



## **DETERMINING CAUSE- SPECIFIC MORTALITY IN MINNESOTA'S NORTHEAST MOOSE POPULATION**

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### **SUMMARY OF FINDINGS**

The primary goal of this study is to improve our understanding of non-anthropogenic (i.e., health-related) mortality of the northeastern Minnesota's declining moose population. Our objectives are to determine causes of non-hunting mortality (i.e., identify specific disease or parasite agents, predation), by responding to mortalities within 24 hours of death, prior to decomposition of tissues. In the first 17 months of this multiyear study, we've captured and radio collared 142 adult moose (mean age = 6 years, range 1 to 14) and a total of 35 collared moose have died since this study began, which included 7 capture-related mortalities that will be censored from subsequent survival analyses. Overall causes of death are as follows: 15 predator-related mortalities (54%), which includes 9 wolf kills, 3 likely wolf kills, and 3 wolf-caused injuries that led to secondary lethal bacterial infections; and 13 health-related mortalities (46%), which includes 3 brainworm (*Parelaphostrongylus tenuis*) infection, 3 winter tick (*Dermacentor albipictus*) infestations, 1 liver fluke (*Fasciola hepatica*) infection with secondary bacterial infection, 1 trauma with associated septicemia, 1 accident (drowning), 1 kidney disease with cachexia, and 3 undetermined health issues. Whole carcasses were retrieved for 9 of these moose, with field necropsies performed on remaining 19 animals. Response times from initial mortality notification (e.g. text message or email) to a team in the field at the death site were  $\leq 24$  hours in 16 cases (58%), between 24 and 48 hours in 6 cases (21%), and  $>48$  hours in 6 cases (21%). Delays in mortality responses  $>24$  hours have been due to collar or technology failures and wolves actively feeding on the moose carcass and thus preventing the collar from sending a mortality alert.

### **INTRODUCTION**

Historically, moose were found throughout the forested zone of north central Minnesota. By the 1960's there were two distinct populations, the northwest (NW) population of the aspen parklands and northeast (NE) population of the boreal forest (Fuller 1986). In the mid-1980's the NW population began a precipitous decline, falling from 4,000 to  $<100$  animals by the early 2000's (Murray et al. 2006, Lenarz 2007). Murray et al. (2006) identified pathogens, including liver flukes (*Fascioloides magna*) and brainworm (*Paralaphostrongylus tenuis*), as the principal cause of death for 37-62% of radio-collared animals; 25% of additional mortalities were likely pathogen-induced, but limited necropsy evidence was inconclusive. They also observed that many moose in NW MN dying of natural causes were malnourished, as evidenced by 51% of

carcasses having bone marrow fat (BMF) contents below a critical threshold (< 30%) and trace mineral deficiencies (i.e., copper and selenium).

Subsequently, in NE MN, Lenarz et al. (2009) reported a 21% average non-hunting mortality rate for radiocollared moose, which was much higher than the 8-12% reported for moose elsewhere in North America (Larsen et al. 1989, Ballard 1991, Kufeld and Bowden 1996). Specific causes of most of the non-anthropogenic mortality (89%) could not be determined, as assessing cause-specific mortality was not the primary objective of the study (Lenarz et al. 2009). Many of the deaths appeared health-related, with prime age animals dying during unusual times of the year or carcasses found intact with little evidence of scavenging.

Aerial surveys also indicate the NE population is declining. Since the estimated peak at 8,840 moose in 2006, the 2014 estimated moose population (4,350) is 50% lower and time series analysis of estimates since 2005 indicate a significant downward trend (DelGiudice 2013). Butler et al. (2013a) documented evidence of exposure of NE MN moose to a variety of disease agents (e.g., West Nile Virus, eastern equine encephalitis, malignant catarrhal fever), which could be potential mortality factors. Additionally, a recent study of sick and vehicle-killed moose ( $n=62$ ) from 2003-2013 had found 85% of animals were undernourished and infected with a variety of disease agents, including brainworm (45%), liver flukes (60%), and winter ticks (*Dermacentor albipictus*) (22%) (Wuenschmann et al., 2014). 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 currently represents a major threat to the NE MN population; clinical signs consistent with brainworm infection were first reported in MN moose in 1912 (Fenstermacher and Olson 1942). Lenarz et al. (unpublished data) found that brainworm may have caused an average 19% (0-32%) of the population's total annual mortality.

## METHODS

Moose were captured within the study area (Fig. 1) by aerial darting (Quicksilver Air Inc., Alaska) with carfentanil (4.5mg or 6.0mg), thyaferentanil (16mg) and xylazine (150mg or 30mg) from a helicopter; immobilizations were reversed with naltrexone (425-575mg) and tolazoline (400mg). Blood (serum and whole blood) was collected at capture by venipuncture of the jugular vein. Serum was screened for evidence of exposure to 10 disease agents following the same protocol as described by Butler et al. (2012b). Additionally, serum was submitted for a large animal serum chemistry profile and reproductive hormones to assess physiological status, overall health, and pregnancy status (Franzmann and LeResche 1978, Haig et al. 1982, Duncan et al. 1994). Serum progesterone levels were determined by the Smithsonian Institute; levels >2.0 ng/mL were considered pregnant. Whole blood in Ethylenediaminetetraacetic acid (EDTA) was used to make blood smears and complete and differential blood cell counts were performed, which may be indicative of condition and health status (Duncan et al. 1994), presence of tick-borne illnesses, and evaluation for the presence of microfilaria. An incisor (I4) was removed for aging by cementum annuli (Sergeant and Pimlott 1959). A general fecal floatation examination for parasites was performed. A thorough physical examination was performed, including assessment of body condition score (very thin, thin, normal, fat), winter tick load, and hair loss. Total body length and girth (cm) were measured to estimate body weight of

moose (Hundertmark and Schwartz, 1998) and hair samples were collected from the withers. Any mortalities that occurred within two weeks of capture were censored from the study.

Moose were fitted with mortality-sensing collars utilizing Global Positioning System (GPS) and Iridium two-way communication technologies (Vectronic Aerospace GmbH; Berlin, Germany). Collars transmit location and status data to a base station (Forest Lake, MN) at user-defined intervals. The base station also analyzes location data to identify animals that have “localized” (e.g., remained within a 20m radius for >24 hours), to assist with detecting sick animals that are potentially moribund. When a mortality or localization event occurred, the mortality response team was notified via text and email messages. Mortality implant transmitters (MITs; Vectronic Aerospace GmbH) were placed orally into the reticulum of a subset of the captured moose. These devices are similar to a cow magnet in size, log internal temperatures every 15 minutes, and transmit a subset of this data through the collar. Additionally, MITs are meant to provide immediate notification of mortality via detection of minimal internal activity (e.g., lack of a heart beat) and this notification is also sent via text and email message to the moose mortality response team. External temperature loggers (Hobo TibdbitV2; Onset Corporation, Bourne, MA) were affixed to the GPS collar and were programmed to collect ambient temperature every 60 minutes.

Moose mortality response teams have 8 primary team leaders that have undergone extensive necropsy training, and they are supported by about 30 secondary and tertiary team members (including Minnesota Department of Natural Resources [MNDNR], tribal, academic, and other personnel) available upon request. Every effort is made to remove carcasses intact from the field and deliver them to the University of Minnesota Veterinary Diagnostic Laboratory (UMN VDL) for a complete necropsy by a board-certified pathologist. Teams are able to utilize special equipment, including trucks with 2000lb winches, an amphibious ARGO, chainsaw winch (e.g. Lewis winch), heavy duty snow machines with long tracks, all-terrain vehicles, and specialized rubber mats with built-in hitches for dragging carcasses. Primary members have also taken specialized training with DNR forestry and fire units to be able to sling out a moose carcass via helicopter. If a moose was found to be alive, but obviously ill, it was euthanized (via gunshot). If carcass extraction is not possible, a thorough and complete field necropsy was performed, guided by an established protocol, and samples were submitted to the UMN VDL for diagnostic evaluation (Butler et al., 2013b).

## **RESULTS AND DISCUSSION**

### **Overall survival and timing of mortalities**

A total of 28 collared moose have died since this study began; which excludes 7 capture-related mortalities that are censored from subsequent survival analyses. Overall survival rate is 74.6% from February 4, 2013 to June 30, 2014. Causes of death are as follows: 15 predator-related mortalities (54%), which includes 9 wolf kills, 3 likely wolf kills, and 3 wolf-caused injuries that led to secondary lethal bacterial infections; and 13 health-related mortalities (46%), which includes 3 *P. tenuis* infection, 3 winter ticks, 1 liver fluke infection with secondary bacterial infection, 1 trauma with associate septicemia, 1 accident, 1 was cachectic with kidney disease and endometriosis, and 3 undetermined health issues (Fig 2). Timing of these mortalities

suggest that most deaths occur in spring (64%, March–May); however, moose died in all seasons (Winter 18%, Summer 14%, and Fall 4%) (Fig 3).

### Annual variation in survival

The annual survival rate for adult moose ( $n=107$ ) in Year 1 (4 February, 2013–5 February, 2014) of this study was 80% (95%CI = 72.0 to 87.9%; Fig 4). A total of 21 collared moose (17 females, 4 males) died. There were 78 moose remaining with active collars, excluding censored animals ( $n=12$ ) at the end of Year 1. Causes of death are as follows: 11 predator-related mortalities (52%), which includes 6 wolf kills (3 of which had predisposing health conditions that likely contributed to their death), 3 likely wolf kills, and 2 wolf-caused injuries that led to secondary lethal bacterial infections; and 10 health-related mortalities (48%), which includes 2 *P. tenuis* infection, 3 winter ticks, 1 liver fluke infection with secondary bacterial infection, 1 trauma with associate septicemia, and 3 undetermined health issues. Whole carcasses were retrieved for 8 of these moose, with field necropsies performed on the remainder. A total of 12 collared moose were censored in Year 1 due to capture-related mortalities ( $n=4$ ), slipped collar ( $n=1$ ), and collars that stopped transmitting ( $n=7$ ).

From February 6-11, 2014, 31 additional moose (12 males, 19 females) and 5 female moose with failed collars (originally captured in 2013) were captured and recruited into the study. Sample size for the start of Year 2 was 104 collared moose. As of June 30, 2014, survival rate is 93.4% (Fig 4). A total of 7 collared moose (6 females, 1 male) have died. Causes of death are as follows: 4 predator-related mortalities (57%), which includes 3 wolf kills and 1 wolf-caused injury that led to a secondary lethal bacterial infection; and 3 health-related mortalities (43%), which includes 1 *P. tenuis* infection, 1 accident (fell through the ice and drowned), and 1 moose that was cachectic with kidney disease and endometriosis. Nine collared moose from Year 2 are censored thus far, which includes 3 capture-related mortalities, 1 slipped collar, and 5 collars that stopped transmitting. Thus far in Year 2 (from capture to 30 June) moose survival is 2x higher than the same time period in Year 1. It appears that winter survival was enhanced by the prolonged winter 2013, which may have suppressed winter tick numbers, as we have not reported any winter tick mortalities during winter 2014. Also, the historically severe winter of 2014 likely reduced deer numbers in our study area, which would lessen disease exposure risks of moose to *P. tenuis* and liver flukes.

### Serologic and Parasite Screening at Capture

Progesterone levels from blood samples obtained during 2014 captures indicated a 77% pregnancy rate; similar to 75% from last winter (Butler et al., 2013b). Biological samples obtained during 2014 captures were analyzed and serologic evidence of exposure to West Nile Virus (8 of 34, 23.5%), malignant catarrhal fever (1 of 34, 2.9%), various serovars of *Leptospira interrogans* (6 of 34, 17.6%) and *Borrelia* (2 of 34, 5.9%) was documented. Fecal floatation was used to screen moose at capture for parasites, and 3 of 28 (11%) were identified as having infections. These parasites included *Nematodirus* sp. (2 moose) and coinfections with Strongyle-type ova (1 moose). Analyses of the CBC and serum chemistry results are pending. Mean age of captured moose was 5.3 years ( $n=23$ , range 1 to 13). The body condition score of each moose was evaluated and recorded whenever possible; 6 were classified as thin (16.7%), 21 as normal (58%), and 9 as fat (25%). None of the animals were classified as very thin. Comparatively, during 2013 captures, a third of all moose captured were either thin or very thin (Butler et al. 2013b). Estimated mean body mass at capture was 395kg ( $n=29$ , range 336-461) in Year 2, compared to 388kg ( $n=99$ , range 269-522) in Year 1.

## **Mortality Response Times**

Whole carcasses were retrieved for 12 of these moose, with field necropsies performed on the remaining 23 moose. Response times from initial mortality notification (e.g. text message or email) to a team in the field at the death site were  $\leq 24$  hours in 20 cases (57%), between 24 and 48 hours in 9 cases (26%), and  $>48$  hours in 6 cases (17%). Delays in mortality responses  $> 24$  hours have been due to collar failures and wolves actively feeding on the moose carcass and thus preventing the collar from sending mortality alert.

## **Collar and MIT Functioning**

Of the 100 collars originally purchased by the MNDNR, 41 have either had mortality signal malfunctions (e.g., collars being locked in mortality mode) or stopped transmitting entirely, and have been replaced under warranty by the manufacturer. The collars locked in mortality (LIM) mode are transmitting locational data; however, staff have to monitor these animals ( $n=21$ ) daily to ensure movement is occurring. To assist with this more intensive monitoring, LIM collars collect and transmit data more frequently and the localization function evaluates movements more often (12 hour intervals). The localization function has increased our ability to recognize animals that are moribund, but are not actually dead, allowing us to euthanize the animal. This allows never before documented clinical signs to be observed (e.g., teeth grinding in a *P. tenuis* infected moose) and key samples (e.g., fresh blood) to be collected, which is vital when trying to determine cause of death. This function has also helped us identify wolf-kills faster, as wolves will feed under the collar and prevent it from going into mortality mode until they have left the carcass.

Initially, we deployed 28 MITs in Year 1 and their functionality is as follows: 4 MITs were spit out by the moose immediately after deployment, 2 stopped functioning after 14 months (presumably failed batteries), 6 were recovered in dead moose, and 16 are currently actively deployed and operational. In Year 2, we attempted to deploy additional MITs and 12 were spit out by moose immediately after deployment (in some cases, capture team witnessed this and recovered MIT) and 18 were successfully deployed. Currently, there are 32 radio collared moose with functioning MITs alive in the study.

## **Preliminary Animal Migration Behavior Analyses**

Year 1 moose movement data were analyzed to assess migration behavior. Out of 70 active moose that survived through the first year of the study, 34% ( $n=24$ ; 17 females, 7 males) migrated to a distinct spring-summer-fall (SSF) range. Average distance from winter range to SSF range was 24km (12.8km for females, 19.9km for males). Average SSF home range (MCP) was 23.8km<sup>2</sup> for females, and 51.8km<sup>2</sup> for males. Average winter home range (MCP) was 11.2km<sup>2</sup> for females and 47.5km<sup>2</sup> for males. Average home range (MCP) for non-migrating moose ( $n=46$ ) was 74.3km<sup>2</sup>. A preliminary analyses of cover types within these moose home ranges suggest that woody wetlands are used the most by moose at all times of the year, but far

more (21.6% higher) in the summer than in winter; moose are using deciduous and coniferous more in the winter. Further analyses of habitat use and movement data are pending.

## **Funding and Future Project Direction**

This \$1.2 million dollar project initially began through funding from the Legislative Citizen Commission on Minnesota's Resources (LCCMR; \$600,000) and in-kind contributions from the MNDNR, University of Minnesota, tribal partners, and nonprofit organizations (e.g., MDHA, Northstar Museum, and Save Minnesota Moose). Additional funding through LCCMR was recently secured for \$600,000 to continue the study and expand on the use of MITs to gain insight at the potential role ambient temperature may play in moose survival and productivity. Sample size of at least 100 collared adult moose will be maintained through 2015 and 2016 by capturing new individuals as needed over the next 2 winters. Further, 30 additional MITs will be deployed. New methods for successful MIT delivery and a calibration of the internal temperature data will be developed in conjunction with the Moose Research Center in Alaska in 2015.

## **ACKNOWLEDGEMENTS**

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Figure 1. Study area in northeast Minnesota where 142 moose have been captured and radio collared (2013–2014) to study cause-specific mortality.

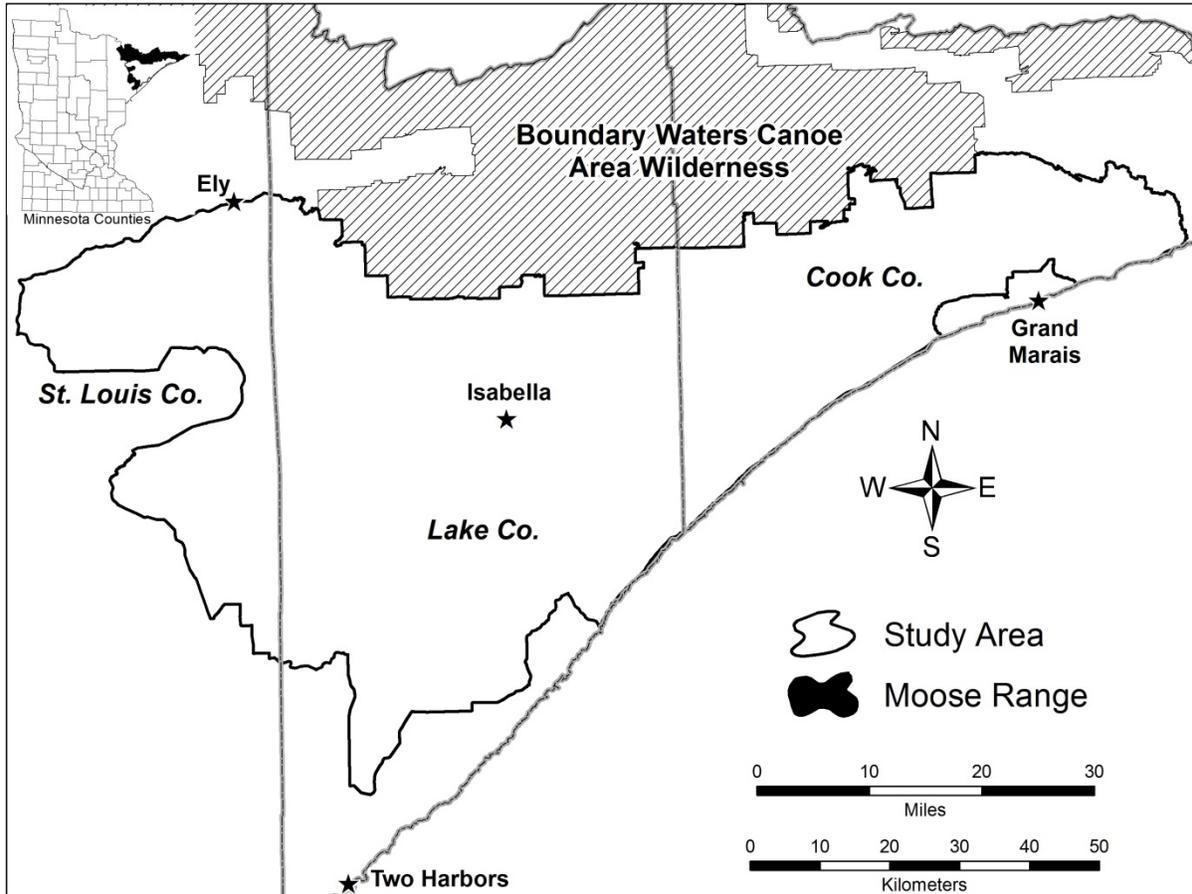


Figure 2. Cause-specific mortality of radiocollared, adult moose ( $n=28$ ) from February 2013 to June 2014, northeast Minnesota.

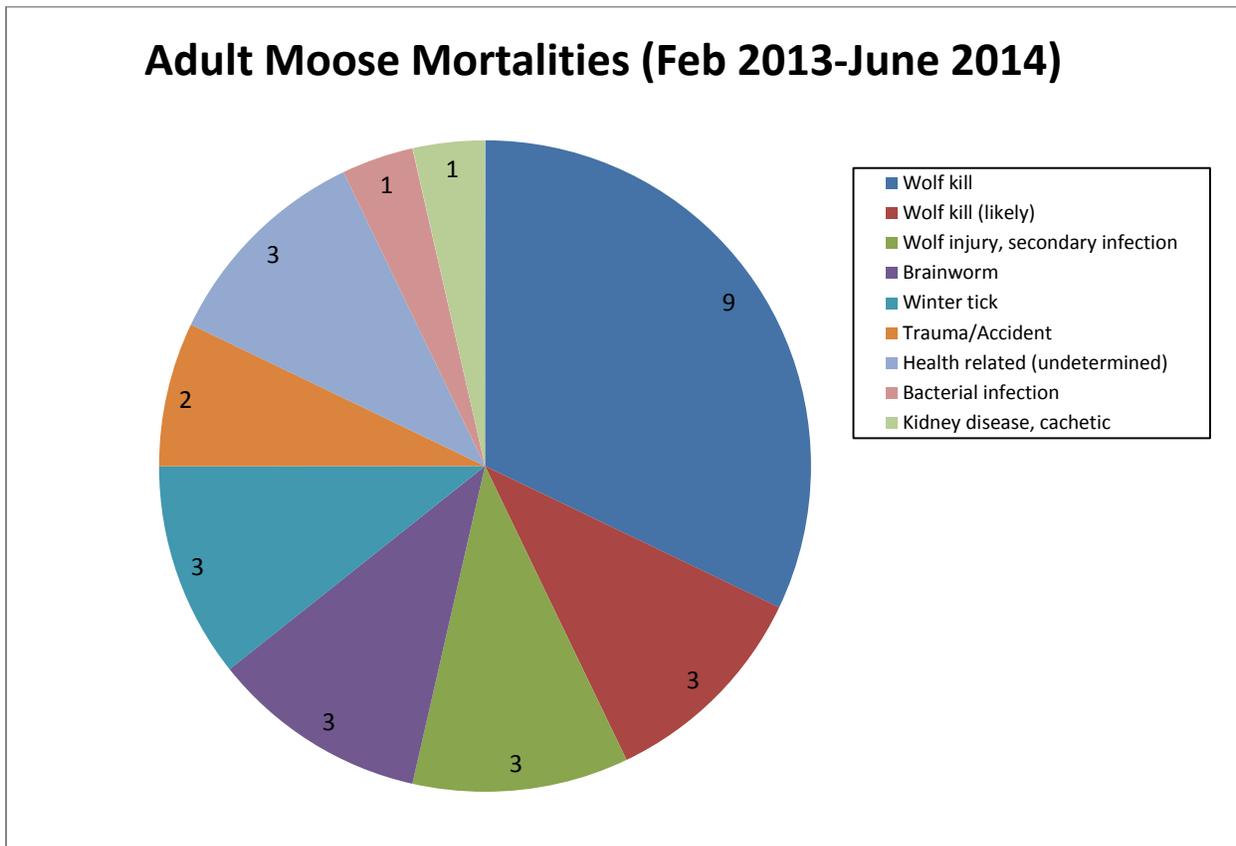


Figure 3. Timing of mortalities for adult moose ( $n=28$ ) from February 2013 through June 2014, northeast Minnesota.

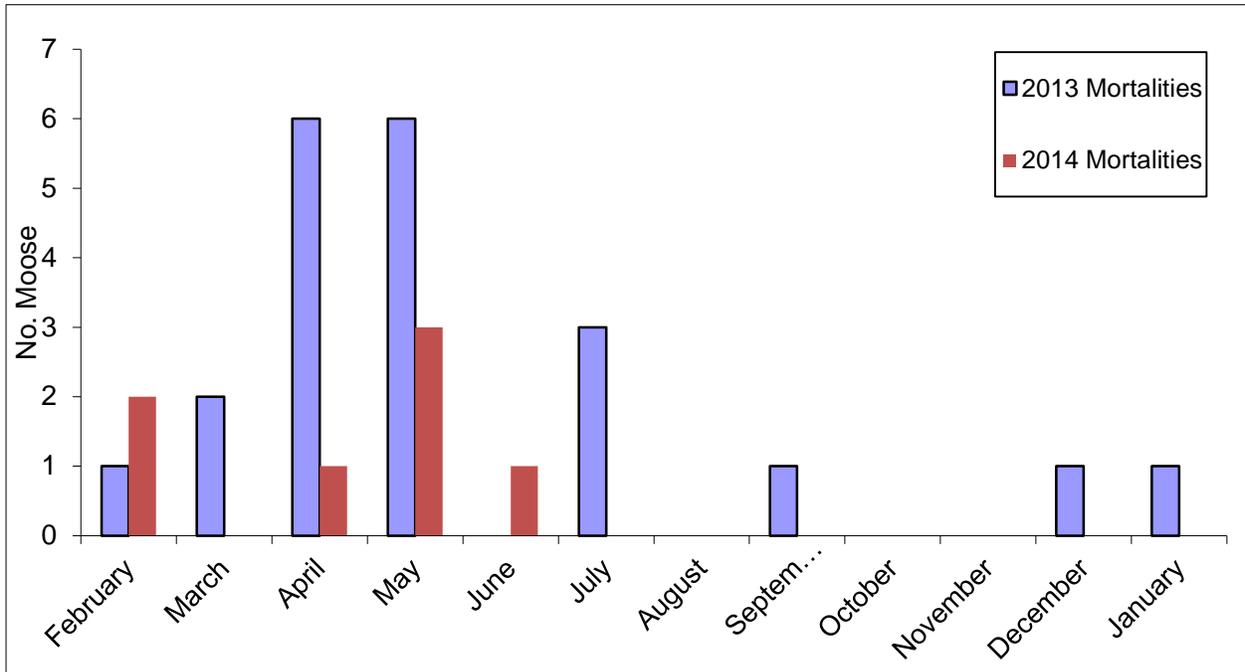
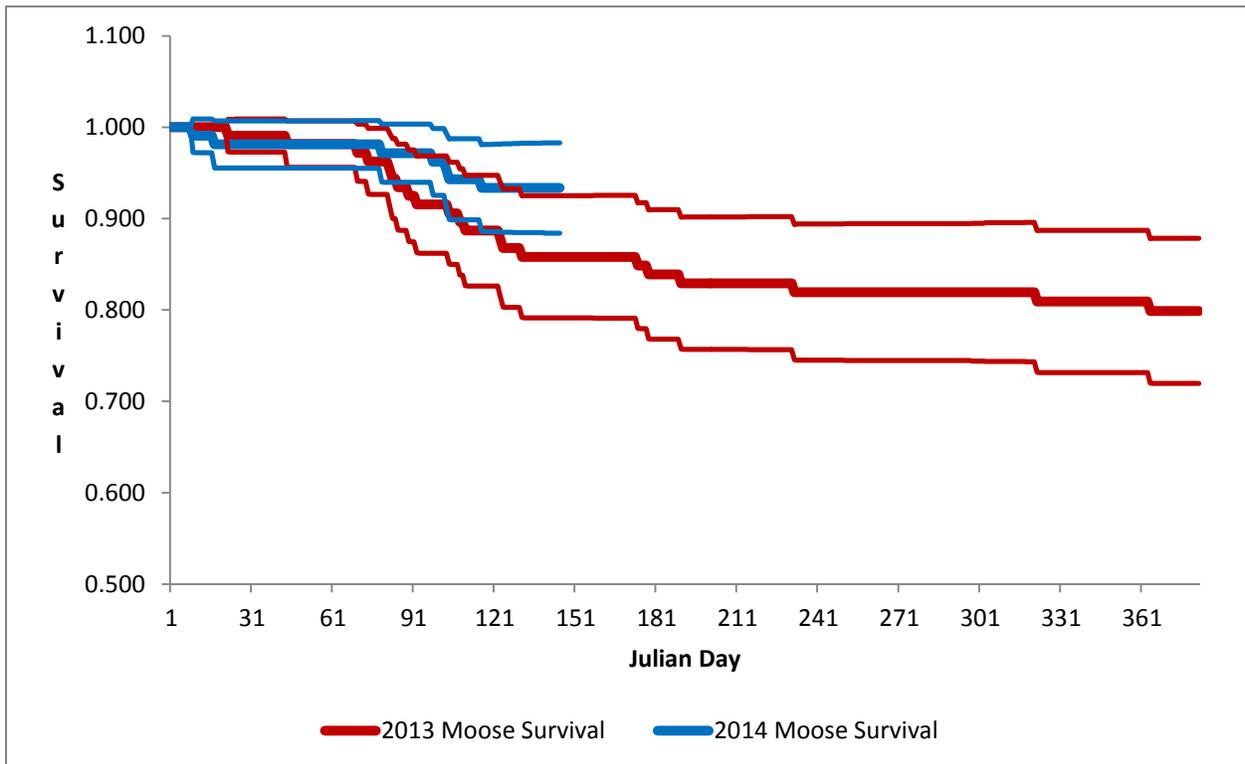


Figure 4. Annual survival of radio-collared moose captured in 2013 and 2014, with 95% confidence intervals included.





## CHRONIC WASTING DISEASE MANAGEMENT IN A MINNESOTA DEER HERD: COORDINATED RESPONSE TO THE SOUTHEAST DETECTION, 2010–2013.

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### INTRODUCTION

In November 2010, the first chronic wasting disease (CWD) positive wild white-tailed deer (*Odocoileus virginianus*) was detected through hunter-harvested surveillance by the Minnesota Department of Natural Resources (MNDNR) in southeastern Minnesota. Surveillance efforts were focused within an area surrounding a CWD-positive captive elk facility in Pine Island, which was discovered infected with the disease in early 2009. This captive elk herd, comprised of about 600 animals, was subsequently depopulated through federal indemnification, and a total of 4 elk were confirmed with the disease. Epidemiological investigations conducted by the United States Department of Agriculture (USDA) and Minnesota Board of Animal Health (BAH) concluded that there was an apparent longstanding infection within this captive elk facility. This wild deer index case was located within 3 miles of the CWD-positive captive elk facility.

In response to this disease detection, MNDNR enacted its CWD Response Plan (available at: [Minnesota DNR CWD response plan](#)). Initial aerial survey results indicated a high density of deer in the area near Pine Island (31 deer/km<sup>2</sup>) as well as numerous recreational feeding sites. The combination of these two factors heightened concern about disease transmission potential. A ban on recreational deer feeding was immediately enacted in a 4-county area, and a supplemental surveillance effort was conducted in February–April 2011 to improve our understanding of disease prevalence and distribution in the local deer herd. To prevent further disease spread the MNDNR (1) created a CWD Management Zone (Deer Permit Area (DPA) 602), (2) restricted whole-carcass movements outside of the zone, and (3) required mandatory sampling of all adult ( $\geq 1.5$  years of age) deer harvested by hunters (4) continued aggressive disease surveillance of hunter-harvested deer in the CWD Management Zone for 3 consecutive years (2011–2013). No additional cases of CWD were discovered in wild deer. Surveillance efforts are complete and DPA 602 was dissolved for the 2014 deer hunting season. In this paper, we describe how MNDNR responded to this detection of CWD in wild deer, including surveillance methods, management strategies, costs, and lessons learned.

### METHODS

#### 2011 Aerial Deer Survey, Recreational Feeding Activity, and Winter

Prior to beginning an intensive sampling effort, MNDNR used a fixed-wing aircraft to conduct an aerial survey of the CWD surveillance area to assess deer numbers and distribution. This survey was conducted in late January–early February 2011, with estimates of 6,200 deer within the 793-km<sup>2</sup> (306-mi<sup>2</sup>) winter surveillance area, equating to an estimated 7.3 deer/km<sup>2</sup> (19 deer/mi<sup>2</sup>) density (Figure 1). Deer densities were highest within a 23-km<sup>2</sup> (9-mi<sup>2</sup>) area surrounding the wild index case; 600 deer were counted using a helicopter census and estimated  $>31$  deer/km<sup>2</sup> (80 deer/mi<sup>2</sup>) (Figure 2). This information was used to guide sharpshooting activities and estimate the percentage of deer removed from the area through these subsequent targeted surveillance efforts.

Another key step in preventing further spread of CWD was to ban the recreational feeding of deer. On February 14, 2011 MNDNR issued a special rule that made recreational deer feeding illegal in a 4-county area (Dodge, Goodhue, Olmsted and Wabasha), surrounding the location of the CWD-positive deer (Figure 3). During the aerial surveys (fixed-wing and helicopter), there were a total of 35 recreational feeding sites observed. The ban was aimed at reducing the potential for the disease spread by eliminating artificially-induced deer concentration sites. MNDNR Enforcement staff began education and

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enforcement of the new rule immediately and compliance was extremely high.

CWD samples were obtained through landowner shooting permits (LSP) February 1-28, 2011, agency-sponsored culling conducted by USDA-Wildlife Services, (USDA-WS) February 22-early April 2011, and opportunistic sampling (e.g., vehicle-killed, sick or found dead deer) in this area. Car-killed deer disposition permits were not given to public persons who hit deer and wanted the carcass in DPA 602; local, county, and state enforcement personnel implemented these guidelines year round.

### **Hunter-harvested Surveillance**

Chronic Wasting Disease testing of hunter-harvested deer in DPA 602 was mandatory and included Special Youth, Archery, Firearm, and Muzzleloader hunting seasons. For the archery and muzzleloader seasons, MNDNR staff placed deer head collection boxes at high-volume registration. Boxes were checked daily, tissue samples were extracted twice weekly by trained MNDNR staff, and shipped overnight to a certified testing laboratory. During the regular firearm season, MNDNR personnel staffed 5 registration stations daily to collect samples from harvested deer, with assistance from veterinary and natural resources students on weekends. Within DPA 602, each registration station collected:

- Retropharyngeal lymph nodes (RLN) from all deer  $\geq 1.5$  years of age, and a small amount of muscle tissue was collected and placed in 95% ethanol for future genetic work.
- A front incisor was extracted from all deer  $\geq 2.5$  years old for aging by cementum annuli.
- Both fawn and adult deer were issued carcass tags by authorized MNDNR staff.
- MNDNR shipped RLN samples daily to the receiving laboratory in order to achieve a 3-business day turnaround time for test results
- Adult carcasses were prohibited from being taken out of DPA 602 until they were confirmed CWD-negative

All samples were inventoried, entered into a database, and sent to either the University of Minnesota's Veterinary Diagnostic Laboratory (St. Paul, MN) or Colorado State University (Fort Collins, CO) for enzyme-linked immunosorbent assay (ELISA) testing. Any presumptive positive samples from ELISA testing would be confirmed using immunohistochemistry (IHC) testing at the National Veterinary Services Laboratory in Ames, Iowa.

Hunters were able to check their test results on the MNDNR website using either their MNDNR number or the carcass tag number issued. At the time of sample collection, hunter information was recorded, including the hunter's name, a telephone number, MNDNR number, and location of kill. Maps were provided to assist the hunters in identifying the location (Township, Range, and Section) of the kill. Cooperating hunters were given a cooperator's patch and entered into a raffle to win a .50 caliber muzzleloader donated by the Minnesota Deer Hunter's Association (MDHA). During the firearm season, individual test results were available on-line within 3 business days of submission. Archery and muzzleloader results took slightly longer because of the bi-weekly sampling strategy.

Carcass movement restrictions within DPA 602 prevented local venison processors outside the zone from processing deer until negative CWD test results were obtained; this affected taxidermists as well. To accommodate hunters seeking a taxidermy mount from deer harvested within DPA 602, we provided local area taxidermists with necessary training and supplies to collect the retropharyngeal lymph node sample needed to test for CWD. At times, taxidermists came into the CWD management zone to cape the deer head for hunters, then the cape and antlers (with cleaned skull cap) were able to leave the zone immediately.

## RESULTS AND DISCUSSION

### Winter 2011 Sampling

Following a well-attended public meeting of >350 landowners, hunters, and concerned citizens in the Pine Island area, a winter sampling effort was initiated to gain a better understanding of disease prevalence and distribution in the local deer herd. Landowners within the boundaries of the winter surveillance area were able to obtain landowner shooting permit (LSP) for culling deer on their property during February 2011. More than 300 LSP were issued, with each landowner allowed to take an unlimited number of deer, designate up to 15 additional shooters under their LSP, use of high powered center-fire rifles, and the use of artificial lights was permitted. This area is historically regulated as a shotgun-only zone during the firearm season, so the ability to use rifles during this sampling effort was viewed as a unique opportunity by many landowners. Once deer were harvested, the landowner was required to contact MNDNR staff within 24 hours; samples were then collected in the field at private residences, each carcass was given a unique identification tag, and landowners were directed not to transport carcasses outside the winter surveillance area until they were notified of test results. Meat that was boned out or cut and wrapped either commercially or privately, and quarters or other portions of meat with no part of the spinal column or head attached were allowed to leave the zone immediately.

Late February 2011 through early April 2011, MNDNR contracted with USDA-Wildlife Services to use sharpshooting at bait piles to obtain additional deer samples. Deer were targeted during late evening and overnight hours, and intact carcasses were transported to the central processing facility located within the winter CWD surveillance. MNDNR and USDA-WS disease biologists eviscerated carcasses immediately upon delivery, collected medial retropharyngeal lymph nodes, a central incisor tooth for aging, muscle sample, recorded pregnancy rates, and issued a unique carcass tag to each individual animal. Entrails were deposited in a lined dumpster. All carcasses were held in a refrigerated trailer at 33-35°F until test-negative results were reported (typically within 3 business days). Once negative results were received, we distributed carcasses to the public from a venison disposition list which consisted of more than 400 people. Notified recipients arrived at the facility daily to load, transport and process one or two deer for consumption.

Through this combined winter surveillance effort, a total of 1,180 deer (752 adults, 428 fawns) were sampled within 16 km (10 miles) of the index wild deer case; all deer were negative for the disease (Figure 4). Sampling included deer taken by landowner shooting permits ( $n = 491$ ), agency-sponsored sharpshooting ( $n = 603$ ), vehicle-kills ( $n = 59$ ), and opportunistic sick deer ( $n = 27$ ). Shooting permits were issued to 323 landowners. Of those, 47% of permit-holders harvested  $\geq 1$  deer. The majority of permittees (57%) took 1 or 2 deer and approximately 5% took >10 deer from their properties.

The estimated total cost of the winter sampling effort was \$419,000. The majority (\$229,000) resulted from the USDA-WS 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.

### Hunter-harvested Surveillance

Following the winter 2011 winter sampling effort, hunter-harvested surveillance became the primary method for obtaining adequate samples for continued CWD monitoring. Fortunately, MNDNR had been conducting hunter-harvested CWD surveillance throughout the state since 2002, with increased focus in southeastern MN due to the infection rate in wild deer in adjacent counties of southern Wisconsin. A recent, intensive surveillance effort in 2009-2010 (which included the discovery of the one positive wild deer in 2010), in which over 3,200 deer were tested for CWD (Figure 5), demonstrated no widespread infection in the region. These data, in combination with historical data from 2002-2008 and the winter 2011 sampling, gave us increased confidence that the disease was not widespread or present at prevalence >0.5%.

The creation of CWD Management Zone, DPA 602, was an important step in efforts to manage

the disease, as it provided an enforceable boundary to restrict the potential flow of prions out of the area. Within DPA 602, MNDNR had the authority to change hunting season lengths, bag limits, offer special disease management tags (unlimited amount at a reduced cost of \$2.50/each) to increase harvest of antlerless deer, and make it mandatory to present the animal for CWD testing upon registration.

From 2011–2013, a total of 4,050 deer ( $n = 1,125, 1,195, \text{ and } 978$  for 2011, 2012, and 2013, respectively) were sampled for CWD within DPA 602 (Figure 6). All deer were negative for the disease. Approximately \$703,000 was spent in efforts to collect and test these hunter-harvested samples over the 3-year period. These results provide strong evidence that Minnesota was on the front end of a CWD outbreak in wild deer. Our inability to detect any additional infected deer in the immediate vicinity of the index case or in surrounding DPA's is encouraging. It is plausible that this disease is recent on the landscape in southeast MN, and that few additional wild deer have been exposed.

## Lessons Learned

The MNDNR had recently responded to an outbreak of bovine tuberculosis (bTB) in cattle and wild deer in northwestern Minnesota (2005-2012) and experiences gained from the management of that disease were very relevant to this CWD outbreak. While bTB and CWD are different diseases with unique transmission routes, initial strategies to manage these outbreaks were similar. Most importantly were efforts to reduce potential transmission pathways by restricting recreational feeding (hunting over bait has been illegal in MN since 1991) and reducing local deer numbers. Some of the tools used in the bTB outbreak, such as agency culling, proved to be highly successful at reducing deer numbers in high-risk areas in both disease outbreaks.

Numerous successes were attained throughout this entire CWD monitoring and response effort, which include:

- Gaining public support for all agency efforts was achieved by maintaining an informed public through publishing/reporting updated information as it became available.
- Having the MNDNR CWD Response Team availability 7 days a week to address concerns and interests expressed by the general public, landowners and hunters.
- The 3-day turnaround time for CWD test results eased hunters' concerns regarding spoilage of their harvested animal.
- The rapid test result time also gave credibility to this project and kept MNDNR staff apprised of additional positive results.
- Accommodating taxidermists and meat processors affected by both the test result reporting time and the carcass movement restrictions, allowed these vendors to perform their work and keep their livelihood and profession active.
- A special page was set up on the MNDNR website for CWD results and updates. Hunters were given a business card with the web address and instructions on how to access their results using either the carcass tag number assigned to their deer or their MNDNR number. Results were posted on the website as soon as they were made available.
- Hunters had the ability to access their results via computer at any time or by phone Monday-Friday during office hours.

While public support for MNDNR's strategies to manage the disease in DPA 602 was evident at the beginning of the outbreak, we did detect rising concerns about continued surveillance efforts once the disease was not widely detected in subsequent years. This "CWD Fatigue" syndrome has been described in other states that have long-standing efforts to manage the disease where public tolerance of control efforts fades over time. Moreover, the high cost of responding to a CWD detection event, which totaled \$1.12 million for this 3-year effort, brings added scrutiny to the need for an aggressive response. The MNDNR believed these efforts were necessary to manage and monitor this CWD outbreak and was thankful for the public and agency support to ensure the health of the local deer herd in southeast MN.

## **Future Surveillance Plans**

Disease surveillance in the CWD Management Zone has been discontinued and DPA 602 will be dissolved by fall 2014. Targeted CWD surveillance of deer exhibiting clinical signs of illness will continue statewide. The MNDNR plans to sample 450 hunter-harvested deer for CWD in DPAs 348 and 349 during the regular firearm season in fall 2014. This effort is in response to a recent detection of CWD in a free-ranging white-tailed deer in Alamakee County, Iowa.

## **ACKNOWLEDGEMENTS**

We would like to thank all the MNDNR Wildlife and Enforcement staff, who volunteered to assist with this disease outbreak surveillance project. Specifically, we'd like to recognize the support received from Rochester Wildlife Staff -- Don Nelson, Mike Tenney, Barb Perry, Nancy Reber, Clint Luedtke, Conservation Officers -- Dan McBroom, Kevin Prodzinski, Phil George and Dean Olson. We also wish to thank the students and faculty from the University of Minnesota, Colleges of Veterinary Medicine and Natural Resources, for assisting in our fall sampling efforts. Special thanks to Julie Hines and Bob Wright for fulfilling our GIS mapping needs and Pete Takash for his efforts in communications and internet-related work. We would also like to thank Beth Martin, Tom Enright, Amanda McGraw, Katie Pilarski, Claire Bagniewski, and Jennifer Dippel for their help through this effort. We appreciate the support of the USDA-Wildlife Services disease biologist Paul Wolf and Danny Storlie and USGS Epidemiologist LeAnn White, along with DNR Pilots Brad Maas and Tom Bucker with flying deer survey work.

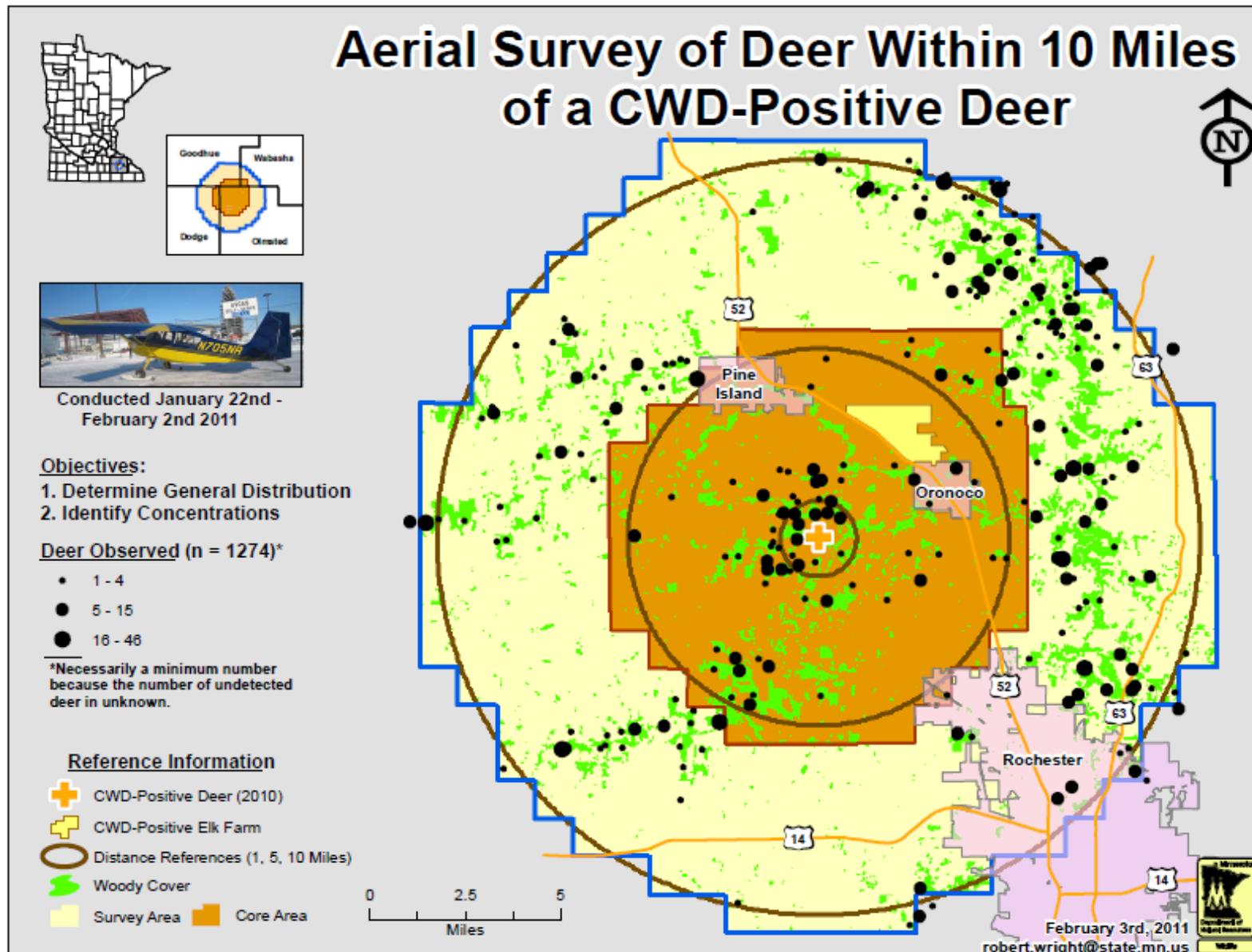


Figure 1. Fixed-wing, aerial survey results for 793-km<sup>2</sup> (306-mi<sup>2</sup>) area surrounding the location of the white-tailed deer that tested positive for chronic wasting disease (CWD), southeastern Minnesota, January–February 2011.

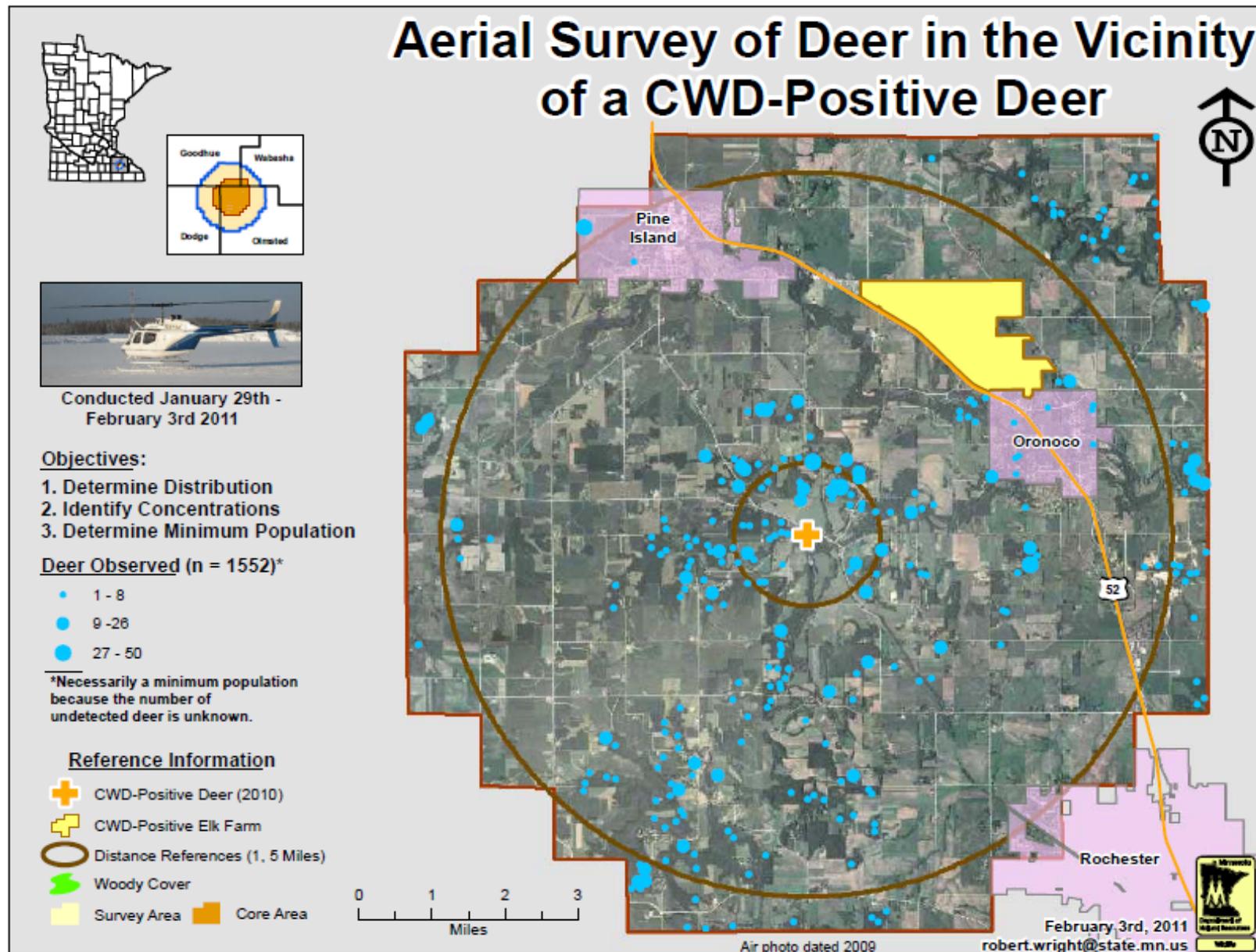


Figure 2. Helicopter white-tailed deer census for the 259-km<sup>2</sup> (100-mi<sup>2</sup>) Core Area within the winter 2011 chronic wasting disease (CWD) surveillance area, southeastern Minnesota, January–February 2011.

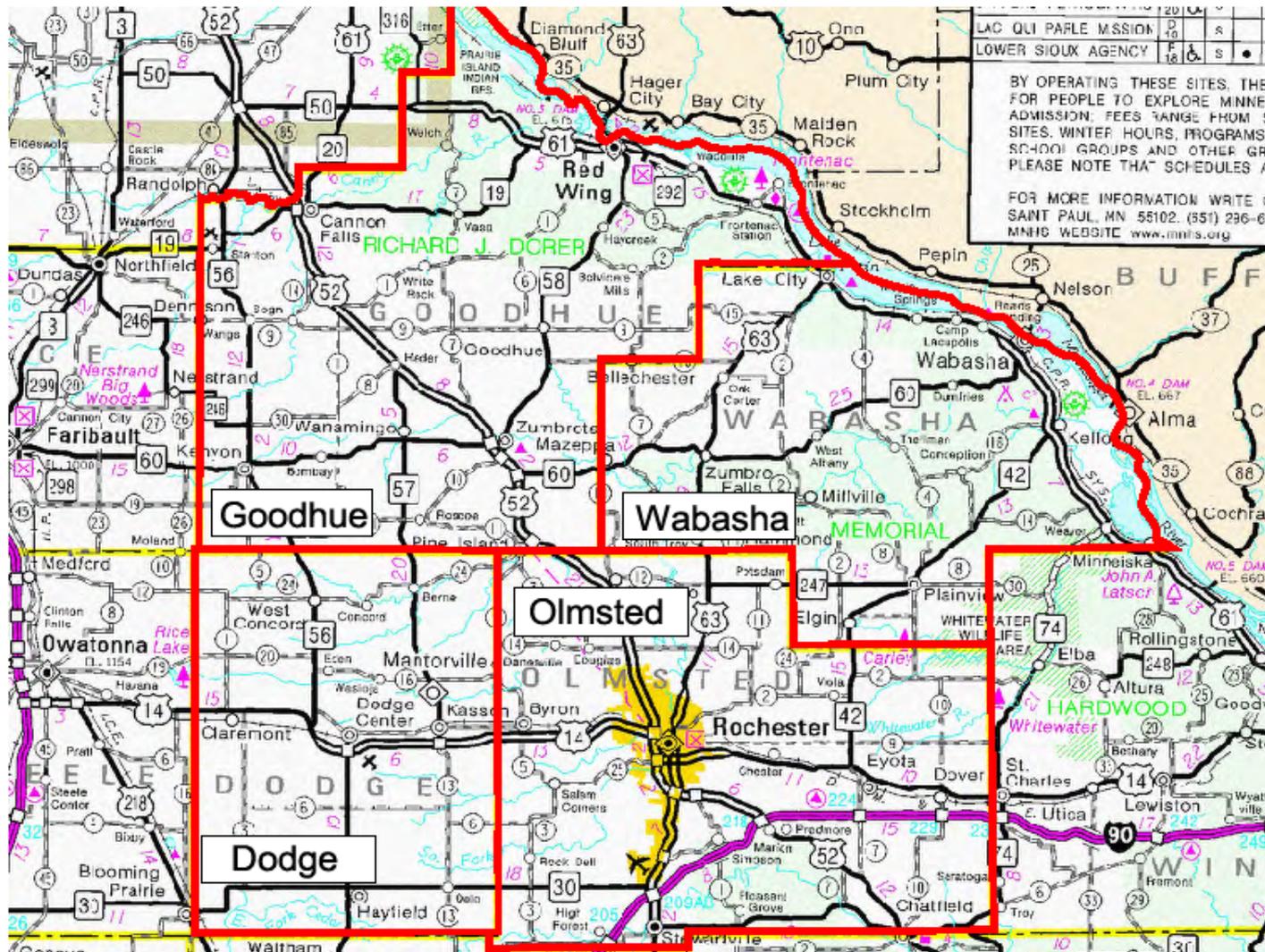


Figure 3. Four-county area in southeastern Minnesota where recreational feeding of wild white-tailed deer was banned in January 2011, following the discovery of chronic wasting disease in Olmsted County.

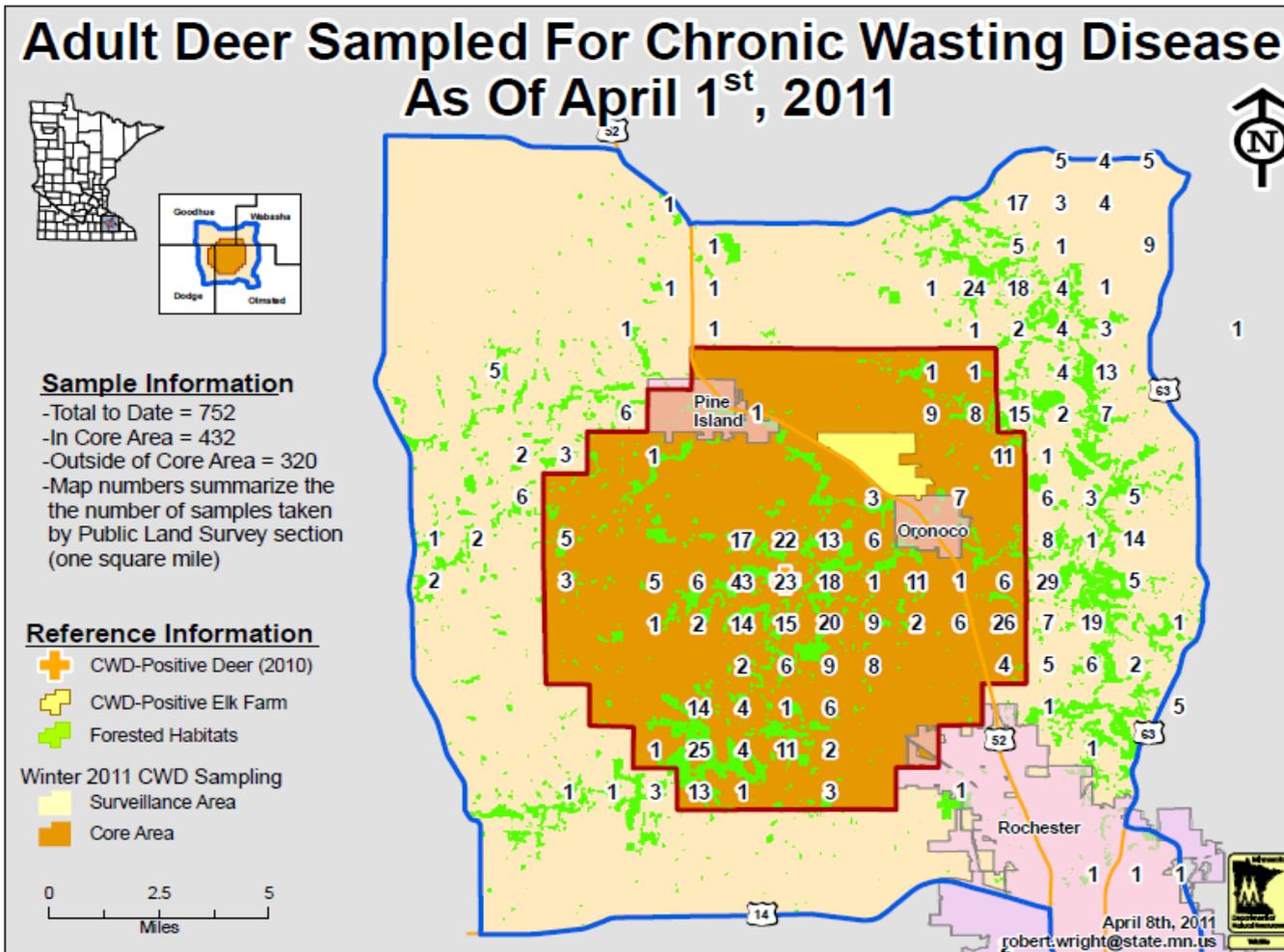


Figure 4. Section totals and distribution of adult (>1 year old) white-tailed deer ( $n = 752$ ) sampled for chronic wasting disease (CWD) during winter 2011, southeastern Minnesota.

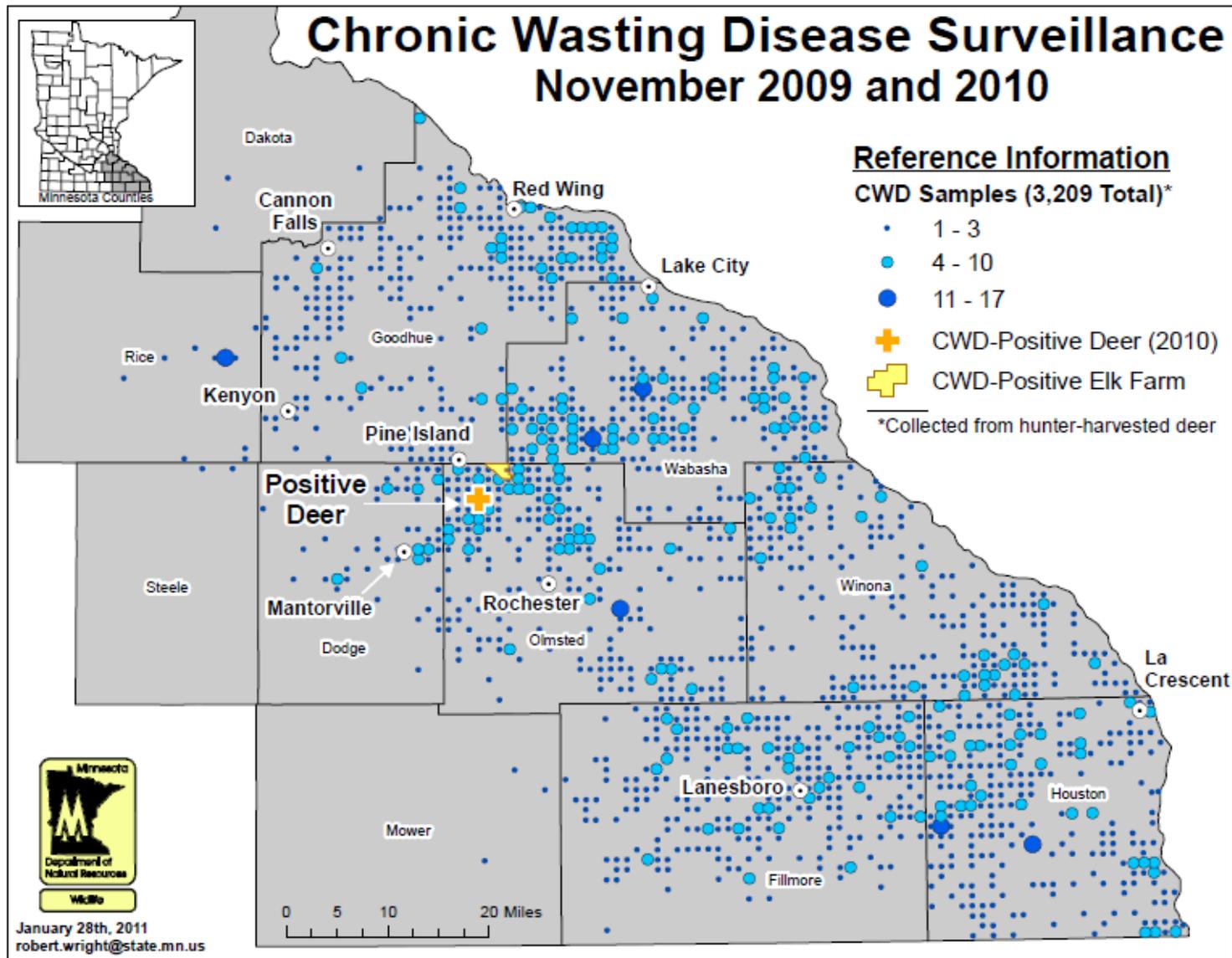


Figure 5. Sampling distribution for all hunter-harvested white-tailed deer ( $n = 3,209$ ) tested for chronic wasting disease (CWD) in southeastern Minnesota, falls 2009 and 2010, in relation to the location of CWD-positive deer.

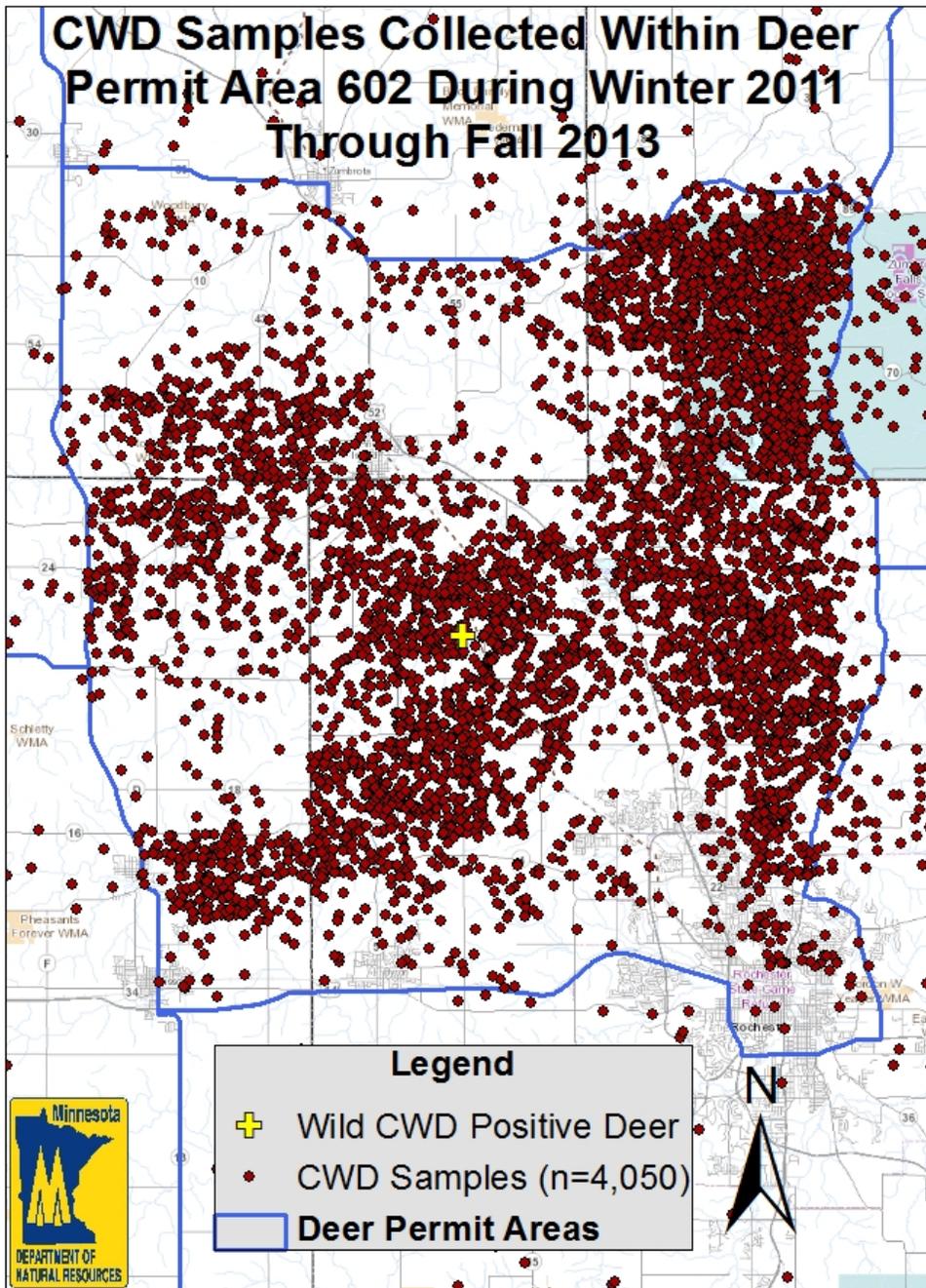


Figure 6. Sampling distribution of deer ( $n=4,050$ ) sampled for chronic wasting disease in deer permit area 602, winter 2011 through fall 2013.

# EPIDEMIOLOGY OF TOXOPLASMOSIS IN WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*): OCCURRENCE, CONGENITAL TRANSMISSION, CORRELATES OF INFECTION, ISOLATION, AND GENETIC CHARACTERIZATION OF *TOXOPLASMA GONDII*<sup>1</sup>

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## ABSTRACT

The prevalence of *Toxoplasma gondii* in white tailed deer (WTD) in the USA is high but little is known of the epidemiology of toxoplasmosis in this host. In the present study, we compared *T. gondii* seroprevalence from 749 WTD collected in 2012 and 2013 from a Metropolitan Park in Ohio and 487 WTD deer shot in Minnesota during 2008, 2009, and 2010. Serum samples were tested for antibodies to *T. gondii* by the modified agglutination test (cut-off titer, 25). Additionally myocardial samples from 123 seropositive WTD from Ohio were digested in pepsin and the digests were bioassayed for the isolation of *T. gondii*. Furthermore, to estimate transplacental rate of transmission, brains from 155 fetuses (included twins) from 148 deer from Minnesota were bioassayed in mice for the isolation of viable *T. gondii*. Seroprevalence of *T. gondii* varied with the year of collection, geography, and the age of deer. Of the Ohio deer sampled in 2012 and 2013 seroprevalences for the two years were similar (73.4% and 75.7%, respectively); remarkably 150 (66.1%) of 227 deer of <1 year of age were seropositive. Of the Minnesota deer, seroprevalence was lowest for the year 2008 (14.8%, 26/175) versus 2009 (27.7%, 59/213), and 2010 (25.2%, 25/99), thought to be related to environmental temperatures. Viable *T. gondii* was isolated in mice from the myocardium of four WTD from Ohio, and brain of one WTD fetus from Minnesota. Tachyzoites from infected mouse tissues were further propagated in cell culture. The DNA isolated from culture-derived tachyzoites of these five *T. gondii* isolates was characterized using 11 PCRRFLP markers (SAG1, 5\_- and 3\_-SAG2, alt.SAG2, SAG3, BTUB, GRA6, c22-8, c29-2, L358, PK1 and Apico). Four genotypes were found, including ToxoDB genotype no. 1 (Type II), no. 2 (Type III), no. 3 (Type II variant) and no. 146. Results indicate fluctuating seroprevalence, probably related to weather and warrant further epidemiological studies.

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<sup>1</sup> Veterinary Parasitology 202, 2014, pg. 270–275

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## CRANIAL AND BRAIN LESIONS OF A FREE-RANGING GRAY WOLF (*CANIS LUPUS*) CONFIRMED IN A HUMAN/WOLF CONFLICT INCIDENT<sup>1</sup>

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### ABSTRACT

We are describing significant brain, cranio-facial, and dental lesions in a wild wolf (*Canis lupus*) involved in the incident that occurred at Lake Winnibigoshish on August 23, 2013, which is the first confirmed wolf attack in Minnesota. DNA forensic analysis confirmed a positive match between the muscle tissue from the wolf and samples obtained from victim. On postmortem examination, the wolf presented asymmetric atrophy and bone remodeling affecting the incisive, maxilla, lacrimal, palatine, frontal, and ethmoid bones. There were dental abnormalities including malocclusion with teeth rotation and deviation, incomplete eruptions, and an odontogenic cyst with an unerupted tooth. Brain changes were bilateral loss and atrophy of extensive cortex regions including olfactory bulb and tract and frontal lobe. We highlight the relevance of a thorough post-mortem examination of wildlife to elucidate disease-based abnormal behavior as the reason for human/animal conflict. In contrast to the majority of cases of this nature, the current case signifies strong, biological, causative evidence for the abnormal behavior of a wild wolf that led to human injury.

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<sup>1</sup> Veterinary Pathology, 2014, *in review*.

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# SPATIAL AND TEMPORAL PATTERNS OF AVIAN PARAMYXOVIRUS-1 EPORNITICS IN DOUBLE-CRESTED CORMORANTS (*PHALACROCORAX AURITUS*) IN THE UNITED STATES<sup>1</sup>

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## ABSTRACT

Newcastle disease is caused by an avian paramyxovirus-1 (APMV-1) virus and is one of the most economically important diseases of poultry worldwide. Outbreaks of APMV-1 in double-crested cormorant (DCCO; *Phalacrocorax auritus*) nesting colonies in the United States and Canada have been sporadically documented in the literature. In this study, we describe the occurrence of APMV-1 associated epornitics in DCCO in the U.S. from first reported occurrence in 1992 through 2012. The frequency of APMV-1 epornitics has increased in the U.S. over the last decade, but the majority of events have occurred in DCCO colonies in the Midwestern U.S. Although morbidity and mortality in co-nesting species has been frequently reported during APMV-1 epornitics, disease caused by APMV-1 was uncommon in species other than DCCO. DCCO populations do not appear to have been significantly affected by this disease. However, since at least 49% the APMV-1 epornitics DCCO in the US have involved APMV-1 strains classified as virulent to poultry (virulent Newcastle Disease virus), its persistence and increased occurrence in DCCO warrants continued surveillance.

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<sup>1</sup> Journal of Wildlife Diseases, 2014, *in review*.

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<sup>3</sup> Minnesota Department of Natural Resources, Wildlife Health Program, Forest Lake, Minnesota

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## NECROPSY FINDINGS IN 62 OPPORTUNISTICALLY COLLECTED FREE-RANGING MOOSE (*ALCES ALCES*) FROM MINNESOTA, USA (2003 – 2013)<sup>1</sup>

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### ABSTRACT

The Minnesota moose population has declined dramatically since the 1990s. All 54 carcasses of moose that died of unknown cause or were euthanized by gun shot by tribal or Department of Natural Resources personnel because of perceived signs of illness between 2003 and 2013 and eight carcasses of moose that died due to vehicular accidents between 2009 and 2013 were submitted to the Minnesota Veterinary Diagnostic Laboratory and included in our study. The majority of the animals were underweight or cachectic ( $n= 53$ ; 85%). Neural migration presumably by *Parelaphostrongylus tenuis* was the most common finding related to disease in the examined animals ( $n = 28$ ; 45%). Moderate to marked *Dermacentor albipictus* (“winter tick”) ectoparasitism with widespread alopecia was the cause or a contributing cause of death in 14 (22%) cases in which grossly apparent anemia was associated with exhaustion of hepatic iron stores. Hepatic lesions associated with *Fascioloides magna* were common ( $n = 37$ ; 60%) but were unlikely to be the cause of death. Environmental factors favoring winter tick survival, habitat expansion of white-tailed deer (*Odocoileus virginianus*) and the survival of terrestrial and aquatic snails, serving as intermediate hosts for *P. tenuis* and *F. magna*, may contribute to the seemingly severe parasitic burden in Minnesota’s moose population.

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<sup>1</sup>Journal of Wildlife Diseases, 2014, *in press*.

<sup>2</sup>Department of Veterinary Population Medicine, Veterinary Diagnostic Laboratory, College of Veterinary Medicine, University of Minnesota, St. Paul, Minnesota

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