

Approximating the Mean Post-mortem Brain Mass and Brain to Body Mass Ratio of Adult Male White-tailed Deer (*Odocoileus virginianus*) in North Dakota, USA

Cami M. Wight¹ and Charlie S. Bahnson^{1,*}

Abstract – Current scientific literature lacks a reference for the average brain mass of male White-tailed Deer (*Odocoileus virginianus*), the most widely distributed cervid in North America. A reference for brain tissue mass may be useful for better understanding transmission risk of chronic wasting disease (CWD), an important neurologic disease of cervids. In a recent study, oral consumption of 100–300 ng of CWD-contaminated brain tissue was sufficient for disease transmission, which is concerning considering the documented persistence of CWD-causing prions within carcasses of infected animals on the landscape. We individually measured the mass of brain tissue from 29 adult male White-tailed Deer from 17 deer hunting units across North Dakota, yielding an average mass of 182.7 grams ($n = 29$; 95% Confidence Interval = 177.2–188.2). These findings demonstrate that a single deer head moved across jurisdictions and left on the landscape could contain infectious doses numbering in the hundreds of millions. This information further supports CWD-related regulations such as carcass transport restrictions and approved disposal requirements. We also present the first known publication of brain to body mass ratio and encephalization quotient of North American White-tailed Deer (1.094) from a subset of our adult male samples.

Introduction

Chronic wasting disease (CWD) is a neurodegenerative prion disease that increasingly threatens wild cervid populations (DeVivo et al. 2017, Edmunds et al. 2016). In addition to direct contact with infected animals, transmission of CWD has also been documented through contact with environments contaminated with infected carcasses (Miller et al. 2004). As a result, many wildlife agencies employ cervid carcass transport and disposal regulations aimed at reducing environmental contamination and anthropogenic spread of the disease (Gillan and Mawdsley 2018). This approach was further supported by a recent study that demonstrated oral exposure to only 100–300 nanograms (ng) of CWD-positive brain material was sufficient to infect *Odocoileus virginianus* Zimmermann (White-tailed Deer) (Denkers et al. 2020). To our knowledge, a published reference does not exist for brain mass in free-ranging, male White-tailed Deer. Such a reference would be helpful to contextualize the amount of potential infectious CWD material in one deer head. Furthermore, the brain to body mass ratio and encephalization quotient reported here may be of use to future morphological or neurodegenerative research.

Methods

Between January 2022 and January 2023, adult male White-tailed Deer aged approximately 2.5 to 4.5 years, based on dentition and tooth wear, were opportunistically selected for the study. Samples were collected from 17 hunting units across the state (Figure 1) and included whole carcasses submitted for mortality investigation ($n = 11$) and heads from

¹ North Dakota Game and Fish Department, Wildlife Health Lab, 3001 E. Main Ave. Bismarck, North Dakota 58501 (USA). *Corresponding author (email: cbahnson@nd.gov).

Associate Editor: Sue Fairbanks, Oklahoma State University.

hunter-harvested animals voluntarily submitted for CWD surveillance conducted by the North Dakota Game and Fish Department (NDGF) during the 16.5-day deer gun season in November ($n = 18$). These hunting units were selected because no confirmed cases of CWD had been detected as of January 2022. Carcasses collected for mortality investigation at the NDGF Wildlife Health Lab (Bismarck, North Dakota, USA) consisted of adult, male White-tailed Deer found dead by members of the public ($n = 9$) or euthanized after appearing moribund ($n = 2$). Cold chain was initiated as soon as possible following carcass collection and was maintained below 4.45 °C throughout the storage period for each sample, which did not exceed 14 days. Due to sub-freezing temperatures throughout much of the 2022 gun season and the outdoor locations of sample collection sites, most samples were subjected to at least one freeze-thaw cycle prior to processing.

All individuals were screened for CWD via enzyme-linked immunosorbent assay (ELISA) testing of the medial retropharyngeal lymph nodes at the Montana Veterinary Diagnostic Laboratory (Bozeman, Montana, USA). CWD was not detected in any study deer, nor were any deer presenting with gross lesions on the cerebrum, cerebellum, or brainstem included in the study. Post-mortem investigations of several animals warranted submission of formalin-fixed tissues to diagnostic laboratories for histological evaluation by a pathologist ($n = 4$), and when brain tissue was included ($n = 2$) no significant findings were noted in the pathology case reports upon examination of the fixed cerebrum, cerebellum, or brainstem.

Carcasses submitted to the Wildlife Health Lab for postmortem examination were weighed intact using a digital hanging crane scale (Castron II Model 2THB) prior to necropsy. Body weight of carcasses that had been scavenged in any capacity was not measured. All major organs were examined grossly for lesions and other abnormalities, including the brain. To extract the brain, skulls were bisected with a cleaver and mallet. Cranial nerves were detached at the point of entry into the cranial cavity, and the brainstem was severed at the point of exit from the foramen magnum. The cerebrum, cerebellum, and brainstem

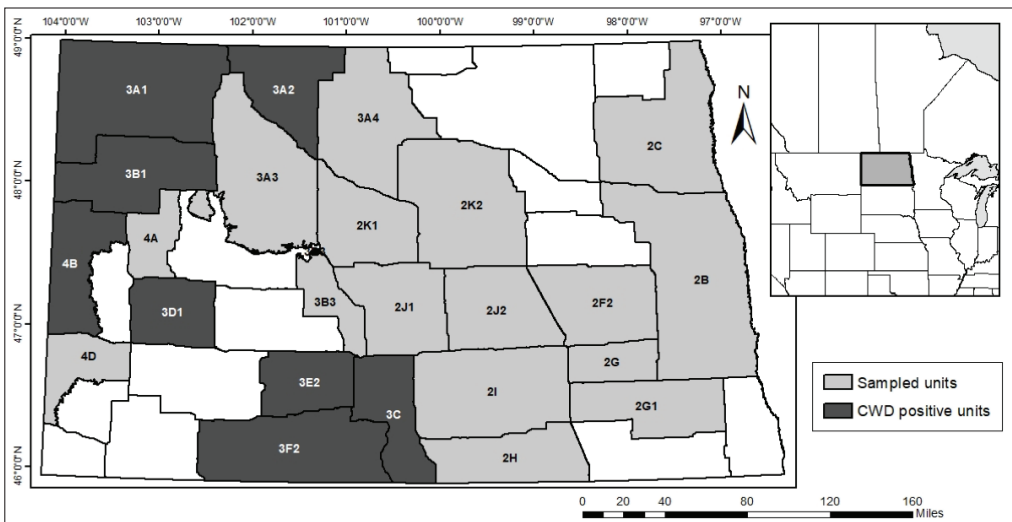


Figure 1. Deer hunting units of North Dakota. Units from which animals in this study were utilized are pictured in light grey, and units with previously confirmed detections of CWD are pictured in dark grey. No CWD-positive units were sampled for the purposes of this study. One sample was collected from units 2G, 2G1, 2J1, 2K1, 2K2, 3A3, 3F1, 4A, and 4B; two samples were collected from 2B, 2F2, and 2J2; three samples were collected from 2C, 2H, and 3A4; and four samples were collected from 2I.

were removed from the skull, placed in a tared container, and weighed in grams (g) using an electronic balance (Brecknell, Model 311). Brain weights were compared between hunter-harvested deer and mortality investigations using the Mann-Whitney test. The mean and 95% confidence intervals were calculated for brain and body weights. For animals in which body weights were also recorded, an encephalization quotient (EQ) was calculated by using the following established formula: $\text{brain mass}/0.12(\text{body mass})^{0.66}$ (Jerison 1973).

Results

Morphometric data were collected from 29 adult, male White-tailed Deer between January 2022 and January 2023 (Table 1). Brain mass was recorded for 29 individuals, and body weight was recorded for nine of those individuals. Brain mass averaged 182.7 g (n = 29; 95% Confidence Interval = 177.2–188.2). There was no statistical difference between mean brain masses from hunter-harvested deer versus mortality investigations (Mann-Whitney $U = 74$, $n_1 = 18$, $n_2 = 11$, $P = 0.271$). Body weight averaged 57.9 kg, with a range of 41–78 kg (n = 9; 95% Confidence Interval = 50.7–65.2). The ratio of brain to body mass calculated for these nine individuals averaged 1:330, with a range of 1:222.8–1:500. The mean brain and body mass values corresponded to EQ of 1.094.

Discussion

The purpose of this study was to fill a gap in the literature while also yielding information necessary to better understand CWD transmission risk. Denkers et al. (2020) demonstrated that 100–300 ng of CWD-contaminated brain homogenate was sufficient to infect White-tailed Deer in an experimental setting. This trace amount may be difficult to conceptualize for managers and policymakers who are charged with developing CWD-mitigating strategies that are both reasonable and effective. While caution must be used to avoid over-interpretation, findings from Denkers et al. (2020), paired with an average brain mass of 182.7 g reported herein, suggest that the number of infectious doses within the brain of a CWD-infected animal could number in the hundreds of millions. Numerous caveats must be considered when extrapolating these implications. For example, the concentration of prions in brain material will vary anatomically and between

Table 1. Mean brain weight, body weight, and brain: body mass ratio of selected adult, male White-tailed Deer collected in North Dakota from January 2022 to January 2023.

Male White-tailed Deer aged 2.5–4.5 yrs					
Measurement	N	Mean	SD	Min.	Max.
Brain – fresh mass (g)	29	182.7	15.1	156.0	211.0
Gross body mass (kg)	9	57.9	11.1	41.0	78.0

animals and depends on where an animal falls within the course of disease (Spraker et al. 2002). The effect of freeze-thaw on moisture retention and weight measurements is unknown. Regardless, this finding further illustrates the potential risk posed by movement and improper disposal of carcasses, both of which may serve as anthropogenic mechanisms that spread disease to novel areas.

Prions persist on the landscape for extended periods of time (Miller et al. 2004, Georgsson et al. 2006), compounding the risk posed by such actions. Following human-caused movement, scattering of carcass parts by scavengers can further exacerbate the contamination of previously CWD-free landscapes (Jennelle et al. 2009, VerCauteren et al. 2012). The brain, spinal column, and lymph nodes are classified as “high-risk” carcass parts and are of particular concern due to patterns in prion accumulation throughout disease progression (Fox et al. 2006). Regulating the transportation or improper disposal of high-risk carcass parts from hunter-harvested animals is an important tool wildlife managers should utilize to reduce CWD introduction and transmission risk (Gillan and Mawdsley 2018).

Morphometrics inform the study of wildlife species by providing quantitative values that may distinguish between normal and abnormal condition of whole animals or major organs. Past studies have reported mean brain masses of 197.7 g (\pm 19 g) for adult free-ranging *Odocoileus hemionus* Rafinesque (Mule Deer) from northcentral Colorado and 159.0 g (\pm 8 g) for adult, female White-tailed Deer bred and raised in captivity in South Dakota (Anderson et al. 1974, Berheim et al. 2019). Our comparable finding of 182.7 g for adult male White-tailed Deer is otherwise absent from current literature, to the best of our knowledge. This information may be of use for future morphological or neurodegenerative research.

Brain to body mass ratio can contextualize encephalization among species. Our value of 1:330, the first available reference for this metric in White-tailed Deer, can be utilized for comparative morphology among species (Jerison 1973). In contrast, the encephalization quotient (EQ) accounts for the scaling relationship between the brain and body mass and is often used as a morphometric means of illustrating the relative intelligence of species (Boddy et al. 2012). Although male-biased and based on averages from a small sample size, this rough approximation of EQ provides marginal context for where White-tailed Deer fall within numerous, well-documented EQ calculations for other species.

Acknowledgements

This project was funded by Federal Aid to Wildlife Restoration administered through North Dakota Game and Fish Department (Study No. W-67-R).

Literature Cited

- Anderson, A.E., D.E. Medin, and D.C. Bowden. 1974. Growth and morphometry of the carcass, selected bones, organs, and glands of mule deer. *Wildlife Monographs* 39:3–122.
- Berheim, E.H., J.A. Jenks, J.G. Lundgren, E.S. Michel, D.G. Grove, and W.F. Jensen. 2019. Effects of neonicotinoid insecticides on physiology and reproductive characteristics of captive female and fawn white-tailed deer. *Scientific Reports* 9:4534. <https://doi.org/10.1038/s41598-019-40994-9>
- Boddy, A.M., M.R. McGowen, C.C. Sherwood, L.I. Grossman, M. Goodman, and D.E. Wildman. 2012. Comparative analysis of encephalization in mammals reveals relaxed constraints on anthropoid primate and cetacean brain scaling. *Journal of Evolutionary Biology* 25:981–994.
- Denkers N.D., C.E. Hoover, K.A. Davenport, D.M. Henderson, E.E. McNulty, A.V. Nalls, and E.A. Hoover. 2020. Very low oral exposure to prions of brain or saliva origin can transmit chronic wasting disease. *PloS One* 15(8), e0237410.

- DeVivo, M.T., D.R. Edmunds, M.J. Kauffman, B.A. Schumaker, J. Binfet, T.J. Kreeger, and T.E. Cornish. 2017. Endemic chronic wasting disease causes mule deer population decline in Wyoming. *PloS One* 12(10), e0186512.
- Edmunds, D.R., M.J. Kauffman, B.A. Schumaker, F.G. Lindzey, W.E. Cook, T.J. Kreeger, and T.E. Cornish. 2016. Chronic wasting disease drives population decline of white-tailed deer. *PloS One* 11(8), e0161127.
- Fox, K.A., J.E. Jewell, E.S. Williams, and M.W. Miller. 2006. Patterns of PrPCWD accumulation during the course of chronic wasting disease infection in orally inoculated mule deer (*Odocoileus hemionus*). *Journal of General Virology* 87(11), 3451–3461.
- Georgsson, G., S. Sigurdarson, and P. Brown. 2006. Infectious agent of sheep scrapie may persist in the environment for at least 16 years. *Journal of General Virology* 87:3737–3740.
- Gillin, C.M., and J.R. Mawdsley. (eds.). 2018. AFWA technical report on best management practices for surveillance, management and control of chronic wasting disease. Association of Fish and Wildlife Agencies, Washington, D. C., USA. 111 pp.
- Jennelle, C.S., M.D. Samuel, C.A. Nolden, and E.A. Berkley. 2009. Deer carcass decomposition and potential scavenger exposure to chronic wasting disease. *The Journal of Wildlife Management* 73:655–662.
- Jerison H.J. 1973. *Evolution of the Brain and Intelligence*. Academic Press, New York, USA, 481 pp.
- Miller M.W., E.S. Williams, N.T. Hobbs, and L.L. Wolfe. 2004. Environmental sources of prion transmission in mule deer. *Emerging Infectious Diseases* 10:1003–1006.
- Spraker T.R., R.R. Zink, B.A. Cummings, C.J. Sigurdson, M.W. Miller, and K.I. O'Rourke. 2002. Distribution of protease-resistant prion protein and spongiform encephalopathy in free-ranging mule deer (*Odocoileus hemionus*) with chronic wasting disease. *Veterinary Pathology* 39:546–556.
- VerCauteren K.C., J.L. Pilon, P.B. Nash, G.E. Phillips, and J.W. Fisher. 2012. Prion remains infectious after passage through digestive system of American Crows (*Corvus brachyrhynchos*). *PLoS One* 7(10), e45774.