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Tuberculosis: A Reemerging Disease at the Interface of Domestic Animals and Wildlife

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Abstract

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Tuberculosis: A Reemerging Disease at the Interface of Domestic Animals and Wildlife

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Abstract Complex interactions involving humans, domestic animals, and wildlife create environments favorable to the emergence of new diseases. Today, reservoirs of *Mycobacterium bovis*, the causative agent of tuberculosis in animals and a serious zoonosis, exist in wildlife. The presence of these wildlife reservoirs is the direct result of spillover from domestic livestock in combination with anthropogenic factors such as translocation of wildlife, supplemental feeding of wildlife and wildlife populations reaching densities beyond normal habitat carrying capacities. As many countries attempt to eradicate *M. bovis* from domestic

livestock, efforts are impeded by spillback from wildlife reservoirs. It will not be possible to eradicate *M. bovis* from livestock until transmission between wildlife and domestic animals is halted. Such an endeavor will require a collaborative effort between agricultural, wildlife, environmental and political interests.

1 Introduction

The emergence of newly recognized diseases in wildlife is often the result of complex and sometimes unintended interactions between wildlife, domestic animals, and humans. Wild animals are susceptible to infection with many of the same disease agents that afflict domestic animals, and transmission between domestic animals and wildlife can occur in both directions. Transmission of *Mycobacterium bovis* from domestic animal populations to wildlife (spillover) and subsequent transmission from wildlife back to domestic animals (spillback) is a theme common in most parts of the world currently attempting eradication of *M. bovis* infection among animal populations. In most cases, both spillover and spillback have been facilitated by anthropogenic factors such as human and domestic animal encroachment on traditional wildlife habitat, translocation of animals, or supplemental feeding of wildlife.

Critical to control of tuberculosis is the understanding of maintenance hosts and spillover hosts. Among spillover hosts, disease does not persist without an external source of reinfection. This external source of infection may be any other population of susceptible hosts, wild or domestic. However, in most cases *M. bovis* was originally introduced by spillover from domestic cattle to a susceptible wild population. Spillover hosts may be dead-end hosts and play no role in disease transmission or may be amplifying hosts that can increase transmission to other wildlife hosts or back to livestock. Disease in spillover hosts will gradually disappear as disease is eliminated in the species acting as the source of infection. In contrast, among maintenance hosts, disease persists without any external source of reinfection. Maintenance hosts may be domestic or wild, but are critical in disease epidemiology and control because without intervention, disease will persist among a population of maintenance hosts (see the chapters by Childs et al. and Childs, this volume). The most efficient disease control efforts are generally aimed at maintenance hosts. There is general acceptance that among wildlife species the European badger (*Meles meles*) in the United Kingdom, the brushtail possum (*Trichosurus vulpecula*) in New Zealand, and the white-tailed deer (*Odocoileus virginianus*) in the United States represent true maintenance hosts.

In the early part of the twentieth century, there were large numbers of tuberculous cattle in industrialized nations in North America, Europe and Australia.

Often an association was made between the number of *M. bovis* -infected humans and the prevalence of tuberculosis in the local cattle population. Infected cattle were generally considered the source of human infection with *M. bovis*, transmission being through direct inhalation or ingestion of unpasteurized dairy products (Grange and Yates 1994; Wigle et al. 1972). Abattoir workers have been infected during the slaughter and processing of cattle (Robinson et al. 1988; Cousins and Dawson 1999). More recently, exposure to tuberculous elk (*Cervus elaphus*) resulted in human infection (Fanning and Edwards 1991). With mandatory pasteurization of milk, tuberculin skin testing of cattle, and slaughter of infected cattle, the incidence of human tuberculosis due to *M. bovis* has declined dramatically in developed countries. However, it is estimated that worldwide approximately 50 million cattle remain infected with *M. bovis*, with a cost to the agricultural community of US $3–4 billion per annum (Steele 1995). In underdeveloped countries, such as many of those in Africa, tuberculosis in cattle is still widespread, as is *M. bovis* infection in humans. Even in developed countries where bovine tuberculosis eradication efforts have been in place for decades, successful eradication of disease from livestock is hampered by several factors, not least of which is the presence of wildlife reservoirs of *M. bovis*. Generally, countries with a documented wildlife reservoir of *M. bovis* have not been successful in eradication of *M. bovis* infection from domestic livestock. Several factors are critical in the development of a wildlife reservoir of disease: disease prevalence, clinical course of the disease, and host ecology. The following three examples illustrate the complex interaction of wildlife, domestic animal, and human factors in the creation and maintenance of wildlife reservoirs of tuberculosis.

2
United Kingdom

2.1
History of *Mycobacterium bovis* Infection in European Badgers

In the 1970s, tuberculosis had been removed from large areas of Great Britain, and eradication was predicted. In 1981, the Wildlife and Countryside Act provided protection to badger populations and resulted in a large increase in the number of badgers. Over the past 10 years, Great Britain has experienced a rising incidence of tuberculosis in cattle, especially in the southwest of England, South Wales, and also in the Republic of Ireland. *Mycobacterium bovis* is endemic among badgers, and although most of the evidence is indirect, it is hypothesized that badgers are a source of infection for cattle and responsible

for the increase in tuberculosis among domestic cattle herds. *Mycobacterium bovis* was first isolated from badgers in Switzerland in 1957 (Bouvier 1963). It is postulated that these badgers were infected by contact with tuberculous roe deer (*Capreolus capreolus*). In 1971, the first tuberculous badger was identified in England (Muirhead et al. 1974) and in 1975 an infected badger was reported in Ireland (Noonan et al. 1975). It is believed that badgers in England became infected with *M. bovis* during the late nineteenth and early twentieth centuries when a large percentage of British cattle were infected with *M. bovis* and infection spilled over from cattle to badgers.

2.2 Badger Ecology

The badger's natural habitat is such that it lives on or near pastures used by cattle where it digs for earthworms and dung beetles. Badgers live in groups of up to 35 animals that defend a communal territory that may include several setts, described as complex, long-lasting networks of tunnels and channels (Tuyttens et al. 2000). Setts provide ideal conditions for the spread of respiratory diseases. Badger social groups may remain stable for years with a low rate of dispersal (Tuyttens et al 2000). Such stability decreases the likelihood of disease transmission between groups, an idea supported by the observation that in undisturbed badger populations disease prevalence is highly localized in clusters (Cheeseman et al. 1988). In extreme cases, badger density can be as high as 25.3 adults per square kilometer; however, there appears to be no correlation between badger density and the prevalence of *M. bovis* infection among badgers (Cheeseman et al. 1989; Rogers et al. 1998).

2.3 Pathology and Transmission

Lesions in tuberculous badgers may be found in the lungs and associated lymph nodes, pharyngeal lymph nodes, mesenteric lymph nodes and kidneys (Gallagher et al. 1976; Gavier-Widen et al. 2001). However, several characteristics distinguish tuberculous lesions in badgers from those typically seen in cattle and may have important implications in disease pathogenesis and transmission. While caseous necrosis, mineralization and peripheral fibrosis are often associated with tuberculous lesions in cattle they are the exception in badgers. Langhans type giant cells, commonly seen in bovine lesions, are rare in badgers, while acid fast bacilli are often numerous. Renal lesions are more common in badgers than in cattle. These lesions can be

extensive, involving several regions of the nephron, and acid fast bacilli can be numerous (Gallagher et al. 1976). Experimental studies demonstrate that badgers can transmit *M. bovis* to cattle (Little et al. 1982); however, the exact route of transmission is unknown. Infected badgers shed large numbers of *M. bovis* in saliva, urine, feces, and exudates from draining lesions (Gavier-Widen et al. 2001). It is suggested that cattle may become infected by inhalation of bacilli from grass contaminated with infected badger urine, feces, or exudates from superficial draining lesions (Hutchings and Harris 1997). Urine is believed to be of greatest risk due to the high numbers of *M. bovis* bacilli present. Badgers urinate either at localized areas used for urination and defecation known as latrines or on pastures where badger paths cross linear features such as hedgerows or ditches known as crossing points (Scantlebury et al. 2004). Both latrines and crossing points are generally accessible to cattle. Moreover, infected badgers can live 3–4 years following the first documented episode of shedding of *M. bovis* (Little et al. 1982), making badgers an ideal maintenance host of *M. bovis*. Experimentally, calves have been infected from contact with experimentally infected, as well as naturally infected badgers (Little et al. 1982), and epidemiological studies have shown that areas with the greatest density of badgers have the highest incidence of tuberculosis among cattle (Muirhead et al. 1974; Cheeseman et al. 1989; Krebs et al. 1998). Badger-to-badger transmission is most likely respiratory and to a lesser extent cutaneous through bite wounds (Cheeseman et al 1989).

2.4 Zoonotic Potential

Recently, the first documented cases of spillover of bovine tuberculosis from animals to humans were reported since the resurgence of the disease in the United Kingdom (Smith et al. 2004). Two siblings residing on a farm were diagnosed with tuberculosis due to *M. bovis*. Cattle on the farm also had been diagnosed with *M. bovis*. The cattle isolate was indistinguishable from the isolates from the two siblings when examined by restriction fragment length polymorphism (RFLP) analysis, spacer oligonucleotide typing (spoligotyping), and variable number tandem repeat (VNTR) analysis, suggesting transmission between cattle and humans. Moreover, the farm supported a large badger population where *M. bovis* infection had been previously diagnosed. It is suggested, although not proven, that cattle became infected through contact with badgers and that humans became infected through contact with cattle.

2.5 Disease Control

Efforts to remove badgers from cattle farming areas have resulted in a decline in bovine tuberculosis (Little et al. 1982). Following the first suggested links between badgers and bovine tuberculosis, farmers were licensed to cull badgers; from 1975 to 1981 hydrogen cyanide gas was used to kill badgers (Donnelly et al. 2003). Gassing with hydrogen cyanide was later replaced with a strategy to identify and remove clusters of infected badgers. From 1986 to 1998, culling occurred only on land where tuberculin test-positive cattle were present (Donnelly et al. 2003). The effectiveness of large-scale culling as opposed to selective culling remained unknown until recently. In 1998, a large experiment was implemented to compare the effects of three different control strategies: no culling of badgers, localized selective culling of badgers in response to identified cases of tuberculosis in cattle, and proactive culling aimed at reducing badger densities to low levels across entire trial areas. Five years into the study, it was determined that reactive culling of badgers resulted in increased levels of tuberculosis in cattle within the trial areas (Donnelly et al. 2003). In response to these findings, reactive culling was discontinued as part of the study, while proactive culling and no culling continue as experimental treatments within the study. The reason for an increased level of tuberculosis in cattle in reactive culling treatment areas is unknown. However, it is known that badger social structures are complex and selective removal of some but not all badgers may result in increased badger movement with badgers using latrines far distant from their original sett, resulting in enlarged social groups with overlapping boundaries (Tuyttens et al. 2000). Such social restructuring among populations with *M. bovis*-infected badgers may result in increased disease transmission among badgers and between badgers and cattle. Increased social restructuring and badger movement has been correlated with increased incidence of *M. bovis* infection among badger populations (Rogers et al. 1998).

Complete removal of any wildlife reservoir of infection is extremely difficult and in the long term (see the chapters by Childs and by Stallknecht, this volume), most believe that the best prospect for control of bovine tuberculosis in Great Britain is a vaccine for cattle, combined with improved diagnostic tests to distinguish vaccinated from infected cattle (Krebs et al. 1998). However, some also contend that a vaccine for badgers should be kept as an option (Anonymous 1997).

Cattle husbandry practices aimed at separating cattle and badgers have also been proposed as a means of tuberculosis control, including keeping cattle away from badger setts, urination trails, and latrines and keeping badgers away from cattle feed troughs and buildings. Studies to design elevated feed troughs that would exclude badgers concluded that the maximum height to which

badgers would climb into a trough was beyond that which would be useable for younger cattle (Garnett et al. 2003). Public attitudes favor no culling of badgers and surveys show that the public generally ranks conservation and animal welfare concerns over those of disease control.

2.6
Other Species as Potential Wildlife Reservoirs of *M. bovis*

In 2004, the results were released of a study to examine numerous species of wildlife in the UK for tuberculosis. Over 4,700 animal carcasses were examined and tissue samples processed for isolation of *M. bovis*. Infection was confirmed in foxes (*Vulpes vulpes*), stoats (*Mustela erminea*), polecats (*Mustela putorius*), common shrews (*Sorex araneus*), yellow-necked mice, squirrels (*Sciurus carolinensis*), roe deer, red deer (*Cervus elaphus*), fallow deer (*Dama dama*), and muntjac deer (*Muntiacus reevesi*). Sample size varied widely, but the highest prevalences were seen in foxes (3.2% of 756), stoats (3.9% of 78), polecats (4.2% of 24), common shrews (2.4% of 41), roe deer (1.0% of 885), red deer (1.0% of 196), fallow deer (4.4% of 504) and muntjac deer (5.2% of 58). A qualitative risk assessment based on prevalence, likelihood of excretion, likelihood of contact with cattle and animal biomass identified fallow deer and red deer as the highest risk, with scores of 0.75 and 0.5, respectively (a score of 1.0 being the highest risk). However, with a regional tuberculosis prevalence as high as 20.5% in badgers, they remain a primary concern for tuberculosis control in the UK. However, other species, particularly deer, may also pose significant risk, especially in regions where deer density is high (http://www.defra.gov.uk/science/project_data/DocumentLibrary/SE3010/SE3010_1628_FRP.doc).

3
New Zealand

3.1
History of Brushtail Possums and *M. bovis* Infection in New Zealand

Prior to the arrival of the first humans to New Zealand, the only mammals present were two species of bats (O'Neil and Pharo 1995). Cattle were introduced approximately 200 years ago and large areas of forest were cleared in the early nineteenth century to accommodate pastoral farming. About the same time, several species of deer were introduced for recreational hunting purposes. By the middle of the twentieth century, deer numbers had climbed to such levels that deer were considered by many as nuisance pests. Deer farming began in

the 1970s as wild deer were captured to establish breeding herds (O'Neill and Pharo 1995). Brushtail possums were first taken to New Zealand from Australia in the mid-nineteenth century to establish a fur trade. Between 1837 and 1922, over 30 groups of possums were imported, maintained in captivity for breeding, and released in over 160 different sites around New Zealand (O'Neill and Pharo 1995). The lack of natural predators combined with abundant food sources resulted in a rapid rise in possum numbers. Currently, possums occupy over 90% of New Zealand land area with an estimated 60–70 million possums nationwide. Possum density estimates range from 1.5 to 25 per hectare, where in some areas the possum density is 20 times greater than that seen in Australia (O'Neill and Pharo 1995).

Mycobacterium bovis was likely introduced to New Zealand with the importation of cattle in the nineteenth century. By the early twentieth century, tuberculosis was recognized as a serious animal and human health problem. Tuberculosis was first diagnosed in farmed deer in 1978 and subsequently spread by movement of untested farmed deer and capture of infected wild deer. The first reported case of tuberculosis in a wild possum in New Zealand was in 1967 (Ekdahl et al. 1970). However, the susceptibility of brushtail possums to infection with *M. bovis* had been determined much earlier (Bolliger and Bolliger 1948). Epidemiological evidence has linked possum tuberculosis and tuberculosis in cattle (Collins et al. 1988). It is likely that possums in New Zealand acquired *M. bovis* from other animals, likely cattle, as *M. bovis* infection has never been seen in Australian possums, the original source of New Zealand's possums.

3.2 Pathology and Transmission

Tuberculous possums often develop disseminated disease, with lymph nodes and lungs being the most common sites of infection. Additionally, one study reported that at least 45% of affected possums had a discharging sinus from a superficial lymph node lesion (Cooke et al. 1995). Lesions can also be seen in the liver, spleen, kidneys, adrenal glands, and bone marrow, suggesting generalized hematogenous spread of bacilli. In one study, lesions were present in one or more of these sites in 86% of 73 tuberculous possums, suggesting that hematogenous dissemination of disease is common in possums (Jackson et al. 1995a). In contrast to lesions in cattle, fibrosis, mineralization and Langhans type giant cells are uncommon, while acid fast bacilli are numerous. The character of the lesions suggests an ineffective host immune response to infection, unable to sequester infection, thereby allowing rapid hematogenous dissemination. In spite of disseminated disease, normal growth of the possum is not significantly

affected until late stages of the disease (Jackson et al. 1995a). Infections among terminally ill possums are, however, characterized by widespread lesions involving numerous organ systems, resulting in a profound effect on behavior and survivability. The disseminated nature of the disease and limited effect on possum growth combined with pulmonary lesions and draining superficial lesions, all of which contain large numbers of *M. bovis*, make possums an ideal maintenance host capable of efficient transmission to other susceptible hosts.

Transmission among possums occurs between mother and offspring as well as direct horizontal transmission among adults. Respiratory secretions are thought to be most important in possum to possum transmission; however, some transmission to offspring through milk also occurs (Jackson et al. 1995b). Infected possums shed *M. bovis* primarily in respiratory secretions and exudate from draining lesions (Jackson et al. 1995b). Shared use of dens would seem a logical point at which transmission of *M. bovis* would occur and indeed in studies using captive possums; den-sharing provided the greatest risk of transmission between possums (Corner et al. 2003). Den-sharing has not been commonly observed in free-living possums; however, sequential den use by different possums has been observed (Paterson et al. 1995) and *M. bovis* has been shown to survive inside possum dens for 7–28 days depending on environmental conditions (Jackson et al. 1995c). The dynamics of possum-to-possum transmission of *M. bovis* appear to be complex and involve individual possum social status. Evidence of this is found in studies demonstrating that naturally infected possums tend to be possums that are central and prominent in the local social hierarchy. Furthermore, experimental infection of such socially dominant possums results in higher levels of disease transmission than experimental infection of possums ranked lower in the societal structure (Corner et al. 2003).

Healthy possums generally avoid contact with cattle (Paterson et al. 1995). Terminally ill possums exhibit abnormal behavior such as increased daytime activity, stumbling, rolling and falling, which attracts attention of inquisitive cattle. Studies using sedated possums to simulate terminally ill possums demonstrated that both deer and cattle exhibit profound interest in abnormally behaving possums. Cattle were seen to be attracted from as far as 50 m to investigate sedated possums (Paterson and Morris 1995). Deer and cattle were shown to spend significant amounts of time within a distance compatible with aerosol transmission (approximately 1.5 m) and to even sniff, touch, lick, roll, lift, chew and kick the possum, creating opportunity for direct transmission (Paterson and Morris 1995; Sauter and Morris 1995). In studies where cattle have been excluded from areas used for denning by tuberculous possums, decreased transmission of *M. bovis* from possums to cattle has been demonstrated. In contrast, where cattle are allowed to graze areas used for denning by tuberculous possums, transmission to cattle continues unabated (Paterson et al. 1995).

3.3 Disease Control

No widespread eradication of a vertebrate host has ever been successful in New Zealand. The attitudes toward possums in New Zealand differ from those of other wildlife reservoirs of tuberculosis in other countries. In New Zealand, possums are viewed as non-native, invasive pests, and widespread removal of possums is desirable for many reasons apart from tuberculosis control. Possums have had a disastrous impact on New Zealand's native flora and fauna. Every night, an estimated 70 million possums consume approximately 21,000 tons of green shoots, leaves, and berries. Possums are omnivorous and also consume bird's eggs, chicks, and insects. While browsing in the forest canopy on fruits and flowers, possums are in direct competition with native nectar feeding birds. While on the ground, possums compete with native kiwi for dens and have been seen eating kiwi eggs. Theoretically, widespread removal of possums from New Zealand's ecosystem would be more socially palatable than removal of native wildlife reservoirs of tuberculosis in other countries. Early control measures included a bounty system on possums, which was minimally effective. Bounties did not allow for prioritization of control efforts and although many possums were removed they were generally not removed from the right places. Possums were generally taken for bounty from easily accessible locations leaving many critical areas unchanged. Recently, aerial distribution of possum baits containing 1080 poison (sodium monofluoracetate) has achieved 90% death rates in some areas (Caley et al. 1999). An effective poison, 1080 causes possums to die of cardiac or respiratory failure. Other poisons that have been used to control possums include brodifacoum, pindone, cyanide, and cholecalciferol. In areas where 1080 baits have been used to decrease possum numbers, tuberculin reactor rates in cattle herds and numbers of tuberculous possums have decreased, only to return to elevated levels in 8–10 years as possum numbers recover through breeding and immigration from surrounding areas (Barlow 1991; Tweddle and Livingstone 1994). Long term (>10 years) maintenance of possum populations below 40% of precontrol densities over widespread areas may be required to affect significant change in cattle tuberculin reactor rates and eradicate tuberculosis from possum populations (Caley et al. 1999)

Although widespread removal of possums through poisoning may decrease the prevalence of tuberculosis in cattle, complete removal of possums from New Zealand may be impractical. It has been suggested that the most promising option for long-term control of tuberculosis in possums is the development of a vaccine combined with a strategy for biological control of possums. *Mycobacterium bovis* BCG vaccine has been administered to possums by subcutaneous, intranasal, and intraduodenal

routes (Aldwell et al. 1995a, 1995b; Buddle et al. 1997; Corner et al. 2001). All routes provide some protection against aerosol challenge with virulent *M. bovis*, evidenced by reduced disease severity, reduced loss of body weight, fewer lung lesions, and decreased bacterial colonization.

Other species such as red deer, feral pigs (*Sus scrofa*), feral cats (*Felis catus*), ferrets (*Mustela furo*), and stoats, goats (*Capra hircus*), rabbits (*Oryctolagus cuniculus*), hares (*Lepus europaeus*), and hedgehogs (*Erinaceus europaeus*) have been found infected with *M. bovis* (Jackson 2002; Tweddle and Livingstone 1994). The role most of these species play in the epidemiology of bovine tuberculosis in New Zealand is not clear; however, of these species, red deer may be another maintenance host of tuberculosis in New Zealand.

4
United States

4.1
History of *M. bovis* Infection in White-Tailed Deer in Michigan, USA

Prior to 1994, there had been only isolated case reports of tuberculosis in white-tailed deer in the United States (Levine 1934; Ferris et al. 1961; Belli 1962; Friend et al. 1963). All reports involved one to two animals and were seen in captive deer, hunter-killed deer, or deer dying of accidental causes. In almost all cases, it was postulated that *M. bovis* had spilled over from tuberculous livestock in the area; however, no follow-up surveys were conducted and no strain comparisons were made to confirm such a hypothesis. In 1975, a free-ranging white-tailed deer in northern Michigan was diagnosed with tuberculosis due to *M. bovis*. Michigan had been declared free of *M. bovis* in livestock in 1975 and was granted TB-free status by the United States Department of Agriculture in 1979. The tuberculous white-tailed deer was thought to be an anomaly and no follow-up surveys of free-ranging deer were conducted. In 1994, a free-ranging, hunter-killed white-tailed deer was identified with tuberculosis due to *M. bovis*. This deer was located just 13 km from the site where the tuberculous deer had been identified in 1975. Subsequent surveys conducted by the Michigan Department of Natural Resources and Michigan State University Animal Health Diagnostic Laboratory identified a focus of *M. bovis* infection in free-ranging white-tailed deer in northeast Michigan (Schmitt et al. 1997). This represented the first known reservoir of *M. bovis* in free-living wildlife in the United States and the first known epizootic of tuberculosis in white-tailed deer in the world. Several factors are thought to have contributed to the establishment and persistence of *M. bovis* in this wildlife reservoir. It is postulated that

M. bovis was transmitted from cattle to deer at some time during the early to mid 1900s when a large number of Michigan cattle were infected with *M. bovis* (Frye 1995). Statistical models estimate that spillover from cattle to deer occurred around 1955 (McCarty and Miller 1998). During this same period, Michigan's deer population was steadily increasing beyond normal habitat carrying capacity. In 1930, there were an estimated 592,000 deer in Michigan. By 1998, the number of deer had grown to over 1.7 million statewide, with focal concentrations of 19–23 deer per square kilometer. The regions of highest deer density were later found to be the center of the current tuberculosis outbreak (Schmitt et al. 1997; O'Brien et al. 2002; Miller et al. 2003). Transmission and maintenance of *M. bovis* among deer is thought to have been facilitated by the common practice in Michigan of long-term winter feeding of large volumes of sugar beets, carrots, corn, apples, pumpkins, and pelleted feed to deer by private citizens to prevent migration and decrease winter mortality in order to keep deer numbers high for hunting purposes (Schmitt et al. 1997). The resulting increased population, combined with prolonged crowding of deer around feeding sites provided increased opportunity for deer-to-deer contact and enhanced transmission of tuberculosis. Supplemental feeding has been documented as a contributing factor to *M. bovis* infection in deer (Miller et al. 2003). Specific risk factors associated with increasing risk of tuberculosis were location of a feeding site near hardwood forest, number of deer fed per year, presence of other nearby feeding sites, and the quantity of grain, fruits, or vegetables fed. DNA fingerprinting through RFLP analysis of *M. bovis* isolates from Michigan white-tailed deer showed that the majority of deer were infected with a common strain of *M. bovis*, suggesting a single source of infection (Whipple et al. 1997). By 2003, over 123,249 deer had been tested by gross necropsy, bacteriologic culture, and histopathology since the identification of the first case in 1994. Of these, 481 cases of confirmed *M. bovis* infection had been identified in 12 counties in northern Michigan.

4.2 Transmission

The presence of *M. bovis* in wildlife is not only detrimental to the health of this wildlife population, but also represents a serious threat to domestic livestock. Thirty-two *M. bovis* -infected cattle herds have been identified in Michigan since the identification of tuberculosis in white-tailed deer. Restriction fragment length polymorphism analysis of *M. bovis* isolates from deer and cattle show that they are identical, suggesting cattle became infected through contact with free-ranging white-tailed deer (Whipple et al. 1999). Surveys of carnivores and omnivores in Michigan have confirmed *M. bovis* infection in coyotes (*Canis latrans*), bobcats

(*Felis rufus*), foxes (*Vulpes vulpes*), black bears (*Ursus americanus*), opossums (*Didelphis virginiana*), raccoons (*Procyon lotor*), and domestic cats (Bruning-Fann et al. 1998, 2001; Kaneene et al. 2002). Restriction fragment length polymorphism analysis suggests that deer and other wildlife are infected with a common strain of *M. bovis* and likely became infected through scavenging of dead deer carcasses; however, infection with limited lesion development in these scavenger species suggests that they are true spillover hosts and not important in the maintenance of the epizootic in deer or transmission to other susceptible hosts.

White-tailed deer experimentally infected with *M. bovis* shed bacilli in saliva and nasal secretions and less frequently in urine and feces (Palmer et al. 1999, 2001). Research has also shown that experimentally infected deer can transmit *M. bovis* to other deer or cattle through indirect contact such as sharing of feed (Palmer et al. 2001, 2004a, b). Furthermore, white-tailed deer experimentally inoculated by the aerosol route do not develop a pattern of lesions similar to that seen in naturally infected deer in Michigan, suggesting that aerosol transmission may not be the primary means of *M. bovis* transmission among Michigan deer (Palmer et al. 2003). Saliva and nasal secretions containing *M. bovis* contaminate feed that can act as a source of infection for other animals. *Mycobacterium bovis* is relatively resistant to environmental factors and under appropriate conditions (cool and protected from sunlight), *M. bovis* may persist in the environment for weeks or months, increasing the likelihood of transmission to other animals (Duffield and Young 1985; Jackson et al. 1995c; Tanner and Michel 1999; Whipple and Palmer 2000). Transmission from doe to fawn, although possible, is probably not important in the maintenance of the disease. Research has shown that fawns can be experimentally infected through consumption of milk containing *M. bovis* (Palmer et al. 2002); however, mammary gland lesions in naturally infected deer have been reported only rarely (O'Brien et al. 2001).

Epidemiologic modeling suggests a two-stage model of transmission. Stage 1 involves transmission within matriarchal groups, allowing disease to persist in the population at a low level (O'Brien et al. 2002). The social structure of white-tailed deer is characterized by family groups consisting of a matriarchal doe and several generations of her daughters and their fawns. Fawns from the previous year leave the dam when she nears parturition. Yearling does often rejoin their dam and her fawns in the fall. Stage 2 involves both supplemental feeding, with resultant increased deer density, and male fawns that disperse to join male groups that travel together at all times except during breeding season (O'Brien et al. 2002). Higher disease prevalence has been observed in adult male deer (Schmitt et al. 2002). Shifting membership by many males in these groups results in males temporarily belonging to several different groups and increased contact with numerous susceptible animals.

4.3 Pathology

Tuberculous white-tailed deer most commonly develop lesions in retropharyngeal lymph nodes, and in lung and pulmonary lymph nodes (Schmitt et al. 1997, Palmer et al. 2000, Fitzgerald et al. 2000). Similar to other species of Cervidae, lesions may grossly resemble abscesses, making differential diagnosis important. Unlike red deer, elk, and fallow deer, draining fistulae from superficial lymph node lesions have not been reported in white-tailed deer (Robinson et al. 1989; Lugton et al. 1998; Beatson 1985; Whiting and Tessaro 1994). Such lesions may be important in disease transmission among these other species of deer.

Microscopically, lesions consist of foci of caseous necrosis with or without mineralization, surrounded by infiltrates of epithelioid macrophages, lymphocytes, and Langhans type multinucleated giant cells. Lesions are often surrounded by variable amounts of fibrous connective tissue and low numbers of acid fast bacilli may be present within the caseum, macrophages, or multinucleated giant cells. Microscopically, lesions in white-tailed deer are similar to those seen in cattle; although subjectively, lesions in cattle may be surrounded by greater amounts of fibrous connective tissue.

4.4 Zoonotic Potential

Although *M. bovis* is a recognized zoonotic agent, no change in incidence of *M. bovis* infections in Michigan's human population has been detected since the epizootic was recognized (Wilkins et al. 2003), and no cases of *M. bovis* in humans have been directly attributed to contact with infected wildlife. Nevertheless, there are potential risks as hunters are exposed to *M. bovis* during the field dressing of deer or the consumption of undercooked venison products. Michigan's Departments of Community Health, Natural Resources and Agriculture have worked cooperatively to educate hunters, farmers, and other Michigan residents on the identification of tuberculosis in deer, personal protective measures hunters can take while field dressing deer, and the importance of thorough cooking of venison prior to consumption (Wilkins et al. 2003).

4.5 Disease Control

In Michigan, wildlife and domestic animal health authorities have adopted control measures that (1) reduce deer density and population through increased hunting, (2) restrict or eliminate supplemental feeding of deer, and (3) monitor

both wildlife and domestic livestock through hunter-killed deer surveys, selected carnivore and omnivore removal and surveillance, and whole-herd cattle testing. These control measures appear to have succeeded in preventing increasing prevalence and geographic spread of tuberculosis in white-tailed deer in Michigan. Supplemental feeding of deer has been banned since 1998 in counties where tuberculous deer have been identified. Enforcement of such a ban has been problematic and universal compliance has not been achieved. Deer numbers have been reduced by 50% in the endemic areas through increased hunting pressure and unlimited harvest of female deer. However, progress toward eradication will likely require further action and more time. Epidemiological modeling suggests that further decreases in deer density and a strictly enforced ban on supplemental feeding will be required to achieve TB-free status.

5 Italy and Spain

Recently, *M. bovis* has been identified in wild boars in Italy. Restriction fragment length polymorphism analysis and spoligotyping have shown that many of the strains isolated from boars are identical to isolates obtained from cattle in the same region (Serraino et al. 1999). The exact means of interspecies transmission is unknown; however, it is speculated that boars are contaminating pastures and thus transmitting the disease to cattle. Similarly, *M. bovis* has been identified in wildlife in Spain, including red deer, fallow deer, wild boar, Iberian lynx (*Lynx pardina*), and hare. Again, transmission between cattle and wildlife is implicated due to similar spoligotyping patterns between livestock and wildlife species (Aranaz et al. 2004).

6 Conclusions

The complex interactions of domestic animals, wildlife, and humans that create emerging disease situations dictate that approaches to disease control will not be simple. Any single approach directed at only one area is not likely to succeed. The test and slaughter policy of tuberculosis, which has been relatively effective in control of tuberculosis in domestic livestock, is recognized as insufficient in areas where wildlife reservoirs exist. Measures to prevent disease transmission are more efficient than efforts required to eliminate an established disease from wildlife or domestic animals. Human involvement in risk reduction

strategies such as education and promotion of biosecurity practices that limit interactions between livestock and wildlife will be required. Serious risk analysis should be conducted prior to introduction or re-introduction of wildlife to new geographic areas. In areas where tuberculosis is endemic in wildlife, certain agricultural practices such as allowing wildlife access to livestock feed may no longer be tolerable if disease is to be eradicated.

The elimination of tuberculosis from free-ranging wildlife is a difficult goal. It will require the cooperation of agricultural and wildlife agencies, legislative bodies, private landowners, and citizens. Because of limited resources, multiple agencies must work collaboratively; assessing blame to one group or organization will be counterproductive. The idea of organizations from different backgrounds working together to address diseases transmitted between domestic animals and wildlife is gaining momentum. This movement is evidenced by a number of recent symposia on diseases at the interface of domestic animals and wildlife, and the creation of wildlife disease committees in traditionally agriculturally based producer groups. Furthermore, resolutions from groups such as the Wildlife Disease Association and the Society for Tropical Veterinary Medicine have come forward that call for funding organizations to encourage projects that foster integration of livestock production and natural resource management, address wildlife, livestock, and rangeland health in environmental impact statements, and use science-based advice when contemplating projects involving wildlife and livestock (Anonymous 2002).

References

Aldwell FE, Keen DL, Stent VC, Thomson A, Yates GF, de Lisle GW, Buddle BM (1995a) Route of BCG administration in possums affects protection against bovine tuberculosis. N Z Vet J 43:356–359

Aldwell FE, Pfeiffer A, de Lisle GW, Jowett G, Heslop J, Keen D, Thomson A, Buddle BM (1995b) Effectiveness of BCG vaccination in protecting possums against bovine tuberculosis. Res Vet Sci 58:90–95

Anonymous (1997) TB in cattle and badgers: review group advocates an investigative approach. Vet Rec 141:636

Anonymous (2002) Resolution by the Wildlife Disease Association and the Society for Tropical Veterinary Medicine calling for international donor community recognition of animal health sciences as critical for the design and management of sustainable wildlife and/or livestock based programs. Ann N Y Acad Sci 969: 364–365

Aranaz A, de Juan L, Montero N, Sanchez C, Galka M, Delso C, Alvarez J, Romero B, Bezos J, Vela AI, Briones V, Mateos A, Dominguez L (2004) Bovine tuberculosis (*Mycobacterium bovis*) in wildlife in Spain. J Clin Microbiol 42:2602–2608

Barlow NE (1991) A spatially aggregated disease/host model for bovine TB in New Zealand possum populations. J Appl Ecol 28:777–793
Beatson NS (1985) Tuberculosis in red deer in New Zealand. In: Fennessy PF, Drew KR (eds) Biology of deer production. R Soc N Z 22:147–150
Belli LB (1962) Bovine tuberculosis in a white-tailed deer. Can Vet J 3:356–358
Bollinger A, Bolliger W (1948) Experimental transmission of tuberculosis to *Trichosurus vulpecula*. Aust J Sci 10:182–183
Bouvier G (1963) Possible transmission of tuberculosis and brucellosis from game animals to man and to domestic animals. Bull Off Inter Epiz 59:433–436
Bruning-Fann CS Schmitt SM, Fitzgerald SD, Payeur JB, Whipple DL, Cooley TM, Carlson T, Friedrich P (1998) *Mycobacterium bovis* in coyotes from Michigan. J Wildl Dis 34:632–636
Bruning-Fann CS, Schmitt SM, Fitzgerald SD, Fjerke JS, Friedrich PD, Kaneene JB, Clarke KA, Butler KL, Payeur JB, Whipple DL, Cooley TM, Miller JM, Muzo DP (2001) Bovine tuberculosis in free-ranging carnivores from Michigan. J Wildl Dis 37:58–64
Buddle BM, Aldwell FE, Keen DL, Parlane NA, Yates G, de Lisle GW (1997) Intraduodenal vaccination of brushtail possums with bacilli Calmette-Guerin enhances immune responses and protection against *Mycobacterium bovis* infection. Int J Tub Lung Dis 1:377–383
Caley P, Hickling GJ, Cowan PE, Pfeiffer DU (1999) Effects of sustained control of brushtail possums on levels of *Mycobacterium bovis* infection in cattle and brushtail possum population from Hohotaka New Zealand. N Z Vet J 47:133–142
Cheeseman CL, Wilesmith JW, Stuart FA, Mallinson PJ (1988) Dynamics of tuberculosis in a naturally infected badger population. Mammal Rev 18:61–72
Cheeseman CL, Wilesmith JW, Stuart FA (1989) Tuberculosis: the disease and its epidemiology in the badger, a review. Epidem Infect 103:113–125
Collins DM, Gabric DM, de Lisle GW (1988) Typing of *Mycobacterium bovis* isolates from cattle and other animals in the same locality. N Z Vet J 36:45–46
Cooke MM, Jackson R, Coleman JD, Alley MR (1995) Naturally occurring tuberculosis caused by *Mycobacterium bovis* in brushtail possums (*Trichosurus vulpecula*): II. Pathology. N Z Vet J 43:315–321
Corner LAL, Buddle B.M, Pfeiffer DU, Morris RS (2001) Aerosol vaccination of the brushtail possum (*Trichosurus vulpecula*) with bacilli Calmette-Guerin: the duration of protection. Vet Microbiol 81:181–191
Corner LAL, Pfeiffer DU, Buddle B.M, and Morris RS (2003) Social-network analysis of *Mycobacterium bovis* transmission among captive brushtail possums (*Trichosurus vulpecula*) Prev Vet Med 59:147–167
Cousins DV, Dawson DJ (1999) Tuberculosis due to *Mycobacterium bovis* in the Australian population: cases recorded during 1970–1994. Int J Tub Lung Dis 3:715–721
Donnelly CA, Woodroffe R, Cox ER, Bourne J, Gettinby G, Le Fevre AM, McInerney JP, Morrison WI (2003) Impact of localized badger culling on tuberculosis incidence in British cattle. Nature 426:834–837
Duffield BJ, Young DA (1985) Survival of *Mycobacterium bovis* in defined environmental conditions. Vet Microbiol 10:193–197

Ekdahl MO, Smith BL, Money DFL (1970) Tuberculosis in some wild and feral animals in New Zealand. N Z Vet J 18:44–45

Fanning A, Edwards S (1991) *Mycobacterium bovis* infection in human beings in contact with elk (*Cervus elaphus*) in Alberta Canada. Lancet 338:1253–1255

Ferris DH, Beamer PD, Alberts JO, Trainer D (1961) Tuberculosis in transported deer. J Am Vet Med Assoc 138:326–328

Fitzgerald SD, Kaneene JB, Butler KL, Clarke KR, Fierke JS, Schmitt SM, Bruning-Fann CS, Mitchell RR, Berry DE, Payeur JB (2000) Comparison of postmortem techniques for the detection of *Mycobacterium bovis* in white-tailed deer (*Odocoileus virginianus*). J Vet Diag Invest 12:322–327

Friend M, Kroll ET, Gruft H (1963) Tuberculosis in a wild white-tailed deer. N Y Fish Game J 10:118–123

Frye GH (1995) Bovine tuberculosis eradication: the program in the United States. In: Thoen CO, Steele JH (eds) *Mycobacterium bovis* infection in animals and humans. Iowa State University Press, Ames IA, pp 119–129

Gallagher J, Muirhead RH, Burn KJ (1976) Tuberculosis in wild badgers (*Meles meles*) in Gloucestershire: Pathology. Vet Rec 98:9–14

Garnett BT, Roper TJ, Delahay RJ (2003) Use of cattle troughs by badgers (*Meles meles*): a potential route for the transmission of bovine tuberculosis (*Mycobacterium bovis*) to cattle. Appl Anim Beh Sci 80:1–8

Gavier-Widen D, Chambers MA, Palmer N, Newell DG, Hewinson RG (2001) Pathology of natural *Mycobacterium bovis* infection in European badgers (*Meles meles*) and its relationship with bacterial excretion. Vet Rec 148:299–304

Grange JM, Yates MD (1994) Zoonotic aspects of *Mycobacterium bovis* infection. Vet Microbiol 40:137–151

Hutchings MR, Harris S (1997) Effects of farm management practices on cattle grazing. Behaviour and the potential for transmission of bovine tuberculosis from badgers to cattle. Vet J 153:149–162

Jackson R (2002) The role of wildlife in *Mycobacterium bovis* infection of livestock in New Zealand. N Z Vet J 50:49–52

Jackson R, Cooke MM, Coleman JD, Morris RS (1995a) Naturally occurring tuberculosis caused by *Mycobacterium bovis* in brushtail possums (*Trichosurus vulpecula*): I. An epidemiologic analysis of lesion distribution. N Z Vet J 43:306–315

Jackson R, Cooke MM, Coleman JD, Morris RS, de Lisle GW, Yates GF (1995b) Naturally occurring tuberculosis caused by *Mycobacterium bovis* in brushtail possums (*Trichosurus vulpecula*): III. Routes of infection and excretion. N Z Vet J 43:322–327

Jackson R, de Lisle GW, Morris RS (1995c) A study of the environmental survival of *Mycobacterium bovis* on a farm in New Zealand. N Z Vet J 43:346–352

Kaneene JB, Bruning-Fann CS, Dunn J, Mullaney TP, Berry D, Massey JP, Thoen CO, Halstead S, Schwartz K (2002) Epidemiologic investigation of *Mycobacterium bovis* in a population of cats. Am J Vet Res 63:1507–1511

Krebs JR, Anderson RM, Clutton-Brock T, Donnelly CA, Frost S, Morrison WI, Woodroffe R, Young D (1998) Badgers and bovine TB: conflicts between conservation and health. Science 279:817–818

Levine PP (1934) A report on tuberculosis in wild deer (*Odocoileus virginianus*) Cornell Vet 24:264–266

Little TWA, Naylor PF, Wilesmith JW (1982) Laboratory study of *Mycobacterium bovis* infection in badgers and calves. Vet Rec 111:550–557

Lugton IW, Wilson PR, Morris RS, Nugent G (1998) Epidemiology and pathogenesis of *Mycobacterium bovis* infection of red deer (*Cervus elaphus*) in New Zealand. N Z Vet J 46:147–156

McCarty CW, Miller MW (1998) A versatile model of disease transmission applied to forecasting bovine tuberculosis dynamics in white-tailed deer populations. J Wildl Dis 34:722–730

Miller R, Kaneene JB, Fitzgerald SD, Schmitt SM (2003) Evaluation of the influence of supplemental feeding of white-tailed deer (*Odocoileus virginianus*) on the prevalence of bovine tuberculosis in the Michigan wild deer population. J Wildl Dis 39:84–95

Muirhead RH, Gallagher J, Burn KJ (1974) Tuberculosis in wild badgers in Gloucestershire: epidemiology. Vet Rec 95:552–555

Noonan NL, Sheane WD, Harper LR, Ryan PJ (1975) Wildlife as a possible reservoir of bovine tuberculosis. Irish Vet J 29:1

O'Brien DJ, Fitzgerald SD, Lyon TJ, Butler JL, Fierke JS, Clarke KR, Schmitt SM, Cooley TM, Berry DE (2001) Tuberculous lesions in free-ranging white-tailed deer in Michigan. J Wildl Dis 37:608–613

O'Brien DJ, Schmitt SM, Fierke JS, Hogle SA, Winterstein SR, Cooley TM, Moritz WE, Diegel KL, Fitzgerald SD, Berry DE, Kaneene JB (2002) Epidemiology of *Mycobacterium bovis* in free-ranging white-tailed deer Michigan USA, 1995–2000. Prev Vet Med 54:47–63

O'Neill BD, Pharo HJ (1995) The control of bovine tuberculosis in New Zealand. N Z Vet J 43:249–255

Palmer MV, Whipple DL, Olsen SC (1999) Development of a model of natural infection with *Mycobacterium bovis* in white-tailed deer. J Wildl Dis 35:450–457

Palmer MV, Whipple DL, Payeur JB, Alt DP, Esch KJ, Bruning-Fann CS, Kaneene JB (2000) Naturally occurring tuberculosis in white-tailed deer. J Am Vet Med Assoc. 216:1921–1924

Palmer MV, Whipple DL, Waters WR (2001) Experimental deer to deer transmission of *Mycobacterium bovis*. Am J Vet Res 62:692–696

Palmer MV, Waters WR, Whipple DL (2002) Milk containing *Mycobacterium bovis* as a source of infection for white-tailed deer fawns (*Odocoileus virginianus*). Tuberculosis 82:161–165

Palmer MV, Waters WR, Whipple DL (2003) Aerosol exposure of white-tailed deer (*Odocoileus virginianus*) to *Mycobacterium bovis*. J Wildl Dis 39:817–823

Palmer MV, Waters WR, Whipple DL (2004a) Investigation of the transmission of *Mycobacterium bovis* from deer to cattle through indirect contact. Am J Vet Res 65:1483–1489

Palmer MV, Waters WR, Whipple DL (2004b) Shared feed as a means of deer-to-deer transmission of *Mycobacterium bovis*. J Wildl Dis 40:87–91

Paterson BM, Morris RS (1995) Interactions between beef cattle and simulated tuberculous possums on pasture. N Z Vet J 43:289–293

Paterson BM, Morris RS, Weston J, Cowan PE (1995) Foraging and denning patterns of brushtail possums, and their possible relationship to contact with cattle and the transmission of bovine tuberculosis. N Z Vet J 43:281–288

Robinson P, Morris D, Antic R (1988) *Mycobacterium bovis* as an occupational hazard in abattoir workers. Aust N Z J Med 18:701–703

Robinson RC, Phillips PH, Stevens G, Storm PA (1989) An outbreak of *Mycobacterium bovis* infection in fallow deer (*Dama dama*). Aust Vet J 66:195–197

Rogers LM, Delahay R, Cheeseman CL, Langton S, Smith GC, Clifton-Hadley RS (1998) Movement of badgers (*Meles meles*) in a high density population: individual, population and disease effects. Proc Ro Soc Lond B 265:1269–1276

Sauter CM, Morris RS (1995) Behavioural studies on the potential for direct transmission of tuberculosis from feral ferrets (*Mustela furo*) and possums (*Trichosurus vulpecula*) to farmed livestock. N Z Vet J 43:294–300

Scantlebury M, Hutchings MR, Allcroft DJ, Harris S (2004) Risk of disease from wildlife reservoirs: badgers, cattle and bovine tuberculosis. J Dairy Sci 87:330–339

Schmitt SM, Fitzgerald SD, Cooley TM, Bruning-Fann CS, Sullivan L, Berry D, Carlson T, Minnis RB, Payeur JB, Sikarskie J (1997) Bovine tuberculosis in free-ranging white-tailed deer from Michigan. J Wildl Dis 33:749–758

Schmitt SM, O'Brien DJ, Bruning-Fann CS, Fitzgerald SD (2002) Bovine tuberculosis in Michigan wildlife and livestock. Ann N Y Acad Sci 969:262–268

Serraino A, Marchetti G, Sanguinetti V, Rossi MC, Zanoni RG, Catozzi L, Bandera A, Dini W, Mignone W, Franzetti F, Gori A (1999) Monitoring of transmission of tuberculosis between wild boars and cattle: genotypical analysis of strains by molecular epidemiology techniques. J Clin Microbiol 37:2766–2771

Smith RMM, Drobniewski F, Gibson A, Montague JDE, Logan MN, Hunt D, Hewinson G, Salmon RL, O'Neill B (2004) *Mycobacterium bovis* infection United Kingdom. Emerg Infect Dis 10:539–541

Steele JH (1995) Regional and country status reports. In: Thoen CO, Steele JH (eds) *Mycobacterium bovis* infection in animals and humans. Iowa State University Press, Ames, IA, pp 169–171

Tanner M, Michel AL (1999) Investigation of the viability of *Mycobacterium bovis* under different environmental conditions in the Kruger National Park. Onderst J Vet Res 66:185–190

Tuyttens FAM, Delahay RJ, MacDonald DW, Cheeseman CL, Long B, Donnelly CA (2000) Spatial perturbation caused by a badger (*Meles meles*) culling operation: implications for the function of territoriality and the control of bovine tuberculosis (*Mycobacterium bovis*). J Anim Ecol 69:815–828

Tweddle NE, Livingstone P (1994) Bovine tuberculosis control and eradication programs in Australia and New Zealand. Vet Microbiol 40:23–29

Whipple DL, Palmer MV (2000) Survival of *Mycobacterium bovis* on feeds used for baiting white-tailed deer (*Odocoileus virginianus*) in Michigan. In: Proceedings of the = 49 th Annual Wildlife Disease Assocociation, p 21

Whipple DL, Meyer RM, Berry DF, Jarnigan JL, Payeur JB (1997) Molecular epidemiology of tuberculosis in wild white-tailed deer in Michigan and elephants. In: Proc U S Anim Health Assoc 543–546

Whipple DL, Jarnagin JL, Payeur JB (1999) DNA fingerprinting of *Mycobacterium bovis* isolates from animals in northeast Michigan. In: Proceedings for the IX International Symposium and World Association of Veternary Laboratory Diagnosis, p 83

Whiting TL, Tessaro SV (1994) An abattoir study of tuberculosis in a herd of farmed elk. Can Vet J 35:497–501

Wigle W, Ashley MJ, Killough EM, Cosens M (1972) Bovine tuberculosis in humans in Ontario. Am Rev Resp Dis 106:528–534

Wilkins MJ, Bartlett PC, Frawley B, O'Brien DJ, Miller CE, Boulton ML (2003) *Mycobacterium bovis* (bovine TB) exposure as a recreational risk for hunters: results of a Michigan Hunter Survey, 2001. Int J Lung Dis 7:1001–1009

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