Effect of Forest Fragmentation on Lyme Disease Risk

BRIAN F. ALLAN,* FELICIA KEESING,†§ AND RICHARD S. OSTFELD‡

*Rutgers University, New Brunswick, NJ 08901, U.S.A.
†Department of Biology, Bard College, Annandale, NY 12504, U.S.A., email keesing@bard.edu
‡Institute of Ecosystem Studies, Millbrook, NY 12545, U.S.A.

Abstract: *Forest destruction and fragmentation in the United States recently have been shown to reduce mammalian species diversity and to elevate population densities of white-footed mice (*Peromyscus leucopus*). One potential consequence of reduced species diversity and high mouse density in small fragments is an increase in human exposure to Lyme disease. Increased risk of exposure to this disease is expected because of the role of the white-footed mouse as the principal natural reservoir of the Lyme bacterium,* Borrelia burgdorferi*. Blacklegged ticks (*Ixodes scapularis*) feeding on mice have a higher probability of becoming infected with the bacterium than do ticks feeding on any other host species. We hypothesized that small forest patches (<2 ha) have a higher density of infected nymphal blacklegged ticks, which is the primary risk factor for Lyme disease, than larger patches (2–8 ha). In the summer of 2000, we sampled tick density and* B. burgdorferi *infection prevalence in 14 maple-dominated forest patches, ranging in size from 0.7 to 7.6 ha, in Dutchess County of southeastern New York state. We found a significant linear decline in nymphal infection prevalence with increasing patch area and a significant exponential decline in nymphal density with increasing patch area. The consequence was a dramatic increase in the density of infected nymphs, and therefore in Lyme disease risk, with decreasing forest patch size. We did not observe a similar relationship between the density of larval ticks and patch size. These results suggest that by influencing the community composition of vertebrate hosts for disease-bearing vectors, habitat fragmentation can influence human health.*

Efecto de la Fragmentación de Bosques sobre el Riesgo de la Enfermedad de Lyme

Resumen: *Se ha mostrado que la destrucción y la fragmentación de bosques en los Estados Unidos reducen la diversidad de especies de mamíferos y elevan la densidad de poblaciones de ratones (*Peromyscus leucopus*). El incremento de la exposición humana a la enfermedad de Lyme es una consecuencia potencial de la reducción en la diversidad de especies y de la alta densidad de ratones en fragmentos pequeños. Se espera el incremento de la exposición a esta enfermedad por el papel del ratón como el principal reservorio natural de la bacteria de Lyme,* Borrelia burgdorferi*. Los ácaros (*Ixodes scapularis*) alimentándose en ratones tienen mayor probabilidad de infectarse con la bacteria que los ácaros que se alimentan en cualquier otra especie huésped. Nuestra hipótesis fue que los fragmentos pequeños (<2 ha) de bosque tienen una mayor densidad de ninfas de ácaros, que son el factor de riesgo primario para la enfermedad de Lyme, que en fragmentos mayores (2–8 ha). En verano de 2000, muestreamos la densidad de ácaros y la prevalencia de infección por* Borrelia burgdorferi *en 14 fragmentos de bosque dominado por arce, con superficie entre 0.7 ha y 7.6 ha, en el Condado Duchess, sureste del estado de New York. Encontramos una declinación lineal significativa en la prevalencia de infección ninfal al incrementar la superficie del fragmento y una declinación exponencial significativa en la densidad de ninfas al incrementar la superficie del fragmento. La consecuencia fue un incremento dramático en la densidad de ninfas infectadas, y por lo tanto en el riesgo de la enfermedad de Lyme, con el decremento de la superficie del fragmento. No observamos una relación similar entre la densidad de ácaros larvarios y el tamaño del fragmento. Estos resultados sugieren que, al influir en la composición de la comunidad de vertebrados huéspedes de vectores de enfermedades, la fragmentación del hábitat puede influir en la salud humana.*

§Address correspondence to F. Keesing.
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Introduction

Human activities in the northeastern United States have resulted in the fragmentation of what was once a predominantly forested landscape. Fragmentation involves a reduction in the average size of remaining forest patches, increasing the distance between patches and increasing the ratio of edge to interior (Andrén 1994; Murcia 1995). One key consequence of the fragmentation of previously continuous forest is a reduction of species diversity in remnant forest patches, a pattern that has been demonstrated for both birds (Blake & Karr 1987; Andrén 1994) and mammals (Andrén 1994; Rosenblatt et al. 1999). However, certain species—typically habitat generalists or species characterized by high population densities and small home ranges—appear to thrive in highly fragmented landscapes (Bender et al. 1998; Debinski & Holt 2000). One such organism, the white-footed mouse (*Peromyscus leucopus*), reaches unusually high densities in small forest fragments, probably resulting from a decrease in abundance of both predators and competitors (Nupp & Swihart 1998). Densities of *P. leucopus* tend to be inversely correlated with forest patch area, with a rapid, nonlinear increase in density in forest patches of <2 ha (Nupp & Swihart 1996; Krohne & Hoch 1999).

We predicted that the reduced species diversity and increased abundance of *P. leucopus* that accompany the fragmentation of forests into small patches would increase the risk of human exposure to Lyme disease. Lyme disease is caused by the bacterium *Borrelia burgdorferi*, which, in eastern and central North America, is transmitted by the bite of an infected blacklegged tick (*Ixodes scapularis)*. Larval ticks typically hatch without *B. burgdorferi* infection and attempt a blood meal from any of dozens of species of mammals, birds, and lizards. Larvae that feed on white-footed mice are highly likely to become infected, but those that feed from most other mammals, birds, and reptiles are unlikely to become infected (Lane et al. 1991; Anderson & Magnarelli 1993; Mather 1993; Ostfeld & Keesing 2000*a*). The white-footed mouse is therefore considered the principal natural reservoir for the bacterium. Larvae that acquire a *B. burgdorferi* infection molt into infected nymphs, which may then transmit the infection to humans, causing Lyme disease. The majority of Lyme disease cases are thought to be transmitted by nymphal ticks, owing to their small size and summer peak in activity, which coincides with human peridomestic and recreational use of the outdoors (Falco & Fish 1989; Barbour & Fish 1993). Adult ticks may also be infected, but they are larger, easier to detect, and active in late fall and early spring when human activity in forests is reduced (Lane et al. 1991).

Lyme disease risk is related to both the proportion of nymphs infected (or the nymphal infection prevalence, NIP) and the density of infected nymphs (DIN) (Ostfeld & Keesing 2000*b*; Ostfeld et al. 2002). The NIP represents the probability of an individual being exposed to the Lyme bacterium if bitten by a tick and is a function of the distribution of larval meals among the community of vertebrate hosts. Because white-footed mice are the most competent reservoirs for the disease, NIP is expected to increase with (1) increasing absolute density of white-footed mice and (2) decreasing species diversity in the host community, which reduces the availability of incompetent reservoir hosts (Ostfeld & Keesing 2000*a*, 2000*b*; Schmidt & Ostfeld 2001). The DIN, on the other hand, represents the probability of being exposed to the Lyme bacterium upon entering tick habitat. Causes of variation in DIN are more complex because DIN is the product of NIP and the total density of nymphs (DON). Several factors can influence DON, including the abundance of larvae in the prior year and the survival of those larvae to the nymphal stage. Larval density, in turn, is determined largely by the distribution of hosts for adult ticks, principally white-tailed deer (*Odocoileus virginianus*) (Wilson et al. 1985, 1988; Daniels et al. 1993; Stafford 1993), because these hosts are the principal site of mating by adult ticks, which determines the sites of larval production (Ostfeld et al. 1996).

Given the presumed effects of forest fragmentation on the vertebrate hosts of ticks and the effects of these hosts on the density and infection prevalence of ticks, we hypothesized that (1) NIP is negatively correlated with forest patch area and (2) DON is negatively correlated with forest patch area and positively correlated with the density of larval ticks. Therefore, (3) DIN, the product of NIP and DON, is negatively correlated with forest patch area and positively correlated with the density of larval ticks.

Methods

We established a field-sampling program in Dutchess County, southeastern New York (lat. 41°50′N, long. 73°45′W), to determine both the distribution of ticks among forest patches of different areas and the prevalence of *Borrelia burgdorferi* infection within ticks. Dutchess County has had the highest number of human cases of Lyme disease and among the highest incidence of Lyme disease (cases per 100,000 population) in the United States over the past several years (Orloski et al. 2000). Based on previous investigations indicating that white-footed mouse densities increased dramatically in small (<2 ha) forest patches compared with larger (>2 ha) patches (Nupp & Swihart 1996; Krohne & Hoch 1999), we selected 14 forest fragments that ranged in area from 0.7 to 7.6 ha.

We selected all forest patches to be a minimum of 1.6 km from the next nearest forested site; average nearest-

neighbor distance was 6.96 km ± 1.37 km (SE). All sites were dominated by sugar maple (*Acer saccharum*) or red maple (*A. rubrum*) because maple-dominated forest has relatively constant interannual densities of black-legged ticks in Dutchess County sites (Ostfeld 1997). Oak-dominated sites were avoided because considerable variation in tick abundance among years exists in this habitat due to masting (Ostfeld et al. 1996, 2001). We chose study sites on the basis of geographic information system maps of Dutchess County, which were prepared from 1994–1995 U.S. Department of Agriculture black and white aerial photos by the Dutchess County Environmental Management Council (Millbrook, New York). Their classification into land-cover types was based on the *LUNR Classification Manual* from Cornell University's Institute for Resource Information Systems (Ithaca, New York). Sites were ground-truthed to confirm area and tree-species composition. Nonforested matrix in the area consisted of a mix between suburban residential and small-scale agricultural land uses.

Nymphal ticks were collected from all sites during the peak in nymphal activity (10 June–15 July 2000), and larval ticks were collected from all sites during the peak in larval activity (August 2000). The timing of our collections coincided with the known peak-activity period of the respective life stages, based on long-term monitoring in Dutchess County (Ostfeld et al. 2001). To avoid a sampling bias, the order of visitation to sites was varied among weeks. Although the nymphal cohort in 2000 arose from larvae present in 1999, we assumed that the 2000 larval cohort would represent average relative abundances in the patches through time. At each site we established a set of parallel line transects totaling 400 m. Transects were at least 10 m apart and varied from 50 to 100 m in length. To determine the density of ticks among patches of different area and to collect ticks for determination of *B. burgdorferi* prevalence, we dragged a 1-m^2 drag cloth, held as close to the ground as possible, the length of each transect. We stopped to examine the cloth every 20 m, and all ticks were removed with forceps and either maintained alive until dissection or preserved in 70% ethanol for later identification. This standard drag-sampling technique has been demonstrated to be both reliable and efficient in surveying *I. scapularis* (Falco & Fish 1992; Ostfeld et al. 1995; Daniels et al. 2000).

We examined at least 20 (21.8 ± 1.16 SE) nymphal ticks from each site for the presence of *B. burgdorferi*. Using immunofluorescence microscopy, we washed ticks once in 70% ethanol and twice in deionized water, placed them in an Eppendorf tube, and ground them in phosphate-buffered saline (PBS). Three 5-mL aliquots of tick suspension were placed in separate wells in multiwell slides, air-dried, and fixed in cold acetone for 10 minutes. Fluorescent rabbit anti-*Borrelia* conjugate was added to wells and incubated for 45 minutes at 37°C. Slides were then washed in PBS, dried, and placed in fluorescent-antibody mounting medium. We examined the slides under an Olympus BH-2 binocular microscope. If spirochetes were not detected immediately, the three wells per individual were examined systematically. Each individual tick was categorized as positive or negative for *B. burgdorferi.*

Our general approach was to use simple regression analyses to test whether DON, NIP, and DIN were a significant function of patch area. Because nymph parameters may be influenced by larval abundance, we also used multiple-regression analyses to test for the effects of both density of larvae (DOL) and patch area (independent variables) on DON, NIP, and DIN (dependent variables).

Results

Nymph density (DON), nymphal infection prevalence (NIP), and their product, the density of infected nymphs (DIN), were all negative functions of patch area. The DON was a significant negative exponential function of patch area ($R^2 = 0.51, p = 0.02, n = 14$ [Fig. 1a], and the R^2 of the exponential model had a 33% better fit than the R^2 of the linear model). Nymph density was more than three times higher in the five smallest fragments (<1.2 ha) than in the larger fragments. On average, small fragments had 0.1 nymphs/m^2, whereas larger fragments had 0.03/m^2. The NIP was a significant negative linear function of patch area ($R^2 = 0.43$, $p = 0.011$, $n = 14$; Fig. 1b), decreasing from an average of 70% in the smallest fragments to an average of 48% in larger fragments. The DIN was significantly related to patch area by a negative exponential function ($R^2 = 0.65$, $p = 0.01$, $n = 14$ [Fig 1c], and the R^2 of the exponential model had a 50% better fit than the R^2 of the linear model). The five smallest fragments supported an average of seven times as many infected nymphs per square meter as did the larger fragments (0.07 infected nymphs/m^2 vs. 0.01 infected nymphs/m^2). A simple regression indicated that the density of larvae (DOL) was not significantly related to patch area ($R^2 = 0.01$, $p = 0.78$, $n = 14$; Fig. 2).

Both the density of larvae (DOL) and patch area appeared to influence the size of nymphal populations. Multiple linear regression of the combined influence of patch area and DOL showed that the density of nymphs was a positive linear function of DOL ($p = 0.047$) and a negative linear function of patch area ($p = 0.029$) (model $R^2 = 0.53$). Multiple linear regression of the combined influence of patch area and DOL on DIN was qualitatively similar. The DIN was a negative linear function of patch area ($p = 0.018$) but was not significantly related to DOL ($p = 0.099$) (model $R^2 = 0.52$). Multiple regression of the combined influence of patch area and

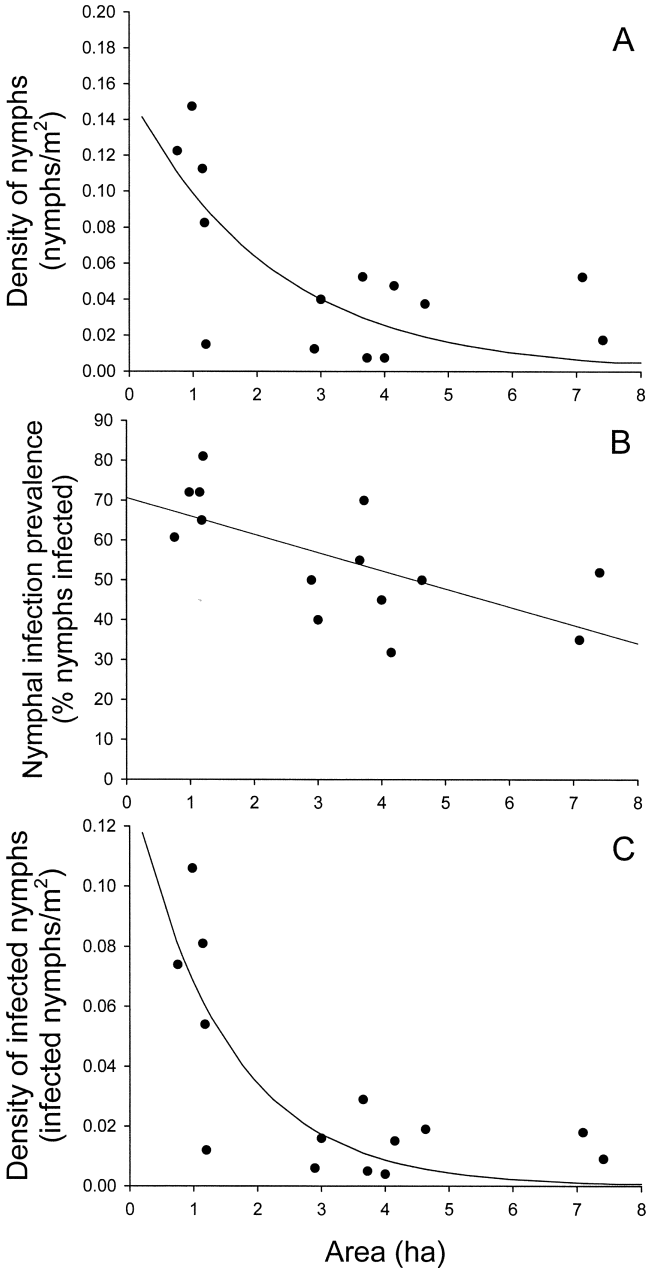

Figure 1. Relationship between measures of Lyme disease risk and forest patch area in a fragmented landscape in Dutchess County, New York: (a) density of nymphal Ixodes scapularis *versus the area of forest fragments (*$R^2 = 0.51$, $p < 0.02$, $n = 14$*); (b) percentage of nymphal* I. scapularis *infected with* Borrelia burgdorferi *versus the area of forest fragments (*$R^2 = 0.43$, $p < 0.01$, $n = 14$*); and (c) density of nymphal* I. scapularis *infected with* B. burgdorferi *versus the area of forest fragments (*$R^2 = 0.65$, $p < 0.01$, $n = 14$*).*

DOL on NIP revealed that DOL had no significant effect ($p = 0.81$), whereas NIP was a negative linear function of patch area ($p = 0.01$; model $R^2 = 0.43$).

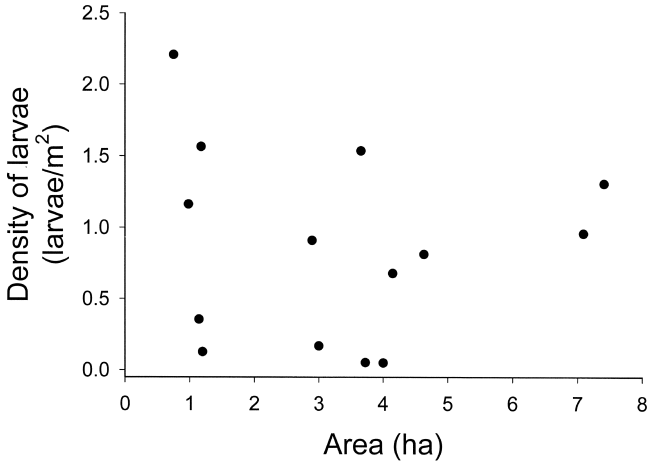

Figure 2. Density of larval Ixodes scapularis *versus the area of forest fragments in a highly fragmented landscape of Dutchess County, New York (*$R^2 = 0.01$, $p = 0.78$, $n = 14$*).*

Discussion

In this highly fragmented landscape, the density of nymphal blacklegged ticks, the prevalence of nymphal infection, and their product, the density of infected nymphs, were inversely correlated with forest patch area. These metrics of the nymphal tick population are ecological indicators of the risk of Lyme disease to the human population that enters tick habitat.

The elevated risk of exposure to Lyme disease in the smallest patches appears to result from two interrelated phenomena. First, forest fragmentation results in the loss of many vertebrate species from the remaining small forest patches (Blake & Karr 1987; Rosenblatt et al. 1999). White-footed mice, however, do not appear to be adversely affected by forest fragmentation (Nupp & Swihart 1996), so the relative abundance of white-footed mice is higher in small patches. Second, because the vertebrate species lost from the smallest forest fragments tend to be predators on and competitors with white-footed mice (Rosenblatt et al. 1999; Schmidt & Ostfeld 2001), regulation of *P. leucopus* may be weaker in smaller patches, resulting in the high absolute densities of mice observed in several studies (Nupp & Swihart 1996, 1998; Krohne & Hoch 1999; Rosenblatt et al. 1999). These two pathways combine to increase the fraction of tick meals that are taken from white-footed mice, the most competent reservoir of Lyme bacteria, and to decrease the fraction of tick meals taken from poor reservoirs. Therefore, the loss of vertebrate species from small forest fragments increases the NIP (Ostfeld & Keesing 2000*a*).

The infection prevalence of nymphs from the forest fragments, especially from the smallest ones, were higher than those determined (using the same immuno-

fluorescence technique) for nymphal tick populations inhabiting more-continuous forest at a nearby site (Ostfeld et al. 2001). In fact, we are not aware of any published values for the prevalence of *B. burgdorferi* infection of nymphal *Ixodes* ticks higher than those observed in this study. Such a result would be expected if larvae had access to dense populations of white-footed mice.

In addition to being more likely to acquire the Lyme bacterium, larval ticks that feed on white-footed mice are more likely to successfully molt into nymphs than are larvae that feed on non-mouse hosts (Davidar et al. 1989; James & Oliver 1990; Mannelli et al. 1993; Mather & Ginsberg 1994; RSO, unpublished data). Consequently, small patches should have elevated DON as well. Because both NIP and DON increase in small fragments, their product, DIN, increases as well.

Although the reduction in NIP with increasing fragment size was linear (Fig. 1b), the decreases in DON and DIN with increasing fragment size resembled an exponential decay (Fig. 1a & 1c). Both DON and DIN were substantially higher in four of the five smallest patches than in any of the larger ones. The shape of the exponential decay curve suggests that forest patches <2 ha in area present an elevated risk of Lyme disease, but that beyond this size risk is less sensitive to variation in patch area. Given the relatively few patches examined and the limited range of patch size, however, the shape of the risk-decay curve must be interpreted with caution. Moreover, elevated values of DON, NIP, and DIN are sometimes observed in more continuous, oak-dominated forest after years of heavy acorn production that lead to population explosions of white-footed mice (Ostfeld et al. 2001).

The density of nymphs (DON) should be a function of both the survival of the larval cohort to the nymphal stage and the density of the prior year's larvae (DOL), a process we call demographic forcing. (We were unable to assess the prior year's DOL, but instead determined the current year's DOL under the assumption that the relative magnitude of DOL remains approximately constant from year to year in maple-dominated forests.) In fact, DON was a significant positive function of DOL in our forest patches. Because of the specialization by adult blacklegged ticks on white-tailed deer, DOL, in turn, is correlated with the abundance of deer (Wilson et al. 1985, 1988). Because DOL was not correlated with patch area in our analysis, we suggest that factors other than forest patch area determine the distribution of white-tailed deer. Preliminary data indicate that larvae can reach high densities in continuously forested areas (B. F. Allan et al., unpublished data) and that the pattern we observed for fragments may not exist within areas of more continuous forest cover. Therefore, we hypothesize that matrix quality, patch quality, or interpatch distance are of greater importance than patch area in determining the abundance of deer and therefore DOL. Such a possibility further suggests caution in extending our results to other landscapes, in which matrix quality and deer abundance may vary.

The incidence of Lyme disease is particularly high in regions where dense human habitation is juxtaposed with forest habitat that supports tick vectors and their hosts (Barbour & Fish 1993). Our results suggest that efforts to reduce the risk of Lyme disease should be directed toward decreasing fragmentation of the deciduous forests of the northeastern United States into small patches, particularly in areas with a high incidence of Lyme disease. The creation of forest fragments of <1–2 ha should especially be avoided, given that these patches are particularly prone to high densities of white-footed mice, low diversity of vertebrate hosts, and thus higher densities of infected nymphal blacklegged ticks.

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