Ecological Applications, 5(4), 1995, pp. 1133–1140
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CONTROLLING LYME DISEASE BY MODIFYING THE DENSITY AND SPECIES COMPOSITION OF TICK HOSTS[1]

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Abstract. We used computer simulation of Lyme disease to test the hypothesis that infection rates of ticks and risk to humans could be reduced by controlling populations of the vertebrate hosts of vector ticks. The model was patterned after the life cycle of the deer tick (*Ixodes scapularis*). Annual survival and fecundity were dependent on the availability of hosts. The likelihood of ticks becoming infected depended on the reservoir competence of hosts and the previous exposure of hosts to infected ticks. Our results demonstrated that the density of ticks was more sensitive to the availability of hosts for juveniles than hosts for adults, suggesting that control of Lyme disease by reducing host densities would prove difficult, since the numerous alternative hosts for juvenile ticks are not amenable to control. The infection rate of ticks depended critically on the reservoir competence of host species parasitized by juveniles. We conclude that measures that alter the species composition of small mammals and birds, and hence modify the average reservoir competence of hosts for juveniles, could be used to manipulate the risk of Lyme disease without actually changing the density of ticks.

Key words: Borrelia burgdorferi; *disease; epizootiology; host–parasite models;* Ixodes scapularis; *Lyme disease; mathematical epidemiology; simulation model.*

INTRODUCTION

The past 15 years have seen good progress in the mathematical theory of disease dynamics in natural populations (e.g., Anderson and May 1978, 1979, Murray et al. 1986, Dwyer et al. 1990). These models have been especially useful for suggesting general principles of disease transmission and evolution, for posing new hypotheses, and for evaluating hypotheses that could not be tested in experimentally intractable host–pathogen systems. We have designed a simple epizootiological model for the dynamics of Lyme disease in tick populations to test hypotheses about how vertebrate hosts contribute to human infection risk. Our modelling approach allows us to explore the likely consequences of particular host combinations that would be difficult or impossible to generate in field situations, and supports several recommendations for environmental management of Lyme disease.

Lyme disease, first diagnosed in 1977, is widespread in Europe and has rapidly become the most prevalent vector-borne human disease in North America (Steere et al. 1977, Burgdorfer et al. 1982, Barbour and Fish 1993). Population densities of the deer ticks, *Ixodes scapularis* (formerly *I. dammini*), which transmit Lyme disease to humans in the eastern United States, have increased substantially over the past several decades, particularly in areas densely inhabited by people (Ginsberg 1993). Adult deer ticks typically parasitize white-tailed deer (*Odocoileus virginianus*), and juvenile ticks parasitize numerous species of mammals, birds, and reptiles. The recent increase in tick numbers is thought to result from increasing population densities of their hosts, particularly deer, but there is uncertainty over whether the densities of ticks could be controlled by artificially reducing the numbers of specific hosts (Lane et al. 1991, Barbour and Fish 1993, Ginsberg 1993, Wilson and Deblinger 1993). The uncertainty persists because critical experiments cannot be done: the principal hosts cannot be experimentally manipulated on relevant temporal and spatial scales. This paper poses a partial solution to this problem. We construct a computer model of the dynamics of Lyme disease, and use it to address the hypothesis that mammal control could help contain the incidence of the disease. The results are important for deciding among management options for Lyme disease, and may have general implications for recent efforts to control diseases through environmental modification (Allen and Bath 1980, Molyneux 1982, Young et al. 1988, Jaenson et al. 1991).

THE MODEL

The model focuses on the population dynamics of ticks, because the risk to humans of contracting Lyme disease is related to the density of infected ticks (Barbour and Fish 1993). The structure of the model is based upon the life cycle of *Ixodes* (Fig. 1). In the northeastern United States, three post-egg stadia occur over a 2-yr period: the first juvenile (larval) stage typically does not carry the bacterium that causes Lyme disease (*Borrelia burgdorferi*), but the second juvenile

[1] Manuscript received 4 March 1994; revised 7 September 1994; accepted 16 November 1994; final version received 3 January 1995.

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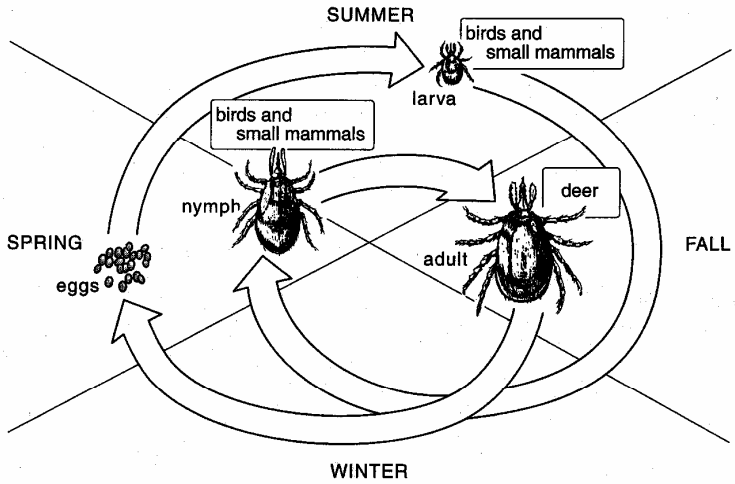

FIG. 1. Diagram of the life history of the deer tick, *Ixodes scapularis,* in the eastern United States, illustrating seasonal patterns of occurrence and the principal hosts for the three stages.

(nymphal) stage and the adult stage can occur in either infected or uninfected forms.

We have written equations for each of the stadia, and for infected and uninfected individuals within stadia, following traditional practice in epidemiological modelling (Anderson and May 1979). In the equations that follow, densities of individuals (number per hectare) in the three stadia are represented by capital letters (L, N, and A for larvae, nymphs, and adults, with u and i referring to uninfected and infected populations). The proportion of individuals surviving between stadia is separated into a probability of finding a suitable host (P, for the probability of finding hosts during the two juvenile stadia) and a term expressing the probability of survival for ticks that successfully locate hosts (s). The production of larvae depends on the probability of locating deer in the adult stage (R) and on fecundity and survival of adult females (both incorporated in f). When parasitizing a host, larvae and nymphs become infected with spirochetes at a frequency of c, which measures the reservoir competence of the host, defined here as the product of the proportion of hosts that are carriers of *Borrelia,* γ, and the transmission rate from infected hosts to uninfected ticks, r. Thus, $c = \gamma r$. The model is:

$$L_{t+1} = fR(Au_t + Ai_t) \quad (1)$$

$$Nu_{t+1} = sPL_{t+1}(1 - c) \quad (2)$$

$$Ni_{t+1} = sPL_{t+1}c \quad (3)$$

$$Au_{t+1} = sPNu_t(1 - c) \quad (4)$$

$$Ai_{t+1} = sP(Ni_t + cNu_t). \quad (5)$$

Subscripts refer to time steps of 1 yr. The entire life cycle is completed in two time steps, as is the case for *I. scapularis* in nature (Fig. 1), because nymphs are produced from larvae without changing the value of t. Steady-state solutions for this set of equations were determined by iterating 1000 generations on a computer; the model always reached a stable point equilibrium because of the strong interference competition among ticks for hosts.

The model includes five functions or constants, which together determine its behavior: survival (s), host-finding (P and R), fecundity (f), and the reservoir competence of hosts (c). We analyzed the model for a series of functional forms and parameter values that were determined from published information on the natural history of *I. scapularis.*

The proportion of individual ticks that successfully locate hosts (P and R) depends on the availability of vertebrates, and our study focused on the consequences of different host densities on tick abundances. There is field evidence for host-density dependence in other tick species (Green et al. 1943, Smart and Caccamise 1988, Young et al. 1988), and we consider it likely in *I. scapularis* as well. We assume that the probability of an individual tick not locating a host is a negative exponential function of host density, so that the probabilities of locating hosts are:

$$P = 1 - \exp\left(-\eta \sum^{n} H\right) \quad (6)$$

and

$$R = 1 - \exp(-\delta D), \quad (7)$$

where H is the density (number per hectare) of the hosts parasitized by juvenile *Ixodes,* D is the density of hosts for adults, n is the number of host species available to juveniles, and η and δ are constants that determine the efficiency of host-finding. For juvenile ticks, the probability of locating a host is a function of the summed

densities of all potential host species. Other monotonically increasing functions relating host-finding to host density generate results similar to those reported here.

We introduced interference competition among ticks for access to hosts by imposing a limit to the number of ticks that could be supported by each individual host during 1 yr. Thus, we used Eq. 6 as long as the number of ticks per host for juveniles did not exceed the limit k_j, and we assumed that all hosts were saturated with juvenile ticks otherwise. The maximum number of ticks per individual host was allowed to vary among host species. Likewise, we used Eq. 7 when the number of ticks per host for adults was less than k_a, and we required that all hosts for adult ticks were saturated otherwise.

Field data suggest that a host for adults can support more adult ticks per year than a host for juveniles can support juvenile ticks (white-tailed deer carry up to 500 adult ticks at a time: Piesman et al. 1979, Anderson and Magnarelli 1980, Wilson et al. 1990; white-footed mice [*Peromyscus leucopus*] carry up to ≈40 juvenile ticks: Levine et al. 1985, Fish and Daniels 1990). Even if adult ticks remain attached to their hosts for a longer period than do juvenile ticks, we can safely assume that each adult host supports at least five times as many ticks as each host for juveniles. For most analyses we set $k_j = 200$ juveniles/yr and $k_a = 1000$ adults/yr.

We assigned values for survival and fecundity based on published information ($s = 0.15$ and $f = 1250$ larvae/female; Carey et al. 1980, Daniels and Falco 1989). Adjusting survival and fecundity estimates had no qualitative impact on the model's behavior. There are no field data suggesting that survival or fecundity of deer ticks are density dependent, and known sources of variation in mortality and fecundity seem unlikely to generate density dependence (Fish 1993).

The reservoir competence of small mammals and birds is defined as $c = \gamma r$, the product of the proportion of hosts for juveniles that are infected with spirochetes (γ) and the proportion of uninfected juvenile ticks that become infected when they parasitize an infected host (the transmission rate, r). Reservoir competence changes seasonally and probably between years, reflecting changing probabilities of parasitism, transmission, and immunity. Field evidence suggests that a positive feedback loop exists between tick infection rates and *Borrelia* incidence in hosts: increasing numbers of infected nymphal ticks transmit the spirochete to an increasing proportion of the host population (Mather 1993, Tälleklint et al. 1993). Our model incorporates this mechanism by allowing the infection rate of hosts for juveniles to vary with the density of infected nymphs:

$$\gamma = 1 - \exp(-\epsilon Ni_{t-1}), \tag{8}$$

where Ni_{t-1} is the density of infected nymphs in the previous generation, and ϵ is an empirically determined constant that controls the efficiency of the feedback process.

The transmission rate from infected hosts to ticks, r, may also depend on the density of ticks if vertebrate hosts acquire immunity to ticks and prevent them from feeding to repletion (Godsey et al. 1987, Brown 1988, Davidar et al. 1989). This process is not well understood and we have not incorporated it into the model, but its probable effect would be to decrease reservoir competence or tick survival.

Analysis of the model focused on the responses of ticks to changes in host density. We are particularly interested in how host density influences the density of infected nymphs, which we interpret as a measure of the risk to humans of exposure to *Borrelia,* because most cases of Lyme disease can be traced to parasitism by an infected *Ixodes* nymph (Barbour and Fish 1993). We also present results for the combined density of infected and uninfected nymphal ticks and the prevalence of *Borrelia* in nymphs, since these two responses together allow us to understand patterns of Lyme disease risk.

Results

Responses to hosts for adult and juvenile ticks

Tick density responded differently to changes in the density of hosts for juveniles vs. hosts for adults (Fig. 2A). Low densities of hosts for either adults or juveniles were insufficient to maintain populations of *Ixodes.* Viable tick populations were supported when the density of hosts for juveniles became sufficiently high for larval and nymphal ticks to locate them, and increased continuously thereafter with increasing host density. Change in the density of hosts for adults was accompanied by change in tick density only at relatively low host densities; once host densities became high enough for all adult ticks to find a host, further increase in host density was inconsequential. Thus, the density of infected ticks was highly sensitive to the density of hosts for adults only within a narrow band of parameter combinations, whereas tick densities were sensitive to the density of hosts for juveniles in most situations, even when both hosts were plentiful (Table 1).

The tick infection rate showed little response to the density of hosts for adult ticks, and was positively related to the density of hosts for juveniles at low densities (Fig. 2B, Table 1). Hosts for adults influenced the infection rate only when they were so scarce that a tick population could not be maintained. Increasing density of hosts for juveniles caused the tick infection rate to increase until all hosts carried *Borrelia,* at which point the infection rate stabilized at a value equal to the infectiveness of hosts to ticks. When density of hosts for juveniles was sufficiently low, the population of ticks carried no spirochetes; in this case there were too few infected ticks to inoculate a substantial fraction

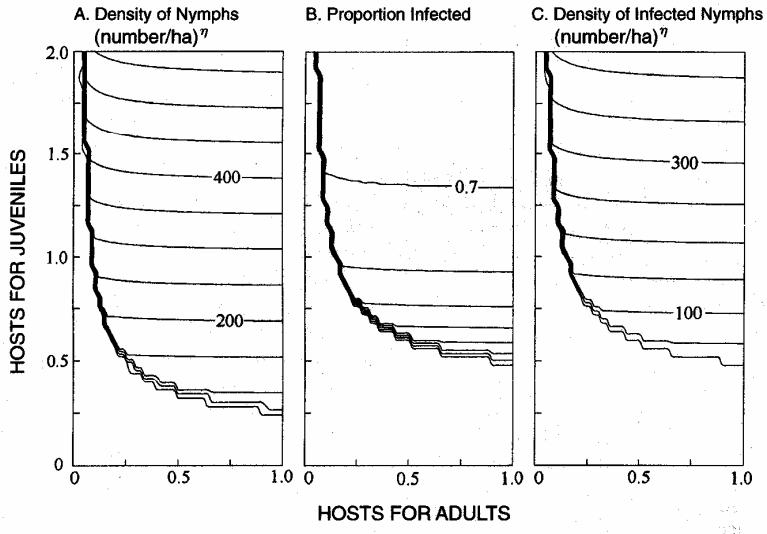

FIG. 2. Results of a simulation of the dynamics of Lyme disease in populations of *Ixodes* ticks, depicting the impact of changing densities of hosts for adult and juvenile ticks. The three panels illustrate responses of the density of nymphal ticks (A), the proportion of nymphs carrying spirochete infection (B), and the density of infected nymphs (C). All responses are at equilibrium after 1000 generations. The number of hosts on the axes of these figures is represented by the power of the exponent in the host-locating functions (Eqs. 6 and 7), equal to the actual density of hosts (number/ha) multiplied by an exponential coefficient (η: hosts for juveniles; δ: hosts for adults; both exponents set equal to 0.1 in these simulations). The responses of adult ticks are similar to those of nymphs.

of hosts, and the disease reservoir could not be maintained in the host population.

The risk of Lyme disease, indicated by the density of infected nymphal ticks, was sensitive to the availability of hosts for juveniles but not hosts for adults (Fig. 2C). Again, there was a region of parameter space in which a tick population existed but no nymphs carried *Borrelia*.

Responses to alternative hosts for juveniles

The impact of alternative species of hosts for juvenile ticks depended on the number of ticks the hosts could support and the reservoir competence of the alternative hosts. If two hosts could support similar numbers of juvenile ticks, then the absolute combined abundance of the hosts determined the equilibrium density of ticks (Fig. 3A). Other analyses showed that if host species differed in their ability to carry ticks, their relative abundance also affected the density of ticks.

The contribution of alternative hosts for juveniles to the infection rate of ticks depended on the hosts' reservoir competence. When one host had a higher infection rate or transmission rate than the other host, the prevalence of infection in nymphs responded more strongly to changes in the density of the more competent reservoir (Fig. 3B). In this analysis, the reservoir competence of one host is 10 times that of the other, which is intended to approximate the difference between the white-footed mouse and the eastern chipmunk (*Tamias striatus*; Mather et al. 1989), two common *Ixodes* hosts in the eastern United States. When the more competent host was common, increasing numbers of the less competent host decreased the infection rate, because the overall reservoir competence of avail-

TABLE 1. Sensitivity analysis of the Lyme disease model. Entries in the table give the proportional change in each response caused by a 10% change in the value of each parameter, while holding all other parameters constant. Under the conditions for which this analysis was carried out (hosts for adults and juveniles are plentiful, with hosts for juveniles 10 times more abundant than hosts for adults), both nymphal density and infection rate are much more sensitive to changes in availability of hosts for juveniles than hosts for adults.

Parameter (symbol)	Initial value	Response in nymph density	Response in proportion infected
Density of hosts for adults (δD)	0.50	0.002	0.0
No. of ticks on hosts for adults (k_a)	1000	0.0	0.0
Density of hosts for juveniles (ηH)	5.00	0.10	0.0
No. of ticks on hosts for juveniles (k_j)	200	0.10	0.0
Transmission from hosts to ticks (r)	0.75	0.0	0.10
Annual survival of ticks (s)	0.15	0.10	0.0
Tick fecundity (f)	1250	0.002	0.0

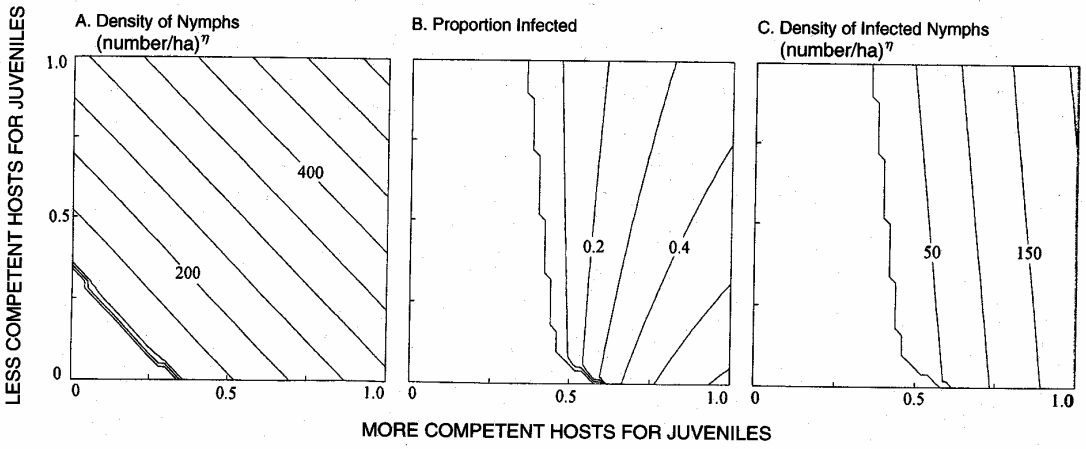

FIG. 3. Results of a simulation of the dynamics of Lyme disease in populations of *Ixodes* ticks, depicting the effect of changing densities of two species of hosts for juvenile ticks differing in their reservoir competence by a factor of 10 (transmission from infected host to tick, r, is 0.75 and 0.075 for the two hosts). Responses and axes correspond to those in Fig. 2. Reservoir competence has no effect on the ability of hosts to support ticks, but has an important impact on tick infection rate and the risk of Lyme disease.

able hosts was diluted by the increasing numbers of the less competent host. Increasing the less competent host had no impact or resulted in slightly higher tick infection rates when the more competent host was rare.

The density of infected nymphs also was more sensitive to the availability of the more competent reservoir species than the less competent one (Fig. 3C). When the host community was dominated by the less competent reservoir (left side of Fig. 3C), there were large numbers of ticks but no *Borrelia* infection.

We performed additional simulations in which a third, even less reservoir-competent, host for juveniles was introduced into the system, and found that the additional host increased the numbers of nymphal ticks but had little impact on the Lyme disease risk. The highest infection rates occurred when the most competent host species was alone in the system; addition of the less competent second and third hosts diluted the proportion of nymphs that carried *Borrelia*. However, those additional hosts also supported higher tick populations, and so their net effect was to increase Lyme disease risk slightly.

DISCUSSION

Our simulation of the dynamics of *Ixodes* ticks and spirochete infection produced two clear results: (1) the abundances of hosts for juvenile and adult ticks had differing effects on tick numbers, and (2) the prevalence of spirochete infection in ticks depended on the average reservoir competence of the collection of hosts available for juvenile ticks. These findings make good biological sense and are in agreement with the literature on Lyme disease ecology; as such, they provide insight into the causes of earlier field observations and inspire confidence in the validity of the model. Furthermore, the results support a novel recommendation for controlling Lyme disease through management of host community structure.

Impact of hosts on tick abundance and infection

Although hosts for both adults and juveniles are essential for completion of the *Ixodes* life cycle, our model suggests that adult hosts make no contribution to controlling tick population densities under most circumstances. At least two processes account for this result. First, there are far more larval and nymphal ticks seeking hosts than adult ticks, so juveniles are much more crowded on their hosts than are adults. Second, each host for adults can support more ticks than can each host for juveniles, as is the case in nature, again making hosts for juveniles more likely to be limiting than hosts for adults. Consequently, limits to the population density of ticks are set by the availability of hosts for juveniles rather than hosts for adults, except when hosts for adults are relatively rare.

In fact, throughout eastern North America hosts for adult *I. scapularis* (mostly white-tailed deer) occur at very low densities relative to hosts for juvenile ticks (many species of vertebrates). It is possible, then, that this system typically lies along the left edge of the parameter space depicted in Fig. 2, near the region where hosts for adults dramatically affect tick population density. Data from field studies do not support this interpretation, however, because hosts for adult ticks are not limiting at natural densities. Deer eradication, reduction, and exclusion experiments demonstrate that extremely low densities of deer are necessary to produce measurable change in the abundance of ticks (Wilson et al. 1988*b*, Deblinger et al. 1993, Stafford 1993, Wilson and Deblinger 1993, Duffy et al. 1994).

Corresponding experiments on hosts for juvenile *I. scapularis* have not been performed.

The model also makes a collection of predictions about how hosts influence tick infection rates. We found that hosts for adult ticks had essentially no effect on the proportion of ticks that carry spirochetes, because hosts for adults do not act as reservoirs for *Borrelia* and have relatively little impact on tick densities. A similar conclusion was reached by Porco (1991), who showed how changes in deer density should be accompanied by only moderate changes in the tick infection rate, except when deer become extremely scarce.

We found instead that the frequency of infection in ticks depended exclusively on the density and species composition of hosts for juveniles, because hosts for adults were not competent reservoirs, and because of positive feedback between spirochete incidence in hosts for juveniles and the ticks that feed on them. In the model, species of hosts for juveniles with high reservoir competence exerted a disproportionate effect on the numbers of infected nymphs. Tick infection rate varied independently from tick density, even to the point of producing dense tick populations that carry no spirochetes, because of the differential impact of host species that differ in their reservoir competence.

Few data exist to assess these predictions. The exceptionally high frequency of tick infection on coastal islands, where the highly reservoir-competent *Peromyscus* dominates the community of potential hosts, supports the notion that juvenile reservoirs influence infection rates (Mather 1993). Also, Lyme disease is rare or absent in the southeastern United States, where there are high populations of *I. scapularis* and diverse communities of lizards and small mammals that are poor reservoirs for *Borrelia* (Lane 1990, Ginsberg 1993, Mather 1993). It appears, then, that Lyme disease occurs most frequently in regions where highly competent reservoir species predominate, which supports the prediction that vertebrate species composition affects the prevalence of spirochetes.

The reduction in tick infection with increased host diversity will be especially likely if the population densities of various hosts are interdependent (e.g., when hosts compete for limiting resources). Competition among host species implies that changes in the densities of coexisting hosts are at least partly compensatory, so that an increase in the density of a less competent host is accompanied by a decrease in the density of more competent hosts, resulting in reduced risk of Lyme disease.

We must emphasize, though, that it would be premature to use this model to predict Lyme disease dynamics in particular locations, because field estimates of host-finding parameters and host densities are not readily available.

Our model embodies a fairly simplified representation of the life cycle of *I. scapularis,* and ignores some factors known to affect disease dynamics in this and other similar systems (e.g., spatial structure, age or stage structure, temperature variation; Dwyer 1991, Adler et al. 1992, Carruthers et al. 1992). However, its ability to generate biologically interpretable results makes us confident that the model captures certain essential features of the dynamics of *Borrelia* in ticks. Furthermore, the model's simplicity improves the ability of field workers to estimate its parameters. All of the parameters in this model can be estimated directly except for the coefficients of the exponential curves relating host-finding to host density (η in Eq. 6 and δ in Eq. 7) and host infection rate to tick density (ϵ in Eq. 8); these parameters must be estimated by regression using field data, or by experimentation. Virtually any other function that describes density dependence in the system will share this same problem of parameter estimation. Experiments that measure the density-dependent responses of ticks to hosts would be particularly useful for efforts to apply this and other models to particular situations (Porco 1991, Sandberg et al. 1992).

Implications for managing Lyme disease

Our results are important for guiding ecologically based efforts to control Lyme disease. In general, the model indicates that it will be easier to lower disease risk by altering the proportion of ticks infected by *Borrelia* than by reducing the absolute population density of ticks through host management. White-tailed deer would be the most feasible host species to manage because of its large body size and the public acceptance of and participation in hunting, but our model demonstrates that the population density of ticks will respond only weakly to manipulation of density of hosts for adults. The densities of the small and medium-sized mammals and birds that act as hosts for juveniles are more difficult to manipulate, but the effects of such manipulation on tick population densities could be substantial. Under most conditions, the model predicts that reduction in the availability of hosts for juveniles would be accompanied by a corresponding reduction in tick density, whereas this is not the case for hosts for adults.

The results suggest that manipulating the species composition of small vertebrates may prove effective for controlling Lyme disease. Disease risk is a function both of the density of ticks and the proportion of those ticks that carry spirochetes. The infection rate of ticks is extremely high when many hosts having high reservoir competence are available for juvenile ticks, as occurs in parts of southern New England and the mid-Atlantic region where the white-footed mouse is the dominant small mammal (Fish 1993, Ginsberg 1993). In our model, infection rates declined when the community of hosts for juveniles included a mixture of competent and less competent species. This finding implies that the incidence of Lyme disease could be reduced, without necessarily affecting the density of

ticks, by encouraging diverse communities of small mammals and birds. Species diversity of small vertebrates tends to be positively related to local and regional habitat diversity (Rosenzweig et al. 1975, Hafner 1977, Anthony et al. 1981), suggesting that ecosystem management for habitat diversity might prove useful for managing Lyme disease.

So far, the most effective host-management strategies are those that locally alter habitats near areas that receive high human use to make them less suitable for tick hosts (Wilson et al. 1988*a*, Wilson and Deblinger 1993). Our results indicate that other measures, which focus on maintaining habitats that support a diverse community of small vertebrates, may also help regulate the incidence of Lyme disease. Therefore, a reduction in Lyme disease incidence may constitute a tangible benefit of increasing biological diversity.

ACKNOWLEDGMENTS

Thanks to Kirsten Hazler for comments on the manuscript. The work was supported by grants from the Mary Flagler Cary Charitable Trust, the David Goodstein Family Foundation, and the General Reinsurance Corporation to the Institute of Ecosystem Studies.

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