Assessment and Management of Disease Risks in Wildlife

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Agriculture and wildlife groups are concerned about the impact of infectious disease on their animals, and each group has apprehension about possible disease introduction from the other's animals. Wild animals generally are susceptible to infection by the same bacteria, viruses, and parasites that infect domestic animals, and transmission of disease agents between wildlife and livestock can occur in either direction. Thus, measures taken to prevent disease transmission in either direction can benefit both wildlife and livestock and are far more efficient than the efforts necessary to eliminate an established disease from domestic or wild animals. The role of wildlife in the epidemiology of domestic animal and human diseases varies greatly, depending on disease agent and host species. Infected wild animals can represent a true risk factor, or they may harbor significant pathogens while posing little or no threat to other species. Consequently, the level of risk must be evaluated to determine whether control programs are necessary or worthwhile. Strategies to assess and reduce risk must be based upon thorough knowledge of the epidemiology of the disease agent in wildlife, humans, and domestic animals; specific information regarding the local situation; and other factors. Risk evaluation and management efforts will involve organizations with differing expertise and cooperation will be essential between wildlife management, public health, and domestic animal health agencies. Risk reduction strategies, when deemed necessary or feasible, may be based upon manipulation of the disease agent, the host, the environment, and/or human activities. Management of human activity, particularly the promotion of biosecurity, may be most efficient strategy because manipulations of the disease agent, host, or environment are more difficult and expensive. The science of risk assessment and disease management in wildlife is growing and evolving as new situations arise and as new methods are developed to meet the needs of wildlife resource, animal agriculture and public health interest groups.

Chronic Wasting Disease: Implications for Domestic Animals

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Chronic wasting disease (CWD) is a disease of deer and elk; natural susceptibility of domestic species has not been identified. However, even though CWD is not recognized to naturally affect domestic animals it still has significant implications for our domestic livestock industries, the game farming industry, and for management of free-ranging populations of deer and elk. Chronic wasting disease is a member of the transmissible spongiform encephalopathies (TSEs) or prion diseases and is related to bovine spongiform encephalopathy (BSE) and the associated fatal human disease, variant Creutzfeldt-Jakob disease (vCJD). Although CWD may have significant negative impacts on populations of free-ranging deer, it is because of its relationship to BSE, vCJD, and the unknowns surrounding the TSEs and species barriers that drive concerns about CWD and domestic animals. A variety of studies have been and are investigating the relationship of CWD to other TSEs and the potential susceptibility of domestic species to the CWD agent. Preliminary evaluation of these studies suggests a closer relationship of CWD to scrapie than to other TSEs. Implications of CWD for domestic animals vary from direct, should it be found that CWD is naturally transmissible to food animals, to indirect, based on perceptions of the public and regulatory agencies to risks associated with the TSEs of animals.

Chronic Wasting Disease in Farmed Deer and Elk

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Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy of North American deer (*Odocoileus* spp.) and elk (*Cervus elaphus nelsoni*). CWD has been detected in wild cervid populations in the U.S. and Canada and in farmed cervids in the U.S., Canada, and South Korea. CWD was first discovered in wildlife research facilities in Colorado and Wyoming in the late 1960's and early 1970's and first detected in wildlife in these States in the 1980's. CWD was first detected in farmed animals in Canada in 1996 and in the U.S. in 1997. Since then the disease has been identified in additional farmed elk herds as well as farmed white-tailed deer herds in a total of eight States in the U.S. (Colorado, Kansas, Minnesota, Montana, Nebraska, Oklahoma, South Dakota, and Wisconsin) and farmed elk herds in two Canadian provinces (Saskatchewan and Alberta). CWD was identified in a farmed elk herd in South Korea in 2001. USDA is working with the States and the farmed cervid industry to develop a CWD herd certification program in the U.S. The goal of the program is to eliminate CWD from farmed cervids. In addition, USDA is working with the U.S. Department of the Interior and State, Federal, and Tribal wildlife agencies in their surveillance and disease management efforts. CWD presents unique disease management and regulatory challenges. These challenges as well as the current distribution and status of CWD in farmed cervids in the U.S. will be summarized.

Management of Chronic Wasting Disease in Wisconsin White-Tailed Deer (*Odocoileus virginianus*)

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Chronic wasting disease (CWD) is a progressively degenerative and ultimately fatal disease of deer (*Odocoileus* spp.) and elk (*Cervus elaphus*) associated with transmissible protease resistant prion proteins.[3] From the recognition of CWD as a transmissable spongiform encephalopathy in 1978 until 2000, the range of CWD in wild cervids was thought to be limited to Colorado, Wyoming and Nebraska. In the last 3 years, CWD has also been detected in wild deer and elk in Illinois, New Mexico, Saskatchewan, South Dakota, Utah, and Wisconsin. CWD has been documented in captive cervids in Alberta, Colorado, Kansas, Minnesota, Montana, Nebraska, Oklahoma, Ontario, South Dakota, Saskatchewan, Wisconsin, and Wyoming.

The detection of CWD in Wisconsin was the first report of CWD in wild cervids east of the Mississippi River. Between 1999 and 2001 the Wisconsin Department of Natural Resources (DNR) had tested 1100 white-tailed deer for the presence of CWD. In 2001, 3 males, 2.5-3.5 years of age and shot within 3 miles of each other, were found to have evidence of CWD on immunohistochemical analysis of brainstem samples. After subsequent sampling of 476 deer within an 11 mile radius of the 3 index cases revealed 15 additional CWD-affected deer, Wisconsin initiated a CWD management program with the goal of controlling spread of the disease and, if possible, eliminating it.[1]

Wisconsin's CWD management to date has included intensive statewide surveillance, significant deer population reduction in the known affected area, the passage of legislation to ban deer feeding and baiting for hunting, and the implementation of an import moratorium and CWD monitoring program for farmed cervids. Since the beginning of 2002, lymph nodes from approximately 40,000 wild white-tailed deer harvested by hunters across the state have been screened by immunohistochemistry for evidence of CWD. Over 200 CWD affected deer have been detected, all within 35 miles of the index cases in south-western Wisconsin.[2] Using liberal hunting seasons and targeted harvesting by agency staff, the deer population in the affected area has been reduced by 25%. Mandatory testing of all farmed cervids transferred live or slaughtered has resulted in the detection of 3 CWD affected farms (1 elk farm, 2 white-tailed deer farms).

The Wisconsin CWD outbreak is part of a significant expansion of the known range of this disease, and evokes particular concern as very high deer densities in southern Wisconsin may facilitate rapid transmission of the agent and may have significant negative impacts on this important wildlife resource.

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Bovine Tuberculosis in Wildlife Species: The Impact on Livestock Agriculture of Finding a 100-Year-Old Livestock Disease in Free-Ranging Animals

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The state of Michigan eliminated its last known bovine tuberculosis infected cattle herd in 1975, and received Bovine Tuberculosis Accredited Free state status in 1979. In 1994, a hunter in Northeastern Lower Michigan harvested a white-tailed deer that was infected with bovine tuberculosis. The only other time that bovine tuberculosis was found in a free-ranging white-tailed deer in Michigan was in 1975, also in Northeastern Lower Michigan. An intensive surveillance program in livestock species and free-ranging wildlife discovered bovine tuberculosis to be endemic in the wild white-tailed deer population of a small area of northeastern lower Michigan, and uncovered a bovine tuberculosis infected cattle herd in the same area in 1998. As of June 2003, thirty cattle herds and 66 individual cattle have been diagnosed to be infected with bovine tuberculosis in Northeastern Michigan.

Controlling and eradicating bovine tuberculosis in a livestock species with an infected free-ranging wildlife population has significant quantifiable, and more difficult to measure, costs and effects. The State of Michigan has undertaken an aggressive program to eradicate this disease from livestock species, and eliminate its presence from wildlife. Since 1998, the Michigan Department of Agriculture (MDA) has utilized resources amounting to over $24 million on activities related to confining the disease to a small area of the state, eliminating any infected livestock and preventing the spread of disease from infected animals, and ensuring trading partners that Michigan animals and products are safe. These activities include conducting over one million bovine tuberculosis tests on 12,000 Michigan farms. This proactive testing, utilizing limitations in currently available diagnostic tests, required the destruction and indemnification of thousands of animals. In addition, over 350 private accredited veterinary practitioners were needed, along with over 70 regulatory personnel, to complete statewide testing, and eradication activities in the infected zone. Michigan State University estimated in 2000 that costs associated with lost milk production, discounts on cattle sold, and inconvenience to producers, would equal $23 million to $26 million annually. A major cost may also be the loss of opportunity for development or expansion of markets that these resources may have supported.

Other costs have occurred, and continue to exist, that are less able to be quantified. Many traditional markets for breeding and feeding livestock were affected or eliminated through restrictive testing or quarantine requirements for movement to other states. The tremendous efforts of private veterinary practitioners to assist with completion of the testing imposed a heavy strain on the veterinary infrastructure in Michigan, and also required an almost complete redirection of activities within the MDA and USDA regulatory veterinary infrastructure to focus on bovine tuberculosis eradication. Producers in the affected area must undergo rigorous testing and reporting prior to receiving permission to move animals, and there has been a restructuring of the industry in the area to change from feeder type animal production, to finishing operations less suited for the region. The total number of cattle farms in the area has seen a reduction in number from approximately 1500 to 1100 farms.

Some positive relationships and activities have developed from this occurrence. The State and Federal agencies in Michigan which oversee livestock and wildlife species have developed a close working relationship, and an understanding of the difficulties and opportunities which exist in each others areas. Diagnostic testing, reporting, and relationships between MDA and private veterinary practitioners has been enhanced. Developing and instituting a comprehensive eradication program has left the state more prepared for potential disease incursions, accidental or intentional. This occurrence has also equipped the State and industry to deal with livestock/wildlife interactions, and future issues that will ultimately develop between these groups of animals.

Bovine Tuberculosis (TB) in Michigan Wildlife

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Since 1995, a total of almost 105,935 deer statewide have been tested and 449 have tested positive. Apparent prevalence in the core area of the outbreak was 2.8% in 2002. In the remainder of the five county area of northeast Michigan where TB is most prevalent, apparent prevalence was 0.5%. Prevalence in both areas remains essentially unchanged since 1998, but is about half the 1997 rate. Prevalence continues to remain highest in older bucks. Of the 449 positive deer found since 1994, 67% have come from only 8 townships, suggesting foci of relatively higher prevalence surrounded by broad areas of much lower prevalence. To date, 1,520 non-cervids of 15 species have been cultured for the disease; 41 have been positive. Seventeen of those have been coyotes. Gross lesions have been quite rare in non-cervids, and none of the positives has shown extensive pathology. Since 1996, 1,187 elk have been tested for TB and 2 positives found. DNA analyses of isolates from infected animals of all species continue to implicate a single strain of *M. bovis*. Strategies for eradication of TB from Michigan wildlife focus on: 1) reducing deer population densities and 2) reducing man-made aggregations of deer by restriction or elimination of baiting and recreational feeding. These strategies have been implemented through provision of extra rifle seasons and unlimited antlerless permits in the former case, and by banning or restriction of deer baiting and feeding in the latter. In the five county area most affected by TB, deer numbers have declined by approximately 35% since 1995, but persistent focal areas of high density, particularly on private land, remain problematic. Compliance with baiting and feeding restrictions has been uneven, and enforcement continues to pose a challenge, though the overall scope of baiting and feeding, have declined substantially since 1997.

Anthrax at the Interface

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In October 2001, several letters containing *Bacillus anthracis* spores were sent through the U.S. Postal Service to recipients in government and private-sector buildings. Consequently, 23 human inhalational or cutaneous anthrax infections occurred. Five of the 11 inhalational anthrax infections were fatal. As a result of this intentional release of *B. anthracis*, anthrax has become a household word in the U.S. and elsewhere. But long before the events of 2001 anthrax has been recognized as a naturally occurring disease in both livestock and wildlife. The interface of livestock and wildlife continues to contribute to the livestock cases of anthrax that occur in the U.S. and elsewhere. While anthrax cases of livestock may be diagnosed, it is often the case that wildlife anthrax may go unnoticed. In this presentation, we will look at examples of the livestock-wildlife anthrax interface in the U.S., Canada and Africa.

Consequences of Mountain Sheep/Domestic Livestock Interactions

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The mountain sheep of North America include Dall's sheep (*Ovis dalli*) and bighorn sheep (*Ovis canadensis*). Dall's sheep inhabit remote areas of northern Canada and Alaska and are infrequently exposed to domestic livestock. Recent population genetic analysis identifies two major subgroups of bighorn sheep, Rocky Mountain bighorn sheep and desert bighorn sheep (Ramey 2000), and these designations will be used in this report.

Multiple-use land management agencies have long provided grazing allotments for domestic livestock within or near bighorn sheep habitat. In addition, feral, abandoned or lost domestic cattle and sheep may be sympatric with bighorn sheep in some areas of the Rocky Mountain and southwestern states.

The impacts of domestic cattle (*Bos taurus*) grazing within bighorn sheep habitat have not been well described. Generally, bighorn sheep avoid areas where cattle are grazed and may not return to these areas for long periods after cattle are removed (King and Workman 1984). The potential for cross species transmission of diseases between cattle and wild ungulates is also unclear and may vary with local environmental conditions. Singer et al (1997) studied cattle, bighorn sheep and mule deer (*Odocoileus hemionus*) in an area where the three species were known to utilize common areas. Only cattle were seropositive to bluetongue virus; deer and bighorn sheep were seropositive to *Babesia sp*.; and *Psoroptes* mites were found only on bighorn sheep. They concluded that cattle did not share similar patterns of exposure to the three pathogens with the wild ungulates and did not constitute a health risk for bighorn sheep in that area. There is insufficient evidence to exclude cattle grazing in or near bighorn sheep habitat in California based on disease considerations, although exclusion may occur for habitat preservation, especially in designated wilderness areas.

Bighorn sheep are closely related to domestic sheep (*Ovis aries*) and share many diseases in common with them, including pneumonias of viral and bacterial origin, contagious ecthyma (CE, soremouth), psoroptic scabies, bluetongue virus infection and others. Domestic sheep, bred and maintained over millennia at high density in close confinement and often under relatively unsanitary conditions, have experienced evolutionary selection pressure for resistance to infectious diseases (Jessup and Boyce 1993). Historically, the high alpine and arid desert environments inhabited by bighorn sheep provided other selection pressures including severe drought, harsh winters, poor forage, and predation. Combined with low herd densities and distribution over large areas, the relative importance of disease as a natural population limiting factor for bighorn was much reduced.

Introduction of domestic sheep grazing into large areas of the Rocky Mountains and the southwestern states in the late 1800's and early 1900's coincided with widespread all age die-offs of bighorn sheep attributed to psoroptic scabies and respiratory disease. There is abundant circumstantial evidence from field outbreaks in free-ranging bighorn indicating that diseases introduced by domestic sheep played an important role in the reductions in bighorn sheep populations throughout their range (Martin et al 1996). Experimental data supports this view.

Controlled contact exposures with domestic sheep (Onderka and Wishart 1988, Foreyt 1989) and inoculations of bighorn sheep with *Pasteurella* isolates or fluids from the respiratory tract of healthy domestic sheep (Foreyt 1990, Foreyt et al 1994) almost invariably have resulted in respiratory disease and death in bighorn sheep. Martin et al (1996) in a review of the literature regarding the compatibility between bighorn and domestic sheep state "almost all wildlife professionals, wildlife veterinarians and researchers have concluded that bighorn sheep and domestic sheep should not occupy the same ranges or be managed in proximity to each other because of the potential adverse effect from disease on bighorn sheep".

Respiratory disease can have devastating and long lasting effects on bighorn sheep populations by direct mortality and by low lamb survival for years following an outbreak. Because pneumonia in bighorn sheep can be of viral, bacterial or parasitic origin and is thought to be influenced by various environmental stressors, it is generally referred to as the bighorn sheep respiratory disease complex. This report will discuss:

1. case studies of respiratory disease in bighorn sheep as seen during apparent spontaneous outbreaks and as a consequence of mountain sheep/domestic sheep interactions.
2. efforts made by wildlife and land management agencies to manage these interactions to protect bighorn sheep.
3. issues related to other livestock species.

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Leptospirosis: A Zoonosis Before Zoonoses Were Cool

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Leptospirosis is a true zoonosis that occurs throughout the world. The epidemiology of the disease is complex as virtually all mammals are susceptible to infection with the approximately 250 pathogenic serovars of the genus *Leptospira*. In particular regions, different leptospiral serovars are prevalent and are associated with one or more maintenance host(s) that serve as reservoirs of infection. Maintenance hosts are often wildlife species and, sometimes, domestic animals and livestock. Contact with an infected maintenance host or areas contaminated with urine from maintenance hosts can cause infection in other species known as incidental hosts. Maintenance hosts are relatively refractory to development of acute disease but remain infected for long periods of time, shedding large numbers of bacteria in urine. Incidental hosts are more susceptible to developing acute disease but they clear the infection and represent little risk of transmission of the infection to other animals or man. The factors which enhance the emergence of leptospirosis in domestic livestock, wildlife, and humans are similar to those which govern other infectious diseases, e.g. climate, ecological disruptions, contact between species not previously in contact, global travel and recreation, animal trade, etc.

Epidemiologic Characteristics of *Brucella abortus* in Wild Ungulates in the United States and Current Status of Vaccines for Prevention of Brucellosis in Livestock and Wildlife

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Although the eradication of brucellosis from cattle in the United States is nearly completed, the prevalence of *Brucella abortus* in bison and elk in the Greater Yellowstone Area are considered reservoirs for reinfecting domestic livestock. Some of the epidemiologic characteristics of brucellosis infections in which bison and elk differ will be discussed. Protection and lasting immunity against brucellosis is achieved with vaccines containing live bacteria which stimulate a strong cell-mediated immune response. An ideal vaccine against brucellosis would persist long enough to induce good immunity without persisting into adulthood, would not cause clinical illness, and would not induce serologic responses which interfere with detection of animals infected with virulent field strains of *B. abortus*. Typically, vaccines against brucellosis are more efficacious in preventing abortions than preventing infection. The recent full licensure of *Brucella abortus* strain RB51 as a vaccine for cattle marks the first new brucellosis vaccine since the approval of strain 19 in the 1940's. Research is being conducted to characterize colonization, immunologic responses, and efficacy of RB51 and S19 in bison and elk. Immunologic differences in responses of bison, elk, and cattle to brucellosis vaccines will be discussed. Experiments evaluating a remote delivery system will be discussed in which the effect of ballistic delivery on immunologic responses and efficacy of brucellosis vaccines in bison and elk were evaluated.

Feral Swine Brucellosis in the United States of America

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Feral swine, which have been deemed an invasive species by executive order, are present in at least 25 US states with the largest concentration being located in the Southeast. Exact population estimates are difficult to ascertain; however, the number and range of feral swine is dramatically increasing due to natural population dynamics and an increasing popularity as a game species in many parts of the US. Existing populations include Eurasian boars, North American feral swine, collared peccaries, and isolated pockets of recently released domestic swine. While brucellosis has been nearly eradicated in the US domestic swine population, brucellosis has been reported in these wild *suid* populations in at least 14 states based primarily on serologic surveillance and is endemic in many of these populations. Limited culture work has shown that both *Brucella suis* biovars 1 and 3 exist in these swine. These infected populations continue to be a source of zoonoses for hunters, capture station workers, and feral swine processing plant workers. These swine also have the potential to infect domestic livestock, and feral swine to domestic swine and feral swine to cattle transmissions of brucellosis have been documented. Current research efforts have been targeted toward controlling the feral swine population dynamics and developing an efficacious *Brucella* vaccine. An overview of feral swine brucellosis and existing research efforts will be presented.

Foot and Mouth Disease: The Interaction of Livestock and Wildlife

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The 2001 foot and mouth outbreak in Great Britain lasted for 9 months and resulted in the destruction of over 3 million sheep, 600,000 cattle, and 138,000 pigs from over 9000 premises. In all, approximately 10 % of the livestock in Great Britain were destroyed. During the early days of the outbreak, movement of sheep caused dissemination of the disease throughout Great Britain and the establishment of small outbreaks in France and the Netherlands. In Great Britain, the possible establishment of foot and mouth disease in wildlife was a concern because this could have prolonged the epidemic and possibly allowed the disease to become endemic in some wildlife species. Testing of a few deer from affected areas failed to identify infected animals, yet farmers and veterinarians in the field expressed concern that wild deer were showing clinical signs of foot and mouth disease.

It is unknown how an outbreak of foot and mouth disease would spread in the United States and what role wildlife might play in spreading the disease. A wide variety of wild species have shown that they are susceptible to experimental infections with the virus. White-tailed deer can be infected with foot and mouth disease and maintain the virus in the oropharynx for up to 11 weeks. Other species of deer in the United States probably would be equally affected. The American buffalo, *Bison bison*, can also be infected with the virus and shed the virus. Hunter killed wildlife have the potential of maintaining the virus in lymphoid tissues for a considerable length of time and consequently, could infect swine if fed infected waste meat. The potential of domestic fowl and geese acting as a transport host for the virus is a concern. Some wild birds, sea gulls (*Larus conus)*, starlings (*Sturnus vulgaris*), and house sparrows (*Passer domesticus*) may become infected with the FMD and shed virus in feces onto their plumage. This may result in some birds spreading the virus over long distance. Since many wild animals have the potential of becoming infected with the virus, both domestic animals and wildlife should be evaluated as to the risks they may play in the spread of the disease to adjacent premises. Consequently, plans should be in place to deal with wildlife issues on infected premises before the depopulation of the domestic animals takes place. Such plans would assist in the prevention of further spread of the virus to other premises from fleeing wildlife species.

Emerging Viral Diseases in Wild and Farmed Cervids

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Several viral diseases are newly recognized in farmed and free-ranging cervids of North America. The emergence of viral diseases is dependent on opportunities for transmission, as well as viral and host factors that influence disease. In general, transmission of viruses is enhanced by contact between infected and susceptible animals, and through contact with contaminated premises or fomites. Increased animal density, herding behavior and a supply of susceptible animals contributes to continued virus transmission within a population. Opportunities for transmission of viruses from domestic to wild species may arise under both range and captive conditions. Viruses, which do not cause disease in domestic host species, may cause more severe disease following introduction into new species. Host factors such as age, acquired immunity, nutritional status, stress and concurrent infections with other agents also influence severity and outcome of disease. In captive cervids, viral transmission is enhanced by increased density of animals, increased contact with contaminated objects and by iatrogenic means. A balance is struck by improved access to feed, better quality forage, shelter from extreme weather, reduction of predation and isolation from domestic animals provided under intensive management. The management of free-ranging cervids shares similarities with that of range beef cattle in the western U.S. Interventions such supplemental winter-feeding, vaccination, culling through hunting and transplantation into new habitats may impact viral diseases in these populations. Increased interest in cervids is anticipated to result in more information about the impact of viral diseases in both free-ranging and farmed animals.

Malignant Catarrhal Fever in Wildlife and Domestic Species

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Malignant catarrhal fever is one of most neglected yet intriguing viral diseases of wild and domestic species. Several factors contributed to this: absence of cell-free ovine herpesvirus-2 (OvHV-2) and the difficulty of reliably reproducing MCF, limited number of diagnostic assays, variable clinical expression among species, relative insignificance of MCF in the cattle industry, the perception that MCF research equals career suicide, and the peculiar nature of mucosal and vascular lesions. Our understanding of the epidemiology, biology and economic impact of MCF increased greatly over the past 10 years.

The MCF family of viruses. In addition to "classic" MCF viruses (OvHV-2 and AlHV-1), many ruminant species are infected with their own well-adapted MCFV strains. Most are not known to cause disease. Infected species include domestic and exotic species, such as the domestic goat, muskox, oryx, and ibex. Positive MCF antibody assay results in less-studied species such as these must be interpreted cautiously until we have a better understanding of their potential impact in other species. The domestic goat was recently recognized as an MCF carrier. Goats endemically infected with caprine herpesvirus-2 (CpHV-2) have caused disease in deer.

MCF is economically important in the bison industry and in zoological collections: MCF is rarely of economic import in cattle, but in the commercial bison industry is the leading cause of acute fatalities in adult animals. Some recent outbreaks have been large. Two illustrations suffice: 27.6% mortality (45/163) over 220 days in an outbreak in SK where bison from multiple sources were exposed to sheep for <24 hours, and death of >800 bison over a 3-month period in feedlot bison in ID exposed to sheep for ~15 days.

MCF is not transmitted from bison to bison. Studies over the past 2 years generated convincing evidence that bison with clinical MCF do not serve as a viral source for clinical MCF in other animals in the herd. These studies include investigations of natural outbreaks, forced co-habitation, and laboratory studies to detect presence or absence of virus in secretions. Bison with MCF or with detectable MCF virus DNA in blood or secretions do not appear to represent a threat to bison herd mates and do not need to be culled.

The spectrum of clinical disease and lesions is not stereotypical. MCF was much beloved by diagnosticians because, like scrapie of old, a "definitive" diagnosis was the prerogative of morphologic pathologists, and not the lowly microbiologist. We increasingly understand that MCF occurs in less stereotyped subacute, chronic and recrudescent forms, in addition to well defined peracute and acute forms. The use of clinical signs, gross lesions and histopathology for diagnosis must be supplemented by PCR and serology.

Nasal secretions are the predominant vehicle by which MCF virus is shed and **the primary source of infection is the 6 - 9 month old sheep**. Molecular and biochemical studies demonstrate high levels of intact virus in ovine nasal secretions during intense, transient shedding episodes that last ~24 hr. Consistent transmission of MCF virus from sheep to sheep by experimental aerosolization of nasal secretions has been accomplished. Sheep aged 6 – 9 months are the most dangerous to susceptible species. This coincides with when sheep are

brought into feedlots for fattening, which has implications for feedlots holding both sheep and susceptible ruminant species.

The incubation period is more clearly defined. Several opportunistic studies of large scale outbreaks establish that the incubation period following natural exposure is ~50 days, with peak mortality at 60 – 70 days and losses continuing for up to 7 months.

MCF can occur in the total absence of sheep, apparently by reactivation of latent infections. Sporadic cases of MCF occur in herds in extremely remote areas, where no sheep exist within hundreds of miles. The only explanation at this time is recrudescence of long-held latent infections.

MCFV can be visualized in tissue sections by IHC and in-situ PCR. A major constraint on understanding the pathogenesis of MCF is paucity of detectable viral particles, antigen or DNA in tissue – ultrastructural studies repeatedly failed to visualize viral particles in even the most florid vascular and epithelial lesions. IHC methods for high-shedding sheep and in-situ PCR methods for affected ruminant species have been developed and will be used in pending experimental pathogenicity studies.

Pseudorabies in Feral Swine

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The decade-long eradication of pseudorabies has cleared infection from our domestic swine but is threatened by the growing reservoir of infected wild swine that will continue to present a risk of reinfection. The virus from wild pigs is very attenuated compared with isolates from domestic pigs and even less virulent than the vaccines used to immunize. Pseudorabies virus can infect most mammals and birds, frequently with lethal consequences. Transmission among wildlife is uniquely suited to survival of the virus in the diverse ecosystem and represents a symbiotic relationship between pathogen and its main porcine host. Wild swine tolerate infection to protect the species from predators and competitors. Mechanisms of transmission include expected oral infection but wild swine virus also moves silently as a venereal infection. Significant involvement of non-porcine wildlife species is still an open question. Virus can move from pigs to several wildlife species and recombination occurs after dual infection. Infection of domestic pigs can occur from infected feral pigs, but steps can be taken to reduce the risk of transmission. Consequences of wildlife infection must be considered as a component of effective biosecurity countermeasures.

Epizootic Hemorrhagic Disease in Wild and Domestic Ruminants

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Epizootic hemorrhagic disease (EHD) is a viral disease of wild and domestic ruminants caused by either epizootic hemorrhagic disease virus (EHDV) serotype 1 or serotype 2. These viruses are orbiviruses closely related to the bluetongue viruses (BTV), the cause of bluetongue (BT), which also cause disease in wild and domestic ruminants. EHDV serotype 2 is the virus that most commonly infects wild ruminants.

In the United States, both the EHDV and BTV are vectored by culicoides midges, thus associated disease is seasonal, typically occurring from early summer into the fall. *Culicoides sonorensis* is the only proven vector of EHDV in the United States although other *Culicoides* species are probably involved in viral transmission and further research is needed in this area.

Virus isolations and serological evidence indicates that EHDV occurs throughout much of the lower 48 states. However, clinical disease due to EHDV infection has a more limited range occurring along the east and Gulf coast areas, in a band across the Midwest and along the west coast. The southeastern United States is an endemic region for EHDV with epizootics occurring in 3 or 8 year cycles. In non-endemic areas (such as the Mid-Atlantic, Northeast, and Midwest) EHDV causes epizootics and severe mortality. In Texas, where numerous serotypes of EHDV and BTV are endemic, disease is rarely observed.

In wild ruminants, EHD is clinically indistinguishable from BT and disease caused by these viruses is typically termed hemorrhagic disease (HD). As the name implies, the disease is often characterized by a hemorrhagic diathesis. However, early in disease pulmonary edema may predominate while later in the course of infection ulceration in the gastrointestinal tract is more characteristic. Lesions could be confused with those of certain foreign animal diseases such as heartwater, foot and mouth disease, and rinderpest.

Although EHDV infect a wide variety of wild and domestic ruminants, there is inter-specific variability in susceptibility. White-tailed deer are extremely susceptible to EHDV infection and of our wild ruminants disease is most often documented in this species, often resulting in mortality. However, disease and mortality have also been documented in mule deer, antelope, elk, and big horn sheep. Domestic cattle rarely develop clinical disease when infected with EHDV and it is typically mild. Disease due to EHDV infection has never been reported in sheep, although they can be infected with viruses. Disease susceptibility does vary in white-tailed deer populations and innate disease resistance may vary at the subspecies level. However, the basis for intra- and inter-specific variability in susceptibility is not totally understood.

The mechanism of overwintering for these viruses has not been established. Recent work in sheep suggests long-term BTV infection of gamma-delta cells in the skin could serve as a source of virus for the *Culicoides* vector. However, *Culicoides* could not be infected with BTVwhen fed on cattle or sheep >21 days post infection. Thus, it seems unlikely that ruminants serve as a reservoir for these viruses.

Vesicular stomatitis: Manmade and *au naturel*

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Vesicular stomatitis (VS) is a New World disease affecting cattle, horses, and swine caused by viruses in the genus *Vesiculovirus,* family *Rhabdoviridae*. The two viruses most associated with VS in domestic livestock include vesicular stomatitis virus New Jersey serotype (VSV-NJ) and vesicular stomatitis virus Indiana serotype (VSV-I). The epidemiology of these viruses is not completely understood but existing knowledge suggests that transmission and maintenance mechanisms differ in wildlife and domestic animal systems. Clinical VS associated with naturally acquired infection has not been reported in any wildlife species. Infection, as determined by the presence of antibodies to VSV-NJ and VSV-I, has been detected in a diversity of wildlife species and high antibody prevalence has been reported from enzootic areas. There is good evidence that both of these viruses are transmitted and maintained in these natural systems through sand fly (Psycodidae) vectors. The specific wildlife amplifying hosts involved in this proposed cycle have not been identified and to date a viremia associated with infection with either virus has been experimentally demonstrated only with rodent species. In contrast, both clinical and subclinical infections with VSV-NJ and VSV-I can occur in cattle, horses, and swine. High rates of infection, as indicated by presence of antibodies, have been detected in livestock populations especially within herds. In addition, multiple transmission routes have been suggested including transmission via biological vectors, mechanical vectors, and contact. Virus has been isolated from many taxa of biting arthropods but to date blackflies (Simuliidae) are regarded as the most likely vectors associated with epizootics. Although a viremia associated with infection in domestic animals has not been reported, infection of blackflies has been documented via co-infection and through feeding on non-viremic hosts (pigs). Although work to date suggests that these viruses (especially VSV-NJ) can move efficiently in domestic animal populations through multiple transmission mechanisms, long-term maintenance of these viruses within these populations appears unlikely.

The interface between the wildlife maintenance systems for these viruses and their transmission into domestic animal populations is poorly understood especially in epizootic areas such as the western United States. The critical questions surrounding this interface relate to better defining the ecological requirements (amplifying host species, vectors, and habitat requirements) needed for virus maintenance and to better understanding host and environmental risk factors that allow these viruses to be efficiently introduced to and transmitted through domestic animal populations.

Need for Wildlife Specific Tools: Characterization of an Adenovirus that Causes Hemorrhagic Disease in Deer

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An adenovirus associated with a newly recognized hemorrhagic disease in deer in North America produces both systemic and localized lesions. Systemic lesions included pulmonary edema and less often hemorrhagic enteropathy. Vasculitis with endothelial intranuclear inclusion bodies was seen primarily in the lungs and alimentary tract and less frequently in the brain, kidney, spleen, pulmonary artery, and urinary bladder. Endothelial intranuclear inclusion bodies were seldom seen in deer with localized lesions. Adenovirus associated with systemic and localized vascular damage was demonstrated by transmission electron microscopy and immunohistochemistry.

Early attempts to propagate the virus in a variety of cell cultures derived from bovine, caprine, and ovine fetuses failed. The production of fetal white-tail deer lung and turbinate cell cultures allowed for propagation of the virus. Because of the lack of vigor of these derived cell cultures, cervine cell culture adapted virus was used to inoculate low passage fetal bovine, caprine and ovine cells, but without success. Development of cervine cell cultures for virus propagation was crucial to virus characterization, development of immunologic based tests for prevalence assessments, development of molecular based tests, and assessment of pathogenicity of this adenovirus.

Concepts of Infectious Disease Surveillance

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In the wake of the foot-and-mouth disease outbreaks in Taiwan and the U.K., and in response to escalating national and international concerns for bioterrorism, an urgent need has emerged for development of disease surveillance systems. Little information has been available, however, on the conceptual foundations surveillance necessary for early detection of foreign and exotic animal diseases. This presentation will outline some of the underlying concepts and parameters of disease surveillance, with emphasis on foreign animal diseases (FAD), as described elsewhere.[1]

Surveillance is defined as an active, ongoing, formal, and systematic process aimed at early detection of a specific disease or agent in a population, or early prediction of elevated risk for a population acquiring an infectious disease, with a pre-specified action that would follow disease detection. The objective of surveillance is to intentionally seek out disease early, before further transmission takes place. Performance attributes for surveillance systems include accuracy (sensitivity and specificity), precision (repeatability/reproducibility), rapidity and timeliness, efficiency (multiple utility), and value. Generally, there are two types of surveillance. One aims to detect an agent after it has already entered a population (*post facto* surveillance), and the other aims to detect the agent or elevated risk of disease before it enters the population (external, preemptive, or risk surveillance). Surveillance systems can be nested or embedded within other systems, whereby data or findings from one system would trigger activation of another more directed surveillance system. Surveillance also might be activated only at times of perceived high risk (hierarchical surveillance).

Three main parameters should be considered in the architecture and design of surveillance systems. These are the biology of the disease, including pathogenesis and epidemiology, the sampling scheme used to capture an infected or positive specimen from an infected animal within an infected herd, and the test or assay used to detect the agent in the sample obtained. Knowledge of the disease pathogenesis and epidemiology is necessary to define disease transition state probabilities, which indicate the likelihood of an infected animal, herd/flock, or population being in a latent, infectious, or clinical disease state. Surveillance sampling schemes should be designed to maximize the probability of early detection (risk sampling), given pragmatic constraints of sample size and frequency. Multiple sampling from the same animal or specimen can be expected to increase overall surveillance sensitivity, while increased sampling frequency will increase the probability of detecting the agent sooner (temporal sensitivity). Diagnostic assays or tests used to screen for a FAD agent should have a high sensitivity. Verification testing to rule-out false positive results should use assays with a high specificity. Sampling architecture developed for a disease with specific disease transition dynamics determines the probability that an agent in a sample or specimen from an infected animal in an infected herd will be presented to the assay for testing. The sensitivity and specificity of the screening and verification assays determine the probability of accurately identifying the agent once it has been presented to the assay. Thus, surveillance is a highly probability-driven

diagnostic process that incorporates disease transition dynamics, directed and strategic sampling, and diagnostic test accuracy and precision.

[1]Thurmond M. Conceptual foundations for infectious disease surveillance. *J Vet Diagn Invest* (in press)

West Nile Virus

Tracy McNamara

Abstract not available at time of printing.

Influenza

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Type A influenza viruses are common pathogens of humans, pigs, horses and poultry. Although the virus is endemic in a number of different species, all type A influenza viruses originate in wild birds, primarily ducks, gulls and shorebirds. Influenza viruses have eight gene segments, with six segments coding for conserved internal proteins and two coding for extremely diverse surface proteins. The hemagglutinin (HA) surface protein is divided into 15 antigenic subtypes and the neuraminidase (NA) protein has 9 antigenic subtypes. All 15 HA and 9 NA subtypes are found in wild birds, but only select subtypes have become endemic in mammalian species. Although many subtypes have been isolated from poultry, chickens and turkeys, only the H5 and H7 subtypes have been associated with the highly pathogenic phenotype. The reason for this subtype selectivity is not known. Influenza viruses are generally promiscuous and many replicate in birds and mammals. However, when these viruses transmit from the natural wild bird reservoir to aberrant species, most don't replicate or transmit well enough to cause either disease or establish a lasting infection in the new host species. On rare occasions the virus strain that crossed the species barrier will have a constellation of genes that allows sufficient replication and transmission in the new species. These viruses may cause disease, and are characterized by a high evolutionary rate as the virus becomes adapted to the new host species. Eventually, the virus will become so adapted to the new species that it will no longer replicate well in the original host species. Influenza because of its segmented nature can also reassort gene segments. This switching of viral genes allows an additional way for viruses to adapt, and if it involves the hemagglutinin gene can result in a "genetic shift" which can result in a new pandemic in humans. One common denominator for the species that influenza infects is that these animals congregate or are reared in large numbers, which likely aids in viral transmission.

Phylogenetic Analyses among Newcastle Disease Virus Isolates from Domestic and Non-Domestic Avian Species

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Newcastle disease virus (NDV) is classified as avian paramyxovirus-1 (APMV1), a member of the family *Parmyxovirdae.* This family is divided into the *Paramyxovirinae* and the *Pneumovirinae*. There are eight other serotypes among avian paramyxoviruses that infect primarily bird species. The International Committee on Taxonomy of Viruses placed NDV and the other APMV types within the Avulavirus genus. NDV is an economically important pathogen of poultry with a worldwide distribution. Outbreaks of virulent forms are List A agents that require reporting to the Office of International Epizootes (OIE). Isolates from several different bird species with origins worldwide were phylogentically analyzed utilizing genomic nucleotide and predicted amino acid sequences. Virulent NDV isolates recovered in the U.S. prior to 1970 only contained neurotropic viruses. All virulent velogenic NDV obtained after that were related to chicken/Australia/AV/32 as the earliest progenitor-type and related to viruses that included chicken/U.S./CA1083(Fontana)/72 and cormorant/U.S.(MN)/40068/92. This included viruses isolated from imported psittacines, poultry in Mexico during 1996 and the recent California isolate from the outbreak that began during the autumn of 2002. NDV isolates from pigeons, classified as pigeon paramyxovirus type 1 (PPMV-1), clustered separate from other isolates phylogenetically. However, there were also pigeon and dove isolates that segregated with other APMV-1 isolates. Therefore, PPMV-1 may be circulating among Columbidae members as a distinct lineage, but they may also harbor a variety of NDV strains. A recent European dove isolate with an aberrant fusion protein cleavage site was an outlying member phylogenetically between the two major groups of APMV-1 isolates.