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# MODELING THE EFFECTS OF RESERVOIR COMPETENCE DECAY AND DEMOGRAPHIC TURNOVER IN LYME DISEASE ECOLOGY

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*Abstract.* Lyme disease risk is related to the abundance of infected nymphal ticks, which in turn depends on the abundance and reservoir competence of wild hosts. Reservoir competence of a host (i.e., probability that an infected host will infect a feeding vector) often declines over time after inoculation, and small mammalian reservoirs typically undergo rapid population growth during the period when vector ticks feed. These processes can affect disease risk in the context of site-specific tick abundance and host community composition. We modeled the effects of reservoir decay and host demographic turnover on Lyme disease risk using a simple yearly difference equation model and a more realistic simulation incorporating seasonal dynamics of ticks and hosts. Both reservoir decay and demographic turnover caused (1) specific infectivity (proportion infected × reservoir competence) of host populations to vary with host community composition, (2) tick infection prevalence and the specific infectivity of reservoirs to be highly sensitive to the abundance of questing nymphs, and (3) specific infectivity and the infection prevalence of ticks to decrease at high host densities. Reservoir competence decay had similar effects in both model formulations, but host turnover had less effect than reservoir decay in the seasonal model. In general, exponential reservoir decay and abrupt loss of reservoir competence had similar effects, although exponential decay caused greater sensitivity to tick density and host community composition. Reservoir decay may explain the observed variability in published field measurements of reservoir competence of a host species. Our results illuminate mechanisms by which host diversity can dilute the impact of a highly competent reservoir and suggest that management to reduce nymphal tick abundance may reap an added benefit by reducing nymphal infection prevalence.

*Key words: black-legged ticks;* Borrelia burgdorferi; *community composition; demographic turnover; dilution effect; epidemiological models; host–parasite interaction;* Ixodes ricinus *complex; Lyme disease; mathematical models; reservoir competence; vector-borne disease.*

## INTRODUCTION

Most human cases of Lyme disease are the result of ecological interactions among at least five species, including the human. The pathogen, tick vector, and two or more wild host species exist in a self-sustaining cycle involving reciprocal transmission between vectors and hosts. The ecological basis of Lyme disease has raised awareness that human health is enmeshed within the processes occurring among species that make up ecological communities. The complexity of Lyme disease epizootiology has stimulated the development of several mathematical models to extract insights into the relationship between tick populations, the host community, and the risk of Lyme disease for humans. All models are simplifications of reality, and simplifying assumptions differ among published models of Lyme disease ecology. Our objective in this paper is to call attention to two processes: decay of reservoir competence (i.e., the probability that an infected host will infect a feeding vector) and host demographic turnover. These processes have been left out of some Lyme disease models and partially incorporated in others. We wish to assess whether reservoir decay and host turnover are nonnegligible components of the natural history of Lyme disease, and have a significant impact on the relationships between tick abundance, host community composition, and Lyme disease risk, with implications for predicting the effects of management actions.

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### *Natural history of Lyme disease*

Lyme disease is the most prevalent vector-borne disease of humans in the United States, Europe, and parts of Asia. Lyme disease is caused by the spirochete *Borrelia burgdorferi* (Bosler et al. 1982) vectored by ixodid ticks (Acari: Ixodidae) of the *Ixodes ricinus* complex (IRC), and the primary ecological risk factor is the abundance of nymphal ticks infected with *B. burgdorferi* (Falco and Fish 1989, Fish 1993, Gray 1998). The most important vector species for human health are *I. ricinus* in northern Europe and North Africa, *I. persulcatus* in eastern Europe and Asia, *I. scapularis*

in eastern North America, and *I. pacificus* in western North America (Gray 1998). *Ixodes ricinus* complex ticks have three mobile life stages: larva, nymph, and adult. Except for adult males, each life stage takes one blood meal before metamorphosis to the next stage or laying eggs and dying (Gray 1998). Immature IRC ticks tend to feed on different species of hosts than adults, and direct transovarial transmission from a female tick to her offspring is very inefficient (Lane et al. 1991, Schoeler and Lane 1993, Patrican 1997*a*). Therefore, adult ticks determine the abundance of immature ticks, but may have little direct effect on the infection prevalence of immature stages (but see Randolph and Craine [1995]).

The enzootic cycle of *B. burgdorferi* is perpetuated by reciprocal transmission between immature IRC ticks and their vertebrate hosts. Immatures feed on various small and medium-sized mammals, birds, and lizards (Lane et al. 1991, Matuschka et al. 1991). Nymphs, some of which were infected with *B. burgdorferi* by their larval blood meal, typically feed one or more months before larvae (Schultze et al. 1986, Fish 1993, Craine et al. 1995). In doing so, nymphs infect the hosts that infect the next generation of larvae. Few hosts of immature IRC ticks survive or retain infection between years (Schug et al. 1991; Randolph and Craine [1995] and references cited therein), so the emergence of nymphs before larvae appears to be critical for perpetuating the enzootic cycle (Spielman et al. 1985, Fish 1993). In a sense, both ticks and hosts act as reservoirs for each other, with ticks sustaining the enzootic during the winter and hosts enabling transmission between tick generations (Talleklint and Jaenson 1995, Lindsay et al. 1997). Host species may vary considerably in the number of ticks each individual feeds as well as their reservoir competence for *B. burgdorferi* (Mather et al. 1989). The efficiency of transmission between tick generations is closely related to the degree to which nymphs and larvae feed on the same competent reservoirs. The relative abundances, tick burdens, and reservoir competences of various species that form the host community are, consequently, important factors influencing the abundance of infected ticks and risk of Lyme disease for humans (Matuschka et al. 1992, Ostfeld and Keesing 2000*a*, *b*, Schmidt and Ostfeld 2001).

In the northeastern United States, where Lyme disease afflicts >10 000 people yearly (Anonymous 2000), white-footed mice (*Peromyscus leucopus*) and eastern chipmunks (*Tamias striatus*) are the primary hosts for immature black-legged ticks (*I. scapularis*) and highly competent reservoirs of *B. burgdorferi* (Anderson et al. 1983, Donahue et al. 1987, Mather et al. 1989, Mannelli et al. 1993, Slajchert et al. 1997, Schmidt et al. 1999, Schmidt and Ostfeld 2001). The abundance of these mammals may substantially affect the abundance of infected nymphs and, consequently, risk to humans (Ostfeld 1997, Ostfeld et al. 2001). However, immature black-legged ticks feed on many other species of terrestrial vertebrates (Piesman and Spielman 1979) that differ in their tick burdens and competence as Lyme disease reservoirs (Mather et al. 1989, Giardina et al. 2000). Alternative hosts also vary in abundance among locations, and populations of white-footed mice and chipmunks vary greatly from year to year in response to fluctuating acorn production (Elkinton et al. 1996, Ostfeld et al. 1996*b*, Wolff 1996, Jones et al. 1998*a*). As a result, the prevalence of competent reservoirs in the host community varies among years and sites (Ostfeld 1997).

### *Models of Lyme disease ecology*

Controversy surrounds the presumption that host community composition can have strong and predictable effects on human Lyme disease risk (Jones et al. 1998*b*, Randolph 1998), and manipulative experiments of appropriately large scale (Ostfeld et al. 1996*b*) have not been done. Several researchers have used mathematical models to explore the potential to alter the risk of Lyme disease in the northeastern USA by reducing tick abundance or manipulating host community composition (Porco 1991, 1999, Van Buskirk and Ostfeld 1995, 1998, Mount et al. 1997*a*, *b*). Such models are difference equation models, but continuous-time analytical models (Caraco et al. 1998) and individual based simulations have also been applied (Deelman et al. 1995).

### *Reservoir decay and host turnover*

Like all models, these have simplifying assumptions. Caraco et al. (1998), Mount et al. (1997*a*, *b*), and Van Buskirk and Ostfeld (1995, 1998) assumed that reservoir competence is a species-specific constant. However, reservoir competence typically declines over time after infection (Donahue et al. 1987, Nakao and Miyamoto 1993, Levin et al. 1995, Shih et al. 1995, Lindsay et al. 1997, Markowski et al. 1998, Richter et al. 2000). We will refer to this phenomenon as *reservoir decay.* Some standard epidemiological models partially account for reservoir decay. For example, susceptible–infected–recovered (SIR; e.g., Porco 1991, 1999) and susceptible–infected–susceptible (SIS) epidemiological models allow for host recovery with complete loss of infectivity, so they incorporate an abrupt loss of reservoir competence. However, transmission rates in SIR and SIS models are still assumed to be constant throughout the infective period, and these models imply that the initial slope of the reservoir decay curve is zero. In addition, some models include the assumption that the host population is demographically stationary (no births, deaths, or migration) during the period when immature ticks are active (Van Buskirk and Ostfeld 1995, 1998). This assumption is possible because infection with *B. burgdorferi* apparently has little effect on the survival or reproduction of wild hosts (Hofmeister et al. 1999). However, populations of rodents and birds typically grow rapidly during spring

and summer, resulting in influx of uninfected host recruits while some infected hosts die. This demographic replacement and dilution of infected hosts by uninfected recruits we term *host turnover*.

Reservoir decay and host turnover can potentially have similar effects on the *specific infectivity* of hosts, defined as the proportion of infected host individuals multiplied by the mean reservoir competence of infected hosts (Mather et al. 1989). From the viewpoint of an individual vector, specific infectivity indicates the overall probability the vector will become infected by biting a host individual selected at random from the population. Both reservoir decay and host turnover cause specific infectivity to decline over time after hosts cease to be bitten by infected ticks (e.g., due to late-summer decline in the abundance of feeding nymphs). In the case of reservoir decay, the decline in specific infectivity would be due to a decline in the actual competence of infected hosts. In the case of host turnover, death and recruitment of hosts would cause a decline in infection prevalence of hosts, even if reservoir competence remained constant.

We expected that reservoir decay or host turnover might have other, less obvious, effects on Lyme epizootiology. For example, both mechanisms could enhance positive feedback in *B. burgdorferi* transmission: increasing prevalence of infected nymphs would increase the frequency at which hosts are reinfected, keeping hosts in a state of high specific infectivity with a greater probability of infecting the next generation of ticks. Could this mechanism establish a threshold infection prevalence, below which the enzootic would diminish? In addition, reservoir decay could cause mean reservoir competence for a particular host species to vary over time and space, depending on the abundance of ticks and other host species. How do reservoir decay and host turnover affect the relationship between host community composition or tick density and prevalence of infected ticks? What is the shape of this relationship and how can it inform strategies for ecological management of Lyme disease risk?

We explored the potential effects of reservoir decay and host turnover in mathematical models of the dynamics of Lyme disease. We employed a simple yearly model, as well as a more detailed simulation that explicitly incorporates seasonal dynamics of rodent and tick populations. Our objectives were to examine how reservoir decay and host turnover affect the relationships between abundance of ticks, composition of the host community, reservoir competence, and prevalence of *B. burgdorferi* infection.

## Methods

Our models are based on the ecology of Lyme disease in the northeastern United States, where only one vector species and one genospecies of *B. burgdorferi* appear to be involved. The ecology of Lyme disease in this region is further simplified by the apparent dominance of white footed mice and eastern chipmunks as hosts for larvae and reservoirs for spirochetes (Mather et al. 1989, Schmidt et al. 1999, Giardina et al. 2000). However, we caution that no study has fully characterized the host community at any one site in terms of the abundance, tick burden, tick feeding success rate, and reservoir competence of every host species. In addition, we caution that the epizootiology of *B. burgdorferi* is considerably different in the southern and western United States, where host community composition, tick species, and seasonal patterns may differ substantially from the northeast (Lane and Loye 1991, Oliver 1996). Therefore, our model may poorly match the ecology of disease transmission in other geographic areas. We used a hierarchical modeling approach, starting with a nonseasonal, one-year time step model (''yearly model''), based on the models of Porco (1991) and Van Buskirk and Ostfeld (1995). Then we built a model that accounted for seasonal host and tick dynamics with a one-week time step (''seasonal model''), similar to the model of Porco (1999). Our rationale for applying two different model structures was to determine whether the patterns that emerge from the yearly model are robust to realistic seasonal dynamics of tick and host populations. Both models were run for a single host species and also for two hosts with different reservoir competences or turnover rates. In the yearly model, host turnover and one type of reservoir decay emerge as mathematically equivalent processes, so we report results for both processes together. In the two-host case, we patterned one host (host A) after the white footed mouse, a highly competent reservoir. The other host (host B) was considered to be a less competent reservoir.

### *Some important assumptions*

A model does nothing but expose the consequences of the assumptions upon which it is based. Although we sought to incorporate realism into our models, some simplifying assumptions were still required (Table 1). Some of these assumptions are supported by empirical data, whereas the validity of others is controversial. Some assumptions are unlikely to affect the qualitative results of the model, whereas others could greatly change the results. Here, we discuss the justification for some of the more critical assumptions.

We assumed that the abundance of adult ticks, and consequently of tick eggs, is unrelated to the abundance of hosts for immature ticks. Therefore, the number of new larvae (yearly model) or nymphs (seasonal model) entering the model each year was kept constant. Moreover, we kept the abundance of each host species constant among years. These assumptions are contradicted by evidence that acorn production positively affects abundance of both larval black-legged ticks and white footed mice in oak forests (Jones et al. 1998*a*), and that abundance of nymphal black-legged ticks is positively correlated with abundance of white-footed mice

TABLE 1. Some important assumptions inherent in our models of Lyme disease ecology.

| Assumption |
|---|
| Both models |
| 1) Each tick bites at most one host during each life stage. |
| 2) Tick bites are distributed among hosts as a Poisson random process. |
| 3) Feeding success of ticks and transmission of *B. burgdorferi* are both independent of the number of ticks attached to an individual host. |
| 4) Adult ticks share no hosts with immature ticks. |
| 5) Ticks that do not feed in a given year do not survive to the next year. |
| 6) Infection with *B. burgdorferi* does not affect survival, reproduction, activity, or immunity of ticks or hosts. |
| 7) *B. burgdorferi* is only transmitted by ticks biting hosts (no vertical or horizontal transmission in ticks or hosts). |
| 8) Infected larval ticks remain infected and infective through the nymphal stage. |
| 9) Infected hosts do not survive or retain infection between years. |
| 10) Reservoir competence returns to the same value each time a host is infected with no immunity or prepatent period. |
| Yearly model |
| 1) Abundances of ticks and hosts are both constant over time. |
| 2) Abundance of immature ticks is independent of the abundance of hosts. |
| 3) The period when larval ticks feed does not overlap with the period when nymphs feed. |
| 4) Hosts die and give birth at equal and constant rates during the nymphal feeding period (setting birth and death rates to zero means no host turnover). |
| Seasonal model |
| 1) Abundances of tick eggs and hosts are both constant among years, but abundances of immature ticks and hosts vary among weeks within a year. |
| 2) Abundance of tick eggs is independent of the abundance of hosts, but abundance of active immature ticks in any given week is affected by the abundance of hosts. |
| 3) Only actively questing ticks encounter and attach to hosts. |
| 4) Survival and encounter rates of each tick life stage are constant over time within a year. |
| 5) Dormant ticks have higher survival rates than engorged or actively questing ticks. |
| 6) Engorged larvae molt into dormant nymphs after four weeks. |
| 7) The proportion of ticks in each life stage that are active increases over the course of each year as a logit-linear function of time, with different functions for larvae and nymphs. |
| 8) Death and birth rates of hosts vary among weeks within a year according to trigonometric functions (setting birth and death rates to zero means no host turnover). |

the previous year (Ostfeld et al. 2001). We expect that such covariance between mouse and tick densities would increase the sensitivity of Lyme disease risk to mouse density, but we leave explicit exploration of these assumptions outside the scope of this paper.

We also assumed that ticks do not transmit *B. burgdorferi* transovarially and that adult and immature ticks feed on different host species. Consequently, we did not explicitly include the adult stage in our models. We assumed that the distribution of tick bites among hosts is random and therefore described by a Poisson process. In reality, tick bites are likely to be clustered (Adler et al. 1992), with some unlucky host individuals receiving many more bites than others. If larval and nymphal bites are statistically independent, then such clustering would tend to reduce tick and host infection prevalence (Porco 1991, 1999). However, positive covariance of larval and nymphal bites would tend to increase infection prevalence, because larval bites would be clustered on the host individuals most likely to be infected and infective (Craine et al. 1995).

We assumed no density dependence in tick feeding success or transmission rate. There is mixed evidence about whether the number of feeding juvenile blacklegged ticks on a host affects their feeding success rate (Hazler and Ostfeld 1995, Levin and Fish 1998). A growing body of evidence indicates that the per-tick rate of transmission of *B. burgdorferi* from host to ticks increases with increasing numbers of ticks feeding on each host (Levin et al. 1997, Ogden et al. 1998), which is a phenomenon well worth exploring in future modeling exercises.

Finally, we assumed that reservoir decay is completely reversible, i.e., reservoir competence returns to the same value after each reinfection. Richter et al. (2000) found that competence of American Robins (*Turdus migratorius*) was similar after reinfection with *B. burgdorferi* as after the birds were initially infected. Otherwise, few data are available to indicate whether or not this assumption is valid.

### *Yearly model*

*Overview*.—Constants, variables, and parameters used in the yearly model are listed in Appendix A. For the yearly model, we kept densities of all host species and all tick life stages constant for each model run. As a consequence, the only states that varied among years were the proportions of hosts and nymphs that are infected and, if reservoir decay was included, the reservoir competence of hosts. We modified the framework of Van Buskirk and Ostfeld (1995) to account for the fact that each tick bites only one host during each juvenile life stage. Therefore, the number of tick bites was limited, and we determined the proportion of hosts

bitten by distributing tick bites among hosts as a Poisson process (Goldfarb 1986, Porco 1991). We assumed that hosts do not survive or retain infections between years, so each yearly cohort of hosts was initially uninfected. The infection prevalence of each host species at the end of the period of nymphal activity was determined by the risk of being bitten by an infected nymph and the probability of transmission from nymph to host. The specific infectivity of each host species was calculated by multiplying infection prevalence by reservoir competence. The specific infectivity of all host species at the end of nymphal activity determined the proportion of feeding larvae that became infected and, consequently, the infection prevalence of nymphs during the next year.

*Nymph feeding and* B. burgdorferi *transmission*.—During the nymphal activity period, the proportion of nymphs that have not fed, $P_u$, is expected to decrease over time, $x$, at a rate determined by host abundance ($H_i$) and the host-specific probability of encountering and successfully feeding on individual hosts ($\alpha_{Ni}$), summed over all host species:

$$\frac{dP_U}{dx} = -P_U\left(\sum_i \alpha_{Ni}H_i\right).$$

For simplicity, we set the length of the nymphal activity period to a value of one, so the rates $\alpha_{Ni}$ (as well as other rate parameters in the yearly model) are scaled to the duration of nymphal activity. Integrating over the nymphal activity period ($0 \le x \le 1$) and subtracting from unity yields the proportion of nymphs that successfully feed during the nymphal activity period ($P_N$), given by

$$P_N = 1 - \exp\left(-\sum_i \alpha_{Ni}H_i\right).$$

Here, $\alpha_{Ni}$ incorporates the rate of encounter between host and nymphal ticks and the probability of a nymph successfully attaching to and feeding on a host it encounters. The proportion of nymphs that acquire meals from host species $i$ ($P_{Ni}$) is given by

$$P_{Ni} = P_N\frac{\alpha_{Ni}H_i}{\sum_i \alpha_{Ni}H_i}.$$

The quantities that vary among years are the proportion of host species $i$ infected with *B. burgdorferi* by feeding nymphs in year $t$ ($\gamma_{it}$), the specific infectivity of host species $i$ ($S_{it}$), and the infection prevalence of nymphs ($\pi_t$). These three variables are interdependent: $\pi_t$ influences $\gamma_{it}$, $\gamma_{it}$ in part determines $S_{it}$, and $S_{it}$ averaged across all host species determines $\pi_{t+1}$. The instantaneous risk of a host of species $i$ receiving an infective bite from a nymph in year $t$ is given by

$$\lambda_{it} = \frac{NP_{Ni}\pi_t\tau_{Ni}}{H_i}$$

where $N$ is the density of nymphs, $\pi_t$ is the proportion of infected nymphal ticks in year $t$, and $\tau_{Ni}$ is the constant probability that a bite from an infected nymph will infect a host of species $i$. Some hosts will receive multiple infective bites, whereas others will escape infection altogether. We assume that all hosts have the same instantaneous risk of receiving an infective bite ($\lambda_{it}$). Therefore, we treat the number of infective bites per host as a Poisson random variable with parameter $\lambda_{it}$, and only those hosts receiving no infective bites remain uninfected. Thus, the overall host infection prevalence after nymphs have fed is given by unity minus the Poisson probability of escaping infection (receiving zero infection events):

$$\gamma_{it} = 1 - \exp(-\lambda_{it}).$$

If reservoir competence is constant, the specific infectivity of host $i$ ($S_{it}$) is obtained by multiplying the host infection prevalence ($\gamma_{it}$) by the reservoir competence of infected hosts ($\tau_{Li}$). The proportion of infected nymphs in the next generation ($\pi_{t+1}$) is given by the average specific infectivity among hosts, weighted by the proportion of total larval meals each host provides:

$$\pi_{t+1} = \frac{S_{1t}\alpha_{L1}H_1}{\sum_i \alpha_{Li}H_i} + \frac{S_{2t}\alpha_{L2}H_2}{\sum_i \alpha_{Li}H_i} + \cdots + \frac{S_{nt}\alpha_{Ln}H_n}{\sum_i \alpha_{Li}H_i} = \frac{\sum_i S_{it}\alpha_{Li}H_i}{\sum_i \alpha_{Li}H_i}. \quad (1)$$

*Reservoir decay*.—In general, reservoir decay enters into the yearly difference equation framework by affecting the specific infectivity of hosts at the end of the nymphal activity period as follows:

$$S_{it} = \int_{x=0}^{1} \tau_{Li}[x]\Pr[x]\,dx \quad (2)$$

where $\tau_{Li}[x]$ is the reservoir competence of a host at time $x$ since it was last infected, $\Pr[x]$ is a probability density function indicating the relative probability of that a host individual will experience an interval of $x$ between infections, and the total length of the nymphal activity period is set to a value of one. The function $\Pr[x]$ depends on the abundance of infected nymphs, their attack rate, as well as their distribution among host individuals; whereas $\tau_{Li}[x]$ is presumably a characteristic of the particular host, vector, and pathogen species, and may depend on modulation of the host's immune response by the vector.

Eq. 2 does not stipulate any particular shape for the reservoir decay curve, $\tau_{Li}[x]$. Intuitively, an important characteristic of the reservoir decay curve is the initial slope. If $\tau_{Li}[x]$ decreases rapidly immediately after infection (small $x$), then a host individual will only remain at a high competence level if it is reinfected very frequently. However, if the $\tau_{Li}[x]$ curve is relatively

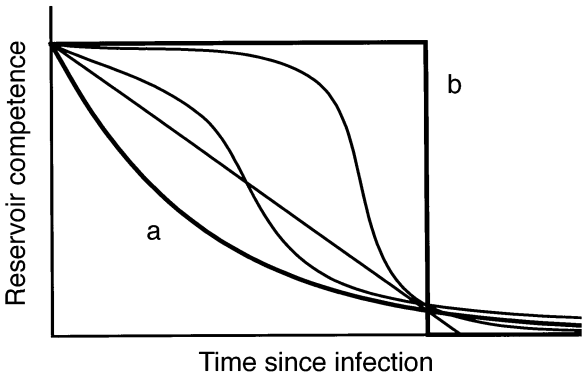


FIG. 1. Hypothetical reservoir competence decay curves: (a) exponential decay and (b) abrupt loss of competence represent extremes on a continuum of curve shapes. The effects of intermediate curve shapes on the behavior of Lyme disease models are likely to lie between the effects of curves a and b.

level for small $x$, then high competence can be maintained at a much lower frequency of reinfection. We chose to examine two specific formulations: exponential decay (which implies rapid initial decrease in competence) and abrupt loss (which implies constant competence for small $x$). These curves represent extremes on a continuum of shapes that encompasses an infinite family of more realistic curves (Fig. 1). We expect that more realistic decay curves are likely to either be similar to one or the other formulation, or to have effects on the behavior of Lyme disease models that are intermediate between the two. The respective formulations of $\tau_{Li}[x]$ are as follows:

$$\tau_{Li}[x] = \tau_{\max,i}\exp(-x\delta_i) \tag{3}$$

$$\tau_{Li}[x] = \begin{bmatrix} \tau_{\max,i} & x \le x_i \\ 0 & x > x_i \end{bmatrix}. \tag{4}$$

Eq. 3 describes an exponential decay curve, controlled by the decay rate, $\delta_i$ (scaled to the duration of the nymphal activity period). Eq. 4 indicates that reservoir competence remains at its maximum value until time $x_i$ has passed, and then drops abruptly to zero. Thus, Eq. 4 represents a susceptible–infected–susceptible (SIS) process in which infected individuals remain infective at a consistent level for a fixed time before becoming susceptible and uninfective again. If infective tick bites are distributed among hosts as a Poisson process, then the time between infections for a particular host has an exponential probability distribution, with the following probability density function:

$$\Pr[x]_{it} = \lambda_{it}\exp(-x\lambda_{it}). \tag{5}$$

Solving Eq. 2 for the cases described by combining Eq. 5 with either Eq. 3 or 4, respectively, yields the following expression:

$$S_{it} = \frac{\tau_{\max,i}\lambda_{it}}{\lambda_{it} + \delta_i}(1 - \exp(-\lambda_{it} - \delta_i)) \tag{6}$$

$$S_{it} = \tau_{\max,i}(1 - \exp(-x_i\lambda_{it})). \tag{7}$$

In both cases, $S_{it}$ is an increasing function of the risk of a host being infected ($\lambda_{it}$), and has an asymptote at $\tau_{\max,i}$. Plugging $S_{it}$ into Eq. 1 yields the infection prevalence of the next generation of nymphs.

*Host demographic turnover*.—To incorporate host demographic turnover, we assumed that birth and death rates are equal and constant over time, as well as being unaffected by infection with *B. burgdorferi*. These are obviously unrealistic assumptions, because host populations typically increase during the period when ticks feed, and vital rates and infection prevalence are necessarily age dependent. However, we feel that this is a reasonable starting point for exploring the fundamental effect of host turnover, which may be extended to an age- or stage-structured formulation (our seasonal model allows birth and death rates to differ and vary over time; see *Methods: Seasonal model*). We also assumed hosts are born uninfected. The rate of change in the infection prevalence of host species $i$ over time ($x$) during the period of tick feeding is given by

$$\frac{d\gamma[x]_{it}}{dx} = (1 - \gamma[x]_{it})\lambda_{it} - \rho_i\gamma[x]_{it} \tag{8}$$

where $\rho_i$ is the instantaneous rate of turnover ($\rho_i$ = births = deaths) in the host population, scaled to the period of nymphal activity. Turnover is applied only to infected hosts, because replacement of uninfected hosts by uninfected recruits has no net effect on the infection prevalence. Integrating Eq. 8 over the period of nymphal activity ($0 \le x \le 1$) yields the host infection prevalence at the end of nymphal activity ($\gamma_{it}$). Multiplying $\gamma_{it}$ by $\tau_{Li}$ yields the specific infectivity:

$$S_{it} = \frac{\tau_{Li}\lambda_{it}}{\lambda_{it} + \rho_i}(1 - \exp[-\lambda_{it} - \rho_i]). \tag{9}$$

Eq. 9 is exactly equivalent to Eq. 6 replacing $\tau_{\max,i}$ by $\tau_{Li}$ and $\delta_i$ by $\rho_i$. These equivalent formulations of host turnover and exponential reservoir decay are also exactly equivalent to an SIS formulation in which infected hosts recover at a constant proportional rate ($\rho_i$ in Eq. 8). Because we have treated transmission between nymphs and hosts as a continuous (rather than instantaneous) process, our formulations are not equivalent to a susceptible–infected–recovered (SIR) formulation. With continuous transmission, an SIR formulation would create a growing class of immune hosts that is not found in our model. In the Discussion, we briefly review evidence that wild reservoir hosts do not develop effective immunity to *B. burgdorferi*, so an SIR formulation probably is inappropriate.

*Running the yearly model*.—We set encounter rates at $\alpha_{Li} = \alpha_{Ni} = 0.05$ (host)$^{-1}$ and nymph-to-host transmission at $\tau_{Ni} = 0.9$ for both host species. Donahue et

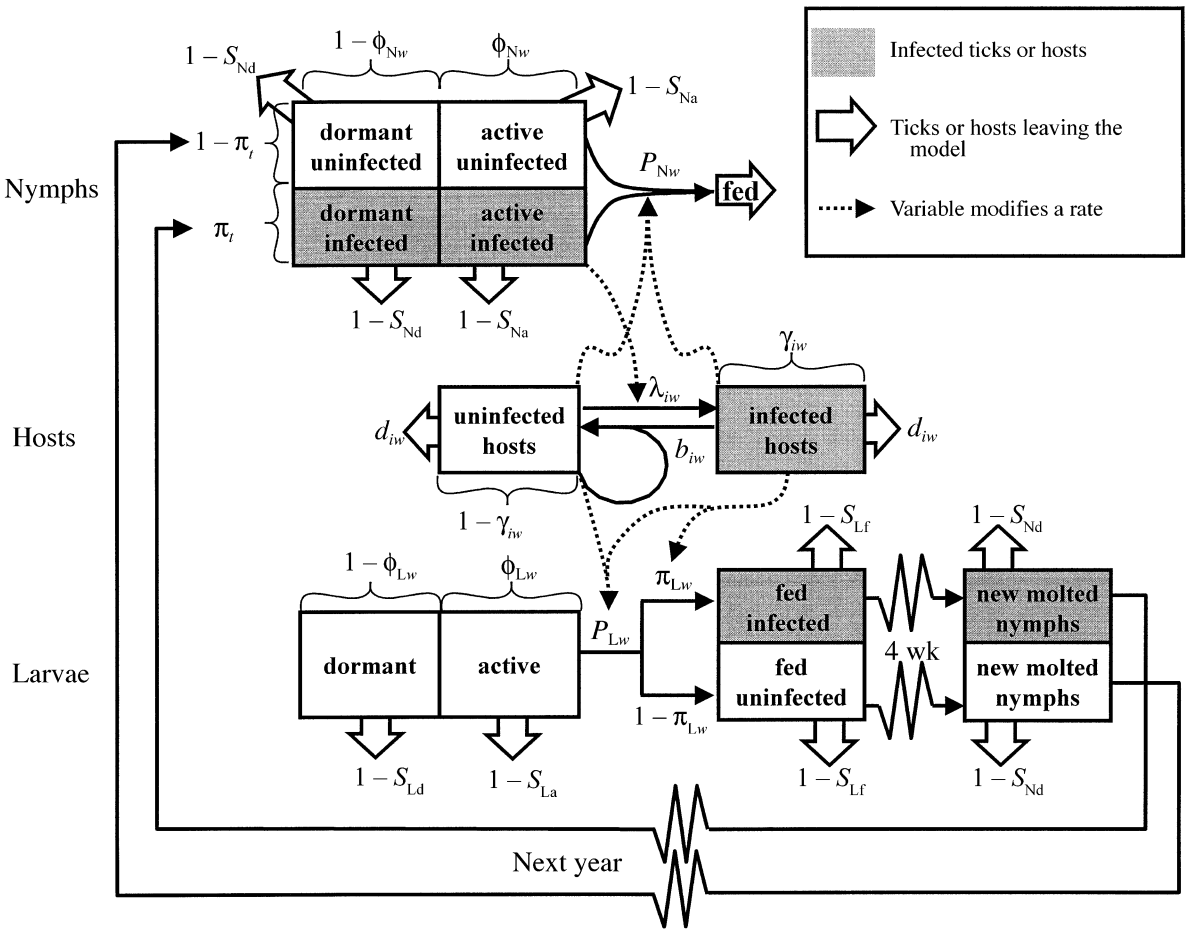


FIG. 2. Schematic representation of processes involved in the seasonal model. Infection with *B. burgdorferi* is passed from nymphal ticks to hosts, and then from hosts to larval ticks. Each yearly cohort of larvae becomes the next year's cohort of nymphs. Solid arrows indicate state transitions, dashed arrows indicate tick–host interactions (feeding and *B. burgdorferi* transmission) that modify state transition rates, and open block arrows indicate ticks or hosts leaving the model (e.g., by dying).

al. (1987) demonstrated that the bite of a single infected nymph has a high probability of infecting a white-footed mouse, so $\tau_{Ni} = 0.9$ is not unreasonable. In this formulation, the absolute value of $\alpha_{Li}$ has no effect (as long as it is between zero and one), although any difference between host species could affect transmission. For runs without reservoir decay or host turnover, we set the competence of host A ($\tau_{LA}$) to 0.9 and determined equilibrium states for several values of $\tau_{LB}$. For all parameter values we used, the simulation converged monotonically to the equilibrium. For runs with reservoir decay, we set $\tau_{max,i} = 0.9$ for both host species. With exponential decay, we set the decay rate for host A ($\delta_A$) to a value of one and determined equilibria for $\delta_B = 1$, 5, and 10. If exponential decay is interpreted as a survival function, then the expected survival time is given by the reciprocal of the decay rate ($\delta^{-1}$). Therefore, to facilitate comparison of exponential and abrupt reservoir decay formulations, we examined values of the duration of infectivity ($x_i$) in the abrupt-loss formulation that are reciprocals of $\delta_i$. We set $x_A = 1$, and determined equilibria for $x_B = 1$, 0.2, and 0.1. We ran the model under conditions of low and high nymphal abundance ($N = 500$ and 5000 nymphs/ha, respectively).

### *Seasonal model*

*Overview.*—Constants, variables, and parameters used in the seasonal model are listed in Appendix B, and Fig. 2 gives a pictorial description of processes and variables involved in the model. In the seasonal model, we used a one-week time step in calculating changes in abundance and infection prevalence of hosts, larvae, and nymphs. The abundance of hosts and ticks varied among weeks within a year, as did the proportion of ticks actively seeking hosts. The initial (week 0) densities of hosts and dormant larvae (eggs) were held constant among years. Over the course of each year, populations of hosts waxed and waned in response to seasonally varying birth and

death rates, resulting in demographic turnover. A proportion of ticks died during each week, and some of the active survivors successfully fed on hosts. Unlike the yearly model, host abundance affected the abundance of questing ticks and the total abundance of nymphs in the seasonal model. High abundance of hosts led to increased success of ticks in finding hosts early in each year and, consequently, to lower abundance of questing ticks later in the year. Also, a high abundance of hosts in a year led to greater nymphal densities the next year, because only those larval ticks that found hosts survived. Early in the summer, more nymphs than larvae were active, and nymphs infected some of the hosts they bit. Later in the summer, high densities of larvae became active, and some larvae that fed on infected hosts became infected themselves. The proportion of feeding larvae that became infected depended on the infection prevalence and reservoir competence of hosts, i.e., the specific infectivity. Fed larvae turned into newly molted nymphs after four weeks, and molted nymphs remained dormant until the next year. Infected larvae remained infected after metamorphosis. At the end of each year, the infection prevalence and density of molted nymphs carried over as the infection prevalence and initial density of nymphs. After nymphs fed, they were no longer included in the model.

*Host seasonal dynamics*.—In natural settings, availability of hosts for juvenile ticks varies substantially across seasons. For example, small mammal densities are typically low in the early spring, but breeding during the growing season causes population growth. In general, host population dynamics within a year can be modeled on the basis of week-specific instantaneous per capita population growth rates ($r_{iw}$):

$$H_{iw} = H_{iw-1}\exp(r_{iw}) \tag{10}$$

where $w$ is the week of the year ($0 < w \leq 52$). The growth rate incorporates week-specific birth and death rates ($r_{iw} = b_{iw} - d_{iw}$). Porco (1999) used a similar formulation (with a monthly time step) in his model, and noted that the host abundance will not change among years if the product of the realized growth rates over the course of a year equals unity:

$$\prod_i \exp(r_{iw}) = \exp\left(\sum_i r_{iw}\right) = 1$$

which implies

$$\sum_i r_{iw} = 0. \tag{11}$$

To model seasonal abundance of hosts, we made weekly per capita death and birth rates trigonometric functions of time (Turchin and Hanski 1997, Turchin and Ostfeld 1997). In the model, weekly per capita death rate of host species $i$ peaks at the beginning and end of each year, based on the following function:

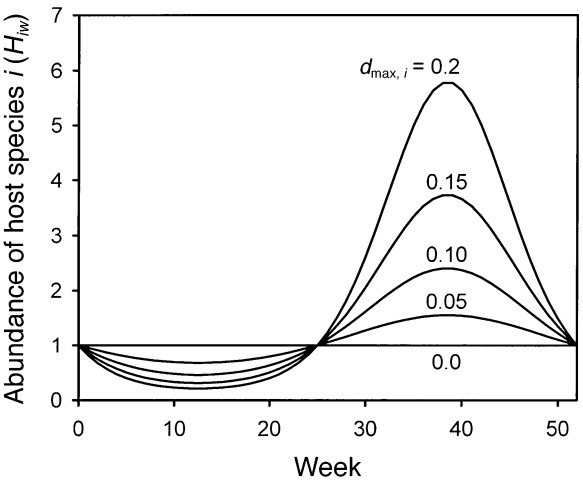


FIG. 3. Seasonal dynamics of a host population in the seasonal model, as generated by Eq. 6. Curves are for different values of maximum per capita death rate, $d_{\max,i}$.

$$d_{iw} = \frac{1}{2}\left(\cos\left[\frac{2\pi w}{52}\right](d_{\max,i} - d_{\min,i}) + d_{\max,i} + d_{\min,i}\right)$$

where $w$ is the week of the year and $d_{\max,i}$ and $d_{\min,i}$ are the maximum and minimum weekly per capita mortality rates, respectively. Weekly per capita birth rates ($b_{iw}$) approach zero during winter and peak at $b_{\max,i}$ during summer:

$$b_{iw} = \frac{b_{\max,i}}{2}\left(1 - \cos\left[\frac{2\pi w}{52}\right]\right).$$

We constrained host birth rates such that $b_{\max,i} = d_{\max,i} + d_{\min,i}$ to satisfy the condition embodied in Eq. 11 and ensure that host abundance does not vary among years, yielding

$$r_{iw} = b_{iw} - d_{iw} = -d_{\max,i}\cos\left[\frac{2\pi w}{52}\right]. \tag{12}$$

Thus, $d_{\max,i}$ determines the range of densities over which the host population fluctuates within a year and sets the rate of demographic turnover. Weekly abundance of host $i$ ($H_{iw}$) (Fig. 3) was calculated by combining Eqs. 10 and 12.

*Tick seasonal dynamics*.—We simulated seasonal changes in the abundance of dormant and questing ticks by modeling the transitions between states of activity (dormant, active, or fed), life stage (larva or nymph), and mortality (alive or dead). We assumed that actively questing and recently fed ticks suffer higher mortality than flat, dormant ticks. The dynamics of ticks seeking and finding hosts or dying was modeled separately for larvae and nymphs. In the model, each year starts with the same density of dormant larvae ($L_{d0}$) all of which are uninfected, whereas the initial abundance and infection prevalence of nymphs is determined by the abundance and infection prevalence of larvae that suc-

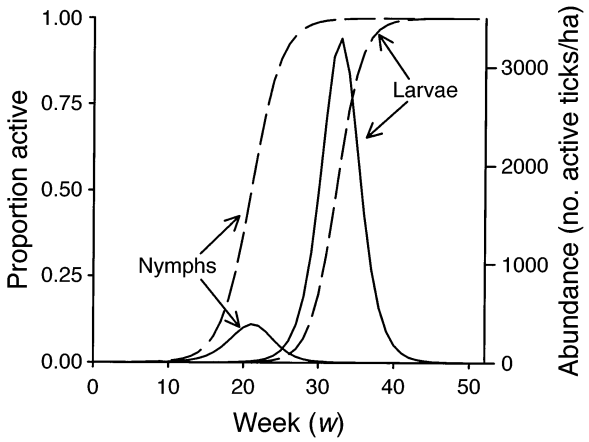


FIG. 4. Phenology of activity and abundance of larval and nymphal ticks in the seasonal model. Dashed lines represent the proportion of ticks of each life stage that are active (left-hand *y*-axis), and solid lines indicate the abundance of active ticks (right-hand *y*-axis).

cessfully fed the previous year. In each week, a proportion of ticks of stage $j$ ($j$ = L for larvae, N for nymphs) are active ($\phi_{jw}$) and the remainder ($1 - \phi_{jw}$) are dormant. Mount et al. (1997*a*) simulated the phenology of immature tick activity as a mechanistic function of ambient temperature. Instead, we mimicked the observed seasonal pattern of larval and nymphal host-seeking activity by making the proportion of active ticks in week $w$ a logit-linear function of $w$ (Fig. 4):

$$\text{logit}(\phi_{\text{L}w}) = -19 + 0.6w$$

$$\text{logit}(\phi_{\text{N}w}) = -11 + 0.5w.$$

Of the dormant, actively questing, and fed ticks of stage $j$, a constant proportion ($s_{\text{d}j}$, $s_{\text{a}j}$, and $s_{\text{f}j}$, respectively) survive to the next week. Based on these equations for larval and nymphal activity and Eq. 12, the peak in host population growth (week 26) occurs just as nymphal activity reaches 88% and nine weeks before larval activity reaches 88%. However, the timing of peaks in density of host-seeking larvae and nymphs vary depending on host abundance.

The proportion of actively questing survivors of life stage $j$ that successfully feed on hosts in week $w$ ($P_{jw}$) is given by

$$P_{jw} = 1 - \exp\left(-\sum_i \beta_{ji} H_{iw}\right)$$

where $\beta_{ji}$ is the instantaneous weekly probability of a tick of life stage $j$ encountering and successfully attaching to a host of species $i$ (analogous to $\alpha_{\text{N}i}$ in the yearly model). Thus, for life stage $j$ in week $w > 0$,

$$j_{\text{u}w} = j_{\text{d}w} + j_{\text{a}w}$$

$$j_{\text{d}w} = j_{\text{u}w-1} s_{\text{d}j}(1 - \phi_{jw})$$

$$j_{\text{a}w} = j_{\text{u}w-1} s_{\text{a}j} \phi_{jw}(1 - P_{jw})$$

where $j_{\text{u}w}$ is the density of unfed ticks in week $w$, $j_{\text{d}w}$ is the density of dormant ticks, and $j_{\text{a}w}$ is the density of active ticks. The resulting distribution of unfed, active tick abundance over time is hump shaped (Fig. 4). In the model, fed larvae survive at a weekly rate of ($s_{\text{Lf}}$) for a molting period of four weeks (Yuval and Spielman 1990), then become dormant nymphs that remain dormant until the next year. The density of fed larvae increases as active larvae feed on hosts, but is reduced by larvae metamorphosing into the nymphal stage:

$$L_{\text{f}w} = L_{\text{f}w-1} s_{\text{Lf}} + L_{\text{u}w-1} s_{\text{La}} \phi_{\text{L}w} P_{\text{L}w} - L_{\text{f}w-4}(s_{\text{Lf}})^4.$$

Meanwhile, the density of newly molted nymphs in week $w$ ($N_{\text{m}w}$) increases by metamorphosis of surviving larvae that fed in week $w - 4$:

$$N_{\text{m}w} = N_{\text{m}w-1} s_{\text{Nd}} + L_{\text{f}w-4}(s_{\text{Lf}})^4.$$

At the end of a year, the abundance of dormant molted nymphs ($N_{\text{m}52}$) becomes the initial density of dormant nymphs for the next year ($N_{\text{d}0}$). We assumed that all ticks that did not successfully feed by the end of a year died, so the abundance and encounter rate of hosts in a year affects the abundance of nymphs the next year.

B. burgdorferi *transmission*.—We calculated the probability of a host becoming infected or reinfected at least once during week $w$ ($\lambda_{iw}$) by distributing nymphal tick bites among hosts. The proportion of active nymphs that acquire meals from host species $i$ in week $w$ ($P_{\text{N}iw}$) is given by

$$P_{\text{N}iw} = \frac{P_{\text{N}w} \beta_{\text{N}i} H_{iw}}{\sum_i \beta_{\text{N}i} H_{iw}}$$

and $\lambda_{iw}$ is given by:

$$\lambda_{iw} = 1 - \exp\left(-\frac{N_{\text{a}w} P_{\text{N}iw} \pi_t \tau_{\text{N}i}}{H_{iw}}\right)$$

where $\pi_t$ is the infection prevalence of nymphs in year $t$ and $\tau_{\text{N}i}$ is the constant probability that the bite of an infected nymphs will infect a host of species $i$ (as for the yearly model). Each week, the infection prevalence of host species $i$ ($\gamma_{iw}$) is increased by infected nymphs biting uninfected hosts and reduced by births of uninfected hosts:

$$\gamma_{iw} = (\gamma_{iw-1} + (1 - \gamma_{iw-1})\lambda_{iw})\exp(-b_{iw}).$$

For constant reservoir competence, specific infectivity in week $w$ ($S_{iw}$) is simply the product of $\gamma_{iw}$ and $\tau_{\text{L}i}$. To incorporate reservoir decay, we determined for each week $w$ and species $i$ the density of hosts last infected $x = 0, 1, 2, \ldots, w$ weeks previously ($H(I_x)_{iw}$) using the recursion:

$$H(I_0)_{iw} = H_{iw-1}\lambda_{iw}(1 - d_{iw})$$

$$H(I_x)_{iw} = H(I_{x-1})_{iw-1}(1 - \lambda_{iw})(1 - d_{iw}) \qquad x > 0.$$

The recursion captures the fact that if a host alive during week $w$ was last infected $x$ weeks ago, then during

the previous week ($w - 1$) it had been last infected $x - 1$ weeks ago, and survived (probability $1 - d_{iw}$) and avoided infection (probability $1 - \lambda_{iw}$) during week $w$. Thus, $H(I_0)_{iw}$ is the density of newly infected or reinfected hosts in week $w$. $H(I_1)_{iw}$ is the density of hosts that were infected or reinfected in week $w - 1$ ($H(I_0)_{iw-1}$) and survived week $w$, but were not reinfected in week $w$. The quantity $H(I_2)_{iw}$ indicates hosts last infected in week $w - 2$ that survived and avoided infection in week $w - 1$ ($H(I_1)_{iw-1}$) and also survived and avoided infection in week $w$, and so on. Specific infectivity is given by the following:

$$S_{iw} = \frac{\sum_{x=0}^{w} H(I_x)_{iw}\tau_{Li}[x]}{H_{iw}}$$

where $\tau_{Li}[x]$ is calculated as in Eq. 3 for exponential decay with $\delta_i$ representing the weekly reservoir decay rate, or as in Eq. 4 for abrupt loss of reservoir competence after $x_i$ weeks. The proportion of successfully feeding larvae that become infected in week $w$ ($\pi_{Lw}$) is given by the weighted average specific infectivity ($S_{iw}$) across all host species:

$$\pi_{Lw} = \frac{\sum_i \beta_{Li} H_{iw} S_{iw}}{\sum_i \beta_{Li} H_{iw}}.$$

The infection prevalence of newly molted nymphs in week $w$ ($\pi_{mw}$) changes as weekly cohorts of fed larvae metamorphose:

$$\pi_{mw} = \frac{\pi_{mw-1} N_{mw-1} s_{Nd} + \pi_{Lw-4} L_{fw-4} (s_{lf})^4}{N_{mw}}.$$

Finally, the infection prevalence of molted nymphs at the end of each year $t$ ($\pi_{m52}$) becomes the nymphal infection prevalence of year $t + 1$ ($\pi_{t+1}$).

*Running the seasonal model.*—For runs with host turnover, we set the minimum death rate ($d_{min,i}$) for both hosts equal to 0.01 and maximum death rate of host A ($d_{max,A}$) to 0.05. We determined equilibrium states for $d_{max,B} = 0.05$, 0.1, and 0.2, because values >0.2 produced unrealistically large (>27-fold) intraannual fluctuations in host density. We set tick weekly survival rates to $s_{Ld} = 1$, $s_{Nd} = 0.99$, $s_{La} = s_{Na} = 0.7$ (Lord 1993), and $s_{Lf} = 0.8$, under the assumptions that dormant larvae are eggs, dormant nymphs minimize mortality by aestivating in moist microsites, active ticks are susceptible to predation or desiccation, and engorged larvae are susceptible to predation and pathogens. Weekly encounter rates were set to equal values for both host species and tick life stages ($\beta_{Li} = \beta_{Ni} = 0.01$ (host)$^{-1}$ for all $i$). These survival and encounter rates resulted in peak nymphal abundances ranging from ~1/15–1/5 of peak larval abundances at host densities of 10–100 hosts/ha, which is similar to the range of relative peak densities of the two life stages observed in the field (Randolph and Craine 1995, Ostfeld et al. 1996*a*). For runs with reservoir decay, we set $\tau_{max,i} = 0.9$ for both host species A and B. With exponential decay, we set the weekly decay rate for host A ($\delta_A$) to 0.1 and determined equilibria for $\delta_B = 0.1$, 0.3, and 0.5. With abrupt loss of reservoir competence, we set the loss time for host A ($x_A$) to 10 wk and determined equilibria for $x_B = 10$, 3.333, and 2 wk. As for the yearly model, values of $x_i$ were equivalent to $\delta_i^{-1}$ to allow comparison of exponential-decay and abrupt-loss results. We input initial values for densities of larvae and hosts and the initial infection prevalence of nymphs, then allowed the model to run until the nymphal infection prevalence equilibrated to a tolerance of $10^{-7}$. We ran the model under conditions of low and high tick densities (10 000 and 100 000 eggs/ha, respectively).

## Results

### *Positive feedback in the yearly model, one-host case, with exponential reservoir decay*

Before exploring equilibrium values, we wanted to determine whether reservoir decay could establish a threshold infection prevalence. Epidemiological models generally predict that the density of susceptible hosts must exceed a threshold level for an epidemic to persist. In the case of a vector-borne disease like Lyme disease, both hosts and vectors must be sufficiently abundant for the pathogen to persist and to invade when it is rare. However, we expected that reservoir decay might increase the force of positive feedback in the Lyme disease system to the point of establishing an infection prevalence threshold. That is, even if vector and host densities exceed thresholds, the disease might require a large inoculum size to invade a disease-free system. Such an infection prevalence threshold can only exist if the proportional rate of increase in infection prevalence between years is an increasing function of infection prevalence over some range, such that the rate of increase is negative when infection prevalence is very low and positive when infection prevalence is higher. We tested for such a threshold in the yearly model with one host, assuming exponential reservoir decay.

If only one host species is available in the yearly model, the infection prevalence of nymphs each year ($\pi_{t+1}$) is equal to the specific infectivity of hosts the previous year ($S_{it}$). In the case of exponential reservoir decay, $S_{it}$ (and hence $\pi_{t+1}$) is given by Eq. 6. Dividing the right-hand side of Eq. 6 by $\pi_t$ yields the proportional change in nymphal infection prevalence between years. The derivative of this proportional change with respect to $\pi_t$ is negative for all $\pi_t > 0$. Thus, the proportional change in nymphal infection prevalence between years is a strictly decreasing function of the initial infection prevalence, and an infection prevalence threshold cannot exist.

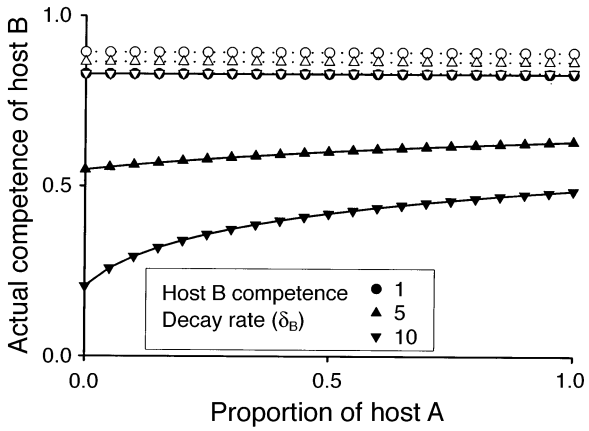


FIG. 5. Effect of tick abundance and host community composition on mean reservoir competence in the yearly model with exponential reservoir decay. Host A is a more competent reservoir (slower decay, $\delta_A = 1$) than host B. Total host abundance ($H_A + H_B$) is fixed at 20 hosts/ha. Solid lines and filled symbols are for low tick abundance (500 nymphs/ha), and dashed lines and open symbols are for high tick abundance (5000 nymphs/ha). Other parameter values are listed in *Methods: Yearly model: Running the yearly model.*

### *Sensitivity to host community composition and tick abundance in two-host case*

*Yearly model*.—We first report results of changing host community composition while keeping the total host density ($H_A + H_B$) constant at 20 hosts/ha. With exponential reservoir decay, mean reservoir competence of hosts at equilibrium depended on both the abundance of immature ticks and on host community composition. In our specific numerical example, mean reservoir competence of host B was positively related to the abundance of host A at low nymphal abundance, but not at high nymphal abundance (Fig. 5). Although host B had a potential reservoir competence of 0.9, its mean reservoir competence was lower at low abundance of ticks and in the absence of a highly competent reservoir, and this effect was much greater when reservoir decay of host B was rapid. At high nymphal abundance, hosts of both species were infected and reinfected very frequently, so reservoir competence was maintained near its maximum and changing the host composition had little effect.

Because exponential reservoir decay and host turnover were mathematically equivalent processes in the yearly model, we report effects on nymphal infection prevalence in the context of reservoir decay, but they apply equally well to host turnover. In the absence of reservoir decay, nymphal infection prevalence at equilibrium was a nearly linear function of the proportional representation of different hosts in the host community (Fig. 6A). However, reservoir decay introduced nonlinearity into this relationship (Fig. 6B, C), with highest slope at low abundance of host A. In addition, nymphal infection prevalence was insensitive to tick abundance when reservoir decay was absent (Fig. 6A), but highly sensitive to tick abundance when reservoir decay occurred (Fig. 6B, C). The specific form of reservoir decay did not affect the qualitative results, but nymphal infection prevalence was lower with exponential decay than with abrupt loss. This quantitative effect of the shape of the reservoir decay curve was greatest when the decay rate of host B was intermediate ($\delta_B = [1/x_B] = 5$).

Next, we kept the density of one host constant at 10 hosts/ha and examined the effect of varying the density of the other host on nymphal infection prevalence at equilibrium. In the absence of reservoir decay, nymphal infection prevalence varied almost linearly with proportional abundance of host A (Fig. 7A) and host B (Fig. 8A), and was insensitive to abundance of ticks. With reservoir decay, however, the relationship between nymphal infection prevalence and host community composition was nonlinear and sensitive to tick abundance. Nymphal infection prevalence was little affected by changing host community composition at high tick abundance (Figs. 7B, C and 8B, C). At low tick abundance, reservoir decay introduced nonlinearities into this relationship. Increasing proportional abundance of the more competent host A had greatest increase nymphal infection prevalence when host A was rare (Fig. 7B, C), and, conversely, the less competent host B caused greatest reduction in nymphal infection prevalence as it came to dominate the host community (Fig. 8B, C). Interestingly, exponential reservoir decay caused nymphal infection prevalence to decrease at high densities of host A (Fig. 7B), because tick bites became diluted among hosts, reducing frequency of reinfection and consequently reducing mean reservoir competence levels. This negative effect of host A abundance on nymphal infection prevalence was negligible when reservoir competence was lost abruptly (Fig. 7C). As expected, increasing abundance of host B (with host A held constant) reduced nymphal infection prevalence, with deterministic extinction of the disease when host B had rapid reservoir decay and high relative abundance in host community (Fig. 8B, C).

*Seasonal model*.—Qualitative effects of reservoir decay in the seasonal model were similar to effects in the yearly model (Figs. 6E, F, 7E, F, and 8E, F). In all cases, reservoir decay introduced nonlinearity and sensitivity to tick abundance into relationships between host community composition and nymphal infection rates. Realistic levels of host turnover, however, had less apparent effect in the seasonal model than did reservoir decay (Figs. 6D, 7D, and 8D). Host turnover introduced a relatively small degree of nonlinearity and a lower degree of sensitivity to tick abundance, especially at low densities of the more competent host A. As was true in the yearly model, the qualitative effects of reservoir decay were robust to the choice of decay formulation (exponential or abrupt), but quantitative effects were not. Even a low exponential decay rate for

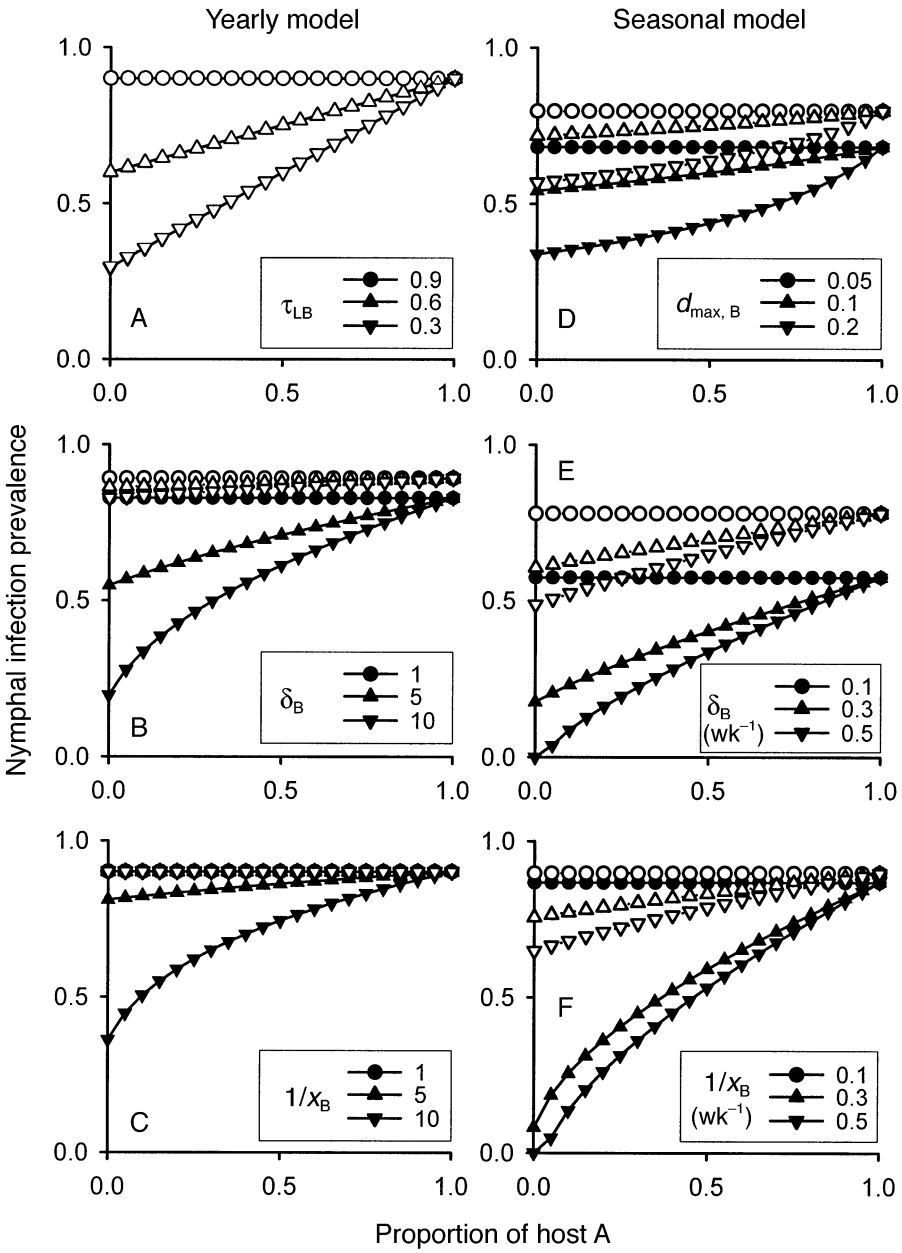


FIG. 6. Effects of tick abundance and host community composition in a two-host case with fixed total host abundance ($H_A + H_B = 20$ hosts/ha) in (A–C) the yearly model, and (D–F) the seasonal model. In all cases, hosts A and B are identical, except that host A generally is a more competent reservoir (maximum competence = 0.9) than host B. Solid lines and filled symbols are for low tick abundance (500 nymphs/ha in yearly model, 10 000 eggs/ha in seasonal model), and dotted lines and open symbols are for high tick abundance (5000 nymphs/ha in yearly model, 100 000 eggs/ha in seasonal model). Low-tick and high-tick symbols and lines overlap almost completely in panel (A). Yearly model formulations: (A) with no reservoir decay, but varying competence of host B; (B) with exponential reservoir decay ($\delta_A = 1$); and (C) with abrupt loss of reservoir competence ($x_A = 1$). Seasonal model formulations: (D) with host demographic turnover ($d_{min,A} = d_{min,B} = 0.01$ wk$^{-1}$, $d_{max,A} = 0.05$ wk$^{-1}$, $\tau_{LA} = \tau_{LB} = 0.9$), (E) with exponential reservoir decay ($\delta_A = 0.1$ wk$^{-1}$), and (F) with abrupt loss of reservoir competence ($x_A = 10$). Other parameter values are listed in *Methods: Yearly model: Running the yearly model* and *Running the seasonal model*. Results from the seasonal model without reservoir decay or host turnover were essentially indistinguishable from equivalent results of the yearly model.

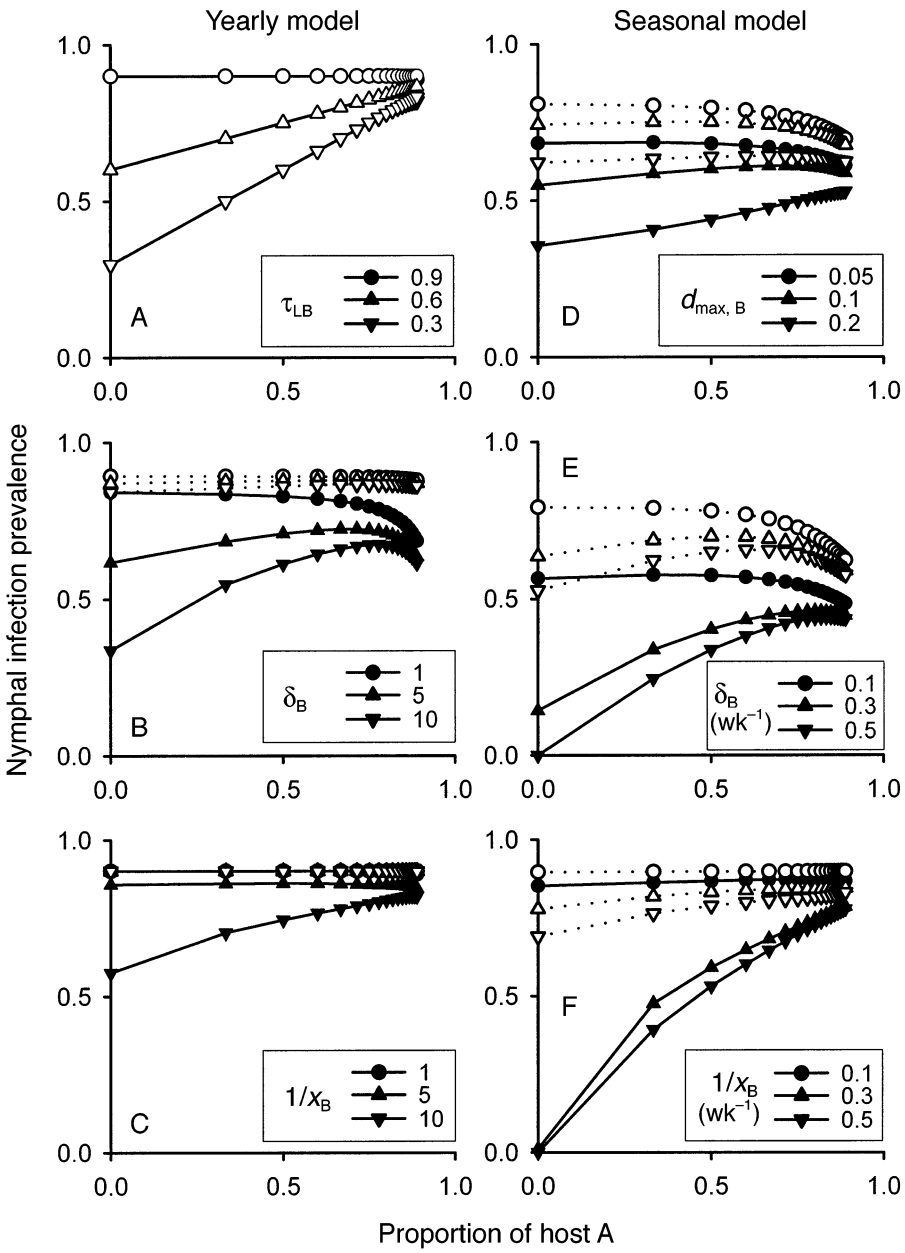


FIG. 7. Effects of tick abundance and host community composition in a two-host case with fixed abundance of the less competent host B ($H_B$ = 10 hosts/ha) in (A–C) the yearly model and (D–F) the seasonal model. Host A abundance varied from 0 to 40 hosts/ha. Parameter values and model formulations are as in Fig. 6.

host B ($\delta_B$ = 0.1 wk$^{-1}$) caused substantial reduction in nymphal infection prevalence at low tick densities (Figs. 6E, 7E, and 8E), but an equivalent decay rate in the abrupt-loss framework ($x_B$ = 10 wk) produced negligible effects on infection prevalence (Figs. 6F, 7F, and 8F). Quantitative results of exponential and abrupt reservoir decay were more comparable for higher decay rates. In addition, exponential decay triggered a negative effect of very high abundances of the more competent host A on nymphal infection prevalence (Fig. 7E) that did not occur in the abrupt-loss framework (Fig. 7F).

## DISCUSSION

Reservoir competence may decay with time since infection, and uninfected hosts can replace infected ones through demographic turnover. We constructed two models to investigate the effects of reservoir decay and host demographic turnover on Lyme disease epizootiology. Our yearly model was more simplistic,

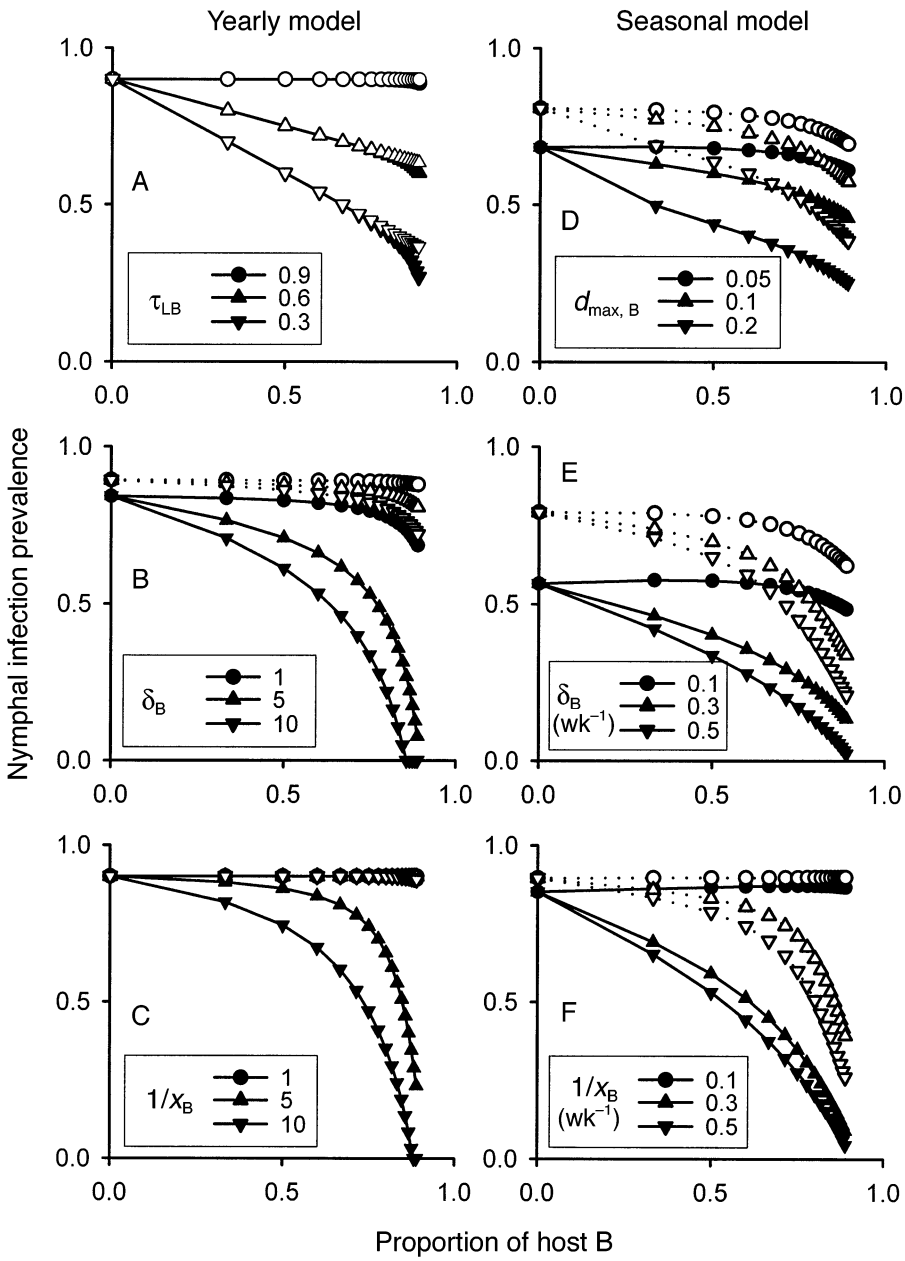


FIG. 8. Effects of tick abundance and host community composition in a two-host case with fixed abundance of the more competent host A ($H_A$ = 10 hosts/ha) in (A–C) the yearly model and (D–F) the seasonal model. Host B abundance varied from 0 to 40 hosts/ha. Parameter values and model formulations are as for Fig. 6.

whereas the seasonal model explicitly included such complicating factors as seasonal birth and death rates of hosts and phenology of tick activity. Nevertheless, both models supported similar conclusions, namely that (1) reservoir decay and turnover have similar effects, although the effects of host turnover are smaller in the seasonal model, (2) neither can establish a threshold infection prevalence of ticks, and (3) both cause specific infectivity of host populations and, consequently, nymphal infection prevalence to be sensitive to host community composition and even more sensitive to tick abundance.

### *Implications for empirical measurement of reservoir competence*

If reservoir competence of a host decays over time since it was last infected, then mean reservoir competence can vary with the frequency at which hosts are bitten by infected ticks. Frequency of infection depends on both the abundance of immature ticks and the rel-

ative abundance of competent reservoir hosts. For example, our results indicate that mean reservoir competence of a host population could vary 0.2–0.9 for a host with a high potential competence (0.9) and a high reservoir decay rate, depending on tick abundance and the presence of a highly competent reservoir (Fig. 5). This phenomenon could explain the wide range of field measurements of reservoir competence for particular host species across sites and times. For example, field-measured reservoir competence values for white-footed mice and eastern chipmunks in Millbrook, New York, USA, were 0.94 and 0.69, respectively (Schmidt and Ostfeld 2001), whereas field-measured competence values from other sites range 0.21–0.875 for white-footed mice and are ~0.20 for chipmunks (reviewed by Giardina et al. 2000). These results suggest that potential competence of chipmunks may be high, but reservoir decay may be more rapid for chipmunks than for white-footed mice. Alternatively, the reservoir characteristics of hosts may vary among populations due to genetic differences in immune response (Brunet et al. 1995). Empirical studies of mean reservoir competence across sites and years are necessary to disentangle the relative influences of genetic composition and community context.

### *Effects on positive feedback and dilution in Lyme disease ecology*

Before constructing these models, we expected that reservoir decay or host turnover could enhance positive feedback to the point of establishing a threshold infection prevalence, below which the epizootic would deterministically vanish. Our logic was that at low infection prevalence, hosts would experience long intervals between reinfections, and thus specific infectivity would decrease. As a result, the nymphal infection the next year would be reduced, and the process repeated. However, analysis of the yearly model indicated that reservoir decay and turnover, as we formulated them, could not establish such a threshold. We found that, at a constant tick density, specific infectivity was a saturating function of nymphal infection prevalence (Eqs. 6, 7, and 9), similar to a type II functional response of predators (Holling 1959). By analogy, we suspect that a threshold could be established by a sigmoid (type III) relationship between specific infectivity and nymphal infection prevalence. A sigmoid relationship could result if the rate of encounter or transmission between nymphs and hosts is not constant, but increases with nymphal infection prevalence, or if reinfection increases the maximum competence of the host. If a threshold infection prevalence existed, it would limit the probability that Lyme disease could become established in tick–host communities where it had been absent. However, the existence of such a threshold seems inconsistent with the rapid spread of Lyme disease (White et al. 1991) and the rapid invasion of the Lyme disease spirochete in colonizing populations of black-legged ticks (Lastavica et al. 1989).

Reservoir decay introduced nonlinearity into the relationship between nymphal infection prevalence and the relative abundance of a poor reservoir. This nonlinearity stemmed from enhanced positive feedback in the transmission cycle: a reduction in the mean specific infectivity of the host community reduced the nymphal infection prevalence, and consequently reduced the frequency at which hosts were infected and reinfected, allowing greater decay in reservoir competence or greater recruitment of uninfected individuals between infection events and further reducing mean specific infectivity. As a result, increasing the proportional abundance of a poor reservoir had the greatest marginal effect on nymphal infection prevalence when poor reservoirs dominate the host community. Conversely, the marginal effect of the abundance of a highly competent reservoir was also greatest when it was relatively rare. Therefore, reservoir decay or host turnover are expected to enhance the dilution effect (Ostfeld and Keesing 2000*a*, *b*, Schmidt and Ostfeld 2001) of poorer reservoirs. Schmidt and Ostfeld (2001) used the discrepancy between the high competence of small mammals and relatively low nymphal infection prevalence to infer that poor reservoirs contributed ~60–70% of larval and nymphal blood meals in forested sites in Millbrook, New York. Other sites typically report nymphal infection prevalence values far below maximum competence values (Lane et al. [1991] and references therein), suggesting that poor reservoirs dominate most host communities in terms of the relative number of tick meals they provide. Consequently, our model predicts that factors that increase the relative abundance of poor reservoirs, by increasing their populations or by reducing those of competent reservoirs, should produce accelerating benefits in terms of reduced nymphal infection prevalence.

Our model does not include a numerical response of ticks to larval and nymphal hosts. Dilution of infection by animals that are good hosts for ticks but poor reservoirs for pathogens could be offset by the added opportunities for tick feeding success, thereby elevating the absolute density of infected nymphs (Schmidt and Ostfeld 2001). This trade-off between dilution and elevated vector densities is involved in vector-borne diseases other than Lyme disease (Rogers 1988, Sota and Mogi 1989, Lord et al. 1996, Norman et al. 1999). However, local density of larval *I. scapularis* is likely to be more strongly linked to the density and movements of large mammalian hosts for adult ticks than to the abundance of hosts for immature stages (Wilson et al. 1985, Duffy et al. 1994, Ostfeld 1997). Also, weather and other abiotic factors can cause variation in tick survival, independent of host density (Lindsay et al. 1995, Jones and Kitron 2000). Therefore, changes in total abundance of hosts for immature tick stages are

likely to have weak effects on nymphal density relative to effects on nymphal infection prevalence.

### *Relationship between competent reservoir abundance and infection prevalence*

Reservoir decay and host turnover increased the likelihood that increasing total host abundance would decrease nymphal infection rates in our models, even if the relative abundance of a competent reservoir increased. This effect resulted from a limited number of tick bites being distributed among more individual hosts, which caused hosts to be infected and reinfected less often. This possibility was raised by Spielman et al. (1984), because a reduced tick-to-host ratio is expected to reduce host infection prevalence. Reservoir competence decay (especially exponential decay) and host turnover exacerbate this effect, by requiring very high densities of infected ticks to ensure that (1) reservoir competence remains near its maximum value, or (2) host recruits become infected before they feed appreciable numbers of larvae. In reality, consistently high host abundance might increase the equilibrium density of immature ticks, increasing the number of available tick bites in the long term. For example, the abundance of nymphal black-legged ticks is positively related to the abundance of white-footed mice the previous year (Ostfeld et al. 2001). However, abundance of immature ticks also varies in space and time due to factors independent of the abundance of their hosts (Lindsay et al. 1999). Abiotic factors can cause survival and abundance of ticks to vary among years and sites (Lord 1993, Lindsay et al. 1995, Ginsberg and Zhioua 1996, Jones and Kitron 2000), as can the abundance and habitat use of hosts for adult ticks, like white-tailed deer (*Odocoileus virginianus*, Wilson et al. 1985, 1990, Duffy et al. 1994). Thus, we hypothesize that the abundances of many host species and of host-seeking nymphs often may be weakly coupled, resulting in reduced nymphal tick burdens, infection frequencies, and, consequently, nymphal infection prevalence at high total host densities. Schmidt et al. (1999) found that high abundance of eastern chipmunks negatively affected burdens of immature black-legged ticks on white-footed mice, which may set the stage for reduced infection prevalence due to reservoir decay or host turnover. Empirical comparisons of infection prevalence with host abundance among years and sites are necessary to test this hypothesis.

### *Relationship between tick density and infection prevalence*

In our models, both reservoir decay and host turnover made specific infectivity and equilibrium nymphal infection rates sensitive to tick abundance. As a consequence, we hypothesize that efforts to control Lyme disease risk by reducing the abundance of nymphal ticks may reap additional benefit by reducing their infection prevalence. However, empirical evidence does not appear to support our hypothesis. Stafford (1993) and Daniels et al. (1993) found that exclusion of white-tailed deer reduced the abundance of nymphal black-legged ticks by 81–98%, and found only suggestive but nonsignificant evidence of reduced nymphal infection prevalence. Mather et al. (1993) found that controlled burning reduced the abundance of nymphs by 49% but may have increased the proportion infected.

The disagreement between our model predictions and the empirical results of tick reduction suggests that one or more of three possibilities may be operating: (1) deer abundance may affect both tick density and the host selection of immature ticks, (2) experimental results reflect nonequilibrium conditions whereas the model predicts equilibrium conditions, and (3) feeding success of immature black-legged ticks may be density-dependent. We consider each of these possibilities in turn.

White-tailed deer are important hosts for adult black-legged ticks, so reducing deer abundance can effectively reduce tick density (Daniels et al. 1993, Stafford 1993). However, deer also act as hosts to immature black-legged ticks (Piesman and Spielman 1979, Main et al. 1981), but are not competent reservoirs of *B. burgdorferi* (Telford et al. 1988). Therefore, reducing deer abundance may increase the proportion of immature ticks feeding on competent reservoirs, thereby reducing the dilution effect (Ostfeld and Keesing 2000*a*). Without complete data on tick burdens and competence decay curves for host species across an entire host community, it is difficult to assess the net effect of reduced tick density and reduced dilution effect.

Regarding the second possibility, our model predicts equilibrium states for hosts and ticks, whereas the results of these perturbation experiments undoubtedly reflect nonequilibrium conditions. However, our model suggests that large reductions in nymphal abundance should produce noticeable effects after one year, even if equilibrium conditions may not be reached for several years. For example, if the yearly model with exponential reservoir decay is equilibrated at 5000 nymphs/ha, $H_A = H_B = 10$, $\tau_{max,A} = \tau_{max,B} = 0.5$, $\delta_A = 1$, $\delta_B = 5$, and other parameter values described in *Methods: Yearly model: Running the yearly model*, and then the nymphal density is reduced to 500 nymphs/ha in year $t$, nymphal infection prevalence drops from 0.48 in year $t$ to 0.36 in year $t + 1$ on its way to a new equilibrium value of 0.32. The possibility remains that the empirical reductions in nymphal abundance were too small to produce detectable effects. However, if 50–98% reductions in nymphal abundance are insufficient to demonstrably reduce nymphal infection prevalence, then the relevance of this prediction of our model is questionable.

The lack of strong evidence for an effect of tick density on nymphal infection prevalence may also point to deficiencies in our model. In particular, our

model does not incorporate density-dependent feeding success of ticks. Density-dependent feeding success would tend to stabilize tick burdens despite variations in tick and host abundance, greatly diminishing the effect of tick abundance on the frequency at which hosts are infected. For example, Goodwin et al. (2001) observed that burdens of immature black-legged ticks on white-footed mice were surprisingly constant, in spite of large variations in mouse and tick densities. Hazler and Ostfeld (1995) observed no effect of density on feeding success of larval black-legged ticks, but Levin and Fish (1998) found that increasing density of larval black-legged ticks induced greater grooming activity by white-footed mice and decreased the proportion of larvae that successfully attached and fed. Perhaps as a result of density-dependent rates at which immature ticks successfully attach to hosts, tick burdens on small mammals tend to vary much less than estimates of questing tick abundance (Ginsberg and Ewing 1989, Ostfeld et al. 1995, Jones et al. 1998*a*), and questing tick density can be a poor predictor of tick burdens (Ostfeld et al. 1996*c*, Schmidt et al. 1999). Thus, if mean tick abundance is high enough that tick burdens on host are always saturated, then variations about that mean may be unimportant. Empirically documenting density dependence in tick feeding success and modeling its effects on Lyme disease dynamics are fruitful avenues for future research.

Porco (1991, 1999) incorporated both host turnover and host recovery into yearly (Porco 1991) and monthly (Porco 1999) Lyme disease models, and reported that peak infection prevalence of black-legged ticks is reduced by host turnover and recovery from infection. He used a susceptible–infected–recovered (SIR) framework, in which infected hosts recovered with complete immunity at a constant proportional rate. This is similar, but not equivalent, to our formulation of exponential reservoir decay. Sensitivity analysis indicated that host recovery rate was the second most important parameter in the monthly model (Porco 1999). Intuitively, reservoir decay and host turnover must reduce infection levels, compared with the case where competence is always at its maximum and infected hosts are never replaced by uninfected hosts. In addition, Porco (1991) concluded that reducing the abundance of white-footed mice would have little effect on the abundance of infected nymphs, unless doing so reduced the survival of larval black-legged ticks. Our results illuminate a possible mechanism underlying Porco's conclusion. We found that reducing the density of an abundant competent reservoir may actually increase nymphal infection prevalence by increasing the numbers of tick bites per host. Thus, our modeling suggests that the most effective strategy for ecological management of Lyme disease risk would be to both decrease tick abundance and increase the abundance of hosts for immature ticks, ideally by increasing the abundance of relatively poor reservoirs. The expected result of this strategy is to decrease the frequency of infective tick bites on each individual host, allowing greater decay of competence and replacement of infected hosts by uninfected recruits. Ultimately, specific infectivity and the next year's nymphal infection prevalence would be reduced. However, density-dependent feeding success could limit the effectiveness of this strategy.

### *Relative importance of reservoir decay and host turnover*

Based on the results of our seasonal model, we suggest that reservoir decay may be a more important factor in the ecology of Lyme disease than host turnover. Host demographic turnover reduces host infection prevalence. Because infected and uninfected hosts are assumed to have equal mortality rates, host infection prevalence is affected primarily by per capita birth rates. Birth rates are strongly linked to individual fitness and constrained by allometric scaling relationships (Begon et al. 1993), reducing the potential variation among host species. However, the seasonal timing of reproduction may contribute substantial variation in host infection rates. If recruitment occurs predominantly in the early summer, with little recruitment between the peak periods of nymphal and larval activity, then a greater proportion of larval hosts (and consequently fed larvae) will be infected than if recruitment occurs immediately before and during the peak larval feeding period.

Unlike demographic turnover, reservoir decay rates are not likely to be tightly constrained by body size or taxonomy, so the potential variation among host species is enormous. Some species remain infective for long periods of time, like white-footed mice (but see Lindsay et al. [1997]), whereas other hosts might be completely incompetent reservoirs, but the spirochete may still be transmitted between cofeeding ticks (Gern and Rais 1996, Ogden et al. 1997, Patrican 1997*b*), yielding an almost infinite effective decay rate.

Randolph and Craine (1995) state that duration of infectivity exceeds average life span for small rodent hosts, which would imply that turnover has a more powerful effect than reservoir decay on *B. burgdorferi* prevalence in hosts and ticks. However, a host may remain measurably infective even as its reservoir competence decays to low levels, thus rendering it less effective in infecting vectors. Also, Schmidt and Ostfeld (2001) inferred that >60% of meals taken by immature *I. scapularis* may be drawn from relatively incompetent hosts, even in forests where highly competent white-footed mice and eastern chipmunks are abundant. Thus, hosts with relatively long life span and potentially rapid reservoir decay may be important in the natural cycle of *B. burgdorferi* transmission. However, further empirical assessment of reservoir decay curves for entire host communities is necessary to fully characterize the relative importance of demographic turnover and reservoir decay.

### *Shape of the reservoir decay curve*

Competence of even the most important reservoirs of Lyme disease is poorly understood. Estimates of maximum competence vary among studies, and little attention has been devoted to characterizing the variability in competence with time and repeated exposures (but see Richter et al. [2000]). Our results pose several questions, for which there are few or no data to provide answers. Is reservoir decay more appropriately represented by an exponential function, by an abrupt-loss susceptible–infected–susceptible (SIS) model, or neither? A sigmoid curve would tend to produce results more similar to the abrupt-loss case, as actual competence remains high for an extended period. Empirical reservoir decay curves for rice rats (*Oryzomys palustris*; Levin et al. 1995) and American Robins (Richter et al. 2000) suggest a sigmoid shape, whereas that of white-footed mice (Donahue et al. 1987) seems nearly linear. In addition, the rate of decay could decrease or increase over time, causing the tail of the decay curve to become thicker or thinner than for an exponential curve. A thick-tailed decay curve would tend to reduce sensitivity to tick abundance, because even individuals that have not been infected for a long time retain substantial infectivity, whereas a thin-tailed curve would increase the importance of tick abundance. Our results indicate that the shape of the reservoir decay curve, especially its initial slope, can affect the quantitative effects of tick abundance and host community composition. In our seasonal model, for example, nymphal infection prevalence is near its maximum irrespective of tick abundance when reservoir competence is lost abruptly after 10 wk (Figs. 6F, 7F, and 8F). However, with exponential reservoir decay at a rate of 0.1 $wk^{-1}$ (equivalent to a 10-wk expected duration of infection), nymphal infection prevalence is ~25% lower at 500 nymphs/ha than at 5000 nymphs/ha. In this example, nearly all hosts are infected within 10 wk, but the rapid initial slope of the exponential decay curve still results in appreciable reduction of competence even at a low decay rate. This quantitative effect of initial slope suggests that merely determining the expected duration of infectivity may not be sufficient to adequately characterize the reservoir decay curve, and that the particular formulation of SIS and related models can be important.

A critical assumption in our formulation of reservoir decay is that reservoir competence returns to the same level each time a host is reinfected. This implies that reservoir hosts are incapable of mounting an effective immune response to *B. burgdorferi*, and that repeated tick bites do not affect *B. burgdorferi* transmission from tick to host. Wikel et al. (1997) reported that repeated bites by uninfected *I. scapularis* nymphs reduce the probability of transmission from infected nymphs to laboratory mice. A growing body of evidence indicates that tick-transmitted *B. burgdorferi* is capable of evading the host immune system via changes in antigen characteristics (Fikrig et al. 1993, Schwan and Piesman 2000) and mediation of host immune response by tick saliva (Ribeiro and Titus 1990, Ramachandra and Wikel 1992, Urioste et al. 1994). Indeed, reservoir competence of European hosts is inversely correlated with the efficacy of immune response (Kurtenbach et al. 1994, 1998). In regions of the USA where Lyme disease is endemic, infection rates of white-footed mice approach 100% (Anderson et al. 1987*a*, *b*) due to frequent bites from infected nymphs. However, reservoir competence remains high (Schmidt and Ostfeld 2001), suggesting that repeated infection via tick bite does not adversely affect reservoir competence. To our knowledge, the only direct evidence that reservoir competence returns to the same value after repeated infection comes from American Robins that were infected twice (Richter et al. 2000). Additional possibilities pertaining to reservoir decay that require further empirical study include the effect of age and taxonomy on decay curve parameters.

## Conclusions

We examined the roles of reservoir competence decay and host demographic turnover in the ecology of Lyme disease, using two model formulations of differing temporal resolution. Both processes tended to reduce transmission of the Lyme spirochete between reservoir hosts and vector ticks, reducing the infection prevalence of nymphal ticks, but enhanced positive feedback in the transmission process. Both processes also increased the sensitivity of infection prevalence to tick abundance and made the relationship between infection prevalence and host community composition highly nonlinear. The shape of the reservoir competence decay curve affected quantitative but not qualitative predictions of the model. Our model predicts that reservoir decay and host turnover can enhance the benefits of managing tick and host abundances. Reducing tick abundance and increasing the relative abundance of poor reservoirs are expected to have synergistic effects on the abundance of infected nymphs and risk of Lyme disease for humans. Empirical attention to characterizing reservoir decay in the context of exposure history, age, and taxonomic category is necessary to develop a robust understanding of the ecology of Lyme disease.

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## APPENDIX A

A list of the state constants, variables, and parameters used in the yearly model is available in ESA's Electronic Data Archive: *Ecological Archives* A012-010-A1.

## APPENDIX B

A list of the state constants, variables, and parameters used in the seasonal model is available in ESA's Electronic Data Archive: *Ecological Archives* A012-010-A2.