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PRELIMINARY RESULTS FROM THE 2010-2011 MOOSE HEALTH ASSESSMENT PROJECT

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SUMMARY OF FINDINGS

This project, which began in 2007, represents the first 2 years of the second phase (2010 – 2012) of an overall health assessment of hunter-harvested moose (*Alces alces*) in northeastern Minnesota (MN The objectives of this project are to: (1) Screen hunter-harvested (and presumably healthy) moose from 2010 to 2012 for select disease agents, (2) Monitor changes in disease incidence or prevalence over time, (3) Assess the clinical impacts of liver fluke (*Fascioloides magna*) infection on moose, and (4) Determine the frequency of histological lesions consistent with brainworm (*Parelaphostrongylus tenuis*) infection. Samples were collected from 199 moose (*n*= 128 in 2010 and *n* =76 in 2011). Moose were screened for West Nile virus, eastern equine encephalitis, western equine encephalitis, St. Louis encephalitis, malignant catarrhal fever, borreliosis (*Borrelia burgdorferi*), anaplasmosis (*Anaplasma phagocytopila*, formerly *Ehrlichia phagocytophila*) and 6 serovars of leptospirosis. There was evidence of exposure to West Nile Virus (25%), malignant catarrhal fever (8%), borreliosis (21%), and leptospirosis (0.6–7.5%). Portions of brain, cerebral spinal fluid, whole blood, and serum were submitted for polymerase chain reaction (PCR) for Flavivirus RNA. Whole livers and brains were collected and examined grossly and histologically for evidence of brainworm and liver flukes; both parasites were documented. Full serum chemistry profiles were conducted on 158 moose and were used to determine if a correlation exists between liver fluke damage and serum liver enzymes. Whole blood samples from 168 moose were submitted for evaluation for tick-borne illnesses; anaplasmosis and piroplasma infections were also documented.

INTRODUCTION

The current aerial survey trend data indicates the moose population in northeastern MN is declining. Since 2002, annual survival and reproductive rates were substantially lower than documented elsewhere in North America (Lenarz et al. 2007). Further, the population estimate has declined over 50% from 2005 (*n*= 8,160) to 2012 (n = 4,230) (Lenarz 2012). Likewise, recruitment and twinning rates have steadily declined since 2002 (Lenarz 2011).

Previous and ongoing research has been unable to determine proximate and ultimate cause(s) of non-hunting moose mortality and the possible related impacts to the long-term viability of the northeastern MN population. In 2007, the MN Department of Natural Resources (MNDNR) began a 3-year moose health assessment project to determine which diseases northeastern MN moose are being exposed to and to establish baseline hepatic mineral levels. Results indicated that hunter-harvested moose in northeastern MN have been exposed to a variety of disease agents such as West Nile virus (WNV), eastern equine encephalitis (EEE), malignant catarrhal fever (MCF), anaplasmosis, borreliosis, and leptospirosis (Butler et al. 2010). While these findings were illuminating, there remained some key factors, the importance of which, we have been unable to determine, including: (1) The role liver damage (due to liver flukes) plays in non-hunting mortality, 2) The impact of arboviruses and how their incidences may be affected by changing climate, and (3) The impact of brainworm on moose survival, due to the difficulty in interpreting brain lesions caused by this parasite. To begin addressing these key factors, a second phase of the moose health assessment project was started in 2010.

Murray et al. (2006) concluded that moose in northwestern MN were dying from high liver fluke loads. However, assessing the extent of liver damage caused by flukes can be subjective. In order to determine if liver damage caused by flukes has clinical implications, serum liver enzymes should be evaluated. Beginning in 2009, we asked hunters to collect whole livers for evaluation. Samples were then ranked for liver fluke loads by a board-certified

veterinary pathologist. Results from this pilot year of liver examinations indicated that 35% of livers had fluke-induced lesions with some having nearly 100% of the liver parenchyma affected (Butler et al. 2010). However, poor blood collection techniques prevented assessment of the clinical impacts of the damage caused by the liver fluke infections. In 2010, we asked hunters to alter their blood collection strategies and began collecting both the whole liver and assessing serum liver enzymes, with the goal of determining whether results of gross evaluation of the liver correlated with enzyme indicators of liver function.

Our moose health assessment during 2007–2009 indicated that moose are being exposed to a variety of arboviruses, including EEE, WNV, borreliosis, and anaplasmosis (Butler et al. 2010). As climate changes, the density and distribution of capable arthropod vectors is expected to change as well (Gould and Higgs 2009). Climate is known to play a key role in determining the geographical and temporal distribution of arthropods, characteristics of arthropod lifecycles, dispersal patterns of associated arboviruses, evolution of arboviruses, and the efficiency with which they are transmitted from arthropods to vertebrate hosts (Gould and Higgs 2009). For example, there has been a substantial increase in tick-borne encephalitis in Sweden since the mid-1980s related to milder winters and earlier arrival of spring (Lindgren and Gustafson 2001). In Phase 2 of the moose health assessment study, serum will be screened for these arboviruses and a few additional select disease agents. Combined with results from our 2007–2009 sampling, we will have 6 years of data on the incidence of arbovirus exposure in our moose herd to evaluate any significant trends relative to fluctuations in climate. Additionally, beginning in 2011, samples were submitted for western equine encephalitis (WEE) and St. Louis encephalitis (SLE).

Diagnostics have shown that moose are dying from brainworm in MN. It is also known that moose are able to survive low-dose infections of brainworm and even develop immunity to subsequent infections (Lankester 2002). Researchers have hypothesized that brainworm was responsible for historic declines in moose populations (Karns 1967, Prescott 1974, Lankester 1987), but it is questionable whether brainworm represents a major threat to the northeastern MN population. In 2008, we began collecting whole brains from hunter-harvested moose to determine the frequency of brain lesions consistent with past brainworm infections in presumably healthy moose. These data would allow for better interpretation of migration tracts and could prevent pathologists from wrongly assigning brainworm as the cause of death based solely on the presence of migration tracts. We will continue to collect whole brains to increase our sample and quantify the number of presumably healthy moose have parasitic migration tracts.

METHODS

Hunters (tribal and state) were asked to collect whole livers, blood, hair, and a central incisor. State hunters were only allowed to harvest bulls while some tribal hunters were able to take either bulls or cows. Wildlife Health Program staff provided a presentation and instructions relative to the moose health assessment project at the mandatory MNDNR Moose Hunt Orientation Sessions and at tribal natural resource offices. Hunters were given a sampling kit with instructions at the sessions. Post-harvest, the sampling kits were dropped off at official registration stations by the hunters at the time of moose registration.

The MNDNR provided hunters with all the equipment needed for sample collection and preservation. Sampling kits included a cooler, 1-60-cc syringe for blood collection, 6-15-cc serum separator tubes, 2-5-cc ethylenediaminetetraacetic acid (EDTA) blood tubes for whole blood collection, 1 heavy-duty bag for liver storage, 2 coin envelopes for the tooth and hair collected, data sheet, protocol, Sharpie marker, 1 pair of large vinyl gloves, and 1 icepack.

Hunters collected blood using the 60-cc syringe after incising the jugular vein as soon after death as possible and recorded time of death and blood collection. Blood was placed in serum-separator tubes and in an EDTA tube and kept cool until they were delivered to official

MNDNR registration stations or tribal natural resource offices. Livers were placed in heavy-duty, pre-labeled bags.

At the stations or offices, serum-separator tubes were centrifuged and the serum decanted. Blood spinning time was recorded. Portable refrigerators were located in advance at the registration stations to maintain the tissue samples. One whole blood sample (EDTA tube) and 1 mL of serum were refrigerated and submitted every 2–3 days to the University of MN (UMN)-College of Veterinary Medicine-Clinical Pathology Laboratory for a full large-animal serum chemistry profile. The remaining whole blood sample was submitted every 2–3 days to the UMN-Department of Entomology for testing for tick-borne illnesses. Remaining serum and the whole livers were frozen. Cerebral spinal fluid was collected when possible. Whole brains were removed with the hunter's permission and placed in formalin. A 1x1x1" piece of brain was removed and frozen. The serum, whole liver, and whole brains were submitted to the UMN Veterinary Diagnostic Laboratory (UMN VDL, St. Paul, MN). The 1x1x1" piece of brain, cerebral spinal fluid, whole blood, and 1 mL of serum were submitted to the Minnesota Department of Health (MDH) for PCR for Flavivirus RNA.

Serum was tested for WNV, EEE, and WEE with a plaque reduction neutralization test (PRNT) and SLE with a serum neutralization test at the National Veterinary Services Laboratory (NVSL) in Ames, Iowa. Serum was screened for leptospirosis (microscopic agglutination test), borreliosis (immunofluorescence assay), anaplasmosis (card test), and MCF via peroxidase-linked assay (PLA) with positive PLA tests further tested with a virus neutralization test (VN) at the UMN VDL. The livers were ranked by a board-certified veterinary pathologist based on parenchymal damage due to liver flukes; ranking included no fluke-induced lesions (no evidence of fluke migration), mild infection (<15% of liver parenchyma is affected with mild prominence/fibrosis of bile ducts and few smaller nodules characterized by peripheral fibrosis and central presence of opaque brown pasty material), moderate infection (15–50% of the liver parenchyma affected by nodules and fibrosis), and marked infection (51–100% of the liver parenchyma affected with deformation of the entire liver by larger nodules with widespread fibrosis). Brains were examined histologically with 4 complete coronary brain, cerebellum, and brain stem sections processed from each moose. An average of 25 histological slides per animal were examined, including the frontal, temporal, parietal, and occipital lobes and the basal nuclei, thalamus, mesencephalon, and brain stem. Central incisors of moose were submitted to Mattson's Laboratory (Milltown, Montana) for aging by cementum annuli (Sergeant and Pimlott 1959).

RESULTS AND DISCUSSION

Samples from 199 moose (n = 128 in 2010 and n = 76 in 2011) were collected (189 males, 7 females, and 3 sex unknown) (Figure 1). Exact age was determined for 196 of these moose (median = 4, range = 1 – 13 years old).

Eastern Equine Encephalitis

Evidence of exposure to EEE was detected in 1/174 (0.6%) moose. The low detection rate in these moose was unexpected as an average exposure rate of 6.1% of the population was documented during Phase 1 of this study (Butler et al. 2010).The continued surveillance for EEE in Phase 2 of this study may provide greater insight into the annual variation in apparent disease prevalence.

A total of 65 moose were sampled (frozen brain, cerebral spinal fluid, serum, and whole blood) by the MDH by PCR for evidence of any Flavivirus RNA. All results were negative.

Mosquitoes spread EEE, which can cause neurologic signs and often death. It poses a greater mortality threat for most species than WNV, although the effects of EEE infection have not been studied in moose.

West Nile Virus

Evidence of exposure to WNV was detected in 44/174 (25%) moose. These results were similar, though slightly lower, to those reported during the first 3 years of the study (35%; Butler et al. 2010). Positive results indicated that animals were exposed to the WNV, but does not necessarily indicate illness. A titer that is greater than 100 is considered a very strong positive and means that the serum was able to neutralize nearly 100% of the virus. Multiple animals had titers ≥100.

Western Equine Encephalitis and St. Louis Encephalitis

Of the 64 sera samples submitted for WEE and SLE testing, none tested positive. Both of these diseases are mosquito-borne. WEE is known to occur infrequently in MN, although when it does, it is often part of a regional outbreak.

Malignant Catarrhal Fever Virus

Evidence of exposure to MCF was detected in 14/174 (8%) moose sampled with PLA. Follow-up testing with VN was negative for 12 of the 14, and the remaining 2 were unsuitable for testing. These PLA results are markedly lower than what we reported from 2007 to 2009 (35%; Butler et al. 2010). The PLA test is more sensitive than VN, meaning it is much better at identifying true positives, whereas VN is more specific and thus better at identifying true negatives. Malignant Catarrhal Fever is a gammaherpes virus, of which there are multiple strains (e.g., wildebeest strain of MCF, sheep strain of MCF, deer strain of MCF). The PLA reacts with multiple gammaherpes viruses. A PLA positive does not indicate the strain of exposure. The VN test only screens for the wildebeest strain (which is exotic to the U.S.) and would be negative if other strains are present. This means a sample that was positive on PLA and negative on VN was likely exposed to MCF, but not the wildebeest strain.

We have been collaborating with researchers (Dr. Hong Li, Washington Animal Disease Diagnostic Laboratory) to determine the strain of MCF exposure in the northeastern MN moose population. To date, all attempts at strain-typing have been unsuccessful.

Gammaherpes viruses have been documented to cause serious illness and death in moose and other ruminants. The clinical symptoms can mimic brainworm infection, including neurological deficits, blindness, and thrashing on the ground prior to death. While infection with MCF frequently results in death, carrier status can occur and is identified with serology. Zarnke et al. (2002) found serologic evidence of exposure in numerous species across Alaska and reported 1% prevalence in moose.

Anaplasmosis

No evidence of exposure to anaplasmosis was detected in moose screened for this disease in 2010 (n = 100). These results are similar to the results of 2007–2009 screening (1/319, 0.3%; Butler et al. 2010), indicating that exposure to this bacterium is likely occurring, albeit at a low rate. Anaplasmosis testing was no longer available in 2011.

Moose are thought to be susceptible to infection with *A. phagocytophilum*. In Norway, anaplasmosis was diagnosed in a moose calf, which displayed apathy and paralysis of the hind-quarters (Jenkins et al. 2001). This moose was concurrently infected with *Klebseilla* pneumonia, to which the calf's death was attributed, though the *Klebseilla* infection was most likely secondary to and facilitated by the primary infection with *A. phagocytophilum*. In sheep, this disease produces significant effects on the immunological defense system, increasing their susceptibility to disease and secondary infections (Larsen et al. 1994).

Borreliosis

Evidence of exposure to borreliosis was detected in 37/174 (21%) moose sampled. These results are similar to results from 2007 to 2009 (23%, Butler et al. 2010).

Borreliosis is a tick-borne bacterial disease that is maintained in a wildlife/tick cycle involving a variety of species, including mammals and birds. While evidence of natural infection in wildlife exists, there has been no documentation of clinical disease or lesions reported in wildlife species.

Leptospirosis

A total of 110 samples were screened for 6 serovars of *Leptospira interrogans*. Results per serovar are as follows:

- *L. interrogans bratislava*:
  - 1/173 (0.6%)
- *L. interrogans canicola*:
  - 1/173 (0.6%)
- *L. interrogans grippothyphosa*:
  - 1/173 (0.6%)
- *L. interrogans hardjo*:
  - 0/173
- *L. interrogans icterohaemorrhagicae*:
  - 1/173 (0.6%)
- *L. interrogans pomona*:
  - 13/173 (7.5%)

While the prevalences are lower for most of the serovars compared with data from 2007–2009, the prevalence of *L. pomona* remained stable (Butler et al. 2010). Leptospirosis is a bacterial disease that can infect a wide variety of mammals, both domestic and wild. Moose could be at an increased risk for leptospirosis, as it is often propagated by mud and water contaminated with urine, not uncommon in moose habitat.

General Tick-Borne Illness Screening

Whole blood samples from 168 (*n* = 109, *n* = 59 in 2010 and 2011, respectively) moose were submitted to the UMN Department of Entomology, where we are collaborating with Dr. Ulrike Munderloh to determine if hunter- harvested moose are infected with tick-borne illnesses. Samples were screened with a variety of PCR techniques. Results, only available for the 2010 samples, indicate that 10% of the moose were infected with anaplasmosis and 32% were positive for prioplasma primers. A hemolytic *Mycoplasma* was also identified in 19 of the samples. Further analysis is pending.

Brain Histopathology

Seventy-one whole brains were collected (*n* = 40 and 31 in 2010 and 2011, respectively). Since 2008, a total of 118 whole brains have been collected and examined. No lesions were found in 101 (86%) of the brains, 12 (10%) had lymphocytic infiltration (unspecific chronic inflammatory lesion), and 5 (4.2%) had lesions consistent with larval migration tracts (mild to moderate meningitis, axonal degeneration, and secondary demyelination).

Whole Liver Evaluation

Whole livers were collected from 169 (*n* = 108 and 61 in 2010 and 2011, respectively). Combined with livers collected in 2009 (*n* = 57), 226 livers have been submitted for gross examination. Of the 226 livers examined, 162 (72%) had no fluke-induced lesions, 34 (15%) had mild infection, 22 (9.7%) had moderate infection, and 8 (3.5%) had marked infection. Collection of whole livers will continue in 2012. Additionally, beginning in 2010, serum was submitted for a serum chemistry profile in an attempt to correlate serum liver enzyme levels with the level of fluke-induced damage. These results have not yet been analyzed.

Serum Chemistries

A total of 158 (*n* = 95 and 63 in 2010 and 2011, respectively) serum samples were submitted for a full large animal serum chemistry profile. Analysis of these results is pending. The purpose of collecting these data is to determine if there is a correlation between the liver ranking and serum liver enzymes, as well as to establish baseline "normals" for animals in this population.

Future Research

This project was the first to document EEE activity in NE MN, though extensive surveillance has not previously occurred. Because of its potential to cause illness and even death in humans and domestic animals, we have initiated a collaborative project with the MDH, the UMN, College of Veterinary Medicine, the UMN, Department of Entomology, and the Metropolitan Mosquito Control District. Mosquitoes will be trapped in a weekly basis at various locations throughout moose zones. The objectives of this project are 1.) Assess spatial and temporal distribution patterns of vectors of EEE throughout NE MN moose range 2.) Assess prevalence of EEE (and other arboviruses) in vector populations. Interestingly there appears to be differences in exposure rates between moose zones. Little is known about vector distribution in this area. This project will identify which mosquito species are present, how the species make-up changes over the summer, whether there is an actual difference in vectors between moose zones, and determine the prevalence rates of the arboviruses in the mosquitoes themselves.

ACKNOWLEDGMENTS

This project would not have been possible without assistance from a number of MNDNR personnel, tribal biologists, and volunteers. We would like to especially thank our crews that worked moose registrations stations: T. Rusch, J. Hines, D. Ingebrigtsen, B. Kirsch, N. Gellerman, W. Gessler, D. Litchfield, D. Pauly, M. Minchak, P. Backman, K. Carlisle, M. Dexter, and J. Hines. J. Hines also made our area maps. We also would like to thank tribal biologists M. Schrage, A. Edwards, L. Overland, and S. Moore for their help with the project.

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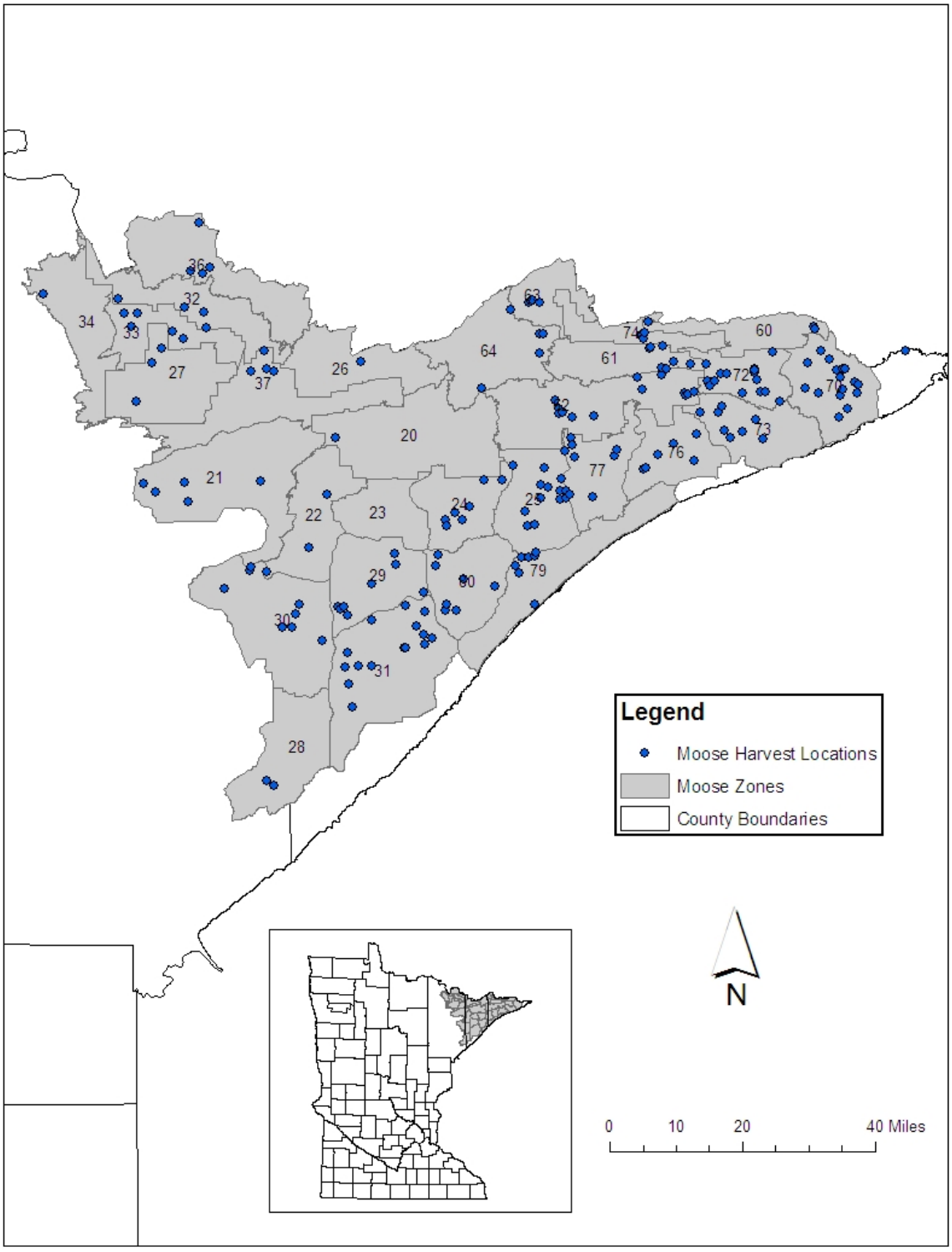

Figure 1. Harvest locations of hunter-harvested moose (*n*=197) included in 2010 and 2011 moose health assessment project, northeastern Minnesota.

MINNESOTA GRAY WOLF DISEASE SCREENING AND MORPHOLOGY

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SUMMARY OF FINDINGS

A total of 442 wolves (*Canis lupus*) were included in this 2-year study to document the apparent prevalence of diseases and parasites in Minnesota's wolf population, as well as provide insight into their genetic makeup. Our results indicated serologic exposure of wolves to 8 diseases: canine parvovirus (72%), canine adenovirus (79%) canine distemper virus (18%), eastern equine encephalitis (3.1%), West Nile virus (32%), heartworm (6.2%), Lyme (76%), and neosporosis (84%). Parasites were discovered in 15% of fecal samples examined. Genetic analyses are pending.

INTRODUCTION

Minnesota's gray wolf population was delisted from the Endangered Species Act in January, 2012. Following that ruling, wolves are managed in Minnesota by state statute, rule, and under the wolf management plan (2001) by the Minnesota Department of Natural Resources (MNDNR) This plan is designed to protect wolves and monitor the population while providing owners of livestock and domestic pets more flexibility in addressing wolf depredation. A primary component of wolf population monitoring is to understand what diseases and parasites might be impacting them. Furthermore, the collection of morphological and genetic data will add current and more spatially comprehensive data to the ongoing debates regarding the genetic identity of wolves in Minnesota.

There are a number of diseases and parasites known to affect wolves that can have population-level impacts. Most notably, relatively high prevalence of canine parvovirus (CPV) has been reported in Minnesota and could be adversely impacting pup survival and limiting population growth (Mech et al. 2008). Although further analysis indicated that the strongest effect of CPV and wolf population change occurred from 1987-1993 and after that had little effect despite higher seroprevalence levels (Mech and Goyal 2011). Other diseases, including canine distemper, adenovirus, and parasites may also kill infected wolves and impact population performance. Furthermore, some diseases, such as neosporosis, are of particular concern to livestock producers; gaining a more thorough understanding of the prevalence and distribution of this disease may benefit wolf management strategies.

There is some uncertainty in the taxonomic and genetic identity of wolves in the Great Lakes Region (Leonard and Wayne 2008, Mech 2008, Koblmuller et al. 2009, Nowak 2009, Schwartz and Vucetich 2009, Wheeldon and White 2009, Mech 2010, vonHoldt et al. 2011). Mech has suggested that non-genetic data support that wolves in Minnesota are hybrids between the gray wolf (*Canis lupus*) and the Eastern wolf (*Canis lycaon*) (2011). This portion of the project will systematically assess both genetic and morphological characteristics of a large sample of wolves in Minnesota. Relating wolf morphology to genetics should help determine the taxonomic identity of wolves throughout Minnesota, and reveal any potential geographic patterns of species introgression. The December 2011 federal delisting rule addressed this issue and determined that wolves in the Western Great Lakes are predominantly gray wolves with some admixture of either coyote or eastern wolf and that there is not sufficient evidence to suggest that there was a significant proportion of the population representative of the purported eastern wolf.

METHODS

The MNDNR entered into a contract with the United States Department of Agriculture (USDA)-Animal and Plant Health Inspection Service (APHIS)-Wildlife Services (WS) to collect biological samples from all dispatched wolves immediately after death. Researchers from the MNDNR, the United States Geological Survey (USGS), Camp Ripley military base, and tribal authorities that capture and radiocollar wolves also were involved in sample collections. Conservation officers and Area Wildlife staff assisted in collecting samples from vehicle-killed wolves. All key personnel were trained in proper sample collection and handling, as well as recording morphological measurements. Sampling kits provided to data collectors included the following items: soft-sided cooler, 1-20cc syringe for blood collection, 6-10-cc serum tubes for blood storage, 1-5-cc EDTA tube for whole blood, 1 whirlpak for fecal collection, 1 ear punch, 1 FTA card, 1-2-mL vial with 95% ethanol, 1-2-mL vial with desiccant, tape measure, caliper, data sheet, protocol, Sharpie, 1 pair of large vinyl gloves, and 1 icepack.

Our goal was to collect samples from wolves throughout the extent of their range in Minnesota; however, the vast majority of samples were collected by USDA-WS with an expected bias toward depredating wolves. Opportunistic sampling (e.g., vehicle kills) was encouraged to help increase sample size and provide a better distribution in more remote areas within wolf range.

Blood was collected from the jugular vein whenever possible (cephalic vein or saphenous vein are also options). For *post-mortem* collections, blood was obtained from the site of a bullet wound, heart, or from the chest cavity as soon after death as possible. In all cases, blood was centrifuged and serum extracted. Whole blood samples were kept cool and sent to an entomologist at the University of Minnesota for tick-related disease research. Fecal samples were collected from the rectum and placed in a whirlpak bag. Heart and brain samples were also collected from dead wolves when possible.

Sera were screened for 8 diseases at the Veterinary Diagnostic Laboratory at the University of Minnesota (UMN-St. Paul) and the National Veterinary Services Laboratory (Ames, Iowa). The presence of CPV was confirmed using a hemagglutination inhibition (HI) test; titers ≥256 were considered positive. Exposure to canine adenovirus 1 (CAV 1) was confirmed using a serum neutralization test (SN); titers ≥8 were considered positive. Canine distemper virus (CDV) was also detected using a SN test; titers ≥25 were considered positive. A plaque reduction neutralization test (PRNT) was used to confirm exposure to eastern equine encephalitis (EEE) and West Nile Virus (WNV), and titers ≥10 were considered positive. Heartworm disease was detected by an antigen test. An immunofluorescence assay (IFA) was used for evidence of exposure to Lyme disease; titers ≥160 were considered positive. The MNDNR is collaborating with Dr. J. P. Dubey (USDA-Agriculture Research Service, Beltsville, Maryland) on a *Neospora* research project. Dr. Dubey used both a modified agglutination test (MAT) and a neospora agglutination test (NAT) on samples of serum, heart, brain, or feces to confirm neospora. A titer ≥25 on either the MAT or the NAT test was considered positive.

This was the final year of the two-year project. Our sampling goals were met as we intended to sample a minimum of 400 wolves and samples were distributed throughout wolf range. While we do intend to continue to sample live-caught wolves, the decision of whether or not we continue to sample dead wolves will be made once final analysis is complete.

RESULTS AND DISCUSSION

Samples from a total of 442 wolves (348 adults, 4 yearlings, 79 pups, and 11 of unknown age; 233 males, 201 females, and 8 unknown sex were included. These included wolves that were euthanized by USDS-WS (n = 255), live-caught research animals (n = 61), vehicle kills (n = 41), found dead (n = 79), and other (n = 6) (Fig. 1). Blood and fecal samples were not collected from wolves that had been dead for an extended period of time.

Serologic Disease Screening

Serological results indicated wolves were exposed to all 8 diseases included in our screening (Table 1). These tests only confirm past exposure, not current infection.

Our results indicated 72% of wolves have been exposed to CPV, which is similar to findings reported by Mech and Goyal (2011) for northeastern Minnesota. Canine parvovirus was first reported in 1967, but it wasn't until 1978 that a new variant of the virus was reportedly killing a high number of newborn wolf pups. It was theorized that this new variant of CPV was a mutation from feline parvovirus. This disease can infect most age classes of canids; however, mortality related to CPV in domestic canids has been primarily associated with younger animals (1–12 weeks of age). Mech and Goyal (2011) evaluated 35 years of relationships between pup survival, population change, and CPV seroprevalence in NE MN. They found the population effect of CPV was temporary, with the strongest effect on pup survival and wolf population change from 1987 to 1993. Following this time frame, little effect was reported and the authors concluded CPV became endemic and the population had acquired enough immunity to negate impacts of infection.

Canine parvovirus is transmitted through the fecal-oral route and causes diarrhea, fever, and dehydration. The disease can be fatal to wolves and is suspected of causing declines or attenuation of wolf populations in Wisconsin (Wydeven et al. 1995) and on Isle Royale, Michigan (Peterson et al. 1998).

Prevalence of CAV1 (78.6%) in wolves in our study was less than the 96% reported in Yellowstone's adult wolf population (Almberg et al. 2009). Canine adenovirus 1 causes hepatitis, a disease of the liver and other body organs. The virus is found worldwide and is spread by body fluids including nasal discharge and urine. Canids of any age are susceptible to the disease. The incubation period is from 6 to 9 days, and signs include fever, loss of appetite, congested mucous membranes, and pain in the region of the liver. Reported mortality in dogs (*Canis familiaris*) is about 10%, and about 25% of the survivors develop a temporary corneal opacity (hepatitis blue eye). Chronic infection may occur, leading to cirrhosis of the liver. It remains unclear how endemic CAV 1 infection might impact wolf populations.

Wolves in Minnesota showed similar exposure to CDV (18%) as Spanish wolves (19%, Sobrino et al. 2007). Canine distemper virus is a *Morbillovirus* that infects a broad class of canids. Animals acquire CDV through inhalation or ingestion of airborne particles (Murray et al. 1999), and clinical signs include pneumonia, encephalitis, and death. Since CDV occurs in several carnivore taxas, there is concern about horizontal transmission among species. Outbreaks of CDV in 1999, 2002 and 2005 in free-ranging wolves within Yellowstone National Park were correlated with high pup mortality rates (Almberg et al. 2009). The CDV appears to be capable of causing dramatic population declines over a short time- frame.

Eastern equine encephalitis is a member of the genus *Alphavirus* in the family *Togaviridae*, which has been a source of epizootics in both domestic and wild animals since the 19th century. Outbreaks are typically concentrated around swampy areas and have been found primarily in the southeastern U. S., but also in Michigan and Wisconsin. Transmission by mosquitoes is thought to be the primary source of exposure; however, direct contact with contaminated blood, feces, vomitus, semen, or assassin bugs also can be a source of infection. Clinical signs vary depending on the species. Little is known about EEE infection in wolves; however, the disease has been documented in domestic dogs (Farrar et al. 2005). Clinical signs in dogs were described as including pyrexia, depression, nystagmus, and lateral recumbency. Farrar et al. (2005) concluded that primarily young dogs are the most susceptible to EEE. This disease had not been known to occur in Minnesota prior to the MNDNR's moose health assessment project initiated in 2007, which discovered 6% of moose (*Alces alces*) in northeastern Minnesota have serological evidence of exposure to EEE (Butler et al. 2010). Our findings suggest northeastern wolves are also exposed to EEE, yet it is unclear what effect, if any, this may have on wolf survival.

West Nile virus is an avian virus that can cause fatal disease in some species of mammals, reptiles and birds. West Nile virus is an arbovirus in the *Flavivirus* genus of the family Flaviviridae. Until 1999, WNV was confined to the eastern hemisphere; however, it has since spread to North America and is now considered established in the U. S. and Canada. West Nile Virus is primarily transmitted by mosquitoes; 59 species are confirmed carriers in North America alone. A recent study of Minnesota's northeastern moose population found nearly 35% serologic prevalence (Butler et al. 2010), and their range overlaps with wolf range. While it remains unclear what effect WNV has on the nearly 32% of wolves that we documented were exposed to the disease, neurological signs have been reported from rare clinical cases in dogs and wolves. For example, a case of WNV was reported in a captive 4-month-old Arctic wolf pup (*C. lupus arctos*, Lanthier et al. 2004) and in a 3-month old wolf pup (Lichtensteiger et al. 2003). Both reportedly exhibited vomiting, anorexia and ataxia prior to death, which occurred 24–48 hours after the onset of neurological signs.

Results from 6.2% of wolves in our study indicated exposure to heartworm, which has been previously documented in Minnesota wolves by Mech and Fritts (1987). Mosquitoes are the major vector of dog heartworm, *Dirofilaris immitis*. Once the worms end up in a canine, they will mature and grow on the right side of the animal's heart and pulmonary arteries. Initial symptoms include detectable heart murmurs and pulse deficits. As the problem progresses, the animal's heart may become enlarged and if the infection becomes severe (up to 200 worms have been found in some animals), blood flow will be blocked. Heart failure may result from a major infection. Heartworm has not been reported in Canada or Alaska, as the mosquitoes that carry it prefer warmer climates.

Our findings indicated a significantly higher prevalence of Lyme disease (76%) than 2.5%, which was previously reported in wolves in Minnesota and Wisconsin (Thieking et al. 1992). Lyme disease is caused by the bacterium *Borrelia burgdorferi*, and can affect dogs, horses and humans. The disease was first discovered in New England in 1975, and has since been reported in at least 43 states and eastern Canada. Infection typically results from bites from infected *Ixodes scapularis* ticks (deer ticks). White-tailed deer (*Odocoileus virginianus*) are the major hosts for the mature ticks, whereas small rodents are the hosts for the immature ticks. These hosts can become infected with *B. burgdorferi*, but never show symptoms of the disease. Wolves in Minnesota and Wisconsin have been found to be infected with the disease, but clinical Lyme disease has not yet been found in wild wolves. A wolf was experimentally infected with *B. burgdrferi* and showed some symptoms of the disease (lymphadenopathy), which suggests that wolves may be susceptible to it (Thieking et al. 1992).

Samples from 239 wolves were submitted for Neospora testing; however, testing hasn't been completed on all the samples. To date, 128 have tested positive. *Neosporas caninum* is a protozoal parasite, which is best known for causing abortion in cattle and neurological disease in dogs. While wild herbivores and canids were thought to act as intermediate and definitive hosts, respectively (Gondim 2006, Dubey et al. 2009), findings originating from this research project confirmed the role of wolves as a natural definitive host (Dubey et al. 2011). While clinical disease due to infection is best described in domestic animals, reports of ill-effects due to *Neospora* infection in wildlife do exist. Gondim et al. (2004) reported that *N. caninum* antibody seroprevalence was detected in 39% of free-ranging gray wolves, 11% of coyotes (*Canis latrans*), 26% of white-tailed deer, and 13% of moose. These data are consistent with a sylvatic transmission cycle of *N. caninum* between cervids and canids. The authors speculated that hunting by humans favors the transmission of *N. caninum* from deer to canids, because deer carcasses are usually eviscerated in the field. Infection of canids, in turn, increases the risk of transmitting the parasite to domestic livestock.

Fecal Parasitology

Fecal samples were collected from 161 wolves and were examined by floatation for any evidence of ova or protozoal infection. Twenty (12 %) of the samples had hookworm ova, 9 (6%) had trematode ova, 41 (25%) had sarcocysts, and 2 (1%) were positive for Neospora. While this provides an idea of the types of parasites present in the wolf population, it does not provide an indication of parasite load or infection rate, as fecal-shedding does not correlate with severity of infection and shedding is often cyclical (Gondim 2006).

Wolves are susceptible to a variety of internal and external parasites. These include at least 24 species of nematodes (roundworms), 21 species of cestodes (tapeworms), 9 species of trematodes (flukes), heartworms, and 3 species of acanthocephalia (spiny-headed worms).

General Tick-borne Illness Screening

A total of 194 blood samples were submitted to the Department of Entomology (UMN), where we are collaborating with Dr. Ulrike Munderloh, to determine if wolves are infected with tick-borne illnesses. Whole blood samples were screened with a variety of polymerase chain reaction (PCR) techniques, which determine disease infection, not just disease exposure (which is detected through serology). Preliminary results from 38 of the 194 samples indicate that 7.9% of the wolves were infected with Anaplasmosis, 40% were positive for prioplasma primers, and 5.3% were infected with Lyme disease. Further analysis is pending.

Morphology and Genetic Analysis

Although 298 skulls have been collected for taxonomic evaluation, presently, only about 75% have been cleaned. We have initiated a collaboration for preparing and curating skulls with Dr. Sharon Jansa at the UMN Bell Museum of Natural History. As collection skulls are prepared for storage, measurements will be made per the protocol described by Nowak (1995). Each skull will be permanently cataloged in the mammal collection at the Bell Museum.

Genetic samples have been collected from 386 wolves. A subset (n = 150) have been submitted to the National Wildlife Forensics Laboratory for analysis, as in Fain et al. (2010). Results are pending. New information has been presented in vonHoldt et al. (2011), which indicates wolves in Minnesota are predominantly gray wolves with admixture from coyotes that dates between 600–900 years ago. However, different sources have presented competing information about the genetic identity of wolves in Minnesota; consequently, additional analyses may be required to enhance our understanding of their genetic makeup. Further, analysis of how skull morphology correlates to genetic identification may also contribute to our understanding of the taxonomic relationships of wolves in the region.

ACKNOWLEDGEMENTS

There have been many people involved in obtaining samples for this study. We greatly appreciate the work of USDA-WS in their collection efforts, as well as MNDNR Area Wildlife and Enforcement staff. Further, samples have been provided by the USGS (S. Barber-Meyer and D. Mech), Fond du Lac Band of Lake Superior Chippewa (M. Schrage and L. Overland), 1854 Treat Authority (A. Edwards), Camp Ripley (B. Dirks and N. Dietz) and MNDNR Forest Populations and Research Group (C. Humpal and B. Sampson). We would like to acknowledge the efforts of our collaborators on diagnostics, Drs. J. P. Dubey (USDA-ARS), U. Munderloh (University of Minnesota), and A. Wuenchmann (Veterinary Diagnostic Laboratory, UMN). A. Ross (Duluth, Minnesota) was instrumental in the establishment of our dermestid beetle colony for skull cleaning. Finally, we thank R. Wright (MNDNR) for assistance with mapping needs.

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Table 1. Serological results for disease screening of wolves sampled in Minnesota, January 2010–March 2012.

Disease	*n*	No. positives	Apparent prevalence (%)
Canine parvovirus	190	136	71.6
Canine adenovirus	192	151	78.6
Canine distemper virus	194	34	17.5
Eastern equine encephalitis	193	6	3.1
West Nile virus	194	62	31.9
Heartworm disease	195	12	6.2
Lyme disease	195	148	75.6
Neospora*	239	128	54

*some test results are pending; collaboration with Dr. JP Dubey, USDA-ARS

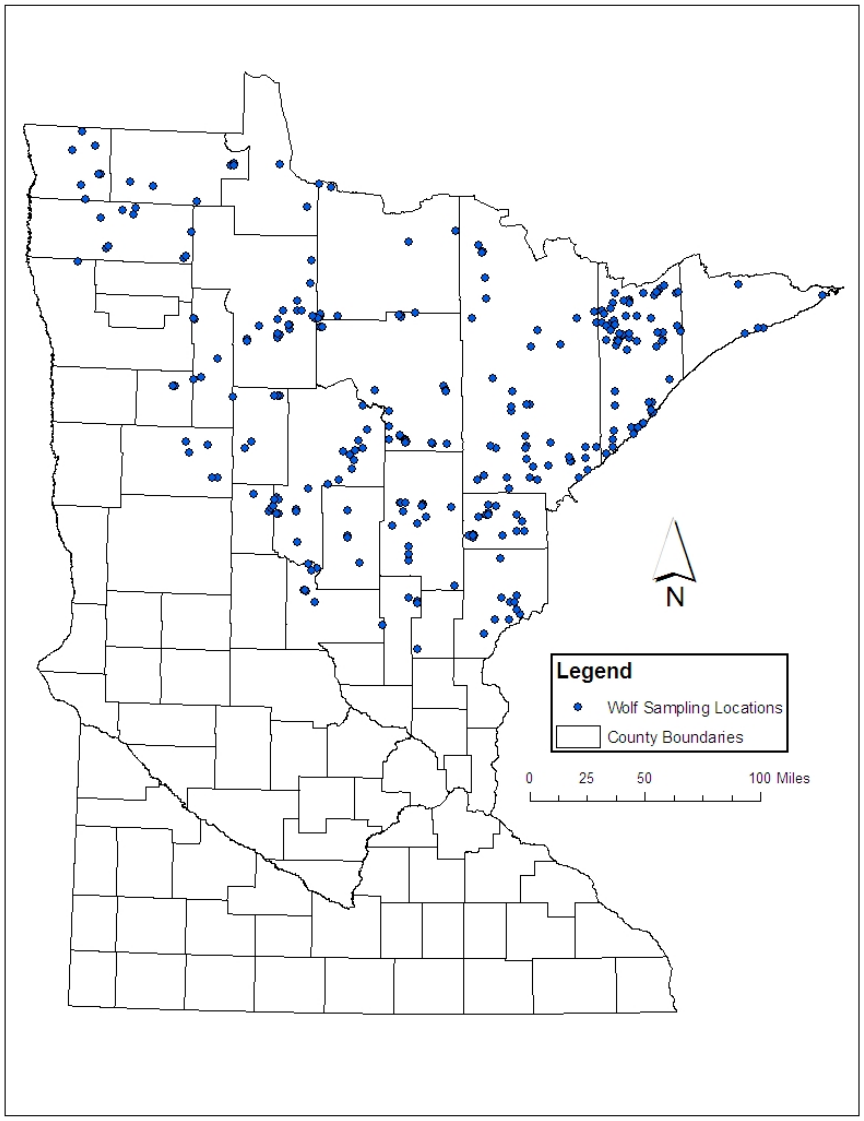

Figure 1. Sampling distribution of wolves (n = 442) included in the study of diseases and genetics of Minnesota's wolf population, 2010-2012.

CHRONIC WASTING DISEASE SURVEILLANCE IN MINNESOTA'S SOUTHEASTERN WILD DEER HERD

Erik Hildebrand, Michelle Carstensen, David Pauly, Erika Butler, and Lou Cornicelli

SUMMARY OF FINDINGS

In fall 2011, the Minnesota Department of Natural Resources (MNDNR) sampled 2,390 hunter-harvested white-tailed deer (*Odocoileus virginianus*) for chronic wasting disease (CWD) in southeastern Minnesota. The surveillance effort focused on testing deer within deer permit area (DPA) 602, and six surrounding DPAs: 233, 293, 341, 342, 343, and 344. All of the samples were negative for CWD. In addition, MNDNR submitted samples from 35 cervids through targeted surveillance, which included sick animals, escaped captive cervids, and roadkills; all these samples were also negative for the disease. The first and only detection of the disease in Minnesota's wild deer population occurred in a hunter-harvested deer from Olmsted county taken during fall 2010. To prevent further disease spread, MNDNR banned recreational feeding of deer in a 4-county area in southeastern Minnesota. MNDNR will continue to conduct CWD surveillance of hunter-harvested deer in fall 2012.

INTRODUCTION

To date, CWD has been diagnosed in 3 captive elk (*Cervus elaphus*) herds and 1 captive white-tailed deer herd within the state of Minnesota. Two of the elk herds (Stearns and Aitkin counties) were discovered in 2002 and depopulated; no additional CWD-positive animals were found. In spring 2006, a captive white-tailed deer from a mixed deer/elk herd in Lac Qui Parle County was discovered to be infected with CWD. That herd was also depopulated without additional infection being detected. In early 2009, a third captive elk herd (Olmsted County) was found infected with CWD and, following depopulation of >600 animals, a total of 4 elk were confirmed with the disease. The United States Department of Agriculture's (USDA) indemnification document noted there was an apparent longstanding infection within this captive elk facility.

Chronic wasting disease belongs to a family of infectious diseases, called transmissible spongiform encephalopathies (TSEs), which alter the morphology of the central nervous system, resulting in a "sponge-like" appearance of this tissue. Chronic wasting disease only affects elk, mule deer (*O. hemionus*), white-tailed deer, and moose (*Alces alces*). The etiological agent of CWD is an infectious protein, called a prion. Incubation time of the disease can range from 1.5 to nearly 3 years, although infected animals have been shown to shed prions in their feces up to a year before showing signs of illness (Tamguney et al. 2009). Clinical signs are non-specific and may include a loss of body condition and weight, excessive salivation, ataxia, and behavioral changes. There is no known treatment or vaccine for the disease and it is always fatal. Experimental and circumstantial evidence suggest that transmission of the disease is primarily through direct contact with infected animals or their infective saliva or excrement (Mathiason et al. 2006, Safar et al. 2008). However, persistence of prions in the environment and resulting indirect transmission has been shown to occur (Miller et al. 2004, Johnson et al. 2007, and Maluquer de Motes et al. 2008).

The Center for Disease Control (CDC) and other public health agencies have concluded there is no known link between CWD and any neurological disease in humans (MaWhinney et al. 2006). However, both the CDC and the World Health Organization (WHO) recommend that no part of a known positive animal should be consumed by humans. Additionally, there is no evidence that CWD can be naturally transmitted to species other than deer, elk, or moose.

Currently, Minnesota has approximately 644 domestic cervid facilities with approximately 20,000 deer, elk, and other cervidae in captivity. As the current statewide population estimate of wild deer approaches one million, there is an element of inherent risk associated with disease transmission between domestic and wild cervids. Overall, risk is difficult to quantify as deer populations are unevenly distributed across the landscape and range in densities from < 1 to 15 deer/km^2. In addition, domestic facilities are sporadically distributed on the landscape and are

mutually exclusive of deer densities.

In response to the discoveries of the first Minnesota CWD-positive captive elk herd in 2002 and CWD in wild Wisconsin white-tailed deer, the MNDNR developed a comprehensive wild deer CWD monitoring program. This included surveillance of targeted animals (e.g., suspect or potentially sick deer exhibiting clinical signs or symptoms consistent with CWD), opportunistic surveillance (e.g., vehicle-killed deer), and hunter-killed deer surveillance. During 2002–2004, nearly 28,000 deer were tested for CWD statewide with no positive results. Following completion of the statewide surveillance, the MNDNR scaled back surveillance efforts and sampled animals in response to elevated risk factors (e.g., detection of CWD-positive animals in captive cervid farms in Minnesota, or proximity of positive CWD cases in wild deer in neighboring states). From 2004 to 2010, an additional 5,700 hunter-harvested deer and over 540 targeted or opportunistic deer were tested for CWD, with no positives detected. Since discovery of our index case, MNDNR has enacted its CWD Response Plan (http://files.dnr.state.mn.us/fish_wildlife/wildlife/disease/cwd/cwdresponseplan.pdf), which indentifies 4 primary goals for managing the disease:

1) Determine and monitor the prevalence and geographic distribution of CWD in the infected area,
2) Prevent or minimize further spread and new introductions of the disease,
3) Support and conduct applied research on CWD and its epidemiology, and
4) Provide accurate and current information about CWD to the public, constituent groups, and agency personnel.

METHODS

Hunter-harvested surveillance was conducted at deer registration stations during the archery, firearm, and muzzleloader seasons within DPA 602. MNDNR also conducted hunter-harvested surveillance within the six surrounding DPAs during four weekends of the regular firearm season. Stations were staffed with MNDNR personnel and students (veterinary medicine and natural resources) trained in lymph node collection during the regular firearm season. Head collection boxes were placed within DPA 602 during the archery and muzzleloader seasons for area wildlife staff to collect necessary samples. All samples were inventoried, entered into a database, and sent to either the University of Minnesota's Veterinary Diagnostic Laboratory (St. Paul, MN) or to Colorado State University (Fort Collins, CO) for enzyme-linked immunosorbent assay (ELISA) testing. Any presumptive positive samples from ELISA testing would be confirmed using immunohistochemistry (IHC) testing at the National Veterinary Services Laboratory in Ames, Iowa.

During fall 2011, registration stations were selected based on deer volume and distribution throughout the surveillance zone to meet a sampling goal of 600 deer minimum within DPA 602 and 300 from each of the surrounding DPA's. At the time of sample collection, hunter information was recorded, including the hunter's name, a telephone number, MNDNR number, and location of kill. Maps were provided to assist the hunters in identifying the location (Township, Range, and Section) of the kill. Cooperating hunters were given a cooperator's patch and entered into a raffle to win a .50 caliber muzzleloader donated by the Minnesota Deer Hunters Association.

Within DPA 602, each registration station staffed for sample collection collected:

- All deer ≥1.5 years of age harvested by hunters were required to be sampled through extraction of retropharyngeal lymph nodes
- Both fawn and adult deer were issued carcass tags by authorized MNDNR staff
- MNDNR mailed samples from deer daily to University of Minnesota's Veterinary Diagnostic Laboratory in order to achieve a three business day turnaround time for results
- Carcasses were prohibited from being taken out of DPA 602 until they were test-negative

In the six surrounding DPA's, hunters were asked to voluntarily submit a retropharyngeal lymph node sample. Those samples were submitted on the Monday following each weekend

during the regular firearm season, with a 7-14 day turnaround time for results. For all samples, hunters were able to check their test results on the MNDNR website using either their MNDNR number or the carcass tag number they were issued at the time of sample collection.

MNDNR continued to sample deer exhibiting clinical symptoms consistent with CWD (targeted surveillance) statewide. Information has been disseminated to wildlife staff regarding what to look for regarding symptomatic deer. Staff were provided the necessary equipment and training for lymph node removal and data recording. The number of samples expected through targeted surveillance is estimated to be less than 100 animals annually, as few reports of sick deer are taken.

RESULTS AND DISCUSSION

During fall 2011, the MNDNR sampled 2,390 hunter-harvested deer within the surveillance area, of which 1,125 were within DPA 602 (Figure 1).

From May 2011 to May 2012, MNDNR collected a total of 35 samples from targeted surveillance efforts. This included samples from 7 escaped captive cervids, 25 free-ranging sick deer, 1 free-ranging elk, and 2 vehicle-killed deer; all samples were negative for CWD.

Another key step in preventing further spread of CWD was to ban the recreational feeding of deer in a 4-county area (Dodge, Goodhue, Olmsted and Wabasha), surrounding the location of the CWD-positive deer found in fall 2010 (Fig. 2). The ban was aimed at reducing the potential for the disease spread by eliminating artificially-induced deer concentration sites. MNDNR Enforcement staff continues to educate and enforce the rule.

Given the results of the CWD surveillance efforts of fall 2011, evidence suggests that Minnesota is on the front end of a CWD outbreak in wild deer. The lack of detecting any additional infected deer in the immediate vicinity of the index case or in surrounding DPAs is encouraging. It may be likely that this disease is recent on the landscape and that few individuals have been exposed. Continued surveillance will be necessary to monitor this outbreak and determine what additional management actions may be needed to prevent CWD from becoming endemic in southeastern Minnesota.

ACKNOWLEDGEMENTS

We would especially like to recognize the tremendous amount of work and commitment by M. Dexter, J. Dippel, K. Pilarski, C. Bagniewski, and A. Wolf for the massive surveillance effort this past fall. We would like to thank all the MNDNR Area Wildlife and Enforcement staff, who volunteered to assist with this project in response to this disease outbreak. We also thank the students and faculty from the University of Minnesota, Colleges of Veterinary Medicine and Natural Resources, for assisting in our fall sampling efforts. Also, a special thanks to J. -Hines and B. Wright for fulfilling our GIS and mapping needs, C. Austin for his efforts in constructing a website for hunters to check their results, and P. Takash for his efforts in communications and internet-related work. We appreciate the support of the USDA-Wildlife Services disease biologist P. Wolf and USGS National Wildlife Health Centers Wildlife Epidemiologist L. White.

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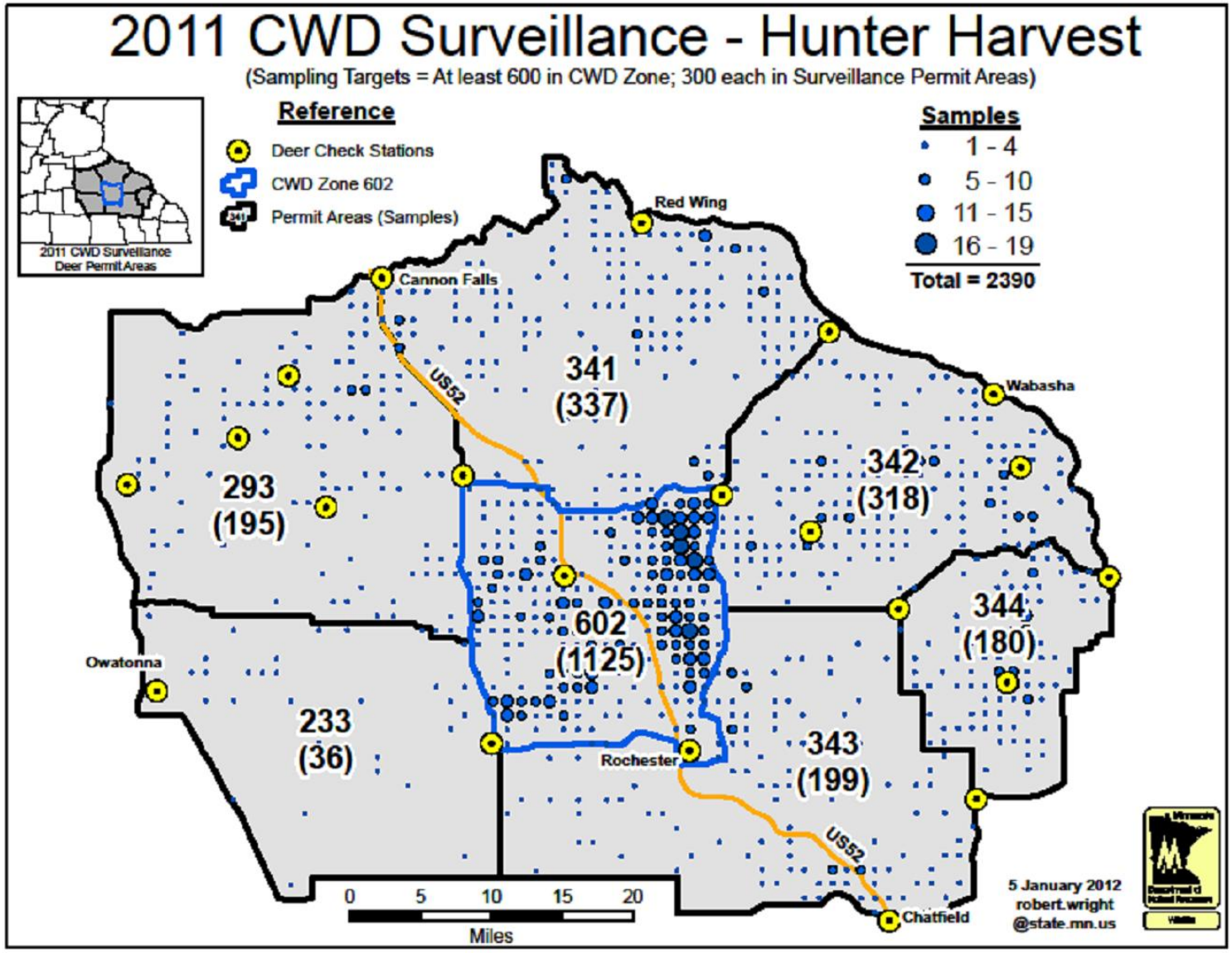

Figure 1. Sampling distribution of hunter-harvested deer (n=2,390) tested for chronic wasting disease in southeastern Minnesota, fall 2011.

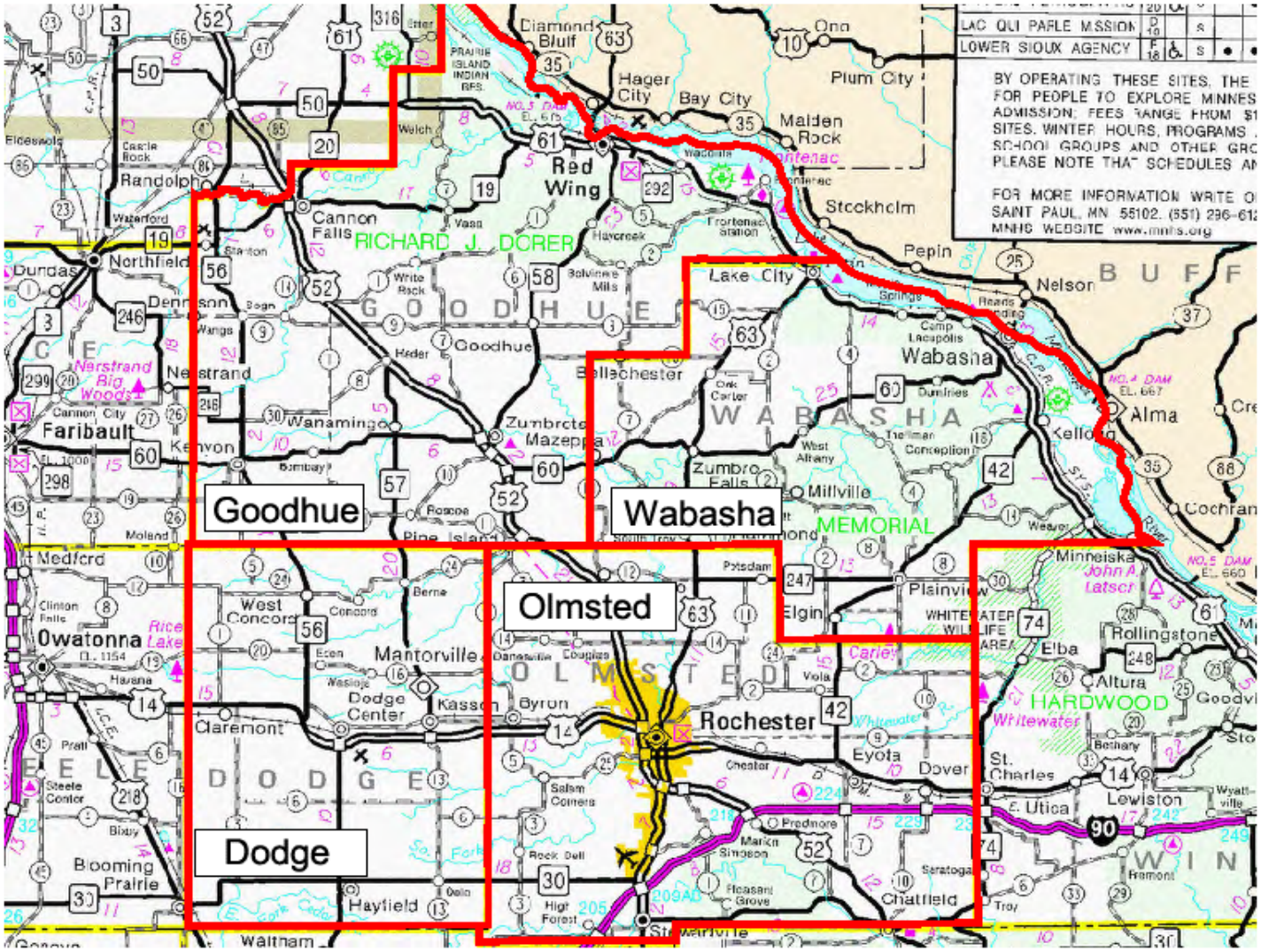

Figure 2. Four-county area in southeastern Minnesota where recreational feeding of wild white-tailed deer was banned in February 2011, following the discovery of chronic wasting disease in Olmsted County.

SPATIAL PATTERNS OF WHITE-TAILED DEER MOVEMENT RELATED TO BOVINE TUBERCULOSIS TRANSMISSION RISK IN NORTHWEST MINNESOTA

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SUMMARY OF FINDINGS

The goal of this pilot research study is to provide a better understanding of white-tailed deer (*Odocoileus virginianus*) movements and habitat use in the transitional landscape of northwestern Minnesota, where a recent outbreak of bovine tuberculosis heightened awareness of disease transmission risks between deer and cattle. In total, 21 deer (5 males, 16 females) were collared during this study and 10 deer (48%) remained alive until the planned collar blow-off date of April 15, 2012. Collar malfunctions occurred in 2 deer (10%), where no movement data were recorded. The overall mortality rate was 53% (n = 10), which was attributed primarily to wolves (n = 8, 80%), as well as hunter-harvest (n = 1, 10%) and unknown cause (n = 1, 10%). Mean home range size for deer (*n* = 9) surviving through the end of the study was 46.7 km^2 (SE = ±10.1). Seven deer were migratory, traveling 4–20 km to distinct winter ranges over 2-3 day periods. Deer visits occurred on 6 farms in the study area, with 1 farm accounting for 61% of the visits. Five deer accounted for all farm visits, including 2 deer visiting only one farm, 2 deer visiting two farms, and 1 deer visiting 3 farms. Over 75% of deer visits occurred in areas where cattle were present, either on a pasture or in an area with a feeding site and/or stored feed (hay bales). Most of the farm visits occurred during the spring (March through May) and primarily during the night (from 12am to 6am). This study provided baseline information regarding cattle-deer interactions critical to transmission of bTB in this region, and highlighted the potential for deer to function as vectors for disease transmission in transitional areas where habitat use between wildlife and livestock overlap.

INTRODUCTION

The Minnesota Department of Natural Resources (MNDNR) and the University of Minnesota (UMN) collaborated on a 15-month pilot study to gain a better understanding of movements and habitat use by white-tailed deer (*Odocoileus virginianus*) in northwest Minnesota. This is an area where continuous forest changes into a more agricultural landscape and deer use of this "transitional" habitat is not as well understood. The 2005 discovery of bovine tuberculosis (bTB) in wild deer in this area also increased concerns that a better understanding on how deer use such a diversified habitat is needed.

We were primarily interested in learning how deer use agricultural lands relative to state forest and wildlife management areas. In addition, we wanted to find out how farming practices, such as feed storage and animal husbandry, influenced deer use of agricultural lands. This project collected thousands of spatial locations of a small number of deer over the course of 15 months. By utilizing this information to improve our understanding of how deer may use farmed and pastured areas differently than natural habitats, we have gained insight into which practices may better minimize the risks of disease transmission between wild deer and cattle.

The UMN's Department of Veterinary Population Medicine previously developed a risk assessment process that was used by the Minnesota Board of Animal Health to evaluate the risk of deer and cattle interactions at farms within the bTB Management Zone (Knust et al. 2011). In this study, the UMN quantified the microhabitat use of deer on farms and the potential for bTB transmission among cattle and deer, and which herds are more at risk for deer-cattle interactions as a consequence of the farm management practices. Further, we hope to leverage the results obtained in this study with another ongoing study evaluating cattle

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movements in northwest Minnesota and potentially across the entire state. Combined, information generated from these studies should allow simulations of how bTB can spread across a network of farms, where disease is introduced by infected cattle and spread by deer as a transmission vector. The research should also provide a further understanding of steps that can be taken to mitigate these risks.

Secondarily, the location data ("fixes") stored on the radiocollars allowed MNDNR to estimate home range size and migration patterns for the study animals. Recognizing that the results will not adequately represent the larger deer population, our findings have provided wildlife managers and researchers with useful information and may help design a larger study in the future, should funding become available.

METHODS

The study area is approximately 360 km^2 and includes a mosaic of state forest and wildlife management lands, private recreational lands, and private farms (including row-crop agriculture, farmsteads, and stored forage). Included in the area are >25 farms with a variety of livestock and agricultural uses (Figure 1). The study area lies just outside the southern boundary of the bTB Management Zone and contains 2 formerly bTB-infected cattle farms; however, the disease has not been detected in wild deer in this area. Deer density ranged from 15-20 deer/km^2. Major predators include gray wolves (*Canis lupus*), black bear (*Ursus americanus*), coyote (*Canis latrans*), and bobcats (*Felis rufus*). Agricultural lands were surveyed to delineate and evaluate parameters (e.g., locations of stored forage, water sources, cattle pastures, etc.) that might attract deer to these areas.

In winter 2011, deer were captured by helicopter netgunning (Quicksilver Air, Inc., Fairbanks, Alaska) and Clover-trapped within the study area. Captured deer were chemically immobilized (100mg xylazine, 400mg ketamine HCl), and blood, urine, and fecal samples were collected for health screening. Methods for serological health screening were described in Carstensen et al. (2011). We also measured rump fat by ultrasound and extracted a last lower incisor to determine exact age by cementum annuli (Mattson's Laboratory, Milltown, Montana). Deer were ear-tagged and fitted with a satellite-linked radiocollar (ARGOS, SirTrack, Hawkes Bay, New Zealand). Body temperature was monitored at 5-min intervals throughout the processing period. A long acting antibiotic (LA-200, oxytetracycline) was administered intramuscularly (1 mL/10kg body weight). Before release, anesthesia was reversed by intravenous injection of 15mg/deer of yohimbine HCl. An observer monitored each deer's recovery and recorded the time deer were up and moving away from the recovery area.

Radiocollars were programmed to record locations every 90 minutes and transmit these "fixes" every 3 days through the ARGOS satellite system. Battery life of radiocollars is expected to be 15 months (to allow for one full year of seasonal movements). Collars were programmed to remotely discharge on April 15, 2012. The research team will retrieve all collars and download the complete set of spatial data. In the interim, fixes are downloaded weekly and examined for temporal and spatial movement patterns to determine mortality, movements, and habitat use. For any study animals that died during the study period, MNDNR wildlife staff investigated the cause of mortality, recovered the collar, and collected medial retropharyngeal lymph node samples from the deer (when possible) for bTB testing.

Deer movements and home range estimates were generated using Home Range Tools (HRT) for ArcGIS® (Rodgers et al. 2007). Minimum covex polygons (MCPs) were constructed by connecting peripheral points containing 99% of available fixes (White and Garrott 1990, Rodgers et al. 2007).

For evaluation of deer use on the agricultural landscape, a descriptive analysis was used to evaluate patterns of deer visits to farms during the entire study period. This will include the number of visits to each farm by season and time of day, number of farms visited by each individual deer, differences in use of farm areas by age and sex of deer, and variation in home range of each deer during the study period. Also, a resource utilization model will be developed

that compares characteristics of locations used by each deer to available locations that are not used; thus, identifying higher risk areas for deer locations based on resource availability.

RESULTS AND DISCUSSION

Deer Capture and Handling

In January 2011, 16 deer (4 males, 12 females) were captured by helicopter netgunning within (n = 11) and slightly northeast (n = 5) of the study area. Capture locations were driven by deer distribution at the time of capture and access to private land to process deer. Due to collar failure immediately following release, one deer (ID 519) was censored from the study because no GPS fixes were transmitted, although its collar was recovered by timed blow-off and the deer remained alive through the end of the study. By the end of February 2011, 3 deer were killed by wolves and one died from unknown causes (Table 1). To compensate for the high winter mortality so early in the study, the sample size was augmented with 5 deer (1 male, 4 females) captured using Clover-traps in March 2011 (Table 1). One of these deer (ID 577) was fitted with a test collar provided by SirTrack (Iridium satellite system prototype), and this collar failed to record or transmit locations immediately after the animal's release. Although this deer was censored from the study, it was killed by wolves in early April and the collar was recovered. A second deer (ID 447) from this group slipped its collar (likely caused by a premature expulsion of the blow-off device) on 22 May, 2011, and was subsequently censored from the study.

In total, 21 deer (5 males, 16 females; 6 yearlings, 15 adults) were collared during this study and 10 deer (48%) remained alive until the planned collar blow-off date of April 15, 2012. Collar malfunctions occurred in 2 deer (10%), where no movement data were recorded. The overall mortality rate was 53% (n = 10), which was attributed primarily to wolves (n = 8, 80%), as well as hunter-harvest (n = 1, 10%) and unknown cause (n = 1, 10%). The collars functioned well, as weekly satellite downloads of these animals obtained approximately one-third of recorded fixes (Table 2). This provided sufficient data to track major animal movements and monitor survival, yet preserves battery life by restricting the amount of time collars communicated with the satellite system. The timed blow-off mechanisms worked perfectly for the 10 deer that survived to the end of the study. The success rate of obtaining fixes was >97% for recovered collars (Table 3).

The number of mortalities we observed from February to April 2011, specifically due to wolf predation, was higher than expected. Winter conditions were moderately severe (WSI = 159, Red Lake Wildlife Management Area) in the study area, with prolonged snow cover of >14 inches from late-January through early April. In Minnesota's forest zone, DelGiudice et al. (2006) reported a 37% winter mortality rate for adult deer during the severe winter of 1995-1996 (WSI = 195), with wolves accounting for 63% of those deaths. During more moderately severe winters (WSI = 124 to 159) in north-central Minnesota, DelGiudice et al. (2006) reported winter mortality rates ranging from 7 to 19%, with wolf predation accounting for 50-80% of the deaths. In contrast, the winter mortality rate for adult female deer in Minnesota's farmland zone has been reported as only 5%; however, there is an absence of wolves and typically more mild winter conditions (Brinkman et al. 2004). Little information exists on winter mortality rates for deer in Minnesota's transition zone, and although the sample size was limited in this study, our preliminary findings suggest there might be some unique attributes in northwestern Minnesota that make deer population dynamics different that both farmland and forest zones. Interestingly, during winter 2012, a historically mild winter with (WSI ≤ 20, Red Lake Wildlife Management Area), none of the remaining study animals were killed by wolves.

Disease Screening and Parasitology

Serological screening of deer at capture for 9 common cattle diseases indicated exposure to bovine parainfluenza 3 virus (24%), malignant catarrhal fever (19%), and infectious bovine rhinotracheitis (9%). Fecal parasitology indicted 13 (65%) of deer had evidence of liver

fluke (*Fascioloides magna*) infection and strongyle-type ova was detected in 4 (20%) deer. Detailed discussion of these findings can be found in Carstensen et al. (2011).

Home Range Size and Deer Movements

Mean home range size for deer (*n* = 9) surviving through the end of the study was 46.7 km^2 (SE = ±10.1; Table 2, Figure 2). Deer that died (or slipped their collar) during the study had significantly smaller home range sizes to survivors (13.9km^2 ± 5.3; Table 3). This apparent difference in home range size might be due to the fact that surviving deer had more than 4x the number of days on the air, thus were tracking movements over a longer time period.

Given the timing of deer capture (mid-January and early March), we assumed these animals were on their winter range (if migratory) or possibly year-round residents at the start of the study. This was an incorrect assumption, as movements to distinct winter ranges didn't occur until late-January or February. Seven deer had home ranges >40km^2 and can be attributed to a few long-distance movements from one end of their range to the other. These movements began in late January for 5 deer, moving 4-20 km in a 2-3 day period. The other 2 deer moved 14-19 km in mid to late March, again over a 2-3 day period. Of these 7 deer, 2 were killed during winter, but the other 5 returned the same distance (in a 2-3 day period of travel) to the area they were originally captured in late March or early April. Interestingly, only 3 of the 5 surviving migratory deer returned to their winter ranges during the mild winter of 2012; however, the start of their movement was much later (late February-early March) and they returned to their spring-summer-fall ranges sooner (mid-March).

Brinkman et al. (2005) reported 78% of deer in Minnesota's farmland zone as migratory (43% obligate and 35% conditional migrators), with a mean migration distance of 10 km. Further, those authors determined mean winter and summer home ranges (95% MCPs) as 5.2km^2 and 2.6km^2, respectively. Conversely, forest zone deer in northeastern and north-central Minnesota were 89% and 68% migratory, respectively (Nelson 1995, Fieberg et al. 2008). Further, migration distances were typically 10-14 km, but ranged from 2-135 km; onset of migrations varied annually, but ranged from early November to January (Fieberg et al. 2008). In both studies of forest zone deer, severe winters coincided with a higher number of conditional migrators making movements to a distinct winter range (Nelson 1995, Fieberg et al. 2008).

Deer Use of the Agricultural Landscape

Data on location of cattle, feeding areas and stored feed were collected by ground-truthing farm landscapes at 4 different times (December 2010 – before the capture; June 2011, October 2011 and March 2012) for the 30 farms within the study area. The farms within the study area are mostly small beef cow-calf operations. Primary variables of interest included locations of cattle, stored feed, and feeding sites.

Results show that deer visits occurred in 6 farms in the study area, with 1 farm accounting for 61% of the visits (Figure 3). Five deer accounted for all farm visits, including 2 deer visiting only one farm, 2 deer visiting two farms, and 1 deer visiting 3 farms (Figure 3). Over 75% of deer visits occurred in areas where cattle were present, either on a pasture or in an area with a feeding site and/or stored feed (hay bales) (Figure 4). Most of the farm visits occurred during the spring (March through May) and in the month of October (although the latter was performed mainly by 1 deer at 1 farm) (Figure 5). Deer visits increased during the crepuscular period achieving its maximum during the night (from 12am to 6am) (Figure 5).

These study results provide baseline information regarding cattle-deer interactions critical to transmission of bTB in this region, and highlight the potential for deer to function as vectors for disease transmission. The large home ranges for many of the study deer overlapped with multiple farms. In this study, 3 deer visited more than one farm which increases potential for disease transmission. Currently, the surveillance system for bTB is not cost-effective in situations of low disease prevalence. Time from infection to detection is extremely long, with the potential for severe consequences in terms of the spread of disease to

other cattle herds and wildlife. Clearly there is need to improve both the sensitivity and cost-effectiveness of the surveillance system by detecting outbreaks faster and reducing the need for extremely costly control measures. When funding sources are allocated for such events, the resources need to be focuses toward the subset of the population that pose the highest risks. Further, the importance of risk mitigation and efforts to prevent of disease transmission between livestock and wildlife are often understated; enhancement and enforcement of appropriate biosecurity measures should be a priority within the agricultural community.

ACKNOWLEDGMENTS

We would like to thank all the folks involved in the deer capture event in subzero conditions, including DNR Wildlife staff: Eric Nelson, Barry Sampson, Erika Butler, Erik Hildebrand, Rick Beito, Emily Dunbar, Mike Hallie, Carolin Humpal, Dawn Torrison, Scott Laudenslager, Arlyn Anderson, Neil Mattson, Donovan Pietruszewski, and Mary Reiswig; UMN's Bryant Carlson and Karin Hamilton; DNR Conservation Officers and Pilots: Angie Warren, Jeremy Woinarowicz, Don Murray, Tom Pfingsten, and Tom Buker; GIS support: Chris Scharenbroich; DNR Communications: Pete Takash, and SirTrack's Chris Kohanny. We also thank the Clover-trapping crew of Dave Kuehn, Erik Hildebrand, Emily Dunbar, and Eric Walberg. We appreciate the support from landowners Mark Sax and Tom Roen for permission to process deer on their properties.

Funding for this study included: USDA NIFA, MN Department of Natural Resources, Minnesota Rapid Agricultural Response Fund, and the Academic Health Center Faculty Research Development Program.

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Table 1. Current status and fate of free-ranging white-tailed deer (*n* = 21) captured and radiocollared in January and March 2011, northwest Minnesota.

Deer ID	Capture Date	Method	Age Class	Age[1] (yr)	Sex[2]	Fate	Cause	Estimated Mortality Date
469	1/15/11	Helicopter	Adult	4.5	F	Alive	collar blow-off	
461	1/15/11	Helicopter	Yearling	1.5	F	Dead	wolf-kill	3/31/11
497	1/15/11	Helicopter	Yearling	1.5	F	Alive	collar blow-off	
467	1/15/11	Helicopter	Yearling	1.5	M	Dead	wolf-kill	2/18/11
466	1/15/11	Helicopter	Adult	8.5	F	Alive	collar blow-off	
496	1/15/11	Helicopter	Adult	2.5	F	Dead	unknown	2/23/11
472	1/15/11	Helicopter	Adult	5.5	F	Alive	collar blow-off	
524	1/15/11	Helicopter	Adult	6.5	F	Dead	wolf-kill	3/10/11
473	1/15/11	Helicopter	Adult	4.5	M	Dead	unknown	7/28/11
495	1/15/11	Helicopter	Adult	2.5	M	Alive	collar blow-off	
471	1/15/11	Helicopter	Yearling	1.5	F	Dead	wolf-kill	4/5/11
491	1/16/11	Helicopter	Yearling	1.5	F	Alive	collar blow-off	
348	1/16/11	Helicopter	Adult	9.5	F	Dead	wolf-kill	2/12/11
460	1/16/11	Helicopter	Adult	2.5	F	Dead	wolf-kill	2/10/11
519	1/16/11	Helicopter	Adult	3.5	M	Alive	collar blow-off, malfunction[3]	
350	1/16/11	Helicopter	Adult	11.5	F	Alive	collar blow-off	
336	3/7/11	Clover-trap	Yearling	1.5	M	Dead	hunter-harvested	11/5/11
578	3/8/11	Clover-trap	Adult	4.5	F	Alive	collar blow-off	
577[4]	3/8/11	Clover-trap	Adult	11.5	F	Dead	wolf-kill	4/10/11
579[5]	3/8/11	Clover-trap	Adult		F	Alive	collar blow-off	
447	3/10/11	Clover-trap	Adult	3.5	F	Unknown	slipped collar	

[1]Age (in years) at capture was determined by cementum annuli. Analysis for adult deer captured in March is pending.
[2]F = female, M = male
[3]Deer 519's collar failed to transmit immediately after capture. No location data were obtained for this deer; however, it did survive through the study period.
[4]Deer 577 was fitted with a SirTrack test-collar (Iridium satellite system) and no movement data was recovered; mortality date is based on a public report of a severely injured deer and carcass remains.
[5]Deer 579 was unable to be aged due to a broken tooth with missing cementum.

Table 2. Fix success rates and home range size of free-ranging deer (*n* = 9) surviving through the end of the study, April 15, 2012, northwest Minnesota.

Deer ID	Days on Air	No. Fixes[1]	Fix Success Rate[2] (%)	99% MCP[3] (km^2)
469	457	7819	99.5	68
497	457	7717	98.3	92
466	457	7811	99.4	3
472	457	7772	98.7	33
495	447	7366	97.9	20
491	456	7785	99.0	76
350	456	7831	99.4	64
578	405	6909	98.0	46
579	405	7024	99.5	18
Mean	444	7559	98.9	46.7
SE	7	122	0.2	10.1

[1]Total number of fixes included only data downloaded from the satellite system from deployment through June 14, 2011.
[2]Fix success rate was calculated by number locations received through the satellite divided by the number of available locations, assuming collars recorded 16 locations/day.
[3]MCP = Minimum Convex Polygon, contained 99% of all locations.

Table 3. Fix success rates, and home range size of free-ranging deer (*n* = 10) that had either died or slipped their collar during the study.

Deer ID	Days on Air	No. Successful Fixes[1]	No. Failed Fixes	Success Rate (%)	99% MCP[2] (km^2)
461	77	1325	8	99.4	40.1
467	43	774	7	99.1	1.0
496	43	773	17	97.8	0.5
524	61	1124	4	99.6	8.0
471	90	1693	82	95.2	10.4
348	28	517	13	97.5	47.9
460	43	763	24	96.8	0.3
447	89	1641	68	95.9	4.1
473	200	3271	167	95.1	10.0
336	244	4807	125	97.5	17.0
Mean	92	1669	52	97.4	13.9
SE	23	429	18	0.5	5.3

[1]Total number of successful fixes included all data from deployment until collar was recovered from the field, which extended beyond the estimated mortality dates.
[2]MCP = Minimum Convex Polygon, contained 99% of all locations.

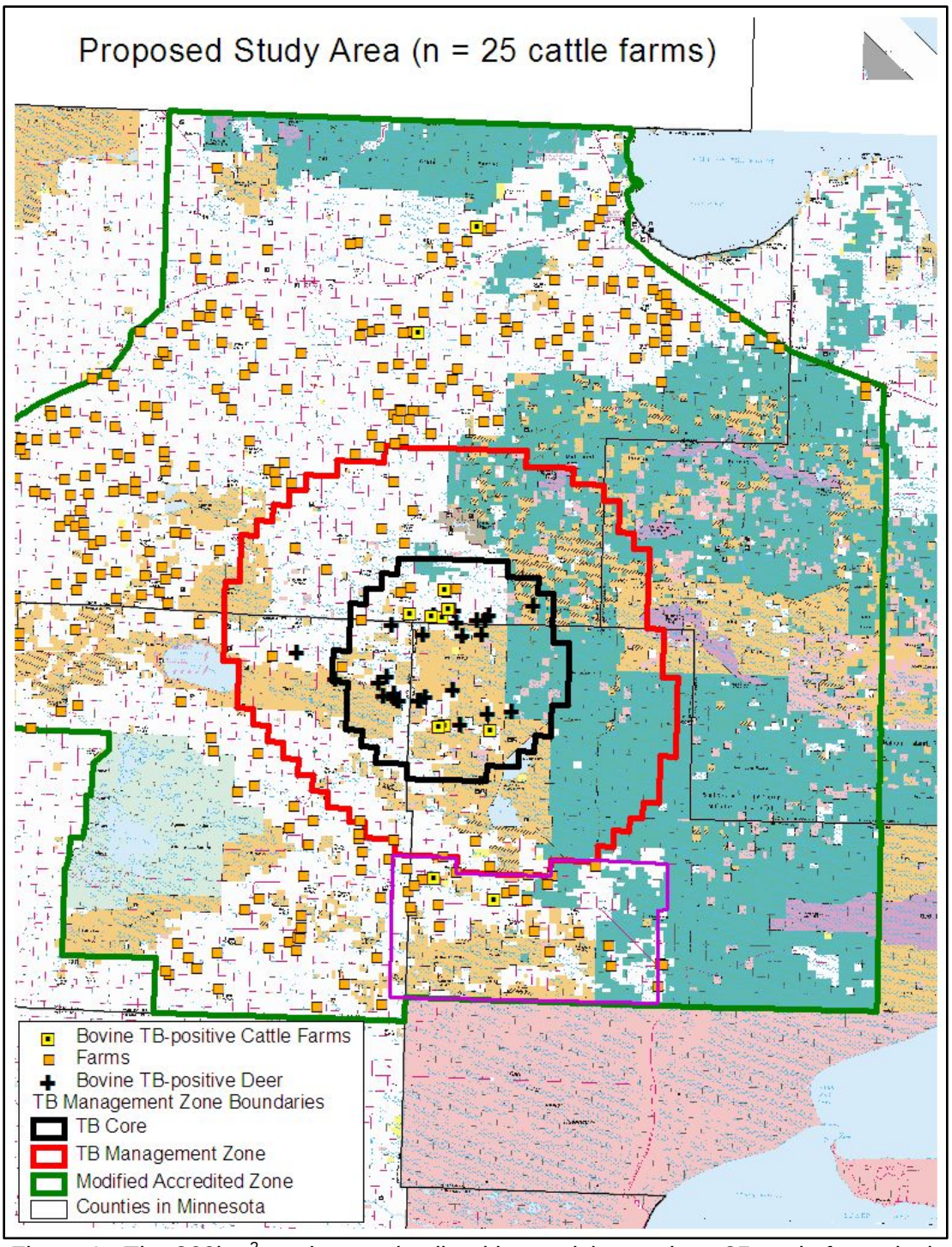


Figure 1. The 360km^2-study area (outlined in purple) contains >25 cattle farms including 2 previously infected with bovine tuberculosis. The study area is immediately south of Bovine Tuberculosis Management Zone, where 27 deer and 8 cattle farms tested positive for the disease.

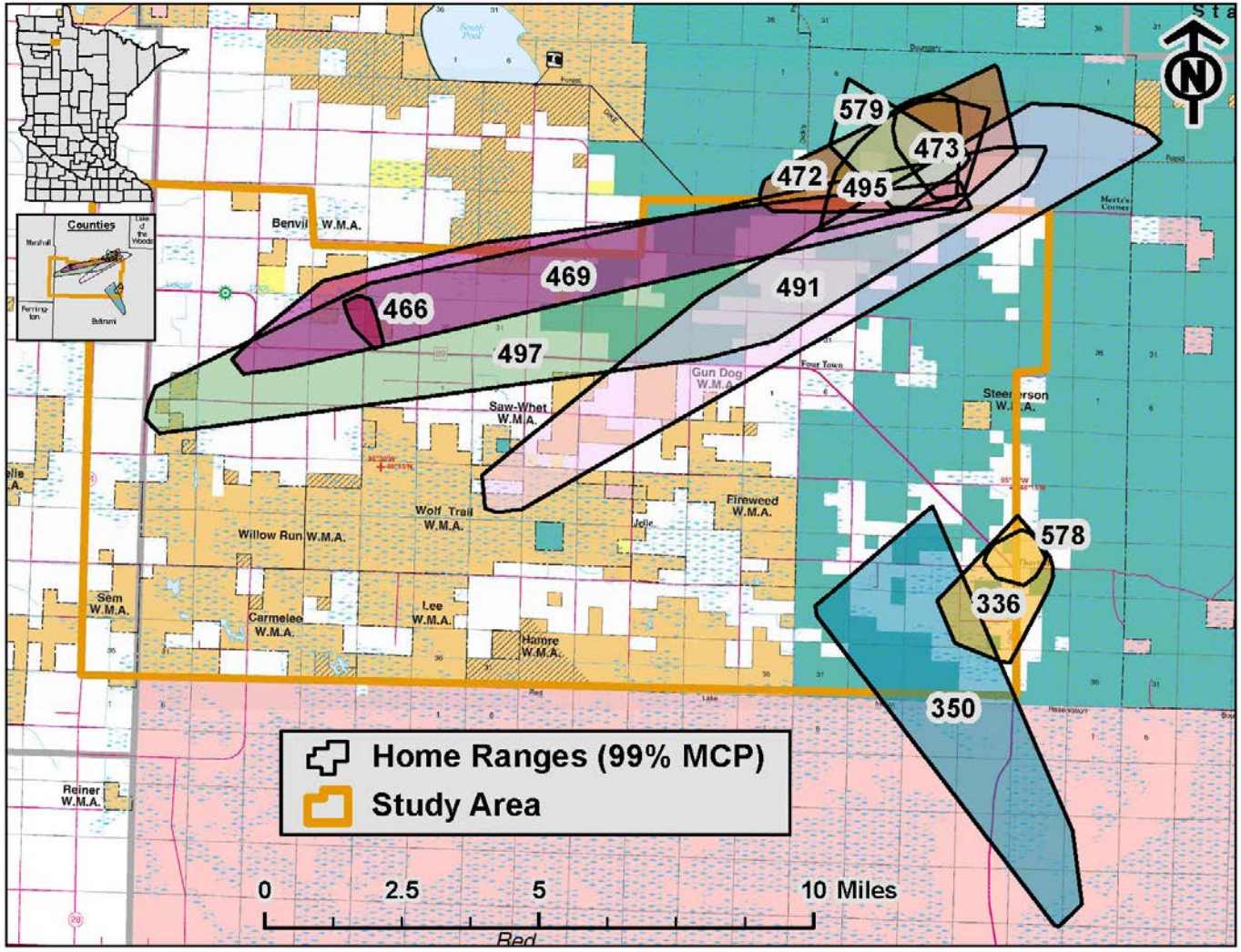


Figure 2. Home ranges, determined by 99% minimum convex polygons, for white-tailed deer (*n* = 11) that survived ≥ 200 days of the study, January 2011–April 2012, northwest Minnesota.

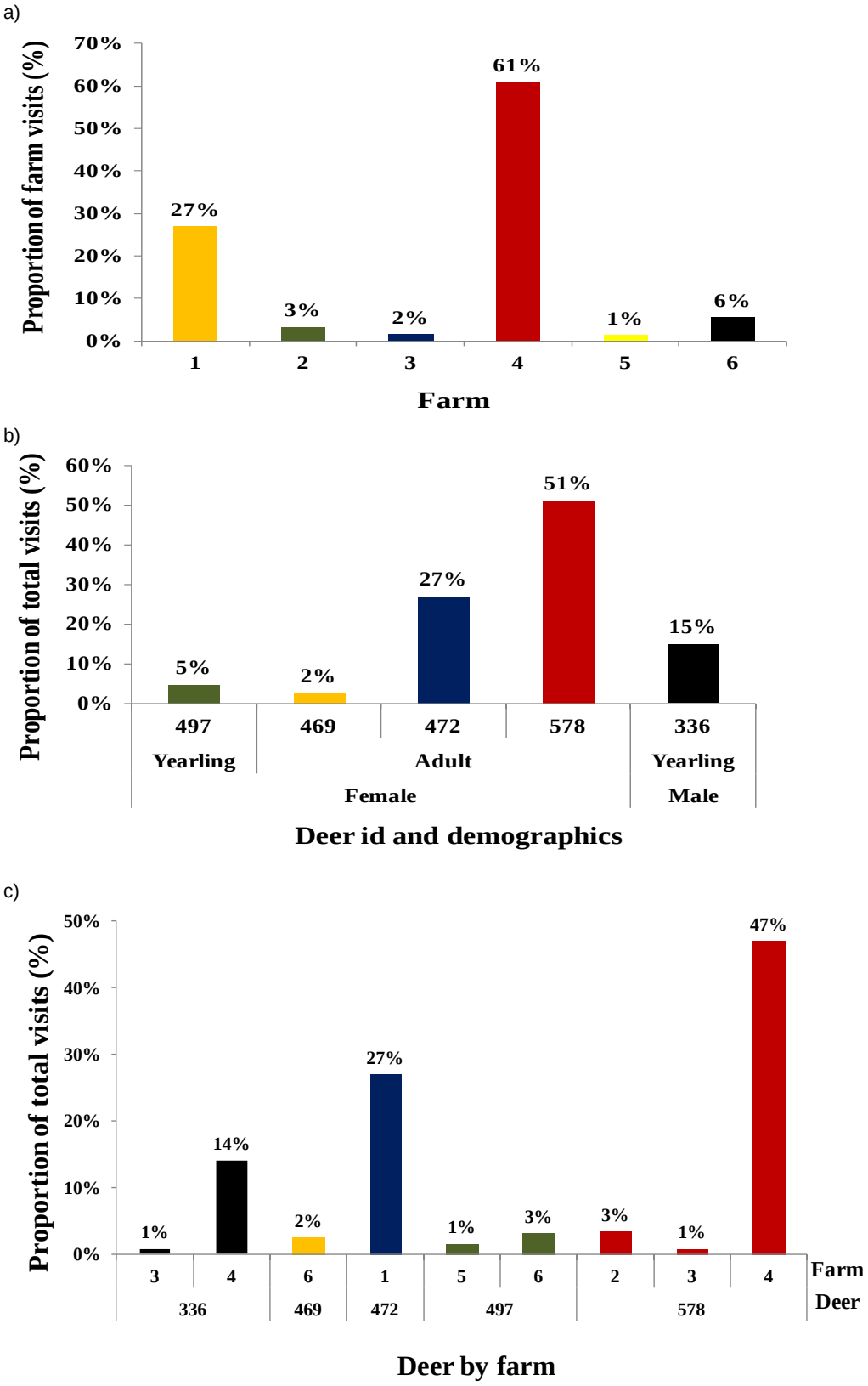


Figure 3. Six farms within the study area received visits by radio-collared deer during the study (a), the age/sex composition of those deer (*n* = 5), and the proportion of total visits by those deer per farm (c), northwest Minnesota.

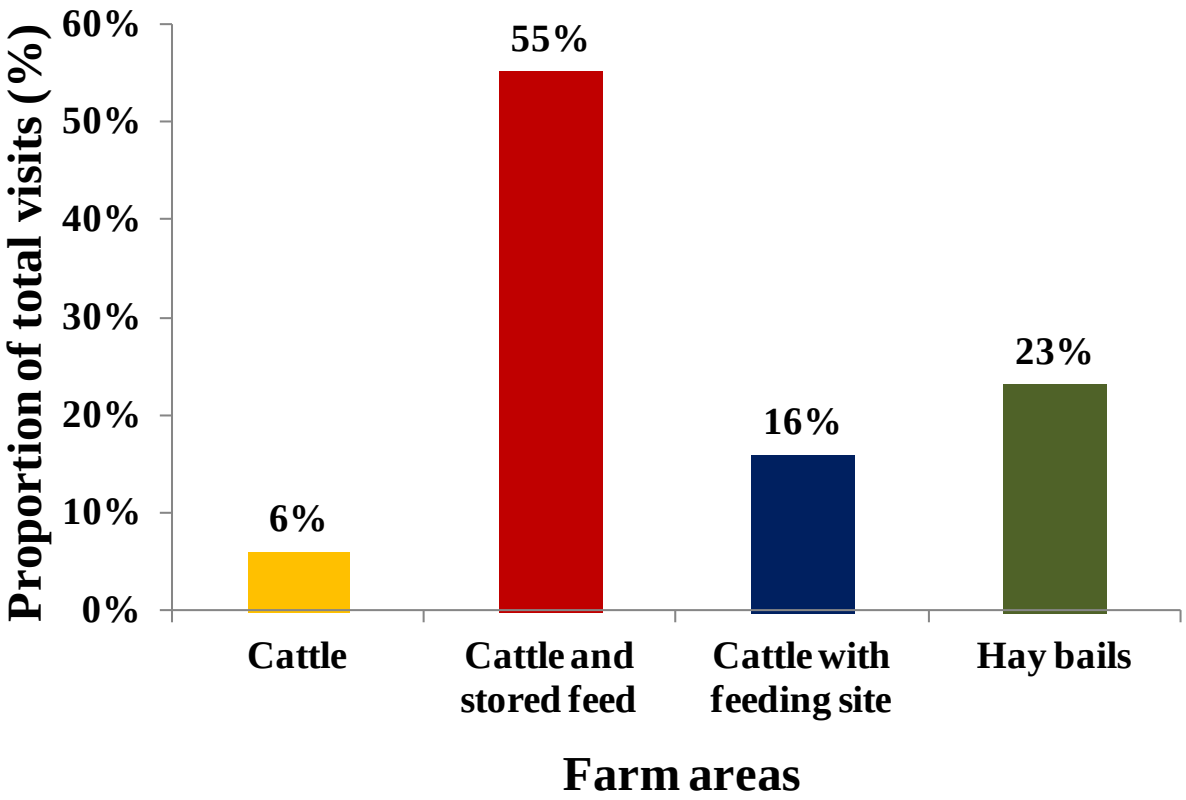
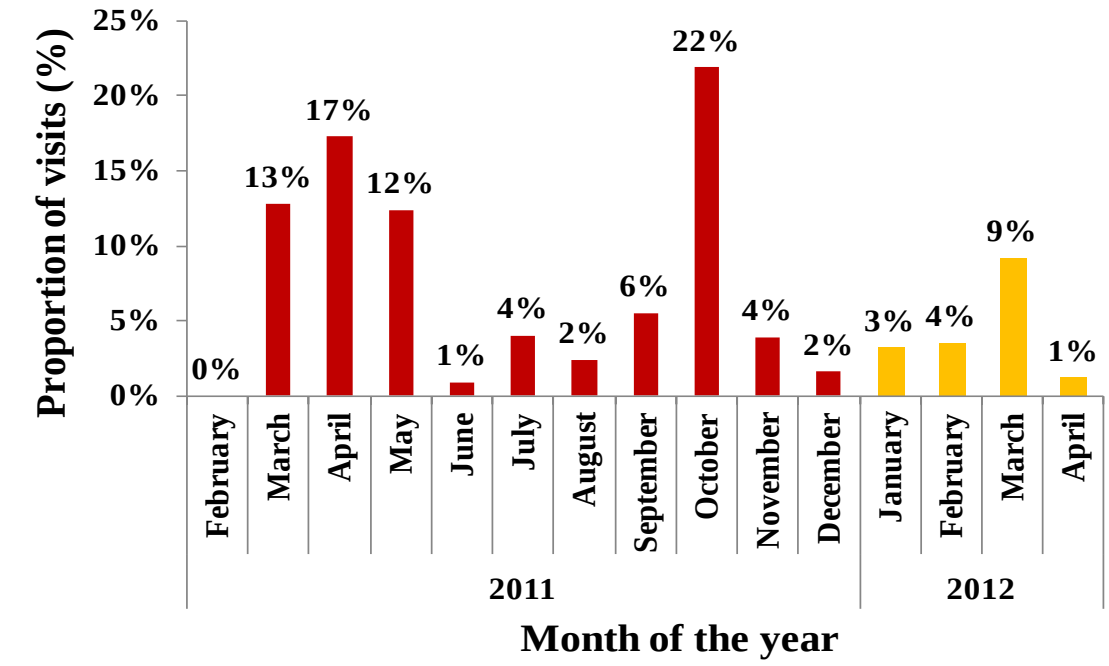


Figure 4. Attributes of farms within the study area and the proportion of their use by radio-collared Deer, January 2011–April 2012, northwest Minnesota.

a)



b)

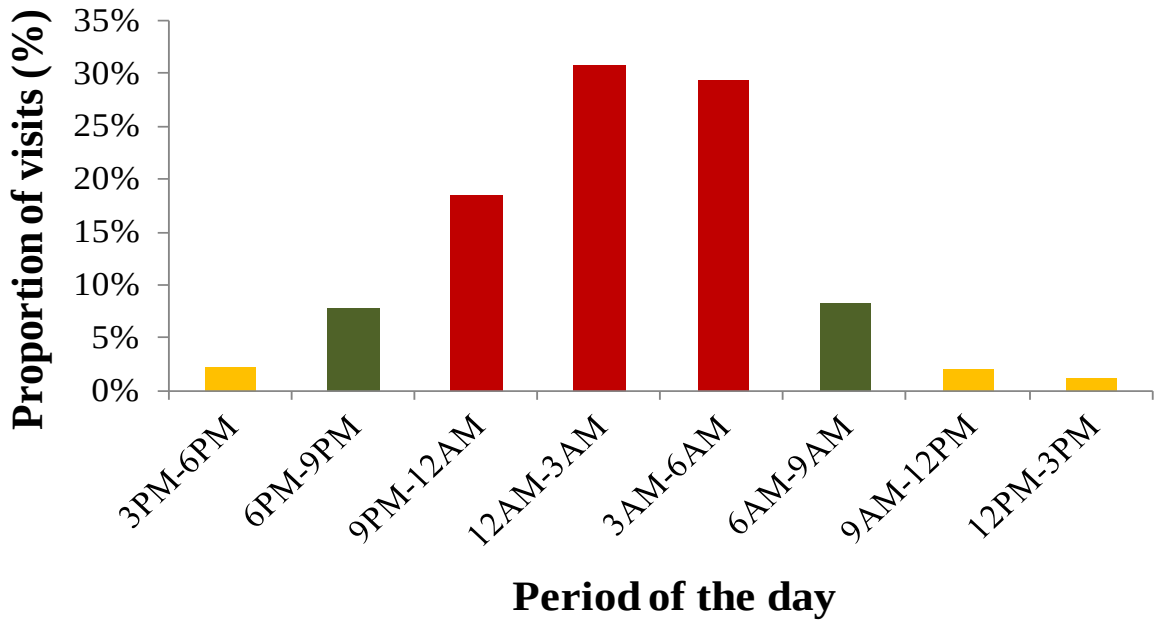


Figure 5. Monthly (a) and daily (b) use of farms by radio-collared deer (*n* = 19) from January 2011–April 2012, northwest Minnesota.

MANAGING BOVINE TUBERCULOSIS IN WHITE-TAILED DEER IN NORTHWEST MINNESOTA: A 2011 PROGRESS REPORT

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SUMMARY OF FINDINGS

A total of 561 hunter-harvested white-tailed deer (*Odocoileus virginianus*) were tested for bovine tuberculosis (bTB) in northwest Minnesota during fall 2011, with no positive cases detected. This marked the 7th consecutive year that the Minnesota Department of Natural Resources (MNDNR) has conducted surveillance for this disease in deer since 2005, when bTB was first detected in a northwest cattle farm. The disease has since been found in a total of 12 cattle operations and 27 free-ranging white-tailed deer (*Odocoileus virginianus*). Both deer and cattle have the same strain of bTB, which has been identified as one that is consistent with the disease found in cattle in the southwestern United States and Mexico. The Board of Animal Health (BAH) has been leading efforts to eradicate the disease in Minnesota's cattle, which have included the depopulation of all infected herds, a buy-out program that removed 6,200 cattle from the affected area, and mandatory fencing of stored feeds on remaining farms. No new infections have been detected in either cattle or deer since 2009. The state regained its bTB-Free accreditation in October 2011; however, some testing requirements remain on cattle herds within the endemic area. MNDNR plans to continue to monitor infection in the local deer population through hunter-harvested surveillance in fall 2012, and any further aggressive management actions (e.g., sharpshooting deer in key locations) will be dependent on future surveillance results.

INTRODUCTION

Bovine tuberculosis (bTB) is an infectious disease that is caused by the bacterium *Mycobacterium bovis*. Bovine tuberculosis primarily affects cattle; however, other mammals may become infected. The disease was first discovered in 5 cattle operations in northwest Minnesota in 2005. Since that time, 7 additional herds were found infected; resulting in a reduction of the state's bTB accreditation to Modified Accredited in early 2008. In fall 2008, Minnesota was granted a split-state status for bTB accreditation that maintained only a small area (2,670mi^2) in northwest Minnesota as "Modified Accredited," allowing the remainder of the state to advance to "Modified Accredited Advanced." To date, 27 wild deer have been found infected with the disease in northwest Minnesota, which can be attributed to a spillover of the disease from infected cattle. In 2010, The United States Department of Agriculture (USDA) upgraded Minnesota's bTB accreditation to Modified Accredited Advanced within the split-state zone and bTB-Free throughout the remainder of the state. With no new infections discovered in MN cattle in 2009 and 2010, USDA upgraded the split-state portion to bTB-Free in October 2011. Although bTB was once relatively common in U.S cattle, it has historically been a very rare disease in wild deer. Prior to 1994, only 8 wild white-tailed and mule deer (*O. hemionus)* had been reported with bTB in North America. In 1995, bTB was detected in wild deer in Michigan and do serve as a reservoir of the disease in that state.

Bovine tuberculosis is a progressive, chronic disease. It is spread primarily through the exchange of respiratory secretions between infected and uninfected animals. This transmission usually happens when animals are in close contact with each other. Animals may also become infected with bTB by ingesting the bacteria from contaminated feed. Incubation periods can vary from months to years from time of infection to the development of clinical signs. The lymph nodes in the animal's head usually show infection first and as the disease progresses, lesions (yellow or tan, pea-sized nodules) will begin to develop throughout the thoracic cavity. In severely infected deer, lesions can usually be found throughout the animal's entire body.

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Hunters do not always readily recognize small lesions in deer, as they may not be visible when field dressing deer. In fact, most infected deer appear healthy. While it is possible to transmit bTB from animals to people, the likelihood is extremely low. Most human tuberculosis is caused by the bacteria *M. tuberculosis*, which is spread from person to person and rarely infects animals.

METHODS

In 2011, a fall hunter-harvested surveillance strategy was developed to collect 500 samples from the bTB Management Zone, which is approximated by Deer Management Area (DMA) 101. When MN regained its bTB-Free accreditation in October, the existing Memorandum of Understanding (MOU) with USDA, signed by both MNDNR and BAH, was no longer in effect. To that end, MNDNR and USDA renegotiated a sampling scheme that would still satisfy our commitment to ensuring the disease is not present in wild deer within the bTB Management Zone at >1.0% with 99% confidence.

At the registration stations, hunters were asked to voluntarily submit lymph node (LN) samples for bTB testing. Hunter information was recorded, including the hunter's name, telephone number, MNDNR number, and location of kill. Maps were provided to assist the hunters in identifying the location (Township, Range, Section, and Quarter-section) of the kill. Cooperating hunters were given a cooperator's patch and entered into a raffle for a firearm donated by the Minnesota Deer Hunter's Association (MDHA). In addition, the Roseau River chapter of MDHA raffled additional firearms and a life-time deer hunting license for hunters that submitted samples from within the bTB Management Zone or bTB Core Area.

Sampling procedures included a visual inspection of the chest cavity of the hunter-killed deer. Three pairs of cranial LNs (parotid, submandibular, and medial retropharyngeal) were visually inspected for presence of gross lesions and collected for further testing. Samples were submitted to the Veterinary Diagnostic Laboratory (VDL) at the University of Minnesota for histological examination and acid-fast staining (when lesions are present only). All samples were then pooled in groups of 5 and sent to the National Veterinary Services Laboratories (NVSL) in Ames, IA for culture. Any suspect carcasses (e.g., obvious lesions in chest cavity or head) were voluntarily surrendered at the registration stations and the hunter was issued a replacement deer license at no charge. Suspect carcasses were transported in their entirety to the VDL for further testing.

In early winter, MNDNR conducted an aerial survey of the bTB Core Area to assess deer numbers and distribution (Figure 1). This information was used to guide future management activities and estimate the percentage of deer removed from the area through hunting and agency culling.

RESULTS AND DISCUSSION

In fall 2011, we collected 561 samples from hunter-harvested deer; 349 samples from within the bTB Management Zone, including 151 samples from within the bTB Core Area (Figure 2). MNDNR collected approximately 70% of the sampling goal from within the bTB Management Zone; however exceeded the overall sampling goal by 12% when including deer tested just outside this zone.

Testing of all lymph node samples at NVSL has confirmed that there were no positive cases of bTB detected during the fall 2011 surveillance. Thus, 2011 marks the second consecutive year in which no new cases of the disease were detected in wild deer. Apparent prevalence of bTB in the local deer population, sampled throughout a 1,730 to 2,670mi^2 Surveillance Zone, indicates a significant decreasing trend from 2006–2011 (Table 1, Figure 3). Further, disease prevalence in the bTB Core Area has decreased dramatically from 2007 to 2010 (Table 1, Figure 3). Although disease prevalence estimates in the TB Core Area are biased due to the limited geographic distribution of bTB-positive deer and the increased

probability of detecting a positive individual, the decreasing trend is consistent with the large-scale surveillance of the local deer populations in the fall.

Aerial survey results from January 2011 estimated that the deer population in the bTB Core Area was a minimum of 160 ± 45 deer (Figure 1). This was markedly lower than the 2011 population estimate of 531 ± 48 (Figure 4, Table 2). This was surprising as winter deer removal efforts have been suspended for 2 years and a lag effect from those operations would be unexpected. However, winter conditions in 2011 were moderately severe in the northwest and over-winter deer survival may have been adversely impacted. In a pilot study involving 16 radio-collared deer south of the bTB Management Zone, 50% of the deer were killed by predators during winter 2011. Further, winter movements of deer are highly influenced by winter weather conditions. It is likely that the bTB Core Area is home to both migratory and resident deer, some of which may move out of the zone to spring-summer-fall or winter ranges during the year. It is further likely that deer from the surrounding area are immigrating into the bTB Core Area as deer numbers are reduced and habitat availability increases. The extremely mild winter of 2012 likely played a role in decreased deer movement into the bTB Core Area, which provides good wintering habitat, and might explain the decrease in estimated deer numbers. Lastly, snow conditions during the March 2012 survey were generally poor and deer visibility may have been compromised.

The proximity of the bTB-infected deer to infected cattle herds, the strain type, and the fact that disease prevalence (<0.1%) is low, supports our theory that this disease spilled-over from cattle to wild deer in this area of the state. To date, we have sampled 10,344 deer in the northwest, and a total of 27 confirmed culture-positive deer (Figure 5). Further, the lack of infected yearlings or fawns and limited geographic distribution of infected adults further supports that deer are not a wildlife reservoir for this disease in Minnesota (Carstensen and DonCarlos, 2011).

In November 2006, a ban on recreational feeding of deer and elk was instituted over a 4,000mi^2 area to help reduce the risk of disease transmission among deer and between deer and livestock (Figure 6). Enforcement officers continue to enforce this rule and compliance is very high within the bTB Management Zone.

With the recent upgrade in status to bTB-Free across the state and a lack of available funding to continue support payments to farms that participated in the buy-out program, BAH has announced that farms will be allowed to repopulate with cattle within the bTB Management Zone beginning July 1, 2012. Although farmers will no longer be required to obtain permits or test individual animals prior to moving cattle, whole-herd testing within the bTB Management Zone will continue. MNDNR will conduct fall hunter-harvested surveillance in 2012, with a sampling scheme similar to what occurred in 2011. If no new cases of bTB are detected in wild deer, the surveillance effort will be suspended. A recheck in 2015 is possible if funding can be identified.

ACKNOWLEDGMENTS

There is no way to complete a project of this scale without the assistance and leadership from St. Paul and regional staff, including Ed Boggess, Dennis Simon, Paul Telander, and John Williams. For all the help with field collections, we'd like to thank area staff from Thief Lake, Red Lake, Roseau River, and Thief River Falls, as well as students and faculty from the University of Minnesota, College of Veterinary Medicine. Also thanks to Bob Wright, Erik Hildebrand, and John Heineman for conducting a deer survey within the bTB Core Area. Also thanks to John Giudice for analyzing the survey data. GIS support was provided by Julie Adams, Bob Wright, and Chris Scharenbroich. A special thanks to David Kuehn for agreeing to temporarily leave retirement for a few weeks of continuous deer surveillance work.

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Table 1. Number of deer sampled for bovine tuberculosis (bTB) and testing results listed by sampling strategy, 2005 to 2011, northwestern Minnesota.

Sampling strategy	2005	2006	2007	2008	2009	2010	2011	Totals
Hunter-harvested (Oct-Jan)	474	942	1,166	1,246	1,488	1,639	561	7,516
# bTB-positive	1	5	5	0	1	0	0	
Apparent prevalence	0.21%	0.53%	0.43%	0.0%	0.07%	0.0%	0.0%	
Sharpshooting (Feb-May)	n/a	n/a	488	937	738	450	n/a	2,613
# bTB-positive			6	6	2	0		
Apparent prevelance			1.23%	0.64%	0.27%	0.0%		
Landowner/tenant	n/a	90	n/a	125	n/a	n/a	n/a	215
# bTB-positive		1		0				
Total deer tested	474	1,032	1,654	2,308	2,226	2,089	561	10,344
Total # bTB-positive	1	6	11	6	3	0	0	27

Table 2. Population estimates[a] and 95% confidence intervals[b] of deer within the Bovine Tuberculosis Core Area, 2007–2012, northwest Minnesota.

Year	Aircraft	Design	Var.est	n	N	Srate	Svar	SE	Xbar	SE	95%CI	**PopEst**	SE	**95% CI**	CV(%)	RP(%)
2007	OH-58	StRS3	SRS	72	164	0.439	NA	NA	5.7	0.46	4.9-6.5	**935**	76.0	**784-1086**	8.1	16.2
2008	OH-58	GRTS.SRS	Local	72	164	0.439	21.94	4.53	4.9	0.56	3.8-6.0	**807**	75.2	**659-954**	9.3	18.3
2009	Enstrom	GRTS.stRS2	Local	79	164	0.482	20.63	2.56	4.1	0.27	3.5-4.6	**664**	44.4	**577-751**	6.7	13.1
2010	OH-58	GRTS.SRS	Local	72	164	0.439	29.30	6.70	2.6	0.39	1.8-3.3	**422**	64.4	**296-548**	15.3	30.0
2011	OH-58	GRTS.SRS	Local	72	164	0.439	21.01	2.80	3.2	0.30	2.7-3.8	**531**	48.6	**436-627**	9.2	18.0
2012	OH-58	GRTS.SRS	Local	72	164	0.439	3.06	0.57	1.0	0.14	0.7-1.3	**160**	22.3	**120-210**	13.6	26.7

[a]**Population estimate = estimated *minimum* number of deer present during the sampling interval.** Estimates are not adjusted for detectability (but intensive survey is designed to minimize visibility bias) and deer movement between sample plots is assumed to be minimal or accounted for via survey software.
[b]**95%CI's** based on sampling variance only (adjusted for spatial correlation in 2008-2011); they do not include uncertainty associated with detectabilty or animal movements (temporal variation due to animals moving onto or off the study area).

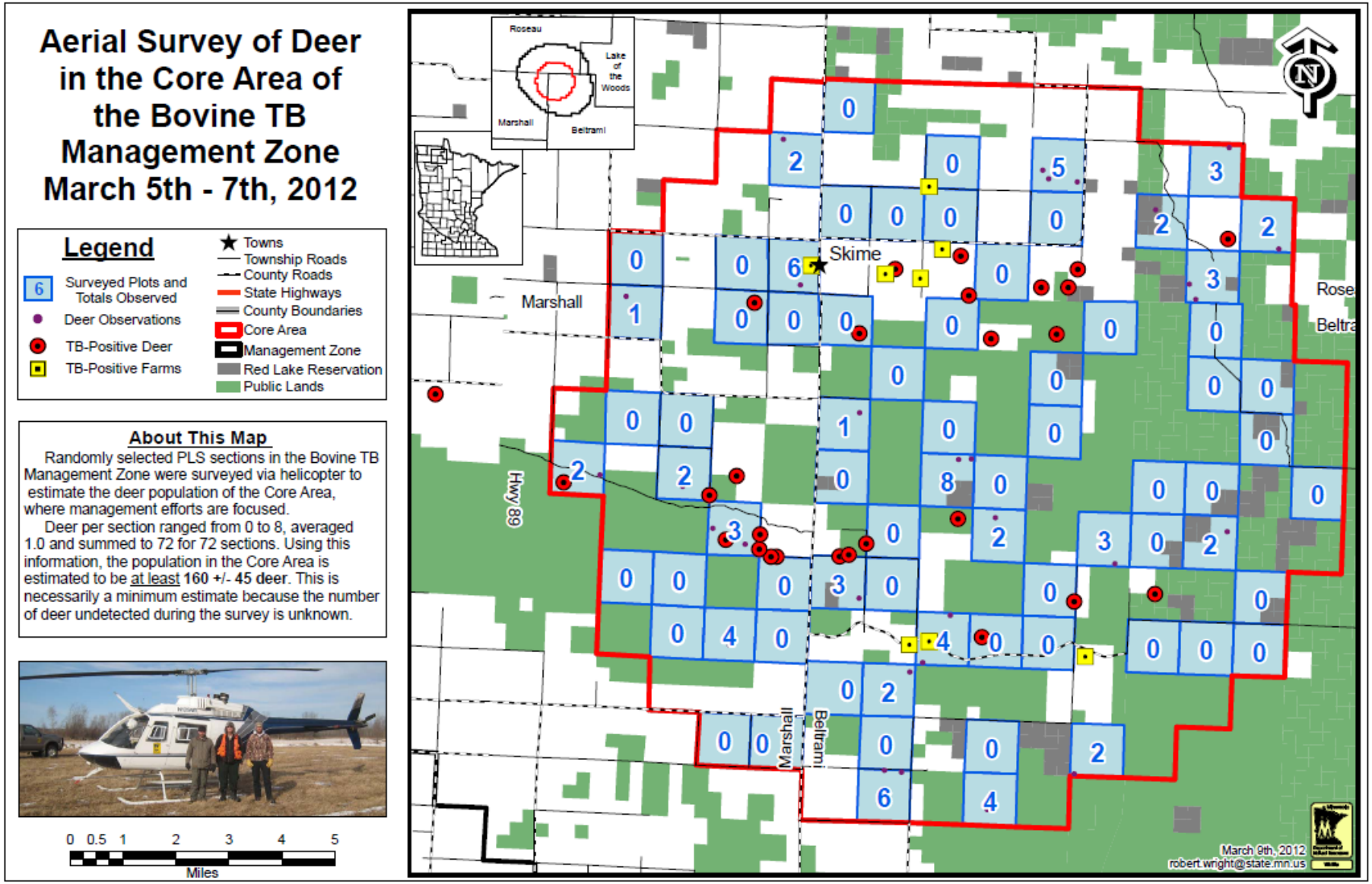


Figure 1. Results of aerial white-tailed deer survey of the Bovine Tuberculosis Core Area in March 2012, northwest Minnesota.

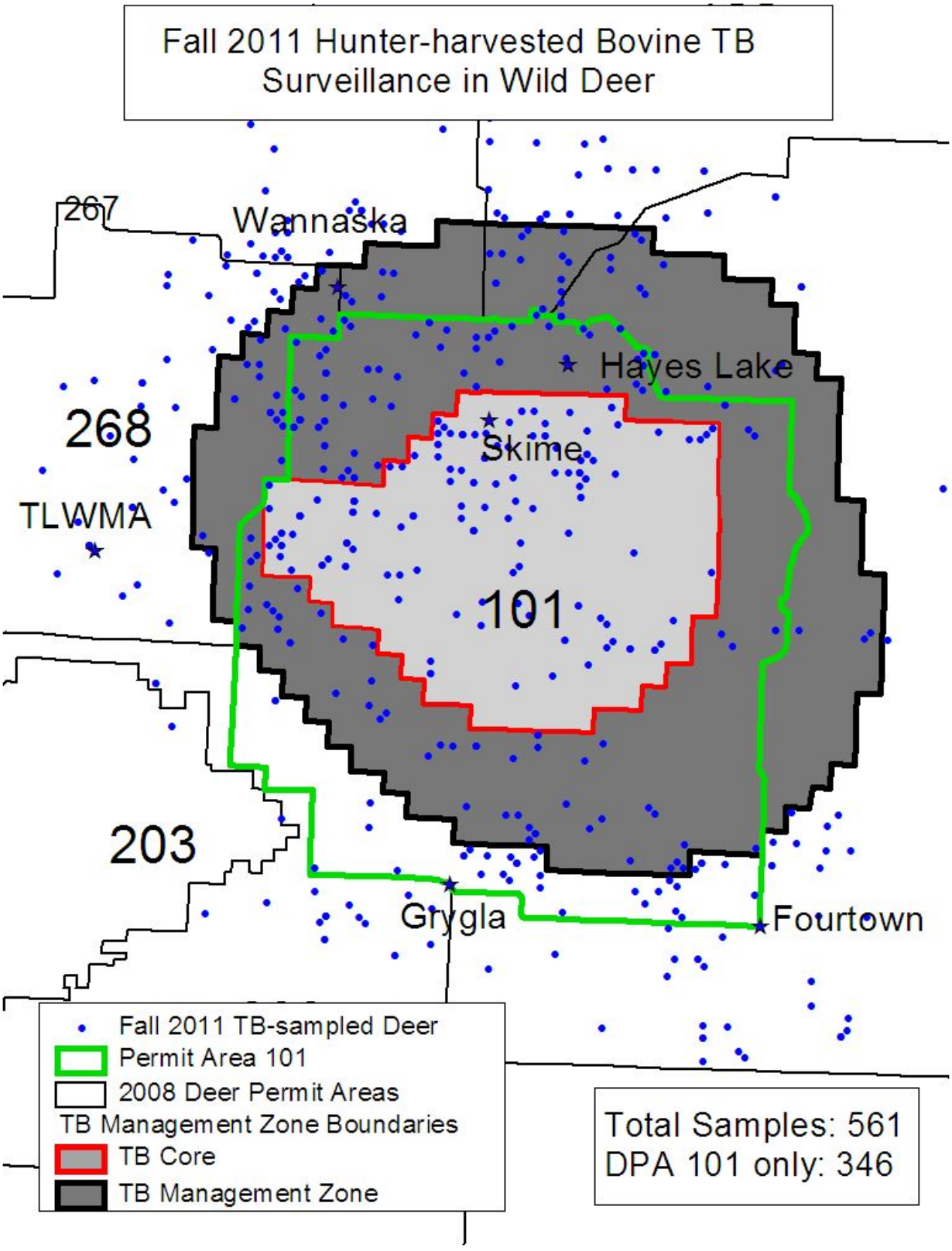
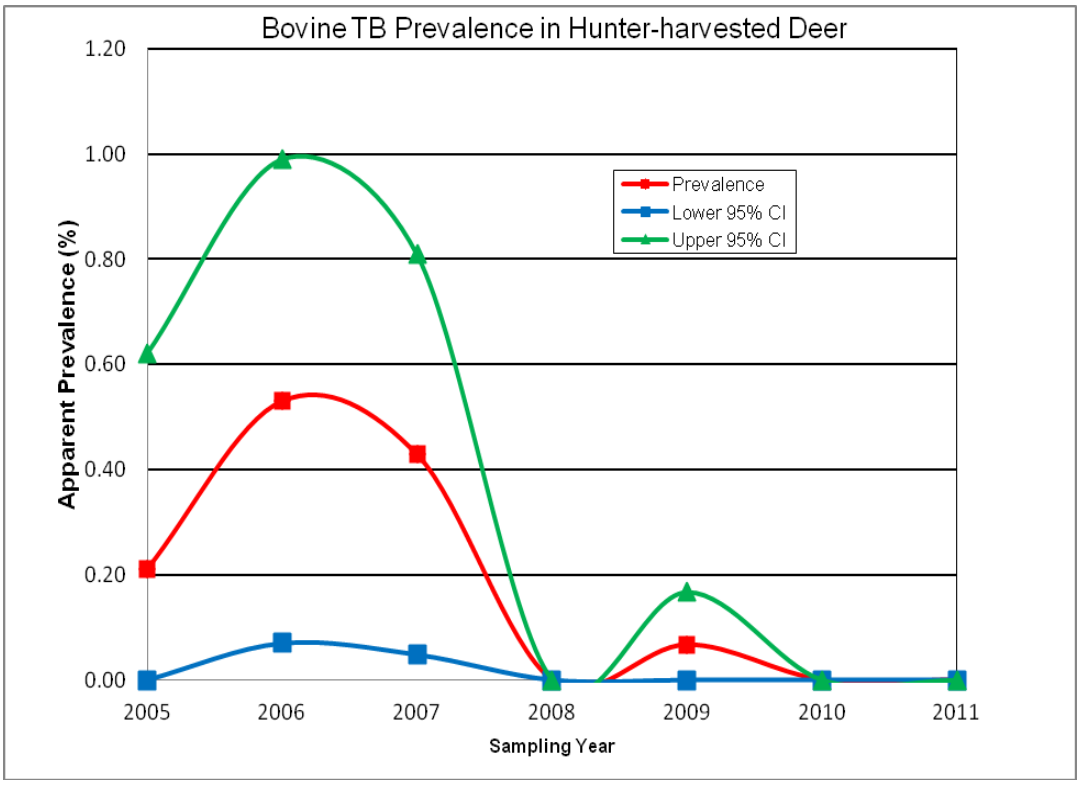


Figure 2. Locations of hunter-harvested deer (*n*=561) sampled for bovine tuberculosis (bTB) during fall 2011 in northwest Minnesota.



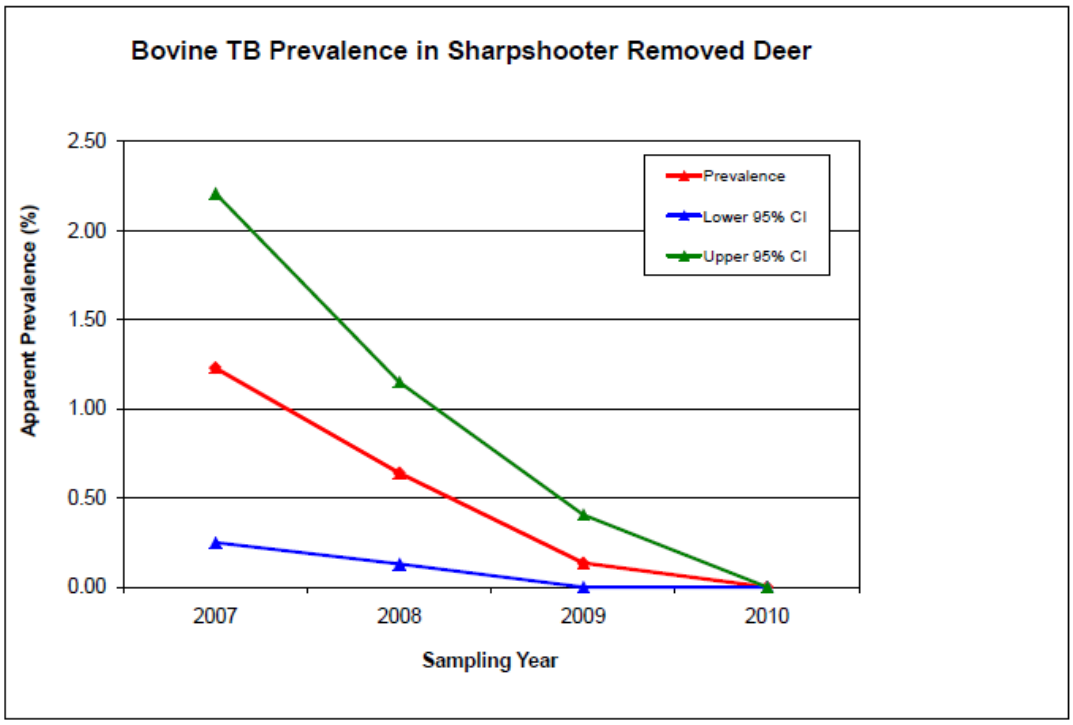


Figure 3. Prevalence of bovine tuberculosis (bTB) in hunter-harvested deer from 2005–2011 in the bTB Surveillance Zone and disease prevalence from sharpshooter removed deer from 2007–2010 in the bTB Core Area, northwest Minnesota.

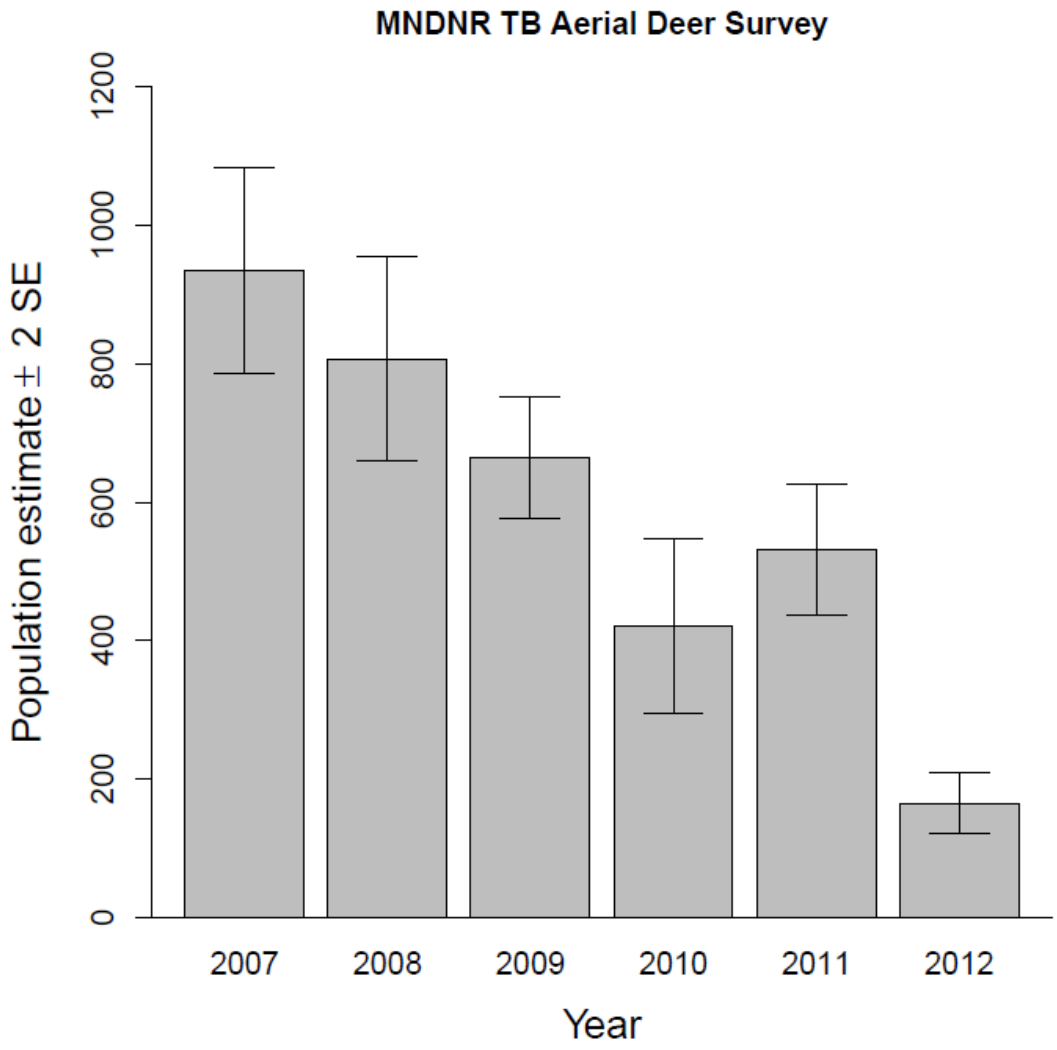


Figure 4. Population estimate of deer within the Bovine Tuberculosis Core Area, winters 2007–2012, northwest Minnesota.

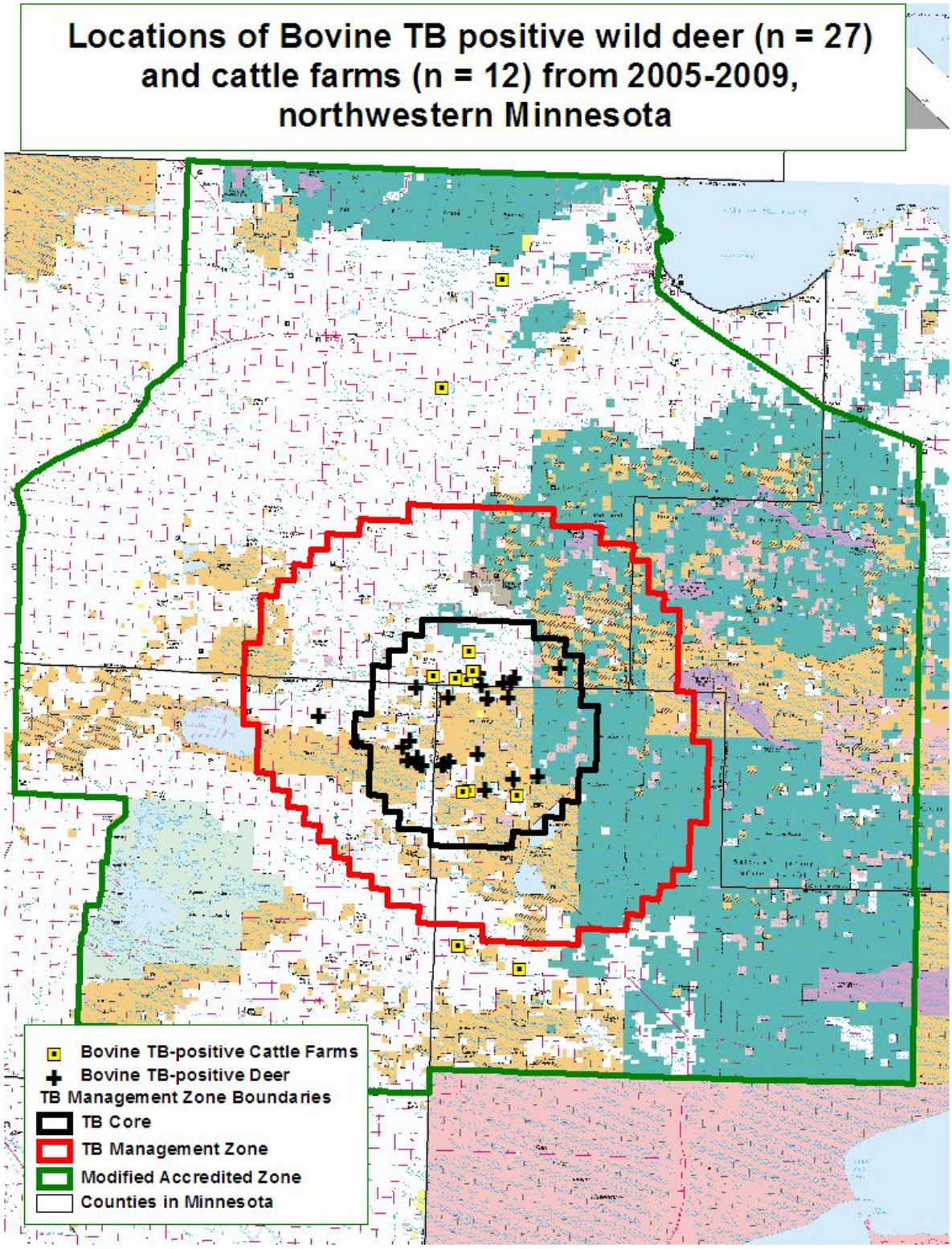
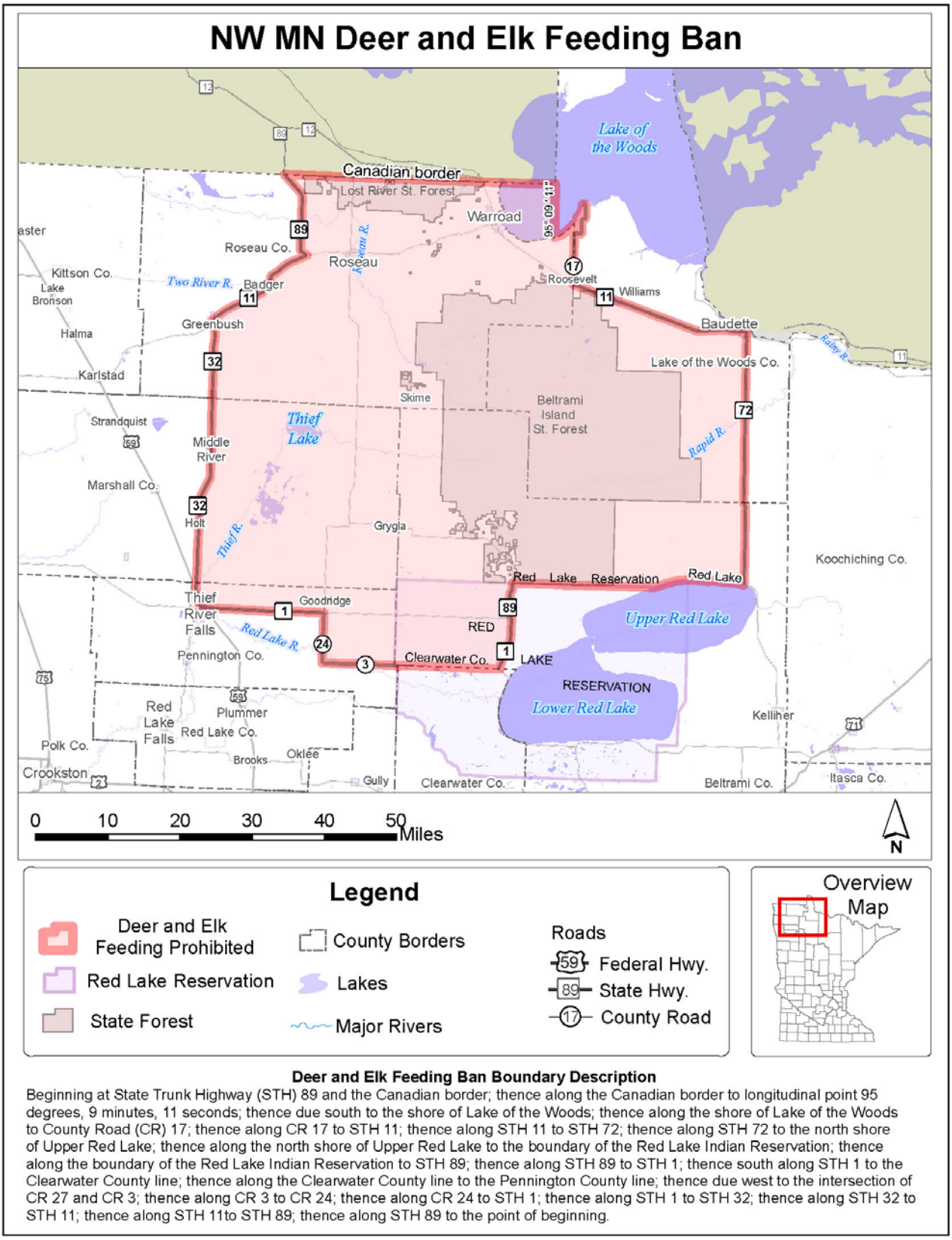


Figure 5. Locations of all white-tailed deer found infected (*n*=27) with bovine tuberculosis (bTB) since fall 2005 in northwest Minnesota, with the 12 previously-infected cattle operations are also included.



Deer and Elk Feeding Ban Boundary Description

Beginning at State Trunk Highway (STH) 89 and the Canadian border; thence along the Canadian border to longitudinal point 95 degrees, 9 minutes, 11 seconds; thence due south to the shore of Lake of the Woods; thence along the shore of Lake of the Woods to County Road (CR) 17; thence along CR 17 to STH 11; thence along STH 11 to STH 72; thence along STH 72 to the north shore of Upper Red Lake; thence along the north shore of Upper Red Lake to the boundary of the Red Lake Indian Reservation; thence along the boundary of the Red Lake Indian Reservation to STH 89; thence along STH 89 to STH 1; thence south along STH 1 to the Clearwater County line; thence along the Clearwater County line to the Pennington County line; thence due west to the intersection of CR 27 and CR 3; thence along CR 3 to CR 24; thence along CR 24 to STH 1; thence along STH 1 to STH 32; thence along STH 32 to STH 11; thence along STH 11to STH 89; thence along STH 89 to the point of beginning.

Figure 6. Area in northwest Minnesota where recreational feeding of deer and elk was banned in November 2006, as a preventative measure to reduce risk of disease transmission.