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SURVEILLANCE FOR HIGHLY PATHOGENIC AVIAN INFLUENZA IN MINNESOTA'S MIGRATORY BIRDS FROM 2006–2010

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SUMMARY OF FINDINGS

As part of a national strategy for early detection of highly pathogenic avian influenza (HPAI) in North America, the Minnesota Department of Natural Resources (MNDNR) and the United States Department of Agriculture's Wildlife Services (USDA-WS) has been conducting surveillance for the virus in waterfowl in the state since 2006. In 2010, 1,016 birds were sampled for HPAI and no positive cases were detected; however, 57 strains of low pathogenic avian influenza (LPAI) were identified. From 2006 to 2010, a total of 9,017 wild birds have been sampled for HPAI throughout Minnesota; no HPAI was detected. Nationwide, approximately 410,600 wild birds have been sampled during 2006–2010, with no evidence of disease, yet this virus remains a major concern in many parts of the world, because of its zoonotic potential and threat to the domestic livestock industry. One particular strain of HPAI, called H5N1, has affected millions of birds and hundreds of people in parts of Asia, Europe, and Africa, and concerns about this strain developing into a worldwide pandemic remain. While concern about the virus entering North America through movements of infected poultry, poultry products, or migrations of wild birds continues, large-scale surveillance in wild bird populations in the United States has been discontinued. Minnesota will continue to monitor the health of wild birds by investigating morbidity and mortality events, and screening for HPAI when appropriate.

INTRODUCTION

Avian Influenza (AI) is a viral infection that occurs naturally in wild birds, especially waterfowl, gulls, and shorebirds. It is caused by type A influenza viruses that have 2 important surface antigens, hemagglutinin (H) and neuraminidase (N), that give rise to 144 possible virus subtypes. Influenza viruses vary widely in pathogenicity and ability to spread among birds. The emergence of an Asian strain HPAI H5N1 virus in 1996 and subsequent spread of the virus in Asia, Africa, and Europe has killed thousands of wild birds and millions of domestic poultry. In 1997, HPAI H5N1 became zoonotic in Hong Kong and to-date has infected at least 552 humans in Eurasia and Africa, resulting in over 322 deaths. The migratory movements of waterfowl and other shorebirds such as from Asia into North America, heightens concern for surveillance of HPAI H5N1, although movements of domestic poultry or contaminated poultry products, both legally and illegally, are believed to be the major driving force in this virus' spread.

Following the spread of HPAI H5N1 from Asia to Europe and Africa in 2006, the National Strategic Plan for early detection of HPAI H5N1 introduction into North America by wild birds was developed. This plan outlined a surveillance strategy that focused on sampling wild bird species in North America that have the highest risk of being exposed to or infected with HPAI H5N1, because of their migratory movement patterns. This includes birds that migrate directly between Asia and North America, birds that may be in contact with species from areas in Asia with reported outbreaks, or birds that are known to be reservoirs of AI.

Recognition that ducks, geese, and swans of the order *Anseriformes* are a primary reservoir for AI, reaffirmed the need for surveillance of these populations to understand the potential for the emergence of pathogenic human and avian strains (Hanson et al. 2003). This risk concern is not focused just on domestic or wild birds in the U. S., but includes the possibility of a worldwide pandemic. Minnesota is rated as a Level 1 state by the Implementation Plan for HPAI Surveillance in the U. S., because of its historic LPAI prevalence, species-specific migratory pathways, geographic size and location, wetland habitat and amount of shoreline, and band recovery information. This means Minnesota was awarded funds to collect an assigned

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number of wild bird species samples for HPAI H5N1 in cooperation with the USDA-WS.

Since 2006, the MNDNR has been working with USDA-WS to collect samples from wild birds for HPAI H5N1 testing. Last year (2010) marked the final year of this surveillance program. In total, $430,000 in federal funds were awarded to Minnesota to collect approximately 7,900 wild bird samples. Sampling goals were as follows: in 2006, 2,000 samples collected under an agreement of $100,000; in 2007, 1,500 samples collected under a $100,000 agreement; in 2008, 1,600 samples collected under a $90,000 agreement; in 2009, 1,400 samples collected under a $70,000 agreement, and again in 2010, 1,400 samples collected under a $70,000 agreement.

METHODS

In 2010, the MNDNR's surveillance goals included 50 common goldeneye (*Bucephala clangula),* 50 ring-neck ducks (*Aythya collaris),* 50 mallards (*Anas platyrhynchos),* and 30 blue-winged teal (*Anas discors)* to sample during the summer months, primarily in conjunction with planned banding activities. In the fall, hunter-harvested surveillance was used to obtain samples from approximately 80 northern pintails (*Anas acuta)*, 80 mallards, 80 American green-winged teal (*Anas crecca*), 80 blue-winged teal, 50 northern shovelers (*Anas clypeata*), and 50 American wigeons (*Anas Americana*). Focus was directed more on fall surveillance, because the prevalence of AI peaks in late summer and early fall, whereas infection rates are often lower than 1% outside of this period (Halvorson et. al 1985).

The USDA-WS planned to sample a similar number of ducks within the species mentioned above, as well as 100 Canada geese (*Branta canadensis).* If sampling goals per species could not be met, other waterfowl species within the same functional group (e.g., dabblers, divers) could be sampled and counted toward the state's total.

Sampling strategies were coordinated between the MNDNR and USDA-WS to maximize access to bird species through handling of live wild-caught birds from waterfowl banding programs, fall hunter-harvested birds at various sites, agency (USDA-WS) harvested birds, and mortality/ morbidity events. Sampling consisted of obtaining 2 cotton swabs cloacal and oropharyngeal for each bird. Both swabs were placed into a vial containing prepared brain heart infusion (BHI) media. These samples were then submitted to the University of Minnesota's Veterinary Diagnostic Laboratory in St. Paul for initial screening for the virus. If positive for AI, samples were forwarded to the National Veterinary Services Laboratories in Ames, Iowa for strain-typing. Environmental (fecal) samples were also collected from 2006 to 2008 in Minnesota and submitted for HPAI testing; this sampling method was suspended in 2009.

RESULTS AND DISCUSSION

From 1 April 2010 through 31 March 2011, the MNDNR and USDA-WS collected a combined total of 1,016 samples from wild birds. This included birds that were live-caught (n = 417), hunter-harvested (n = 552), agency-harvested (n = 40), and mortality/morbidity events (n = 7) throughout Minnesota (Table 1, Figure 1). No positive cases of HPAI H5N1 were identified; however, 7 American green-winged teal, 32 mallards, and 2 northern pintails tested positive for LPAI subtype H5 (Figure 2). The testing protocol limited the screening for H5, H7, and N1 subtypes only; however, in some cases other subtypes were identified and reported elsewhere (Table 2).

According to the latest numbers from the United States Geologic Survey's (USGS) website (http://wildlifedisease.nbii.gov/ai/), approximately 40,660 birds were sampled for HPAI H5N1 in the U. S. in 2010. No positive cases were found. From 2006 to 2010, over 410,000 wild birds have been sampled for HPAI H5N1 throughout the U. S., including 9,017 in Minnesota, and no HPAI H5N1 has ever been detected. Despite multiple wild bird mortality events in Asia and Europe, it does not appear that HPAI H5N1 has been introduced via migratory birds into the U. S.

From 2006 to 2010, of the 9,017 samples collected in Minnesota, there were 146 positive LPAI H5 subtypes and 7 LPAI N1 subtypes (Table 3). Approximately 26% of the total samples collected were in the summer months (presumably from resident/local birds), while 48% were from fall hunter-harvested birds that were migrating into Minnesota.

There has been additional AI research conducted by the Southeastern Cooperative Wildlife Disease Study (SCWDS) since 2006 in northwestern Minnesota. Primary focus areas include Roseau River Wildlife Management Area (WMA), Thief Lake WMA, and Agassiz National Wildlife Refuge (NWR). Sampling has also occurred at lakes around the Bemidji and Fosston areas. From 2006 to 2010, SCWDS sampled over 9,200 ducks, and based on virus isolation in embryonating chicken eggs, found 1,254 positive samples, of which 30 were LPAI H5 subtypes, and 20 LPAI H7 subtypes (Table 4). Throughout all testing, there was no HPAI H5N1 virus detected. Sampling in Minnesota will continue by SCWDS at least through 2013.

Other AI research has been conducted throughout the state by University of Minnesota (UMN) since 2008, mostly in conjunction with MNDNR's sampling efforts. From 2008 to 2010, the UMN sampled over 3,100 ducks, have analyzed 3,092 to-date, and used both a plaque reduction neutralization test (PRNT) and a virus isolation (VI) test; 72 LPAI isolates have been detected. Sub-types isolated by species to-date include LPAI H1N1, H6N1, H1N1, H3N8, and H3N2 in mallards; LPAI H4N8, H4N2, H3N8, H3, and H11N9 in blue-winged teal; and LPAI H4N8 in ring-necked ducks. No H5 or H7 LPAI or HPAI has been encountered to-date.

Federal AI funding for most wild bird surveillance in the U. S. is no longer available; however, federally-funded efforts to monitor for the disease in domestic poultry will likely continue. Even though USDA-WS and MNDNR will no longer be conducting large-scale surveillance for HPAI H5N1 in wild birds, AI samples will continue to be collected at all mortality/morbidity events involving wild birds in the state.

ACKNOWLEDGEMENTS

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Table 1. Bird species sampled in Minnesota for highly pathogenic avian influenza H5N1 by the Minnesota Department of Natural Resources (MNDNR) and United States Department of Agriculture-Wildlife Services (USDA-WS), 2010. These wild birds were live-caught, hunter-harvested, agency-harvested, or subjects of morbidity/mortality events.

Agency	Species sampled	*n*
MNDNR	Black duck (American)	2
	American green-winged teal	86
	American coot	6
	American wigeon	26
	Blue-winged teal	84
	Common goldeneye	50
	Common merganser	3
	Gadwall	4
	Greater scaup	2
	Hooded merganser	1
	Lesser scaup	40
	Mallard	218
	Northern pintail	39
	Northern shoveler	35
	Redhead	6
	Ring-necked duck	127
	Ruddy duck	1
	Wood duck	25
	Total	755
USDA-WS		
	American green-winged teal	2
	American wigeon	4
	Blue-winged teal	11
	Canada goose	84
	Double-crested Cormorant	57
	Mallard	82
	Northern shoveler	2
	Wood duck	19
	Total	261
Grand Total		1016

Table 2. Subtyping results of bird species sampled in Minnesota by the Minnesota Department of Natural Resources and United States Department of Agriculture-Wildlife Services, 2010.

Species	H10N7	H3N2	H3N8	H4N6	H5N2	H6N1	N2	N4	N8	TOTAL
American green-winged teal				1		1	1	1		4
Mallard	2	1		1	3	1	1		1	10
Northern pintail				1						1
Wood duck			1							1
Total	2	1	1	3	3	2	2	1	1	16

Table 3. Low pathogenic avian influenza strains detected in wild birds sampled in Minnesota by the Minnesota Department of Natural Resources and United States Department of Agriculture's Wildlife Services, 2006–2010.

Year	TotaL samples	Species	LPAI H5	LPAI N1
2006	2,065			
		American green-winged teal		1
		Northern pintail	1	
		Ring-necked duck	1	
		Total	2	1
2007	2,264			
		American green-winged teal 8		1
		American wigeon	5	
		Blue-winged teal	6	
		Lesser scaup	3	
		Mallard	8	1
		Northern pintail	9	1
		Northern shoveler	1	
		Total	40	3
2008	2,263			
		American green-winged teal 4		
		American wigeon	4	
		Bufflehead	1	
		Blue-winged teal	4	
		Gadwall	2	
		Lesser scaup	1	
		Mallard	24	1
		Northern pintail	2	
		Northern shoveler	1	
		Total	43	1
2009	1,409			
		American green-winged teal 3		
		American wigeon	1	
		Blue-winged teal	5	1
		Mallard	2	1
		Northern pintail	4	
		Ring-necked duck	4	
		Wood duck	1	
		Total	20	2
2010	1,016			
		American green-winged teal 7		
		Mallard	32	
		Northern pintail	2	
		Total	41	0
Total	9,017	Grand Total	146	7

Table 4. Avian influenza samples collected in Minnesota by the Southeastern Cooperative Wildlife Disease Study , Athens, Georgia, 2006–2010.

Year	Total samples	Positive (%)	# of Subtypes	LPAI H5	LPAI H7
2006	130	17 (13%)	4	0	0
2007	2,441	222 (9%)	27	2	15
2008	2,452	438 (18%)	31	16	2
2009	2,341	238 (10%)	Pending [a]	6	3
2010	1,896	339 (18%)	Pending [a]	6	0

[a] All H5 and H7 viruses recovered during these years have been tested by National Veterinary Services Laboratories.

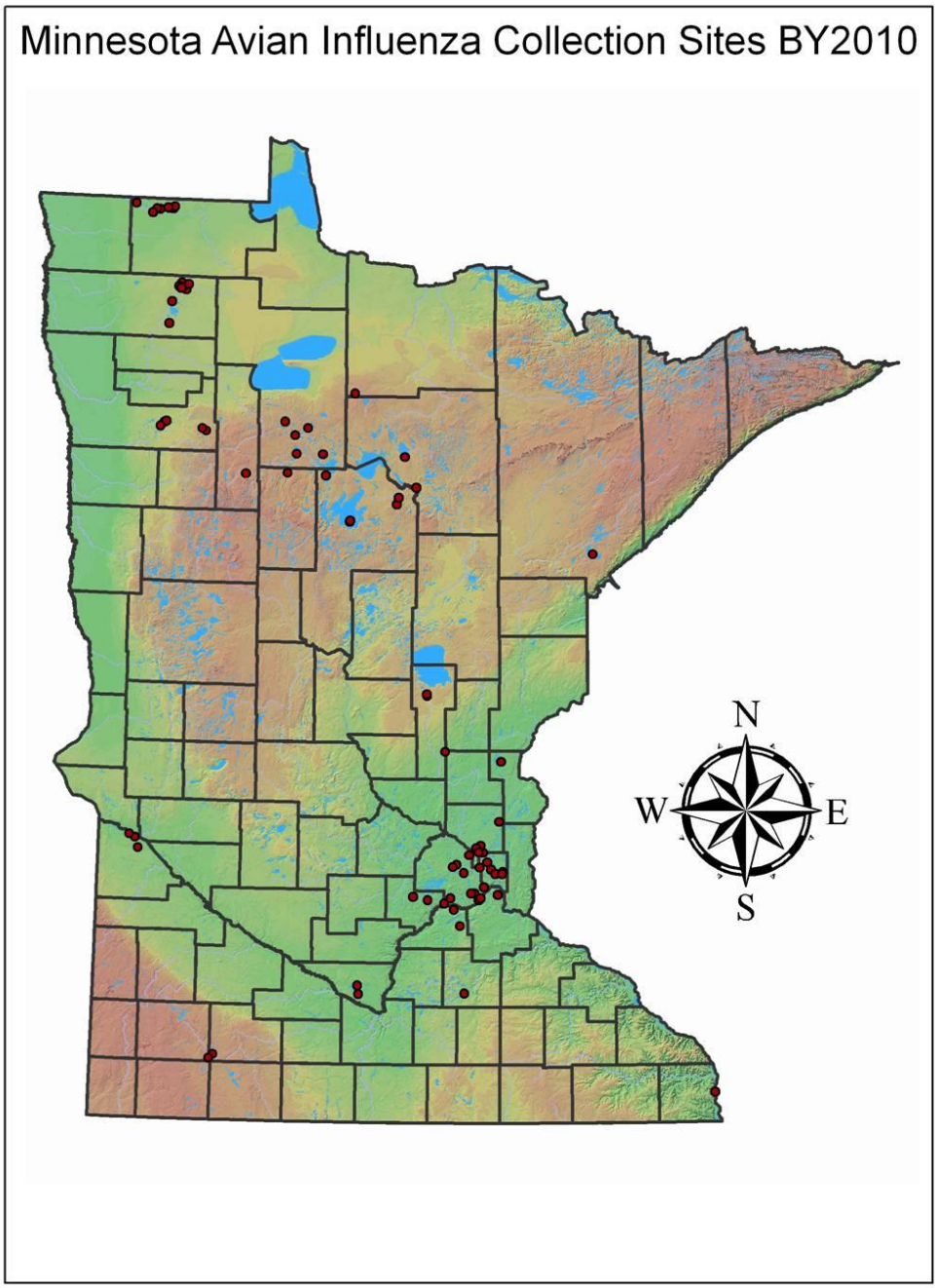

Figure 1. Sites in Minnesota from which wild bird samples (*n* = 1,016) were collected and tested for highly pathogenic avian influenza by the Minnesota Department of Natural Resources and United States Department of Agriculture's Wildlife Services, 2010.

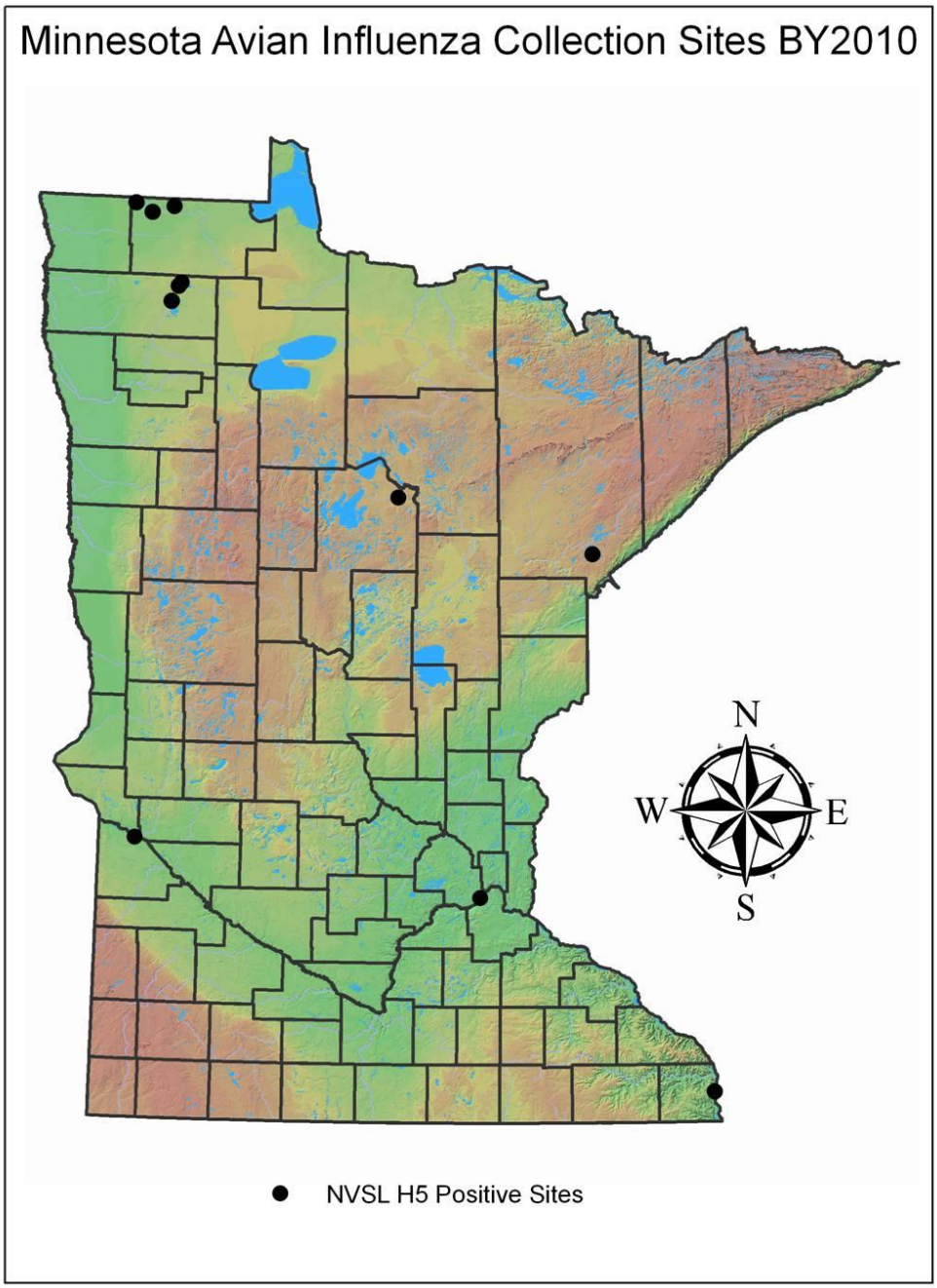

Figure 2. Collection sites in Minnesota where a low pathogenic avian influenza H5 strain was detected among the waterfowl (*n* = 41) sampled by the Minnesota Department of Natural Resources and United States Department of Agriculture's Wildlife Services, 2010.

PRELIMINARY RESULTS FROM THE 2010 MOOSE HEALTH ASSESSMENT PROJECT

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SUMMARY OF FINDINGS

This project represents the second phase of an assessment on the overall health of hunter-harvested moose (*Alces alces*) in northeastern Minnesota (MN), which began in 2007. The purpose of this project is to: (1) continue to screen hunter-harvested (and presumably healthy) moose from 2010 to 2012 for select disease agents to monitor changes in disease incidence or prevalence over time, (2) assess the clinical impacts of liver fluke (*Fascioloides magna*) infection on moose, and (3) determine the frequency of histological lesions consistent with brainworm (*Parelaphostrongylus tenuis*) infection. Samples were collected from 130 moose in 2010. Moose (*n* = 110) were screened for West Nile virus, eastern equine 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 (29.1%), malignant catarrhal fever (3.6%), borreliosis (21.8%), and leptospirosis (0.9–9.2%). 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 (*n* = 95) were used to determine if there is a correlation between liver fluke damage and serum liver enzymes. Whole blood samples (*n* = 109) were submitted for evaluation for tick-borne illnesses; anaplasmosis and piroplasma infections were documented.

INTRODUCTION

Several lines of evidence suggest 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, modeling based on these vital rates indicated that the population has been declining by approximately 15% per year since 2002 (Lenarz et al. 2010). Likewise, recruitment and twinning rates (1%) have steadily declined since 2002; recruitment was reported at its lowest rate in 2011. In 2011, the bull:cow ratio (0.64) was at the lowest value in the last 27 years. Lastly, hunter success rates have steadily decreased, from 84% in 1993 to 51% in 2010 (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 launched in 2010.

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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. A large portion of the liver can be destroyed, yet have no clinical impact to the health of the animal. 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 and ranking of their liver fluke load 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 liver function.

Our moose health assessment during 2007–2009 indicated that our moose are being exposed to a variety of arboviruses, including EEE, WNV, borreliosis, and anaplasmosis (Butler et al. 2010). As climate changes, the density annd distribution of capable vectors is expected to change as well. 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.

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 migration tracts.

METHODS

Hunters (both 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. We asked hunters to locate their kill-sites on maps we provided.

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. Whole brains were removed with the hunter's permission and placed in formalin. The serum, whole liver, and whole brains were submitted to the UMN Veterinary Diagnostic Laboratory (UMN VDL, St. Paul, MN).

Serum was tested for WNV and EEE with a plaque reduction neutralization test (PRNT) 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 130 moose (125 males, 4 females, 1 sex unknown) were submitted for diagnostic screening in 2010 (Figure 1). Exact age was determined for 124 of these moose (median = 4, range = 1–11 years old).

EEE

One hundred and ten serum samples were tested for EEE and all were negative. The absence of EEE exposure 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. 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.

WNV

Evidence of exposure to WNV was detected in 32 of 110 (29.1%) moose. These results were similar to those reported during the first 3 years of the study (34.8%, 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. Little is known about the effects of WNV in moose.

MCF

Evidence of exposure to MCF was detected in 4 of 110 (3.6%) moose sampled with PLA. Follow-up testing with VN was negative for 2 of the 4, 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. The PLA reacts with multiple gammaherpes viruses (e.g., wildebeest strain of MCF, sheep strain of MCF, deer strain of MCF). 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 a gammaherpes virus, 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.

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 24 of the 110 (21.8%) moose sampled. These results are similar to results from 2007 to 2009 (22.9%, 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. bratislava*:
  - 1/109 (0.9%)
- *L. canicola*:
  - 0/109
- *L. grippothyphosa*:
  - 1/109 (0.9%)
- *L. hardjo*:
  - 0/109
- *L. interrogans* serovar *icterohaemorrhagicae*:
  - 0/109
- *L. pomona*:
  - 10/109 (9.2%)

While the prevalences are lower for most of the serovars compared with data from 2007–2009, the prevalence of *L. pomona* actually increased from 6.9% to 9.2% (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 109 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 polymerase chain reaction (PCR) techniques. Preliminary results indicate that 10.1% of the moose were infected with anaplasmosis and 32.1% were positive for prioplasma primers. Further analysis is pending.

Brain Histopathology

Forty whole brains were collected in 2010. Since 2008, a total of 87 whole brains have been collected and examined. No lesions were found in 74 (85.1%) of the brains, 9 (10.3%) had lymphocytic infiltration (unspecific chronic inflammatory lesion), and 4 (4.6%) had lesions consistent with larval migration tracts (mild to moderate meningitis, axonal degeneration, and secondary demyelination).

Whole Liver Evaluation

In 2010, 108 whole livers were collected. Combined with livers collected in 2009 (n = 57), 165 livers have been submitted for gross examination. Of the 165 livers examined, 120 (72.7%) had no fluke-induced lesions, 28 (16.9%) had mild infection, 15 (6.7%) had moderate infection, and 6 (3.6%) had marked infection. Collection of whole livers will continue in 2011–2012. 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 95 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.

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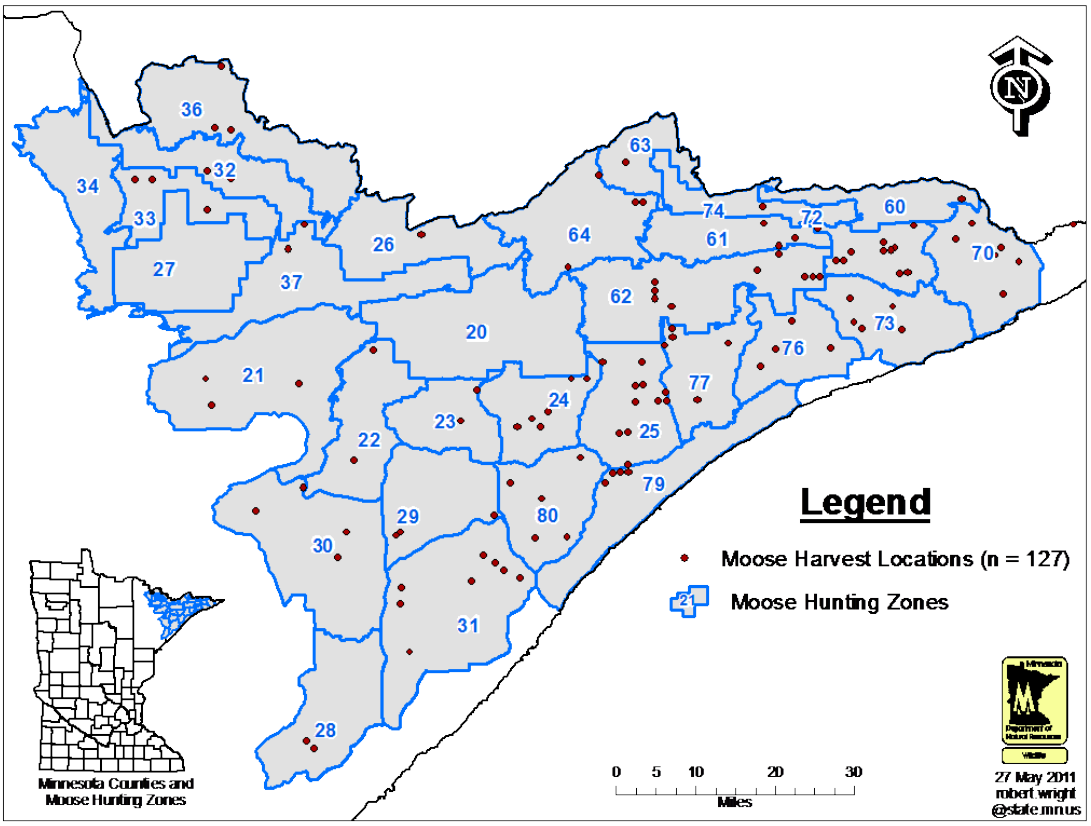

Figure 1. Locations of hunter-harvested moose (*n* = 127) included in the 2010 moose health assessment project, northeastern Minnesota.

MINNESOTA GRAY WOLF DISEASE SCREENING AND MORPHOLOGY

Michelle Carstensen[1], Erika Butler, Dan Stark, Erik Hildebrand, and John Erb

SUMMARY OF FINDINGS

A total of 206 wolves (*Canis lupus*) were included in the first year of a 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 (75.4%), canine adenovirus (76.1%) canine distemper virus (16.4%), eastern equine encephalitis (2.8%), West Nile virus (13.7%), heartworm (9.6%), Lyme (94.5%), and neosporosis (83.6%). Parasites were discovered in 14.5% of fecal samples examined. Genetic analyses are pending.

INTRODUCTION

Minnesota's gray wolf population is currently managed under the authority of the U.S. Fish and Wildlife Service. Wolves in Minnesota are classified as threatened under the federal Endangered Species Act. The Minnesota Department of Natural Resources (MNDNR) anticipates a decision to delist gray wolves in Minnesota within the next year. Following that ruling, wolves will be managed in Minnesota by state statute, rule, and under a wolf management plan. This plan is designed to protect wolves and monitor the population while giving owners of livestock and domestic pets more flexibility in addressing wolf depredation. A primary component of monitoring the wolf population 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). 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 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). No one has attempted to relate Minnesota wolf morphology with genetics. 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 or subspecies occurrence. Information will have both scientific and management value, and depending on the timing of the results, may better inform ongoing efforts to delist the wolf in Minnesota.

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 within the MNDNR, the United States Geological Survey (USGS), 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 euthanized wolves, blood was collected from the site of a bullet wound, heart, or from the chest cavity as soon after death as possible. 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 euthanized wolves when possible. Once properly preserved, the serum and genetic samples collected during this study can be stored indefinitely.

Serums 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). 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.

Morphological measurements of cleaned skulls from dead wolves followed procedures described in Nowak (1995). The 10 measurements were (1) greatest length of skull, (2) zygomatic width, (3) alveolar length from P1 to M2, (4) maximum width of rostrum across outer sides of P4, (5) palatal width between alveoli of P1, (6) width of frontal shield, (7) height from alveolus of M1 to most ventral point of orbit, (8) depth of jugal, (9) crown length of P4, and (10) greatest crown width of M2 (illustrations of the measurements and a more detailed explanation of statistical procedures is described in Nowak [1995]). For all wolves, including live captures, we recorded coat color, body weight, and measurements of ear length, shoulder height, body length, tail length, and foot length and width.

To assess mtDNA and microsatellites, genetic samples (e.g., ear punch, FTA® card, and muscle samples) were collected from each wolf. Muscle samples were preserved in both 95% ethanol and desiccant, and stored at room temperature. Genetic samples will be evaluated by the U. S. Fish and Wildlife Forensics Laboratory in Ashland, Wisconsin. Details of the statistical analyses used to identify or group individuals based on DNA or morphology will be outlined when results are available. Herein, we simply note that the focus will be on elucidating any spatial differences or patterns in molecular or morphological attributes, and on assessing whether any observed molecular patterns translate into meaningful morphological differences.

Anticipated project duration is 2 years. We hope to sample a minimum of 400 wolves over the 2-year study period, with samples distributed throughout wolf range. Detection of any disease will be assessed at an assumed prevalence level; >1% prevalence would be significant.

As a pilot study, the significance of any disease detection from this health survey would require more formal study to ascertain its significance relative to population demography.

RESULTS AND DISCUSSION

Samples from a total of 206 wolves (149 adults, 4 yearlings, 42 pups, and 11 of unknown age; 105 males, 100 females, and 1 unknown sex) were included in the first year of this study. These included wolves that were euthanized by USDS-WS (*n* = 103), live-caught research animals (*n* = 31), vehicle kills (*n* = 22), found dead (*n* = 45), and euthanized due to sickness (*n* = 5) (Figure 1). Genetic samples were obtained from all wolves; however, 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 75% of wolves have been exposed to CPV, which is similar to findings reported by Mech et al. (2008) 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). Little is known about the epidemiology of CPV in wild canid populations or its potential to negatively impact populations. Mech et al. (2008) reported that annual pup survival was reduced by 70% in northeastern Minnesota, and wolf population change was related to CPV antibody prevalence. These authors further speculated that CPV may reduce pup survival by 40–60% in the greater Minnesota population, and that this reduction limited rate of population increase to about 4% per year (compared with increases of 16–58% in other wolf populations). 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 in wolves in our study was slightly 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 as Spanish wolves (18.7%, 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 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 14% 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 nearly 10% 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 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).

Neospora caninum is a protozoal parasite, which is best known for causing abortion in cattle and neurological disease in dogs. Wild herbivores and canids also are thought to act as intermediate and definitive hosts, respectively (Gondim 2006, Dubey et al. 2009). 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

A total of 62 fecal samples were examined by floatation for any evidence of ova or protozoal infection. Nine of the samples had hookworm ova, 2 had trematode ova, 13 had sarcocysts, and 2 were positive for both sarcocyts and hookworm. 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 38 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 indicate that 7.9% of the wolves were infected with Anaplasmosis, 39.5% were positive for prioplasma primers, and 5.3% were infected with Lyme disease. Further analysis is pending.

Morphology and Genetic Analysis

Although 206 skulls have been collected for taxonomic evaluation, presently, only about 15% have been cleaned. We have initiated a collaboration for preparing and curating skulls with Dr. Sharon Jansa at the Bell Museum of Natural History (UMN). As collection skulls are prepared for storage, measurements will be made as in Nowak (1995). Each skull will be permanently cataloged in the mammal collection at the Bell Museum.

Genetic samples are being stored until the National Wildlife Forensics Laboratory can conduct analyses, as in Fain et al. (2010). We intend to submit these samples for analyses in July 2011. 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.

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Table 1. Serological results for disease screening of wolves sampled in Minnesota, January 2010–February 2011.

Disease	*n*	No. positives	Apparent prevalence (%)
Canine parvovirus	69	52	75.4
Canine adenovirus	71	54	76.1
Canine distemper virus	73	12	16.4
Eastern equine encephalitis	72	2	2.8
West Nile virus	73	10	13.7
Heartworm disease	73	7	9.6
Lyme disease	73	69	94.5
Neospora	73	61	83.6

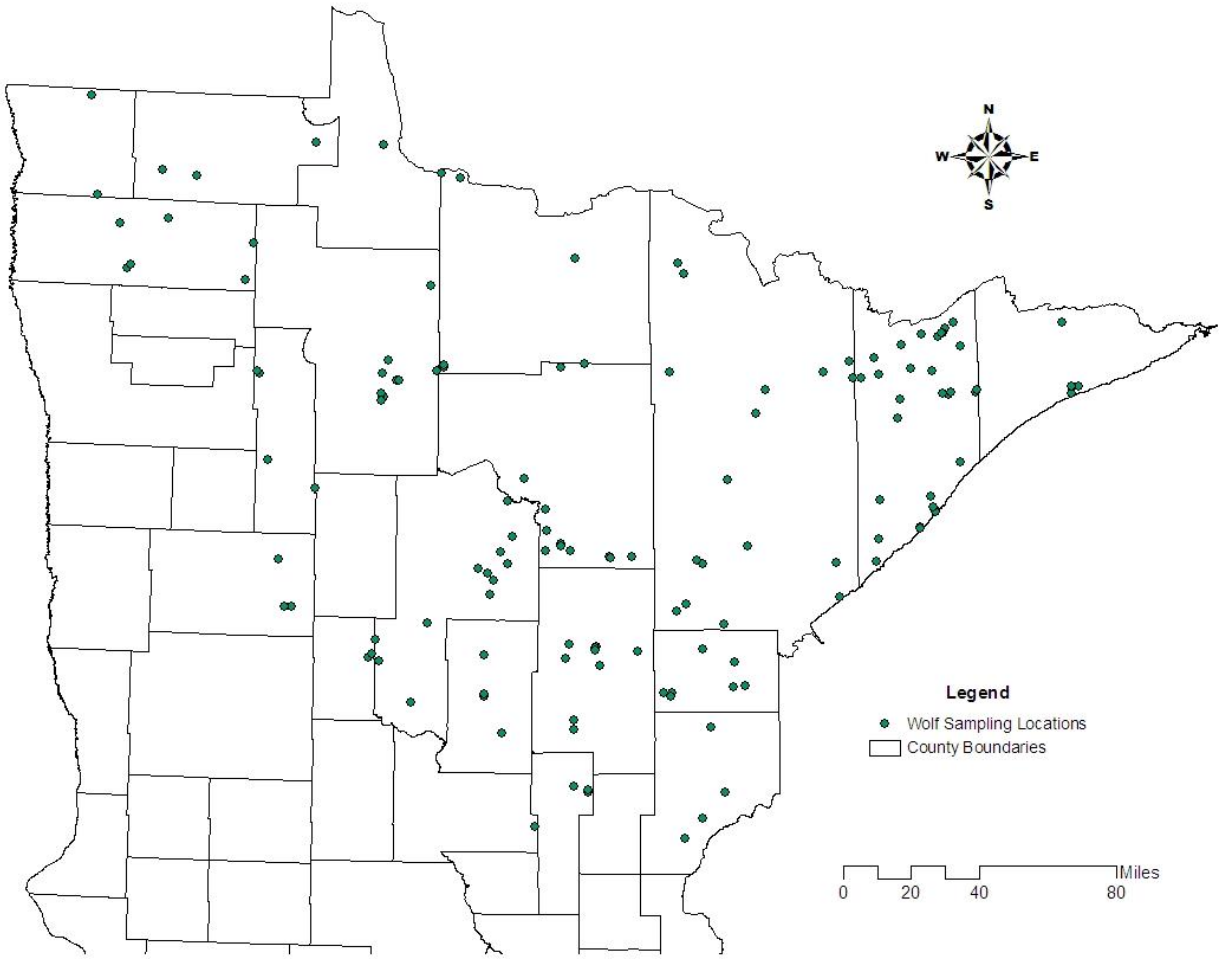

Figure 1. Sampling distribution of wolves (*n* = 206) during the first year of study of diseases and genetics of Minnesota's wolf population, 2010.

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

Michelle Carstensen[1], Louis Cornicelli, David Pauly, Erik Hildebrand, and Erika Butler

SUMMARY OF FINDINGS

In November 2010, the Minnesota Department of Natural Resources (MNDNR) sampled 564 hunter-harvested white-tailed deer (*Odocoileus virginianus*) for chronic wasting disease (CWD) in southeastern Minnesota. This surveillance effort focused on a 32.2-km (20-mi) radius around a CWD-positive captive elk facility near Pine Island, discovered in 2009. One deer tested positive for CWD (0.2% apparent prevalence), marking the first detection of the disease in Minnesota's wild deer population. In response to this disease detection, MNDNR conducted a fixed-wing aerial deer survey in a 16.0-km (10-mi) radius of the index case in late January 2011 and estimated 6,200 deer (7.3 deer/km^2, 19 deer/mi^2). A supplemental surveillance effort was conducted in February–March 2011; 752 adult deer samples were collected and all tested CWD-negative. 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 2011.

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.

Overall, Minnesota has approximately 580 domestic cervid facilities with approximately 15,100 deer, elk, and other cervidae behind fences. 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 over the landscape and range in densities from (< 1-15 deer/km^2, 1–40 deer/mi^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 2009, an additional 5,200 hunter-harvested deer and over 500 targeted or opportunistic deer were tested for CWD, with no positives detected.

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

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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.

METHODS

Hunter-harvested surveillance was conducted at deer registration stations during the regular firearm hunting season and first weekend of the muzzleloader season. Stations were staffed with MNDNR personnel and students (veterinary medicine and natural resources) trained in lymph node collection. Hunters were asked to voluntarily submit medial retropharyngeal lymph node samples for CWD-testing. All samples were inventoried, entered into a database, and sent to the University of Minnesota's Veterinary Diagnostic Laboratory (St. Paul, MN) for enzyme-linked immunosorbent assay (ELISA) testing. Positive samples from ELISA testing would be confirmed using immunohistochemistry (IHC) testing at the National Veterinary Services Laboratory in Ames, Iowa.

During fall 2010, registration stations were selected based on deer volume and distribution throughout the surveillance zone to meet a sampling goal of 500 deer within a 20-mile radius of the former CWD-positive elk farm near Pine Island. 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 firearm donated by the Minnesota Deer Hunter's Association.

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.

Additionally, MNDNR implemented efforts to obtain an additional 900 samples during winter 2011 in a 793-km^2 (306-mi^2) area surrounding a newly detected CWD-positive deer. Landowner shooting permits, agency-sponsored culling (conducted by USDA-Wildlife Services), and opportunistic sampling (e.g., vehicle-killed, sick or deer found dead) were used to collect samples from deer in this area. Landowner authorized by permit contacted trained MNDNR staff within 24 hours of harvesting deer; samples were collected in the field at private residences. All agency-harvested deer were transported intact to a central processing facility located within the winter CWD surveillance area. Sample collection and handling was similar to that described above. Carcasses were held in a refrigerated trailer at 33-35°F until test-negative results were reported (typically within 3 business days), then were salvaged for venison and made available to the public.

Prior to beginning the winter-sampling effort, MNDNR used a fixed-wing aircraft to conduct an aerial survey of the winter CWD surveillance area to assess deer numbers and distribution (Figure 1). A helicopter census of the CWD Core Area was conducted as well (Figure 2). This information was used to guide sharpshooting activities and estimate the

percentage of deer removed from the area.

RESULTS AND DISCUSSION

During fall 2010, MNDNR sampled 438 hunter-harvested deer within 52 km^2 (20 mi^2) of the CWD-positive elk farm in Olmsted county, and an additional 86 deer in the periphery (Figure 3). In mid-January 2011, MNDNR was notified that an adult female harvested by a hunter on 28 November 2010, tested positive for CWD. This was the first case of CWD detected in a wild cervid in Minnesota. It was harvested approximately 4.8 km (3 mi) southwest of the former CWD-positive elk farm in Pine Island (Figure 4). Initial prevalence estimated the infection at < 0.2% of the local deer population. Further, over 3,200 deer were sampled in the southeast during falls 2009 and 2010 combined (Figure 4), which included about 400 deer within a 16-km (10-mi) radius of the index case.

From May 2010 to May 2011, MNDNR collected a total of 47 samples from targeted surveillance efforts. This included samples from 7 escaped captive cervids, 34 free-ranging sick deer, 2 free-ranging elk, 2 vehicle-killed deer, and 2 wild deer removed from within a captive cervid facility; all samples were negative for CWD.

Since discovery of our index case, the 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.

As a first critical step in responding to CWD detection in the wild, the MNDNR conducted an aerial survey to gain an understanding of local deer abundance and distribution on the landscape. An aerial deer survey in late January-early February estimated 6,200 deer within the 793-km^2 (306-mi^2) winter surveillance area, equating to 7.3 deer/km^2 (19 deer/mi^2) density (Figures 1 and 2). Deer densities were highest within a 23-km^2 (9-mi^2) area surrounding the index case; 600 deer were counted with an estimate of >31 deer/km^2 (80 deer/mi^2) (Figure 2).

In order to gain further confidence in the apparent prevalence and geographic extent of the CWD infection in the local deer population, an additional 1,180 deer (752 adults, 428 fawns) were sampled within 16 km (10 miles) of the index case in winter 2011 (Figure 5); all deer were negative for the disease. Sampling included deer taken by landowner shooting permits (*n* = 491), agency-sponsored sharpshooting (*n* = 603), vehicle-kills (*n* = 59), and opportunistically (*n* = 27). Landowner shooting permits authorized landowners, or their designees, to take deer on their property. The permits had no bag limits and landowners were encouraged to take multiple deer. Ultimately, 323 landowner shooting permits were issued, and 47% of permit-holders harvested ≥1 deer. Overall, 57% of permitees took 1 or 2 deer and only 5% took >10 deer from their properties.

Another key step in preventing further spread of CWD was to ban the recreational feeding of deer. On February 14, MNDNR issued a special rule that made recreational deer feeding illegal in a 4-county area (Dodge, Goodhue, Olmsted and Wabesha), surrounding the location of the CWD-positive deer (Figure 6). The ban was aimed at reducing the potential for the disease spread by eliminating artificially-induced deer concentration sites. MNDNR Enforcement staff began educating and enforcing the new rule immediately and compliance was extremely high.

The estimated cost of the winter surveillance effort was $419,000. The majority ($229,000) resulted from the USDA sharpshooting contract, staff overtime ($82,000), and diagnostic testing ($30,000). The remaining expenditures were related to staff travel, building leases, and equipment leases or rentals.

Given the results of the CWD surveillance efforts of 2010 and winter 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 is encouraging.

It may be plausible 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, and H. Bloom for the massive surveillance effort this past winter. Equally important was excellent leadership provided by E. Boggess, M. DonCarlos, D. Simon, T. Bremicker, C. B. Bylander and G. Salo. We would like to thank all the MNDNR Area Wildlife and Enforcement staff from Regions 3 and 4, 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. Adams and B. Wright for fulfilling our demanding GIS needs, J. Giudice, B. Haroldson, B. Moss, and T. Buker for conducting the aerial survey work, and P. Takash for his efforts in communications and internet-related work. We appreciate the support of the USDA-Wildlife Services program in their deer removal efforts (J. Hart and his team of wildlife professionals), as well as the Disease Program, who provided disease biologists (P. Wolf-MN, E. Blizzard-CO, J. Caudel-IN, and C. Hicks-OH). Their dedication and commitment to the successful completion of this project was tremendous.

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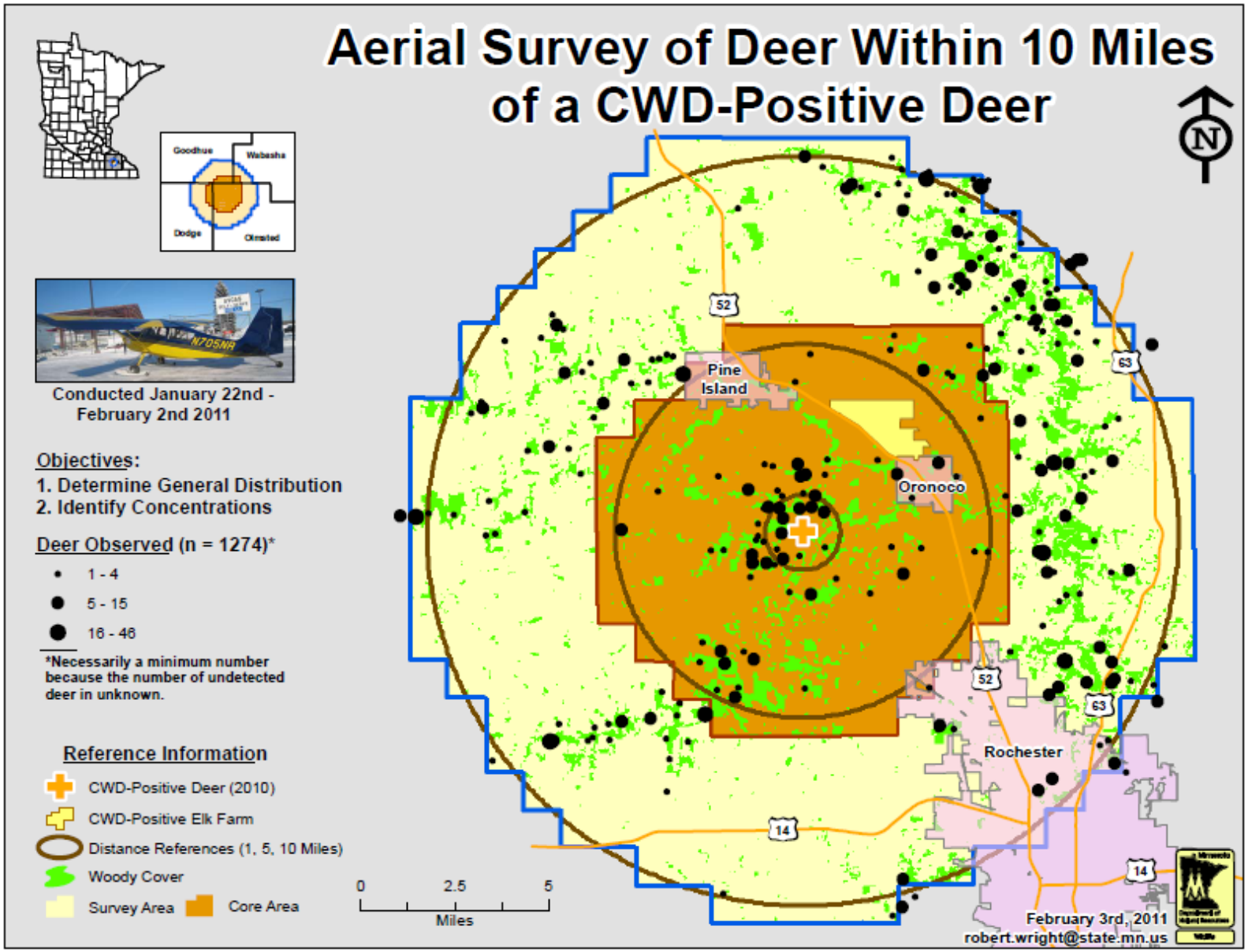

Figure 1. Fixed-wing, aerial survey results for 793-km^2 (306-mi^2) area surrounding the location of the white-tailed deer that tested positive for chronic wasting disease (CWD), southeastern Minnesota, January–February 2011.

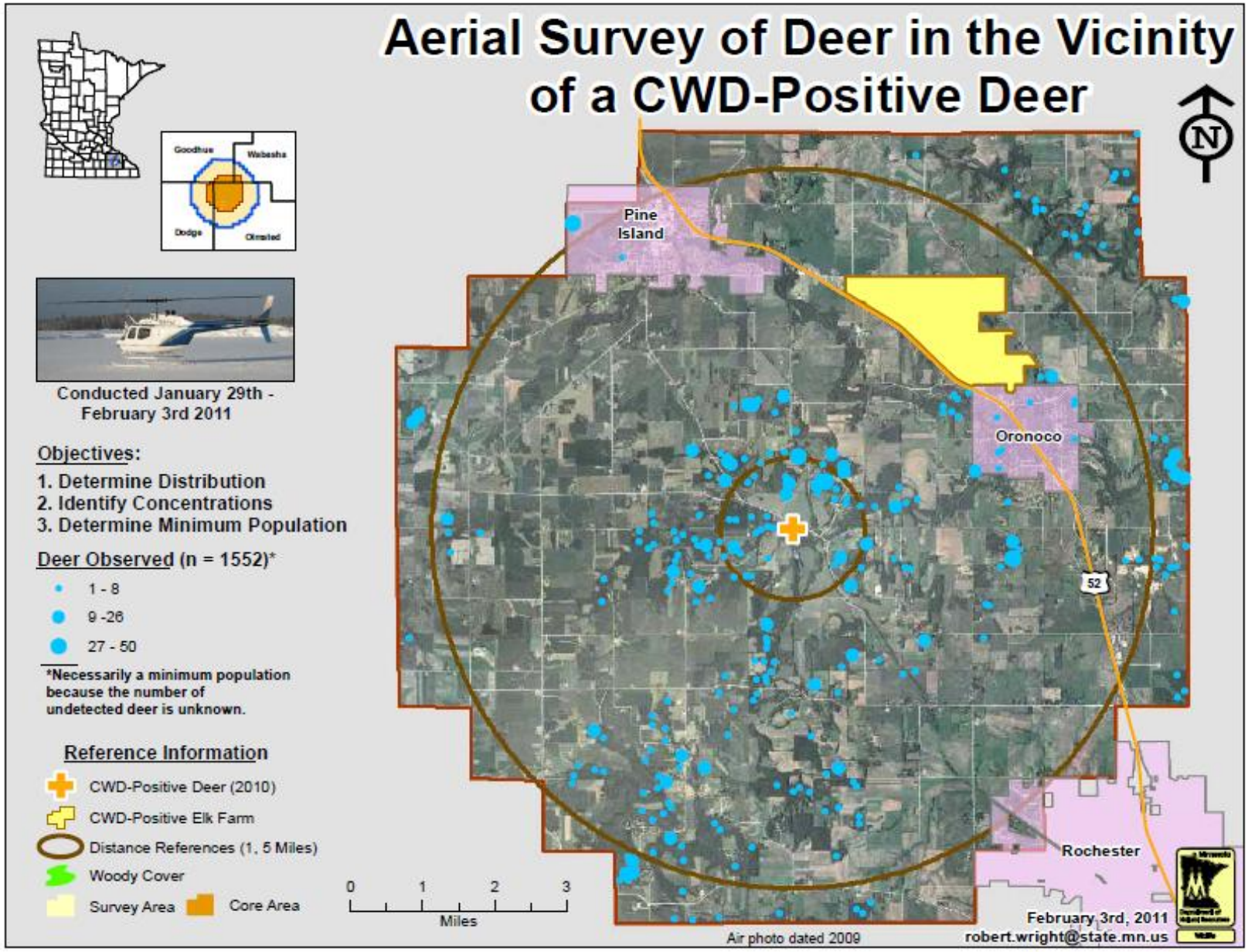

Figure 2. Helicopter white-tailed deer census for the 259-km^2 (100-mi^2) Core Area within the winter 2011 chronic wasting disease (CWD) surveillance area, southeastern Minnesota, January–February 2011.

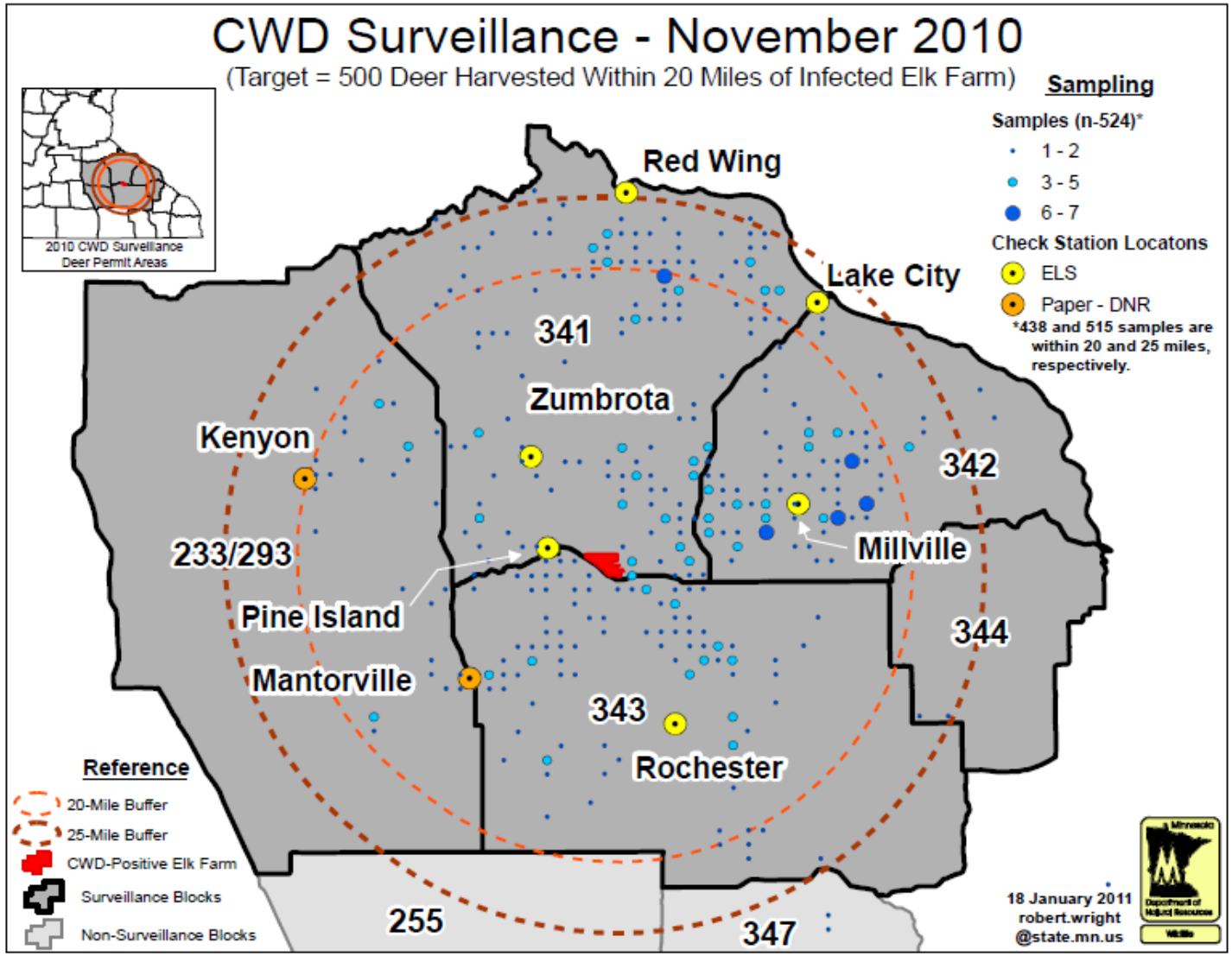

Figure 3. Sampling distribution for hunter-harvested white-tailed deer (*n* = 524) tested for chronic wasting disease (CWD) within 32 and 40 km (20 and 25 mi) of a formerly positive captive elk farm, southeastern Minnesota, fall 2010.

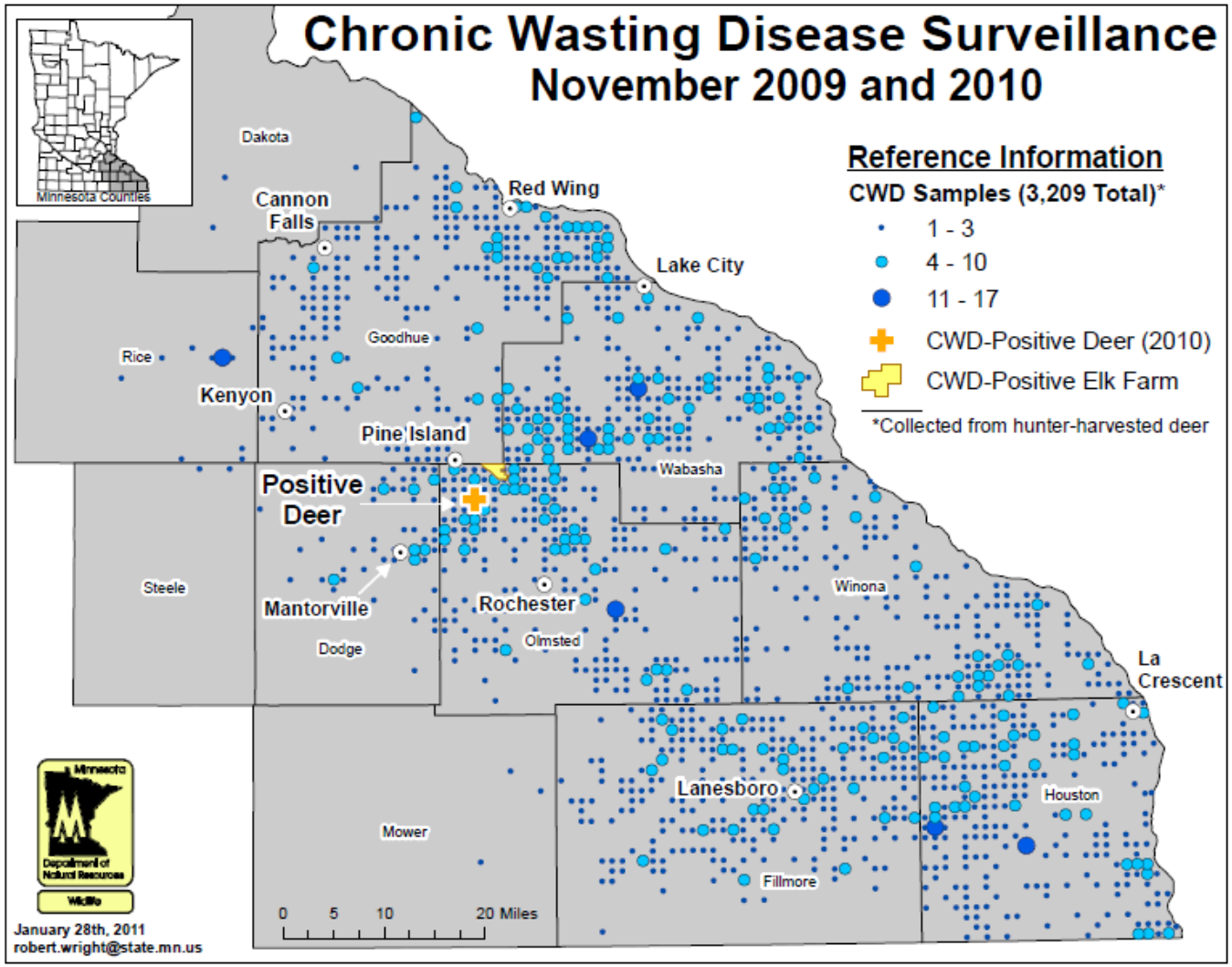

Figure 4. Sampling distribution for all hunter-harvested white-tailed deer (*n* = 3,209) tested for chronic wasting disease (CWD) in southeastern Minnesota, falls 2009 and 2010, in relation to the location of CWD-positive deer.

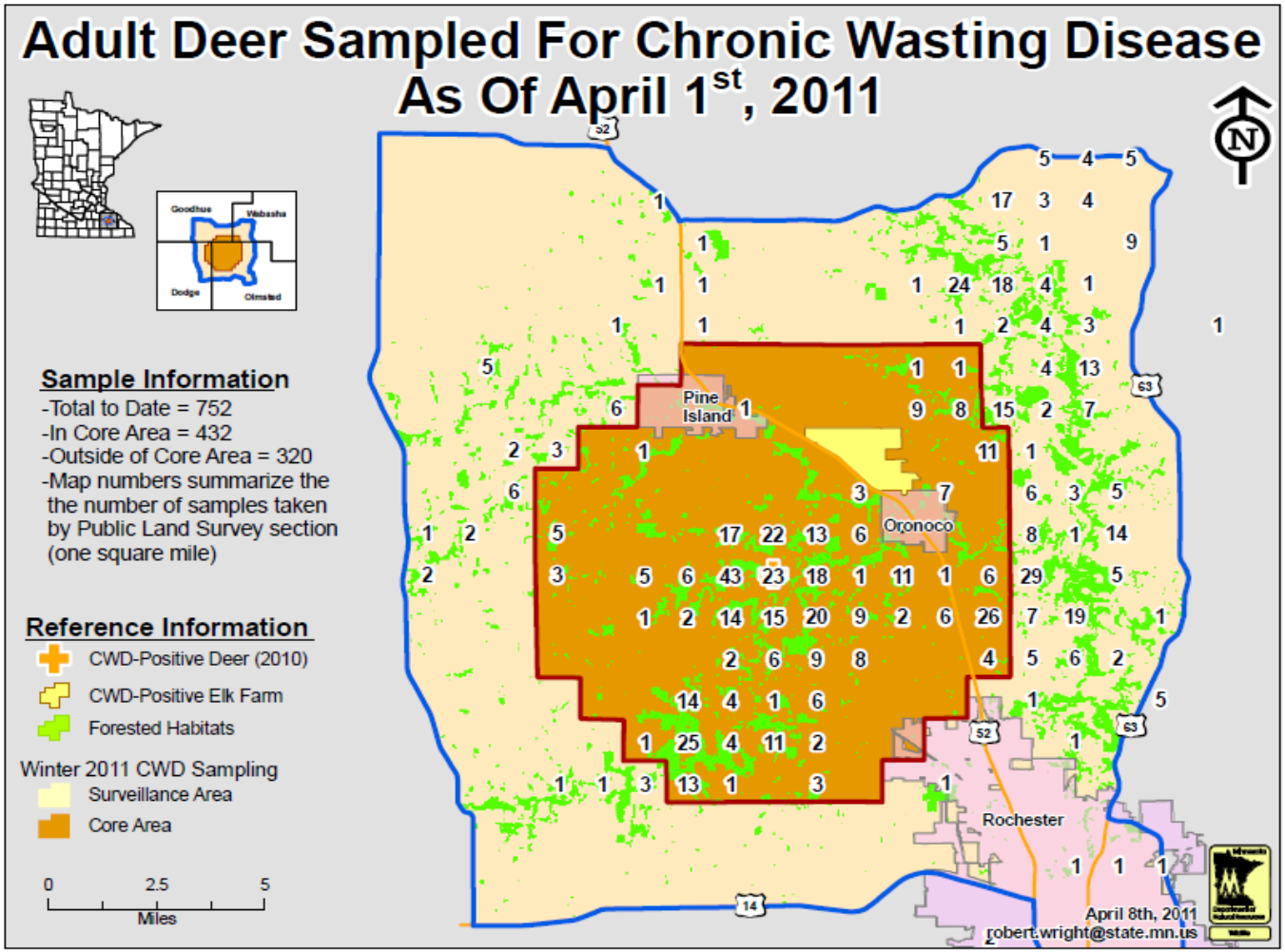

Figure 5. Section totals and distribution of white-tailed deer (*n* = 752) sampled for chronic wasting disease (CWD) during winter 2011, southeastern Minnesota.

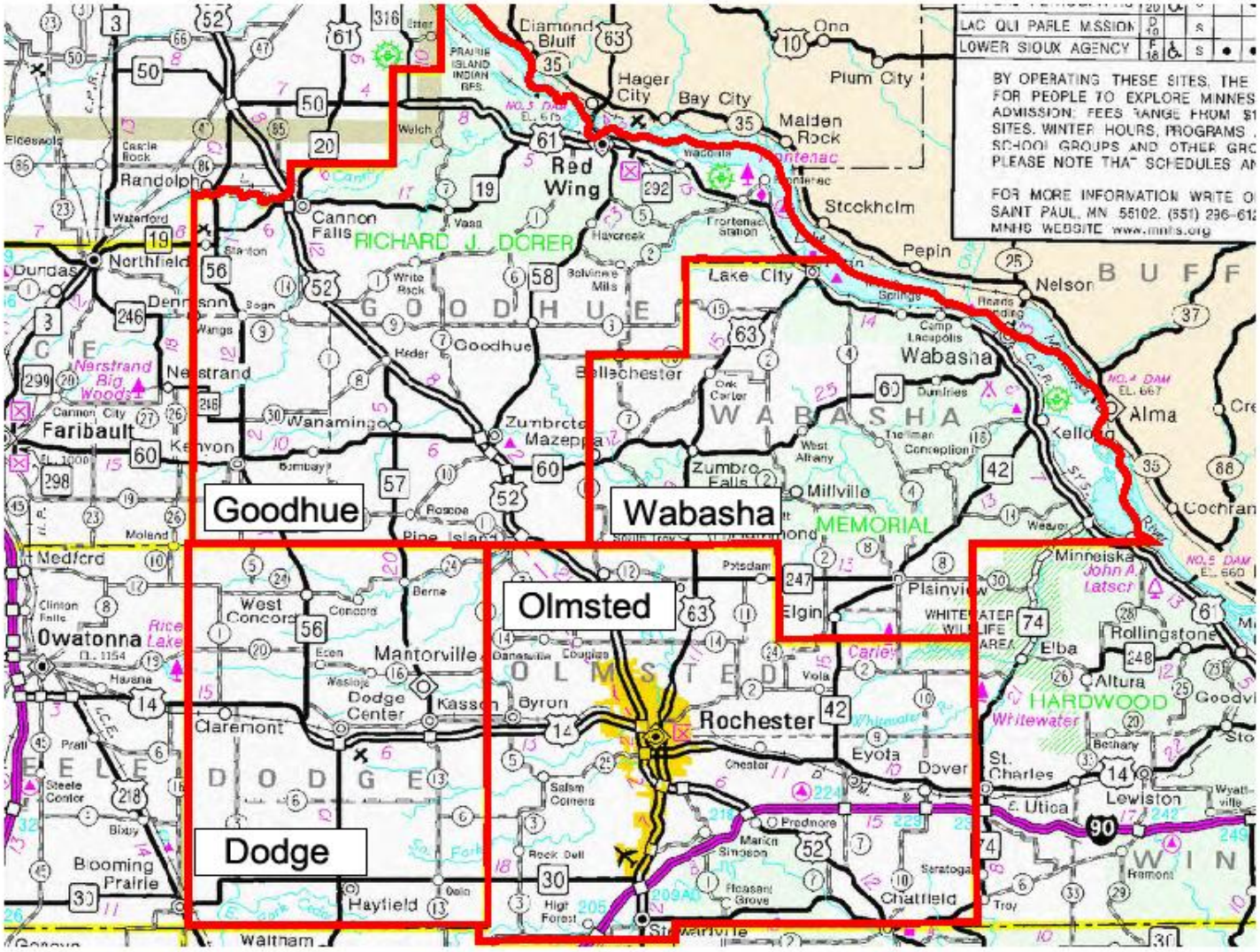

Figure 6. Four-county area in southeastern Minnesota where recreational feeding of wild white-tailed deer was banned in January 2011, following the discovery of chronic wasting disease in Olmsted County.

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

Michelle Carstensen[1], Robert Wright, Joao Ribeiro Lima[2], Louis Cornicelli, Eric Nelson, Scott Wells[2], and Marrett Grund

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 January 2011, 16 deer were captured by helicopter net-gunning and fitted with satellite-linked global positioning system (GPS) collars. A second, ground-based capture effort in March added 5 deer to the study to compensate for a high winter mortality rate (47%), caused primarily by wolf predation. Preliminary findings for the first 5 months of this 15-month study indicated a mean winter home range size for deer (n = 19) from mid-January through mid-June of 19.9 km^2 (SE = 5.4) and a mean minimum cumulative distance traveled of 97 km (± 13). Serological screening of deer at capture for 9 common cattle diseases indicated exposure to bovine parainfluenza 3 virus (PI3, 24%), malignant catarrhal fever (MCF, 19%), and infectious bovine rhinotracheitis (9%). Fecal parasitology analyses indicated 13 (65%) of deer had evidence of liver fluke (*Fascioloides magna*) infection and strongyle-type ova was detected in 4 (20%) deer. Analysis of deer use of agricultural landscapes is pending.

INTRODUCTION

The Minnesota Department of Natural Resources (MNDNR) and the University of Minnesota (UMN) are collaborating on a 15-month pilot study to gain a better understanding of movements and habitat use by white-tailed deer in northwestern Minnesota. This is an area where continuous changes of forest into a more agricultural landscape and deer use of this "transitional" habitat are not partiularly 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 are primarily interested in learning how deer use agricultural lands relative to state forest and wildlife management areas. In addition, we want to find out how farming practices, such as feed storage and animal husbandry, influence deer use of agricultural lands. This project intends to collect thousands of spatial locations of a small number of deer over 15 months. Utilizing this information to improve our understanding of how deer use farmed and pastured areas differently than natural habitats, we hope to gain greater 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 plans to quantify the microhabitat use of deer on farms, the potential for bTB transmission among cattle and deer, and to determine which herds are more likely to interact with deer 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 movements in northwestern Minnesota and possibly 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 facilitate further understanding of steps that can be taken to mitigate these risks.

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Secondarily, the location data ("fixes") stored on the radiocollars will allow the MNDNR to estimate home range sizes and dispersal rates, and describe migration patterns for the study animals. While we recognize that the results may not adequately represent the larger target deer population, they will provide wildlife managers and researchers with useful information and contribute to the design of 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). Within 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 to 20 deer/km^2. Major predators include gray wolves (*Canis lupus*), black bears (*Ursus americanus*), coyotes (*Canis latrans*), and bobcats (*Felis rufus*). Agricultural lands were surveyed to delineate and evaluate parameters that might attract deer to these areas (e.g., locations of stored forage, water sources, cattle pastures).

In winter 2011, deer were captured by helicopter net-gunning (Quicksilver Air, Inc., Fairbanks, Alaska) and Clover trap. We chemically immobilized (100 mg xylazine HCl, 400 mg ketamine HCl) captured deer and collected blood, urine, and fecal samples for health-screening. We also measured rump fat thickness by ultrasound and extracted a last lower incisor to determine exact age by counting cementum annuli (Mattson's Laboratory, Milltown, Montana). We ear-tagged and fitted deer with a satellite-linked radiocollar (ARGOS, SirTrack, Hawkes Bay, New Zealand). Body temperature was monitored at 5-min intervals throughout the processing period. We administered a long-acting antibiotic (LA-200, oxytetracycline) intramuscularly (1 mL/10 kg body weight). Before release, we reversed anesthesia by intravenous injection of 15 mg/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.

We programmed radiocollars 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 1 full year of seasonal movements). Collars were programmed to drop off in mid-April 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 study animals that die during the study period, MNDNR wildlife staff investigate the cause of mortality, recover the collar, and collect medial retropharyngeal lymph node samples from the deer (when possible) for bTB testing.

Serums were tested for malignant catarrhal fever via peroxidase-linked assay (PLA); positive PLA tests werethen tested with a virus neutralization test (VN) at the National Veterinary Services Laboratory (Ames, Iowa). All other serology was conducted at the UMN's Veterinary Diagnostic Laboratory (VDL) in St. Paul, Minnesota, which included screening for leptospirosis (6 serovars, microscopic agglutination test), anaplasmosis (card test), brucellosis (card test), and bovine parainfluenza 3 (hemagglutination inhibition test). Exposure to bluetongue virus and neosporosis were determined by enzyme-linked radioimmunoassay (ELISA). Exposure to bovine viral diarrhea (BVD, Types 1 and 2) and infectious bovine rhinotracheitis (IBR) were confirmed using serum neutralization tests (SN); titers ≥8 were considered positive. In addition, whole blood and serum were submitted to the UMN-College of Veterinary Medicine-Clinical Pathology Laboratory for a full large-animal serum chemistry profile and hematology; analyses of these results are pending.

We examined deer movements and made home range estimates using Home Range Tools (HRT) for ArcGIS® (Rodgers et al. 2007). Minimum convex polygons (MCPs) were constructing 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 will be performed to evaluate patterns of deer visits to farms throughout the 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 variations in home range characteristics 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; the intention is to identify 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 net-gunning within (n = 11) and slightly northeast (n = 5) of the study area (Figure 2). 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, 1 deer (ID 519) was censored from the study and its fate remains unknown. As of June 2011, 7 of these remaining deer (47%) were killed by wolves (n = 6) or died from unknown causes (n = 1) (Table 1).

To compensate for the high winter mortality, 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 triggering of the blow-off device) on 22 May 2011, and subsequently was censored from the study.

As of June 2011, 11 radiocollared deer remain in the study. The collars appear to be functioning 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. For collars that have been recovered, the success rate of obtaining fixes has been >95% (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 (Winter Severity Index [WSI] = 159, Red Lake Wildlife Management Area) in the study area, with prolonged snow cover of >36 cm 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 factors in northwestern Minnesota imposing a unique influence on on deer population dynamics different than in the farmland and forest zones.

Disease Screening and Parasitology

Serological results indicated deer were exposed to bovine parainfluenza 3 virus (24%), MCF (19%), and infectious bovine rhinotracheitis (9%). There was no evidence of exposure to anaplasmosis, bluetongue virus, bovine viral diarrhea (Types 1 or 2), brucellosis, leptospirosis,

or neosporosis. These tests only indicate deer have been exposed to these diseases, and thus, developed an immune response in which antibodies were detected through the various testing methods. We are not able to confirm current infection or illness from any of these diseases in these deer.

Exposure to PI3 in this study was not unexpected, as our prevalence was similar to the 20% reported by Ingebrigsten et al. (1986) for deer throughout Minnesota. Parainfluenza 3 virus is an RNA virus classified in the paramyxovirus family and is most commonly associated with cattle. Although PI3 is capable of causing disease, it is usually associated with mild to subclinical infections. The most important role of PI3 is to serve as an initiator that can lead to the development of secondary bacterial pneumonia. Little is known about PI3 infection in white-tailed deer. Thorsen et al. (1977) demonstrated PI3 was infective in both captive and free-ranging pronghorn (*Antilocapra americana*) in Alberta. In a serologic survey of wild cervids in national parks in the U. S., 58% of mule deer (*Odocoileus hemionus*) and 57% of elk (*Cervus elaphus*) were exposed to PI3 (Aguirre et al. 1995).

Our findings of 19% prevalence for MCF in deer is lower than what has been recently reported for wild elk in northwestern Minnesota (29%, Hildebrand et al. 2010) and northwestern moose (35%, Butler et al. 2010). Malignant catarrhal fever is caused by a Gammaherpes virus and affects many species in the family Artiodactyla (even-toed ungulates), including cattle, bison (*Bison bison*), deer, moose (*Alces alces*), exotic ruminants and pigs (*Sus scrofa domesticus*). At least 10 MCF viruses have been recognized worldwide, including 2 well-known viruses carried by sheep (*Ovis aries*) and wildebeest (*Connochaetes taurinus*); 5 MCF viruses have been linked to disease, while the others have been found, to date, only in asymptomatic carriers. The deer strain of MCF is typically carried asymptomatically, but it can cause disease in other susceptible species or in rare cases, in the reservoir host itself. In deer, MCF is usually acute and affected deer die within 1–2 days; however, more typically, MCF symptoms include corneal opacity, hemorrhagic diarrhea and bloody urine, shedding of the hoof in some animals, and death within 3 weeks of disease onset (Center for Food Security and Public Health 2008).

Infectious bovine rhinotracheitis is a highly contagious, infectious disease of cattle that is caused by bovine herpesvirus-1. Primarily a respiratory disease, IBR but can also cause conjunctivitis, abortions, encephalitis, and generalized system infections. Not much is known about IBR virus in deer. While we report a 9% prevalence, a higher prevalence (15%) was noted in a statewide serologic survey of Minnesota deer by Ingebrigsten et al. (1986). Infectious bovine rhinotracheitis exposure has also been reported in Minnesota's moose (Johnson et al. 1973). Sadi et al. (1991) reported a 57% prevalence of IBR in white-tailed deer on Anticosti Island (Quebec, Canada) and suggest it was the cause of an unusual mortality event among a 3–4 year-old cohort. While clinical signs associated with IBR in wild white-tailed deer are not known, researchers have demonstrated mild clinical signs in captive mule deer, including anorexia, depression, excessive salivation, increased respiratory rate, and occasional cough (Chow and Davis 1964).

Fecal samples from 20 deer were screened for evidence of parasites by fecal floatation. Thirteen (65%) of deer had evidence of liver fluke (*Fascioloides magna*) infection and strongyle-type ova were detected in 4 (20%) deer. Negative results do not necessarily mean the animal was parasite-free, only that it was not actively shedding at the time the feces were collected. Also, culture of fecal samples did not detect any evidence of Johne's disease (*Mycobacterium paratuberculosis*).

Home Range Size and Deer Movements

Mean home range size for all deer (n = 19) from mid-January through mid-June was 19.9 km^2 (SE = 5.4) and the mean cumulative distance traveled was 97 km (SE = 13). However, since deer were captured during mid-late winter, we are uncertain whether or not this represents solely winter ranges for these deer or also included all or portions of their spring-summer-fall ranges. Further, while deer that died (or slipped their collar) during the study had similar mean home range sizes to survivors (14 km^2 ± 6.7 and 24 km^2 ± 8.0, respectively;

Figures 3 and 4), the mean cumulative distance traveled by survivors was nearly twice as high as those that died (Tables 2 and 3), likely due primarily to being tracked over a longer time period. Six deer had home ranges >36 km^2,attributable to a few long-distance movements from one end of their range to the other (Figures 3 and 4). These movements began in late January for 3 deer, moving 11–21 km in a 2–3-day period. The other 3 deer moved 14–21 km in mid- to late March, again over a 2–3 day period. Of these 6 deer, 2 were killed, but the other 4 returned the same distance (in a 2–3 day period of travel) to the area in which they were originally captured in late March or early April. However, the majority of deer (63%) had home ranges ≤ 10 km^2.

Given the timing of deer capture (mid-January and early March), we assumed these animals were either on their winter range (if migratory) or were possibly year-round residents at the start of the study. Therefore, it is too early in the study for a thorough interpretation of the deer movement and home range data generated thus far. 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 5.2 km^2 and 2.6 km^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 most 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

No results have been generated yet, as only 5 months of spatial data are available. Analysis will occur at the completion of the 15-month project.

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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, northwestern 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		
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		
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		
496	1/15/11	Helicopter	Adult	2.5	F	Dead	unknown	2/23/11
472	1/15/11	Helicopter	Adult	5.5	F	Alive		
524	1/15/11	Helicopter	Adult	6.5	F	Dead	wolf-kill	3/10/11
473	1/15/11	Helicopter	Adult	4.5	M	Alive		
495	1/15/11	Helicopter	Adult	2.5	M	Alive		
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		
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	Unknown	collar malfunction	
350	1/16/11	Helicopter	Adult	11.5	F	Alive		
336	3/7/11	Clover-trap	Yearling		M	Alive		
578	3/8/11	Clover-trap	Adult		F	Alive		
577[3]	3/8/11	Clover-trap	Adult		F	Dead	wolf-kill	4/10/11
579	3/8/11	Clover-trap	Adult		F	Alive		
447	3/10/11	Clover-trap	Adult		F	Unknown	slipped collar	

[1]Age (in years) was determined by cementum annuli. Analysis for deer captured in March is pending.
[2]F = female, M = male
[3]Deer 577 was fitted with a SirTrack test-collar (Iridium satellite system) and no movement data was recovered; mortality date are based on a public report of a severely injured deer and carcass remains.

Table 2. Fix success rates, home range size, and cumulative distance traveled by free-ranging deer (*n* = 11) remaining in the study, as of June 14, 2011, northwestern Minnesota.

Deer ID	Days on air	No. fixes[1]	Fix success rate[2] (%)	99% MCP[3] (km^2)	Cumulative distance traveled (km)
469	150	941	39.2	38.1	209.5
497	150	748	31.2	84.4	162.7
466	150	635	26.5	1.0	87.4
472	150	983	41.0	18.4	151.1
473	150	616	25.7	5.7	82.9
495	150	627	26.1	10.4	123.4
491	149	905	38.0	54.9	137.8
350	149	900	37.8	36.5	111.2
336	99	397	25.1	8.5	128.3
578	98	500	31.9	2.1	121.3
579	98	526	33.5	8.3	67.8
Mean	136	707	32.4	24.3	125.7
SE	7	60	1.8	8.0	12.1

[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 of 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, home range size, and distance traveled by free-ranging deer (*n* = 8) 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)	Cumulative distance traveled (km)
461	77	1325	8	99.4	40.1	111
467	43	774	7	99.1	1.0	18
496	43	773	17	97.8	0.5	14
524	61	1124	4	99.6	8.0	53
471	90	1693	82	95.2	10.4	128
348	28	517	13	97.5	47.9	29
460	43	763	24	96.8	0.3	9
447	89	1641	68	95.9	4.1	100
Mean	59	1076	28	97.7	14.0	58
SE	8	156	11	0.6	6.7	17.1

[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.

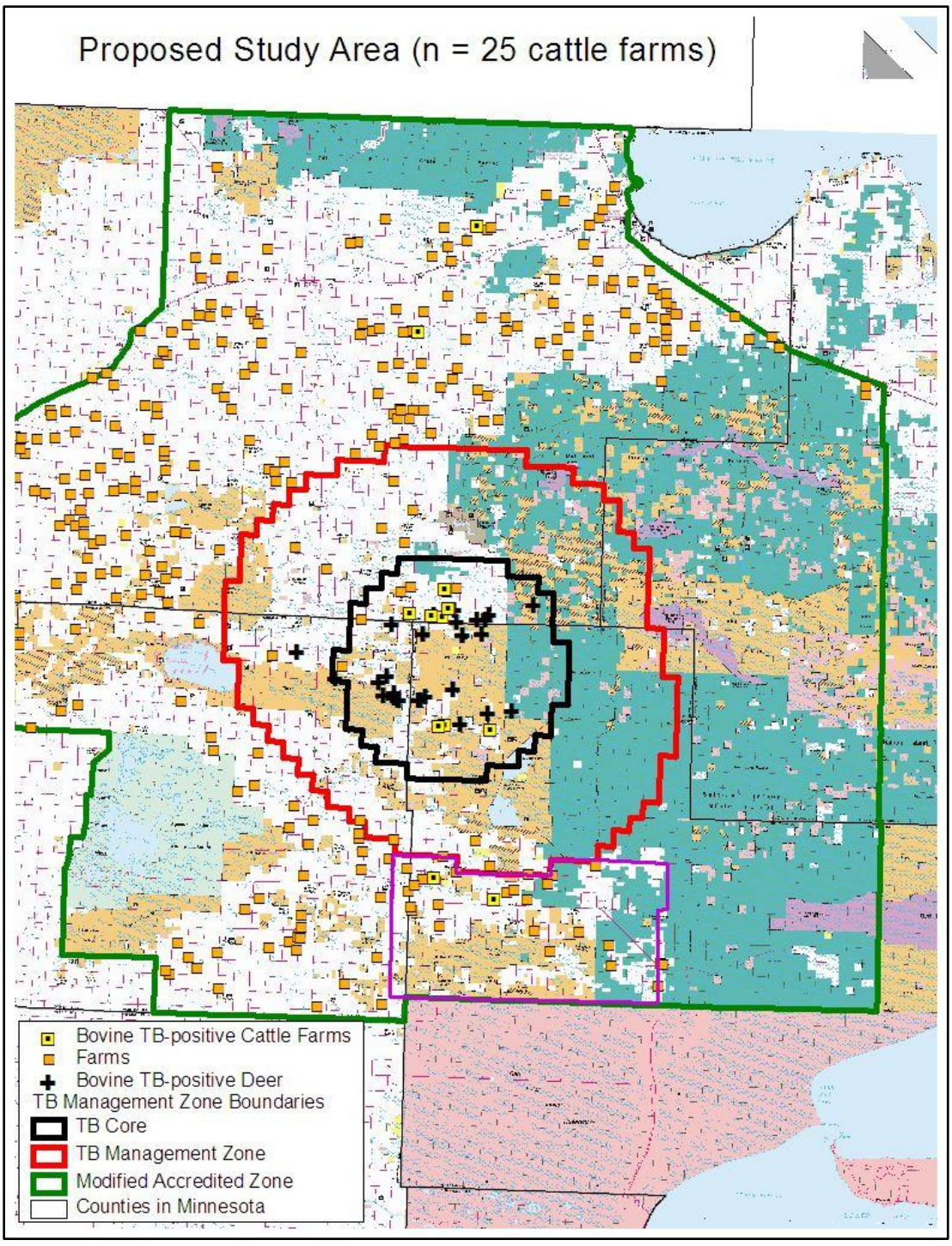

Figure 1. The 360-km^2-study area (outlined in purple) contains >25 cattle farms including 2 previously infected with bovine tuberculosis. The study area is immediately south of the Bovine Tuberculosis Management Zone, where 27 deer and 8 cattle farms tested positive for the disease.

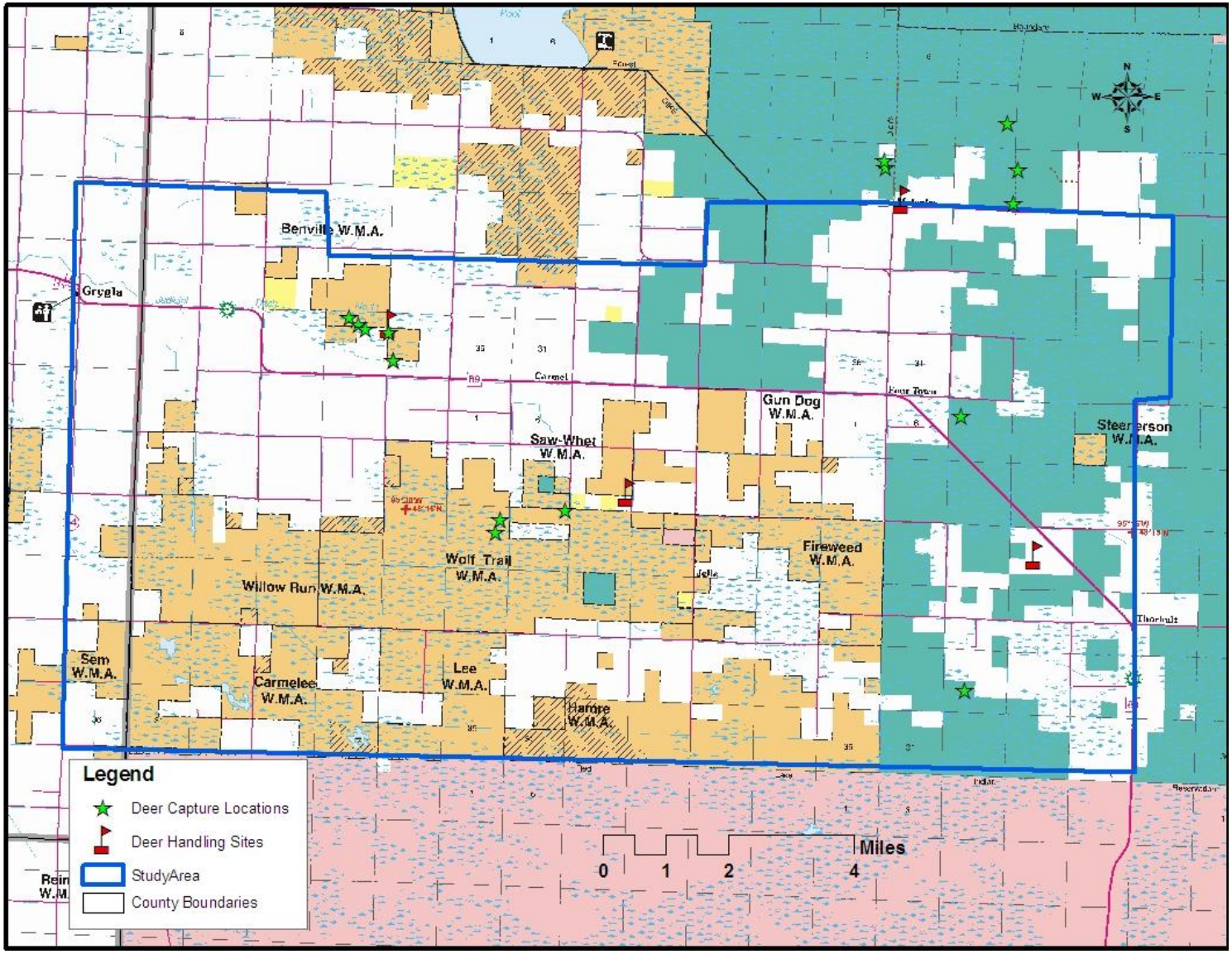

Figure 2. Capture locations and handling sites for free-ranging white-tailed deer (*n* = 16) captured by helicopter net-gunning in January 2011, northwestern Minnesota.

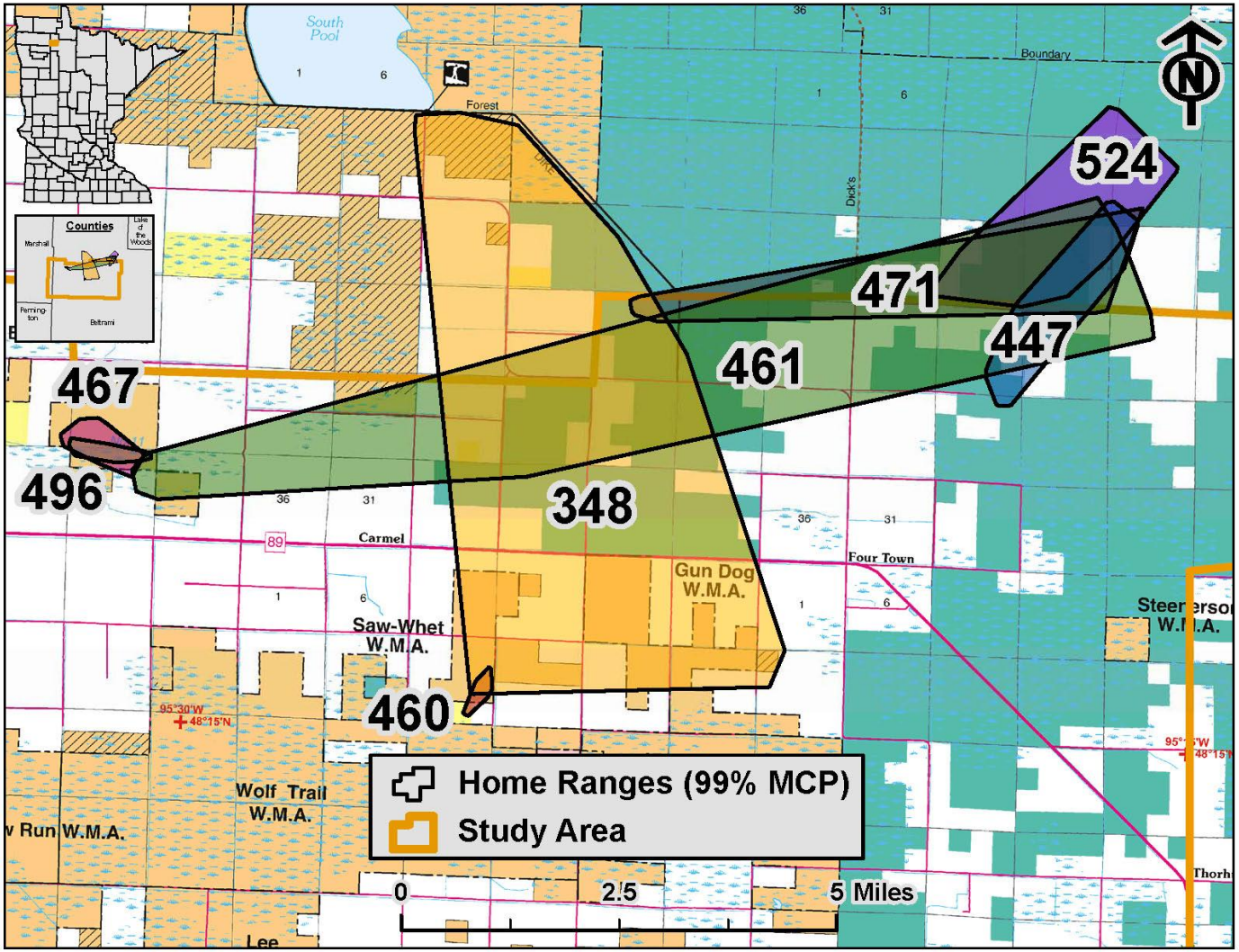

Figure 3. Home ranges, determined by 99% minimum convex polygons, for white-tailed deer (*n* = 8) that died or slipped their radiocollar during the study period, January–April 2011, northwestern Minnesota.

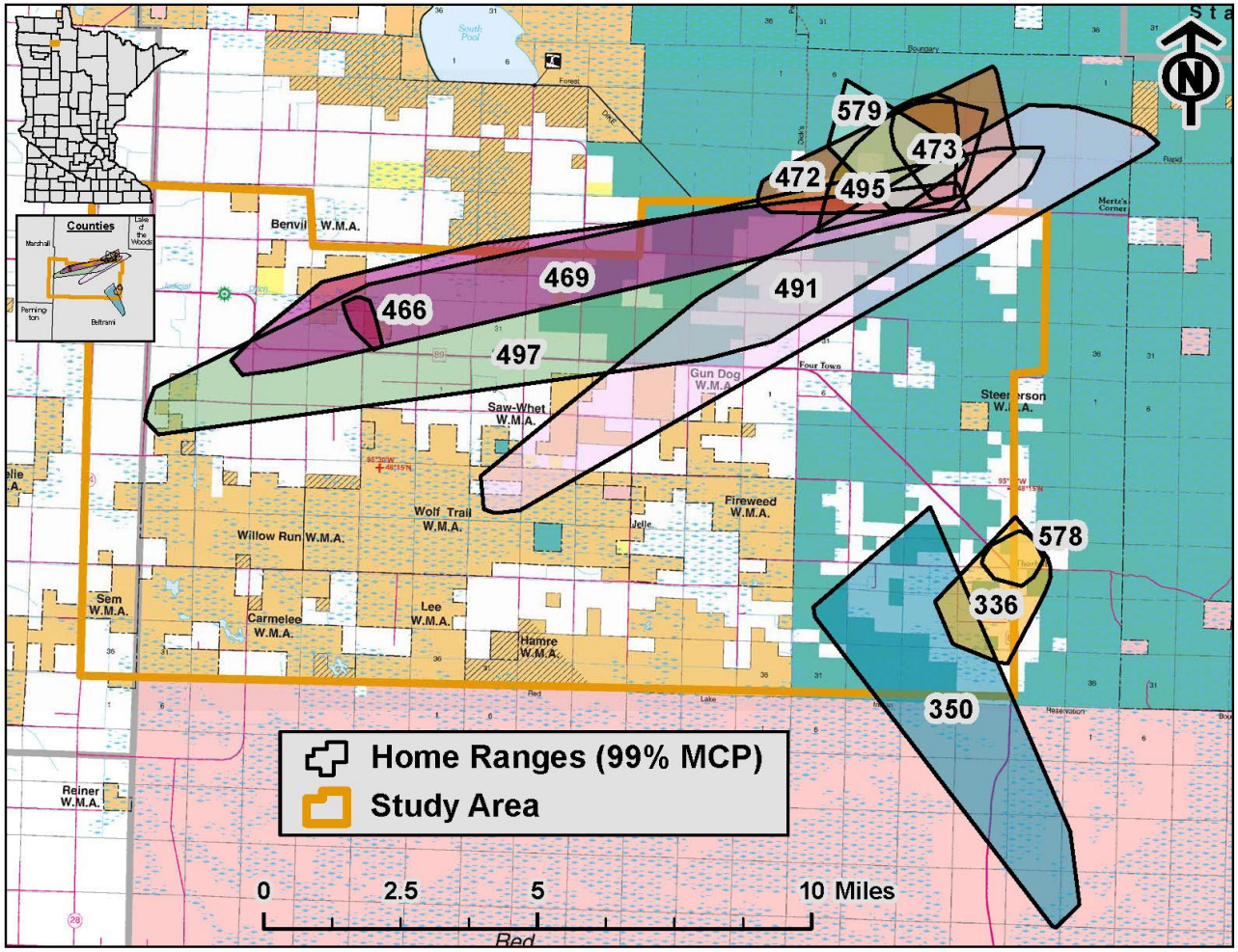

Figure 4. Home ranges, determined by 99% minimum convex polygons, for white-tailed deer (*n* = 11) alive as of 14 June 2011, northwest Minnesota.

MANAGING BOVINE TUBERCULOSIS IN WHITE-TAILED DEER IN NORTHWESTERN MINNESOTA: A 2010 PROGRESS REPORT

Michelle Carstensen[1], Erika Butler, Erik Hildebrand, and Louis Cornicelli

SUMMARY OF FINDINGS

Bovine tuberculosis (bTB), first detected in northwestern Minnesota in 2005, has since been found in 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. In response to the disease being detected in cattle, the Minnesota Department of Natural Resources (MNDNR) began surveillance efforts in free-ranging white-tailed deer within a 24-km (15-mi) radius of the infected farms in fall 2005. To date, 26 of the 27 deer infected with bTB were sampled within a 425-km^2 (164-mi$^{2)}$ area, called the bTB Core, which is centered in Skime, Minnesota, and encompasses 8 of the previously infected cattle farms. In total, 1,639 hunter-harvested deer were tested for bTB in northwestern Minnesota during fall 2010, with no positive cases detected. This marks the first year that no new infected cases were detected in wild deer. An aerial survey estimated the population of the bTB Core to be 531 (SE = 95) deer in January 2011. The absence of new infected deer resulted in a suspension of targeted removal operations using ground sharpshooting over winter. A recreational feeding ban, instituted in November 2006 in a 10,360-km^2 (4,000-mi^2) region in northwestern MN to help reduce the risk of deer to deer transmission of the disease, remains in effect. Under a current agreement among the United States Department of Agriculture (USDA), BAH, and MNDNR, hunter-harvested deer surveillance will continue to monitor infection in the local deer population, and any further aggressive management actions (e.g., sharpshooting deer in key locations) will be dependent on future surveillance results.

INTRODUCTION

Bovine tuberculosis 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 northwestern 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 (6,915 km^2 [2,670 mi^2]) in northwestern 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 northwestern MN, which can be attributed to a spillover of the disease from infected cattle. In 2010, The USDA upgraded Minnesota's bTB accreditation to Modified Accredited Advanced within the split-state zone and bTB-free throughout the remainder of the state. Although bTB was once relatively common in U. S. cattle, historically, it has 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 eating contaminated feed. Incubation periods

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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. 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 2010, we developed a fall hunter-harvested surveillance strategy to meet the sampling goals established in a recently renegotiated Memorandum of Understanding (MOU) between the USDA and both the MNDNR and BAH. It requires 1,000 deer to be tested for bTB within the Modified Accredited Advanced Zone (MAAZ). Distribution of these samples was to include 500 from within the bTB Management Zone and 500 from the area outside the bTB Management Zone, but within the MAAZ. The MNDNR further defined these goals to specify that the 500-sample goal from within the bTB Management Zone must include at least 200 samples from the bTB Core Area.

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 the 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. Six 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. All samples were then pooled in groups of 5 and sent to the National Veterinary Services Laboratories (NVSL) in Ames, Iowa 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 2010, we collected 1,639 samples from hunter-harvested deer; 1,437 samples from within the MAAZ and 202 samples outside the zone (Figure 2). Thus, MNDNR collected nearly 1.5x the overall sampling goal set forth by the MOU with USDA. Further, the sampling distribution met the guidelines of the MOU for samples collected within the bTB Management Zone (n = 575) and outside this zone, but within the MAAZ (n = 862) (Figure 2). The MNDNR achieved 92% of the specified goal of collecting at least 200 samples from within the bTB Core Area.

Testing of all lymph node samples at NVSL confirmed that there were no positive cases of bTB detected during the fall 2010 surveillance. Thus, 2010 marks the first complete year (including winter 2010 sharpshooting in the bTB Core Area) 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 4,481–6,915-km^2 (1,730–2,670 mi^2) Surveillance Zone, indicates a significant decreasing trend from 2006 to 2010 (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 TB-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 531 (SE = 95 deer, Figure 1). This was slightly higher than the February 2010 population estimate of 422 (SE = 64; Figure 4, Table 2). Aggressive deer removal in the bTB Core Area by liberalized hunting, disease management permits, landowner shooting permits, and targeted sharpshooting allowed us to reduce the deer population in this 425-km^2 (164-mi^2) area by approximately 55% from 2006 to 2010, but clearly, maintaining deer numbers at a low level will remain difficult. 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 moderately severe winter of 2010–2011 may have played a role in increased deer movement into the bTB Core Area, which provides good wintering habitat, and might explain the slight increase in estimated deer numbers.

The proximity of the TB-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 9,783 deer in the northwest since 2005;27 were 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 (*Cervus elaphus*) was instituted over a 10,360-km^2 (4,000-mi^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.

As part of the requirements to regain bTB-Free accreditation, the USDA has required BAH to test all cattle herds within the Modified Accredited Advanced Zone annually, with additional movement restrictions for farms located within the bTB Management Zone. The BAH has submitted an application for status upgrade to USDA, and a decision is expected by September 2011. If approved, Minnesota would regain its bTB-free status throughout the entire state, removing our current split-state status entirely. What this will mean for continued surveillance in both cattle and deer is unknown. The MNDNR is committed to assisting BAH in regaining and maintaining Minnesota's bTB-free status. The MNDNR will conduct fall hunter-harvested surveillance in 2011, although surveillance goals and a timeline for continued surveillance beyond 2011 will likely be negotiated with USDA this fall.

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Carstensen, M., and M. W. DonCarlos. 2011. Preventing the establishing of a wildlife disease reservoir: a case study of bovine tuberculosis in wild deer in Minnesota, USA. Veterinary Medicine International, Volume 2011, Article ID 413240, 10 pages, doi:10.4061/2011/413240.

Table 1. Number of deer sampled for bovine tuberculosis (TB) and testing results listed by sampling strategy, 2005 2010, northwestern Minnesota.

Sampling strategy	2005	2006	2007	2008	2009	2010	Totals
Hunter-harvested (Oct-Jan)	474	942	1,166	1,246	1,488	1,639	6,955
# TB-positive	1	5	5	0	1	0	
Apparent prevalence (%)	0.21	0.53	0.43	0.0	0.07	0.0	
Sharpshooting (Feb-May)	0	0	488	937	738	450	2,613
# TB-positive			6	6	2	0	
Apparent prevelance			1.23	0.64	0.27	0.0	
Landowner/tenant	0	90	0	125	0	0	215
# TB-positive		1		0			
Total deer tested	474	1,032	1,654	2,308	2,226	2,089	9,783
Total # TB-positive	1	6	11	6	3	0	27

Table 2. Population estimates and 95% confidence intervals of deer within the Bovine Tuberculosis Core Area, 2007–2011, northwestern Minnesota.[a,b]

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	2.70	3.2	0.30	2.7–3.8	531	48.6	436–627	9.2	18.0

[a]Population estimate = estimated *minimum* number of deer present during the sampling interval. Estimates are not adjusted for sightability (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% confidence intervals (CI) are based on sampling variance only (adjusted for spatial correlation in 2008–2011); they do not include uncertainty associated with sightabilty or animal movements (temporal variation due to animals moving onto or off the study area).

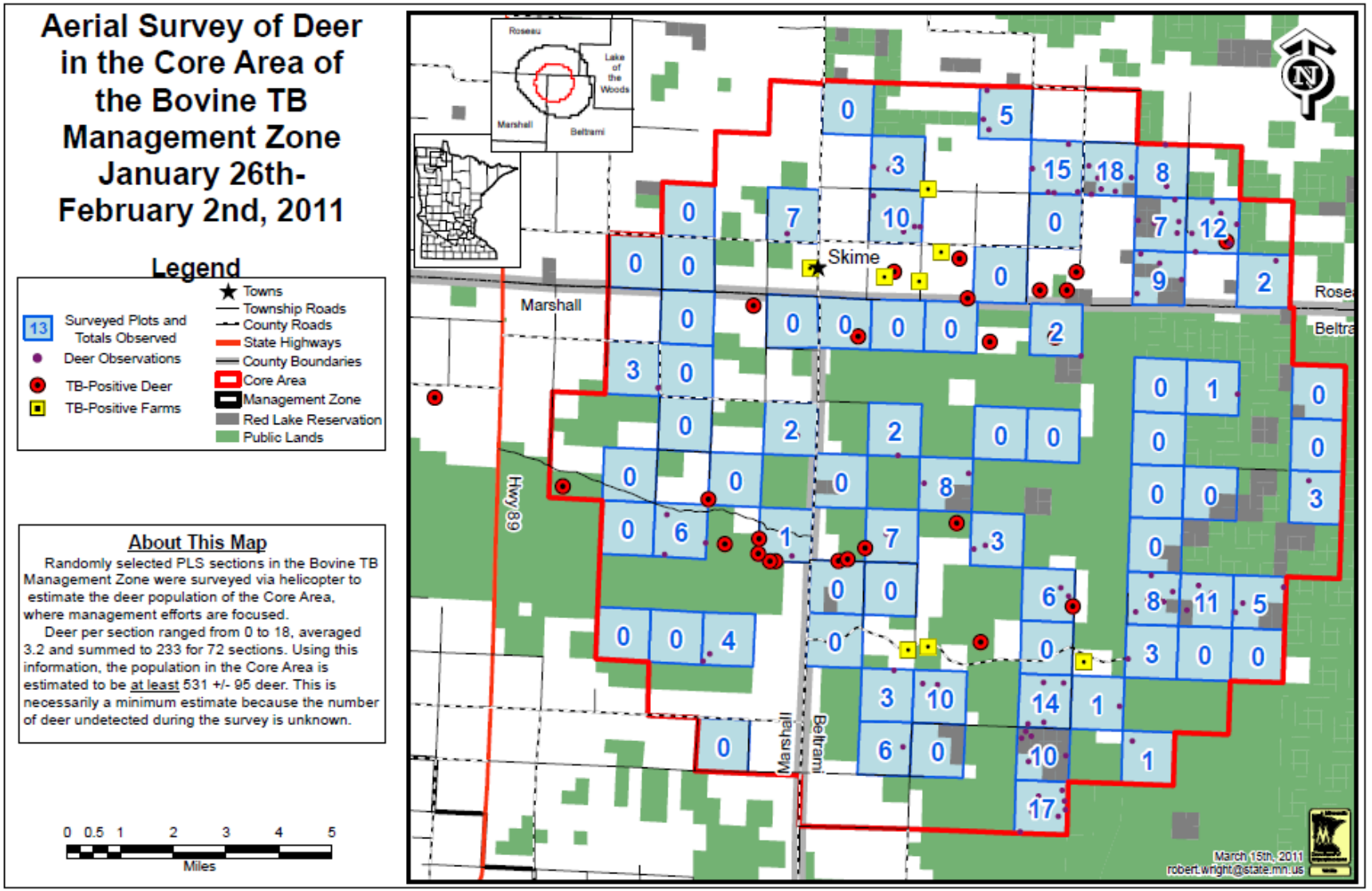


Figure 1. Results of aerial white-tailed deer survey of the Bovine Tuberculosis Core Area in January 2011, northwestern Minnesota.

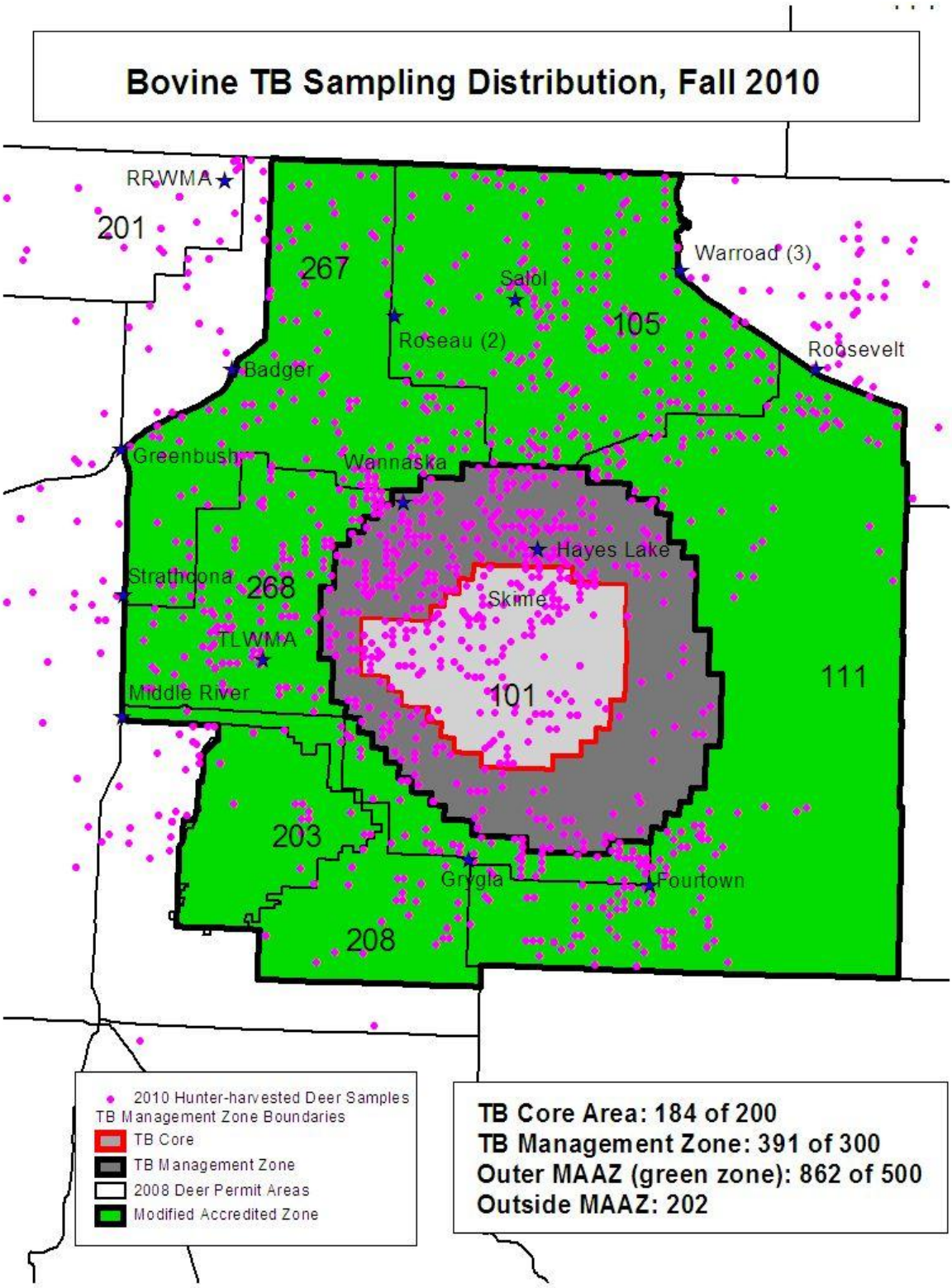
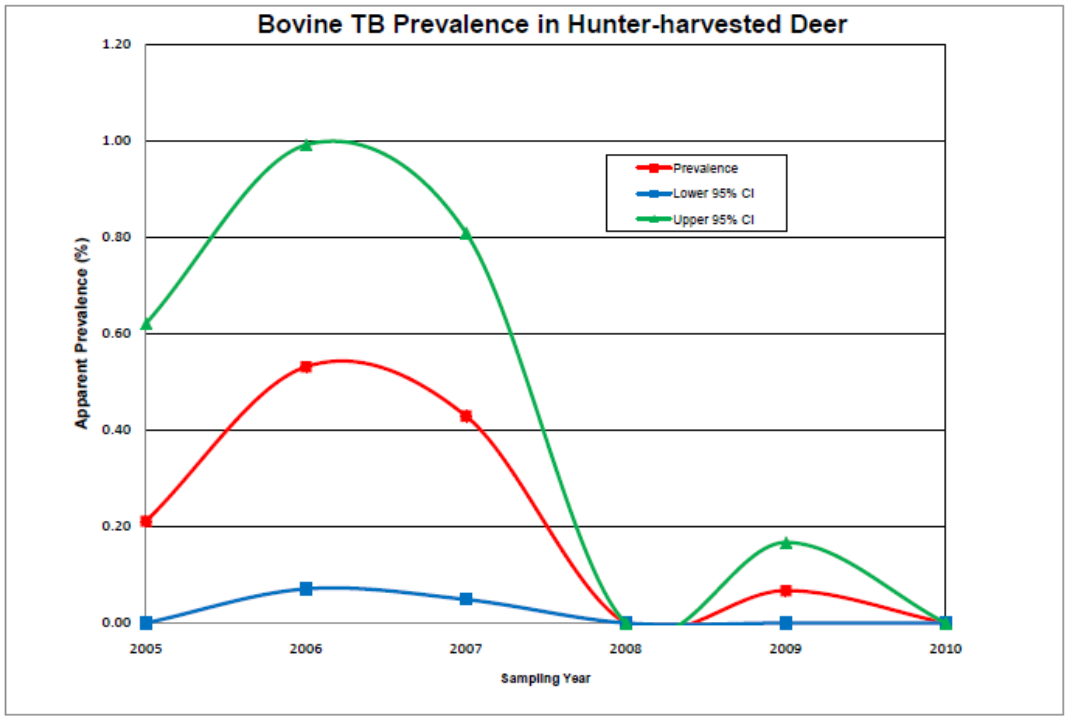


Figure 2. Locations of hunter-harvested deer (*n* = 1,639) sampled for bovine tuberculosis (TB) during fall 2010 in northwestern Minnesota.



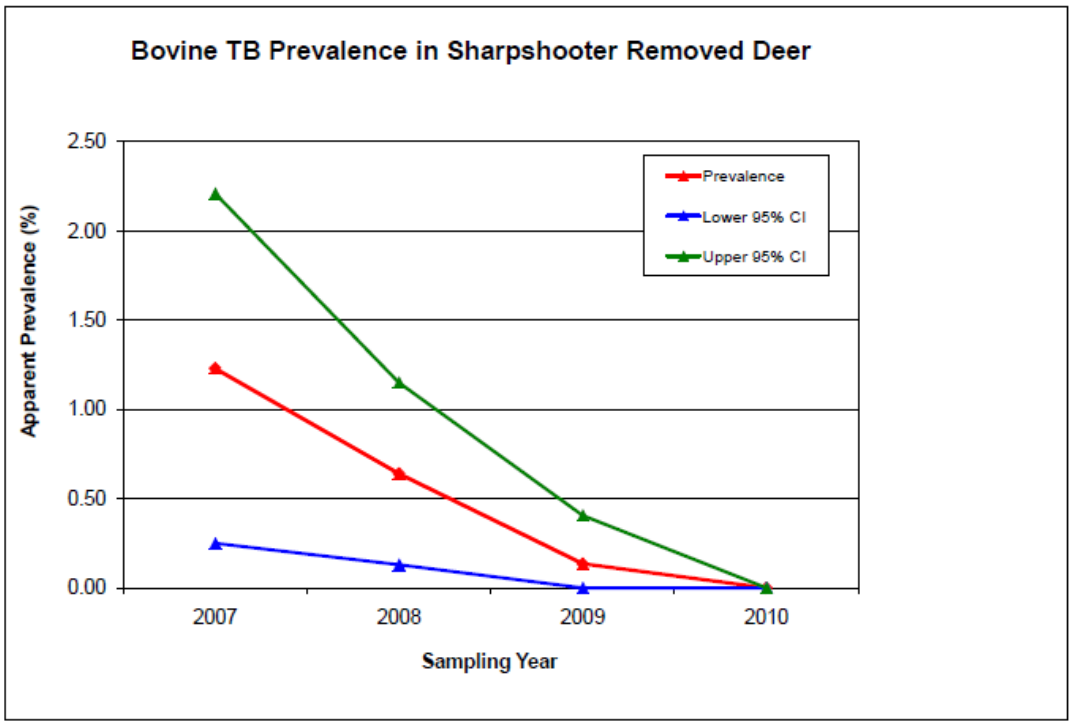


Figure 3. Prevalence of bovine tuberculosis (TB) in hunter-harvested deer from 2005 to 2010 in the BovineTB Surveillance Zone and disease prevalence from sharpshooter removed deer from 2007 to 2010 in the Bovine TB Core Area, northwestern Minnesota.

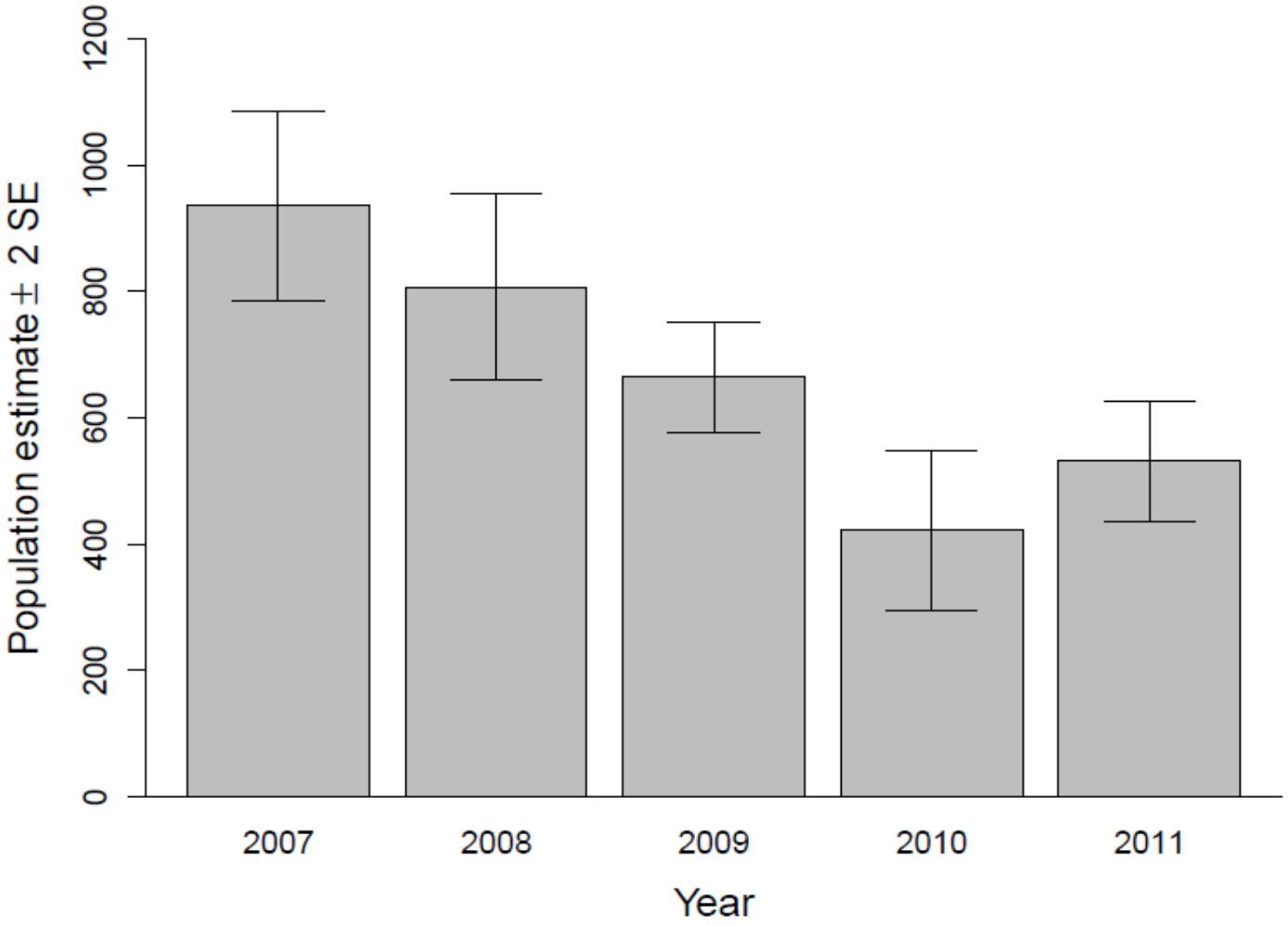


Figure 4. Population estimate of deer within the Bovine Tuberculosis Core Area, winters 2007–2011, northwestern Minnesota.

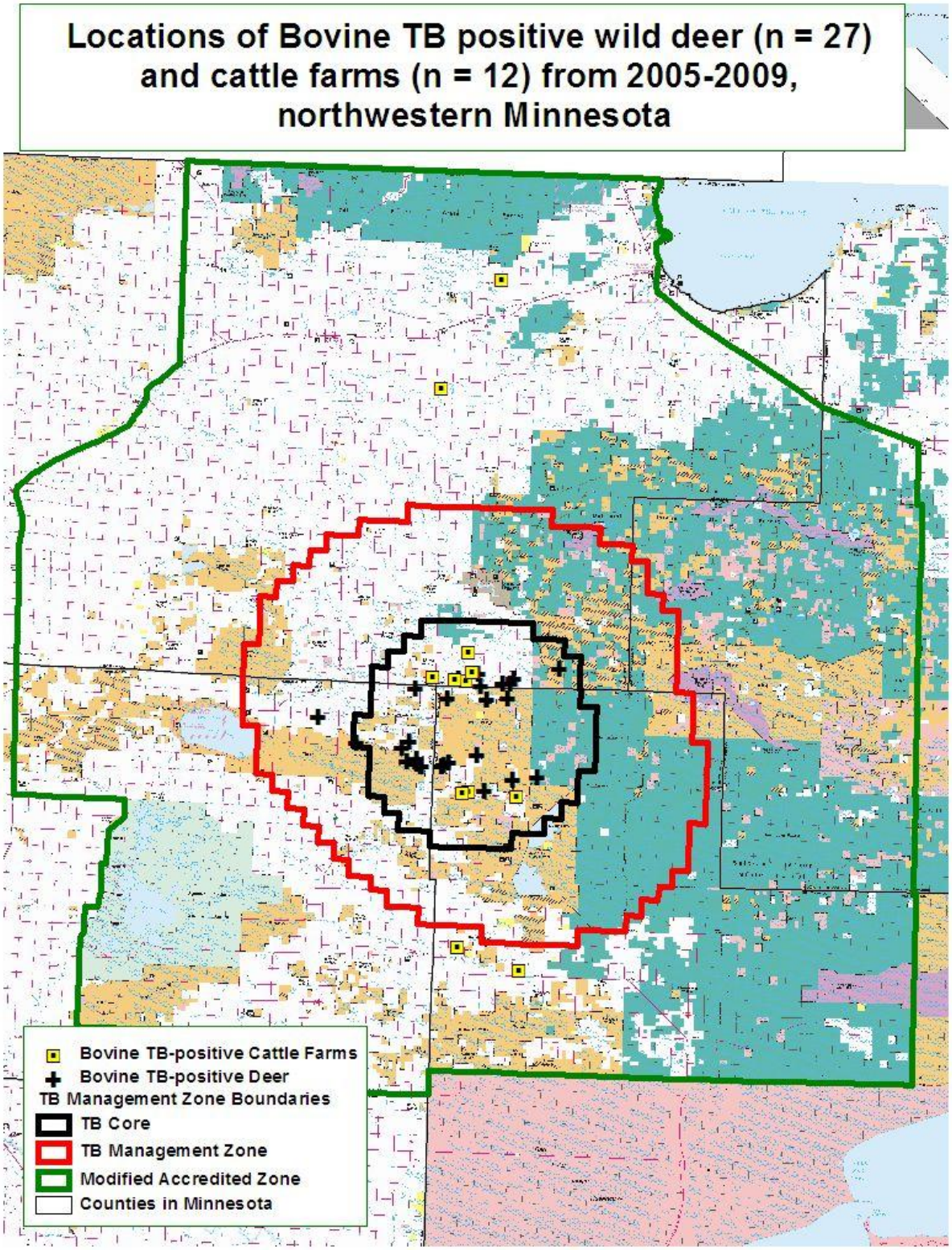
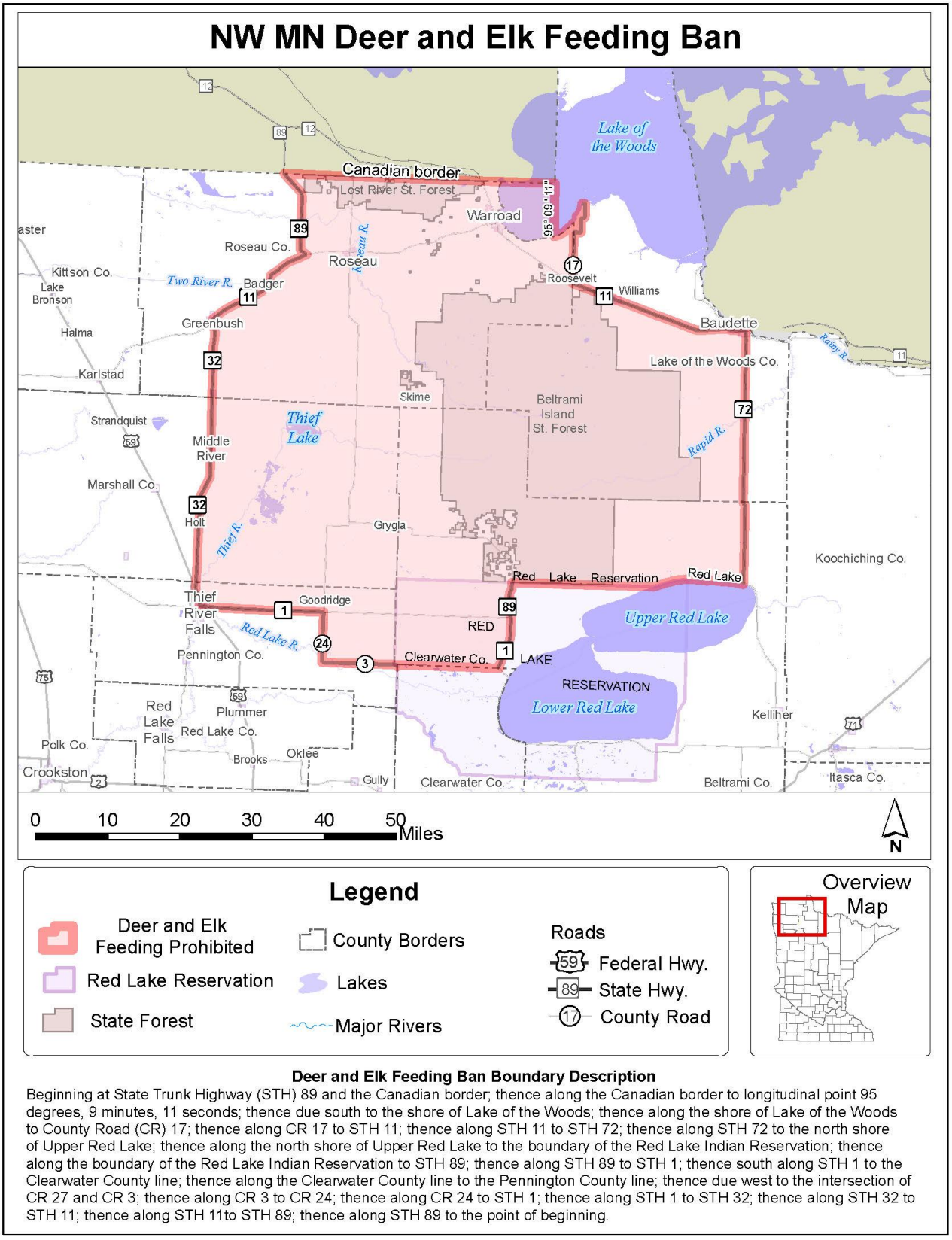


Figure 5. Locations of all white-tailed deer found infected (*n* = 27) with bovine tuberculosis (TB) since fall 2005 in northwestern Minnesota; the 12 previously-infected cattle operations are included.



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

PREVENTING THE ESTABLISHMENT OF A WILDLIFE DISEASE RESERVOIR: A CASE STUDY OF BOVINE TUBERCULOSIS IN WILD DEER IN MINNESOTA, USA[1]

Michelle Carstensen and Michael W. DonCarlos

ABSTRACT

Bovine tuberculosis (bTB) has been found in 12 cattle operations and 27 free-ranging white-tailed deer (*Odocoileus virginianus*) in northwestern Minnesota, following the state's most recent outbreak of the disease in 2005 in the northwestern part of the state. Both deer and cattle have the same strain of bTB. The Minnesota Board of Animal Health has been leading efforts to eradicate the disease in Minnesota's cattle, which have included the depopulation of all infected herds, a cattle buy-out program, and mandatory fencing of stored feeds. The Minnesota Department of Natural Resources (MNDNR) began surveillance efforts in free-ranging white-tailed deer in fall 2005. All bTB-infected deer have been found within a 16-km^2 area in direct association with infected cattle farms. Aggressive efforts to reduce deer densities through liberalized hunting and sharpshooting have resulted in a 55% decline in deer densities. Also, recreational feeding of wild deer has been banned. Disease prevalence in deer has decreased from 1.2% in 2005 to an undetectable level in 2010. Minnesota's primary goal has been the eradication of bTB from both deer and cattle. The aim of this paper is to describe the primary management strategies implemented by MNDNR to prevent the establishment of a wildlife disease reservoir in free-ranging white-tailed deer. These strategies included, (1) rapid response to initial disease detection, (2) follow-through on monitoring the outbreak with adequate surveillance, (3) recognizing when monitoring must switch to management, (4) aggressively reducing transmission potential by reducing deer densities, limiting recreational feeding and mitigating risks at the cattle-wildlife interface, and (5) evaluation of efforts and adjusting as needed.

[1]From published paper: Carstensen, M., and M. W. DonCarlos. 2011. Preventing the establishment of a wildlife disease reservoir: a case study of bovine tuberculosis in wild deer in Minnesota, USA. Veterinary Medicine International, Volume 2011, Article ID 413240, 10 pages, doi:10.4061/2011/413240

PUBLIC ACCEPTANCE AS A DETERMINANT OF MANAGEMENT STRATEGIES FOR BOVINE TUBERCULOSIS IN FREE-RANGING U.S. WILDLIFE[1]

Michelle Carstensen, Daniel J. O'Brien[2], and Stephen M. Schmitt[2]

ABSTRACT

When bovine tuberculosis (bTB) is detected in free-ranging wildlife populations, preventing geographic spread and the establishment of a wildlife reservoir requires a rapid, often aggressive response. Public tolerance can exert a significant effect on potential control measures available to managers, and thus on the success of disease management efforts. Separate outbreaks of bTB in free-ranging white-tailed deer (*Odocoileus virginianus*) in 2 midwestern states provide a case study. In Minnesota, bTB was first discovered in cattle in 2005 and subsequently in deer. To date, 12 beef cattle farms and 26 white-tailed deer have been found infected with the disease. From 2005 to 2008, disease prevalence in deer has decreased from 0.4% (SE = 0.2%) to < 0.1% and remained confined to a small (< 425 km^2) geographic area. Deer population reduction through liberalized hunting and targeted culling by ground sharpshooting and aerial gunning, combined with a prohibition on baiting and recreational feeding, have likely been major drivers preventing disease spread thus far. Without support from cattle producers, deer hunters and the general public, as well as politicians, implementation of these aggressive strategies by state and federal authorities would not have been possible. In contrast, Michigan first discovered bovine bTB in free-ranging deer in 1975, and disease management efforts were not instituted until 1995. The first infected cattle herd was diagnosed in 1998. Since 1995, disease prevalence in free-ranging deer has decreased from 4.9% to 1.8% in the 1500-km^2 core outbreak area. Culture positive deer have been found as far as 188 km from the core area. Liberalized harvest and restrictions on baiting and feeding have facilitated substantial reductions in prevalence. However, there has been little support on the part of hunters, farmers or the general public for more aggressive population reduction measures such as culling, and compliance with baiting and feeding restrictions has been variable and often problematic. We compare and contrast the Minnesota and Michigan outbreaks with respect to temporal, social, economic, and logistical factors that shape public attitudes toward aggressive disease control strategies, the limitations these factors place on management, and the implications for bTB eradication from wildlife reservoirs in the U. S.

[1] Abstract from published paper: Carstensen, M., D. J. O'Brien, and S. M. Schmitt. 2011. Public acceptance as a determinant of management strategies for bovine tuberculosis in free-ranging U. S. wildlife. Veterinary Microbiology 151:200–204, doi:10.1016/j.vetmic.2011.02.046

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