

A VERSATILE MODEL OF DISEASE TRANSMISSION APPLIED TO FORECASTING BOVINE TUBERCULOSIS DYNAMICS IN WHITE-TAILED DEER POPULATIONS

Authors: McCarty, C. W., and Miller, M. W.

Source: Journal of Wildlife Diseases, 34(4) : 722-730

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-34.4.722>

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

A VERSATILE MODEL OF DISEASE TRANSMISSION APPLIED TO FORECASTING BOVINE TUBERCULOSIS DYNAMICS IN WHITE-TAILED DEER POPULATIONS

C. W. McCarty¹ and M. W. Miller²

¹ Graduate Degree Program in Ecology, Colorado State University, Fort Collins, Colorado 80523, USA (e-mail: cmccarty@lamar.colostate.edu)

² Colorado Division of Wildlife, Wildlife Research Center, 317 West Prospect Road, Fort Collins, Colorado 80526, USA (e-mail: mike.miller@state.co.us)

ABSTRACT: A model was derived for disease transmission in dynamic host populations and its application was demonstrated in forecasting possible outcomes of a bovine tuberculosis (*Mycobacterium bovis*) epidemic in a white-tailed deer (*Odocoileus virginianus*) population. The approach was mechanistic, based disease transmission on the probability of each susceptible individual becoming infected per unit time, and afforded the flexibility necessary to model epidemics in dynamic wildlife populations. This approach was applied to a sex- and age-structured deer population model. This model predicted that tuberculosis prevalence in a white-tailed deer population could rise from approximately 3% to about 21% over 25 yr, and that neither lowered deer survival nor lowered transmission would be completely effective in eliminating disease from the population. Maternal transmission appeared unimportant to modeled tuberculosis dynamics; in contrast, disease was not maintained for >15 yr in models lacking lateral transmission.

Key Words: Bovine tuberculosis, epidemic modeling, modeling, epidemiology, *Mycobacterium bovis*, *Odocoileus virginianus*, white-tailed deer.

INTRODUCTION

Modeling is an important tool for describing and predicting the dynamics and outcomes of a wide variety of ecological processes, including infectious disease transmission (Starfield and Bleloch, 1986; Grenfell and Dobson, 1995). Epidemic models have been used to investigate the role of disease in population processes, to compare disease management strategies, and to assess risk of disease transmission within and among species (Anderson and May, 1979, 1982; Grenfell and Dobson, 1995; Barlow, 1996).

Dynamic wildlife populations present unique challenges to traditional epidemic modeling approaches. A combination of features, including environmental or demographic stochasticity, nonconstant survivorship, differential susceptibility or infectiousness, individual covariates, and density-dependence may be required to realistically portray epidemics in free-ranging wildlife populations. The differential equation approach to epidemic modeling (e.g., Anderson and May, 1979) becomes mathematically intractable when such fea-

tures are incorporated (e.g., Bailey 1975; Heesterbeek and Roberts, 1995). Similarly, the Reed-Frost approach is limited by parameters that are conditional on a fixed population size throughout the duration of the epidemic (Bailey, 1975; Becker, 1981); the latter assumption may be appropriate in stable human populations, but is of limited utility in applications to fluctuating wildlife populations. In addition to these difficulties, few epidemic models can be readily parameterized from data typically provided by wildlife studies. Consequently, models depicting chronic epidemics spanning many generations of wildlife hosts are relatively uncommon (Barlow, 1995). Despite these challenges, wider availability of epidemic models could be useful to those responsible for managing wildlife disease problems.

Herein, we describe a versatile mechanistic model of disease transmission and demonstrate its application in forecasting possible outcomes of a bovine tuberculosis (*Mycobacterium bovis*) epidemic in a dynamic white-tailed deer (*Odocoileus virginianus*) population.

GENERAL MATHEMATICAL APPROACH

We represent the transition from the susceptible to the infected state with a simple mechanical model. Two assumptions are necessary for this model to apply. First, we assume the infectious subpopulation produces some number of infectious contacts per member (i) per unit time (j), denoted as $\beta_{i,j}$. We define an infectious contact as any interaction between an infectious individual and any other individual that would result in disease transmission if the other individual were susceptible. Second, we assume all individuals within the host population have equal or known probabilities of contacting any infectious individual per unit time. The probability of any one susceptible individual being infected by one or more of the I members of the infectious subpopulation per unit time ($P_{(s \rightarrow i)}$) is calculated as

$$P_{(s \rightarrow i)} = 1 - (1 - P)^I \quad (1),$$

where P is the probability of any one susceptible individual becoming infected by receiving one or more infectious contacts from a single infectious individual per unit time. We calculate the latter probability as:

$$P = 1 - \left(1 - \frac{1}{N}\right)^\beta \quad (2),$$

where β is the number of infectious contacts per infectious individual per unit time and N is the total population size. Here, $(1 - 1/N)$ is the probability of the susceptible individual *not* receiving a single infectious contact from that single infectious individual. Substituting equation 2 into equation 1 yields the probability of a susceptible individual becoming infected per unit time:

$$P_{(s \rightarrow i)} = 1 - \left(1 - \frac{1}{N}\right)^{I\beta} \quad (3).$$

Our model has two key features. First, disease transmission is driven by the number of potential infectious contacts made by an infectious individual during a given time step (β). Because these contacts are randomly allocated across the population,

some may be “wasted” on individuals that are immune or are already infected. This parameter is measurable. Moreover, β itself can be modeled to reflect changes in the nature of interactions within the host population, between host species, and with the environment. Second, this model recognizes that host population size also influences the probability of interactions between individuals during a given time step. By incorporating total population size into estimating the probability that a susceptible individual will become infected, this model allows populations to fluctuate without compromising baseline assumptions about disease transmission. It follows that three factors act in concert to affect the probability that any susceptible individual becomes infected during any given period of time: the number of infectious individuals, the number of infectious contacts each is capable of producing, and the size of the population where potential interactions may occur.

APPLICATION TO BOVINE TUBERCULOSIS EPIDEMIOLOGY

In 1994, tuberculosis was first detected among free-ranging white-tailed deer in northeastern Michigan; subsequent investigations revealed that tuberculosis was well-established in the affected deer population (Schmitt et al., 1997). Because this epidemic potentially threatened local cattle herds and presented significant obstacles to long-term wildlife resource management, Michigan's Departments of Agriculture and Natural Resources (MDNR, Lansing, Michigan, USA) requested an assessment of various associated risks (United States Department of Agriculture, unpubl. report). Using the mathematical approach outlined above, we constructed the following epidemic model as a foundation for the quantitative components of subsequent risk assessments.

MODELING METHODS

We modeled epidemic and host population dynamics as a multivariate Markov

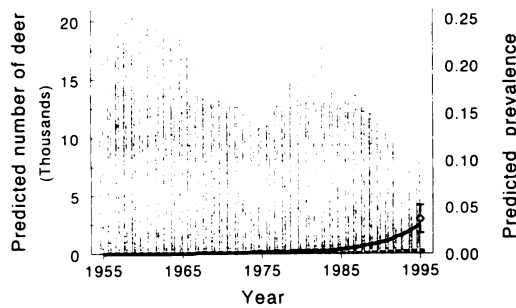


FIGURE 1. The deterministic model simulated historic trends in deer population (left axis) and tuberculosis (right axis) dynamics. Transmission coefficients ($\beta_F = 0.5$, $\beta_M = 8.1$) were selected to yield a predicted prevalence of about 3.1% 40 yr after introduction of one infected female. Gray bars are total numbers of deer predicted, black bars are numbers of infected deer predicted, and the line represents simulated prevalence. The diamond is an independent prevalence estimate derived from field data (Schmitt et al., 1997; MDNR, 1998); capped bars bound the estimate's 95% confidence interval.

process (Sharpe, 1988), with each dimension representing the number of individuals in a particular sex, age, and health class. Transitions between consecutive vectors were governed by a series of simple rules determined by population dynamics of the host species (analogous to the difference equations employed in matrix population models; Caswell, 1989) and host-parasite interactions.

Initially, we constructed a deterministic white-tailed deer population and tuberculosis epidemic model generally fitted to trends observed in the affected deer population in Michigan over the last 40 yr (Fig. 1). This preliminary model served two purposes. First, it ensured reasonable representation of historic trends in population and tuberculosis dynamics observed in the affected white-tailed deer population. Second, it provided estimated transmission coefficients for use in subsequent projections of epidemic trends. Our model featured two sexes (male, female), two age classes (fawn, adult), and four health states (susceptible, first and second yr of incubation, infectious), and operated on an annual time step. Within each time step, fawns were recruited, maternal and lateral

tuberculosis transmission occurred, yearlings and adults of all health states were removed via hunting and natural mortality, and the disease progressed in incubating animals. Yearling and adult survival rates were fixed (male = 0.45, female = 0.75) to produce male:female ratios (about 1 male:2 females before annual mortality and about 1 male:4 females after mortality) that matched field observations (MDNR, unpubl. data). To mimic processes driving white-tailed deer population dynamics (e.g., McCullough, 1979), fawn recruitment rates (the combination of fawn birth and survival rates) were adjusted annually (range 0.35 to 0.7) to fit the simulated population to historic trends (range about 7,600 to about 20,000 deer; Fig. 1) (MDNR, unpubl. data). We assumed recruitment and adult survival rates were reduced by both hunting and natural mortality, but their effects were combined in the model; we further assumed no compensation in recruitment to counterbalance reduced survival (e.g., Bartmann et al., 1992). Because average deer life spans were relatively short under these survival regimes (about 2 yr for males, about 4 yr for females), and because severe, disseminated tuberculosis was rarely observed in affected individual white-tailed deer (Schmitt et al., 1997), we assumed tuberculosis did not affect recruitment or survival.

Health states reflected exposure to and duration of infection with *M. bovis*. We modeled the transition from the susceptible to the infected state using the mechanical model described in equation 3. We assumed all deer in the population were equally susceptible to tuberculosis prior to infection. However, based on sex-related differences in prevalence initially observed in the affected population (males = 0.08, females = 0.02; S. Schmitt, pers. comm.), we assumed that males were somehow more likely than females to contact an infectious individual at each time step. We allowed fawns born to infectious

females additional opportunity to become infected via maternal transmission (T_{MAT}).

Once susceptible deer became infected, we assumed a 2 yr incubation period before infected deer could transmit *M. bovis* to other deer; based on descriptions of lesion size and distribution in white-tailed deer (Schmitt et al., 1997), it appeared to us that deer in early stages of infection would be unlikely to shed infectious doses of *M. bovis*. During the second year of incubation, we assumed tuberculosis infections would be detectable by conventional postmortem diagnostic approaches (e.g., Schmitt et al., 1997). Infectious deer remained infectious for life, each transmitting disease at set rates expressed as infectious contacts per time step between infectious individuals and females and fawns (β_F) and between infectious individuals and adult males (β_M). Females were additionally capable of transmitting infection to their fawns at a fixed rate ($T_{MAT} = 0.25$). We assumed no immunity to or recovery from tuberculosis once deer were infected.

At each time step, the probability of a susceptible deer entering the first year of incubation was determined by equation 3; the total number of first year incubators was the sum of deer newly infected via lateral transmission and via maternal transmission. The probabilities of deer moving from the first to second year of incubation and from the second year of incubation to the infectious class were sole functions of sex- and age-specific survival rates; consequently, the overall probability of infected deer surviving to become infectious was about 0.20 for males and about 0.56 for females. The model tracked total numbers of deer in each health state at each time step, and also calculated prevalence estimates: we defined the numerator for prevalence as the total number of infected deer detectable at postmortem exam (=second year incubating + infectious) to allow comparison to field data, and used total population less fawns as the denominator. The model also tracked the total number

of infected deer (=first year incubating + second year incubating + infectious).

Initial conditions were set such that population size was about 15,000 deer and increasing. We apportioned the initial population using observed sex ratio data (about 33% males:67% females before annual mortality; MDNR, unpubl. data) and adjusted fawn recruitment rates to increase and decrease population size to track historic trends (MDNR, unpubl. data). We then added one infectious female to the population, and fit the model to estimate transmission coefficients necessary to yield observed prevalences of about 2.4% in ≥ 1 -yr-old females and 8.1% in ≥ 1 -yr-old males (3.1% overall for ≥ 1 -yr-old deer) 40 yr later (Fig. 1). These transmission coefficients ($\beta_F = 0.5$, $\beta_M = 8.1$) were used as baseline epidemic conditions in our projection models.

Next, we allowed stochastic processes to influence the dynamics of the population and the epidemic to explore the range of possible future trajectories. Initial conditions for forecasting were taken from year 40 (=1995) of the deterministic model. We used Monte Carlo methods (Ross, 1997) to investigate the range of possible outcomes and to derive variance estimates of relevant functions of the population vector (e.g., yearly prevalence and probability of transition from susceptible to infected). Alternative management scenarios also were modeled. As a baseline for comparisons, we left parameters describing population and epidemic dynamics unmodified from the deterministic model and projected progress of tuberculosis in the simulated herd for 30 yr. We then considered the effects of two types of management: the first reduced survival rates, and the second reduced transmission; we examined these alone and in combination.

We also ran simulations to examine model sensitivity and effects of temporal variation on model outcomes. Because the adult survivorship of deer in North America is relatively stable but recruitment rates vary widely across time both within and

among populations (McCullough, 1979; Bartmann et al., 1992), we examined effects of temporal variation in recruitment for each scenario by modeling yearly recruitment as a Beta random variable (Ross, 1997) with mean = 0.48 and 80% of the probability mass between 0.20 and 0.75. Further, scenarios were examined with and without maternal transmission.

For each case of each scenario, we generated 1,000 possible sample paths. FORTRAN source code for these calculations is available from the authors upon request or at <http://lamar.colostate.edu/~cmccarty>.

MODELING RESULTS

Under our deterministic model's survival and transmission assumptions, the basic reproductive ratio (R_0 ; Heesterbeek and Roberts, 1995) of tuberculosis in white-tailed deer was >1.0 . Consequently, our stochastic model predicted that, given the foregoing assumptions, there is a high likelihood that tuberculosis will persist in the infected white-tailed deer population in Michigan. Model outcomes also illustrated the potential effects of prospective management interventions on the predicted trajectory of this epidemic (Figs. 2, 3). In general, reducing survival was predicted to be less effective in reducing prevalence than reducing transmission until the lower threshold for population persistence was reached (Fig. 2); reducing both survival and transmission appeared more effective than either approach alone. For example, the model predicted that prevalence may rise to about 21% over a 25 yr period in the absence of management intervention (Fig. 3A). Reducing adult survival by 10% projected drastic population declines over 25 yr, from 7,926 to 1,105 deer on average (standard deviation = 71; range 886 to 1,293) (Fig. 3B). Lowering adult survival by 10% appeared slightly less effective in reducing predicted prevalence than lowering transmission coefficients by 10%, but lowering survival reduced the average number of infected deer to about one fifth the average number predicted by lowering

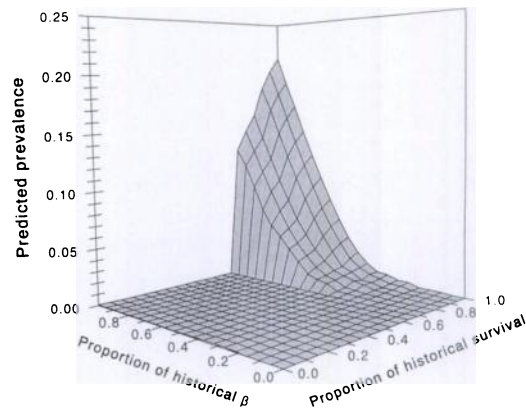


FIGURE 2. The effects of two alternative management approaches on tuberculosis prevalence were examined using our stochastic model. In general, the model predicted that lowering survival would be proportionately less effective in reducing predicted prevalence than lowering transmission until the threshold for population persistence was reached; lowering both survival and transmission appeared more effective than either approach alone.

transmission (Fig. 3B, C). The model predicted that reducing both survival and transmission by 10% would diminish both prevalence and total numbers of infected animals (Fig. 3D). Reducing survival beyond 70% of historic rates eliminated both the epidemic and the population by year 25 (Fig. 2).

Temporal variation in recruitment increased the uncertainty of observed prevalence dramatically: standard deviations increased 3.8- to 4.5-fold. However, the epidemic still persisted in all sample paths of most scenarios. Because of low recruitment rates and the long incubation period of bovine tuberculosis, maternal transmission was not a significant factor in modeled epidemics: reducing maternal transmission to zero had virtually no effect on model outcomes. In contrast, tuberculosis was not maintained for more than 15 yr in simulated populations in the absence of lateral transmission.

DISCUSSION

We believe it useful to distinguish two broad categories of modeling: model fitting and selection, and forecasting. We

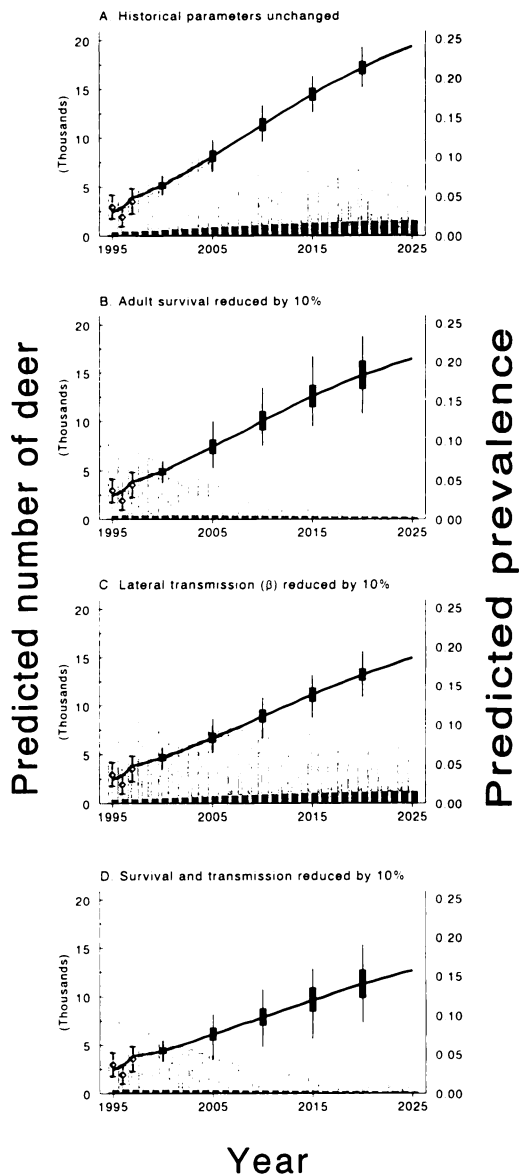


FIGURE 3. (A) Models with unaltered parameters predicted that tuberculosis prevalence (right axis) would increase to about 21% over the next 25 yr. (B) Models that reduced survival by 10% produced only a modest reduction in predicted prevalence, but forecast dramatic declines in mean population size (left axis) and total infected animals (left axis). (C) Models with transmission coefficients reduced by 10% also produced a modest reduction in predicted prevalence without affecting population numbers. (D) Models that reduced both survival and transmission by 10% diminished both prevalence and total numbers of infected animals. Gray bars are total numbers of deer predicted, black bars are numbers of infected deer predicted, and the line represents predicted preva-

consider fitting and selection as a branch of statistics concerned with the efficient use of data when inferences are limited to the process that created those data. Here, the usual goal is to select a model, from among candidate models, that achieves the best compromise between bias and precision (Burnham and Anderson 1992). In contrast, forecasting is predicting outcomes in the absence of data generated from the process being investigated (Caswell, 1989). Thus, all models of future events (in our context, modeling the possible trajectories of epidemics) are exercises in forecasting. The foregoing description of our deer-tuberculosis model illustrates several important facets of a general approach to epidemic modeling: model selection, fitting, and forecasting. As a final step, we encourage careful appraisal of model assumptions and outcomes. We believe this last step to be perhaps the most useful product of any modeling exercise.

Our deer-tuberculosis model illustrates the importance of appraising model outcomes. For example, our model predicted that tuberculosis is likely to persist in the affected deer population for >25 yr under virtually any plausible management strategy and that even a substantial increase in deer harvest is unlikely to eliminate tuberculosis without imperiling long-term population survival. Although the model forecast combined reductions in transmission and survival as the most effective management strategy, no proven methods for such intervention have been identified. It is conceivable that severely reducing adult survival (e.g., >10%) will also reduce tuberculosis transmission, but we can offer no unequivocal support for this prediction.

←
 lence; vertical lines span the entire range of prevalence predictions, and the wider portions of those lines are ± 1 standard deviation of prevalence estimates from 1,000 sample paths. Diamonds are independent prevalence estimates derived from field data (Schmitt et al., 1997; MDNR, 1998); capped bars bound the estimates' 95% confidence intervals.

Also, because the effects of increased harvest on deer social structure are largely unknown, reducing the overall number of deer may not reduce average group size or social interactions per unit time; in the absence of such reductions, culling may be relatively ineffective as a disease management tool (Barlow, 1996). Clearly, management experiments designed to test these predictions are warranted.

Assumptions about disease transmission also deserve retrospective scrutiny, as our deer-tuberculosis model illustrates. As in all epidemic models, prevalence dynamics in our model were driven by transmission coefficients, the true values of which remain unknown. We estimated these coefficients by fitting the deterministic model to our and others' perceptions of both population and disease trends. One perception influencing transmission coefficients was that of the epidemic's historic duration. We were forced to choose a somewhat arbitrary date for the epidemic's origin because no one actually knows when bovine tuberculosis was first introduced into the deer population in Michigan, or by what means that introduction occurred. Available information strongly suggested that *M. bovis* had been enzootic in the affected deer population for at least 20 yr: in 1975, a seemingly isolated case of bovine tuberculosis was diagnosed in a hunter-killed deer from this same population (Schmitt et al., 1997). We believed it highly unlikely that this was the affected population's first tuberculosis case. Several plausible sources of infection for free-ranging deer existed in Michigan about four decades ago: bovine tuberculosis occurred in cattle, and in captive exotic and white-tailed deer in Michigan in the 1950's (Ferris et al., 1961, Towar et al., 1965). Consequently, we believed 1955 (40-yr-ago) was a reasonable but conservative estimation of the epidemic's origin. Observed data generally support this assumption. A more recent introduction (e.g., 5- to 10-yr-ago) would have required considerably higher transmission rates to elevate

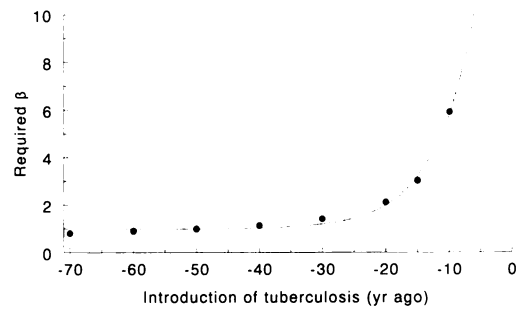


FIGURE 4. The deterministic model also was used retrospectively to examine the likely historic duration of the tuberculosis epidemic in Michigan. Assumptions about epidemic duration influenced the values of transmission coefficients (β) necessary for the model to approximate observed prevalence data (Schmitt et al., 1997). We assumed tuberculosis was introduced 40-yr-ago; a more recent introduction (e.g., 5- to 10-yr-ago) would have required considerably higher coefficients to elevate prevalence to levels observed in 1995.

prevalence to levels observed in 1995 (Fig. 4). Using such transmission rates, our model projected that prevalence should now be doubling annually; the latter has not been observed (Schmitt et al., 1997; MDNR, 1998) (Fig. 3). Although more distant introductions (e.g., 50- to 70-yr-ago) seemed feasible (Fig. 4), such scenarios might have tended to underestimate transmission rates and subsequently overestimate potential efficacy of management interventions. Because these assumptions have such profound effects on epidemic dynamics, experimental data on or independent estimates of tuberculosis transmission among white-tailed deer would greatly enhance the reliability of future modeling efforts.

The violation of many other less obvious model assumptions also could affect forecasts. For example, although possible (Morris and Pfeiffer, 1995), for lack of evidence we assumed that no secondary infectious reservoir existed or will exist among sympatric species. The presence of a secondary reservoir could cause our model to underestimate future prevalence and probability of persistence. Similarly, an environmental source of infection (e.g.,

contaminated food sources; Schmitt et al., 1997) also could cause the model to underestimate prevalence and persistence in the future. To address such possibilities, equation 3 could be modified to include a parameter representing constant background risk posed by an additional source of infection:

$$P_{(s \rightarrow i)} = 1 - \alpha \left(1 - \frac{1}{N}\right)^{I\beta} \quad (4),$$

where α is the probability of *not* becoming infected via contact with the environmental source of infection during each time step (C. W. McCarty, unpubl. data) (note that when $\beta = 0$, all transmission comes from environmental sources). It is also conceivable that β is density-dependent. For lack of evidence, our model assumed tuberculosis transmission coefficients were density-independent. As a result, the probability of disease persistence may have been overestimated.

Assumptions about population structure and white-tailed deer behavior could also affect model outcomes. Based on local perceptions and anecdotal evidence, we modeled the study population as closed. Emigrating infected deer, possibly searching for new sources of food after cessation of supplemental feeding, could cause new epidemics in neighboring populations. Also, because white-tailed deer groups are matriarchal (McCullough, 1979; Lagory, 1986; Nelson and Mech, 1987), estimates of transmission parameters made under a random mixing assumption may be unsuitable for forecasting. Disruption of matriarchal groups via increased harvest or other processes could affect tuberculosis transmission in ways not accounted for in our model, causing the model to either over- or underestimate prevalence trends. Moreover, lack of spatial structure in our model may have oversimplified disease dynamics by neglecting potential heterogeneity of tuberculosis distribution and prevalence (e.g., Cheeseman et al., 1981; Barlow, 1991); as a result, culling focussed in high prevalence areas could be more ef-

fective than predicted by our model. Despite its potential shortcomings, we believe this model has at least served to raise questions about tuberculosis epidemiology in white-tailed deer that may stimulate further empirical investigations.

In conclusion, we suggest equation 3 as a plausible general model of the transition from the susceptible to the infected state. This model offers several desirable features. For example, β has a mechanistic interpretation: in equation 3, β is the mean number of infectious contacts between infectious individuals and any other member of the host population per unit time. It follows that values for β could be measured via controlled experiments. Unlike the transmission parameter of the Reed-Frost and Greenwood models, β is independent of population size. However, by holding N constant across time steps, equation 3 simplifies to the Reed-Frost and Greenwood models (Fine, 1977), the only epidemic models to date that have been subjected to rigorous model fitting and selection (Bailey 1975; Becker, 1981). In the context of individual-based models (DeAngelis and Gross, 1992), equation 3 can be interpreted as the probability of a susceptible individual receiving one or more of the available infectious contacts ($I\beta$) per time step. This model can be adapted from discrete to continuous time, although the resulting differential equations are intractable (C. W. McCarty, unpubl. data). Finally, we believe our approach addresses at least two of the needs identified in Barlow's (1995) critical review of wildlife disease modeling: equation 3 is relatively simple, and it allows reconciliation of deterministic and stochastic models.

ACKNOWLEDGMENTS

This work was supported by Federal Aid in Wildlife Restoration Project W-153-R. We thank S. Schmitt, E. Carlson, and T. Carlson for access to unpublished MDNR data and assistance with model development; B. Corso, B. Wagner, D. Norden, H. S. Hurd, C. Bruning-Fann, L. Paisley, O. Williams, and M. Chad-dock also provided useful suggestions during

the modeling process. J. Gross, T. Shenk, R. Bartmann, V. Apanius, and two anonymous reviewers provided valuable reviews of earlier manuscript drafts.

LITERATURE CITED

- ANDERSON, R. M., AND R. M. MAY. 1979. Population biology of infectious diseases: Part I. *Nature* 280: 361–367.
- , AND ———, (editors). 1982. Population biology of infectious diseases. Life sciences research report # 25. Springer-Verlag, Berlin, Germany, 314 pp.
- BAILEY, N. T. J. 1975. The mathematical theory of infectious diseases and its applications, 2nd ed. Charles Griffin & Co., London, UK, 413 pp.
- BARLOW, N. D. 1991. A spatially aggregated disease/host model for bovine Tb in New Zealand possum populations. *Journal of Applied Ecology* 28: 777–793.
- . 1995. Critical evaluation of wildlife disease models. *In Ecology of infectious disease in natural populations*, B. T. Grenfell and A. P. Dobson (eds.). Cambridge University Press, Cambridge, UK, pp. 230–259.
- . 1996. The ecology of wildlife disease control: Simple models revisited. *Journal of Applied Ecology* 33: 303–314.
- BARTMANN, R. M., G. C. WHITE, AND L. H. CARPENTER. 1992. Compensatory mortality in a Colorado mule deer population. *Wildlife Monographs* 121: 39 pp.
- BECKER, N. 1981. A general chain binomial model for infectious diseases. *Biometrics* 37: 251–258.
- BURNHAM, K. P., AND D. R. ANDERSON. 1992. Data-based selection of an appropriate biological model: The key to modern data analysis. *In Wildlife 2001: Populations*, D. R. McCullough and R. H. Barret (eds.). Elsevier Science Publishers, Ltd., London, UK, pp. 16–30.
- CASWELL, H. 1989. Matrix population models, construction, analysis and interpretation. Sinauer Associates, Sunderland, Massachusetts, 328 pp.
- CHEESEMAN, C. L., G. W. JONES, J. GALLAGHER, AND P. J. MALLINSON. 1981. The population structure, density and prevalence of tuberculosis (*Mycobacterium bovis*) in badgers (*Meles meles*) from four areas of south-west England. *Journal of Applied Ecology* 18: 795–804.
- DEANGELIS, D. L., AND L. J. GROSS. 1992. Individual based models and approaches in ecology: Populations, and ecosystems. Chapman Hall, New York, New York, 525 pp.
- FERRIS, D. H., P. D. BEAMER, J. O. ALBERTS, AND D. TRAINER. 1961. Tuberculosis in transported deer. *Journal of the American Veterinary Medical Association* 138: 326–328.
- FINE, P. E. M. 1977. A commentary on the mechanical analogue to the Reed-Frost epidemic model. *Journal of Epidemiology* 106: 87–100.
- GRENFELL, B. T., AND A. P. DOBSON (editors). 1995. Ecology of infectious disease in natural populations. Cambridge University Press, Cambridge, UK, 521 pp.
- HEESTERBEEK, J. A. P., AND M. G. ROBERTS. 1995. Mathematical models for microparasites of wildlife. *In Ecology of infectious disease in natural populations*, B. T. Grenfell and A. P. Dobson, (eds.). Cambridge University Press, Cambridge, UK, pp. 90–122.
- LAGORY, K. E. 1986. Habitat, group size, and the behaviour of white-tailed deer. *Behavior* 98: 168–179.
- MCCULLOUGH, D. R. 1979. The George Reserve deer herd, population ecology of a K-selected species. University of Michigan Press, Ann Arbor, Michigan, 271 pp.
- MICHIGAN DEPARTMENT OF NATURAL RESOURCES. 1998. Northeast Michigan surveillance activities for bovine tuberculosis in the livestock and free-ranging deer populations. Update: January 20, 1998. Michigan Department of Natural Resources, Wildlife Division, East Lansing, Michigan, 10 pp.
- MORRIS, R. S., AND D. U. PFEIFFER. 1995. Directions and issues in bovine tuberculosis epidemiology and control in New Zealand. *New Zealand Veterinary Journal* 43: 256–265.
- NELSON, M. E. AND L. D. MECH. 1987. Demes within a northeastern Minnesota deer population. *In Mammalian dispersal patterns: The effects of social structure on population genetics*, B. D. Chepko-Sade and Z. T. Halpin, (eds.). University of Chicago Press, Chicago, Illinois, pp. 27–40.
- ROSS, S. 1997. Simulation. 2nd ed. Academic Press, San Diego, California, 282 pp.
- SCHMITT, S. M., S. D. FITZGERALD, T. M. COOLEY, C. S. BRUNING-FANN, L. SULLIVAN, D. BERRY, T. CARLSON, R. B. MINNIS, J. B. PAYEUR, AND JAMES SIKARSKIE. 1997. Bovine tuberculosis in free-ranging white-tailed deer from Michigan. *Journal of Wildlife Diseases* 33: 749–758.
- SHARPE, M. 1988. General theory of Markov processes. Boston Academic Press, Boston, Massachusetts, 419 pp.
- STARFIELD, A. M., AND A. L. BLELOCH. 1986. Building models for conservation and wildlife management. Macmillan Publishing Company, New York, New York, 253 pp.
- TOWAR, D. R., R. M. SCOTT, AND L. S. GOYINGS. 1965. Tuberculosis in a captive deer herd. *American Journal of Veterinary Research* 26: 339–346.

Received for publication 30 May 1997.