

# Chronic Wasting Disease in Free-Ranging Wisconsin White-Tailed Deer

Damien O. Joly,\* Christine A. Ribic,\*  
Julie A. Langenberg,† Kerry Beheler,†  
Carl A. Batha,† Brian J. Dhuey,‡  
Robert E. Rolley,‡ Gerald Bartelt,‡  
Timothy R. Van Deelen,§;  
and Michael D. Samuel\*¶

Three White-tailed Deer shot within 5 km during the 2001 hunting season in Wisconsin tested positive for chronic wasting disease, a prion disease of cervids. Subsequent sampling within 18 km showed a 3% prevalence (n=476). This discovery represents an important range extension for chronic wasting disease into the eastern United States.

Chronic wasting disease (CWD) is degenerative and usually considered to be fatal in White-tailed Deer (*Odocoileus virginianus*), Mule Deer (*O. hemionus*), and Elk (*Cervus elaphus*) associated with the presence of transmissible protease-resistant prion proteins (PrP<sup>Sc</sup>) (1,2). Although the transmission route of PrP<sup>Sc</sup> is unknown, it may be transmitted in deer and elk by direct contact or indirectly from the environment (1,2). In experiments, clinical signs have appeared as early as 15 months after exposure (1) and include weight loss, anorexia, repetitive behaviors, hyperesthesia, and intractability. Signs progress to severe emaciation, extreme behavioral changes, excessive salivation, tremors, and mild ataxia (1,2). CWD was first recognized in captive Mule Deer in Colorado (3) and subsequently described in the free-ranging cervid populations of Colorado and Wyoming (1); prevalence in these disease-endemic areas varies spatially and among the three sympatric cervid species (4). Before its discovery in Wisconsin, CWD was detected in captive cervid farms in Colorado, Nebraska, South Dakota, Oklahoma, Kansas, Montana (USA), as well as Alberta, Saskatchewan (Canada), and South Korea (1). Apart from the contiguous

areas of Colorado, Wyoming, and Nebraska, CWD had previously only been detected in two free-ranging Mule Deer from Saskatchewan, one Mule Deer from South Dakota, and in a number of Mule Deer from the western slopes region of Colorado (1). Previously, no cases of CWD were reported east of the Mississippi; however, subsequent to our research, CWD-positive cervids were found in Minnesota (captive Elk), Wisconsin (captive White-tailed Deer and Elk), and Illinois (free-ranging White-tailed Deer). Further, west of the Mississippi, the following CWD-positive animals have been found: Mule Deer in New Mexico and Utah; free-ranging Mule and White-tailed Deer in Saskatchewan, Canada; and captive Elk and White-tailed Deer in Alberta, Canada.

## The Study

In autumn of 1999 and 2000, the Wisconsin Department of Natural Resources (WDNR) submitted to the National Veterinary Services Laboratories (NVSL) (Ames, Iowa) brain material (obex) from 657 hunter-killed White-tailed Deer registered at hunter check stations across the state. None came from the study area we describe. Samples were tested for CWD prion by immunohistochemistry (IHC) (5). Prion was not detected in any samples. However, 3 of 445 White-tailed Deer shot in autumn of 2001 were positive for CWD. These deer were males, 2.5 years of age, and were shot within 5 km in south-central Wisconsin. WDNR subsequently conducted a sampling program to assess the distribution and prevalence of CWD in the vicinity of these three positive deer. We report the results of this sampling program.

Samples were collected from 500 adult (>1 year of age) White-tailed Deer within an approximate 18-km radius, and all samples were tested for CWD. Deer were submitted by hunters who were issued scientific collection permits, collected at roadside after vehicular collision, or collected by WDNR U.S. Department of Agriculture sharpshooters. Data from collected deer included the geographic location based on the Wisconsin Public Land Survey System (township-range-section), sex, and age (estimated by using tooth eruption and tooth wear patterns [6]). Location of kill was indicated on a map by hunters during interviews by DNR staff. Samples of brain stem (obex) and retropharyngeal lymphatic tissue were fixed in 10% buffered formalin and submitted to NVSL for testing using IHC. We considered a deer to be CWD positive if either obex or retropharyngeal samples were IHC positive (1).

We used the spatial scan statistic provided by Kulldorff and Nagarwalla (7) (program SaTScan available from: URL: <http://www3.cancer.gov/prevention/bb/satscan.html>) to assess the presence and location of CWD clusters within the surveillance area. Location data were collected to the survey unit "section" (approximately 2.6 km<sup>2</sup>). We pooled

\*United States Geological Survey-Wisconsin Cooperative Wildlife Research Unit, University of Wisconsin-Madison, Madison, Wisconsin, USA; †Wisconsin Department of Natural Resources, Madison, Wisconsin, USA; ‡Wisconsin Department of Natural Resources, Monona, Wisconsin, USA; §Wisconsin Department of Natural Resources, Rhinelander, Wisconsin, USA; and ¶United States Geological Survey-National Wildlife Health Center, Madison, Wisconsin, USA

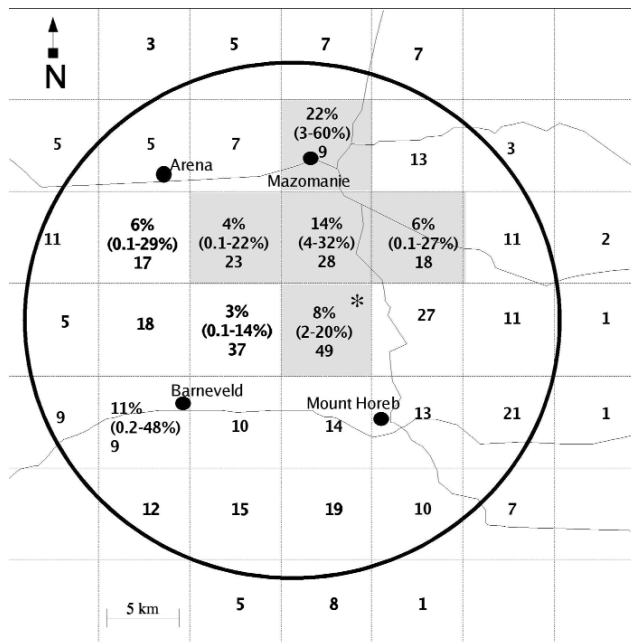


Figure. Spatial distribution of chronic wasting disease in White-tailed Deer sampled in Wisconsin (February–April 2002). Locations for sampled deer were recorded by using the Wisconsin Public Land Survey System (township-range-section); analysis was conducted on pooled 4X4 sections (41 km<sup>2</sup>), as indicated by the dashed grid lines. Prevalence, 95% confidence limits (CI), and sample size for each quadrat are indicated, as well as sample size only for quadrats in which positive deer were not detected. A cluster of higher than expected prevalence was detected in the north-central region of the sampling area indicated by shading (prevalence 9.4%, 95% CI 5.0% to 16.0%, n=127). The asterisk indicates the quadrat in which the three initial positive deer were found. The circle represents the targeted surveillance area.

locations into 4X4 section quadrats for analysis to compensate for sections from which no deer were collected. In a separate analysis, sex and age were assessed as predictors of CWD status by using logistic regression (function glm in program R v. 1.5.0; available from: URL: <http://www.r-project.org>) (8). Model selection uncertainty was incorporated into the odds ratio (OR) estimates by using model averaging (9).

## Results and Discussion

From March 2 to April 9, 2002, samples were collected from 505 deer; however, 29 deer were not included in the analysis because of sample autolysis, inappropriate tissue submission, or lack of availability of appropriate tissues (e.g., deer with no intact cranium or those shot in the head). Of the remaining 476 deer (87 males, 386 females, and 3 for which sex was not recorded), 15 (3.2%; 95% confidence limit [CI] 1.7% to 5.1%) were IHC positive, 11 in both obex and retropharyngeal lymph node samples and 4 from lymph nodes only. We inferred that deer that were only lymph node positive were in the earlier states of

infection (1). Estimated prevalence varied spatially within the surveillance area. A cluster of higher than expected prevalence was detected in the north-central region of the sampling area (prevalence 9.4%; 95% CI 5.0% to 16.0%;  $p=0.003$ ;  $n=127$ ) (Figure).

Prevalence did not vary by sex (males: 3.4%, 95% CI 0.1% to 9.7%,  $n=87$ ; females: 3.1%, 95% CI 1.6% to 5.3%,  $n=386$ ; male vs. female OR 1.1, 95% CI 0.56 to 2.19), a pattern consistent with Mule Deer sampled in Colorado and Wyoming (4). Increasing prevalence with age was suggested, although we could not distinguish whether the OR differed from 1 (OR 1.13, 95% CI 0.93 to 1.39). We had a small sample ( $n=32$ ) of older animals (>5 years of age), which weakened our ability to detect an increase in prevalence with age statistically. Miller et al. (4) found that CWD prevalence increased with age in male Mule Deer and then abruptly declined in older age classes. We did not have a sufficient sample size to evaluate a sex difference in prevalence by age.

The known range of CWD was extended by its detection in Wisconsin, which is the first report of the disease east of the Mississippi River. Although we do not know how the free-ranging deer population of Wisconsin became affected by CWD, the most commonly suggested hypothesis is that CWD in Wisconsin may have emerged through importing of an affected cervid. The current enzootic of CWD in free-ranging deer and elk is paralleled by an enzootic in the captive cervid industry, and the relationship between CWD-affected elk farms and recent (2000–2002) diagnoses of CWD in free-ranging deer in Nebraska, South Dakota, and Saskatchewan remains under investigation (1). Elk were imported to Wisconsin from CWD-affected herds in Colorado during the 1990s, and recently (September and October 2002) captive White-tailed Deer were found to be positive on two separate farms in central and southern Wisconsin (10). Furthermore, during epidemiologic investigations of these positive farms, WDNR discovered that deer had escaped in March 2002 from one of these farms, one of which was later shot and found to be CWD positive (9). We stress that these positive captive deer are likely not the source of CWD in this free-ranging White-tailed Deer outbreak because of the captive deer's distance from the area where the CWD-positive free-ranging deer are (approximately 130 km). No direct evidence exists that CWD came to Wisconsin by the captive cervid industry. However, further investigation on possible links between CWD cases in captive and free-ranging cervids in Wisconsin is ongoing.

## Conclusions

The state of Wisconsin is undertaking an integrated research, surveillance, and management program to determine the distribution of CWD in the Wisconsin free-rang-

ing deer population and eventually eliminating the disease from the known affected area of south-central Wisconsin (10,11). As of March 2003, a total of 39,636 deer had been sampled statewide for CWD as part of this surveillance and management program (data are available from: URL: <http://www.dnr.state.wi.us/org/land/wildlife/whealth/issue/cwd/>). Computer simulation of CWD dynamics in western cervid populations (12) indicated that CWD could severely reduce deer numbers. Disease transmission may occur at a greater rate and consequently have a larger impact on the population in the eastern United States, where White-tailed Deer densities are typically an order of magnitude larger than western deer and elk populations (e.g., deer densities in the CWD-affected area are estimated to be currently >720 deer per km<sup>2</sup>) (WDNR, unpub. data). Deer and deer-related activities, such as hunting, wildlife viewing, and other social factors, are an important component of the Wisconsin culture and economy (approximately \$1 billion/year) (13), prompting an aggressive research and management strategy to combat CWD in Wisconsin's free-ranging deer population.

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Dr. Joly is a research associate with the United States Geological Survey-Wisconsin Cooperative Wildlife Research Unit, University of Wisconsin-Madison. His research interests include the ecology of infectious diseases of wildlife.

#### References

1. Williams ES, Miller MW, Kreeger TJ, Kahn RH, Thorne ET. Chronic wasting disease of deer and elk: a review. *J Wildl Manage* 2002;66:551-63.
2. Williams ES, Kirkwood JK, Miller MW. In: Williams ES, Barker IK, editors. *Transmissible spongiform encephalopathies. Infectious diseases of wild mammals*. 3rd edition. Ames (IO): Iowa State University Press; 2001. p. 292-301.
3. Williams ES, Young S. Chronic wasting disease of captive mule deer: a spongiform encephalopathy. *J Wildl Dis* 1980;16:89-98.
4. Miller MW, Williams ES, McCarty CW, Spraker TR, Kreeger TJ, Larsen CT, et al. Epizootiology of chronic wasting disease in free-ranging cervids in Colorado and Wyoming. *J Wildl Dis* 2000;36:676-90.
5. Hamir N, Cutlip RC, Miller JM, Williams ES, Stack MJ, Miller MW, et al. Preliminary findings on the experimental transmission of chronic wasting disease agent of mule deer to cattle. *J Vet Diagn Invest* 2001;713:11-90.
6. Severinghaus CW. Tooth development and wear as criteria of age in white-tailed deer. *J Wildl Manage* 1949;13:195-216.
7. Kulldorff M, Nagarwalla N. Spatial disease clusters: detection and inference. *Stat Med* 1995;14:799-810.
8. Ihaka R, Gentleman R. A language for data analysis and graphics. *Journal of Computational and Graphical Statistics* 1996;5:299-314.
9. Burnham KP, Anderson DR. *Model selection and inference: a practical information-theoretic approach*. New York: Springer; 1998.
10. Wisconsin Department of Natural Resources. Environmental impact statement on rules to eradicate chronic wasting disease from Wisconsin's free-ranging white-tailed deer herd; 2003. [cited Mar 10, 2003] Available from: URL: <http://www.dnr.state.wi.us/org/land/wildlife/whealth/issues/cwd/>
11. Wisconsin Department of Natural Resources. Wisconsin regulations related to chronic wasting disease. 2002 [cited Aug 12, 2002]. Available from: URL: <http://www.dnr.state.wi.us/org/land/wildlife/regs/02CWDregs.pdf>
12. Gross JE, Miller MW. Chronic wasting disease in mule deer: disease dynamics and control. *J Wildl Manage* 2001;65:205-15.
13. U.S. Department of the Interior, Fish and Wildlife Service, and U.S. Department of Commerce, Bureau of the Census. 1996 national survey of fishing, hunting, and wildlife-associated recreation: Wisconsin. FWS/96-WI; issued April 1998.

Address for correspondence: Damien O. Joly, Department of Wildlife Ecology, University of Wisconsin-Madison, 218 Russell Labs, Madison, WI 53706, USA; fax: (608) 270-2415; email: [dojoly@wisc.edu](mailto:dojoly@wisc.edu)

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