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12. We performed molecular modeling using MACROMODEL 5.5 (Amber* force field): F. Mohamadi *et al*., *J. Comput. Chem.* **11**, 440 (1990). The volumes for the guests and the cavities were obtained from the GRASP program [A. Nicholls, K. A. Sharp, B. Honig, *Proteins* **11**, 281 (1991)].
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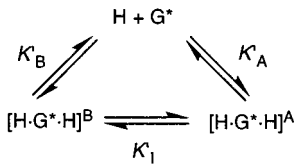
14. We obtained all measurements by ^{1}H NMR experiments using the integrals for the peaks of the guest inside and outside the capsules. There is an estimated 10% error in these measurements. The equilibrium may be described as follows:



The following assumptions were made: (i) the amount of dimer (unfilled or filled with solvent) present before addition of the guest is negligible; (ii) after addition of the guest, all of the host material not assembled into the capsule is in the aggregate state; and (iii) the association of the guest with itself is negligible.

$$K'_A = \frac{[H{\cdot}G^*{\cdot}H]^A}{[H][G^*]} = \frac{aV}{[h - 2(a + b)][g - (a + b)]} \quad (1)$$

$$K'_B = \frac{[H{\cdot}G^*{\cdot}H]^B}{[H][G^*]} = \frac{bV}{[h - 2(a + b)][g - (a + b)]} \quad (2)$$

$$K'_I = \frac{[H{\cdot}G^*{\cdot}H]^A}{[H{\cdot}G^*{\cdot}H]^B} = \frac{K'_A}{K'_B} \quad (3)$$

$$\Delta(\Delta G^0) = -RT \ln K'_I \quad (4)$$

$$a = g(I_{gA}/I_{gT}) \quad (5)$$

$$b = g(I_{gB}/I_{gT}) \quad (6)$$

$$I_{gT} = I_{gO} + I_{gA} + I_{gB} \quad (7)$$

where K'_A and K'_B are the apparent association constants for the predominant and the subordinate complexes, respectively, and K'_I is the apparent isomerization constant between the two complexes. In these equations H is the host; G* is the chiral guest; $[H{\cdot}G^*{\cdot}H]^A$ and $[H{\cdot}G^*{\cdot}H]^B$ are the concentrations of the predominant and the subordinate complexes, respectively: ΔG^0 is the free energy of formation; T is temperature; and R is the ideal gas constant; I_{gT} is the sum of all the integrals corresponding to the guest (subscript T stands for total); I_{gO} is the integral for the signal of the guest outside the capsule, I_{gA} is the integral for the signal of the guest in complex A; I_{gB} is the integral for the signal of the guest in complex B; h is the initial amount of monomer (in millimoles); g is the amount (in millimoles) of guest added to the solution; a is the amount of guest (in millimoles) in complex A; b is the amount of guest (in millimoles) in complex B; and V is the total volume (in milliliters).

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16. This research was supported by the Skaggs Research Foundation and NIH. We thank U. Obst for advice on molecular dynamics studies. Administración de Fomento Económico of Puerto Rico provided fellowship support to J.M.R. The Ministerio de Educación y Cultura of Spain provided fellowship support to T.M.

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Chain Reactions Linking Acorns to Gypsy Moth Outbreaks and Lyme Disease Risk

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In eastern U.S. oak forests, defoliation by gypsy moths and the risk of Lyme disease are determined by interactions among acorns, white-footed mice, moths, deer, and ticks. Experimental removal of mice, which eat moth pupae, demonstrated that moth outbreaks are caused by reductions in mouse density that occur when there are no acorns. Experimental acorn addition increased mouse density. Acorn addition also increased densities of black-legged ticks, evidently by attracting deer, which are key tick hosts. Mice are primarily responsible for infecting ticks with the Lyme disease agent. The results have important implications for predicting and managing forest health and human health.

Oak trees (*Quercus* spp.) produce large autumnal acorn crops (masting) every 2 to 5 years, producing few or no acorns during intervening years (*1*–*4*). Acorns are a critical food for white-footed mice, *Peromyscus leucopus* (*1*, *4*–*6*). Mice are important predators of pupae of the gypsy moth, *Lymantria dispar* (*1*, *6*–*10*). This introduced insect periodically undergoes outbreaks (*11*, *12*) that defoliate millions of hectares of oak forests, decreasing tree growth, survival, and mast production (*13*). An abundance of acorns draws white-tailed deer, *Odocoileus virginianus*, into oak forests (*14*, *15*). Mice and deer are the primary hosts of the black-legged tick, *Ixodes scapularis*, which is the vector of spirochete bacteria (*Borrelia burgdorferi*) that cause Lyme disease in humans (*16*–*18*). Here we report the results of experimental removal of mice and addition of acorns, which demonstrate how acorn production is connected to gypsy moth outbreaks and Lyme disease risk.

Masting is associated with increased survival and breeding of mice in winter and spring (*19*), with peak densities occurring the following midsummer (*1*, *4*, *6*). High mouse density correlates with high predation rates on moth pupae (*1*, *6*), which may prevent low-density moth populations from increasing (*1*, *6*–*8*). Conversely, mast crop failure correlates with low mouse densities and low rates of pupal predation the following summer (*1*, *4*, *6*), which may initiate moth outbreaks (*7*, *9*).

Moth populations at our research site reached peak densities in 1990, declined by four orders of magnitude to 0.2 egg masses ha^{-1} by 1992, and remained between 6 and 38 egg masses ha^{-1} in 1993–1994 (*1*). A large red oak (*Q. rubra*) acorn crop in autumn 1994 led to high mouse densities in summer 1995 (*1*). We took advantage of low moth and high mouse densities to remove mice during moth pupation, testing the chain of interactions linking acorns to mice to moths. Mice were removed from three grids of approximately 2.7 ha but were left unmanipulated on three control grids (*20*). Mouse densities did not differ between control and experimental grids in June 1995, just before mouse removal (Fig. 1; $P = 0.18$, paired t test) (*21*). Continuous live trapping reduced mouse densities on experimental grids to less than half those on control grids by the midpoint of a 32-day removal period in June–July coincident with female moth pupation (Fig. 1; $P =$

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0.018, one-tailed unpaired *t* test).

Densities of late-stage moth larvae (*22*) did not differ between treatments at the start of the experiment (Fig. 2A). Predation on female pupae was estimated by monitoring survival of the native population and by recording attacks on freeze-dried pupae (*23*). On control grids with high mouse densities, no living female pupae were found, and 100% of freeze-dried pupae were attacked by predators in 2 to 4 days, which is much less than the 13 days required for eclosion to the adult stage. Over 99% of attacks on freeze-dried pupae that could be attributed to vertebrates or invertebrates were caused by vertebrates, and 97% of vertebrate attacks where the predator species was identifiable were made by mice. In contrast, on experimental grids, 42% of native female pupae survived for 13 or more days, and 22% of freeze-dried pupae were unattacked at 14 days; 77% of these attacks were caused by vertebrates, with 89% being mouse attacks. The number of successfully eclosed female pupae and resulting egg masses on trees (*24*) was respectively 45-fold (Fig. 2B) and 43-fold higher (Fig. 2C) on experimental than on control grids. Comparison of control grids in 1995 and 1994 showed that oak masting in 1994 led to a 15-fold increase in July mouse densities, a 34-fold increase in mouse predation on freeze-dried pupae, and a decrease by a factor of 26 in moth egg mass densities (*25*).

The increase in moth density that resulted from simulating mast failure by removing mice was similar in magnitude to that observed at the start of natural moth outbreaks, and the decrease in moth density on control grids was similar in magnitude to that previously observed after masting-induced increases in mouse density (*1*, *6*).

Lyme disease in the northeastern and north central United States is transmitted to humans by black-legged ticks infected with *B. burgdorferi* (*16*, *26*). Adult ticks feed and mate on white-tailed deer before dropping to the ground in autumn, laying eggs the following spring or early summer (*17*, *27*). Larvae hatch in midsummer and are free from infection with *B. burgdorferi* because of extremely low rates of transovarial transmission (*28*). White-footed mice are primarily responsible for infecting ticks with *B. burgdorferi* during the larval blood meal (*29*, *30*). Larvae then molt to nymphs that overwinter on the forest floor. In spring or early summer 1 year after egg hatch, infected nymphs seek vertebrate hosts, including humans, and may transmit *B. burgdorferi* to the host at this blood meal (*16*–*17*). The abundance of infected nymphs is the primary determinant of Lyme disease risk (*16*). Nymphs molt into adults that seek a deer host in the autumn. The location of deer in autumn determines the location of egg-laying adults and thus where host-seeking larvae should occur the following summer (*1*, *31*–*32*).

In the autumn of mast years, deer spend more than 40% of their time in oak stands feeding on acorns but spend less than 5% of their time there in non-mast years (*15*). Larval tick density in oak forests reaches peak levels the summer after mast production but is low during the summer after mast failure (*1*), corresponding to predictions based on habitat use by deer. Increased densities of mice in oak forests during the summer after masting coincide with peak densities of larval ticks (*1*). Because mice are the principal reservoirs for Lyme disease spirochetes, high densities of infected nymphal ticks and a high risk of exposure to Lyme disease should occur 2 years after heavy acorn production (*32*).

We took advantage of mast crop failure in the autumn of 1995, when acorn production was lower by a factor of 18 than in 1994, to add acorns to the three experimental grids but not to the three control grids (*33*), testing the chain of interactions linking acorns to mice, deer, and ticks. We added more than 811,000 acorns (>3500 kg) to experimental grids at densities of 60 m^{-2} of oak canopy, approximating the 1994 acorn crop. We also simulated food caching by periodically supplementing mouse nest boxes on experimental grids with acorns, leaving boxes on control grids unsupplemented. Mouse density and reproductive status were monitored, and each month we measured the numbers of host-seeking ticks and ticks infesting mice (*34*). Although mice had been removed from the experimental grids in June–July, densities had returned to the levels measured on control grids by early October 1995, before acorn additions (Fig. 1; $P = 0.98$, unpaired *t* test).

Acorn addition significantly increased mouse densities from March–August 1996

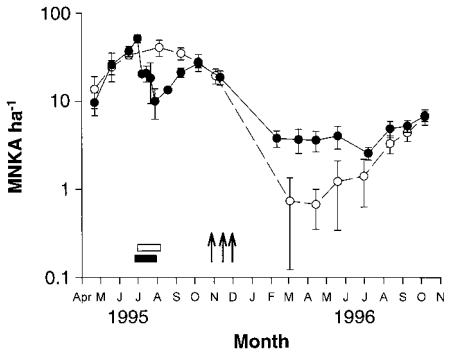

Fig. 1. White-footed mouse densities on three control (○) and three experimental (●) grids as mean (±SE) MNKA ha^{-1}, April 1995–October 1996, showing (i) 1995 densities before, during, and after the period of mouse removal from experimental grids (black bar) at the time of native female gypsy moth pupation (white bar); (ii) 1995 densities before and during acorn additions (arrows) to experimental grids, October–November 1995, when there were very low numbers of acorns produced on control grids; and (iii) 1996 densities after acorn additions to experimental grids in 1995. High 1995 mouse densities were associated with autumn 1994 masting, and mouse densities typically decline during winter (*1*).

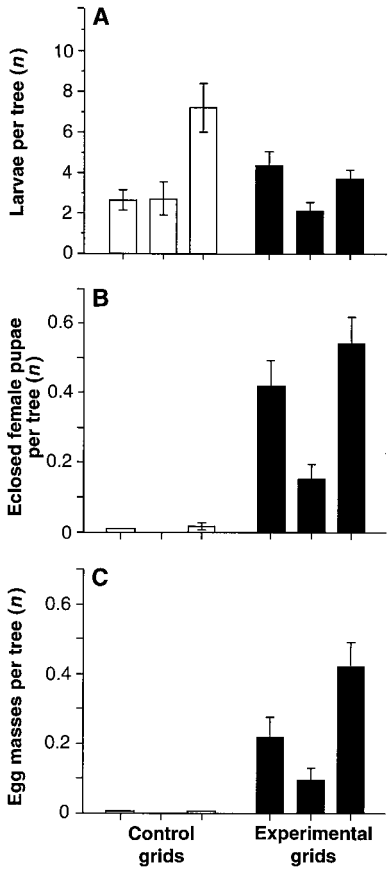

Fig. 2. Densities of gypsy moth life stages on or under burlap bands on trees on control grids (open bars) versus experimental grids (solid bars) where mice were removed. The bars show grid means and within-grid SEs. Across-grid control and experimental means (±SE) and statistical comparisons are also shown for each graph. (**A**) Number of living late-stage larvae per tree just before mouse removal. Control grids, 4.17 (±1.51); experimental grids, 3.36 (±0.68); $P = 0.83$, Mann-Whitney *U* test. (**B**) Number of female pupae per tree successfully eclosing to adults after mouse removal. Control grids, 0.008 (±0.005); experimental grids, 0.370 (±0.115); $P = 0.02$, one-tailed Mann-Whitney *U* test. (**C**) Number of egg masses per tree after mouse removal. Control grids, 0.006 (±0.003); experimental grids, 0.245 (±0.095); $P = 0.02$, one-tailed Mann-Whitney *U* test.

(Fig. 1; $P = 0.032$, one-tailed F test), with approximately three- to sevenfold greater densities on experimental grids than on control grids in March–May. Densities converged on control values after August. From February to May, 75% of the adult mice on experimental grids ($n = 72$) were in breeding condition versus 59% ($n = 17$) on control grids ($P = 0.09$, one-tailed χ^2 test). Although the low numbers of mice on control grids limited our ability to detect reproductive differences, the increase in mouse density caused by acorn addition was evidently mediated by both enhanced survival and reproduction. Our small-scale acorn additions may have had less effect on mouse populations than do natural masting events that typically occur over thousands of hectares (*2*, *3*). For example, predators could have been attracted to the locally elevated mouse densities (*35*) or mice could have emigrated to surrounding areas with lower mouse densities (*32*).

Densities of host-seeking ticks in August 1996, the time of peak larval host-seeking activity, were over eight times higher on acorn addition grids than on control grids (Fig. 3A). Although deer habitat use was not directly monitored, larval tick distribution is largely determined by deer distribution the previous autumn (*1*). Consequently, adding acorns would have increased the time deer spent in autumn feeding on acorns in experimental grids as compared with control grids. The number of attached larval ticks per mouse was 40% higher on acorn-addition grids as compared with control grids (Fig. 3B). Adding acorns not only increased the densities of mice up to 9 months later but also increased larval tick burdens on mice because of the effects of acorn additions on deer habitat use and the effects of deer habitat use on the abundance of host-seeking larval ticks (*36*).

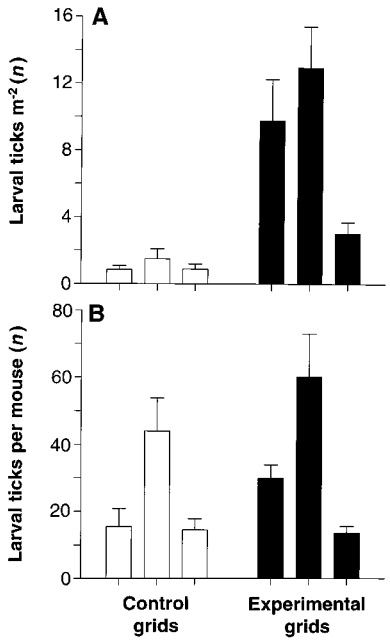

Fig. 3. Tick densities on control grids (open bars) and experimental grids (solid bars) in August 1996 after acorn additions to experimental grids in October–November 1995. The bars show grid means and within-grid SEs. Across-grid control and experimental means (±SE) and statistical comparisons are also shown for each graph. (**A**) Number of host-seeking larval ticks per square meter. Control grids, 1.07 (±0.21); experimental grids, 8.59 (±2.93); $P = 0.017$, one-tailed paired t test. (**B**) Number of larval ticks per mouse. Control grids, 24.67 (±9.67); experimental grids, 34.58 (±13.64); $P = 0.046$, one-tailed paired t test.

Our results provide strong support for the idea that a chain of events links acorns to gypsy moth outbreaks and Lyme disease risk. The experiments demonstrate first that acorns determine overwinter survival, reproduction, and the resulting density of mice. Second, that high or low mouse density, at low gypsy moth population density, can respectively suppress or release moth populations through altered pupal predation. Third, that acorns determine larval tick densities by effecting the use of oak forests by deer, resulting in high densities of both host-seeking uninfected ticks and ticks parasitizing mice at the time when spirochete-infected mice are most abundant.

It may be feasible to predict the risk of contracting Lyme disease from infected nymphal ticks in oak forests on the basis of masting events, with the risk being greatest 2 years after an abundant acorn crop. Similarly, suppression or initiation of moth outbreaks may be predictable from mast production or failure when moth populations are at low densities. However, because other mortality agents, not mice, appear to control moth populations at higher densities (*12*, *37*), outbreak initiation by mast failure and the collapse of mouse populations is probably necessary, but may not always be sufficient, to cause moth populations to rise to levels that cause defoliation. An additional, important long-term feedback to Lyme disease may exist, because moth defoliation reduces acorn production and can reduce oak abundance in forests (*13*). Our studies indicate that attempting to simultaneously prevent moth outbreaks and minimize Lyme disease risk, by using silvicultural practices that alter acorn production, would be unlikely to succeed because decreasing the likelihood of moth outbreaks could increase the risk of Lyme disease and vice versa.

Ecologists have hotly debated the relative importance of direct versus indirect species interactions as a cause of contingent ecological outcomes (*38*). Our studies clearly demonstrate that both gypsy moth dynamics and Lyme disease risk have contingent outcomes arising from a complex chain of strong pairwise interactions among taxonomically diverse species that are all interconnected within an ecosystem.

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20. Three pairs of open grids (165 by 165 m and 180 by 110 m for one grid) 100 to 250 m apart, with pairs separated by 1 to 3 km, were located in upland oak forests (57 to 70% oak relative basal area) at the Institute of Ecosystem Studies. Grids were 11-by-11 arrays (12 by 10 for one grid) of trap stations 15 m apart, with two Sherman live traps per station at the centers of 15-by-15-m grid cells used for gypsy moth sampling. A wooden nest box, containing cotton batting nesting material replaced twice a year, was attached at 1.3 m to a large tree [26 to 58 cm in diameter at breast height (DBH)] in each of 20 grid cells; boxes were stratified to maximize intercell distances. These nest boxes have no effect on mouse densities [J. O. Wolff and D. S. Durr, *J. Mammal.* **67**, 409 (1986); D. T. Krohne, J. F. Merritt, S. H. Vessey, J. O. Wolff, *Can. J. Zool.* **66**, 2170 (1988)] and were colonized by mice only. Mouse densities, measured as minimum number known alive (MNKA) per hectare, were estimated from (i) monthly mark-recapture over 2-day periods from the 120 or 121 trap stations on all grids, April–November 1995 and 1996, and July 1994 for two control grids; (ii) trapping of new and recaptured

animals during the removal period every 2 to 4 days on experimental grids, 26 June–30 July 1995; and (iii) monthly mark-recapture from nest boxes, February–April 1996. Traps were baited daily with oats, set 2 to 3 hours before dusk, and checked the following two mornings. Traps were not operated during the day or in December–March to avoid risks of overheating or hypothermia of animals. During mark-recapture, all mice were given numbered metal eartags at first capture. Trap station or nest box, tag number, gender, body mass, and age and reproductive condition (for females, vaginal patency and evidence of pregnancy and lactation; for males, whether testes were descended) were recorded at all captures, and animals were released at the point of capture. During the removal period, the same variables were recorded, but tagged and untagged animals were moved to a site 4.5 km away. One hundred forty, 230, and 239 mice were removed from the three experimental grids, respectively. None of the tagged relocated animals were recaptured on grids. Care of animals was in accordance with institutional guidelines.

21. Unless otherwise noted, response variables were examined for grid pair effects, using an appropriate parametric or nonparametric comparison, followed by treatment comparisons using paired tests if $P < 0.05$ for grid pair effects and unpaired tests if $P > 0.05$ for grid pair effects. Where one-tailed treatment comparisons were used because there were a priori hypotheses, this is reported; otherwise, comparisons were two-tailed. A single time point was used to compare densities before mouse removal; interpolated densities were used for comparisons at the midpoint of the mouse removal period. Mouse densities were ln-transformed before grid effect analyses of variance (ANOVAs) and *t* tests.
22. Two hundred forty to 242 trees, greater than 7 cm DBH and greater than 2 m in height, without nest boxes, one tree pair per grid cell, were used for moth sampling on trees. Half the trees were banded with slitted, folded, burlap skirts (30 cm) tied at 1.3 m. One-third of all tree pairs were oak-oak, one-third were oak–non-oak, and one-third were non-oak–non-oak. Banded and unbanded tree pairs were alternated across each grid. Late-stage larvae use burlap bands as daytime refuges (*11*), and their development was monitored until fourth and fifth instars were prevalent. The number of living larvae of all instars on or under burlap bands on all banded trees was counted during dry days between 8 and 15 June and was expressed as the mean number of larvae per tree, with a tree-to-tree within-grid variance estimate. Larval, pupal, and most egg mass density data were analyzed by Kruskal-Wallis ANOVA by ranks and showed no grid pair effects ($P > 0.05$). Treatment effects were analyzed with Mann-Whitney *U* tests unless otherwise noted.
23. Pupation, eclosion, mating, and egg-laying occur in the daytime resting locations of late-stage larvae, and the number of flightless females determines egg mass density. Female pupae took a mean (±SE) of 12.7 (±0.4) days ($n = 42$) to develop to adults, before immediately mating and laying egg masses. The time taken for female pupae to eclose was determined by monitoring individuals every other day from 30 June to 7 August on grid cells with banded oak-oak tree pairs ($n = 34$ monitored survivors). Because no monitored pupae survived on control grids, additional female (sixth-instar) larvae ($n = 8$) were enclosed in small wire mesh cages as they began to pupate, to prevent predator access. Caged adults and egg masses were removed and are excluded from density estimates. The fate of individual native female pupae on a given date (uneclosed or eclosed, based on characteristic splitting and adult emergence holes), and characteristic signs of mortality agents on attacked pupae (predation, insect parasitism, fungal hyphae, and other or unknown), were recorded every other day on banded oak-oak tree pairs. Predation on freeze-dried female pupae was measured with a technique modified from Smith (*10*). Mass-reared female pupae (USDA Animal and Plant Health Inspection Service, MA) were freeze-dried and affixed with beeswax, which retains an imprint of the species-specific tooth marks of vertebrate predators, in groups of five onto burlap panels 20 by 15 cm. Twenty or 21 panels per grid were stapled under the bands of one of the pair of oak-oak banded trees on 10 July. Pupae were monitored daily to day 9 (except day 6) and then every other day to day 18 or until all pupae had been attacked. Judging by tooth marks and damage characteristics, we recorded attacks as being due to vertebrates, invertebrates, both, or an unknown agent. (*10*). The total number of successfully eclosing pupae was determined from counts on all banded trees at the end of the eclosion period and was expressed as the mean number per tree, with a tree-to-tree within-grid variance estimate.
24. New egg masses on or under burlap bands were counted on all banded trees and expressed as the mean number per tree, with a tree-to-tree within-grid variance estimate. Twelve randomly selected 15-by-15-m grid cells per grid were also censused for egg masses on or under bands, at heights less than 2 m on banded and unbanded trees, on small saplings, dead trees, woody debris, litter, and rocks. Firm new egg masses were distinguished from spongy old egg masses with visibly hatched eggs by gentle pressing. Mean (±SE) new egg mass densities per grid cell were 0.028 (±0.028) on control grids and 0.305 (±0.121) on experimental grids ($P = 0.036$, one-tailed Mann-Whitney *U* test). Most egg masses are laid at heights less than 2 m in low-density moth populations [P. Skaller, *Environ. Entomol.* **14**, 106 (1985)].
25. Only two control grids were fully operational in 1994, limiting the statistical power of pairwise comparisons between 1994 and 1995. Mean (±SE) mouse densities, MNKA ha^{-1}, were 2.59 (±0.36) in July 1994 and 38.81 (±9.24) in July 1995 (one-tailed paired *t* test on ln-transformed data, $P = 0.045$). Mice attacked a mean (±SE) of 1.06 (±0.44) freeze-dried pupae per day in 1994 and 36 (±14) in 1995 (one-tailed paired *t* test on ln-transformed data, $P = 0.003$). Mean (±SE) egg mass densities per tree on or under burlap bands were 0.11 (±0.02) in 1994 and 0.004 (±0.004) in 1995 (one-tailed paired *t* test, $P = 0.0862$).
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33. Acorn production (mostly red oak) on control grids in 1995 versus 1994 averaged 7.6 (range 7.2 to 8) versus 97 (range 59 to 134) acorns per square meter of canopy (estimated from 0.5-m^2 seed traps, of which five per grid were under canopy oaks), from an average of 98 versus 142 masting oaks per grid, based on canopy inspection. The acorn density added was the average of 1994 values and that found by Sork *et al.* (*3*) for red oak (25 m^{-2} of canopy). To calculate the amount of acorns to add per grid, we used the mean DBH of masting oaks in 1994 to calculate average crown radius (in meters) {$1.71 + 7.92 \times$ DBH [C. D. Canham, A. C. Finzi, S. W. Pacala, D. H. Burbank, *Can. J. For. Res.* **24**, 337 (1994)]} and the average number of oaks masting in 1994. Based on these calculations, we added an average of 270,525 (1172 kg) acorns [primarily red oak, locally collected and donated as well as purchased (Sheffield Seed Co., Locke, NY)] to each of the three experimental grids. Acorns were added in three events between late October and late November 1995 and were evenly scattered in a 4.45-m average crown radius circle around the center of each of the inner 80 grid cells. The 20 nest boxes on each experimental grid received acorn supplements of 10 per box every month from mid-September to December 1995; then 5 per box every 2 weeks to early June 1996.
34. Ln-transformed mouse density before acorn addition was analyzed at a single time point (grid effect ANOVA, *t* test). In calculating the effects of acorn additions on mouse densities (ln-transformed) we used repeated measures ANOVA (rmANOVA) to test for grid pair effects ($P > 0.05$; rmANOVA Wilkes' Lambda for time and treatment by time interaction effects, $P > 0.05$; *F* test for treatment effect). Sample sizes for mouse breeding frequencies on control grids (analyzed by χ^2) precluded analysis of grid pair effects. Tick densities were sampled monthly on all grids, April-November 1996, by dragging a 1-m^2 white corduroy cloth, supported by a wooden dowel at one end and lead weights at the other end [R. C. Falco and D. Fish, *Exp. Appl. Acarol.* **14**, 165 (1992)] along 15-by-30-m transect segments per grid, with transects randomly selected at each sample time. Every 30 m, ticks were removed and preserved in 70% ethanol for identification. Mean larval tick density (ticks per square meter), averaged over transect segments with a segment-to-segment within-grid variance estimate, were ln-transformed (grid effect ANOVA, *t* test). The numbers of larval ticks attached to live-trapped mice were counted at each trap session. Mean larval ticks per mouse, averaged over each grid, with differences among mice giving the within-grid variance estimate, were ln-transformed (grid effect ANOVA, *t* test). Data for August 1996 are reported because this is the peak period of host-seeking activity by larval ticks, as determined by 7 years of monitoring data (*30, 31*).
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36. The abundance of infected host-seeking nymphal ticks is the primary determinant of Lyme disease risk (*15*). Because acorn addition caused higher densities of host-seeking larval ticks, higher densities of ticks on spirochete-infected mice, and higher densities of mice, Lyme disease risk is expected to be substantially increased 2 years after masting. This inference was supported by our observation that the average density of nymphs in June 1997 was 73.9% higher and in July was 31.3% higher on experimental (acorn-supplemented) grids than on control grids. However, because of high variability among sites, a drought causing small sample sizes of nymphs, and possible density-dependent emigration by mice from our experimental grids during the 1996 period of larval infestation, neither of these differences was statistically significant (paired *t* tests, $P = 0.21$ and 0.15 for June and July, respectively).
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