



Chronic wasting disease in deer and elk: a critique of current models and their application

Eric M. Schaubert and Alan Woolf

Abstract Chronic wasting disease (CWD), a fatal transmissible spongiform encephalopathy of deer (*Odocoileus* spp.) and elk (*Cervus elaphus*), presents a challenge to wildlife managers because little is known about its transmission, yet it could severely threaten wildlife populations if action is not taken rapidly. Published mathematical models predict that CWD could devastate populations of free-living deer and elk, prompting wildlife managers to attempt large-scale eradication of deer in hopes of containing CWD outbreaks. Our objective is to critically examine the theoretical and empirical support for current models of CWD epizootiology, in light of herd health-management actions. We identify a critical, untested premise (i.e., strictly frequency-dependent transmission) that underlies the dire model predictions. We re-evaluate published comparisons of model output with field data and find little support for published model structures. Given the uncertainty surrounding the future effects of chronic wasting disease on deer and elk populations, and the potential costs of unnecessarily culling large numbers of charismatic and valuable animals, we propose that consideration of alternative models and management actions in a decision-theoretic framework is necessary for wildlife management actions to retain their scientific basis.

Key words assumptions, *Cervus elaphus*, chronic wasting disease, deer, density-dependence, elk, epizootiology, eradication, frequency-dependence, models, transmission

Chronic wasting disease (CWD) has recently emerged as a major concern of wildlife managers, biologists, and stakeholders throughout North America (Enserink 2001, Williams et al. 2002). Chronic wasting disease is a fatal transmissible spongiform encephalopathy (TSE; Williams and Young 1980) that has been observed in free-living and captive deer (*Odocoileus* spp.) and elk (*Cervus elaphus*) and is the only TSE known to persist in free-living wildlife populations (Spraker et al. 1997, Miller et al. 2000). Although CWD appears to have persisted for decades at relatively low prevalence in an enzootic region in parts of Colorado and Wyoming, recent data suggest that its prevalence in

free-living mule deer (*O. blemionus*) and elk may be increasing (Miller et al. 2000, Gross and Miller 2001). Outside this enzootic area, CWD has been detected in free-living mule deer, white-tailed deer (*O. virginianus*), or elk in Illinois, Nebraska, New Mexico, Saskatchewan, South Dakota, and Wisconsin and appears to be spreading (Williams et al. 2002). No link between CWD and disease in humans or noncervid livestock has been found, but these risks cannot be dismissed with absolute certainty (Bartz et al. 1998, Raymond et al. 2000, Hamir et al. 2001). Compounding the potential impact on wildlife and human health, CWD threatens to erode favorable public perception of wildlife resources

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and the fundamental importance of sport hunting as both a tool for management of free-ranging deer and elk populations and a major monetary source for wildlife management agencies and local economies.

While much is known about the empirical epizootiology of CWD (Williams et al. 2002), critical parameters and processes related to modes and patterns of transmission are unknown. Also, CWD epizootics in wild herds have not been observed long enough to know what their ultimate population-level effects will be. Therefore, mathematical models are critical tools for assessing the potential impact of CWD on deer and elk populations and weighing this risk against the costs of alternative management actions. Mathematical models have been developed to synthesize existing knowledge of CWD epizootiology in wild mule deer; these models uniformly predict that CWD can cause extinction of host populations (Miller et al. 2000, Gross and Miller 2001). In the face of dire model predictions, scarce data, and uncertainty, experts have recommended strong and rapid steps to contain and eradicate CWD outbreaks (Gross and Miller 2001, Williams et al. 2002). The Wisconsin Department of Natural Resources has begun an attempted eradication of all white-tailed deer within a >900-km² area where CWD has been detected (Nolen 2002), representing a prominent management philosophy and a strategy likely to be considered by many agencies responsible for managing populations at risk for CWD.

We believe it useful to critically examine the premises and empirical support of published CWD models. While we acknowledge that rapid management action to control CWD may be warranted and that wildlife managers invariably must act without the luxury of complete knowledge, we propose that science-based wildlife management will advance if competing models and management alternatives are carefully explored in a decision-theoretic framework. All scientific knowledge is tentative and provisional, and science advances by repeatedly confronting hypotheses and models with logic and data. It is in this spirit that we offer the following critique of the premises and support of current CWD models.

Theoretical foundation

A model is a formal construct that illuminates the logical consequences of the assumptions upon

which it is based, and the validity of the model as a representation of reality depends on how closely its assumptions reflect the characteristics of the real system. The published models of CWD epizootiology in wild mule deer (Miller et al. 2000, Gross and Miller 2001) share a common assumption: the number of effective contacts between an infectious individual and other individuals per unit time (β) is constant and independent of population size or density. This premise results in frequency-dependent transmission, where the force of transmission is a function of the frequency (i.e., proportion) of infectious individuals within the population (Appendix A). The idea of frequency-dependent transmission is based on the premise that opportunities for contact between an infectious individual and susceptible individuals are unaffected by population size (de Jong et al. 1995). An important distinction exists between population size and density, depending on the scale over which transmission occurs. For example, if transmission occurs exclusively within social groups and the number of individuals per group is constant, changes in the number of groups inhabiting an area will change the population density on a large scale but may not affect the local density or contact rate experienced by an individual within a group. The assumption of frequency-dependent transmission for CWD has been justified on the basis of the aggregative, migratory, and habitat-selection behaviors of wild deer (Gross and Miller 2001), but its importance to model output has not been discussed.

Epidemiological theory indicates that pure frequency-dependent transmission strongly promotes unstable host-pathogen dynamics (Getz and Pickering 1983), such that the disease either dies away ($\beta < \beta^*$, where β^* is a threshold value) or drives the host and itself to extinction ($\beta > \beta^*$). However, if β is not constant but decreases as the host density decreases, known as density-dependent transmission, the disease and its host can reach a stable equilibrium or exhibit regular cycles (Anderson and May 1978, May and Anderson 1978). The presumption of frequency-dependent versus density-dependent transmission is critical to the predicted outcome of an epizootic: host-pathogen extinction versus host-pathogen coexistence. Gross and Miller (2001:213) report "a disturbing result of this modeling exercise was our inability to identify a set of realistic parameters that permits sustained coexistence of CWD in a wild deer population." We emphasize that this dire outcome of CWD models

is entirely a predictable consequence of the frequency-dependent assumption and does not stem from any particular known characteristics of CWD.

Whether transmission is frequency-dependent or density-dependent is determined by the primary mechanism of transmission and the spatial structure of host populations. Frequency-dependent transmission is particularly likely in cases of venereal or vector-borne transmission (May and Anderson 1978, Getz and Pickering 1983) because the number of mates per individual host or host-bites per vector may be essentially independent of host density in many species. Frequency-dependent transmission also is promoted when a host population is subdivided into groups of nearly constant size, so that changes in overall population size or density do not affect the local density within groups (de Jong et al. 1995). Due to their matrilineal social structure (Hawkins and Klimstra 1970; Geist 1981, 1982; Nelson and Mech 1999), deer and elk appear to be candidates for frequency-dependent transmission. However, the CWD agent is most likely transmitted via bodily fluids both through direct contact and indirectly because the agent appears to persist in the environment (Miller et al. 1998). We argue that this combination of direct and indirect transmission is unlikely to be strictly frequency-dependent. Also, mule deer and elk (particularly females) congregate on winter range (Geist 1982, 1998), where the exudates of an infected animal potentially can contact a larger number of animals if more animals congregate in or migrate along the same area, suggesting that some form of density-dependent transmission is feasible. Group size and social structure of deer and elk also respond to changes in population density (Kie and Bowyer 1999, Hebblewhite and Pletscher 2002). Finally, if CWD transmission is strictly frequency-dependent, other diseases of deer or elk transmitted by the oral-fecal route should exhibit similar dynamics and should cause host extinction. This has not been observed.

Classical epidemiological models have been based on the premise that β is directly proportional to the population density of hosts (Kermack and McKendrick 1927, Anderson and May 1978), and such strictly linear density-dependence is clearly unrealistic for wild deer and elk. However, it also is unrealistic to presume that β is completely independent of host density. For example, CWD has become much more prevalent in captive cervid herds maintained at high densities than in free-liv-

ing herds (Williams and Young 1980, Williams et al. 2002), suggesting that population density has a positive influence on the probability of transmission. For group-living species like cervids, β may be approximately constant over a range of moderate population densities but is likely to change when population density is very low or high. Thus, β might rise to an asymptote as population density increases (Dietz 1982, Heesterbeek and Metz 1983, Antonovics et al. 1995, Heesterbeek and Roberts 1995, Ramsey et al. 2002) or could vary as a power function or some other nonlinear function of density (Figure 1). Incorporating these nonlinear forms of density-dependent transmission into the model of Miller et al. (2000) would result in persistent host-pathogen coexistence if β drops below β^* as host density drops. Output of CWD models is very sensitive to changes in the value of β (Miller et al. 2000, Gross and Miller 2001), so even weakly density-dependent transmission may enable host-pathogen coexistence. The many unknown aspects of CWD transmission prohibit robust prediction of the population impact.

Empirical support

Miller et al. (2000) tested the validity of their frequency-dependent model by comparing its predictions with empirical data relating to changes in CWD prevalence over time and patterns of CWD prevalence across sex and age classes in mule deer

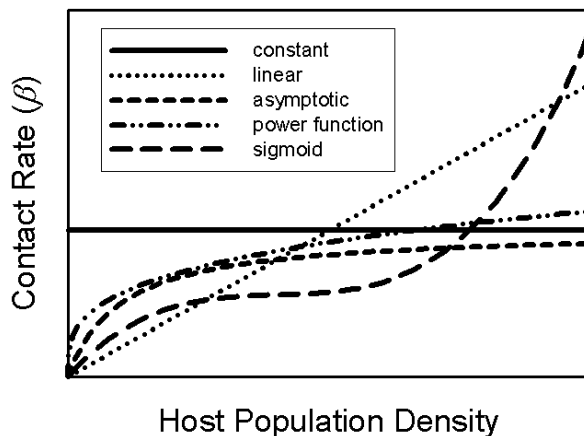


Figure 1. Plausible relationships between host population density and the number of effective contacts per unit time between each host individual and others. Current published models of chronic wasting disease assume no relationship (constant), whereas Anderson-May type models assume a linear relationship. However, there exists a range of intermediate and more complex relationships that are biologically feasible.

in Colorado. However, the apparent concordance reported by Miller et al. (2000) between observed and predicted CWD prevalence across age–sex classes is in error. As the model is described by Miller et al. (2000), it is incapable of producing a pattern in which infection prevalence differs across age classes >4 years old or between sexes (Appendix B). This contrasts with their field data and their purported model output (Miller et al. 2000: figure 4). The empirical age–prevalence relationship of CWD in free-living mule deer, particularly its rarity in older male deer, cannot be explained by the model and indicates that some important biological processes are missing from the model.

The concordance with empirical data also is questionable for the model of Gross and Miller (2001), who used the same age–prevalence data for mule deer as Miller et al. (2000) but lumped the data between sexes. The authors claimed that the nearly flat age–prevalence relationship that emerged from the model “closely matched independent field observations,” (Gross and Miller 2001:210) despite the prominent differences in observed CWD prevalence among age groups. Prevalence of CWD, averaged across age classes, was similar in model output and field data, but whether that similarity truly represents concordance with independent data is unclear. In the model of Gross and Miller (2001), CWD prevalence generally increased over time during simulation runs, so the model output would closely match observed CWD prevalence only at certain time steps. The authors neglect to indicate from what time step in the simulation the model output came and whether that time step was chosen based on criteria other than similarity to observed data.

Implications for management

Culling has been used often in attempts to contain or eliminate wildlife diseases by driving host populations below a threshold density (Barlow 1996, Wobeser 2002). However, unlike density-dependent transmission, strict frequency-dependent transmission does not permit the existence of a threshold host density below which the pathogen cannot persist (Getz and Pickering 1983). Therefore, incomplete host eradication (i.e., partial culling) can deterministically cause elimination of a disease with density-dependent but not a frequency-dependent transmission, unless infected individ-

uals can be identified and culled selectively (Gross and Miller 2001). If transmission is truly frequency-dependent, incomplete eradication might only hasten the ultimate extinction of that host population without preventing disease spread to other populations. In other words, even if the frequency-dependent assumption upon which eradication programs are based is true, it implies that eradication is unlikely to successfully control the spread of the disease unless nearly 100% of hosts are eliminated.

Given that the assumption of strict frequency-dependent transmission is both critically important and untested, it seems prudent to consider what management options might be appropriate if this assumption is untrue. If transmission is not frequency-dependent, then a threshold host density may exist. If so, that threshold host density may be high or low, relative to current densities of deer and elk. If the threshold density is high, the disease will not substantially reduce wild populations, and CWD does not endanger deer herds. If it is low, the host population must be reduced to even lower densities to locally eliminate the disease. It remains an open question whether such extreme culling programs will be logistically or politically feasible, particularly if CWD introduction is not a one-time occurrence or the CWD agent persists in the environment. Complete elimination of CWD from all North American deer and elk herds is unlikely, despite the best efforts of humans, suggesting that it could be reintroduced relatively frequently into disease-free populations (because both CWD epidemiology and proposed management actions occur on the scale of decades, “relatively frequently” might mean once per 20 years). If CWD introduction in a region is not a one-time occurrence, then CWD establishment could be prevented in the long term only by suppressing host densities for an indefinite period below the densities that would result if the disease took its course. Even if necessary and successful, defeating CWD via host eradication would come at a cost, not only economic but also in terms of public perception of wildlife resources, acceptance of management paradigms, and interruption of the hunting tradition. These costs of success and the uncertainty surrounding the necessity of host eradication should be accounted for when weighing alternative management actions.

We do not intend to imply that any attempt to manage wildlife diseases by reducing host density

is wrong or undesirable. However, the fact that all wildlife diseases are not intensively managed implies that managers implicitly weigh the costs of various actions against the risks of inaction. For CWD, models are the best available tools for estimating the risks of inaction and therefore the appropriate magnitude of response. The existence of a model of CWD epizootiology that predicts certain extinction of the host could be interpreted as justification for whatever management action is deemed most likely to prevent this dire outcome. However, any modeler with understanding of fundamental ecological theory and no consideration for validity of assumptions could produce models predicting certain extinction of one or all species for any host–pathogen (Getz and Pickering 1983), host–parasite (Hassell and May 1973), predator–prey (Murdoch and Oaten 1975), or other ecological interaction. Therefore, predictions of alternative models need to be considered and judged on the validity of their assumptions and concordance with data in order to evaluate the appropriate magnitude of response.

Our objective is to call attention to a critical untested premise (i.e., frequency-dependent transmission) of current CWD models and to temper acceptance of model predictions with the uncertainty surrounding the validity of that premise and the weakness of empirical support. Gross and Miller (2001:213) claimed that “to the extent that modeled mechanisms of CWD transmission appear to offer at least a reasonable approximation of disease processes occurring in nature, it follows that this model provides plausible forecasts of future epidemic trends.” We agree with the logic of the first part of this statement but question whether theory or data permit the acceptance of these models as a reasonable approximation of CWD transmission dynamics. The predictions of frequency-dependent models of CWD epizootiology (Miller et al. 2000, Gross and Miller 2001) represent a small set of possible outcomes of CWD epizootics in wild populations. Other outcomes are also plausible, and their actuality depends on the true (but unknown) relationships between transmission and population density, sex and age structure, and spatial structure. Current frequency-dependent models are consistent with the observed long-term persistence of CWD at low prevalence in free-living deer and elk, but this observation also is consistent with other hypotheses (e.g., CWD will remain at relatively low prevalence indefinitely). The range of possible reality states and the potential benefits and

costs of alternative management actions may be best analyzed in a decision–theoretic framework where potential costs of inaction and alternative actions are explicitly weighed and point to the urgent need for research into the transmission dynamics of CWD to firmly base management decisions on the best possible science.

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Appendix A

If the number of effective contacts per individual per year (β) is constant and population size (N) is a finite integer, then the probability of an uninfected

host becoming infected during a time step (i.e., force of infection) is given by $1 - (1 - 1/N)^{\beta}$, where I is the number of infectious hosts (McCarty and Miller 1998, Miller et al. 2000; note typographical error in Gross and Miller 2001:208). As N approaches infinity (e.g., if a large geographic area is examined) but infection prevalence ($1/N$) remains constant, this probability converges to the zero term of a Poisson distribution subtracted from unity: $1 - \exp(-\beta/N)$. Numerical analyses indicate that the finite- N and Poisson transmission probabilities differ by a factor of ~ 0.05 or less for $N > 10$. Thus, the force of transmission is primarily a function of infection prevalence and not the absolute of number of infecteds. For time steps < 1 year, as steps (and hence β) become small, the Poisson probability converges to β/N .

Appendix B

Model output reported in figure 4C–D in Miller et al. (2000) cannot be produced by the model they describe; see figure 4 in Gross and Miller (2001) for an age–prevalence relationship consistent with model structure. Below, we describe the model of Miller et al. (2000), which represents the spread of CWD in a free-living population of mule deer and prove that their model necessarily produces sex- and age-independent prevalence for above age class 4. Gross and Miller (2001) use a slight modification of the same model structure.

Yearly survival of uninfected yearling and adult deer is assumed to differ between sexes but not age classes (except that no deer survives past age class 15), and the probability of a susceptible deer becoming infected in a given year is equal for all sex and age classes except fawns (which may receive vertical transmission from their mothers). After a deer is infected, it spends 1 year in the latent stage before becoming infectious. After an animal becomes infectious, survival is successively halved in each of 3 subsequent years irrespective of sex and age. No infectious deer are allowed to live > 3 years in this model. For each sex the number of infected individuals in age class $j > 4$ in year t ($I_{t,j}$) is a function of the number of uninfected individuals in age class $j - 4$ in year $t - 4$ ($S_{t-4,j-4}$), the sex-specific yearly survival rate (ψ_{sex}), and the yearly probabilities of becoming infected for the previous 4 years ($P_{t-4}, P_{t-3}, P_{t-2}, P_{t-1}$):

$$\begin{aligned}
 I_{t,j} &= S_{t-4,j-4} \times P_{t-4} \times \psi_{\text{sex}} \times \frac{\psi_{\text{sex}}}{2} \times \frac{\psi_{\text{sex}}}{4} \times \frac{\psi_{\text{sex}}}{8} \\
 &+ S_{t-4,j-4} \times (1 - P_{t-4}) \times \psi_{\text{sex}} \times P_{t-3} \times \psi_{\text{sex}} \times \frac{\psi_{\text{sex}}}{2} \\
 &\times \frac{\psi_{\text{sex}}}{4} + S_{t-4,j-4} \times (1 - P_{t-4}) \times (1 - P_{t-3}) \times \psi_{\text{sex}} \\
 &\times \psi_{\text{sex}} \times P_{t-2} \times \psi_{\text{sex}} \times \frac{\psi_{\text{sex}}}{2} \\
 &+ S_{t-4,j-4} \times (1 - P_{t-4}) \times (1 - P_{t-3}) \times (1 - P_{t-2}) \\
 &\times \psi_{\text{sex}} \times \psi_{\text{sex}} \times \psi_{\text{sex}} \times P_{t-1} \times \psi_{\text{sex}} \\
 &= S_{t-4,j-4} \left(\psi_{\text{sex}} \right)^4 \left(\frac{P_{t-4}}{64} + [1 - P_{t-4}] \right. \\
 &\quad \times \left[\frac{P_{t-3}}{8} + [1 - P_{t-3}] \right] \\
 &\quad \left. \times \left[\frac{P_{t-2}}{2} + [1 - P_{t-2}] P_{t-1} \right] \right)
 \end{aligned}$$

The total number of individuals of that sex in age class $j > 4$ in year t ($N_{t,j}$) is given by the sum of $I_{t,j}$ and those individuals that were uninfected in year $t - 4$, avoided infection entirely, and survived for 4 years:

$$\begin{aligned}
 N_{t,j} &= I_{t,j} + S_{t-4,j-4} \left(\psi_{\text{sex}} \right)^4 \\
 &\quad \times (1 - P_{t-4})(1 - P_{t-3})(1 - P_{t-2})(1 - P_{t-1})
 \end{aligned}$$

The product $S_{t-4,j-4} \times (\psi_{\text{sex}})^4$ can be factored out of both $I_{t,j}$ and $N_{t,j}$, so infection prevalence ($I_{t,j}/N_{t,j}$) is function only of $P_{t-4}, P_{t-3}, P_{t-2}$, and P_{t-1} , which are constant across sex and age classes. We conclude that, given the assumptions upon which this model is based, infection prevalence cannot differ between sexes or among age classes above age class 4.

