, or is destroyed. This usually takes from 2 weeks to 6 months. Most cases in Great Britain have occurred in dairy cows (Friesians) between 3 and 6 years of age (50). The youngest confirmed

case occurred in a 20-month-old heifer, and the oldest case was found in a cow 18 years of age.

Clinical Signs

Cattle affected by BSE develop a progressive degeneration of the nervous system. Affected animals may display changes in temperament, abnormalities of posture and movement, and changes in sensation. More specifically, the signs include apprehension, nervousness or aggression, incoordination, especially hind-limb ataxia, tremor, difficulty in rising, and hyperaesthesia to sound and touch. In addition, many animals have decreased milk production or loss of body condition, or both, despite continued appetite.

Gross Lesions

There is no gross lesion associated with BSE.

Morbidity and Mortality

In Great Britain, 19 percent of the dairy herds and 1.6 percent of the beef herds have had one or more cases of BSE. This difference is believed to result from the fact that dairy calves were fed a higher level of protein supplement. The average incidence in herds in Great Britian has been 1.75 cases. However, there have been a few herds with over 30 cases. Affected animals die.

Diagnosis

Field Diagnosis

A field diagnosis of BSE is based on the occurrence of clinical signs of the disease. A bovine animal that has signs of a CNS disturbance should be observed over time (at least 2 weeks) to determine whether the signs become progressively more severe. If, after this interval, improvement or recovery has not taken place, BSE should be suspected and the animal humanely euthanized. As a USDA FSIS Veterinarian, you will not be diagnosing BSE in this manner.

Specimens for Laboratory

Because the BSE agent is considered a human pathogen, protective clothing, gloves, and face protection should be worn when performing the necropsy. The entire brain should be removed intact with a portion of the cranial cervical spinal cord attached. Portions should be placed in a plastic bag and submitted unfixed. The remainder of the brain should be fixed in 10 percent buffered formalin solution. One cerebral hemisphere is removed by cutting the brain stem through the space between the cerebellum and cerebrum with a longitudinal cut between the cerebral hemispheres.

Laboratory Diagnosis

Bovine spongiform encephalopathy currently must be confirmed by histopathological examination of brain tissue. Bilaterally symmetrical degenerative changes are usually seen in the gray matter of the brain stem. These changes are characterized by vacuolation or microcavitation of nerve cells in the brain stem nuclei. The neural perikarya and axons of certain brain stem nuclei contain intracytoplasmic vacuoles of various sizes, that give the impression of a spongy brain. Hypertrophy of astrocytes often accompanies the vacuolation (49). A diagnosis may also be made by the detection of SAF's using electron microscopy.

Two supplemental tests are available to enhance the diagnostic capabilities for BSE. These are immunohistochemistry and the Western blot technique. In the past, if the brain tissue was not harvested shortly after the animal's death, autolysis often made it very difficult to confirm a diagnosis by histopathology. These tests allow for the possibility of confirming a diagnosis of BSE by detecting PrP^{BSE} even if the brain has been frozen or autolyzed .

Differential Diagnosis

Differentials for BSE include rabies, listeriosis, nervous ketosis, milk fever, grass tetany, lead poisoning, and other toxicities or etiological agents affecting the nervous or musculoskeletal system of adult cattle.

Treatment

There is no known treatment for BSE or any of the TSE's.

Vaccination

There is no preventative vaccine.

Control and Eradication

Bovine spongiform encephalopathy from foreign sources may be prevented by the implementation of import regulations prohibiting live ruminants and ruminant products (especially meat, bone meal, and offal) from countries where BSE may exist. Because the origin of BSE remains unknown, preventing an epidemic of BSE would involve, at a minimum, the prohibition of feeding ruminant proteins to ruminants. The prevention program of any country should also include an active surveillance effort focused on high-risk cattle for the early detection of BSE. Most countries of the world have prohibited the importation of cattle and bovine products from countries known to have BSE. In addition many countries have taken steps to enact regulations prohibiting the feeding of ruminant proteins to ruminants. This is true even in countries such as Australia and New Zealand with no known animal TSE's.

Agricultural officials in countries known to have BSE have taken a series of actions to control and, it is to be hoped, eradicate BSE. These include making BSE a notifiable disease, prohibiting the inclusion of certain animal proteins in ruminants' rations (the feed bans vary

depending on the amount of BSE detected), and depopulating certain populations of cattle thought to be of higher risk because of epidemiological findings.

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

U.S. Actions

With an active surveillance program in place for 8 years, BSE has been detected in one cow in the United States. The United States Department of Agriculture (USDA), Food and Drug Administration (FDA), and industry groups are actively working to prevent any additional cases. The measures USDA, Animal and Plant Health Inspection Service (APHIS), has taken in this regard include prohibitions or restrictions, or both, on certain animal and product imports, ongoing surveillance for the disease in the United States, preparation of an emergency response plan in the unlikely event an introduction were to occur, and continuing educational efforts. The Animal and Plant Health Inspection Service actively shares information and coordinates closely with other Federal agencies, as well as the States, livestock and affiliated industries, veterinary and research communities, and consumer groups, to ensure that the United States has a uniform approach to transmissible spongiform encephalopathies based on sound scientific information.

A comprehensive surveillance program has been implemented by APHIS in the United States to ensure timely detection and swift response in the unlikely event that an introduction of BSE were to occur. This surveillance program entails the location of imports from countries known to have BSE and targeted active and passive surveillance for either BSE or any other TSE in cattle.

To locate each of the 496 British cattle that were imported into this country between January 1, 1981, and July 1989, APHIS has conducted a traceback effort. In July 1989, the United States prohibited the importation of ruminants from countries affected with BSE. As of March 1998, only 17 of these animals are known to be alive in the United States, and these are being carefully monitored by APHIS personnel on an ongoing basis. In addition, five head of cattle imported from Belgium in 1996 are now under quarantine. In cooperation with the states and industry, APHIS continues to purchase these animals for diagnostic purposes. No evidence of BSE has been found in any of these imported animals.

The United States has had an aggressive, active surveillance program for BSE since May 1990. Bovine spongiform encephalopathy is a notifiable disease, and there are more than 250 Federal and State regulatory veterinarians specially trained to diagnose foreign animal diseases, including BSE. The Animal and Plant Health Inspection Service leads an interagency surveillance program, which includes the Food Safety Inspection Service (FSIS) and the Centers for Disease Control (CDC). The surveillance samples include field cases of cattle exhibiting signs of neurological disease, cattle condemned at slaughter for neurological reasons, rabies-negative cattle submitted to public health laboratories, neurological cases submitted to veterinary diagnostic laboratories and teaching hospitals, and random sampling of cattle that are nonambulatory at slaughter. As of February 21, 1998, over 6,600 brains had been examined for BSE or another form of a transmissible spongiform encephalopathy in cattle. No evidence of either condition has been detected by histopathology or immunohistochemistry.

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

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

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

Public Health

BSE and CJD — Human Health Concerns

On March 20, 1996, the U.K.'s Spongiform Encephalopathy Advisory Committee (SEAC) announced the identification of 10 cases of a new variant form of CJD (nvCJD). All of the patients developed onset of illness in 1994 or 1995. The following features describe how these 10 cases differed from the sporadic form of CJD:

· The affected individuals were much younger than the sporadic CJD patient. Typically, sporadic CJD patients are over 63 years old. The average patient age for the variant form of CJD is 27.5 (range of 16 to 42) years.

· The course of the disease in the nvCJD averaged 13 months. Sporadic CJD cases average a 6-month duration.

· In the variant cases, electroencephalographic (EEG) electrical activity was not typical of sporadic CJD.

· Although brain pathology was recognizable as CJD, the pattern was different from normal CJD, and evidenced large aggregates of prion protein plaques.

Epidemiologic and case studies have not revealed a common risk factor among the cases of nvCJD. According to the SEAC, all victims were reported to have eaten beef or beef products in the last 10 years, but none had knowingly eaten brain material. One of the affected individuals had been a vegetarian since 1991 (52).

The SEAC concluded that, although there was no direct scientific evidence of a link between BSE and nvCJD, on the basis of current data and in the absence of any credible alternative, the most likely explanation was that the cases were linked to exposure to BSE before the introduction of control measures; namely, the specified bovine offal (SBO) ban in 1989.

Research reported in later 1996 and 1997 has presented further evidence to support a causal association between nvCJD and BSE. Two significant studies published in the October 2, 1997, edition of Nature led the SEAC to conclude that the BSE agent is very likely to be the cause of nvCJD. Dr. Moira Bruce and colleagues at the Institute for Animal Health in Edinburgh, Scotland, inoculated three panels of inbred mice and one panel of crossbred mice with BSE, nvCJD, and sporadic CJD. Interim results indicate that mice inoculated with BSE show the same pattern of incubation time, clinical signs, and brain lesions as mice inoculated with tissues from patients with nvCJD. This provides evidence that BSE and nvCJD have the same signature or are the same "strain." In addition classical CJD and known scrapie strains were not similar to nvCJD or BSE (9).

Results from another study published by Dr. John Collinge and colleagues of Imperial College School of Medicine, London, United Kingdom, strongly support Bruce's results. Collinge's paper reports experimental transmission of BSE to transgenic mice expressing only human PrP (20).

The Health and Safety Executive in the United Kingdom now advises that BSE must be considered a biological agent (human pathogen) within the meaning of the Control of Substances Hazardous to Health Regulations 1994 (45).

FSIS BSE Rules: Federal Register, January 12, 2004 (Volume 69, number 7, pages 1861-1892)

On December 23, 2003, one BSE positive cow was identified in the state of Washington. This cow had been randomly sampled for BSE and the carcass not held. Therefore, the product from this cow was distributed into commerce, resulting in a recall.

As a result, FSIS will no longer pass and apply the mark of inspection to the carcasses and parts from cattle that are selected for testing by USDA's Animal and Plant Health Inspection Service (APHIS) for BSE until the sample is confirmed negative.

FSIS is requiring that all non-ambulatory disabled cattle presented for slaughter be condemned.

The stunning method of penetrating captive bolt, which deliberately injects air into the cranial cavity of cattle, is no longer acceptable. FSIS is concerned that such stunning devices may force visible pieces of brain, known as macro-emboli, into the circulatory system of stunned cattle.

Specified risk materials (SMR's) are include: brain, skull, eyes, trigeminal ganglia, spinal cord, vertebral column (excluding the lumbar vertebrae and the wings of the sacrum) and dorsal root ganglia of cattle 30 months of age or older, and the tonsils and distal ileum of the small intestine of all cattle. These materials are inedible and are prohibited for use as human food. All federally inspected establishments that process the carcasses or parts of cattle must develop, implement, and maintain written procedures for the removal, segregation, and disposition of SRMs. Establishments must incorporate these procedures into their HACCP plans or in their SSOPs or other prerequisite program. Infectivity has never been demonstrated in the muscle tissue of cattle experimentally or naturally infected with BSE at any stage of the disease.

Advanced Meat Recovery (AMR) systems must not introduce central nervous system tissue into product labeled as "meat".

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M. Contagious bovine pleuropneumonia

Definition

Contagious bovine pleuropneumonia (CBPP) is a highly infectious acute, subacute, or chronic disease, primarily of cattle, affecting the lungs and occasionally the joints, and caused by *Mycoplasma mycoides mycoides*.

Etiology

Contagious bovine pleuropneumonia is caused by *M. mycoides mycoides* small colony type (SC type). *M. mycoides mycoides* large colony type is pathogenic for sheep and goats but not for cattle. *M. mycoides mycoides* (SC type) survives well only in vivo and is quickly inactivated

when exposed to normal external environmental conditions. *M. mycoides mycoides* does not survive in meat or meat products and does not survive outside of the animal in nature for more than a few days. Many of the routinely used disinfectants will effectively inactivate the organism.

Host Range

Contagious bovine pleuropneumonia is predominantly a disease of the genus *Bos*; both bovine and zebu cattle are naturally infected. There are many reported breed differences with respect to susceptibility. In general, European breeds tend to be more susceptible than indigenous African breeds (8). There does seem to be some age resistance, for animals less than 3 years of age are less resistant to experimental challenge (5). In zoos the infection has been recorded in bison and yak. Although it has been reported that the domestic buffalo (Bubalus bubalis) is susceptible, the disease is difficult to produce experimentally in this species (7).

Geographic Distribution

Contagious bovine pleuropneumonia is endemic in most of Africa. It is a problem in parts of Asia, especially India and China. Periodically, CBPP occurs in Europe, and other outbreaks within the last decade have occurred in Spain, Portugal and Italy. Contagious bovine pleuropneumonia was eradicated from the US in the nineteenth century. It is of historical interest that the Bureau of Animal Industries, which was a forerunner of USDA's APHIS was formed in 1884 specifically to eradicate CBPP. The United States was declared free of CBPP only 9 years later in 1893. Currently CBPP is not present in the Western Hemisphere.

Transmission

Contagious bovine pleuropneumonia is spread by inhalation of droplets from an infected, coughing animal. Consequently, relatively close contact is required for transmission to occur. Outbreaks usually begin as the result of movement of an infected animal into a naïve herd. It is widely believed that recovered animals harboring infectious organisms within a pulmonary sequestrum may become active shedders when stressed. Although this may be a factor in some outbreaks, it has not been substantiated experimentally (9). There are limited anecdotal reports of fomite transmission, but this means of transmission is not generally thought to be a problem.

Incubation Period

The time from natural exposure to overt signs of disease is variable but generally quite long. It has been shown that healthy animals placed in a CBPP-infected herd may begin showing signs of disease 20-123 days later (7). Experimentally, subsequent to instillation of large quantities of infective material at the tracheal bifurcation, the incubation period was 2-3 weeks.

Clinical Signs

Usually the first abnormality noticed is a depressed, inappentent animal with fever. Coughing may be the next sign followed by evidence of thoracic pain and an increased respiratory rate. As pneumonia progresses and animals become increasingly dyspenic, animals are inclined to stand with elbows abducted in an attempt to decrease thoracic pain and increase chest capacity. Auscultation of the lungs reveals any of a wide variety of sounds, depending on how severely the pulmonary parenchyma is affected. Crepitations, rales, and pleuritic friction rubs are all possible. Percussion over affected areas reveals dullness. When pulmonary involvement is extensive and severe, there will be very labored respiration and sometimes open mouthed breathing. Occasionally in calves, pneumonia may be accompanied by a polyarthritis. Animals affected in this manner may be very reluctant to move and stand stiffly with an arched back. Getting up and down may cause obvious discomfort. Large joints may be distended and warm on palpation. If joint pain is severe, animals may be so reluctant to bend the joints that they lie in lateral recumbency with legs outstretched. Contagious bovine pleuropneumonia often evolves into a chronic disease. This form, characterized by ill thrift and recurrent low-grade fever, may be difficult to recognize as pneumonia. Forced exercise may precipitate coughing.

Gross Lesions

The gross pathologic features of CBPP are quite characteristic (3). If the animal dies, there is usually extensive and marked inflammation of the lung and associated pleura. In severe cases there can be abundant fluid in the thoracic cavity. The inflammation is not uncommonly unilateral. The initial focus can be in any part of the lung, and in fatal cases, usually has spread locally and extensively to include a sizeable segment. The affected pulmonary parenchyma is odorless and often has all stages of lesions with both acute and chronic pulmonary changes adjacent to one another. The predominant gross change is consolidation, or thickening, of individual lobes which become encased in markedly widened interlobular septa, resulting in very characteristic marbled appearance. Interlobular septa become distended first by edema, then by fibrin, and finally by fibrosis. The overlying pleura may be very thickened by an irregular layering of yellow fibrin which, with time, becomes fibrosed, often resulting in adhesions between parietal and visceral pleurae. Not uncommonly, within an affected lung will be found a sequestrum - a focus that has undergone coagulative necrosis, and is effectively sealed off from the rest of the lung. Such sequestra may even be found in recovered animals. It has been shown that *M. mycoides mycoides* (SC-type) can survive within these sequestra for months or possibly longer.

Morbidity and Morality

The attack rate with CBPP is variable. It is not thought to be a highly contagious disease. With increased confinement of animals, morbidity rises. The mortality rate with CBPP is quite varied and ranges from 10-70 percent in various outbreaks. As with many subacute and chronic infectious diseases, mortality may depend on other intercurrent factors such as plane of nutrition, level of parasitism, and general body condition.

Diagnosis

Field Diagnosis

Clinical diagnosis of CBPP is difficult. At postmortem the gross lesions of CBPP are somewhat distinct. Often there is extensive deposition of fibrin and a large quantity of straw colored fluid in the thoracic cavity with prominent marbling of pulmonary parenchyma. Generally, all stages of pathologic changes from acute through to chronic are present in one animal. In some chronic cases the nodules of inflammation may not be readily apparent from the pleural surface but can be palpated within the parenchyma. Unlike many other pneumonias CBPP is often unilateral.

Specimens for Laboratory

From a live animal, nasal swabs, transtracheal washes, or pleural fluid obtained by thoracic puncture all provide good samples for isolation attempts. From a dead animal that has had severe clinical disease, the best specimens to submit are affected lung, swabs of the major bronchi, tracheo-bronchial or mediastinal lymph nodes, and joint fluid from those animals with arthritis. All samples should be collected aseptically and, if possible, placed in transport medium (heart infusion broth, 20 percent serum, 10 percent yeast extract, benzylpenicillin at 250 to 1000 IU/ml). Samples should be kept cool and shipped on wet ice as soon as possible. If transport to the laboratory is delayed (more than a few days), samples may be frozen (1). Blood should be collected for serum.

Laboratory Diagnosis

A definitive diagnosis is made by isolating and identifying the organism. Serology is helpful in the diagnosis of CBPP. Because CBPP is a subacute or chronic disease, most animals will have developed antibodies by the time of clinical disease.

Differential Diagnosis

Clinically, CBPP may be confused with other pneumonic conditions, especially bovine pasteurellosis. However, bovine pasteurellosis would likely spread much more rapidly and consequently the epidemiologist picture would be distinct.

Treatment

Mycoplasma mycoides mycoides (SC-type) is susceptible to a variety of antimicrobials, including streptomycin, oxytetracycline and chloramphenical. However, antimicrobial therapy[y may only serve to slow the progression of the disease or may even in some cases favor the formation of sequestra. In the case of chronically affected animals, or subclinically affected carriers, the organisms may be in an inaccessible location with an area of coagulative necrosis, which by definition is not served by a blood supply.

Vaccination

A modified live virus vaccine is available for use in enzootic areas. A major drawback of this vaccine is that it generates an unpredictable local reaction. For this reason it is often given in the tail tip, which may become necrotic and slough. Immunity subsequent to vaccination is generally good and lasts at least 12 months. The CBPP vaccine is often given in combination with the vaccine for rinderpest.

Prevention

Because CBPP is a chronic disease that may exist subclinically in carrier animals, it is important to maintain sufficient regulatory restrictions to prevent its introduction in apparently healthy animals. Serologic testing of susceptible animals for importation is a recommended safeguard.

Control and Eradication

Successful control of the spread of CBPP rests on removing susceptible animals from any possible contact with CBPP-infected animals whether they are clinically affected or subclinical carriers only. On-farm quarantine of suspicious and contact animals would be very advantageous in stemming the spread of disease. In an outbreak situation, testing, slaughter, and quarantine would be methods of choice.

Public Health Aspects

There is no evidence to indicate that humans are susceptible to this disease.

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N. Rinderpest

Definition

Rinderpest is a contagious viral disease of cattle, domestic buffalo, and some species of wildlife. It is characterized by fever, oral erosions, diarrhea, lymphoid necrosis, and high mortality.

Etiology

Rinderpest virus (RPV) is a single-stranded RNA virus in the family Paramyxoviridae, genus Morbillivirus. It is immunologically related to canine distemper virus, human measles virus, peste des petits ruminants virus, and marine mammal morbilliviruses. There is only one serotype of rinderpest virus, but field strains vary widely in virulence, ease of transmission, and host affinity.

Rinderpest virus is a relatively fragile virus. Sunlight is lethal and the vaccine must therefore be kept in a brown bottle and protected from light; virus in a thin layer of blood is inactivated in 2 hours. Moderate relative humidity inactivated the virus more quickly than wither high or low humidity. The virus is very sensitive to heat and both lypholized and reconstituted virus should therefore be kept cold; lypholized virus stored at -20° C is viable for years. Vaccine reconstituted in pure water quickly loses potency. Vaccine is more stable in a saline solution; reconstitution in a molar concentration of sulfate ions greatly increases resistance to heat.

Rinderpest virus is rapidly inactivated at pH 2 and 12 (10 minutes); optimal for survival is pH 65.-7. The virus is inactivated by glycerol and lipid solvents.

Host Range

Most wild and domestic cloven footed animals can be infected.

Geographic Distribution

Rinderpest is present in the Indian subcontinent, Near East and sub-Saharan Africa.

Transmission

Rinderpest was established as an infectious disease in 1754 when susceptible animals were infected by placing bits of material previously dipped in morbid discharge into an incision made in the dewlap. In 1899, cattle were infected with a bacteria-free infiltrate.

Secretions and excretions, particularly nasal-ocular discharges and feces, 1 to 2 days before clinical signs to 8 to 9 days after onset of clinical signs contain large quantities of virus. Spread of RP is by direct and indirect (contaminated ground, waters, equipment, clothing) contact with infected animals; aerosol transmission s not a significant means of transmission (it occurs only in a confined area and over a short distance). A major reason RP spreads in Africa is that herds are nomadic. Cattle follow the grass and thus move great distances, and during the dry season, many herds will use the same well or watering area, thus there is ample opportunity for cross-infection. It is said that a good fence will control RP.

There is only one serotype of RPV: recovered or properly vaccinated animals are immune for life, and there is no vertical transmission, arthropod vector, or carrier state. For these reasons, RPV is an ideal virus to be targeted for eradication.

Highly virulent strains of RPV are responsible for epizootics in susceptible animals and tend to die out. Milder strains tend to persist in an area, and the disease is not recognized as RP unless serology is performed.

The roles the various hosts can play in the disease are as follows:

- Cattle and domestic buffalo - highly susceptible
- Sheep and goats in Africa - subclinical infection and seroconversion, but there is no transmission to other animals
- Sheep and goats in India - when infected by low-passage goat RP vaccine will transmit to domestic buffalo
- Pigs - Swayback pigs in Thailand and the Malay Peninsula can be naturally infected and may die. European pigs can be infected by ingestion of RPV-infected meat and will transmit to cattle and other pigs.
- Wild ungulates - African buffalo, wildebeest, kudu, eland, giraffe, and wart hog are highly susceptible; Thompson gazelle, hippopotamus are fairly susceptible. Wild ungulates are infected by contact with cattle and can transmit to cattle. IN the absence of RP in cattle, the disease dies out in wildlife.

Incubation Period

The incubation period 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.

Clinical Signs

Depending on the strain of virus, resistance of the animal affected, and the concurrent infection, RP can appear as a peracute, acute or mild infection.

Peracute form

This form is seen in highly susceptible and young animals. The only signs of illness are fever of 104° to 107° F (40-41.7°C), congested mucous membranes, and death within 2-3 days after the onset of fever.

Acute or Classic form

This form of disease progresses as follows:

- Small amounts of virus may be in nasal and ocular secretions before the onset of fever
- Fever of 104-106°F (40-41.1°C)
- Serous to mucopurulent ocular discharge
- Serous to mucopurulent nasal discharge
- Leukopenia
- Depression
- Anorexia
- Constipation
- Oral erosions - salivation may be abundant and frothy
- Fever decreases and viral titer drops
- Diarrhea - may be very watery and hemorrhagic or both
- Dehydration, emaciation
- Prostration and death in 6-12 days after onset of illness.

Gross Lesions

Oral lesions are variable; some isolates cause good oral lesions and with others there is no oral lesion. Oral lesions start as small grey foci that may coalesce. The grey (necrotic) epithelium then sloughs off and leaves a red erosion.

- Mouth - lesions occur on the gums, lips, hard and soft palate, cheeks, and base of tongue. Early lesions are grey, necrotic, pinhead-sized areas that later coalesce and erode and leave red areas.
- Esophagus - Brownish necrotic or eroded areas
- Rumen and reticulum - lesions are rare
- Omasum - erosions and hemorrhage are rare

- Abomasum - congestion and edema
- Small intestine-necrosis or erosion of Peyer's patches in the jejunum; necrosis or erosions over the lymphoid area in the ileum (ingesta adhering to the intestinal mucosa indicates areas of necrotic epithelium)
- Cecum and colon - the wall may be edematous, and there may be blood in the lumen and blood clots on the mucosa. Lesions are usually more severe in the upper colon (edema of the wall, erosions in the mucosa, and congestion). The lesions may be accentuated at the cecocolic junction. Further down the colon, the colonic ridges may be congested; this is referred to as "tiger striping". Tiger striping can occur in other diarrhea and probably results tenesmus.
- Severity of intestinal lesions varies between isolates.
- Lymph nodes - Generally swollen and edematous.
- Liver- There may be petechial to ecchymotic hemorrhages in the gall bladder.
- Lung -there may be emphysema, congestion, and areas of pneumonia.

Diagnosis

Field Diagnosis

Rinderpest should be considered in all ages of cattle whenever there is a rapidly spreading acute febrile disease accompanied by the preceding clinical signs and lesions of RP. The all ages stipulation is important because this will be one of the major differences between bovine virus diarrhea-mucosal, which predominantly affects animals between 4 and 24 months of age.

Specimens for Laboratory

Because the viral titer drops when the fever falls and diarrhea starts, specimens could preferably be collected from animals with high fever and oral lesions. The following samples should be collected from live animals:

Blood in EDTA or heparin
Blood for serum
Swabs containing lacrimal fluid
Necrotic tissue from the oral cavity
Aspiration biopsies of superficial lymph nodes

For the best specimens, a febrile animal should be slaughtered and specimens collected. If this cannot be done, then collect specimens from moribund animals. Collect the blood samples listed above and sections of spleen, lymph nodes, and tonsil.

The preceding samples should be transported to the laboratory on wet ice - -NOT FROZEN. A complete set of tissues, including sections of all lesions, should be collected in 10 percent formalin.

Laboratory Diagnosis

To confirm the initial diagnosis in a free area, the virus has to isolated and identified.

Differential Diagnosis

The differential diagnosis for RP should include bovine rhinotracheitis, malignant catarrhal fever, foot and mouth disease, vesicular stomatitis, salmonellosis, paratuberculosis and arsenic poisoning.

Vaccination

The following types of RP vaccine have been used:

1. Lapinized in China and Korea
2. Avianized-lapinized in Korea
3. Goat-adapted in India
4. Cell-culture adapted in Africa, Middle Eats, and India.

An experimental vaccine-vectored vaccine containing the F and H genes of RPV has protected against a challenge inoculation of virulent virus.

The two most commonly used vaccines in 1996 were the goat-adapted and cell-culture adapted vaccines. The goat adapted vaccine is only partially attenuated; it will cause disease in animals with low innate resistance or concurrent latent disease and kills sheep and goats. The cell-culture attenuated vaccine was developed by Plowright in Kenya in the 1960's. This is a safe vaccine for many species and produces life long immunity in cattle (animals challenge-innoculated 7 years after vaccination were protected). In endemic areas where cattle have been vaccinated, colostral immunity will interfere with the vaccination of calves up to 11-12 months of age. Because the duration of colostral immunity is variable, the recommendation is to vaccinate calves annually for 3 years.

One of the biggest problems with the cell-culture adapted vaccine has been stability. The lypholized virus has to be kept cold (cold chain) until used. The combination of maintenance of the cold chain and remoteness of vaccination sites made RP vaccination very expensive. Because of the uncertainty that the vaccine being used was viable, the areas in Africa it is and was the policy to vaccinate animals every year in the hope that one of the vaccinations would immunize the animal. Researchers at Plum Island in the early 1990's greatly increased the stability of the lypholized vaccine by modifying the stabilizers and lyophilization process. This change in production is now being used in some production facilities in Africa.

Experimentally, the vaccine-vectored RP vaccine protected cattle against challenge inoculation with RPV. This vaccine is undergoing field testing. This vaccine could be particularly useful in an eradication program because vaccine-vectored RP vaccine immunized animals can be differentiated serologically from animals having antibody induced by the live virus. The vaccine-vectored vaccine would enable a country toward the end of an eradication program to maintain herd immunity to RP without using a live RP virus.

Control and Eradication

Countries and areas free of RP should prohibit unrestricted movement of RP- susceptible animals and uncooked meat products from areas infected by RP or practicing RP vaccination.

Because recovered animals are not carriers, and there are good serological techniques, zoological ruminants and swine can be imported with proper quarantine and testing. If an outbreak occurs, the area should be quarantined, infected and exposed animals slaughtered and buried or burned; and ring vaccination considered.

Experimentally it has been shown that RPV will not be transmitted by bovine embryo transfer if the embryos have been processed by the technique recommended by the International Embryo Transfer Society and the OIE.

High risk countries (those trading with, or geographically close to, infected countries) can protect themselves by having all susceptible animals vaccinated before they enter the country or vaccinating the national herd, or both. If an outbreak occurs, the area should be quarantined and ring vaccinated.

Endemic countries should vaccinate the national herd. Owing to the uncertainty of vaccine potency, the recommendation is to vaccinate annually for at least 4 years, followed by annual vaccination of calves. Foci of infection should be quarantined and stamped out. Wildlife, sheep and goats should be monitored serologically. Serological monitoring of sheep and goats could be complicated by using RP vaccine to protect against pest des petits ruminants.

Public Health

There is no report of RPV infection in a human.

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O. Heartwater

General

Heartwater is an infectious, noncontagious, tick-borne disease of domestic and wild ruminants, including cattle, sheep, goats, antelope, and buffalo. The disease is caused by an intracellular rickettsial parasite, Ehrlichia ruminantium, and is transmitted by a number of species of ticks in the genus Amblyomma.

Heartwater is usually an acute disease and is commonly fatal within a week of onset of clinical signs. The disease is widespread in most of Africa and on several islands in the West Indies. With increased trade and movement of animals in today's global market, Heartwater may present a significant threat to the domestic livestock industry in the United States.

Livestock owners should monitor their animals for exotic ticks and for clinical signs of the disease. If Heartwater is suspected, owners should immediately report these findings to a veterinarian or to a State or Federal animal health official.

History

Heartwater was first identified in sheep in South Africa in the 1830s. By 1898, it was shown to be a transmittable disease, and in 1900, the tropical bont tick was identified as a vector. In 1980, Heartwater was reported for the first time in the Western Hemisphere on the Caribbean island of Guadeloupe, although the vector tick was probably introduced from Africa much earlier. The disease is also present on the Caribbean islands of Marie Galante and Antigua.

The tropical bont tick has spread to several other islands in the Caribbean, although a definitive diagnosis of Heartwater has not been made to date on those islands.

Signs

The acute form of heartwater is the most commonly observed presentation of the disease. A sudden high fever (107° F) is followed by loss of appetite, depression, and respiratory problems. Animals may initially have an increased respiratory rate, followed within a few days by severe respiratory distress. Nervous disorders often follow the respiratory signs and can include a variety of abnormal behaviors such as excessive chewing movements, incoordination, head tilting upward, overly rigid posture, and walking with a high-stepping gait.

Some animals may undergo convulsions or be unable to rise. These nervous signs usually last for no more than 24 to 48 hours, followed by the animal's death. In some cases, the nervous signs may not be noticed prior to death.

A mild form of the disease, known as Heartwater fever, is present in some affected regions among indigenous breeds with a natural or acquired resistance to Heartwater. The only clinical sign of the mild form of the disease is a transient fever, and animals with this form usually recover.

Postmortem Lesions

Heartwater derives its name from a common postmortem finding of excessive fluid in the sac surrounding the heart. More commonly, the fluid accumulates within the lungs, thus the lungs appear "wet" and heavy. The fluid may also accumulate within the chest cavity itself, outside the lungs.

Confusion with Other Diseases

The observed nervous system abnormalities suggest other diseases (such as rabies, tetanus, meningitis, or encephalitis) or toxic poisoning. A definitive diagnosis of heartwater is made by microscopic examination and observation of the causative rickettsia in a brain tissue smear.

How It Spreads

Heartwater is transmitted only by ticks of the genus Amblyomma, with the tropical bont tick as one of the most important vectors. This tick is widely distributed throughout Africa, Yemen, the Cape Verde islands, and several islands in the Caribbean.

The life cycle of Amblyomma ticks may take from 1 to 4 years. Thus, the infection may persist, inside the tick for a long time. In its immature stages, the tick will feed on a wide variety of livestock, wild ungulates, ground birds, small mammals, reptiles, and amphibians.
Rapid spread of the tropical bont tick in the West Indies has occurred since the 1960s. Movement of tick-infested livestock was incriminated in some cases, but overall, the cause of the spread of these ticks has not been determined.

Cattle egrets became established in the region in the 1950s and have been implicated in much of the recent spread of heartwater. Small numbers of tick-infested cattle egrets have been shown to move among islands in the region, but these birds are not considered to be efficient disseminators of the tick.

Susceptible Species

Animals susceptible to heartwater include cattle, sheep, goats, and buffalo. Some breeds of cattle (e.g., Jerseys and Brahmas) may be more susceptible than others. Exotic ruminants can also contract the disease.

In laboratory tests in the United States, the white-tailed deer (*Odocoileus virginianus*) has been shown experimentally to be highly susceptible to heartwater. Amblyomma maculatum, another potential vector, is a common parasite of white-tailed deer in the Southern United States. However, there is no evidence that heartwater is present in wildlife in this country.

Prevention and Control

Preventive measures by livestock owners should include implementation of an effective tick-control program, including regular inspection of animals and pastures for ticks and elimination of the vector through the use of acaricides.

To prevent introduction of heartwater or any other foreign animal disease, the U.S. Department of Agriculture's (USDA) Animal and Plant Health Inspection Service (APHIS) tests imported animals for heartwater and other diseases and ensures that all animals are free of ticks or other potential insect vectors before entry into the United States is permitted.

P. Sheep and Goat Pox

Definition

Sheep and goat pox (SGP) is an acute to chronic disease of sheep and goats characterized by generalized pox lesions throughout the skin and mucous membranes, a persistent fever, lymphadenitis, and often a focal viral pneumonia with lesions distributed uniformly throughout the lungs. Subclinical cases may occur.

Etiology

The virus that causes SGP is a capripoxvirus, one of the largest viruses (170-260nm by 300-450nm) (10). It is closely related to the virus that causes lumpy skin disease; SGP Virus and lumpy skin disease virus cannot be distinguished serologically. There is only one serotype of SGP virus (SGPV). Various strains of SGPV cause disease only in sheep, others only in goats and some in both sheep and goats (2,3,9).

The SGPV is very resistant to physical and chemical agents.

Host Range

Sheep and goat pox virus causes clinical disease in sheep and goats. The virus replicates in cattle but does not cause clinical disease. The disease has not been detected in wild ungulate populations.

Geographic Distribution

The disease is endemic in Africa, the Middle East, the Indian subcontinent, and much of Asia.

A goat pox like disease was reported in the western United States (15), but no attempt was made to identify the agent with reference serum against SGPV. Serum samples from animals representing the affected group of goats were submitted to the Foreign Animal Disease Diagnostic Laboratory (FADDL) at Plum Island, NY, and tested for antibody to SGPV; no antibodies were found against SGPV. The serums were not tested for antibodies to bovid herpesvirus 2 or contagious ecthyma at the FADDL. Unfortunately, the viral isolate was not available for study. It is conclusive that what was reported in the literature was not goat pox.

Transmission

Contact is the main means of transmission of SGPV. Inhalation of aerosols from acutely affected animals, aerosols generated from dust contaminated from pox scabs in barns and night holding areas, and contact through skin abrasions either by fomites or by direct contact are the natural means of transmitting SGPV. Insect transmission is possible. The virus can cause infection experimentally by intravenous, intradermal, intranasal, or subcutaneous inoculation.

Incubation Period

Under field conditions, the incubation of SGP is between 4 and 8 days. Experimentally, the first sign (fever) may appear within 3 to 5 days after inoculation. The course of the disease is 4 to 6 weeks with various stages of pox lesions present at the same time. Full recovery may take up to 3 months.

Clinical Signs

Sheep and goat pox virus may cause subclinical infection; clinical cases vary from mild to severe (3). The course of the disease in sheep and goats is similar. The first signs may include fever, depression, conjunctivitis, lacrimation, and rhinitis. Within a few days of the prodromal signs, pox lesions develop in the skin. These are more easily observed on the wool-free or hair-free parts of the body such as the perineum, inguinal area, scrotum, udder, axilla, and muzzle. Lesions do occur in wooled or haired skin. Generally more severe (extensive) skin lesions correlate with more severe illness. The skin lesions first appear as an erythematous area (macula). The lesions progress to a raised, slightly blanched lesion that presents erythema with edema in the central part of the lesion (papule). Pox lesions with a transudate, representing the vesicular stage of the lesion, may be noted, but rarely is there any gross vesicle in the skin. The center of the lesion then becomes depressed and gray (necrotic) and is surrounded by an area of hyperemia. Late in the course of the disease (2 to 4 weeks after the first signs, the lesion becomes dry and a scab forms. A characteristic feature of a pox lesion is that lesions involve the entire epidermis and dermis and penetrate into the subcutaneous tissue; it feels like a nodule. Depending on the severity of the skin lesion, there may be a scar, an area devoid of wool or hair, after the lesion heals. Secondary bacterial infection may complicate the healing process. The muzzle may be swollen, and the nares and oral mucosa may have extensive lesions. IN many cases, pneumonia may occur with labored breathing and a respiratory rate

approaching 90 per minute. Depression, anorexia, and emaciation are common and may persist. Nervous signs may occur, but how these are related to the SGPV infection is not clear.

Lambs and kids under 1 month of age may suffer a very severe generalized form of SGP. The signs described above for older animals are exaggerated, and there is an increased mortality.

Gross Lesions

At necropsy, skin lesions have congestion, hemorrhage, edema, vasculitis and necrosis and will be seen to involve all layers of the epidermis, dermis, and in severe cases, extend into the adjacent musculature. Lymph nodes draining affected areas are enlarged up to eight times their normal size owing to extensive lymphoid proliferation, edema, congestion and hemorrhage.

Mucous membranes of the eye, mouth, and nose have pox lesions that, in severe cases, may coalesce. In severe cases of SGP, the eyelids may be so seriously affected that the proliferative lesions and inflammation cause the eyes to close. Lesions on the muzzle and mares may coalesce, and proliferative changes and inflammation may be extensive. Pox lesions may occur in the pharynx, epiglottis, and trachea. These usually appear as rounded blanched areas surrounded by an area of hyperemia. Occasionally there may be lesions in the epithelium of the rumen and Omasum.

Pox lesions in the lungs may be severe and extensive; the lesions are focal and uniformly distributed throughout the lungs as the result of hematogenous infection. Early lesions are congested areas; these progress to discreet areas of congestion and edema and finally to white nodules. Areas distal to the pox lesions have lobular atelectasis. Mediastinal lymph nodes are often enlarged up to five times their normal size and may be congested, hemorrhagic, and edematous.

Pox lesions also may be present on the vulva, prepuce, testicles udder, and teats.

Morbidity and Mortality

The severity of SGP varies depending on the strain of the virus and the age and breed of the animals affected (5). In adult sheep and goats, morbidity may range from 70-90 percent with some subclinical infections. Mortality can approach 50 percent. In endemic areas, mortality rates range from 5-10 percent, but it can approach 100 percent in imported animals (OIE). In susceptible lambs and kids under 1 month of age, morbidity may approach 100 percent, and mortality may be as high as 95 percent. Factors that may complicate the course of the disease and increase the mortality are poor nutrition, heavy parasitism, and severe climatic conditions.

Diagnosis

Field Diagnosis

A tentative diagnosis of SGP can be made on the basis of clinical signs consisting of skin lesions, which on palpation involve the whole thickness of the skin, a persistent fever, lymphadenitis, and often pneumonia; mortality may approach 50 percent in adults and 95 percent in lambs and kids under 1 month of age.

Specimens for Laboratory

For laboratory diagnosis of SGP, skin biopsies of early lesions can be used for virus isolation and histopathologic and electron microscopic studies. Samples aspirated from enlarged lymph nodes can be used for virus isolation. Necropsy samples should include a full set of tissues, but samples of the lungs, trachea, and rumen containing gross lesions are especially valuable for histopathology. Samples for virus isolation should be shipped under wet ice if they will arrive within 2 days and shipped under dry ice if delivery will take longer (send in screw capped vials with the caps secured with electrical tape). Samples for histopathology should be preserved in 10 percent buffered formalin (DO NOT FREZE). Serum samples should be taken from acute and chronic cases. Follow-up serum samples from acute cases may be taken 2 to 3 weeks after the first sample.

Laboratory Diagnosis

The laboratory procedures for the diagnosis of SGP include identification of the agent by cell inoculation and identification by immunofluorescence staining of intracytoplasmic inclusion bodies, inhibition of the cytopathic effect using positive serum, and antigen detection ELISA. Serological tests include agar gel immunodiffusion, ELISA, detection of antibody by virus neutralization, the indirect fluorescent antibody test (4) or both; in combination with characteristic histopathologic lesions (3).

Differential Diagnosis

Following are several diseases to consider in the differential diagnosis for SGP:

- Bluetongue - Animals are depressed and have a non-purulent conjunctivitis. The muzzle is swollen, congested, and edematous, and there may be a coronitis. Deformed aborted fetuses and deformed newborn sheep and goats may be encountered.
- Peste des Petits Ruminants - Conjunctivitis, rhinitis, and oral lesions that are white, raised, and necrotic are common. Pneumonia, diarrhea, and mortality approaching 90 percent in lambs and kids under 1 month of age are characteristic signs.
- Contagious ecthyma (contagious pustular dermatitis, ORF) - this disease is most severe in lambs and kids. The proliferative pox disease is most severe in lambs and kids. The proliferative pox lesions are common on the muzzle ad eyes of affected neonates; mortality may approach 50 percent. Nursing females may have proliferative pox lesions on the teats and muzzle. This is a zoonotic disease; lesions in attendants are not uncommon.
- Photosensitization - dry, flakey, inflamed areas are confined to the non-pigmented parts of the skins.

- Insect Bites - the trauma from insect bites may cause local inflammation, edema, and pruritis. Insects seldom bite mucous membranes.
- Parasitic pneumonia - severe signs of respiratory distress may occur with extensive parasitic lesions; in these cases, there is no pox lesion in the skin.
- Caseous lymphadenitis - focal, raised lesions ion the skin represent caseous abscesses; abscesses are not seen in SGP.
- Streptothricosis (*dermatophilus congolensi* infection) - lesions are superficial and often moist. Lesions are common in the skin of the neck, axillary region, inguinal region, and perineum. The organism may be demonstrated by Geisma staining.
- Mange - scab-like skin lesions are seen with psoroptic mange. Itching and scratching are not seen in SGP.

Vaccination

In endemic areas, vaccination is an effective means of controlling losses from SGP. Killed vaccines have not proven to be practical under field conditions because they do not provide solid, long lasting immunity. Several modified live virus vaccines have been used for protection against SGP. The most widely used employed vaccine is probably the Romanian strain that has been used effectively for many years (14, 16). The Kenya O 180 strain (6) is possibly the vaccine with the best safety and efficacy.

Control and Eradication

Prevention

The most likely manner for SGP to enter a new area is by introduction of infected animals. Restrictions on the movement of animal and animal byproducts (meat, hair, wool, and hides) are essential to prevent introduction of SGP. Wool, hair, and hides must be subjected to suitable decontamination procedures before entry into non-endemic areas.

Control

If a new case is confirmed in a new area before extensive spread occurs, the area should be quarantined, infected and exposed animals should be slaughtered, and the premises cleaned and disinfected. Vaccination of susceptible animals on premises surrounding the infected flock(s) should be considered.

If the disease has spread over a large area, the most effective means of controlled losses from SGP is vaccination; however, consideration should be given to eliminating infected and exposed flocks by slaughter; properly disposing of animals and contaminated material; and cleaning and disinfecting contaminated premises, equipment, and facilities.

Eradication

A carrier state has not been shown for SGPV. However, the virus may persist for many months on contaminated premises. The imposition of quarantines on areas and premises containing infected or exposed animals is required to prevent disease spread. Depopulation of infected and exposed flocks should be used if limited spread has occurred. If the disease has spread extensively, massive vaccination followed by cessation of vaccination and control of animal movements from the area represent a strong strategy to control and then eradicate SGP.

Public Health

There is no conclusive evidence that SGPV infects humans. A report from India (17) that implied that goat pox caused human infection was merely based on clinical signs. There was no attempt to isolate the causative virus or perform serology on the convalescent serums of three patients to differentiate the infection from contagious ecthyma, which is a known zoonotic agent that occurs worldwide. A report from Sweden (1) indicated that human infection occurred during an outbreak of goat pox. Although serological studies seemed to indicate that the apparent causative agent of the outbreak was not vaccine or contagious ecthyma, no virus was isolated. Therefore it cannot be said that goat pox caused human infection.

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Q. Exotic Newcastle Disease

(Velogenic Newcastle disease, Asiatic Newcastle disease**)**

Definition

Velogenic Newcastle disease (VND) is the most severe form of Newcastle disease and is likely the most serious disease of poultry throughout the world (2,4,13). In chickens it is characterized by lesions in the brain or gastrointestinal tract, morbidity rates near 100 percent, and mortality rates as high as 90 percent in susceptible chickens. Neurologic signs or severe depression are the most obvious clinical sign, and some nonvaccinated birds may be found dead with no detected sign of prior illness.

Etiology

Newcastle disease viruses (NDV's) occur as three pathotypes: lentogenic, mesogenic, and velogenic, reflecting increasing levels of virulence. The most virulent (velogenic) isolates are further subdivided into neurotropic and viscerotropic types. The velogenic isolates are considered exotic to the United States, and the disease caused by these VND isolates is the subject of this chapter.

The Newcastle disease viruses belong to the *Paramyxoviridae* virus family and, like other members of this group, possess two surface proteins that are important to the identification and behavior of the virus. The first, hemagglutinin/neuraminidase (HN) is important in the attachment and release of the virus from the host cells in addition to its serologic identification. The other very important surface protein is the fusion (F) protein, which has a critical role in the pathogenesis of the disease. There are at least nine known types of avian paramyxoviruses based on the antigenic makeup of the hemagglutinin. NDV is the prototype virus for Type 1 avian paramyxoviruses.

Host Range

Inapparently infected carriers that are the most likely source for introduction of VND include numerous species of exotic pet and exposition birds, waterfowl, and domestic poultry (18). A persistent carrier state has been demonstrated in psittacine (8) and in certain other wild birds (19) whereas virus can be recovered from chickens for shorter periods of time, usually 14 days or less.

Geographic Distribution

Velogenic Newcastle disease is endemic in many countries of Asia, the Middle East, Africa, and Central and South America. Some European countries are considered free of VND. VND has caused high mortality in wild cormorants in Canada and the United States.

Transmission

In many parts of the tropics VND is recurrent in the poultry populations. One possibility is that they are infected from a wild bird reservoir. Additional studies will be required before it can be established which species, if any, are true carriers and which are only transiently infected. It is not known whether the occurrence of VND in wild birds moving in international trade can be reduced by avoiding the capture of certain species or their collection at certain time periods or places. Once introduced into poultry, the virus spreads farm-to-farm by the movement of inapparently infected poultry species; on contaminated objects such as boots, sacks, egg trays, and crates; or by flies (5) or mice. Reports from England (11) that the virus can be wind-borne under certain conditions should be considered even though there was no evidence of airborne transmission between premises with the virus that caused the 1971 outbreak in California. Free-flying wild birds apparently had no role in the spread of VND during that outbreak (16).

Incubation Period

The incubation period for Newcastle disease after natural exposure varies from 2 to 15 days. For VND in chickens, an incubation period of 2 to 6 days is common. The incubation period in other species of birds may be longer.

Clinical Signs

Velogenic Newcastle disease is a devastating malady in unvaccinated chickens of any age. The first sign in laying chickens is usually a marked drop in egg production followed within 24 to 43 hours by high death losses. At the onset, 10-15 percent of a flock may be lost in 24 hours. After 7 to 10 days, deaths usually subside, and birds surviving 12 to 14 days generally do not die but may display permanent paralysis and other neurologic signs. The reproductive system may be permanently impaired, and thus egg production does not return to previous levels. In vaccinated chickens, or chicks protected by parental antibodies, the clinical signs are less severe and are proportional to the level of protective antibodies.

With viscerotropic strains (VVND), edema of the head, especially around the eyes may become apparent after birds have been sick for 2 or 3 days (9). This edema usually does not involve the comb and wattle to the extent of highly pathogenic avian influenza (HPAI). A dark ring sometimes forms around the eye, probably due to cyanosis and poor blood circulation in the edematous tissue. This "black eye" appearance is especially visible in white chickens.

Bile-stained, greenish-dark diarrhea may be noted 2 to 3 days after onset of illness. Some birds in an affected flock usually have diarrhea throughout the course of the disease.

The most noteworthy clinical sign in unvaccinated flocks is sudden death without prior indications of illness. The peracute onset often causes the owner to suspect poisoning.

Respiratory distress and signs of neurological disturbances, such as drooping wings, torticollis, and ataxia. may not be as marked as they are with the neurotropic forms of the disease. However, these neurologic signs are frequently observed in chickens that survive infection with the viscerotopic strains for 2 or 3 weeks. Because of lack of experience with viscerotropic

strains, poultry owners throughout the United States and Canada may not consider Newcastle disease as a possible diagnosis unless they see the neurologic signs they have seen with the domestic neurotropic viruses.

Neurotropic strains cause respiratory signs soon followed by neurologic signs, including muscular tremors, paralysis of legs or wings, torticollis, and opisthotonos. There is a marked decline in egg production but usually no diarrhea. Disease signs may differ markedly, depending on the host species. Psittacines or pigeons infected with the viscerotropic strains of virus may display neurologic signs typical of the disease caused by the strains of neurotropic ND in chickens (7). These same viscerotropic viruses may cause typical signs and lesions of VVND when inoculated into chickens (6). In some species, such as finches and canaries, clinical disease may not be observed.

Gross Lesions

No gross lesion may be observed in many of the first birds dying in a commercial poultry operation. Peracute deaths are generally due to collapse or dysfunction of the reticuloendothelial system before discernible gross lesions have developed. There is no pathognomonic gross lesion for VVND, but, generally, sufficient lesions can be found to make a tentative diagnosis if enough birds are examined (14). Because of the marked similarities between the gross lesions of VVND and highly pathogenic avian influenza, a final diagnosis in the first flocks must await virus isolation and identification. In a continuing outbreak where numerous flocks are involved, gross observations may eventually be all that is necessary when typical lesions are present.

Edema of the interstitial tissue of the neck, especially near the thoracic inlet, may be marked. After the trachea and esophagus are exposed during necropsy examination, straw colored fluid may drip from these tissues. Congestion and occasionally hemorrhage may be seen in the trachea generally corresponding to the rings of cartilage.

Proventriculus

Petechial and small ecchymotic hemorrhages may be present on the mucosa of the proventriculus. These small hemorrhagic foci tend to be found near the base of the papillae and concentrated around the posterior and anterior orifices.

Intestine

Peyer's patches, cecal tonsils, and other focal aggregations of lymphoid tissue in the gut wall usually are markedly involved and are responsible for the term viscerotropic applied to this form of Newcastle disease. These areas progressively become edematous, hemorrhagic, necrotic, and ulcerative. In chickens that have died from VVND, these involved lymphoid areas can often be observed without opening the gut.

Reproductive System

Ovaries may be edematous, hemorrhagic, or degenerated. Yolk peritonitis can frequently be observed in layers as a result of VVND, and rough, misshapen eggs are frequently laid by recovering hens.

Neurotropic strains of VND may cause few gross lesions other than in the trachea and lungs. There will be no gross lesion in the brain of diseased birds. Gross lesion patterns usually differ markedly between the disease caused by the viscerotropic and neurotropic velogenic viruses.

Morbidity and Mortality

Clinical VND is most severe in chickens, peafowl, guineas, pheasant, quail and pigeons. Turkeys may develop a milder form of the disease. Severity of disease in psittacine and passerine birds is variable. In susceptible chickens, the morbidity and mortality rates can be as high as 100 percent and 90 percent, respectively. In some species such as finches and canaries, clinical disease may not be observed.

Diagnosis

Field Diagnosis

A tentative diagnosis of VND may be made on the basis of history, clinical signs, and gross lesions, but because of similarities to other diseases such as fowl cholera and highly pathogenic avian influenza, confirmation requires virus isolation and identification.

Specimens for Laboratory

Virus can readily be recovered from sick or recently dead birds. Swabs are the most convenient way to transfer VND virus from tissues or secretions of the suspect bird to brain and heart infusion broth or other cell culture maintenance medium containing high levels of antibiotics (1). Trachea, lung, spleen, cloaca, and brain should be sampled. Swabs should be inserted deeply to ensure obtaining ample epithelial tissue. If large numbers of dead or live birds are to be sampled, cloacal swabs from up to five birds can be pooled in the same tube of broth. An alternate technique is to place 0.5 cm^3 of each tissue into the broth. If the specimens can be delivered to a laboratory within 24 hours, they should be placed on ice. If delivery will take longer, quick-freeze the specimens and do not allow them to thaw during transit.

Laboratory Diagnosis

In the laboratory, virus isolation is attempted by inoculating 9- to 11-day-old embryonating chicken eggs. Chorioallantoic fluid (CAF) is collected from all embryos dying after 24 hours postinoculation and tested for hemagglutination (HA) activity. If positive, the hemagglutination-inhibition (HI) test is used with known NDV-positive serum to confirm the presence of NDV in the CAF (3). If NDV is found, it is characterized by inoculating 4- to 6-week-old chickens free of ND antibodies with the suspect CAF by swabbing the cloaca, instilling into the nares or conjuctival sac, or injecting into the thoracic air sac. If VVND virus is present, the inoculated chicks usually die in 3 to 7 days, revealing typical visceral lesions on postmortem examination. Neurotroph ic VVD viruses will cause severe neurologic and respiratory signs in inoculated chickens but no visceral lesions. If no bird dies in 10 days, the NDV is not considered to be the velogenic, viscerotropic type but is either a lentogen or mesogen.

Differential Diagnosis

The viscerotropic, velogenic Newcastle disease in poultry can be confused with highly pathogenic avian influenza, infectious laryngotracheitis, fowl cholera, and coryza.

Vaccination

Vaccination with viable or inactivated oil emulsion vaccines, or both, can markedly reduce the losses from VND in poultry flocks. If eradication of the virus is not the goal of the control program, vaccines can be used to lessen the impact of the disease. Their use, however, can make the complete eradication of the virus much more problematic by increasing the difficulty of identifying infected flocks. There is little doubt, however, that vaccination makes the flock more refractive to infection when exposed and reduces the quantity of virus shed by infected flocks.

Control and Eradication

Before 1972, VND was introduced into the United States on several occasions by unrestricted introduction of exotic pet birds, especially psittacine birds. Because pet birds are not usually associated with domestic poultry, VND outbreaks were rare (20). Since 1973, restrictions on the importation of exotic birds requiring the quarantining and testing of imported birds in approved quarantine facilities have reduced but not eliminated the threat of VND in the United States. Illegally imported exotic bird species remain the source of frequent outbreaks of VND in private or commercial aviaries.

The establishment of a strict quarantine and destruction of all infected and exposed birds with financial indemnification for losses followed by thorough cleaning and disinfection of premises were the main features necessary for eradication of VND virus from the poultry producing area of southern California. Flocks may be safely and humanely destroyed using carbon dioxide in air-tight chambers and the carcasses disposed of by burying, composting, or rendering, depending upon the geographic area and the numbers involved. The VND virus has been recovered from effluent water for as long as 21 days and from carcasses for 7 days when the daytime temperatures were over 90o F. It is recommended that premises be kept free of domestic poultry for an additional 30 days after cleaning and disinfection are completed.

Insects and mice associated with the poultry should be destroyed before depopulation of a flock begins (5,12). Usually 48 hours is sufficient to control these vectors. As soon as all birds are killed and the manure and feed removed, all equipment and structural surfaces should be thoroughly cleaned using high-pressure spray equipment. The entire premises should then be sprayed with an approved residual disinfectant such as the cresylics or phenolics. Preliminary disinfection will probably inactivate most of the viruses on the surface of floors, equipment, cages, etc., but no disinfectant is effective unless it is applied to scrupulously cleaned surfaces free of all organic material.

Cleaning and disinfecting commercial poultry premises are time-consuming and expensive operations. All manure must be removed down to a bare concrete floor. If the floor is earthen, at least the top inch of soil should be removed with the manure. Manure can be safely disposed of by burying it at least 5 feet deep or by composting. If composting is used, the manure piles

should be tightly covered with black polyethylene sheets in a manner to prevent access by birds, insects, and rodents during composting. These piles of manure should remain tightly covered and undisturbed at least 90 days during warm weather and for longer periods during cold weather. Recent studies indicate that proper composting can decompose carcasses and manure, and thus inactivate viruses in only a few weeks.

Feathers, usually numerous around commercial poultry premises, can be burned outside the buildings, and in some cases inside, with the careful use of a flame thrower, or they can be removed and the area wet down with disinfectant. The hot sun and high daytime temperatures will assist in destroying the virus in the area of the houses. Extremely cold temperatures will make the cleaning and decontamination process much more difficult, and the results more uncertain.

In 1997, because neither the neurotropic or viscertropic strain of velogenic Newcastle disease was known to exist in the United States, USDA-APHIS declared both types to be exotic and therefore indistinguishable as to the response of disease control officials should they occur in the United States.

Surveillance

The most difficult part of the VND eradication program is locating inapparently infected and exposed birds.

Repeated vaccination at 30 to 50 day intervals protects most chickens against clinical manifestation of VND. However, vaccine does not prevent all chickens in a flock from becoming infected, showing no disease sign, or shedding virulent virus. As individual chickens become susceptible and get exposed to the virus, they become infected and also shed the virus for a time. Thus, the virulent virus continues to be present in apparently healthy, vaccinated flocks. The advantages of using vaccines as part of a VND eradication program must be weighed against the difficulty created in finding asymptomatic but infected and virus-shedding flocks. In such instances owners should be encouraged to observe strict biosecurity measures to reduce the chances of their flocks being exposed to VND virus.

Infected carriers in vaccinated flocks can be detected using one of two systems. In the first, all birds dying during a 24-hour period are collected twice a week, and cloacal swabs and brains are collected and cultured for the presence of VND virus using the diagnostic sampling procedures described earlier. Birds in VND-infected flocks that die from Marek's disease, leukosis, gout, and numerous other disease conditions may yield VND virus—especially if their immune system was impaired by those diseases before death. In the second virus detection system, susceptible sentinel birds are placed in vaccinated flocks (18). The sentinel birds must be unvaccinated and obtained from a specific pathogen-free source to be certain that they do not inadvertently serve as a source of diseases for the suspect flock. In most instances the sentinel birds die from VND within a week or so after placement if there is VND virus present in the flock; however, in some cases it is sometimes difficult to place sentinel birds so they are adequately exposed to any VND virus that may be in the flock — especially in caged-layer flocks.

Public Health

Although people may become infected with VND virus, the resulting disease is typically limited to a conjunctivitis. Recovery is usually rapid, and the virus is no longer present in eye fluids after 4 to 7 days. Infections have occurred mostly in laboratory workers and vaccinating crews with rare cases in poultry handlers. No instance of transmission to humans through handling or consuming of poultry products is known. Individuals with conjunctivitis from VND virus should not enter poultry premises or come in contact with live avian species.

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R. Avian Influenza (Fowl Plague)

General

The historic and dreaded synonym used to describe the disease caused by highly pathogenic (HP) avian influenza virus is fowl plague. Less pathogenic strains of avian influenza have caused problems in many US turkey flocks and live poultry markets since the 1960's, while few commercial chicken flocks were involved. However in 1983, a virus originally characterized as relatively nonpathogenic began to produce a fowl-plague-like disease with high death losses in Pennsylvania. Control and eradication of this 1983 outbreak cost over $63 million in Federal funds and an additional $350 million in increased consumer costs. As of 1998, viruses related to those involved in the 1983 outbreak continue to circulate in North America and to pose a threat to the US Poultry industry. A highly publicized outbreak of H5N1 avian influenza (AI) in chickens and people in Hong Kong illustrates the potential public health concerns that may surface as a result of AI infections.

Definition

Avian influenza (AI) is a disease of viral etiology that ranges from a mild or even asymptomatic infection to an acute, fatal disease of chickens, turkeys, guinea fowls, and other avian species, especially migratory waterfowl (1,2,3,4,8,9,10,11).

Etiology

Fowl plague was described in 1878 as a serious disease of chickens in Italy. It was determined in 1955 that fowl plague (FP) virus is actually one of the influenza viruses. The AI viruses, along with the other influenza viruses, make up the virus family Orthomyxoviridae. The virus particle has an envelope with glycoprotein projections with hemagglutinating and neuraminidase activity.

These two surface antigens, hemagglutinin (HA) and neuraminidase (NA), are the basis of describing the serologic identity of the influenza viruses using the letters H and N with the appropriate numbers in the virus designation e.g., H7N2. There are now 16 hemagglutinin and 9 neuraminidase antigens described among the Type A influenza viruses. The type designation (A, B, or C) is based upon the antigenic character of the M protein of the virus envelope and the nucleoprotein within the virus particle. All influenza viruses affecting domestic animals (equine, swine, avian) belong to Type A, and Type A influenza virus is the most common type producing serious epidemics in humans. Types B and C do not affect domestic animals.

Classical fowl plague viruses have H7 as one of the surface antigens but can have different N antigens. It was once believed that all H7 viruses are highly pathogenic fowl plague viruses and that no other avian influenza viruses could produce a fowl-plague-like disease. When avirulent AI viruses with the H7 antigens were demonstrated in turkeys in 1971 and highly virulent AI viruses with the H5 antigen were first found in chickens in 1959, the necessity for redefining the term fowl plague or using other terminology became apparent. Because there are highly virulent AI viruses that possess H antigen other than the H7 and H7 AI viruses that do not produce clinical fowl plague, an international assembly of avian influenza specialists proposed that the term fowl plague no longer be used. They suggested that any AI virus, regardless of its HA designation, meeting specified virulence requirements in the laboratory be designated highly pathogenic avian influenza (HPAI). The criteria that serve as the basis for classifying an AI virus as HPAI has more recently been modified to include molecular considerations such as the type of amino acids at the cleavage site of its HA. This chapter will be limited to describing the HPAI and not the AI viruses of less virulence and pathogenicity.

Host Range

Most avian species appear to be susceptible to at least some of the AI viruses. A particular isolate may produce severe disease in turkeys but not in chickens or any other avian species. Therefore, it would be impossible to generalize on the host range for HPAI, for it will likely vary with the isolate. This assumption is supported by reports of farm outbreaks where only a single avian species of several species present on the farm became infected. Swine appear to be important in the epidemiology of infection of turkeys with swine influenza virus when they are in close proximity. Other mammals do not appear to be involved in the epidemiology of HPAI. The

infection of humans with an H5 avian influenza virus in Hong Kong in 1997 resulted in a reconsideration of the role of the avian species in the epidemiology of human influenza.

Geographic Distribution

Highly pathogenic avian influenza viruses have periodically occurred in recent years in Australia (H7), England (H7), South Africa (H5), Scotland (H5), Ireland (H5), Mexico (H5), Pakistan (H7), and the United States (H5). Because laboratory facilities are not readily available in some parts of the world to differentiate Newcastle disease and HPAI, the actual incidence of HPAI in the world's poultry flocks is difficult to define. It can occur in any country, regardless of disease control measures, probably because of its prevalence in wild migratory waterfowl, sea birds and shore birds.

Avian influenza has produced losses of variable severity, primarily in turkeys in the United States, since the mid-1960's. The disease outbreaks in turkeys in the United States have been caused by AI viruses with many of the HA designations. It was in the fall of 1983 that a highly virulent H5 virus produced severe clinical disease and high mortality in chickens, turkeys, and guinea fowl in Pennsylvania. This severe disease, clinically indistinguishable from classical fowl plague, occurred after a serologically identical but apparently mild virus had been circulating in poultry in the area for 6 months.

Outbreaks of less virulent AI have frequently been described in domestic ducks in many areas of the world. The AI viruses are often recovered from apparently healthy migratory waterfowl, shore birds, and sea birds worldwide. The epidemiologic significance of these isolations relative to outbreaks in domestic poultry has led to the generally accepted belief that waterfowl serve as the reservoir of influenza viruses.

Transmissions

There is a considerable body of circumstantial evidence to support the hypothesis that migratory waterfowl, sea birds, or shore birds are generally responsible for introducing the virus into poultry. Once introduced into a flock, the virus is spread from flock to flock by the usual methods involving the movement of infected birds, contaminated equipment, egg flats, feed trucks, and service crews, to mention a few. Preliminary trapping evidence indicates that garbage flies in the Pennsylvania outbreak were sources of virus on the premises of the diseased flocks. Virus may readily be isolated in large quantities from the feces and respiratory secretions of infected birds. It is logical to assume, therefore, that because virus is present in body secretions, transmission of the disease can take place through shared and contaminated drinking water. Airborne transmission may occur if birds are in close proximity and with appropriate air movement. Birds are readily infected via instillation of virus into the conjunctival sac, nares, or the trachea. Preliminary field and laboratory evidence indicates that virus can be recovered from the yolk and albumen of eggs laid by hens at the height of the disease. The possibility of vertical transmission is unresolved; however, it is unlikely infected embryos could survive and hatch. Attempts to hatch eggs in disease isolation cabinets from a broiler breeder flock at the height of disease failed to result in any AI-infected chickens. This does not mean that broken contaminated eggs could not be the source of virus to infect chicks after they hatch in the same

incubator. The hatching of eggs from a diseased flock would likely be associated with considerable risk.

Incubation Period

The incubation period is usually 3 to 7 days, depending upon the isolate, the dose of inoculum, the species, and age of the bird.

Clinical Signs

Infections of HPAI result in marked depression with ruffled feathers, inappetance, excessive thirst, cessation of egg production, and watery diarrhea. Mature chickens frequently have swollen combs, wattles, and edema surrounding the eyes. The combs are often cyanotic at the tips and may have plasma or blood vesicles on the surface with dark areas of ecchymotic hemorrhage and necrotic foci. The last eggs laid, after the onset of illness, are frequently without shells. The diarrhea begins as watery bright green and progresses to almost totally white. Edema of the head, if present, is often accompanied by edema of the neck. The conjunctivae are congested and swollen with occasional hemorrhage. The legs, between the hocks and feet, may have areas of diffuse hemorrhage. Respiratory signs can be a significant feature of the disease, depending on the extent of tracheal involvement. Mucus accumulation can vary. It is not unusual in caged layers for the disease to begin in a localized area of the house and severely affect birds in only a few cages before it spreads to neighboring cages.

Death may occur within 24 hours of first signs of disease, frequently within 48 hours, or be delayed for as long as a week. Some severely affected hens may occasionally recover.

In broilers, the signs of disease are frequently less obvious with severe depression, inappetence, and a marked increase in mortality being the first abnormalities observed. Edema of the face and neck and neurologic signs such as torticollis and ataxia may also be seen.

The disease in turkeys is similar to that seen in layers, but it lasts 2 or 3 days longer and is occasionally accompanied by swollen sinuses.

In domestic ducks and geese the signs of depression, inappetence, and diarrhea are similar to those in layers, though frequently with swollen sinuses. Younger birds may exhibit neurologic signs.

Gross Lesions

Birds that die with the peracute disease and young birds may not have significant gross lesions other than severe congestion of the musculature and dehydration. In the less acute form, and in mature birds, significant gross lesions are frequently observed. They may consist of subcutaneous edema of the head and neck area, which is evident as the skin is reflected. Fluid may exit the nares and oral cavity as the bird is positioned for postmortem examination. The conjunctivae are severely congested— occasionally with petechiation. The trachea may appear

relatively normal except that the lumen contains excessive mucous exudate. It may also be severely involved with hemorrhagic tracheitis similar to that seen with infectious laryngotracheitis. When the bird is opened, pinpoint petechial hemorrhages are frequently observed on the inside of the keel as it is bent back. Very small petechia may cover the abdominal fat, serosal surfaces, and peritoneum, which appears as if it were finely splattered with red paint. Kidneys are severely congested and may occasionally be grossly plugged with white urate deposits in the tubules.

In layers, the ovary may be hemorrhagic or degenerated with darkened areas of necrosis. The peritoneal cavity is frequently filled with yolk from ruptured ova, causing severe airsacculitis and peritonitis in birds that survive for 7 to 10 days.

Hemorrhages may be present on the mucosal surface of the proventriculus — particularly at the juncture with the gizzard. The lining of the gizzard peels easily and frequently reveals hemorrhages and erosions underneath. The intestinal muscosa may have hemorrhagic areas — especially in the lymphoid foci such as the cecal tonsils. The gross lesions are not distinctly different from those observed with velogenic viscerotropic Newcastle disease (VVND). The lesions in turkeys and domestic ducks are similar to those in chickens but may not be as marked.

Morbidity and Mortality

The prognosis for flocks infected with HPAI is poor. Morbidity and mortality rates may be near 100 percent within 2 to 12 days after the first signs of illness. Birds that survive are usually in poor condition and resume laying only after a period of several weeks.

Diagnosis

Field Diagnosis

Highly pathogenic avian influenza is suspected with any flock where sudden deaths follow severe depression, inappetence, and a drastic decline in egg production. The presence of facial edema, swollen and cyanotic combs and wattles, and petechial hemorrhages on internal membrane surfaces increases the likelihood that the disease is HPAI. However, an absolute diagnosis is dependent upon the isolation and identification of the causative virus. Commercially available type A influenza antigen-capture enzyme linked immunosorbent assay kits designed for use in human influenza have recently shown promise as a possible rapid diagnostic test for poultry.

Specimens for Laboratory

Specimens sent to the laboratory should be accompanied by a history of clinical and gross lesions, including any information on recent additions to the flock. Diagnosis depends upon the isolation and identification of the virus from tracheal or cloacal swabs, feces, or from internal organs (5). Specimens should be collected from several birds. It is not unusual for many of the

submitted specimens to fail to yield virus. Swabs are the most convenient way to transfer AI virus from tissues or secretions of the suspect bird to brain and heart infusion broth or other cell culture maintenance medium containing high levels of antibiotics. Dry swabs should be inserted deeply to ensure obtaining ample epithelial tissue. Trachea, lung, spleen, cloaca, and brain should be sampled. If large numbers of dead or live birds are to be sampled, cloacal swabs from up to five birds can be pooled in the same tube of broth. An alternative technique is to place 0.5 cm^3 of each tissue into the broth. Blood for serum should be collected from several birds. If the specimens can be delivered to a laboratory within 24 hours, they should be placed on ice. If delivery will take longer, quickfreeze the specimens and do not allow them to thaw during transit.

Laboratory Diagnosis

Nine to 11-day-old embryonated chicken eggs are inoculated with swab or tissue specimens. Avian influenza virus will usually kill embryos within 48-72 hours. If the virus isolated is identified as a Type A influenza virus, through the AGP or ELISA tests, it is then tested using a battery of specific antigens to identify its serologic identity (HA and NA type).

Sera from infected chickens usually yield positive antibody tests as early as 3 or 4 days after first signs of disease.

Differential Diagnosis

Highly pathogenic avian influenza is easily confused with VVND, because the disease signs and postmortem lesions are similar, and may also be confused with infectious laryngotracheitis and acute bacterial diseases such as fowl cholera and *Escherichia coli.* However, in an area where AI is prevalent, such as during an outbreak, sound presumptive diagnoses can be made by flock history, signs, and gross lesions.

Treatment

Amantadine hydrochloride has been licensed for use in humans to treat influenza since 1966. The medication is effective in reducing the severity of influenza Type A in humans. Experimental evidence indicated possible efficaciousness in poultry when the drug was administered in drinking water to reduce disease losses, but drug-resistant viruses quickly emerged, negating the initial beneficial effects. Thus, the drug is not recommended for use in poultry.

Vaccination

Inactivated oil-emulsion vaccines, although fairly expensive, have been demonstrated to be effective in reducing mortality, preventing disease, or both, in chickens and turkeys (7). These vaccines may not, however, prevent infection in some individual birds, which go on to shed virulent virus. More economical viable vaccines prepared using naturally avirulent or attenuated strains have the disadvantage of the possible creation of reassortant influenza viruses with unpredictable characteristics. These reassortants could result when a single host bird is simultaneously infected with both the vaccine and another AI virus. Owing to the segmented nature of the influenza virus genome, a reassortment of genetic material can readily occur,

creating new influenza viruses. The basic drawback to any vaccine approach for the control of HPAI is the large number of HA subtypes that can cause the disease. Because there is no cross-protection among the 15 known HA subtypes, either a multivalent vaccine will be needed or vaccination postponed until the prevalent disease-causing subtype in the area is identified. A recombinant fowl pox virus vaccine containing the gene that codes for the production of the H5 antigen has recently been licensed. The use of a recombinant insect virus containing the gene for either the H5 or H7 antigen has been used to make these vaccine proteins in insect cell cultures.

Control and Eradication

The practice of accepted sanitation and biosecurity procedures in the rearing of poultry is of utmost importance. In areas where waterfowl, shore birds, or sea birds are prevalent, the rearing of poultry on open range is incompatible with a sound AI prevention program (12).

Appropriate biosecurity practices should be applied, including the control of human traffic and introduction of birds of unknown disease status into the flock. Cleaning and disinfection procedures are the same as those recommended in the chapter on velogenic Newcastle disease.

Public Health

The AI viruses are Type A influenza viruses, and the possibility exists that they could be involved in the development, through genetic reassortment, of new mammalian strains. An influenza virus isolated from harbor seals that died of pneumonia had the HA and NA surface antigens of an influenza virus isolated from turkeys a decade earlier. The infection and deaths of 6 of 18 humans infected with an H5 avian influenza virus in Hong Kong in 1997 resulted in a reconsideration of the portentous role that the avian species have on the epidemiology of human influenza. Previously there was only one report of a human becoming infected with an H7 AI virus. It is impossible to predict the importance of AI virus in determining the strains of virus that infect humans. There was no evidence to indicate that humans coming in contact with large quantities of the H5N2 virus during depopulation efforts in the HPAI outbreak of 1983 in Pennsylvania became infected with the virus.

In the current news, an HPAI virus has been spreading across Asia, Europe and Africa. This highly pathogenic H5N1 virus has infected people as well as bird populations. Since December 2003, when the first bird-to-human transmission occurred in Viet Nam, 166 people have been infected. The World Health Organization reports as of February 9, 2006 the H5N1 strain of the bird flu has killed 88 people in 7 countries. At this time, the most recently confirmed death has occurred in Iraq. This H5N1 virus spread west from Southeast Asia to the fringe of Europe in late 2005. In early February 2006, it reached Iraq. On February 8, 2006 it was identified in bird populations in Nigeria.

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Originally written by C.W. Beard, D.V.M., USDA, ARS. Southeast Poultry Research Laboratory, Athens, GA. In "Foreign Animal Diseases", by US Animal Health Association, Richmond, VA. Modified 1/10/06 by Center for Learning, FSIS.

III. Reporting Procedures

If, while conducting routine ante mortem or postmortem inspection of animals, identify a condition that you feel is suspicious of a reportable or foreign animal disease, you are to notify the District Office immediately. The DO will contact the State Animal Health Official and/or the APHIS Veterinary Services Area Veterinarian In Charge (AVIC) immediately. Veterinary Services is obligated to respond to your concern immediately, and will be there to assist you with diagnosis of this condition. Your importance in this role cannot be overemphasized: FSIS employees many more veterinarians than does Veterinary Services, and we look at many more

animals through routine slaughter procedures. Therefore, we have a unique opportunity to identify reportable and foreign animal diseases, and can play a critical role in disease control and eradication, and in prevention of potential bioterrorist activities. A complete list of AVIC's and State Veterinarians is in sections VI and VII of these materials for your reference.

IV. MOU

MEMORANDUM OF UNDERSTANDING
BETWEEN
UNITED STATES DEPARTMENT OF AGRICULTURE
FOOD SAFETY AND INSPECTION SERVICE
AND
UNITED STATES DEPARTMENT OF AGRICULTURE
ANIMAL AND PLANT HEALTH INSPECTION SERVICE

Relative to
Cooperation with Respect to Surveillance Programs

The parties to this Memorandum of Understanding are the United States Department of Agriculture, Food Safety and Inspection Service, hereinafter called FSIS, and the United States Department of Agriculture, Animal and Plant Health Inspection Service, hereinafter called APHIS.

WHEREAS, the parties to this Memorandum of Understanding agree to cooperate in meeting their responsibilities relative to information exchange regarding animal and public health disease surveillance, diagnostic testing, investigations, trace backs, recalls, animal welfare, and animal and public health concerns and of providing a safe, secure, wholesome, and economical food supply; and

WHEREAS, FSIS has qualified personnel available to inspect, observe, and report evidence of communicable diseases at the time of slaughter; and

WHEREAS, APHIS has laboratory expertise, facilities, and personnel available to conduct tests on blood samples and tissue specimens, conduct sample collection for surveillance purposes, conduct epidemiological traces of animals and animal products, respond to disease outbreaks and animal welfare concerns; and

WHEREAS, both parties have responsibilities in these areas, are willing to enter into a mutually beneficial reimbursable agreement for certain services, and are committed to developing further plans for implementation of said agreement; and

WHEREAS both parties are committed to utilizing this agreement to build stronger and mutually supportive working relationships to better protect human and animal health,

NOW, THEREFORE, in consideration of the agreements and mutual covenants herein contained, the parties hereto do mutually agree:

FSIS Agrees:

1) Upon notification of a potential concern related to the administration of veterinary biologics, to notify APHIS, Veterinary Services (VS), and the FSIS Technical Services Center when evidence is found at slaughter that abnormal findings may have resulted from the administration of veterinary biologics.

- To cooperate with APHIS-VS on routine inspection of restricted meats imported into the United States, including sampling and laboratory examination as required for certain products, produced in specific establishments, which are suspected of being undercooked. (Refer to Appendix 1.)

- To cooperate with APHIS-VS in the breaking of official seals applied to shipments of restricted livestock and poultry arriving at slaughter and to continue to provide APHIS with information concerning violations of animal health or animal welfare regulations relative to movement of livestock and poultry observed by FSIS inspectors.

- To notify APHIS-VS of animals suspicious for screwworm infection, scrapie, and other animal diseases of interest to APHIS.

- To cooperate with APHIS on bovine spongiform encephalopathy (BSE) surveillance. (Refer to Appendix 9.)

- To collect and submit for laboratory testing blood samples from test eligible adult cattle and swine and to collect suspected tuberculosis lesions from cattle and suspected tuberculosis thoracic lesions from swine disclosed at slaughter, and to collect all manmade identification devices from all cattle and swine from which blood samples or lesions are collected. (Refer to Appendices 3 and 4.)

2. Where APHIS has contracts for blood collection to ensure that all manmade animal identification devices are collected.

    - To collect all manmade identification devices and report to APHIS all known, identified, or permitted brucellosis or tuberculosis reactors slaughtered.

    - To collect tissues from animals upon special request for *Brucella* culturing. (Refer to Appendices 3 and 4.)

    - To promptly notify APHIS-VS when signs and/or lesions of foreign animal diseases are noted in livestock or poultry during antemortem and/or postmortem inspection(s). FSIS will inform the appropriate APHIS-VS official prior to processing animals suspected of a foreign animal disease and will follow existing FSIS regulatory procedures. (Refer to Appendix 6.)

    - To cooperate with the collection and submission of tissue samples that may be suspected of tuberculosis (mycobacteria). (Refer to Appendix 4.)

    - To provide APHIS with read only access to the data obtained from the electronic Animal Disposition Reporting System (eADRS) slaughter data as per the MOU signed November 2004. (Refer to Appendix 7.)

    - To provide APHIS with read only access to the data obtained from the eADRS slaughter data for animal disease surveillance programs. (Refer to Appendix 10.)

3. When livestock presented for slaughter are to be released for a purpose other than slaughter, the operator of the official establishment or the owner of the livestock shall first

obtain permission for the movement of such livestock from the local, State, or Federal livestock sanitary official having jurisdiction as per Title 9, *Code of Federal Regulations* (9CFR), section 309.2(p).

- To notify APHIS-VS of imported meat, poultry, or egg products suspected of being tampered or containing toxic industrial chemicals, foreign animal disease agents, or other potential biological or chemical contamination.

- To promptly notify APHIS-VS when agents of biological or chemical warfare/terrorism are suspected in animal-based food product.

- To perform food security verification procedures at certain threat levels that includes coordinating security concerns on live animals with APHIS-VS officials.

- To cooperate with APHIS-VS on the Brucellosis Eradication Program through blood sample collection at slaughter under a quarterly reimbursable agreement. FSIS will provide APHIS-VS with a report of the number of samples collected by FSIS personnel quarterly by establishment.

B. APHIS Agrees:

1. To notify FSIS when administration of certain veterinary biologics may be related to abnormal reactions or that certain animals slaughtered have been exposed to a biologic of interest to APHIS.

2. To arrange and coordinate with State authorities for the collection and submission of specimens from animals potentially infected with diseases of interest to APHIS for diagnosis and to provide FSIS with current lists of diseases of interest to APHIS and guidelines to report such diseases.

3. To, in consultation with FSIS, provide clearance for new adjuvants or other ingredients of biologics where the safety of meat of animals or poultry for human consumption following their use may be questionable, and to establish withholding periods for biological products which produce temporary residues in animals and poultry.

4. To conduct field investigations and to advise FSIS of outbreaks of diseases that affect the health of animals including those of public health significance such as brucellosis, tuberculosis, ornithosis, anthrax, rabies, BSE, or other zoonotic or potentially zoonotic diseases or syndromes of interest to APHIS and to report progress in eradicating these diseases.

5. To conduct field investigations and to advise FSIS in a timely manner of outbreaks of vesicular or other reportable or exotic diseases of foreign origin.

6. To provide, upon request, assistance in the inspection of swine or other animals at slaughter when vesicular or other reportable or exotic diseases of foreign origin are suspected.

7. To provide information relative to traceback of animals to points of origin as requested

by FSIS inspectors and to conduct field investigations for those incidences that are of mutual interest to both parties.

8. To provide FSIS inspectors, via telephone, fax, or e-mail, timely laboratory results on tissue specimens submitted to the National Veterinary Services Laboratories for examination for tuberculosis on retained carcasses or other public health diseases that result in retention of carcasses.

9. To provide FSIS, upon request, reports on the number of blood samples received by APHIS laboratories for analysis from their respective areas.

10. To notify FSIS inspectors when known tuberculosis reactors are shipped to slaughter plants and to assist FSIS inspectors upon their request in the collection of laboratory samples from tuberculosis reactors.

11. To provide the FSIS inspector feedback regarding any actions and/or findings that result from inspector contributions to investigations of communicable diseases.

12. To notify appropriate FSIS officials of any findings of residue or chemical substances in livestock or poultry, or in the tissues or products thereof, which may indicate the potential for adulteration of the meat or poultry supply, including specific available information as to the origin or location of livestock or poultry associated with such findings.

13. To arrange for the Permit for Movement of Restricted Animals or Materials (VS Form 1-27) when applicable animals need to be moved from the slaughterhouse to alternate locations.

14. To provide FSIS training slots in each applicable class to attend the Foreign Animal Disease Diagnostician Course held at Plum Island, New York, at FSIS expense.

15. To notify appropriate FSIS officials of any findings suggestive of biologic or chemical warfare or terrorist actions against livestock or poultry.

16. To provide FSIS with recommended on-farm, market, and transportation biosecurity measures.

17. To notify FSIS of any live imported food animal suspected of having been exposed to potential terrorist/warfare agents.

18. To cooperate when requested on the tracing of recalled products should there be an animal, public health, or food security emergency.

19. To notify FSIS in a timely manner when samples of imported meat, poultry, or egg products are required because they may not meet APHIS regulatory requirements.

20. To assist FSIS when notified of serious livestock animal welfare concerns and when inhumane transportation is observed, especially if it concerns imported animals shipped under an APHIS seal.

21. To provide certificate forms to certify inedible byproducts for export when APHIS/VS

personnel are not available and has prearranged the certification with FSIS.

22. To reimburse FSIS quarterly for blood samples collected by FSIS personnel.

23. To make available, upon request, electronic copies of reports and to consult with FSIS prior to publicly releasing data derived from the eADRS as per Appendix 7.

C. It is Mutually Understood and Agreed:

1. That the details of this cooperative undertaking shall be jointly planned through an APHIS-FSIS Implementation Working Group and executed by the cooperating parties.

2. This Memorandum of Understanding is to define in general terms the basis on which parties concerned will cooperate and does not constitute a financial obligation to serve as a basis for expenditures. Each party will acquire and expend its own funds. Any and all expenditures from Federal funds in the Department of Agriculture made in conformity with the plans outlined in the Memorandum of Understanding must be in accord with Department rules and regulations and in each instance based upon appropriate financial procedures. Expenditures made by either party will be in accord with its particular rules and regulations. Reimbursable agreements will be developed to provide for the exchange of funds between agencies as required.

3. Either party shall be free to furnish such equipment as may be needed without cost to the other party. Any such equipment furnished shall remain the property of the providing party and subject to its disposition.

4. The responsibilities assumed by each of the parties hereto are contingent upon funds being available from which expenditures legally may be met.

5. Both parties will share training resources involving subjects of mutual interest; tuberculosis postmortem training by FSIS (refer to Appendix 8) and foreign animal disease diagnostician training by APHIS.

6. The results of the work herein may be published jointly by the parties hereto or by either party separately, but manuscripts prepared for publication by either shall be submitted to the other party for suggestions and approval prior to publication. In the event of disagreement, either party may publish results about its own responsibility, giving proper acknowledgment to the other cooperator. Both parties shall share with each other proposed policies or procedures related to this MOU that may directly impact the other prior to the public release with sufficient time for the other to respond appropriately prior to publication.

7. This Memorandum of Understanding supersedes previous FSIS-VS Memoranda of Understanding, including #MU 12-37-MU-334.

8. The provisions of this Memorandum of Understanding shall be reviewed annually.

9. This Memorandum of Understanding shall become effective upon the date of final signature and shall continue indefinitely but may be modified or discontinued at the

request of either party. Each party shall provide in writing 60 days notice in advance of the effective date desired for termination of this agreement or any major modification.

UNITED STATES DEPARTMENT OF AGRICULTURE
FOOD SAFETY AND INSPECTION SERVICE

_____

Date Administrator

UNITED STATES DEPARTMENT OF AGRICULTURE
ANIMAL AND PLANT HEALTH INSPECTION SERVICE

_____

Date Administrator

Appendix 1

Food Safety and Inspection Service (FSIS)
Animal Plant Health Inspection Service (APHIS)
Veterinary Services (VS)

Inspect, collect and examine imported cooked meats for underprocessing.

Cooked meats are allowed to be imported from certain countries where exotic animal diseases occur only when these meats are thoroughly cooked. FSIS has qualified import inspection personnel assigned to the ports of entry (POE) that are available to inspect and report evidence of meat products that are suspected of being insufficiently cooked during processing in the country of origin. Because APHIS-VS has a regulatory responsibility to prevent the distribution of underprocessed meat products from these countries, both parties agree as follows:

A. FSIS Agrees:

1. To immediately notify APHIS-VS via telephone or fax, followed up by e-Mail, of any suspect lots and findings; and retain/detain suspect lots when available. FSIS will retain or control any related lots of products that APHIS-VS considers necessary. Suspect lots include, but are not limited to, those displaying evidence of inadequate processing, such as bones and undercooking.

2. To assist APHIS-VS in the examination of suspect lots and collect samples as directed by APHIS-VS.

3. As directed by APHIS-VS or the Department of Homeland Security to refuse entry of any lot not meeting APHIS-VS requirements during import inspection and to impose restrictions on future lots.

4. To provide, when appropriate, representatives from various FSIS staffs to serve on emergency situation teams.

B. APHIS Agrees:

1. To accept responsibility when a suspect lot is in violation of its import requirements and that APHIS-VS is the final authority for the disposition of suspect lots.

2. To provide FSIS as quickly as possible with oral and written instructions on sampling plans and action requested such as depth of recall, retention or detention of suspect lots, and final disposition of product.

3. To determine and inform FSIS of any additional information required to ensure complete enforcement of APHIS-VS standards and import requirements.

4. To notify the foreign government, the brokers/importers, and the appropriate FSIS office of the findings and actions being taken by APHIS-VS as a result of violations of regulatory requirements.

5. To provide representatives to work with FSIS emergency response teams.

C. It is Mutually Understood and Agreed:

1. That APHIS-VS and FSIS will cooperate in accordance with applicable laws and regulations to allow resolution of each incident.

2. FSIS will coordinate action in cases where the product has not completed FSIS import reinspection.

Appendix 1
Attachment A

Standard Procedures for Handling Imported Cooked Meat Products in Which Pink Juices are found at an approved FSIS Import Establishment.

A. The Food Safety and Inspection Service (FSIS) will retain, if available, the entire shipment, including the sample, and notify the local Department of Homeland Security, Customs and Border Protection, as well as the Animal and Plant Health Inspection Service (APHIS), Protection and Quarantine (PPQ), Veterinary Regulatory Support (VRS), Animal Quarantine Inspection Veterinarian (AQIV), or the VRS headquarters office by telephone, fax, or e-Mail and the FSIS Office of International Affairs and provide the following information:

    1. Production code (complete tube and carton identification).

    2. Country of origin and establishment number.

    3. Type and amount of product.

B. Location of retained product

C. Any specific decontamination procedures related to intentional or un-intentional contamination issues.

D. Department of Homeland Security-Customs and Border Control (DHS-CBP) and/or APHIS-PPQ-VRS-AQIV at the local port office will immediately notify the APHIS-PPQ-VRS headquarters office, the Director, Animal Products, National Center for Import-Export (NCIE), Animals and Products Staff, Veterinary Services (VS), Riverdale, Maryland, and provide the same data listed under A (above) by telephone.

E. The APHIS Director of the NCIE will:

    1. Establish communication with the following;

        a. U.S. representative of the foreign establishment or the U.S. importer.

        b. The appropriate FSIS office.

    2. Notify appropriate government officials in the country of origin through the Agricultural Attaché.

    3. Investigate the extent of the problem by determining if other shipments are involved.

    4. Initiate appropriate action to:

a. Refuse entry in accordance with APHIS regulations and policy.

b. Coordinate with direct assistance of APHIS-PPQ-VRS headquarters, with local APHIS, PPQ Office of International Affairs to ensure that satisfactory disposition of product is made as per VS policy and to ensure that all appropriate PPQ, VS, and FSIS personnel are notified as is appropriate.

c. Coordinate appropriate actions with regional and local VS offices when pink juices are found in commerce; i.e., in U.S. establishments or wholesale warehouses (after passing port-of-arrival inspection).

F. APHIS-VS field personnel shall not take action to dispose of product without first obtaining instructions through channels from VS, NCIE.

APHIS will consider any contamination hazards and coordinate with FSIS on specific procedures related to potential food security contamination issues.

Standard Procedures for Handling Perishable Cooked Pork Products from Restricted Countries as indicated in 9 CFR, Part 94.

A. When Food Safety and Inspection Service (FSIS) laboratory results indicate the cooked pork product was undercooked (155 degrees F. or less), the laboratory shall immediately notify the appropriate FSIS office which will immediately notify the Animal and Plant Health Inspection Service (APHIS), National Center for Import/Export (NCIE). The laboratory should report the production codes, specific type of product, and any other pertinent information.

B. APHIS, NCIE,-(note-spell out please, and the FSIS officials managing recalls will coordinate to retain/detain all of the available products in the lot and to recall products that have been shipped from the import establishment.

C. The Director of NCIE will:

1. Immediately contact the U.S. representative of the foreign establishment or importer.

2. Notify appropriate government officials in the country of origin through the Agricultural Attaché.

3. Request information (records) for location, etc., of identified production code product.

4. Coordinate with appropriate FSIS or APHIS, Plant Protection and Quarantine (PPQ), Veterinary Regulatory Support (VRS), headquarters office any action to control recall, destroy, or export product.

5. Involve APHIS-Veterinary Services (VS), APHIS-PPQ-VRS- Animal Quarantine Inspection Veterinarian, and Department of Homeland Security-Customs and Border Control field personnel in tracing product, if necessary.

D. The movement and/or destruction of the product will be under APHIS-VS or APHIS-PPQ-VRS supervision.

Appendix 2

Food Safety and Inspection Service (FSIS)
Office of International Affairs (OIA)

Animal and Plant Health Inspection Service (APHIS)
Veterinary Services (VS)

Activities by FSIS and APHIS-VS to ensure that meat and poultry products imported from foreign countries into the United States meet applicable animal health and inspection control standards.

FSIS and APHIS-VS have separate but closely related statutory responsibilities regarding the eligibility for import into the United States of meat and poultry products from foreign countries. Both agencies maintain separate staffs of experts to ensure that these important requirements are met on a continuous basis. To maximize efficiency and effectiveness, the following statements are agreed to by the parties:

A. FSIS and APHIS-VS will exchange information on plants certified and approved to export to the United States and provide updates of the plant listings.

B. Regular monthly telephone conferences will be scheduled between APHIS' National Center for Import and Export, Technical Trade Services, Plant Protection and Quarantine, and FSIS.

C. APHIS-VS will provide FSIS with copies of product restriction information when this information is not contained in Title 9, *Code of Federal Regulations*, part 94, or when more detailed information is available.

D. APHIS-VS and FSIS will inform each other of changes in foreign country disease status and export eligibility status.

E. APHIS-VS and FSIS will exchange addresses of program officials stationed in foreign countries. Notification of changes will be made in a timely manner.

F. APHIS-VS will inform FSIS of acceptable interpretations of regulations affecting product production in restricted countries. This information will be updated on a periodic basis when FSIS personnel are attending area meetings or when there are significant changes in policy. FSIS, through its regular reviews, will gather information and notify APHIS-VS of any deviations from acceptable practices.

G. APHIS-VS and FSIS will exchange information regarding the disease status of countries exporting to the United States and information on their respective audit plans. FSIS will notify APHIS-VS of any information learned relative to animal health concerns found during the course of review activities.

H. APHIS-VS and FSIS will exchange travel plans for foreign country visits on a quarterly basis. When possible, travel will be coordinated to avoid taxing resources of any foreign country.

I. APHIS-VS will conduct reviews of foreign plants to determine the adequacy of proposed procedures for processing product to mitigate risk due to animal disease. APHIS-VS will provide to FSIS detailed interpretations of requirements and how they must be met in the establishments. When requested by APHIS-VS, FSIS will collect information regarding animal disease issues and in-plant processes during regularly scheduled audits and will report relevant information to APHIS.

J. APHIS-VS and FSIS agree to brief their respective foreign program personnel on the various facets of each other's program.

There will be no reimbursement for these activities.

Food Safety and Inspection Service (FSIS)
Animal and Plant Health Inspection Service (APHIS)
Veterinary Services (VS)

Brucellosis Eradication: Title 9, *Code of Federal Regulations*, part 78: Collection of samples for *Brucella* isolation.

FSIS collects all manmade identification devices and reports to APHIS all known brucellosis or tuberculosis reactors slaughtered, and collects tissues from animals upon special request for culturing *Brucella*.

APHIS-VS documents animal movements from brucellosis infected and/or suspect herds on VS Form 1-27. In order to confirm or prove the existence of *Brucella* organisms in the herd or herds of origin, FSIS is requested to collect tissue samples from such animals when specifically identified and agreed upon with VS. In most instances, VS personnel will participate in or conduct the collections.

FSIS Agrees:

1. To submit tissue samples to the National Veterinary Services Laboratories (NVSL) for *Brucella* isolation in accordance with established procedures, provided sufficient personnel are available or if personnel are not available to notify VS immediately.

- To make no charges to VS for collecting and submitting subject tissue samples.

- To utilize the most current VS sampling procedures and official forms.

APHIS-VS Agrees:

1. To process samples submitted for *Brucella* isolation as promptly as possible.

2. To furnish shipping containers and preservatives for submitting specimens to NVSL.

3. To notify FSIS in advance of the arrival of animals to be sampled.

4. To provide FSIS with the most current sampling procedures and forms.

It is Mutually Understood and Agreed:

1. FSIS and VS will cooperate to minimize the work of collecting, submitting, and reporting laboratory results on specimens identified for *Brucella* culturing.

2. That both VS and FSIS, insofar as possible, will inform the other of impending changes in procedure that are likely to affect the submission or handling of specimens.

Food Safety and Inspection Service (FSIS)

Animal and Plant Health Inspection Service (APHIS)
Veterinary Services (VS)

Tuberculosis Eradication: Title 9, *Code of Federal Regulations*, part 77: Collect and submit suspected tuberculosis lesions or thoracic granulomas found in cattle carcasses along with all accompanying manmade identification devices.

The goal of national tuberculosis surveillance is to collect and submit a minimum of one suspicious lesion per 2,000 adult cattle slaughtered to the National Veterinary Services Laboratories (NVSL), Ames, Iowa, or to another laboratory officially approved by VS for detailed inspection.

FSIS Agrees:

1. To submit specimens (plus all manmade non-FSIS identification devices) to NVSL to be examined for tuberculosis from carcasses where lesions resembling tuberculosis or thoracic granulomas are found; in nonreactor cattle at a rate of at least one lesion per 2,000 adult cattle slaughtered and from reactor cattle as mutually agreed upon.

2. To make no charge to VS for collecting and submitting specimens resembling tuberculosis and thoracic granulomas.

3. To submit: (a) completed VS Form 6-35 for each nonreactor animal from which specimens are submitted, and (b) completed VS Form 10-4 (or FSIS Form 6000-1) with specimens from reactors sent to NVSL.

APHIS-VS Agrees:

1. To provide adequate personnel in plants where it is mutually agreed that assistance is required to meet program goals.

2. To examine specimens submitted by FSIS as promptly as possible for tuberculosis.

3. To report laboratory findings to FSIS by telephone, fax, or e-mail within 3 working days of receipt of specimens from carcasses retained for disposition because of suspected tuberculosis or thoracic granulomas; such telephone call, fax, or e-mail to be followed by a written summary of the histopathologic findings on VS Form 10-17 and mycobacteriologic results on VS Form 10-2.

4. To send slides from selected specimens as mutually agreed upon to the FSIS Field Service Laboratories in Athens, Georgia, or to other FSIS laboratories upon request.

5. To furnish shipping containers and preservative for submitting specimens to NVSL.

It is Mutually Understood and Agreed:

1. To work together to minimize the work of collecting, identifying, submitting, and reporting laboratory results on specimens resembling tuberculosis and thoracic granulomas.

2. That both VS and FSIS, insofar as possible, will inform the other of impending changes in procedure that are likely to affect the submission or handling of specimens.

3. That personnel from both Agencies will exchange visits to the laboratory and facilities of the other agency for professional interchange and uniformity.

4. That the two Agencies will collaborate in furnishing summaries to appropriate personnel of results obtained in this operation of mutual benefit.

Food Safety and Inspection Service (FSIS)

Animal and Plant Health Inspection Service (APHIS)
Veterinary Services (VS)

Identification of Mycobacterium Isolates

FSIS has qualified personnel available to analyze, and report evidence of mycobacterium in postmortem pathologic samples of domestic animals.

APHIS has laboratory expertise, facilities, and personnel available to identify several varieties of mycobacterium isolates and has an interest in these microorganisms due to their invasiveness for both animals and humans.

In that both parties have responsibilities is these areas, the parties agree with each other as follows:

FSIS Agrees:

- To coordinate efforts with APHIS-National Veterinary Services Laboratories (NVSL) to provide access to necessary pathology samples and related electronic data to promote the surveillance and eradication of tuberculosis.

- To cooperate with APHIS on routine inspection of meat and poultry including sampling and laboratory examination by APHIS-NVSL as required to isolate and identify mycobacteria.

- To collect and submit to APHIS-NVSL postmortem samples for microbiological identification of approximately 100 mycobacteria cultures per year during routine and special meat and poultry mycobacteria surveillance program activities.

APHIS-VS Agrees:

1. To advise FSIS of significant increases or decreases in mycobacteria infections or mortalities in domesticated animals.

2. To provide FSIS with monthly reports on cultures received from FSIS by APHIS.

3. To cooperate with FSIS to establish improved ways to share information gained via pathology samples to promote the eradication of tuberculosis.

It is Mutually Understood and Agreed:

1. That the details of this cooperative understanding shall be jointly planned and executed by the cooperating parties.

Appendix 6

Food Safety and Inspection Service (FSIS)

Animal and Plant Health Inspection Service (APHIS)
Veterinary Services (VS)

Foreign Animal Diseases and Reportable Diseases: Notification to VS when vesicular or other reportable conditions and/or lesions of foreign animal diseases are suspected in livestock or poultry during antemortem and/or postmortem inspection.

When vesicular or other reportable diseases or exotic diseases of foreign origin are suspected in a packing plant/slaughter establishment during antemortem or postmortem inspection, the Area Veterinarian in Charge (AVIC) for VS shall immediately be notified. Upon receipt of such a report, the following procedures are to be implemented by VS:

1. The AVIC immediately requests an investigation by the nearest trained foreign animal disease diagnostician (FADD).

2. The FADD will immediately respond to the request according to established APHIS protocols.

3. The FADD will do a thorough investigation, including areas such as case evaluation, possible traceout and premises of origin evaluation, and sample collection, as indicated.

4. All samples collected will be submitted to the Foreign Animal Disease Diagnostic Laboratory on Plum Island or the National Veterinary Services Laboratories at Ames, Iowa, depending upon the results of the investigation.

5. Serology test results will be available within 24 hours of receipt of the samples at the laboratory. Other tests, such as cell culture or animal inoculation, will take longer. However, in most cases, serology results or polymerase chain reaction should be sufficient for correct disposition of the carcasses and/or live animals.

6. VS will execute prompt response to FSIS plant personnel notifying them of the test results of submitted samples.

7. VS, upon request, will provide assistance in the inspection of livestock and poultry at slaughter when vesicular or other reportable or exotic disease of foreign origin are suspected

Appendix 7

APHIS Agreement No. 05-9208-0141-MOU
MEMORANDUM OF UNDERSTANDING
BETWEEN
UNITED STATES DEPARTMENT OF AGRICULTURE
FOOD SAFETY AND INSPECTION SERVICE (FSIS)
And
UNITED STATES DEPARTMENT OF AGRICULTURE
ANIMAL AND PLANT HEALTH INSPECTION SERVICE (APHIS)

ARTICLE 1 - PURPOSE

The purpose of this Memorandum of Understanding (MOU) is to document the requirements of the collaborative effort between APHIS AND FSIS through the sharing of the FSIS eADRS database system used to permit APHIS to access data for BSE Surveillance work.

ARTICLE 2 - BACKGROUND

The USDA BSE Surveillance program is reliant on data collected on-farm, and at slaughter plants and other sample collection facilities. Data for the program is primarily entered into the USDA BSE Surveillance system however; slaughter data is also entered daily by FSIS personnel into the electronic Animal Disease Reporting System.

ARTICLE 3 - AUTHORITIES

Under the Farm Security and Rural Investment Act of 2002, P.L. 107-171, Subtitle E, Animal Health Protection, Section 10401-10418, the Secretary of Agriculture, in order to protect the agriculture, environment, economy, and health and welfare of the people of the United States by preventing, detecting, controlling, and eradicating diseases and pests of animals, is authorized to cooperate with foreign countries, States, and other jurisdictions, or other persons, to prevent and eliminate burdens on interstate commerce and foreign commerce, and to regulate effectively interstate commerce and foreign commerce.

ARTICLE 4 - OBJECTIVE

APHIS has requested FSIS provide direct electronic access to the eADRS database to:

1) Validate information concerning animals sampled as a result of the BSE Surveillance program.
2) Assure comparability between data recorded on eADRS and the BSE Surveillance Information System
3) Reference data as needed for other surveillance programs and emerging animal health issues identification.

ARTICLE 5 – MUTUAL AGREEMENT

APHIS and FSIS jointly agree to assist in promptly troubleshooting access to eADRS problems incurred by APHIS and resolving the system conflicts identified as a result.

ARTICLE 6 - FSIS RESPONSIBILITIES

FSIS will provide APHIS with read only access to the data obtained from the electronic Animal Disease Reporting System (eADRS) database. Provide electronic access capability including network authorization, and system and application user ID's/passwords to two APHIS/VS/CEAH personnel.

Assist in promptly troubleshooting access problems and resolving system conflicts identified.

Initial contact: Steve Weber
USDA:APHIS:VS:CEAH
2150 Centre Ave, Building B
Fort Collins, CO 80526
Telephone (970) 494-7271
Fax (970) 472- 494-7269____
Internet: steve.weber@aphis.usda.gov

ARTICLE 7 – APHIS: VS: CEAH RESPONSIBILITIES

APHIS will use the data to:

1) Validate information concerning animals sampled as a result of the BSE Surveillance program.
2) Assure comparability between data recorded on eADRS and the BSE Surveillance Information System
3) Reference data as needed for other surveillance programs and emerging animal health issues identification.

APHIS (CEAH) agrees to protect the confidentiality and sensitivity of the data being provided to the extent required by Federal regulations and FOIA. Furthermore, USDA APHIS will not release, publish, or publicly report any proprietary information originating from the eADRS, and will consult with FSIS prior to proposing policy or program direction based on the data obtained. USDA APHIS will make available to FSIS, upon request, electronic copies of internal reports derived from eADRS data.

- Limit electronic data access only to two authorized personnel.
- Access system only for retrieval or analysis of identified information and log off eADRS system promptly after retrieving necessary data.
- Utilize data retrieved from eADRS only for purposes identified above.

ARTICLE 8 - STATEMENT OF NO FINANCIAL OBLIGATION

Signature of this MOU does not constitute a financial obligation on the part of APHIS or FSIS. Each signatory party is to use and manage its own funds in carrying out the purpose of this MOU.

ARTICLE 9 - LIMITATION OF COMMITMENT

This MOU and any continuation thereof shall be contingent upon the availability of funds appropriated by the Congress of the United States. It is understood and agreed that any

monies allocated for purposes covered by this MOU shall be expended in accordance with its terms and in the manner prescribed by the fiscal regulations and/or administrative policies of the party making the funds available. If fiscal resources are to transfer, a separate agreement must be developed by the parties.

ARTICLE 10 - CONGRESSIONAL RESTRICTIONS

Not applicable.

ARTICLE 11 - AMENDMENTS

This MOU may be amended at any time by mutual agreement of the parties in writing.

ARTICLE 12 - TERMINATION

This MOU may be terminated at any time by mutual agreement of the parties in writing, or by one party within 30 days notice in writing of the other party.

ARTICLE 13 - EFFECTIVE DATA AND DURATION

This MOU will be in effect upon final signature and will continue until December 31, 2005

NAME: Kenneth Petersen
TITLE: Acting Deputy Assistant Administrator
USDA:FSIS:OFO
CITY, STATE Washington, DC

/s/ Kenneth Petersen ___ 11/23/04
Signature Date

NAME: Dr. John Clifford
TITLE: Deputy Administrator
USDA:APHIS Veterinary Services
Washington DC

/s/ John R.Clifford 11/19/04
Signature Date

United States Department of Agriculture

Marketing and Regulatory Programs

Animal and Plant Health Inspection Service

Food Safety

Food Safety and Inspection Service

Washington, DC 20250

Appendix 8

VETERINARY SERVICES AND OFFICE OF FIELD OPERATIONS
MEMORANDUM NO. 552.7

Subject: Post Mortem Techniques for Tuberculosis Reactors and Suspects

To: VS Management Team
Area Veterinarians in Charge, VS
State Veterinarians
FSIS Office of Field Operations Headquarters Executive Team
FSIS District Office Managers
OPPD Assistant Administrator

I. PURPOSE

This memorandum establishes a training program with the Food Safety and Inspection Service (FSIS), Office of Field Operations that will provide Animal and Plant Health Inspection Service (APHIS), Veterinary Services (VS), veterinarians an opportunity to become skilled in the proper techniques of conducting post mortem examinations of tuberculin test reactors and suspects.

II. CANCELLATION

This memorandum replaces and cancels Animal Health Division Memorandum 552.7, dated February 20, 1970.

III. GENERAL

VS remains committed to the longstanding practice that FSIS provides specialized training to selected VS veterinarians in proper post mortem techniques for tuberculosis reactors and suspects.

When tuberculosis reactors or suspects are slaughtered in federally or State inspected slaughtering establishments, the VS Area Veterinarian in Charge (AVIC) is responsible for confirming that thorough post mortem examinations and proper disposition or disposal of tuberculin reactor cattle were conducted according to official procedures. These procedures are of mutual benefit to FSIS and VS and are prerequisites to the payment of Federal indemnity.

VS and OFO Memorandum No. 552.7 2

In order to provide the thorough and high quality post mortem examinations necessary, VS veterinarians must receive appropriate instruction and be provided opportunities for practical experience. To achieve this, FSIS will provide in-plant training without cost to VS, and VS will pay the travel, per diem, and similar costs for VS personnel participating in the training.

This training will provide FSIS with professional assistance, particularly when large numbers of reactors or suspects are slaughtered at an establishment. As needed, VS veterinarians would be available to assist with the post mortem examination of tuberculosis reactors or suspects being slaughtered at federally inspected slaughter establishments. The appropriate AVIC will inform the appropriate FSIS District Managers and State and local meat inspection managers that VS veterinarians are available to assist with post mortem examinations.

IV. TRAINING PROGRAM

A. FSIS agrees to assist in classroom training at the National Bovine Tuberculosis Epidemiology Training Course in Ames, Iowa.

VS periodically conducts the National Bovine Tuberculosis Epidemiology Training Course for VS veterinarians and State veterinarians at the National Veterinary Services Laboratories (NVSL), Ames, Iowa. Instruction is provided by tuberculosis epidemiologists; FSIS veterinarians experienced in tuberculosis slaughter surveillance; university professors involved with the pathology, immunology, and epidemiology of tuberculosis; and tuberculosis laboratory experts from NVSL. This 1 week course has limited hands-on post mortem training activity. APHIS will pay for travel and per diem expenses for FSIS instructors participating in this course.

B. APHIS agrees to coordinate field training with FSIS.

The AVICs will submit nominations for post mortem training to the National Tuberculosis Program Coordinator, National Center for Animal Health Programs (NCAHP), Eradication and Surveillance Team, VS, who will arrange for the training with the FSIS Center for Learning (CFL) in College Station, Texas. Notification of training dates will be provided well in advance.

After the formal training at NVSL, selected VS veterinarians (in groups of two or three) will be detailed to official establishments for training. FSIS will determine and schedule the training dates. The training period may require approximately 1 week (3 days training and 2 days travel). If more or less time is required, the FSIS veterinary field mentor may determine the time required to achieve the necessary results. APHIS will

pay all travel costs for the trainees. FSIS will issue a copy of the most current guidelines to each VS trainee upon arrival at the designated establishment.

VS veterinarians will spend time on the line in the slaughterhouse working with and learning from FSIS veterinarians skilled in post mortem techniques. VS veterinarians should be able to observe a significant amount of pathology and learn from their FSIS colleagues how to locate and evaluate the appropriate lymph nodes and proper procedures to collect, prepare, and ship various laboratory specimens. VS veterinarians will be able to appreciate the multitude of activities occurring in slaughterhouses and learn how to interact effectively with FSIS and industry in the slaughterhouse environment. FSIS veterinarians and food inspectors will receive additional information regarding the importance of their role in the Cooperative State-Federal Tuberculosis Eradication Program and will be exposed to epidemiological tracing, tuberculosis testing, prevalence and incidence of bovine tuberculosis, etc.

V. EQUIPMENT

APHIS will supply the appropriate equipment and clothing, such as scabbard, knife, stone, steel, hook, white smocks or coveralls, helmet, and rubber boots, to the trainee well in advance of the scheduled training.

VI. CERTIFICATE OF TRAINING

FSIS, CFL, will collaborate with VS to appropriately certify the VS trainees. The training report signed by the FSIS mentor and the SF-182 (Request, Authorization, Agreement, and Certification of Training) will document the training. The title of the course is to be shown as "Post Mortem Techniques for Tuberculosis Reactors or Suspects." The CFL will track training completion in the training database.

1/21/04 /s/ Ron DeHaven	1/21/04 /s/ William Smith
W. Ron DeHaven	William Smith
Deputy Administrator	Assistant Administrator
Veterinary Services	Office of Field Operations
Animal and Plant Health Inspection Service	Food Safety and Inspection Service

Appendix 9

MEMORANDUM OF UNDERSTANDING
BETWEEN
UNITED STATES DEPARTMENT OF AGRICULTURE
FOOD SAFETY INSPECTION SERVICE
AND
UNITED STATES DEPARTMENT OF AGRICULTURE
ANIMAL AND PLANT HEALTH INSPECTION SERVICE

Relative to Bovine Spongiform Encephalopathy (BSE) Sampling of Cattle Condemned on Ante mortem Inspection

The parties to this Memorandum of Understanding are the United States Department of Agriculture, Food Safety and Inspection Service, hereinafter called FSIS, and the United States Department of Agriculture, Animal and Plant Health Inspection Service, hereinafter called APHIS.

WHEREAS, BSE is a reportable animal disease in the United States,

WHEREAS, FSIS has qualified personnel available to inspect, condemn, and obtain samples from condemned animals; and

WHEREAS, APHIS has laboratory expertise, facilities, and personnel available to conduct tests on cattle brains for BSE; and

WHEREAS, both parties have responsibilities in these areas,

NOW, THEREFORE, in order to ensure that all cattle condemned on ante mortem inspection are sampled for BSE, the parties hereto do mutually agree to the following:

A. The FSIS agrees:

1. To collect brain samples from all cattle that are condemned by FSIS upon ante mortem inspection at federally inspected establishments. Veal calves condemned for reason other than CNS disorders are exempt from this collection.
2. To provide training for FSIS Public Health Veterinarians designated to carry out such sampling.
3. To provide for shipping of theses samples to an APHIS laboratory designated by APHIS for the purpose of BSE testing.

B. The APHIS agrees:

1. To obtain samples from animals at locations other than federally inspected establishments, including animals that are dead on arrival-but not offloaded-through their routine sampling and agreements with dead-stock facilities, renderers, and other animal disposal facilities.

2. To coordinate with the FSIS State Liaison Directors, through memoranda of understanding or other such agreements, to ensure sample collection from State-inspected facilities.

3. In cases where APHIS has provided funding for a technician to remain on the premises and take samples at federally inspected establishments, that technician when conducting BSE sampling is under the supervisory oversight of the FSIS inspector-in-charge. The FSIS inspector–in-charge will have the authority to direct the APHIS technician to select samples for BSE submission.

4. To provide FSIS with the following equipment and supplies:
   a. special sample boxes, pre-addressed shipping labels, and shipping instructions,
   b. equipment needed to harvest samples, and
   c. protective equipment for FSIS personnel who will be taking samples.

5. To test samples collected by FSIS at the National Veterinary Services Laboratory (NVSL) in Ames, Iowa, or another APHIS-designated laboratory.

6. To promptly report the results of testing in accordance with the BSE communications plan.

7. To provide FSIS with access to the APHIS database containing sample results and associated information.

8. To reimburse FSIS for the following costs:
   a. $40 per sample for samples collected by FSIS personnel for FY 2004. In subsequent fiscal years, this amount will be adjusted for inflation and increases in pay and benefits.
   b. Up to $75,000 in FY 2004 to provide initial training for its Public Health Veterinarians to carry out the sample collection.
   c. Up to $1.6 million initially and up to $100,000 annually thereafter for rabies vaccination of Public Health Veterinarians who request such vaccination.
   d. Transfer $2.0 million in FY 2004 to provide FSIS inspection personnel with the necessary telecommunications and hardware for the electronic transfer of data to APHIS. FSIS will use these funds to provide for electronic transfer of data in Federal establishments that slaughter 50 or more cattle per month.
   e. Transfer up to $1.0 million per year, beginning in FY 2005 for the annual telecommunication service costs associated with the electronic transfer of data. This annual funding will continue for the duration of FSIS participation in BSE surveillance testing.

C. <u>It is Mutually Understood and Agreed:</u>

1. That the details of this cooperative undertaking shall be jointly planned and executed by the cooperating parties.

2. Expenditures made by either party will be in accord with its particular rules and regulations.

3. The results of the work herein shall be shared between parties, and any manuscripts prepared for publication by wither shall be submitted to the other party for suggestions and approval prior to publication.

4. The provisions of the Memorandum of Understanding shall be reviewed annually.

5. By mutual agreement, this Memorandum of Understanding shall become effective upon the date of final signature and shall continue indefinitely but may be modified or discontinued at the request of either party. Requests for termination or any major modification shall be submitted to the other party in writing for consideration not less than 60 days in advance of the effective date desired.

UNITED STATES DEPARTMENT OF AGRICULTURE
FOOD SAFETY INSPECTION SERVICE

2/12/02 /s/ Barbara J. Masters
Date Acting Administrator

UNITED STATES DEPARTMENT OF AGRICULTURE
ANIMAL AND PLANT HEALTH INSPECTION SERVICE

7/15/04 /s/ Kevin Shea
Date Acting Administrator

Appendix 10

Food Safety and Inspection Service (FSIS)

Animal and Plant Health Inspection Service (APHIS)
Veterinary Services (VS)

Electronic reporting of slaughter data (eADRS) to APHIS

Statistics concerning the number and class of animals slaughtered and estimated prevalence of disease in these animals are collected by FSIS personnel in slaughter plants. These statistics are compiled and entered into the FSIS database. These slaughter surveillance data are critical for VS to establish animal disease program status, evaluate the effectiveness of animal identification and blood sample collection, and to measure program targets and goals.

In addition, condemnation data is important to VS to identify disease trends and risk areas that VS needs to investigate as possible foreign animal disease incursions or emerging disease outbreaks/issues.

FSIS Agrees:
FSIS agrees to provide APHIS with read only electronic access to the data obtained from the eADRS database including network authorization, and system and application user ID's/passwords to two APHIS/VS/NCAHP personnel.

APHIS agrees use the data to:

1) Validate information concerning animals sampled as a result of animal disease and surveillance programs.

2) Protect the confidentiality and sensitivity of the data being provided to the extent required by Federal regulations and the Freedom of Information Act. Furthermore, APHIS will not release, publish, or publicly report any proprietary information originating from the eADRS and will consult with FSIS prior to proposing policy or program direction based on the data obtained. APHIS will make available to FSIS, upon request, electronic copies of internal reports derived from eADRS data.

3) Limit electronic data access only to two authorized personnel.

4) Access system only for retrieval or analysis of identified information and log off eADRS system promptly after retrieving necessary data.

5) Utilize data retrieved from eADRS only for purposes identified above.

APHIS and FSIS jointly agree to assist in promptly troubleshooting access to eADRS problems incurred by APHIS and resolving the system conflicts identified as a result.

V. BOVINE TUBERCULOSIS ERADICATION AWARDS PROGRAM

January 24, 2001
VETERINARY SERVICES MEMORANDUM NO. 540.6

Subject: Bovine Tuberculosis Eradication Performance Awards Program
To: Directors, VS Regions Area Veterinarians in Charge, VS

I. PURPOSE

A. Revise and update the special performance awards program for bovine tuberculosis eradication.

B. Authorize issuance of awards to Food Safety and Inspection Service (FSIS) Food Inspectors and Public Health Veterinarians (PHV's) assigned to Federally inspected cattle, bison, and cervid slaughtering establishments for their significant contributions to the eradication of tuberculosis in cattle, bison, and cervids.

C. Provide policies and procedures for nominating, selecting, and rewarding such employees under this program.

D. Describe the types of awards to be given.

II. BACKGROUND

Instructions for the Bovine Tuberculosis Eradication Performance Awards Program were previously outlined in the Animal and Plant Health Inspection Service (APHIS) Directive 540.6, June 18, 1996. Recommendation for a cash award under the Bovine Tuberculosis Eradication Program is a part of the Comprehensive Strategic Plan for the Eradication of Bovine Tuberculosis, dated October 2000.

III. GOAL

The goal of this awards program is to reward timely detection of bovine tuberculosis and increase the quality and number of laboratory specimens submitted by FSIS personnel.

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IV. COVERAGE AND AREA OF CONSIDERATION

This award recognizes FSIS employees for their superior contributions in support of the Bovine Tuberculosis Eradication Program. Food Inspectors (grades GS-7 through GS-9) and PHV's (grades GS-9 through GS-13) are eligible**.**

V. TYPE OF AWARD

FSIS Food Inspectors and PHV's will be considered for:

A. A cash award of $100 for steers and $500 for adult animals to be shared equally each time Mycobacteriosis is reported on histopathology by the National Veterinary Services Laboratories (NVSL). If the specimen is positive for *Mycobacterium tuberculosis (complex)* on Polymerase Chain Reaction (PCR) test, or *M. bovis* is isolated, the cash award will be increased to a total of $200 for steers and fed heifers and $1,000 for adult animals. Tissues submitted only to FSIS field service laboratories or to other approved, diagnostic laboratories that are indicative of tuberculosis shall be forwarded to NVSL for reconfirmation in order to qualify for an award.

B. A second cash award of $6,000 to be shared equally when an infected herd located in the United States is initially found as a result of the information provided to Veterinary Services (VS) regarding the identification of the lesioned animal.

Note: Each award is to be shared with the Food Inspector or Inspectors responsible for retaining the affected carcass and the PHV initiating the VS 6-35 report. In the event of multiple cases in the same slaughter lot, awards will be granted for as much as three cases from such a lot. Specimens from animals sent to slaughter under permit because of tuberculosis (reactors, suspects, animals from quarantined herds, exposed animals being depopulated, and exposed animals traced to new herds) will not qualify as a basis for an award.

C. A team award of **$**300 per team member will be awarded annually to high submitting FSIS slaughter inspection groups irrespective of histopathology results. High submitting establishments will qualify, at the end of each 12-month period (Fiscal Year), when the plant is credited with one or more suspicious tuberculosis lesions or thoracic granulomas submitted per 1,000 cattle killed.

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The accounting will be kept according to the following procedure: Each time a VS Form 6-35 is completed, the recording official (initiating PHV) and Food Inspector will place their signatures and print legibly their names on the form. VS will keep an account of names by submission and establishment. For each establishment attaining the goal of one submission per 1,000 cattle killed, all participants in that achievement group will be considered team members warranting the team award. An individual inspector is eligible for no more than one team award per year. Dollar amounts of the team award are expected to remain at **$**300 per member, but may change periodically, up or down, according to available budgetary allowances. FSIS District Managers may participate in determining the team makeup.

The monetary provisions of these awards are effective on the issuance date of this memorandum.

Note: There is a direct correlation between successful tracebacks and the accuracy of systems correlating identification devices with the correct carcass. In the experience of VS, "countback" systems of recovering ID have resulted in about 50 percent unsuccessful tracebacks to the correct herd of origin. More positive carcass/ID correlation systems are highly recommended.

Note: Submitting specimens (and ID) from cattle, bison, and cervidae condemned for granulomatous conditions would contribute to numbers of submissions needed to fulfill team awards criteria.

VI. CRITERIA FOR NOMINATION

To be eligible for consideration for an award:

A. The Food Inspector must:
1. Detect lesions of possible tuberculosis in a regular kill animal and hold for further examination by the PHV; and
2. Collect identification devices and coordinate identification with the affected carcass.

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B. The Public Health Veterinarian must:
1. Make the determination that the lesions may be tuberculosis and should be submitted for examination;
2. Collect and submit the specimens to NVSL, Ames, Iowa, for histopathologic and mycobacteriologic examination;
3. Collect all identification devices (such as ear tag, back tag, sale tag) with any supporting information (slaughter permit, brands, herd of origin) and submit them with the specimens to NVSL; and
4. Complete VS Form 6-35, “Report of Tuberculosis Lesions or Thoracic Granulomas in Regular Kill Animals.” All identification collected (see paragraph VI.B.3 above) should be noted in the report.

All identification devices should be placed in a shipping container with the specimens. The better the quality of specimens submitted, the greater likelihood of mycobacterial confirmation (paragraph V.A). The more complete and accurate the identification of the animal, the greater likelihood of finding the infected herd of origin (paragraph V.B).

VII. ADMINISTRATIVE PROCEDURES

A. Animal Health Programs Staff (AHPS), VS, will prepare the appropriate documents for award consideration when an FSIS employee meets the requirements outlined in section VI.

B. Senior Staff Veterinarian for Tuberculosis Eradication will review the award recommendation to ensure all criteria have been met, and forward documentation to Marketing and Regulatory Programs-Business Services (MRP-BS), Minneapolis Business Site (MBS), Personnel Services, Processing Team, Minneapolis, Minnesota.

C. MBS, Personnel, Processing Team, by authority outlined in a Reimbursable Agreement, will input this data into their payroll system and electronically transmit the data to the National Finance Center (NFC), New Orleans, Louisiana, for issuance of the check. NFC will forward the check to the appropriate VS Area Office.

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D. Area Offices will:

1. Prepare a letter of appreciation for signature by the Area Veterinarian in Charge (AVIC);
2. Forward a copy of the letter of appreciation to MBS, Minneapolis, Minnesota, for the employee's official personnel folder; and
3. Arrange for an appropriate presentation. The AVIC will personally present the award to the employees when possible.

VIII. PUBLICIZING AWARDS

The Animal Health Programs Staff, VS, will provide information to the Administrators of APHIS and FSIS for publicizing the awards within each Agency. They will also provide information to the National Association of Federal Veterinarians for publicizing the awards in their monthly newsletter. Local newspaper coverage is also encouraged.

IX. EFFECT ON OTHER AWARDS

No previous TB incentive award should in any way jeopardize or enhance subsequent incentive awards or any other performance award for which the employee may be eligible.

/s/Chester A. Gipson for
Alfonso Torres
Deputy Administrator
Veterinary Services

VI. List of APHIS Veterinary Services, Area Veterinarians In Charge (AVIC's)

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VIII. Paper: "Surveillance of Zoonotic Diseases"

Background:

Legal Authority/Statutory Directive:

The Public Health Security and Bioterrorism Preparedness and Response Act of 2002 ("the Act") (PL 107-188) was signed into law on June 12,2002. The Act instructs the Secretary of Health and Human Services, through the Commissioner of FDA and the Director of CDC, and the Secretary of the U. S. Department of Agriculture to coordinate surveillance of zoonotic diseases. This terminology appears in Title III of the Act, Subtitle A, "Protection of the Food Supply," Section 313.

The Act provides little guiding language to define the scope of Congressional intent in this section. In its broadest terms, "zoonotic diseases" includes all infections, toxico-infections, and intoxications related to microorganisms that may cause disease in humans and are transmitted either directly or indirectly from animals. Inasmuch as Section 313 emerges in Title III, "Protecting Safety and Security of Food and Drug Supply," there is a natural extension of the definition to include infectious diseases of animals that would threaten our food sources (e.g., exotic animal diseases) and human nutrition, even if these additional diseases are not caused by human pathogens. Therefore, the most robust response by HHS and USDA to Section 313 will address all of the diseases caused by bacteria, parasites, viruses, and prions that are shared by humans and other animals (domestic and wild), plus those diseases of domestic animals that pose a substantial risk of large epidemics in our livestock and poultry populations or have characteristics that make them likely (but unrecognized) agents of human disease. The latter category of potential zoonotic agents will be useful sources of hypotheses when syndromic surveillance of human illnesses identifies clusters without known etiologic agents. While a little further afield, natural toxins, such as mycotoxins and marine biotoxins, fit well within the counter-terrorism context of zoonoses, and could be included as well.

Discussion:

Summary of Proposed Action:

Passive surveillance of certain zoonotic diseases in humans and other animals, and microbiologic monitoring for commonly encountered zoonotic agents in foods, feeds, cosmetics, biologics, and medical devices already occurs to some extent for communicable disease control and regulatory purposes. In the U. S. many zoonotic diseases are of low incidence, and the programs related to them tend to be under-resourced, uncoordinated, and unattended by decision-makers at the highest levels. In addition to known zoonotic agents, there exists, probably in low frequency, infectious agents of xenogeneic origin that may become pathogenic in humans under certain circumstances. Ratcheting up the current FDA programs to counter-terrorism levels and coordinating them for maximum public health benefit will require additional human and other resources, and an expanded scope. Broadly speaking, the surveillance interest and expertise in this range of zoonoses is distributed among agencies in HHS and USDA as follows:

- CDC/NCID: Human diseases caused by all zoonotic pathogens, regardless of route of transmission, and clinical isolates of the agents.
- USDA/APHIS: Diseases of domestic animals caused by zoonotic pathogens or by agents that threaten to cause large epidemics in livestock and poultry populations, and clinical isolates of agents in both categories.
- FDA/ORA:
- FDA/CFSAN: Foodborne zoonotic agents isolated from food, dietary supplements, special nutritionals, and cosmetics.
- FDA/CVM: Feed isolates of agents that are transmissible to humans or that can cause epidemic disease in animals; surveillance of antimicrobial resistance.
- USDA/FSIS: Meat, poultry and egg product isolates of agents that are transmissible to humans or that can cause epidemic disease in animals.
- FDA/CBER: Xenogeneic infectious agents that can contaminate non-human origin cell cultures or tissue cultures used in vaccine production, or transmit from biologic-producing or xenotransplantation products to a human recipient. This includes, in addition to known zoonotic agents, xenogeneic agents that might not otherwise be pathogenic in humans or have the opportunity to infect humans.
- FDA/CDRH: Zoonotic agents that can contaminate non-human origin xenotransplantation products and be transmitted to human recipients.
- FDA/CDER: Zoonotic agents that contaminate non-human drug ingredients.

Broad interpretation of Section 313 will result in an interagency program that incorporates a number of different surveillance approaches and priorities, with each activity informing and enhancing the effectiveness of the others (a ZooNet, perhaps?). The combined data from all of these related surveillance systems will create a baseline to characterize “normal” so unusual events can be rapidly identified, characterized, and contained. Developing such a program requires a number of activities, including the following:

- Prioritizing agents to allow for the rational development of surveillance programs.
- Prioritizing products likely to be terrorist targets for the high priority agents.
- Development of sensitive, specific, cost-effective and practical diagnostic tools.
- Validation of diagnostic tools and training in their use.
- Development of survey instruments and systems for data management and analysis.
- Implementation into surveillance programs.
- Coordination of data management, sharing, and use by interagency collaborators.

Within this mix of surveillance activities, CFSAN’s initial response to Section 313 will be to develop guidance to the FDA field staff related to procedures to be followed for microbiologic surveillance of zoonotic agents in foods, dietary supplements, special nutritionals, and cosmetics regulated by FDA. Further development of this concept to incorporate a broader perspective (CBER, CDRH, CVM, CDER, CDC) and inter-departmental (HHS and USDA) approach will be achieved through participation in drafting the concept paper by representatives of other Centers and agencies. Developing this guidance will include risk ranking for agents and agent/product combinations, assessing current methods capabilities, implementing current methods and a sampling plan in the field, developing laboratory methods as needed and employing these new methods in the field, and establishing procedures for collating, analyzing, communicating, and responding to the results.

The guidance from CFSAN will be considered together with related guidance from the other Centers to establish FDA surveillance priorities and a work plan for inspections and sample

analyses. FDA priorities and work plans will be coordinated with those of CDC, APHIS, and FSIS. Clinical, product, and environmental isolates derived from microbiologic surveillance systems can be pooled in interagency strain sets (for subtyping, GIS, and other collaborative analyses).

Time line: **[Note: This time line relates only to the CFSAN related issues].**

Event	Due Date
1. Develop concept paper	October 18, 2002
2. Clear concept paper through FDA	November 4, 2002
3. Develop risk ranking	November 29, 2002
4. Determine current methods capabilities	December 13, 2002
5. Implement current methods in the field, with appropriate sampling plan	March 2003
6. Develop procedures for collating, analyzing, communicating, and responding to the results.	June 2003
7. Develop and validate first round of new methods (those at the top of the risk ranking)	September 2003
8. Implement new methods in the field with appropriate testing program.	December 2003
9. Develop and validate second round of new methods	September 2004
10. Implement new methods in the field with appropriate testing program.	December 2004

Preliminary Cost/Benefit Analysis: Initial evaluation suggests that economic analysis is not necessary for implementing Section 313.

Small Entity Analysis: Not applicable.

Stakeholder Interest: Section 313 imposes no new regulatory or record-keeping requirements on industry, and this will limit the immediate impact and level of interest. However, zoonotic agents have media appeal and cause public concern, so enhanced detection of these agents and their association with products in commerce will create at least a moderately high level of stakeholder interest. More active and coordinated interagency surveillance programs will improve control of zoonoses in humans and other animals generally, decrease the response time to intentional and unintentional health emergencies involving zoonoses, and increase the likelihood that infections and epidemics can be predicted and prevented.

IX. Internet Resources:

World trade organization agreement on the association of sanitary and phytosanitary measures

http://www.wto.org/goods/spsagr.html

Codex Alimentarius for Food Safety Health Concerns:

http://www.codexalimentarius.net

Organization International des Epizooties for Animal Health Concerns:

http://www.oie.int/

International Plant Protection Convention for Agriculture Health Concerns:

http://www.fao.org/ag/agp/agpp/pg/

International Animal Health Code: recommended rules for agricultural commerce:

http://www.oie.int/eng/normes/mcode/a_summary.htm

Manual of Standards for Diagnostic Tests and Vaccines:

http://www.oie.int/eng/publicat/en_standards.htm

International Aquatic Animal Health Code and Diagnostic Manual for Aquatic Animal Diseases:

http://www.oie.int/eng/publicat/en_aqua.htm

Regulations base for import and export – Title 9 Code of Federal Regulations:

http://www.access.gpo.gov/nara/cfr/waisidx_01/9cfrv1_01.html

Communicates international requirements:

http://aphis.usda.gov/guidance/regulations/animal/

FSIS import-export:

http://www.fsis.usda.gov/OFO/export/explib.htm

Regionalization in APHIS:

http://www.aphis.usda.gov/vs.reg-request.html

Andean Community:

http://www.comunidadandina.org/

http://www.comunidadandina.org/normativa/RES/R449.HTM

OIRSA – Regional Organization for Agricultural Health

http://ns1.oirsa.org.sv/

Mercosur – Common Market of South America

http://www.mercosur.org/english/default.htm

North American Free Trade Agreement

http://www.sice.oas.org/trade/nafta/naftatce.asp

X. WORLD ORGANIZATION FOR ANIMAL HEALTH (OIE) NOTIFIABLE DISEASE LIST

Multiple species diseases

- Anthrax
- Aujeszky'sdisease
- Echinococcosis/hydatidosis
- Heartwater
- Leptospirosis
- Q fever
- Rabies
- Paratuberculosis
- New world screwworm (*Cochliomyia hominivorax*)
- Old world screwworm (*Chrysomya bezziana*)
- Trichinellosis
- Foot and mouth disease (FMD)
- Vesicular stomatitis
- Lumpy skin disease
- Bluetongue
- Rift Valley fever

Cattle diseases

- Bovine anaplasmosis
- Bovine babesiosis
- Bovine brucellosis
- Bovine genital campylobacteriosis
- Bovine tuberculosis
- Bovine cysticercosis
- Dermatophilosis
- Enzootic bovine leucosis
- Hemorrhagic septicemia
- Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis
- Theileriosis
- Trichomonosis
- Trypanosomosis (tsetse-transmitted)
- Malignant catarrhal fever
- Bovine spongiform encephalopathy
- Rinderpest
- Contagious bovine pleuropneumonia

Sheep and goat diseases

- Ovine epididymitis (*Brucella ovis*)
- Caprine and ovine brucellosis (excluding *B.ovis*)
- Caprine arthritis/encephalitis
- Contagious agalactia

- Contagious caprine pleuropneumonia
- Enzootic abortion of ewes (ovine chlamydiosis)
- Ovine pulmonary adenomatosis
- Nairobi sheep disease
- Salmonellosis (*S.abortusovis*)
- Scrapie
- Maedi-visna
- Peste des petits ruminants
- Sheep pox and goat pox

Swine diseases

- Atrophic rhinitis of swine
- Porcine cysticercosis
- Porcine brucellosis
- Transmissible gastroenteritis
- Enterovirus encephalomyelitis
- Porcine reproductive and respiratory syndrome
- Swine vesicular disease
- African swine fever
- Classical swine fever

Avian diseases

- Avian infectious bronchitis
- Avian infectious laryngotracheitis
- Avian tuberculosis
- Duck virus hepatitis
- Duck virus enteritis
- Fowl cholera
- Fowl pox
- Fowl typhoid
- Infectious bursal disease (Gumboro disease)
- Marek's disease
- Avian mycoplasmosis (*M.gallisepticum*)
- Avian chlamydiosis
- Pullorum disease
- Highly pathogenic avian influenza (HPAI)
- Newcastle disease

Equine diseases

- Contagious equine metritis
- Dourine
- Epizootic lymphangitis
- Equine encephalomyelitis (VEE, EEE and WEE)
- Equine infectious anemia
- Equine influenza
- Equine piroplasmosis
- Equine rhinopneumonitis

- Glanders
- Horse pox
- Equine viral arteritis
- Japanese encephalitis
- Horse mange
- Surra (*Trypanosoma evansi*)
- African horse sickness