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# The spread and potential control of disease across the domestic cattle-wildlife interface

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A constant threat to disease control in domestic animals and wildlife is the potential spread between species, particularly across the domestic-wildlife interface. Between domestic cattle and wild ruminants there are multiple diseases which can be transferred, causing a major area of potential risk for the control of disease. Some common examples include bovine tuberculosis, bovine viral diarrhea, malignant catarrhal fever, brucellosis, hemorrhagic disease, and Johne's disease.

Bovine tuberculosis is caused by the bacterium *Mycobacterium bovis* (*The Merck Veterinary Manual*, 2010-2013). Bovine tuberculosis most commonly affects cattle but can easily spread to many other mammal species, both domestic and wild. The easy spread is due to a variety of transmission methods, including inhalation of aerosols, ingestion, or entry through open wounds. It also has the ability to survive for many months in the environment. Clinical signs of bovine tuberculosis are consistent across species and can progress from slight emaciation, low grade fluctuating fever, and weakness to extreme emaciation and acute respiratory distress (*The Merck Veterinary Manual*, 2010-2013). The variable, and often mild clinical signs, make detection difficult. In domestic species the disease is often identified with periodic tuberculin skin tests. In wildlife it is usually detected through histopathology of collected bodily fluids. Bovine tuberculosis is controlled primarily through detection from routine testing, separation of infected individuals, and elimination of infected animals (OIE, 2012). However, its presence in wildlife makes it difficult to control successfully. Detection is not as accessible in wildlife. Therefore, practices that could increase transmission should be prevented. Supplemental feeding of white-tailed deer (*Odocoileus virginianus),* which causes

large populations to aggregate, has been shown to increase incidence of the disease (Davidson, 2006).

Bovine Viral Diarrhea (BVD) is caused by a *Pestivirus*. There are multiple strains of this virus, but they can be broadly categorized into cytopathic and noncytopathic based on their ability to destroy non-lymphatic cells upon infection. Cattle are the primary reservoir host but can transmit it to any even-toed undulate (order *Artiodactyla*). Transmission can occur vertically in utero, horizontally through infected fecal-oral and oral-nasal contact, or through insect vectors. The severity of infection varies greatly and can be divided into four basic categories: clinically acute, clinically severe, persistent, and mucosal. Clinically acute infections generally only last 1-2 days and are highly transmissible. Clinical signs include depression, fever, and diarrhea. Clinically severe cases have higher morbidity rates and generally last 3-7 days showing signs of high fever, dehydration, diarrhea and leukopenia. Persistent infections generally only affect young individuals, less than 2 years, and are only caused by noncytopathic strains. Persistently infected cattle are usually asymptomatic but have stunted growth and shortened life spans. Mucosal infections are the most severe and can be highly fatal. They occur when persistently infected cattle are also infected by a cytopathic strain. Clinical signs include fever, leukopenia, diarrhea, dehydration, lesions of the nares and mouth, and can lead to death.

Just as in bovine tuberculosis, the most important step in control is identification of infected animals followed by separation and treatment. (*The Merck Veterinary Manual*, 2010-2013). When identifying and working to prevent the spread of the disease it is important to consider all possible reservoirs. While BVD is most prevalent in cattle, studies have shown that both mule deer (*Odocoileus hemionus*) and white-tailed deer have the potential to contract and spread the disease (*Diseases and Parasites of White-Tailed Deer*, 1981). Deer infected with

disease present clinical signs similar to those of cattle ranging from weakness, dehydration, and diarrhea to abnormal gait, emaciation, and death in chronic cases. A study in North Dakota showed that sick and dead deer diagnosed with BVD were found in close association with pastures used by infected cattle (*Diseases and Parasites of White-Tailed Deer*, 1981). Such an association could be due to either cattle or deer acting as a reservoir and spreading it to the other species. Further investigations in North Dakota showed that each species, deer (*Odocoileus spp.*) and cattle, could transfer the disease to the other. Generally, when deer acted as the reservoir, only a mild form of the disease manifested in cattle. When cattle were the reservoir, the disease was much more serious in deer, often leading to death. Understanding the ability of the disease to be passed between these species is important in the control of its spread.

Malignant Catarrhal Fever (MCF), a third disease able to cross the domestic wildlife interface, is caused by two different herpes viruses, one for which wildebeest is the reservoir host and the other with sheep as the primary host. Neither of these hosts are affected by the disease, but both can transmit it to other species (Iowa State University, 2012). The non reservoir species most commonly affected in the United States are cattle and captive wild ruminants. The prevalence of MCF in both of these groups is low (Iowa State University, 2012; Li, et al., 1996). Both species present similar clinical signs, but the disease progresses much more rapidly in deer (Iowa State University, 2012). Deer positive for MCF usually die following a few days of debilitating clinical signs including depression, weakness, and diarrhea. In cattle, the clinical signs are similar but also incorporate discharge from the nose and eyes and open-mouthed breathing. It is possible that these additional symptoms are due to the more gradual progression of the disease which lasts up to 1-2 weeks before causing death.

A fourth disease, Brucellosis, caused by bacteria of the genus *Brucella*, is one of the most serious and costly to livestock. (NAHP, 2005) Brucellosis is transmissible between cattle and wild ruminants but the incidence is low because transmission requires direct contact with vaginal or birthing fluids of infected animals (*Diseases and Parasites of White-tailed Deer,* 1981). The effects of this disease are consistent across species with the most common clinical signs being abortion or birth of weak calves. In cattle this can result in serious losses of potential livestock and decreased milk production (*The Merck Manual,* 2010-2013). Currently in the US, Brucellosis is successfully managed through an eradication program involving routine testing of milk four times per year and routine blood testing of non-diary animals (NAHP, 2005). If an animal is identified as positive, the individual animal is slaughtered. Depopulation occurs when entire herds are affected. Because cattle are the primary reservoir hosts, successful control in this species has led to low incidence of disease in wildlife as well.

Hemorrhagic disease is another example of a disease that can cross the domestic- wildlife interface. It is a viral disease transmitted by a virus of the genus *Orbivirus*. White-tailed deer are most affected by the disease in the United States, although it can be passed to cattle (*Diseases and Parasites of White-tailed Deer,* 1981). Spread of this virus is not contagious and requires a biological vector, most commonly biting midges (*Culicoides spp.).* Because of the requirement of a vector, hemorrhagic disease often occurs in a seasonal pattern with the life cycle of the vector. The disease occurs at the highest incidence in late summer and early fall when weather is typically warmer (OIE, 2009). In cattle, the disease is often asymptomatic; however, in white-tailed deer, the disease can be very severe. Some cases in white-tailed deer result in rapid death, but more commonly, the disease will progress over several months with clinical signs such as depression, inactivity, and emaciation followed by a slow recovery (*The Merck Manual,* 2010-

2013). Currently there is no effective cure for hemorrhagic disease and most treatments involve supportive therapy, providing a calm environment, and providing soft palatable food (*The Merck Manual,* 2010-2013). Research is being conducted to develop vaccines as a possible control mechanism but currently a strong enough immune response to vaccination has only been shown with a modified live vaccine with a high rate of reaction (OIE, 2009). The most effective control method is the control of the biological vectors which transmit the disease (Davidson, 2006).

While many diseases, such as those discussed thus far, are transmissible between domestic cattle and wildlife, this paper will focus in more detail on Johne's (Yo-neez) disease (JD). This disease is caused by the bacterium, *Mycobacterium avium* subspecies *paratuberculosis* (MAP) (*The Merck Veterinary Manual,* 2010-2013). In the United States, JD has affected beef and dairy and has been detected in select populations of wild ruminants (Davidson et al., 2004). The possibility of JD transfer from wildlife to herds of domestic animals and what conditions promote JD require further investigation. The low prevalence in wildlife means limited economic effect in these populations, however, costs can be high in domestic cattle. In their 2008 study, O'Brien, et al. estimated that 68% of dairy herds in the United States had at least one positive case of MAP, with a cost per cow in a low prevalence herd around $40 and as high as $200 per cow in herd with high incidence of disease. O'Brien et. al (2013) estimated JD costs the US dairy industry $200 to $250 million annually. The beef industry has a prevalence of only 10% in herds nationwide (NAHMS 2007). Bhattara et al. (2013b) surveyed beef producers and veterinarians in Texas and estimated the average loss per infected cow was $275, and when prevalence of JD is 7%, the annual herd loss was $1920. The greatest loss came from lowered weaning weights (Bhattarai, et. al, 2013b). Bhattarai et al. (2013a) evaluated 4,842 beef herds currently enrolled in the USDA National Johne's Demonstration Herd Project and

found the average weaning weight of calves from cows with strongly positive MAP ELISA was 21.48 kilograms less than those from cows with negative ELISA results. Lowered weaning weight can affect both beef and dairy industries leading to serious economic losses.

Besides the serious economic losses that can be associated with JD, this disease poses a serious threat to the health of the animal. Often because of the difficulty of detection this results in years of poor health with no proper treatment, as well as years of an infected animal actively spreading the disease. To combat the threat to animal welfare and economic losses an effective program for detection and control is necessary. Development of such a program will require more in depth understanding of the disease and the economic feasibility of the different control options (Wells and Wagner, 2000).

Animals less than six months of age are at greatest risk for infection from MAP. (*The Merck Veterinary Manual*, 2010-2013). The most common route of transmission is fecal-oral. Usually young animals contract the disease after contacting the infected feces of adult animals; however, it is possible to contract MAP transplacentally (Wells and Wagner, 2000). Horizontal transmission can also occur when young animals are grouped together. Sweeney (1996) found that an individual can begin shedding infectious levels of the bacterium in the feces within 24 hours of infection, making horizontal transmission a serious risk (Sweeney, 1996).

In cattle, the disease usually remains dormant until the animal reaches about two years of age (Manning, 2001). At this point, it develops into a chronic wasting disease that results in diarrhea and weight loss as well as decreased milk production and fertility. In wild ruminants, such as deer (*Odocoileus spp.*) and elk (*Cervus canadensis*), young animals less than two years of age are still the most susceptible. However, in contrast to cattle, once contracted, the disease often leads to clinical signs much more quickly, at 8-10 months of age. Additionally, in

deer and elk, diarrhea is not always present; however, they do exhibit muscle wasting and may develop lesions in the gastrointestinal tract and lymph nodes (http://www.usask.ca/wcvm/herdmed/specialstock/deer/Johne_info.html, March 5, 2014).

There is no apparent trend in the geographical distribution of Johne's Disease in American cattle (NAHMS, 2007). The infection happens throughout the country and is often maintained at low levels. In deer and elk, the disease is generally absent from populations for extended periods, then will occur in sporadic outbreaks in various locations affecting many animals at once (http://www.usask.ca/wcvm/herdmed/specialstock/deer/Johne_info.html, March 5, 2014). In wildlife, distribution seems random with no trend to the location of the disease. Collins and Manning (2010) detected MAP in elk (*C. elaphus nannodes*) in Point Reyes National Seashore in California, but it was not detected in similar populations of elk tested in Wyoming, Montana, and Arkansas. MAP was also detected in Florida key deer (*Odocoileus virginianus clavium*) in 1996 and has remained present in this population. The disease was also discovered in two male white-tailed deer in Virginia (Collins and Manning, 2010). While lack of testing in wildlife may mean that the prevalence and distribution is greater than what has been reported, the information available suggests that MAP is much less prevalent in these species. However, even if the prevalence is low in wildlife, infected individuals do have the potential to spread the disease to other species.

Stevenson et al. (2009) performed genomic testing of MAP in both positive domestic cattle and nineteen different wildlife species and found animals living in the same location shared the same strain of MAP. This correlation indicates that transmission between wildlife and domestic cattle is possible. Currently, there are limited studies on the prevention of MAP in wildlife. However, more extensive research has been done on bovine tuberculosis (TB), a closely

related disease. In white-tailed deer in Michigan, Miller et al. (2003) found numerous risk factors associated with supplemental feeding and the spread of bovine TB. Any method of supplemental feeding that increased population density or used foods such as fruits, which are often shared by more than one animal, were correlated to increased incidence of bovine TB. Due to the similar nature of TB and MAP it is likely that such patterns characterize JD as well. Therefore, it is advisable to prohibit those practices as a method of control of JD.

Shaughnessy et al. (2013) focused on the potential of wild rabbits to spread MAP through their feces and found a significant correlation between the number of infected wild rabbits on a farm and the prevalence of MAP in domestic livestock. This indicates that rabbits spread MAP through their feces leading to infection of domestic cattle and other wildlife which contact it providing evidence that wildlife do act as a reservoir for JD. This trait is important to consider when planning effective control of the disease.

A successful control program must consider the effectiveness and availability of diagnostic tests, risk factors of the disease, producers' willingness to participate, and the potential for successful eradication. Currently, there are multiple diagnostic tests available for MAP. The most common is fecal culturing. Other options include ELISA of serum and milk samples, environmental cultures, and PCR analysis. While these other tests can provide useful information, their specificity and sensitivity is limited when compared with the fecal culture and are therefore not relied on as primary detection methods (Lavers, et al., 2014, O'Brien, 2013). Fecal culturing is most commonly used due to its high sensitivity, or true positive rate, and specificity, or true negative rate, especially when performed on pooled fecal samples (Lavers, et al., 2014). However, this test can be somewhat expensive and other options have been explored. In the dairy industry there is interest in milk ELISA testing. For herds which participate in Dairy

Herd Improvement Association or similar testing and record keeping programs, testing the milk for MAP antibodies would be convenient. Lavers (2014) found the effectiveness of milk ELISA tests had low herd sensitivity levels of 58-63% and that within and overall prevalence in a particular area greatly affected the results. False positives increased as herd prevalence increased (Lavers, et al., 2014). O'Brien et al. (2013) examined the possibility of using fecal PCR to supplement milk ELISA testing using farmed red deer. This test is appealing due to its low cost and high speed in comparison with fecal culturing. However, increased labor required for the test is not practical on a herd wide basis but would be useful in confirming positive milk ELISAs.

It is also important to consider education of producers and risk factors for developing JD before establishing a nationwide control program. The 1996 National Animal Health Monitoring System (NAHMS) survey on JD in the dairy industry found 45.7% of producers were unfamiliar with the disease. While this number decreased in the 2007 survey, many management practices have not changed (Wells and Wagner, 2008). Clearly further education about both the disease and best management practices is needed. Additionally, it is important to identify risk factors for the spread of the disease. Wells and Wagner (2000) identified the top risk factors in US cattle herds including introduction of new individuals to the herd with unknown MAP status, group housing of periparturient cows or calves prior to weaning, and the size of the herd, with larger herds being more likely to harbor infections. None of the greatest risk factors are unique to JD and all play a role in the spread of other infectious diseases. In order to better control JD more specific risk factors must be identified. For example, higher prevalence of Johne's Disease in calves born in the summer was recently identified as a unique risk factor (Zare, Shook, et. al, 2013). To gain a more thorough understanding of what promotes JD, research should also focus on infected wildlife populations. Incidence of JD in wild ruminants is sporadic. This could be

due to lack of testing or detection. However, if the sporadic distribution is accurate, identifying commonalities in the areas where JD is prevalent in wildlife could provide information on risk factors for the spread of the disease.

Producers' willingness to participate and potential of program success are closely related. Programs perceived as beneficial should be cost effective and have a high potential to succeed. The cost to both infected dairy herds and beef herds can be extremely high, with much of the cost coming from premature culling, loss of milk production, and decreased weaning weight (Bhattarai, Fosgate, et. al, 2013). Therefore, the potential benefit of a control program may be highest in the high prevalence herds. Cho et al. (2013) evaluated multiple control models using computer simulations and statistics and determined that the most effective protocol would be biannual pooled fecal culturing with culling of infected individuals and proper hygiene management. Separation of cow and calf, maintaining a clean nursery environment, and prevention of contact between calves and adult cow manure are important hygiene management practices. These practices are only feasible for the dairy industry, thus requiring other options to be considered for prevention in beef. Such practices, which are applicable to the dairy industry as well, should limit the accumulation of manure and the maintenance of conditions which can harbor MAP (www.johnes.org/beef/control.html, April 18, 2012). Any standing bodies of water, particularly those which drain from potentially contaminated fields, should be fenced off. These ponds can harbor MAP for over a year. Hay for feeding should be distributed in multiple areas to prevent congregating and accumulation of manure. Overcrowding in muddy lots, especially around calving, should also be avoided. If cows are contained during calving, the dam and calf should be removed to a lower risk area as soon as possible. The complete removal of the causative agent, MAP, may not be possible due to replacement animals continually added to the

herd, but there are other options to help prevent reintroduction of the disease (Cho, Tauer, et al., 2013). A strategy involving routine testing and culling of infected individuals has been extremely successful in Australia where the control program is nationwide and also involves a certification program. National level certification of JD free herds will help eliminate reintroduction of the bacteria because producers can make the choice to only bring in confirmed JD negative individuals. Certification also has the potential to encourage non-participating producers to enroll in the program and provide confidence to international markets.

Currently a Voluntary Bovine Johne's Disease Control Program is available in the United States (Bhattarai, et al., 2013). Bhattarai, et al. (2013) evaluated the opinion of veterinarians and beef producers in Texas enrolled in the program. All acknowledged the potential loss of income due to JD, but only 25% of producers of low risk herds saw a significant benefit in the control program. Wells, et al., (2000) performed a similar study in Minnesota, which has the highest enrollment rate of both dairy and beef herds. They revealed that the program has been successful in lowering the prevalence and risk. However, low prevalence and smaller herds were less likely to see the value and participate in the program. While producers of these low prevalence herds may not see the direct benefits of the program, their participation is necessary for potential eradication (Wells, et al., 2000). Herds which are not regularly testing may be harboring subclinical MAP infections and may unknowingly assist in the introduction or reintroduction of the disease to other herds during the sale of animals. Establishing a nationwide certification program would help control this problem as producers would be less likely to buy cattle from herds that were not certified. This lost potential income, due to discouraged sales of cattle, could then encourage those producers with low prevalence, that are not currently participating, to enroll in the program.

While establishing a nationwide testing and certification program would be highly effective in controlling JD, the implementation of such a program may not be feasible due to the cost and limited value seen by all producers. Those producers involved in the sale of cattle would likely see the benefit of such a program, but for those with a closed herd or whose business does not depend on the sale of cattle may not be willing to pay for a certification which would not provide an immediate and recognizable benefit. For this reason, I think control should focus on establishing effective management practices for prevention. On both dairy and beef farms this includes maintaining a clean and dry calving environment, limiting accumulation of manure, and preventing access to contaminated water. Specifically on dairy farms, where JD is more prevalent, additional management practices include rapid separation of calves from adult cows and limiting contamination of calves with antigens from adults. This can occur through access with contaminated manure or feed. One of the most important practices for preventing this contamination is the order in which age groups are cared for. Calves and young cows should always be accessed before older cows in order to prevent exposure of MAP, and many other antigens, to their immature immune systems. Most of these practices are well known to producers, and the challenge is making sure they are implemented effectively on farms.

As mentioned throughout this paper, in addition to contamination within a farm, there is the risk of spread of JD between cattle and wildlife. Based on current research, prevalence of JD in wildlife is not consistently high throughout the United States. For this reason, I do not think it would be cost effective to establish a large scale control program in wildlife. The spread of JD and many other diseases within wildlife populations can be limited greatly by prohibiting practices encouraging congregation of large numbers, such as supplemental feeding. While this practice is valued by the hunting industry, I believe that the benefit of stopping such activities

would outweigh the advantage it provides to hunters if they are properly educated of the effects on the spread of disease. With prevalence of JD being low overall for wildlife, I do not believe that they pose a great risk to domestic cattle as reservoirs of the disease. Any prevention of spread between the species should be focused in areas where the disease is known to exist. In these areas extra precautions, such as upright fences of heights greater than 8 feet or forward slanting fences, to prevent access of wildlife to farms and preventing cattle from accessing water sources that could be accessed by wildlife should also be implemented. While this will result in a greater cost to the producer, the benefit to animal welfare and the prevention of loss through illness should justify the investment.

When working in any animal industry, whether domestic or wild, there are two major concerns. First, and most important, is the welfare of the animals involved, second is the success of the business. Important in maintaining both of these, is the monitoring and control of disease. This becomes increasingly difficult when dealing with diseases that can cross the interface between species. Studying the trends in these diseases and understanding their risk factors can lead to effective control and support the goals of the animal industry.

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