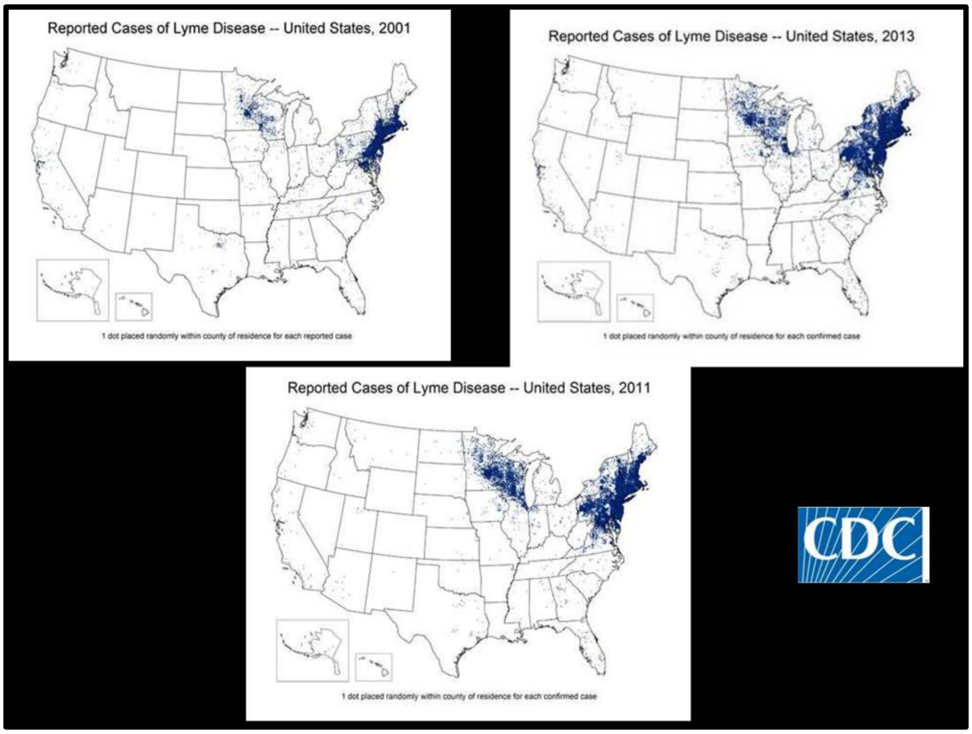
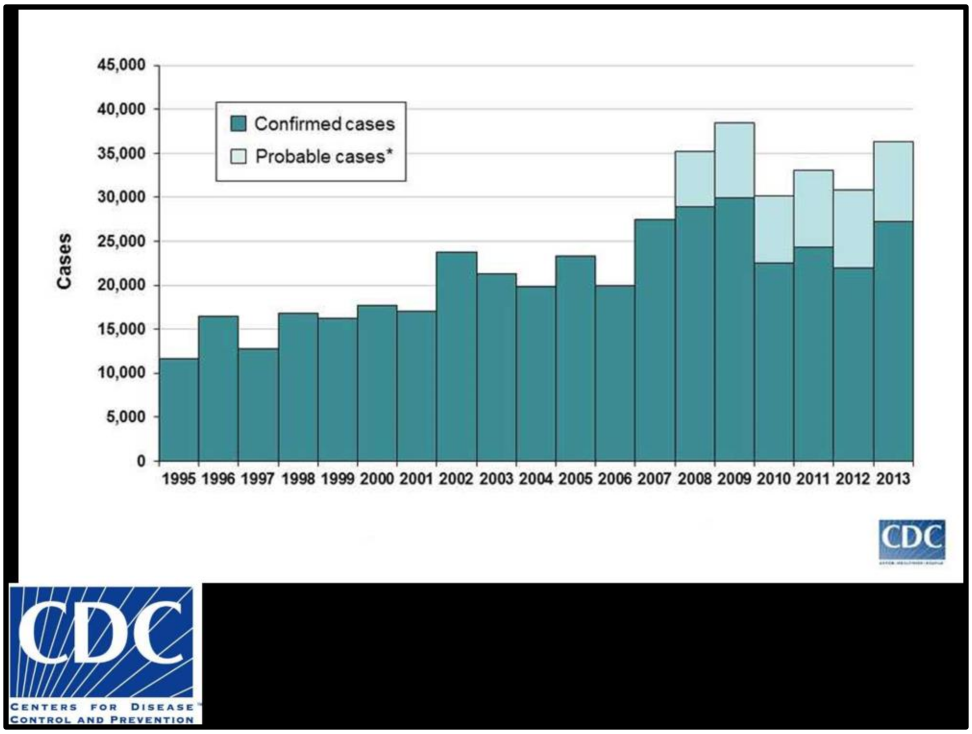
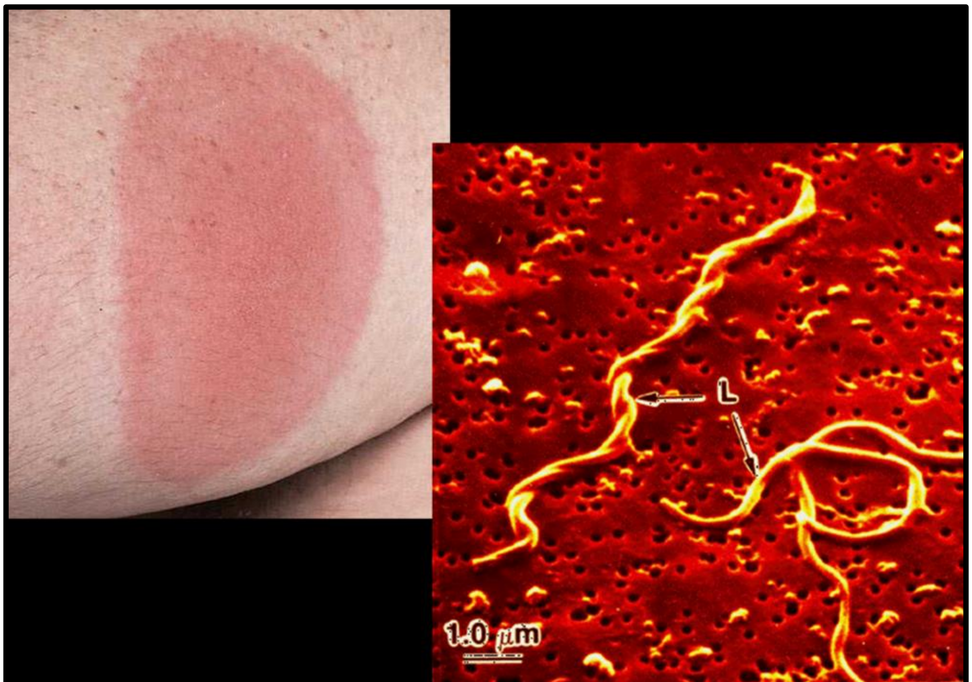


AN APPLIED CASE STUDY of the complexity of ecological systems and process: Why has Lyme disease become an epidemic in the northeastern U.S. over the last few decades? Where did it come from? What causes Lyme disease?



Frequency of DIAGNOSED Lyme disease cases have increased several-fold in the last couple of decades – an *apparent* outbreak or epidemic – an 'emerging pathogen'. Many cases certainly remain unreported; in the last few years, the CDC has used various means to estimate true numbers, but some researchers believe these estimates are still much too low.

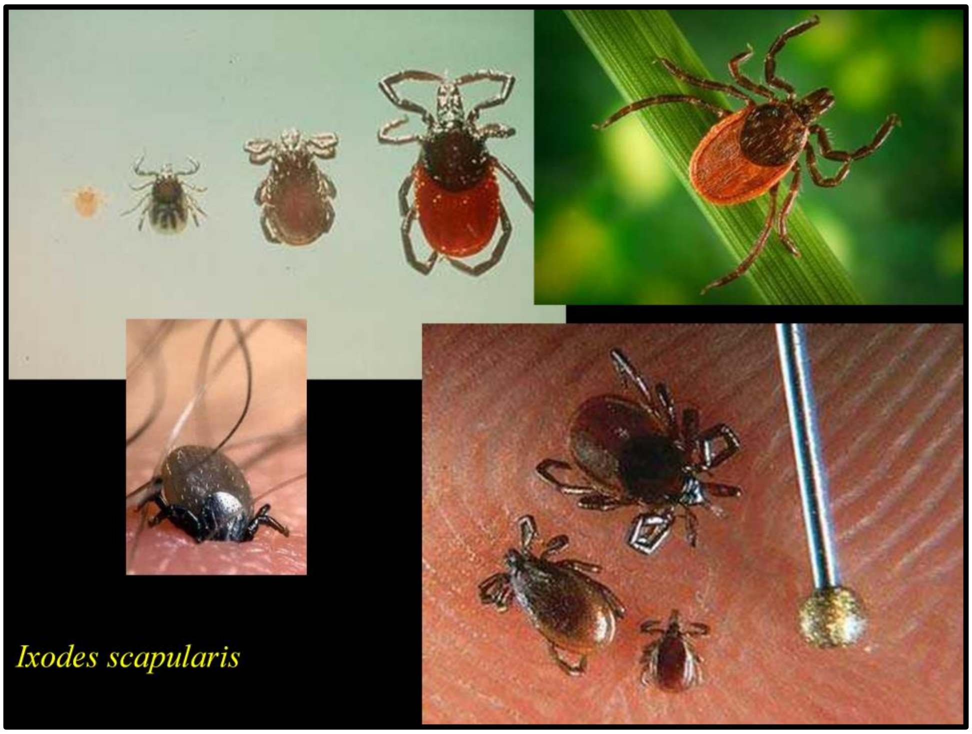
Public health concerns have become wide-spread



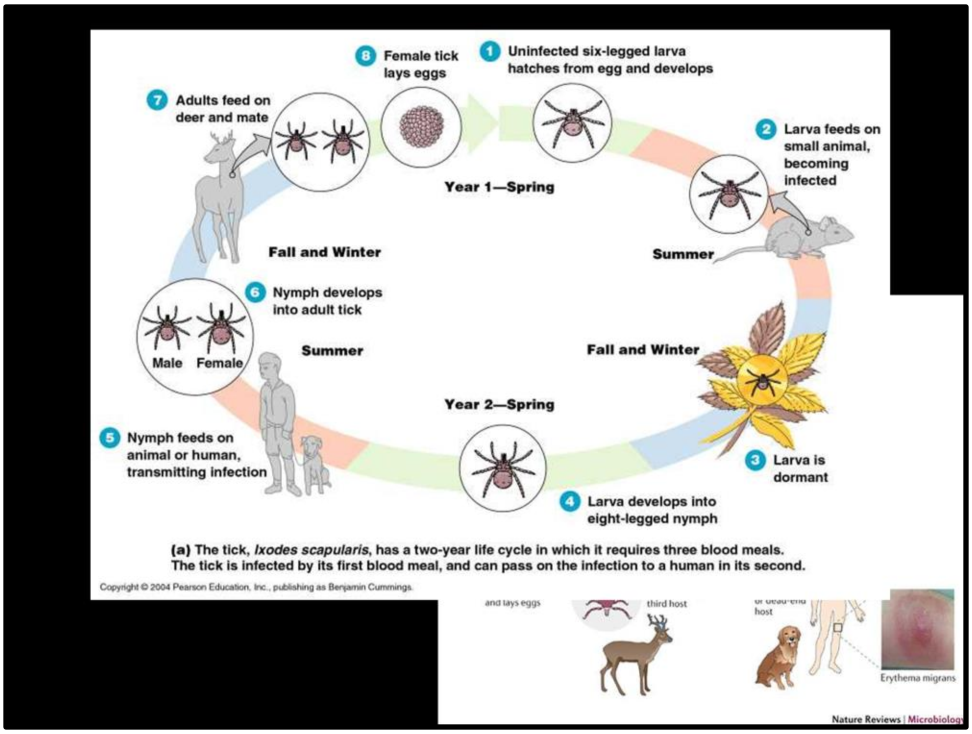
Borrelia burgdorferi

Proximal causation: The *disease* – the symptoms we experience under the name 'Lyme disease' – is induced by infection with a spirochaete bacterium (in the same group as the bacterium that causes syphilis). The proper name of the disease is borreliosis. But a 'disease' is just a collection of symptoms...

Recent genetic analyses show that the Ötzi the Iceman – a mummy preserved beneath a glacier in the European Alps for about 5300 years carried *Borrelia*. Further genetic analyses of *Borrelia* itself suggest that the species originated in Europe and is quite ancient. It is not, as had been supposed, a 'new' pathogen from North America (although it may have evolved greater virulence recently here).



Causality at one remove: The bacterium is *vector-transmitted* – it must be transferred between hosts via a bite by the deer tick *Ixodes scapularis*. Ticks must, in turn, acquire the bacterium from a previous hosts. Ticks hatch as larvae without infection; in order to pass to the next life-cycle stage – the nymph – larvae must successfully gorge on blood from a mammal or bird host. Ticks seeking a host ('questing') perch on grass, twigs or weeds and simply wait; when a suitable host brushes past, they quickly release and attach to the host. They can wait for months. Nymphs must have a blood meal to molt and become adults. Adult females must have a blood meal to reproduce. If the tick acquires *Borrelia* from its first or second host, it can transfer it to the next host.



The tick's life-cycle has four stages; egg, larva, nymph, and (reproductive) adult. The life-cycle is typically extended over two years. Ticks can pick up the bacterium in year one and infect a new host in second year.

White-footed mice or deer mice (genus *Peromyscus*) are the most common and ubiquitous mammal in eastern woodlands and forests. They are also the ‘competent reservoir’ of *Borrelia*;, meaning that the bacterium can persist and thrive in the mouse population from which it can be transferred to other species. A large proportion of deer mice are infected with the bacterium, which doesn’t appear to cause disease symptoms in the mice, and it is readily transferred to ticks feeding on the mice. Deer mice cause Lyme disease… But what regulates the abundance of deer mice (and so the likelihood of a tick using a deer mouse as one of its first hosts)? What do deer mice eat?

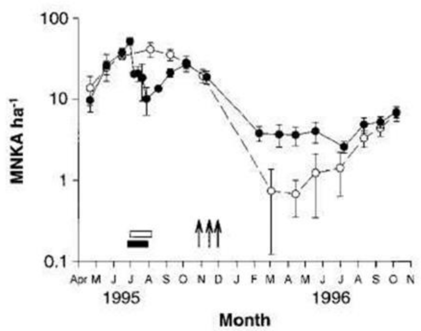
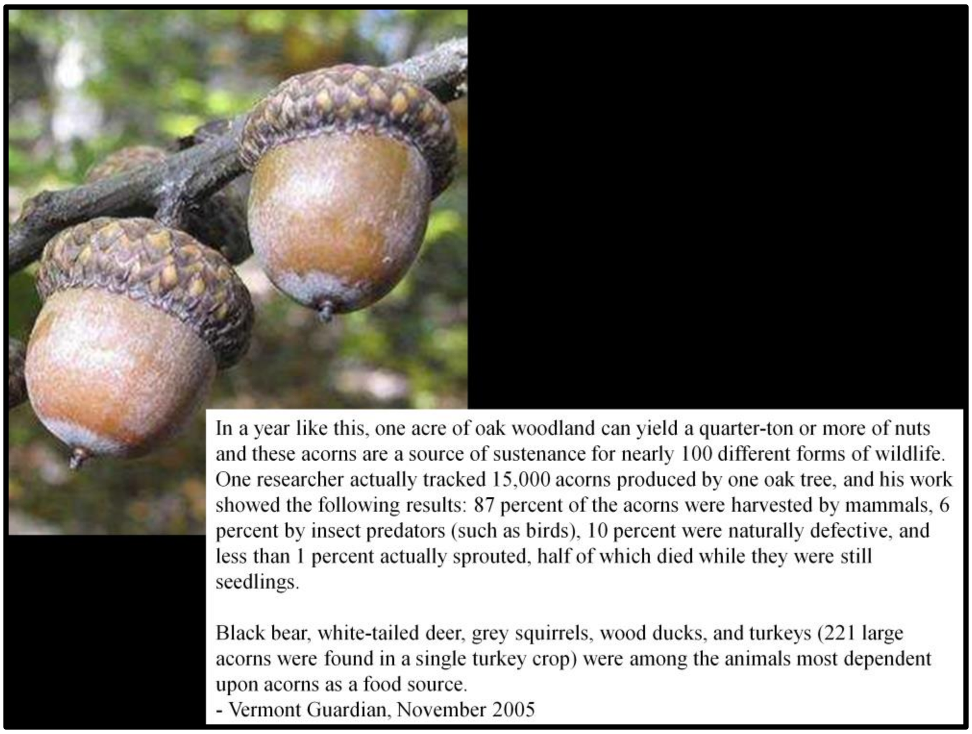
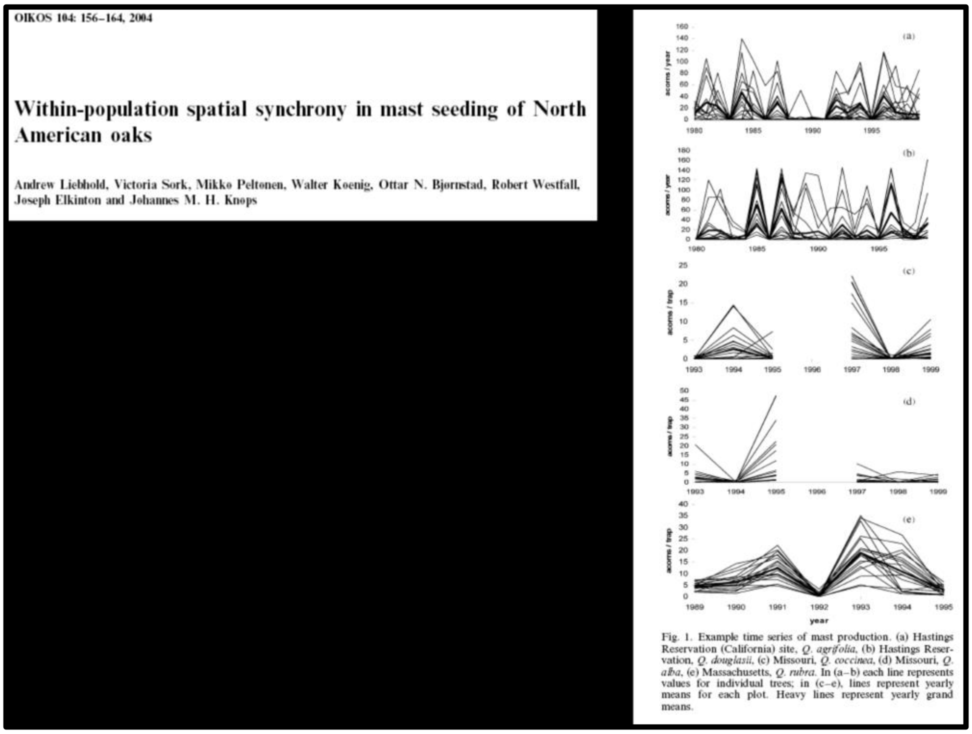


Fig. 1. White-footed mouse densities on three control (○) and three experimental (●) grids as mean (±SE) MNKA ha^{-1}, April 1995–October 1996, showing (i) 1995 densities before, during, and after the period of mouse removal from experimental grids (black bar) at the time of native female gypsy moth 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*).

“MNKA” = 'minimum number known alive‘ Read the graph caption and make sure you understand what it is showing.



A favored food for deer mice (and many other species), when available, is acorns. Acorns are highly nutritious and easily stored. However they are not produced in constant quantities. Typically, an oak tree will produce very large crops every few years and virtually none in the intervening years.


OIKOS 104: 156–164, 2004

Within-population spatial synchrony in mast seeding of North American oaks

Andrew Liebhold, Victoria Sork, Mikko Peltonen, Walter Koenig, Ottar N. Bjørnstad, Robert Westfall, Joseph Elkinton and Johannes M. H. Knops

Fig. 1. Example time series of mast production. (a) Hastings Reservation (California) site, *Q. agrifolia*, (b) Hastings Reservation, *Q. douglasii*, (c) Missouri, *Q. coccinea*, (d) Missouri, *Q. alba*, (e) Massachusetts, *Q. rubra*. In (a–b) each line represents values for individual trees; in (c–e), lines represent yearly means for each plot. Heavy lines represent yearly grand means.

Oak trees in a particular area tend to exhibit synchrony in their acorn production patterns. Years when most trees produce large crops of nuts are called “mast” years (the word is an old English term for ‘meaty’ fruits produced by trees).

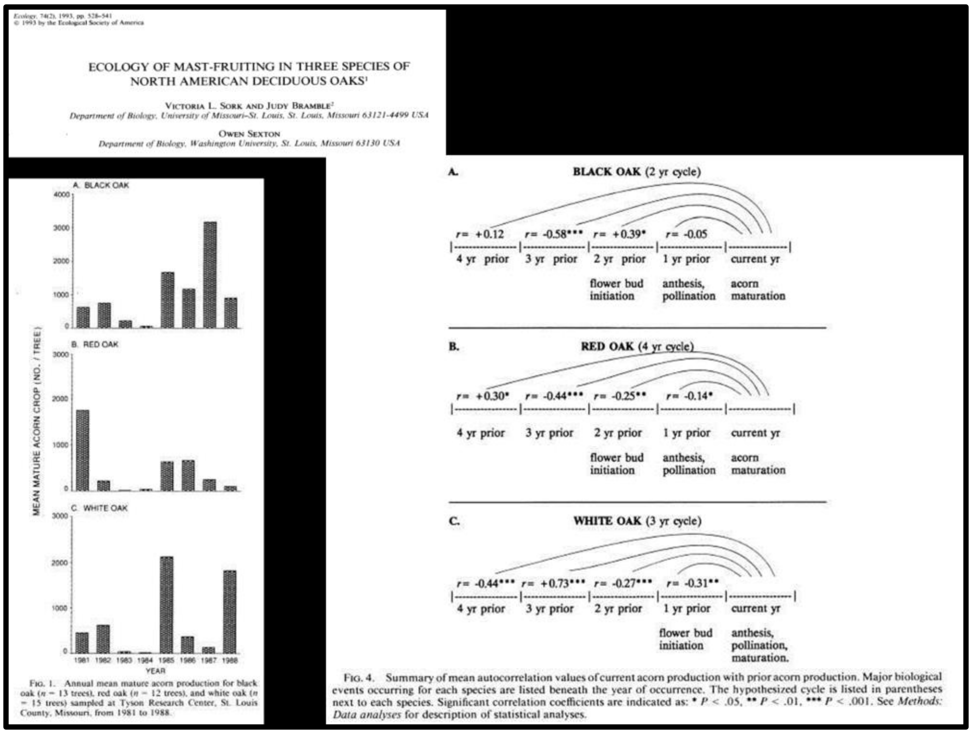


Fig. 1. Annual mean mature acorn production for black oak (n = 13 trees), red oak (n = 12 trees), and white oak (n = 15 trees) sampled at Tyson Research Center, St. Louis County, Missouri, from 1981 to 1988.

Fig. 4. Summary of mean autocorrelation values of current acorn production with prior acorn production. Major biological events occurring for each species are listed beneath the year of occurrence. The hypothesized cycle is listed in parentheses next to each species. Significant correlation coefficients are indicated as: * $P < .05$, ** $P < .01$, *** $P < .001$. See *Methods: Data analyses* for description of statistical analyses.

And oaks of the same species in one region tend to be synchronized in their fruiting 'pulses'. This is referred to as 'mast fruiting' (because farmers used to rely on such crops of tree nuts to feed livestock, and they called this food source 'mast'). Many types of trees that produce large seeds or fruit mast. WHY DO THEY DO THIS?

Journal of Ecology 1995, **83**, 683–696

Alternate bearing, predator satiation and seedling recruitment in *Quercus robur* L.

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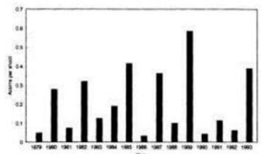


Fig. 1 Acorn production by *Quercus robur* in Silwood Park, Berkshire, England over the 15-year period 1979–93. Acorn production is recorded as the mean number of acorns (including acorns galled by the cynipid wasp Andricus *quercuscalicis*) per shoot averaged over 30 trees. Notice that acorn production during the 'high' years is extremely variable, and that there were two consecutive 'low' years in 1983 and 1984 (see text for details). Different trees were used for the periods 1979–81 and 1982–93.

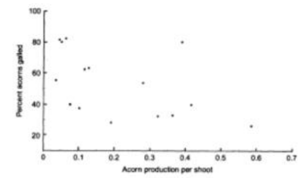
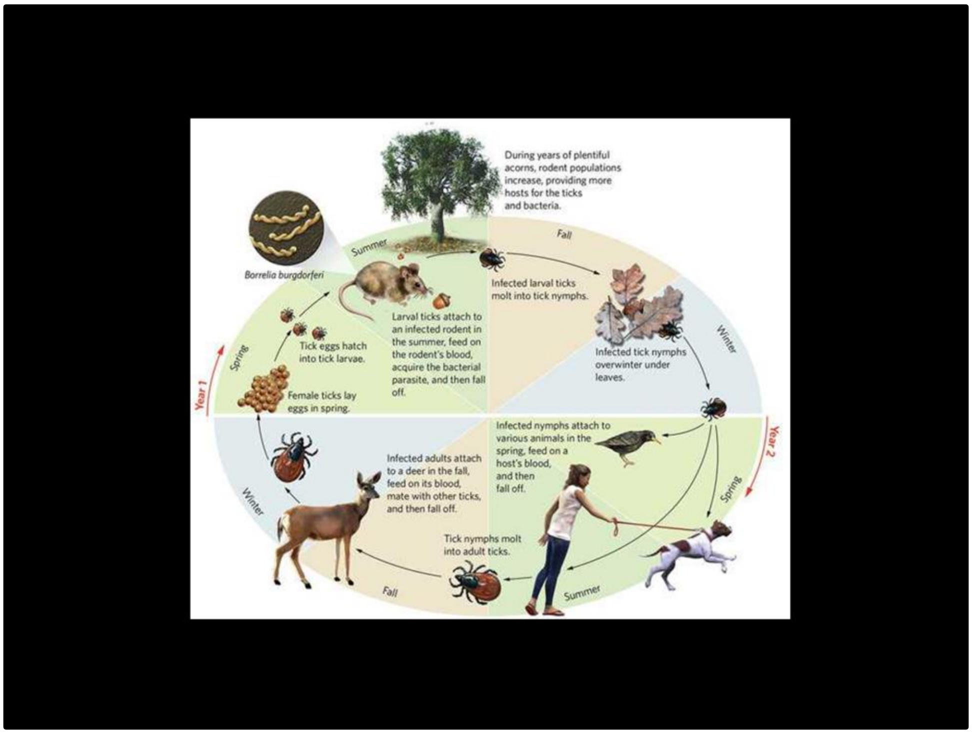


Fig. 3 The relationship between mean acorn crop size and average mortality ($P \times 100\%$) inflicted by the gall wasp *Andricus quercuscalicis* (averaged across 30 trees; each point represents one year of the study). This broad pattern of inverse density dependence is consistent with the hypothesis of predator satiation during the 'high acorn years' (mean loss rate = *c.* 30%) and competition for access to young female flowers in the low years (mean loss rate = *c.* 70%), but note that the significance of the logistic regression (up to 1992) was destroyed by the coincidence of high acorn crop and high percentage galling in 1993 (see text for details). In most years, however, the probability of an acorn escaping knopper gall attack is proportional to acorn crop size. There is no evidence for nonlinearity in the relationship between galling and acorn crop size on the logit scale [$\ln(P/(1-P))$] on which the regression was carried out.

Table 3 The fate of acorns, assessed by a comparison of predator-proof acorn traps with canopy assessments and counts of open-ground quadrats. The densities of sound acorns, weeviled acorns and knopper galls on four occasions between 1984 and 1989 (average of 60 traps; six beneath each of 10 trees). Note the marked difference percentage sound acorns and percentage weevily acorns removed from the ground during the first 24 h

Year:	1984	1985	1988	1989
Average acorn crop:	low	peak	trough	peak
Sound acorns				
(a) on the tree (per shoot)	0.14	0.25	0.06	0.43
(b) in traps (m^{-2})	3.2	6.3	4.2	18.3
(c) on the ground (m^{-2})	0	1.3	0.4	10.1
(d) % predation on ground	100	79	90	45
Galls of *A. quercuscalicis*				
(a) on the tree (per shoot)	0.05	0.17	0.04	0.15
(b) in traps (m^{-2})	1.7	4.4	3.1	5.8
(c) on the ground (m^{-2})	2.0	4.0	3.4	5.3
(d) % acorns galled (tree)	21	35	33	23
(e) % acorns galled (ground)	69	56	72	29
Acorns with *C. glandium*				
(a) on the tree (per shoot)	0.05	0.06	0.02	0.07
(b) in traps (m^{-2})	1.1	2.1	0.9	3.5
(c) on the ground (m^{-2})	0.9	1.9	0.9	2.9
(d) % nongalled acorns with weevils (on the tree)	26	25	18	16
(e) % nongalled acorns with weevils (on the ground)	100	59	69	22
(f) % weevily acorns removed	18	10	0	17

The prevailing theory for the EVOLUTIONARY reason for mast fruiting is the 'predator satiation' hypothesis. Seed-eater populations are limited in the 'sparse' years to levels that can't consume all of the available nuts in mast years; some nuts 'escape' predation to produce new trees. Thus, individual trees that mast, and individuals that respond to the same 'cues' for a mast crop as other trees in the area, are most likely to produce seeds that survive to make new trees (i.e., these traits will be *selected for*). This is the predator satiation hypothesis. Some evidence supports it (i.e., conforms with predictions that emerge from the hypothesis).



Adding oak mast dynamics to the picture may explain fluctuations in Lyme disease frequency from year to year. Is it appropriate to say that oaks cause Lyme disease?

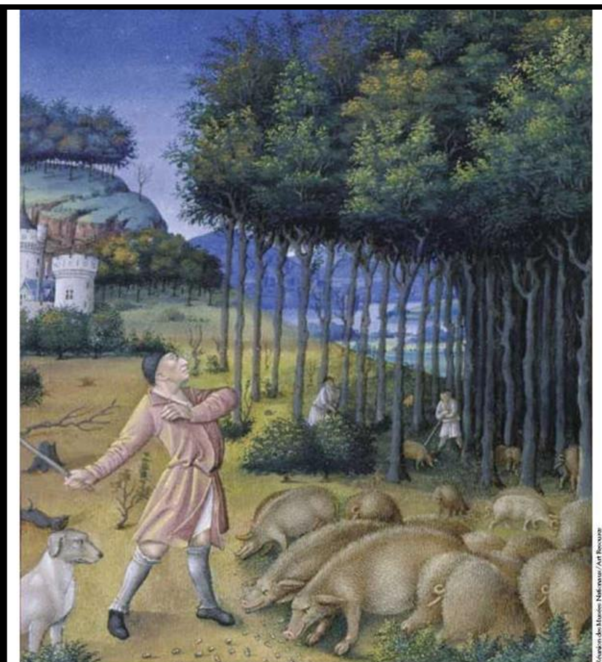


Figure 1. Oak trees have long provided sustenance for people and their livestock. During a mast year, entire populations of oaks produce huge crops of acorns, and farmers have taken advantage of this bounty since at least medieval times. Painted in 1416, this picture for November from the calendar section of the *Très Riches Heures*, by the Limbourg brothers from Flanders, shows a peasant throwing a stick to knock down acorns on which his pigs are feeding. Only recently have scientists begun to understand the ecological significance and underlying biology of masting.

Pigs eating acorns, being watched by stern dog and a ham actor. A tangential comment; a number of organisms may have evolved as specialists in exploiting resources that are extremely abundant when they occur, but that occur infrequently (‘pulsed resources’). Pigs may be one of these (check out Dan Janzen's *Why do bamboos wait so long to flower*). Certainly human pig-herders have long recognized and exploited the phenomenon of masting to fatten their hogs..

THESE PATTERNS have been used to suggest a HYPOTHESIS FOR WHY LYME DISEASE HAS BECOME SUCH A PROBLEM ONLY RECENTLY (or did we just not notice it before)? Consider what's changed. For example, passenger pigeons were another species that evolved to exploit resources that are occasionaly extremely abundant (in this case by being able to travel great distances in huge flocks to where a resource 'pulse' might be available at any given time). They are known to have eaten really vast quantities – millions of tons -- of mast crops. Maybe they consumed the acorns before the mice could get them – in which case exinction of pigeons → increasing mouse populations → greater prevalence of *Borrelia burgdorferi*?

Let us now, kind reader, inspect their place of nightly rendezvous. One of these curious roosting-places, on the banks of the Green river in Kentucky, I repeatedly visited. … I rode through it upwards of forty miles, and, crossing it in different parts, found its average breadth to be rather more than three miles. … I arrived there nearly two hours before sunset. Few Pigeons were then to be seen, but a great number of persons, with horses and wagons, guns and ammunition, … Two farmers from the vicinity of Russelsville, distant more than a hundred miles, had driven upwards of three hundred hogs to be fattened on the pigeons which were to be slaughtered. … The dung lay several inches deep, covering the whole extent of the roosting-place. Many trees two feet in diameter, I observed, were broken off at no great distance from the ground; and the branches of many of the largest and tallest had given way, as if the forest had been swept by a tornado ….. Suddenly there burst forth a general cry of "Here they come!" The noise which they made, though yet distant, reminded me of a hard gale at sea, passing through the rigging of a close-reefed vessel. As the birds arrived and passed over me, I felt a current of air that surprised me. Thousands were soon knocked down by the pole-men. The birds continued to pour in. The fires were lighted, and a magnificent, as well as wonderful and almost terrifying, sight presented itself. The Pigeons, arriving by thousands, alighted everywhere, one above another, until solid masses were formed on the branches all round. Here and there the perches gave way under the weight with a crash, and, falling to the ground, destroyed hundreds of the birds beneath, forcing down the dense groups with which every stick was loaded. It was a scene of uproar and confusion. I found it quite useless to speak, or even to shout to those persons who were nearest to me. Even the reports of the guns were seldom heard, and I was made aware of the firing only by seeing the shooters reloading.

No one dared venture within the line of devastation. … The Pigeons were constantly coming, and it was past midnight before I perceived a decrease in the number of those that arrived. The uproar continued the whole night; …. Towards the approach of day, the noise in some measure subsided: …, the Pigeons began to move off in a direction quite different from that in which they had arrived the evening before, and at sunrise all that were able to fly had disappeared. The howlings of the wolves now reached our ears, and the foxes, lynxes, cougars, bears, racoons, opossums and pole-cats were seen sneaking off, whilst eagles and hawks of different species, accompanied by a crowd of vultures, came to supplant them, and enjoy their share of the spoil.

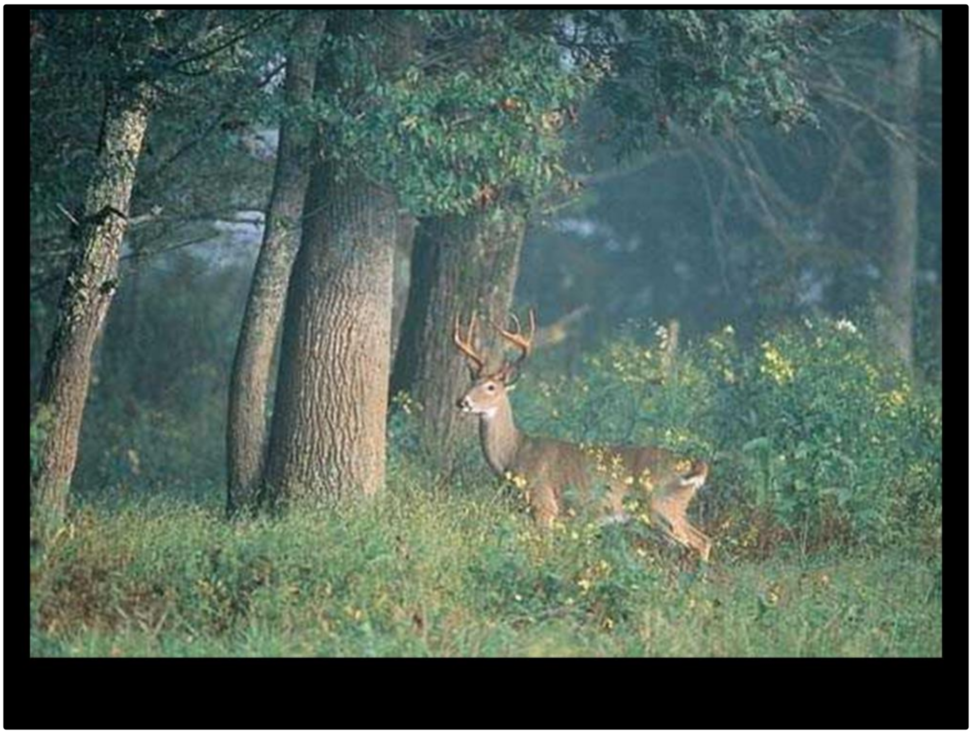
It was then that the authors of all this devastation began their entry amongst the dead, the dying, and the mangled. The Pigeons were picked up and piled in heaps, until each had as many as he could possibly dispose of, when the hogs were let loose to feed on the remainder.

- *J. J. Audubon*

John James Audubon’s comments on the extreme abundance of passenger pigeons

Let us take a column of one mile in breadth, which is far below the average size, and suppose it passing over us without interruption for three hours, at the rate mentioned above of one mile in the minute. This will give us a parallelogram of 180 miles by 1, covering 180 square miles. Allowing two pigeons to the square yard, we have one billion, one hundred and fifteen millions, one hundred and thirty-six thousand pigeons in one flock. As every pigeon daily consumes fully half a pint of food, the quantity necessary for supplying this vast multitude must be eight millions seven hundred and twelve thousand bushels per day.

- *J.J. Audubon* 1813



ADD DEER: Because the tick that is the *vector* for *Borellia* is called a 'deer tick', people tend to assume deer are the actual source of the disease. It's a bad rap. Deer don't carry the bacterium much (it doesn't seem to thrive in their blood), BUT they often host many ticks of all ages, including gravid adult females. And deer can travel large distances carrying their ticks along for the ride. And they like acorns a lot. Because they are large and mobile, deer TRACK food pulses spatially. In mast years they travel to oak forests to feed on acorns, where the gravid female ticks they're carrying can drop to the ground and lay eggs that will hatch the following spring. When they are likely to find one of the now-abundant deer mice as their larval host, and acquire the bacterium… Do DEER CAUSE LYME DISEASE?

NORTHWESTERN NATURALIST 85:16–19 SPRING 2004

MOVEMENT OF MALE AND FEMALE BLACK-TAILED DEER FORAGING ON ACORNS

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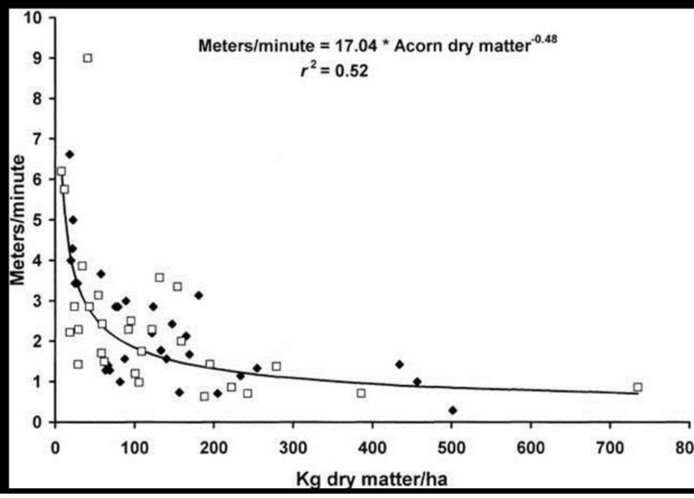
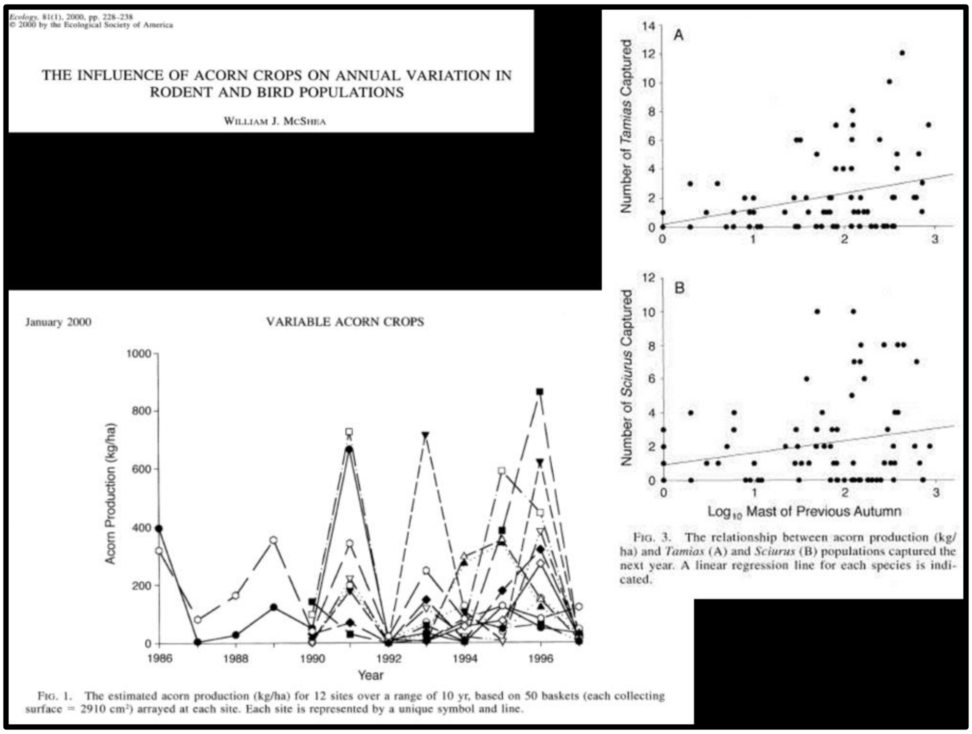


FIGURE 1. Scatter plot of acorn abundance and movement rate of male and female black-tailed deer. Closed diamonds represent males and open squares represent females.

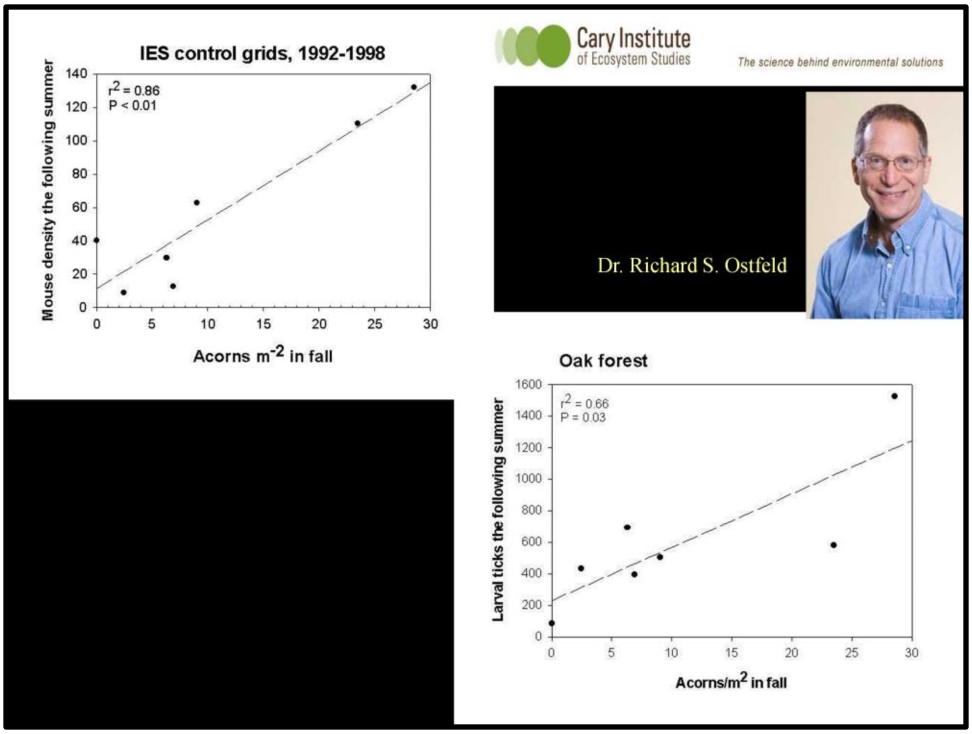
The more nuts available, the less deer travel (this is a prediction of ‘optimal foraging theory’, a central ecological concept that emerges from game theory).



But small, local populations of things like rodents and song birds can't 'follow' the crop; instead their populations respond NUMERICALLY, increasing dramatically in response to mast years and crashing between.

Mother white-footed mouse with babies attached to her nipples.

Following mast years, deer mice produce larger litters of young more frequently. They breed through the winter, feeding on stored acorns, when normally they stop breeding due to scarcity of food by late winter and populations crash before spring. Following mast years, deer mouse populations continue to increase through the winter and so, in spring, when tick eggs hatch and questing larvae are numerous, they're very likely to encounter a deer mouse – the *Borrelia* reservoir – and become an infected nymph that might bite you. OAKS CAUSE LYME DISEASE… (NOTE that if this chain of reasoning – hypothesis – is correct, it would predict that Lyme disease cases should be more frequent about a year after a mast crop of acorns…)



A great deal of work on this system and how it relates to Lyme disease has been done in Rick Ostfeld's lab at the Institute for Ecosystem Studies in Millbrook, NY (in Dutchess County – the epicenter of Lyme disease). Many of the results reviewed here are from the work of Ostfeld and collaborators.

None of this happens in isolation; Garrett Hardin's 'first law of ecology' is 'you can't do just one thing'. Deer mice are also predators on the eggs and young of song-birds that nest on the ground or in low shrubs. Do high population of hungry mice (in years right after mast crops) have negative effects on song-bird populations? Sharp-shinned hawks eat song-birds. Does the effect propagate up the food chain?

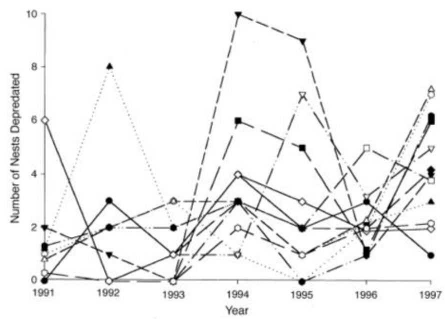


FIG. 4. The number of nests depredated at each site during the study. Ten nests of two quail eggs each were pl
each site for one week during June of each year. Each site is represented by a unique symbol and line.

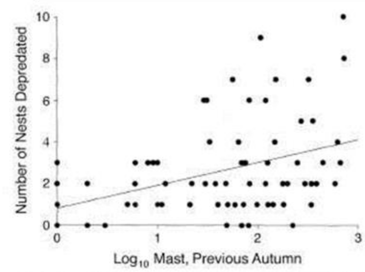
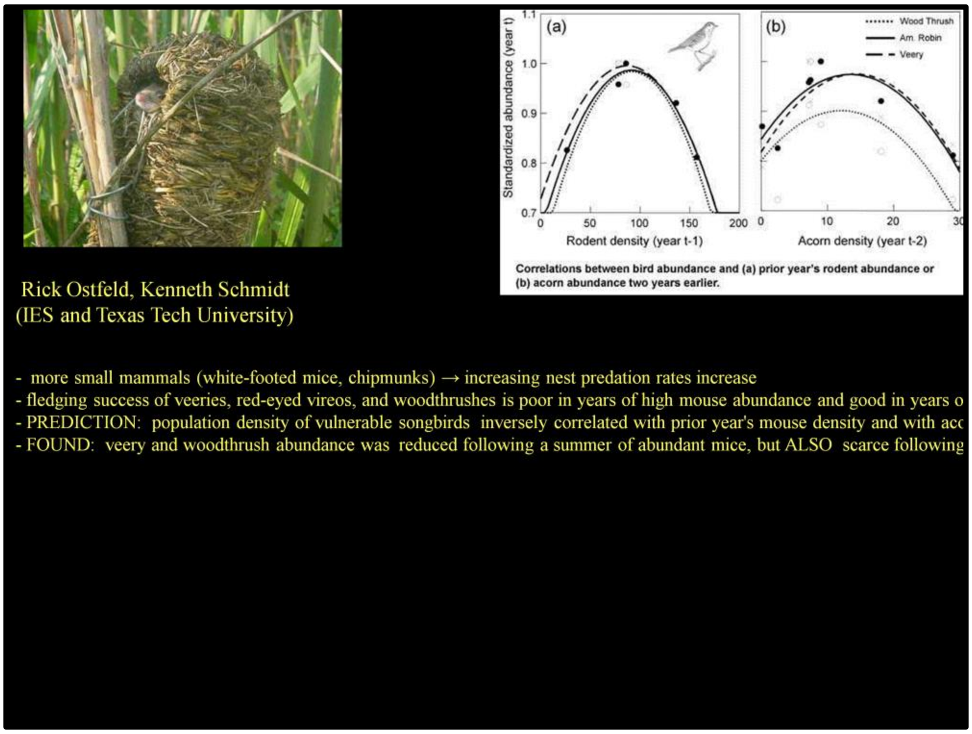
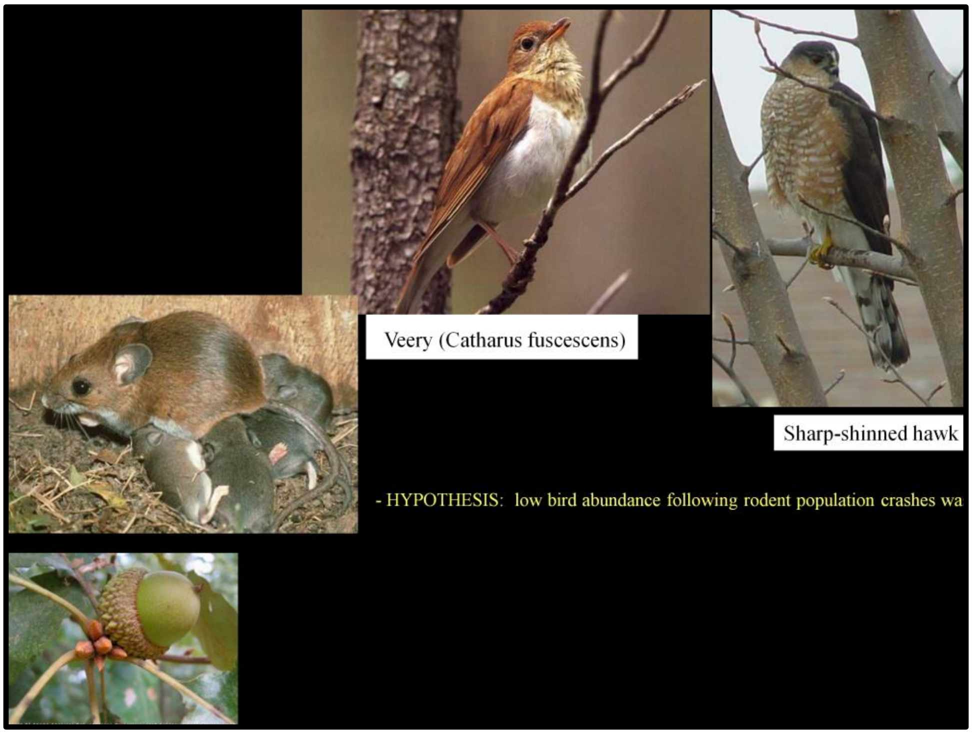


FIG. 5. The relationship between the amount of artificial nest predation at a site and the amount of acorns (kg/ha) produced at the site the previous autumn. A linear regression line is plotted.

Maybe; an experiment with artifical nests



But it's odd that songbird breeding success is ALSO low in years following very low rodent populations. Is there another layer of feedback?



Avian predators also eat songbirds AND mice; perhaps their populations build up following rodent population surges, then when rodent populations crash (a year after mast), hungry hawks focus on song-birds? (This propagation of effects up and down the food-chain is common. Ecologists talk about 'top-down' and 'bottom-up' control on ecological structures. If acorn abundance is what controls all these populations, that would be an example of 'bottom-up' control...) Such complex feed-backs make system behavior very hard to model!

Ecology, 84(2), 2003, pp. 406–415

SONGBIRD POPULATIONS IN FLUCTUATING ENVIRONMENTS: PREDATOR RESPONSES TO PULSED RESOURCES

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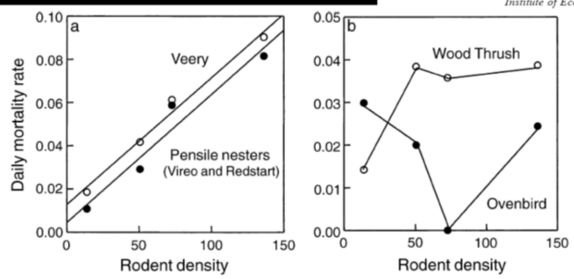

Daily nest mortality rates on songbird nests as a function of the summer rodent density (number of animals/2.25 ha).

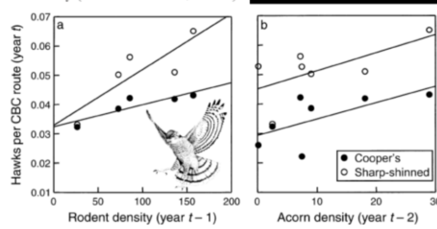

FIG. 4. Sharp-shinned and Cooper's Hawk abundance (standardized to party hour) from 37 Christmas Bird Count regions surrounding Dutchess County (New York), as a function of (a) rodent or (b) red oak acorn abundance measured at the Institute of Ecosystem Studies. Hawk abundances are plotted against rodent densities (number of animals/2.25 ha) from the previous summer or against acorn densities (acorns/0.5 m²) from two autumns previous.

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SONGBIRD POPULATIONS IN FLUCTUATING ENVIRONMENTS: PREDATOR RESPONSES TO PULSED RESOURCES

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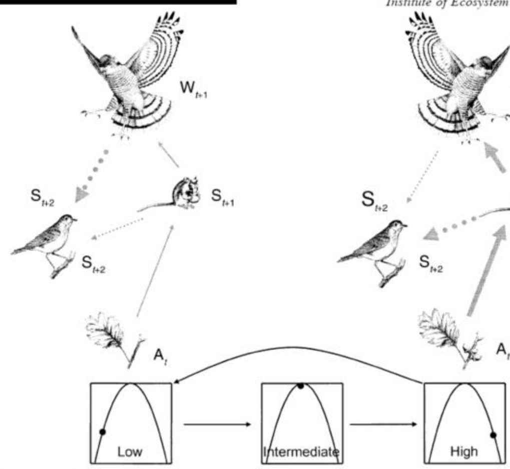
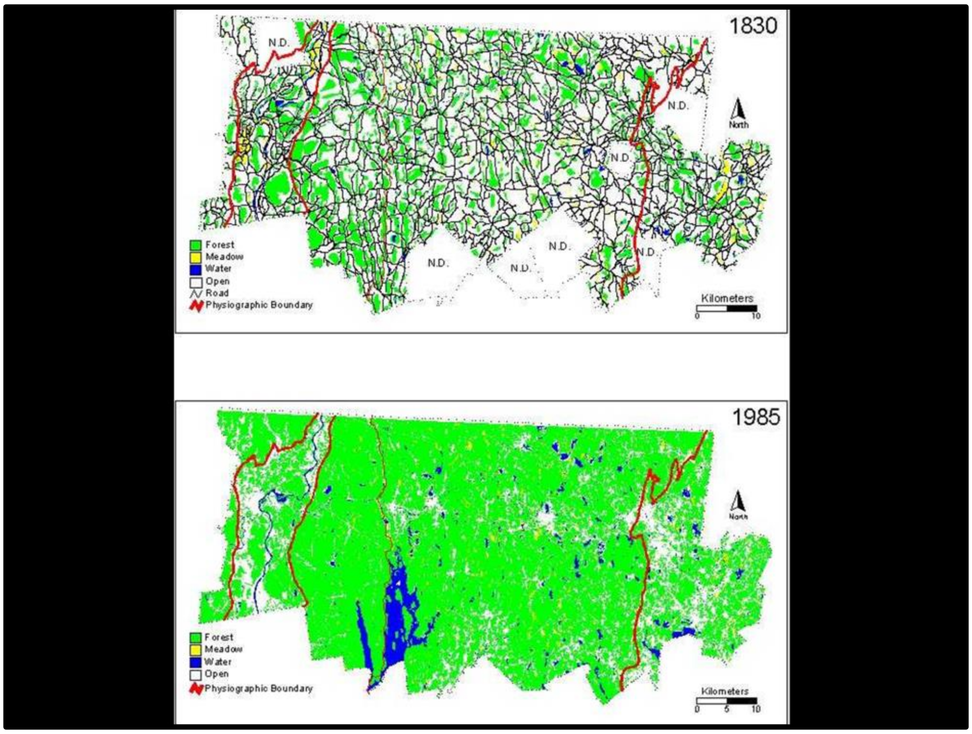


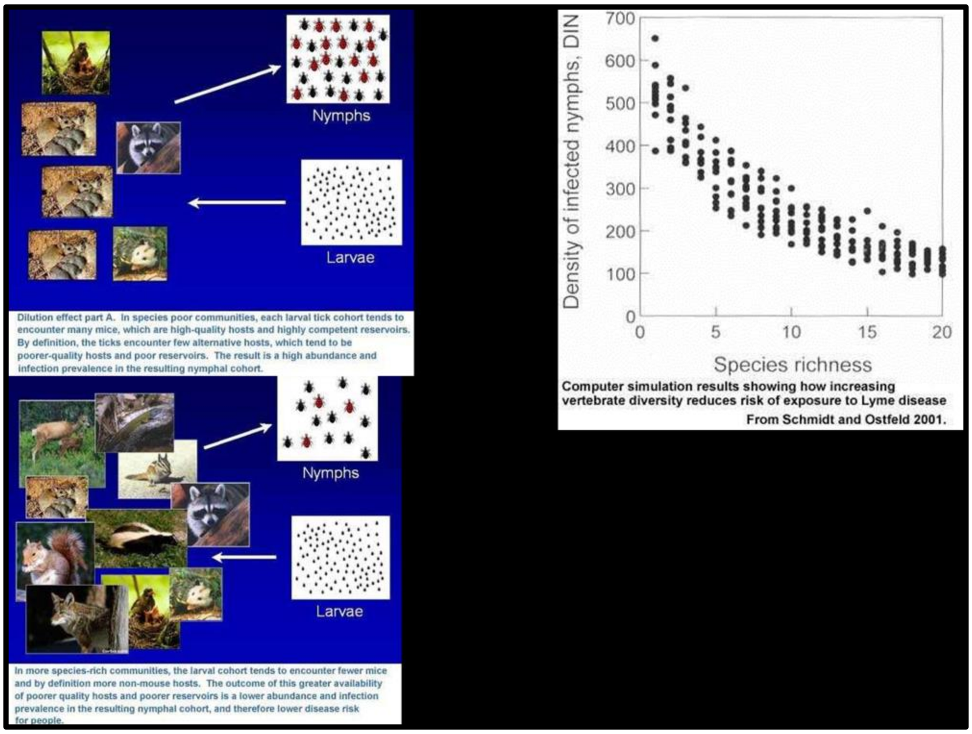
FIG. 5. Schematic diagram of the connections between oaks, mast-consuming rodents, songbirds (thrushes in particular), and hawks. The figure goes through a masting cycle (left to right) from low to intermediate to high acorn production. Positive and negative effects highlighted in the study are designated by solid and dashed arrows, respectively. The notations beside the arrows designate the period over which abundances were examined: A_t = Autumn of year t, S_{t+1} = Summer year t + 1, W_{t+1} = Winter year t + 1. Songbirds have two negative inputs: rodents, primarily via nest predation, and hawks, primarily via predation on fledglings, juveniles, and adults.



But there are other changes in the landscape. In the 19th century the northeast was essentially completely deforested and large mammal populations (including deer) almost eliminated. Since then, forests have recovered with agricultural abandonment. Oaks are often a prominent component of post-agricultural forests in southern new england and the lower Hudson valley – the areas where Lyme is most prominent. LYME DISEASE IS CAUSED BY HUMAN SETTLEMENT AND LAND-USE?? (These maps for north-central Mass. show forest cover in green for 1830 and 1985; most of the northeast would show a similar pattern.)

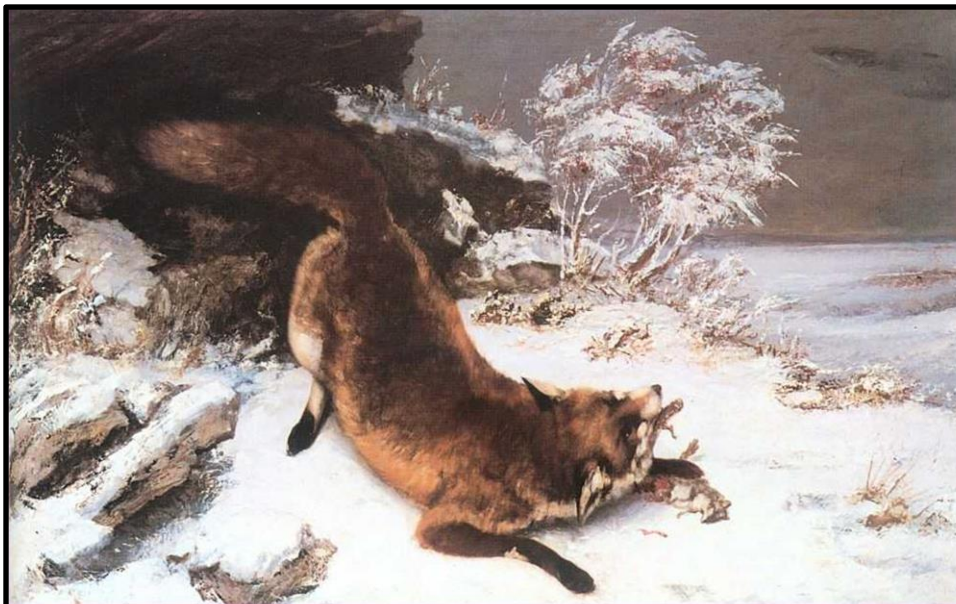
Fragmented landscape illustrating forest patches of different sizes embedded within a non-forested matrix. Small patches appear to pose higher risk of exposure to Lyme disease.

BUT WHY HAS LYME disease become so much more serious NOW? Perhaps it's because passenger pigeons are gone, so rodent populations get larger – but that should have happened a century ago... Another aspect of the modern landscape of these regions is that forests are often highly FRAGMENTED – broken up by patterns of human settlement (farms or suburban development depending on area). Additionally, residential patterns have changed; for esthetic reasons, people choose to live in dispersed developments interspersed with wooded areas.



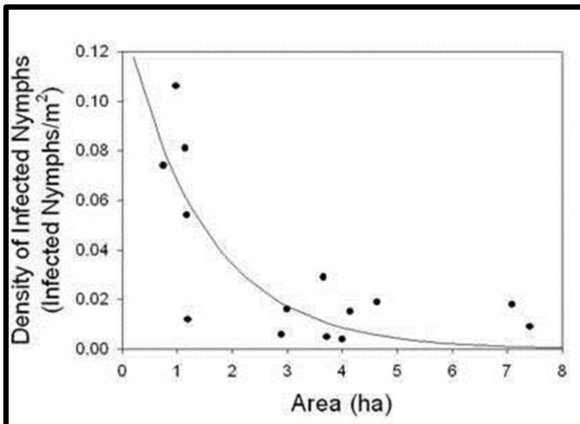
Computer simulation results showing how increasing vertebrate diversity reduces risk of exposure to Lyme disease
From Schmidt and Ostfeld 2001.

Fragmentation has many effects on the ecological structure of forest communities. Fo example, as forest patch size decreases, diversity of mammal species – potential hosts for ticks – decreases. But deer mice don't require large patches and are present everywhere; in the smallest patches, they're about the only mammal around. SO, in small patches, tick larvae are most likely to attach to a mouse and become infected nymphs that can give you Lyme disease; in larger patches, larvae are likely to encounter other hosts, none of which are as likely to infect them with *Borrelia*.



Gustave Courbet, *The Fox in the Snow*

And large patches of forests are more likely to support predators that will eat deer mice, while they're among the first to go extinct when habitat area shrinks.



Data from Dutchess County, New York, showing that small forest fragments have elevated Lyme disease risk. From Allan et al. 2003.

So researchers have predicted that exposure to infected nymphal ticks is more likely in landscapes dominated by small forest patches. There's some evidence to support this, as well. SO, it seems that the risk of human infection is particularly high where humans live close to small, fragmented, isolated patches of forest. SUBURBANIZATION CAUSES LYME DISEASE?

The ecology of infectious disease: Effects of host diversity and community composition on Lyme disease risk

Kathleen LoGiudice*†‡, Richard S. Ostfeld*, Kenneth A. Schmidt*§, and Felicia Keesing*¶

PNAS | January 21, 2003 | vol. 100 | no. 2 | 567–571

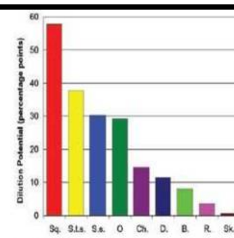


Fig. 2. The ability of each species to reduce the effect of white-footed mice (the most competent reservoir) on NIP. Dilution potential is the difference (in percentage points) between the expected NIP in a two-host community consisting of mice plus the focal species and a community in which mice are the only possible host. Sq., squirrel; S.t.s., short-tailed shrew; S.s., *Sorex* shrew; O, opossum; Ch., chipmunk; D, deer; B, birds; R, raccoon; Sk, skunk.

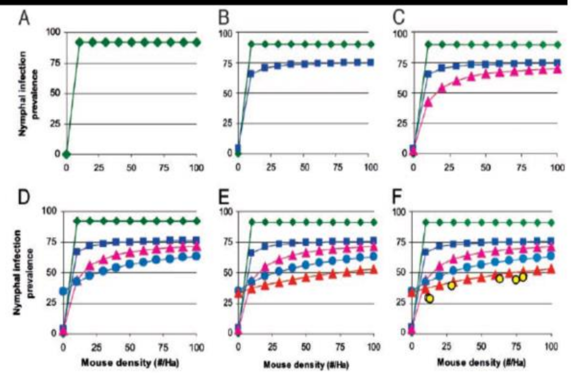
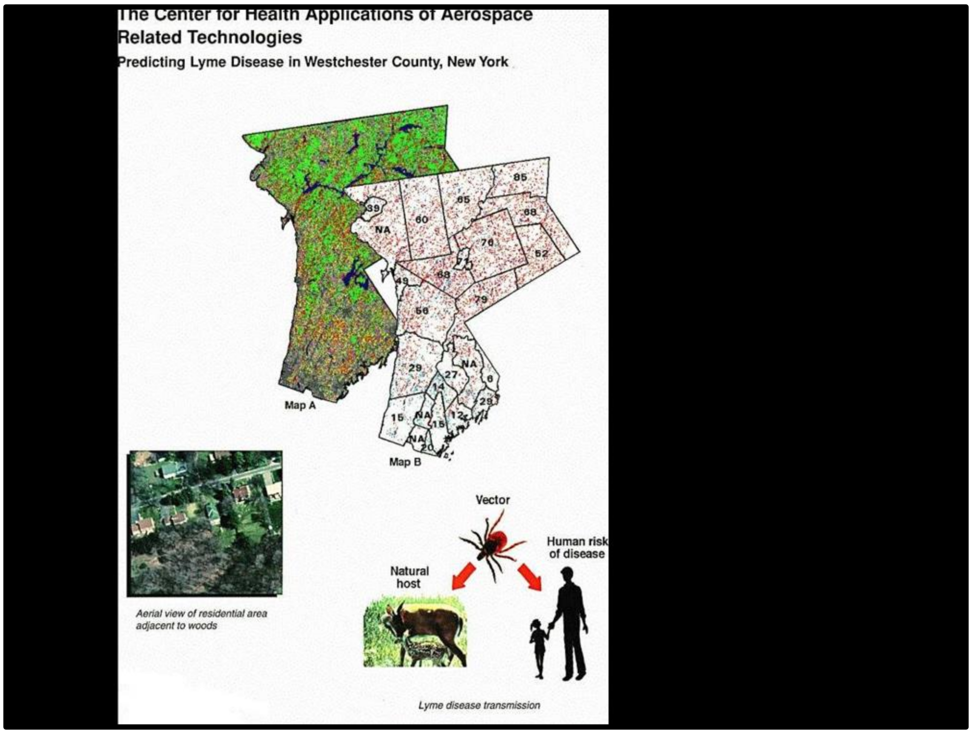
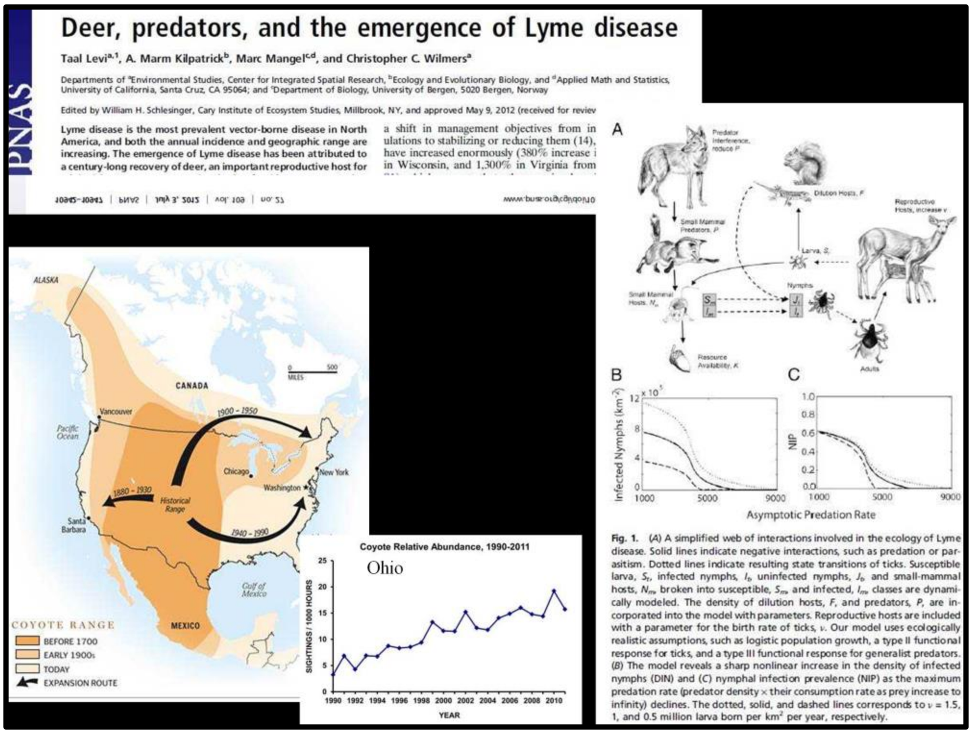


Fig. 1. Predicted NIP across a realistic range of mouse densities as host diversity is increased and host community composition is concomitantly changed. Host community consisting of only white-footed mice (green diamonds) (*A*); white-footed mice, chipmunks, and white-tailed deer (dark blue squares) (*B*); all hosts in *B* plus raccoons, opossums, and skunks (pink triangles) (*C*); all hosts in *C* plus short-tailed shrews and four species of ground nesting birds (light blue circles) (*D*); all hosts in *D* plus tree squirrels and *Sorex* shrews (red triangles) (*E*); and field data collected at our site over 7 years, showing the mouse density during the larval peak in year *t* and NIP for host-seeking nymphs in year *t* + 1 (yellow circles) (*F*).



So human risk of Lyme disease is a function of exposure to tick nymphs or adults that have acquired *Borrelia* from a previous host. Can we take this a step further and see if the risk of Lyme disease can be quantitatively modeled/predicted? What are the factors that might increase risk? Can these be incorporated in quantitative structures and computer models?



It gets even more complicated as complexity is added to the food web. It appears that MEDIUM-SIZED predators – like coyotes (whose abundance has increased dramatically in the northeast in recent years) – can lead to decreased abundance of SMALL predators (like foxes, either eating or just competing with them. Foxes are mouse specialists, so reducing fox numbers may actually FAVOR deer mouse populations... COYOTES CAUSE LYME DISEASE?

Deer, predators, and the emergence of Lyme disease

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Edited by William H. Schlesinger, Cary Institute of Ecosystem Studies, Millbrook, NY, and approved May 9, 2012 (received for review March 16, 2012)

Lyme disease is the most prevalent vector-borne disease in North America, and both the annual incidence and geographic range are increasing. The emergence of Lyme disease has been attributed to a century-long recovery of deer, an important reproductive host for

a shift in management objectives from increasing deer populations to stabilizing or reducing them (14), Lyme disease cases have increased enormously (380% increase in Minnesota, 280% in Wisconsin, and 1,300% in Virginia from 1997 to 2007; Fig.

10942–10947 | PNAS | July 3, 2012 | vol. 109 | no. 27

www.pnas.org/cgi/doi/10.1073/pnas.1204536109

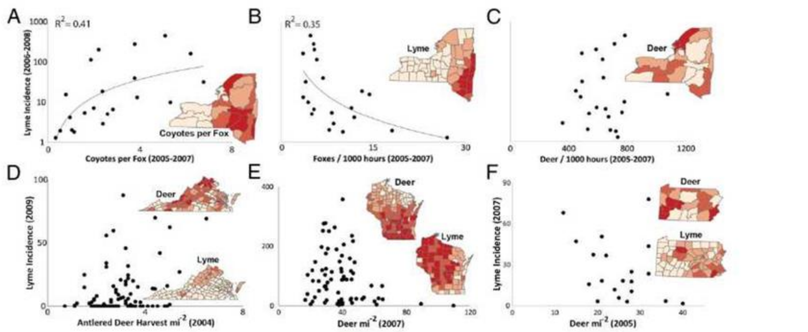
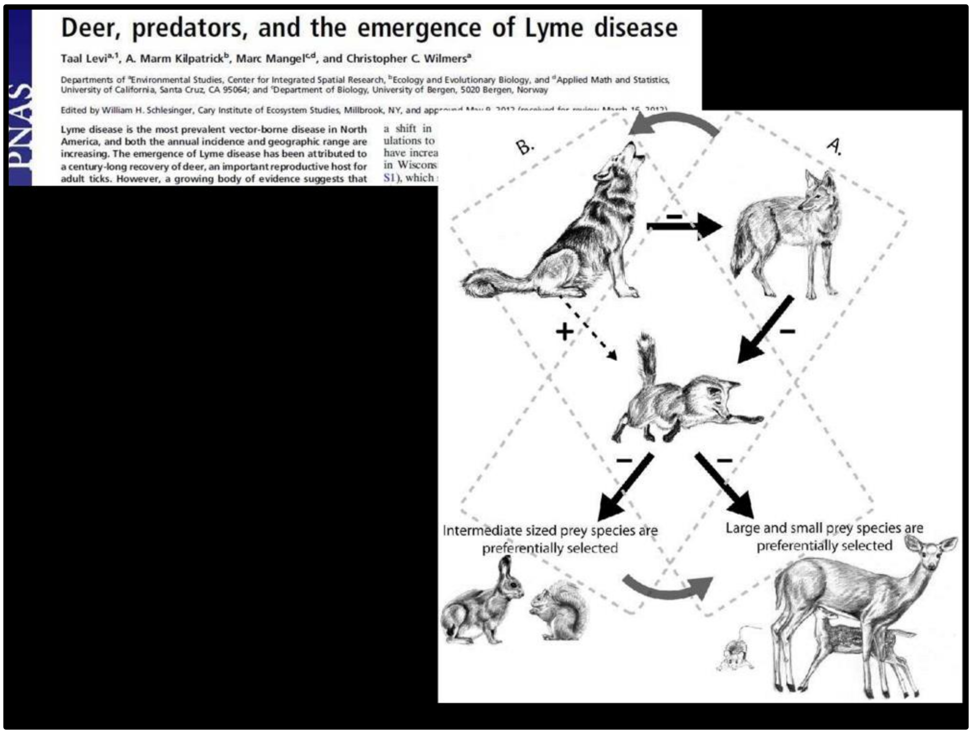
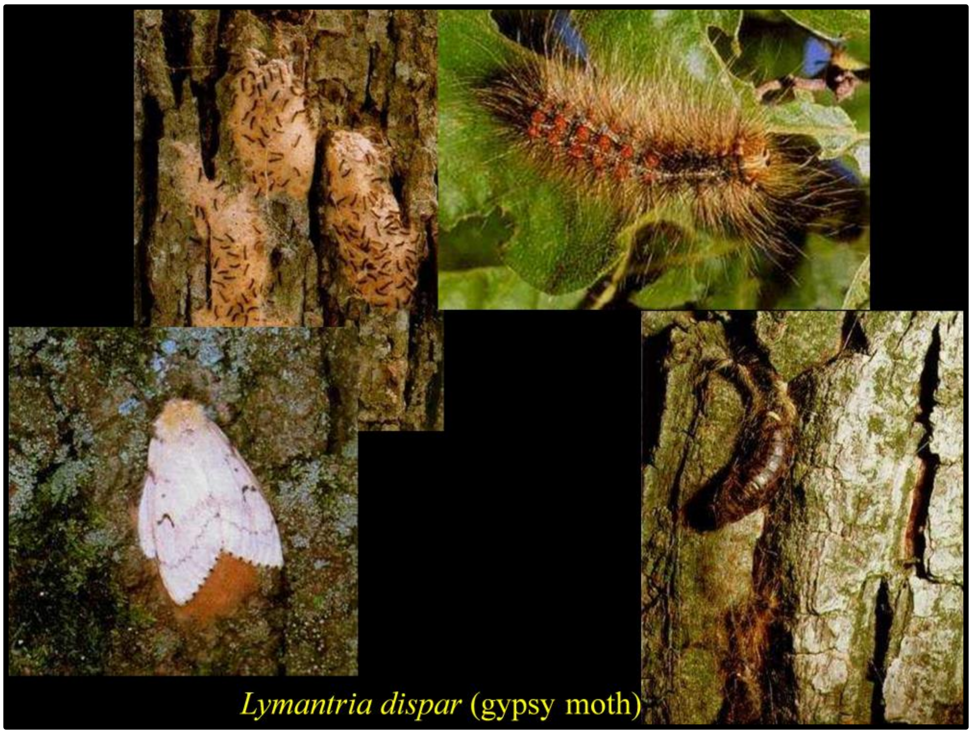


Fig. 4. Spatial relationships among deer, predators, and Lyme disease. (*A*) In New York, observation rates from the bow-hunter wildlife survey indicate that Lyme disease incidence (cases per 100,000) is positively correlated with coyotes, (*B*) negatively correlated with foxes, and (*C*) unrelated to deer. Coyote observations are scaled by foxes to highlight the transition in the predator community and its impact on Lyme disease. (*D*) Deer as estimated by the buck harvest density are positively (but weakly) correlated with Lyme disease incidence in Virginia counties (R^2 = 0.1, P = 0.001). (*E*) In contrast, deer density estimates (from sex-age-kill models) are negatively correlated with Lyme incidence in Wisconsin counties (R^2 = 0.06, P = 0.05, but driven by few data points—not significant when removed) and (*F*) negatively correlated in Pennsylvania deer management units (R^2 = 0.14, P = 0.09), where the unit with the lowest deer density has the second-highest Lyme incidence. (*Insets*) Darker red indicates more-abundant wildlife populations and higher Lyme incidence (in four classes: 0–10, 10–50, 50–100, and >100 cases per 100,000).

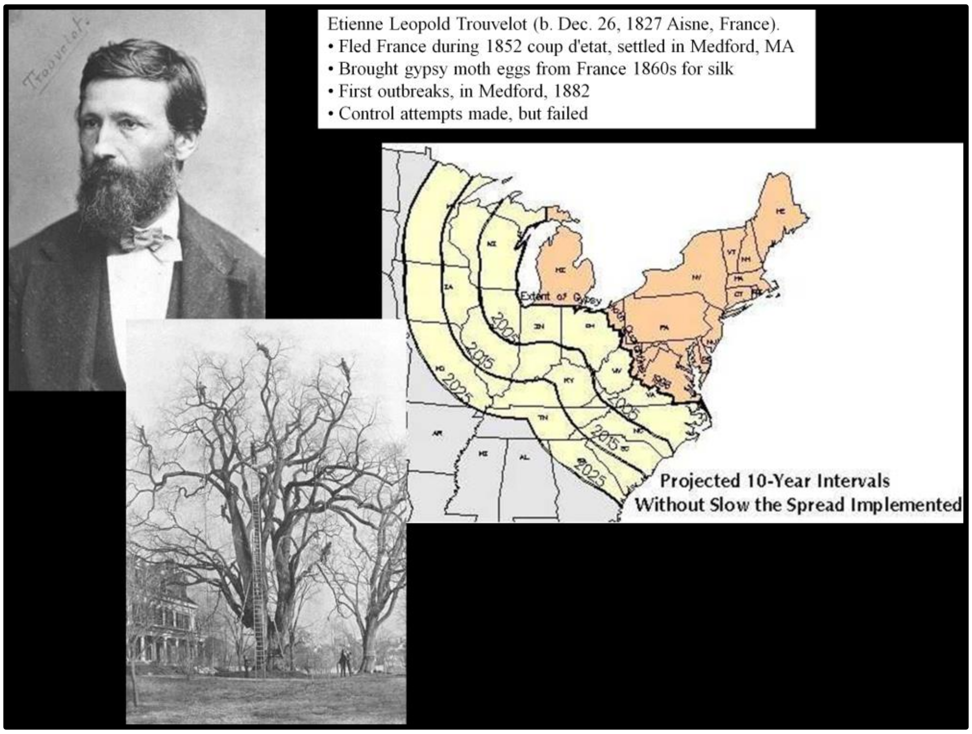
Lyme disease frequencies are better predicted by COYOTE and FOX abundance than by DEER abundance. STUDY THESE GRAPHS and think about what they mean…



BUT WHY has this system changed in recent decades? Why the increase in coyotes? One possibility is the elimination, by humans, of wolves. Wolves tended to exclude coyotes from forested habitats – partly by hunting and eating them (even though they're close relatives and can interbreed). Wolves are specialists on large prey, like deer, but they don't bother things as small as foxes. So, replacing wolves with coyotes may 'shift the state' of the whole food chain (what's sometimes called a 'tipping point' phenomenon, or a system with 'alternative stable states'). It can be either 'wolf and fox eat a lot of deer and mice (and maybe squirrels and rabbits benefit), OR 'coyote s eat a lot of medium-sized animals like foxes, squirrels, rabbits, and deer and mice benefit'. COYOTES CAUSE LYME DISEASE?

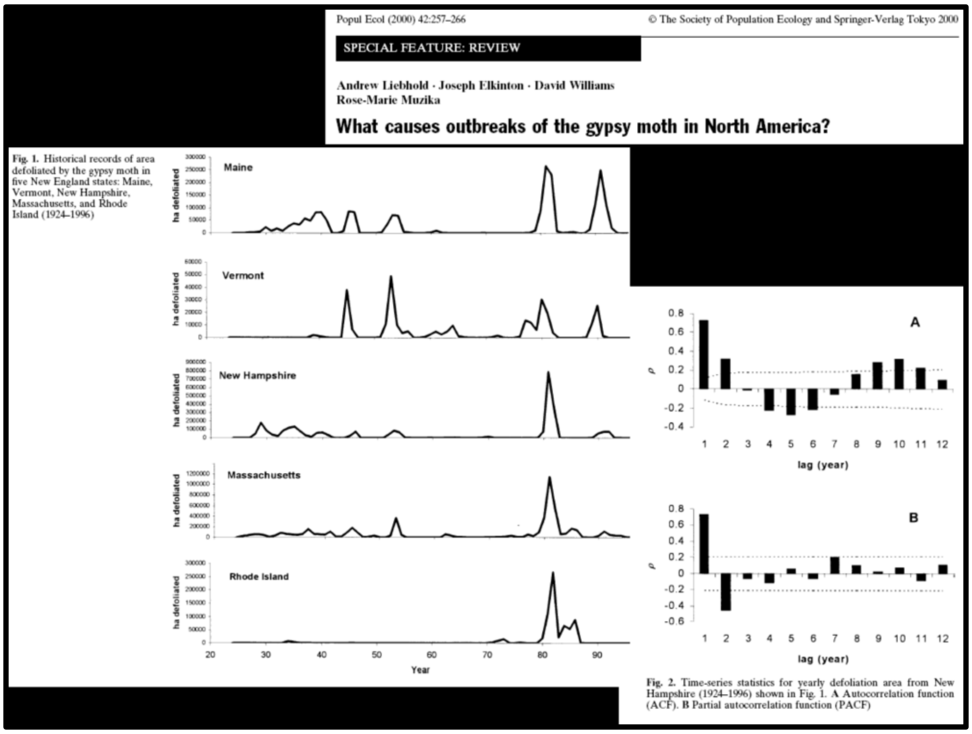


There are yet other dynamics linked to these processes that remain to be fully understood. For example, Gypsy moths are a non-indigenous species, introduced originally as a potential source of silk.

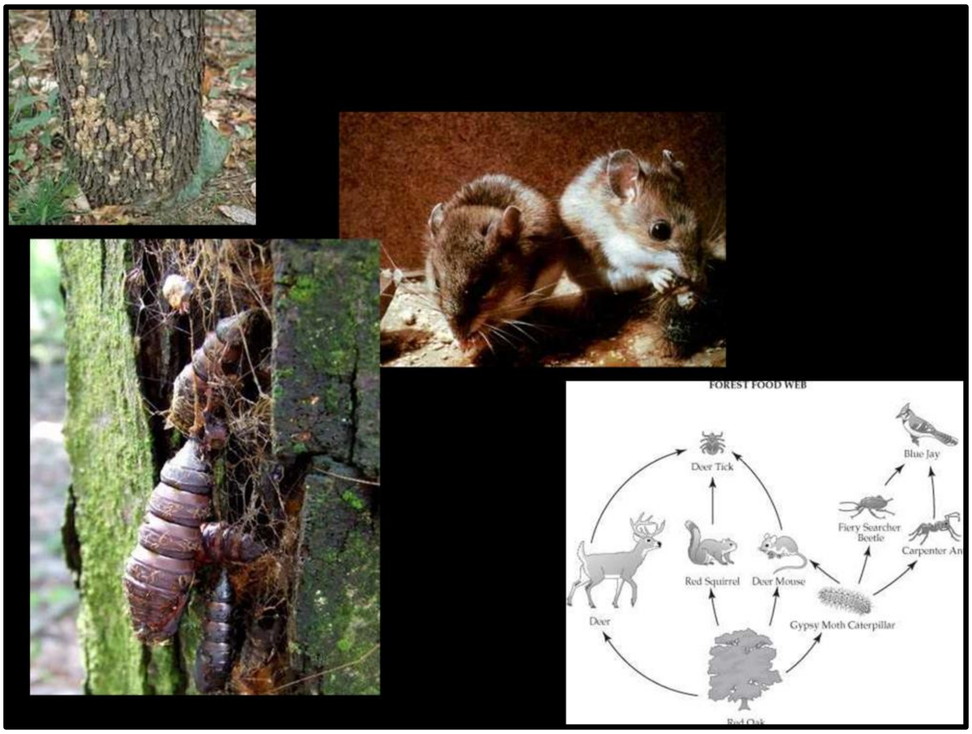


After intentional introduction as silk producers, they escaped confinement and have become one of our most problematic forest pests.

Gypsy moths are an ‘outbreaking’ species. Populations can fluctuate between very low background levels and occasional massive outbreaks that can cause landscape-scale defoliation of trees and, sometimes, significant tree mortality (certainly stress for the defoliated trees). There are a number of species of insects that have similar population cycles; they are not all introduced.



The causes of gypsy moth outbreaks have been a long-time object of study; they're still not well understood, but I won't get into that here (it's an area that would be of interest for those who are mathematically inclined – one of the first 'real-world' applications of complexity theory).



Gypsy moth egg cases and cocoons can be extremely abundant when there's an outbreak; deer mice love to eat them. So gypsy moths decrease oak vigor by defoliating trees, thus decreasing likelihood of a mast crop – but they also support larger deer mouse populations. Gypsy moths both cause and prevent Lyme disease. This is an example of a system with FEEDBACK LOOPS – a 'complex system' – and the behavior of such systems is extremely difficult to predict. All ecological systems have such feedback loops. (Mice are also a likely factor in helping to regulate gypsy moth populations; when large deer mouse populations are getting hungry in a summer AFTER a mast crop, they may really hit gypsy moth populations hard.)

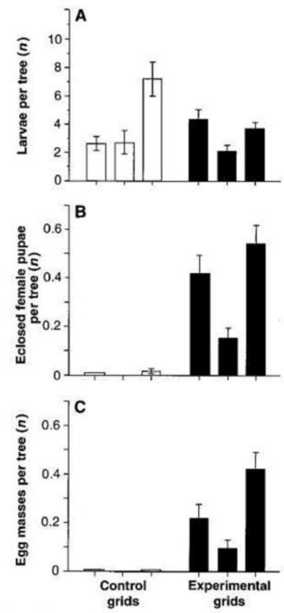


Fig. 2. Densities of gypsy moth life stages on or inder burlap bands on trees on control grids open bars) versus experimental grids (solid bars) vhere mice were removed. The bars show grid neans and within-grid SEs. Across-grid control

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

Clive G. Jones,* Richard S. Ostfeld, Michele P. Richard, Eric M. Schauber, Jerry O. Wolff

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.

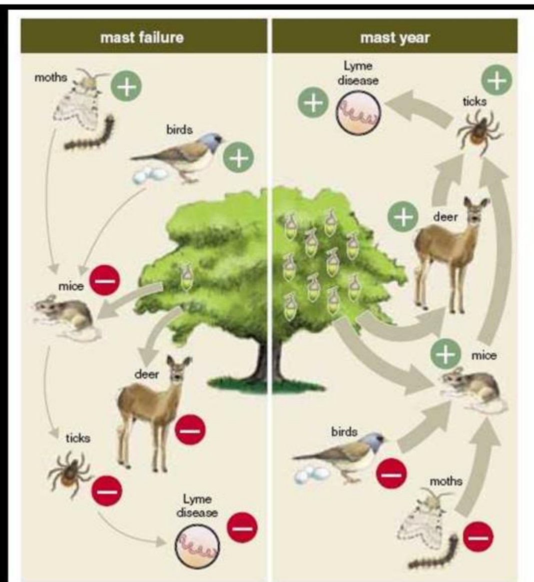
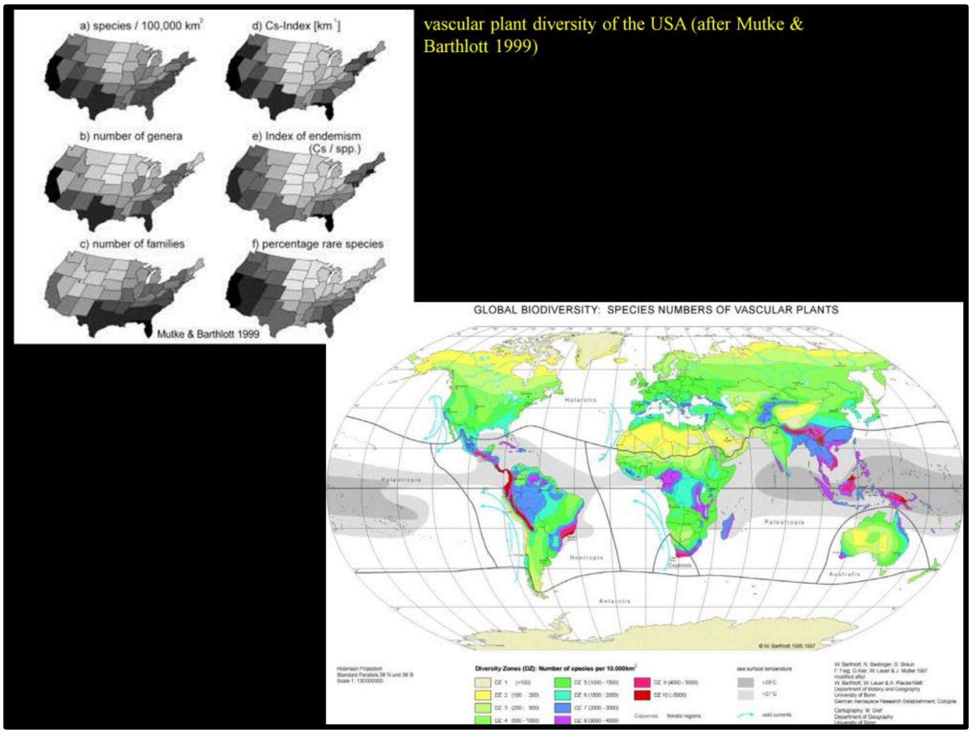


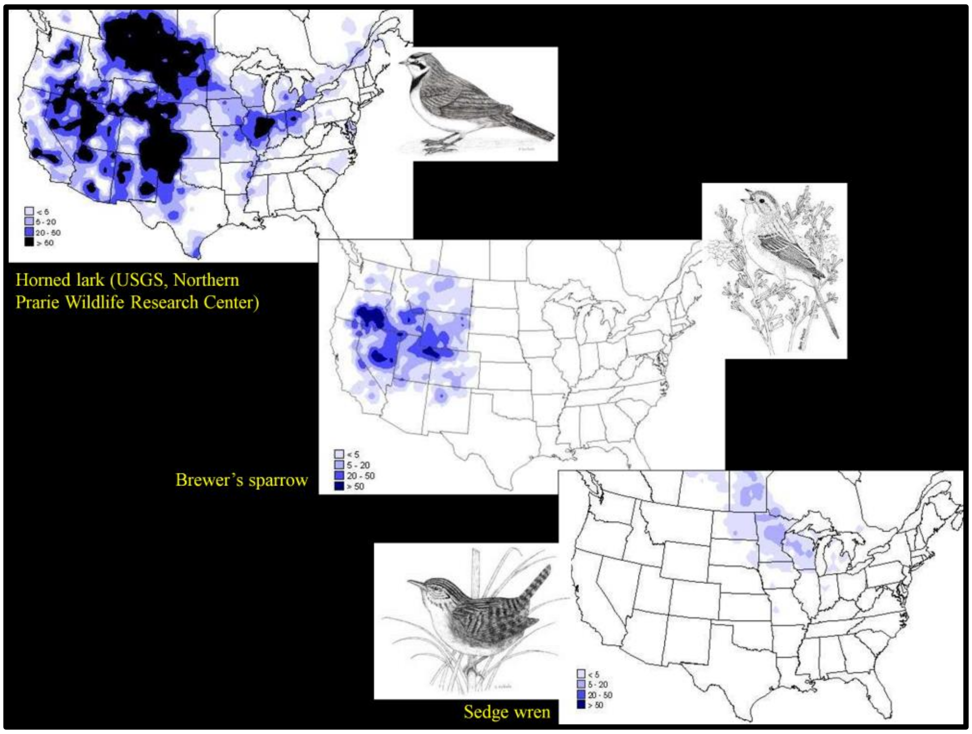
Figure 4. Multiple ecological interactions between species in a model Eastern deciduous forest result in a trophic cascade in which the effects of acorn crop size ripple through the food web, setting off a chain of events that cause animal populations to change. Large amounts of energy *(arrows)* flow through the system during a mast year *(right)* when a large acorn crop is produced by an oak *(center)*, causing populations of deer, mice and ticks to increase and raising the incidence of tick-borne Lyme disease (+). Populations of ground-nesting birds and gypsy moths decrease (–) owing to predation by mice. During a mast failure *(left)* effects of an acorn shortage on the food web are negative for acorn-eaters and their predators and positive for birds and moths, whose eggs and larvae are eaten by mice.

So, a SMALL PART of all of this can be shown in a cause-effect diagram. A sort of *causal model (or hypothesis) of the system.* Here, signs indicate positive or negative population influences; if the organism at the bottom of the arrow increases, it will have the indicated effect on population of the organism at the other end.. The signs change depending on resource (acorn) supply. Imagine what other links might exist (or have changed), and think about how you'd assess their importance.



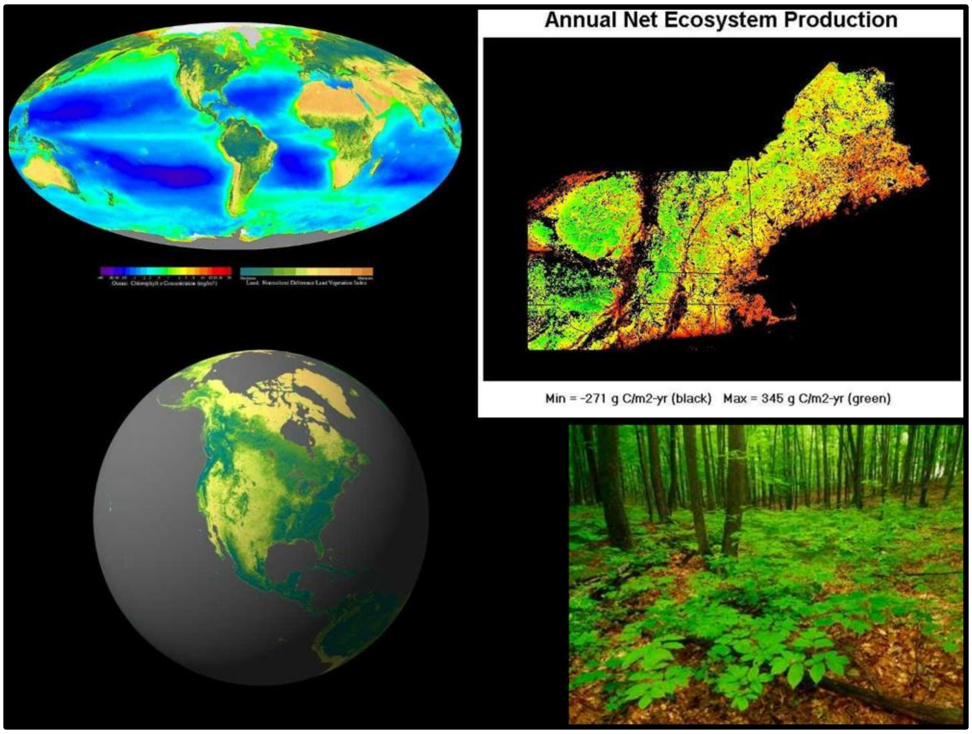
The Lyme disease story touches on many of the kinds of questions ecologists ask. It's hard to simplify what ecology is too far, but it's possible to put most of those questions in one of three or four big categories:

BIG QUESTION ONE: *DIVERSITY*. What regulates diversity? Why do some areas support more species than others? What factors favor high diversity (what allows species to coexist?) or constrain diversity (what prevents species from coexisting?)? Are there patterns? Explanations? Does diversity have practical consequences for human well-being?

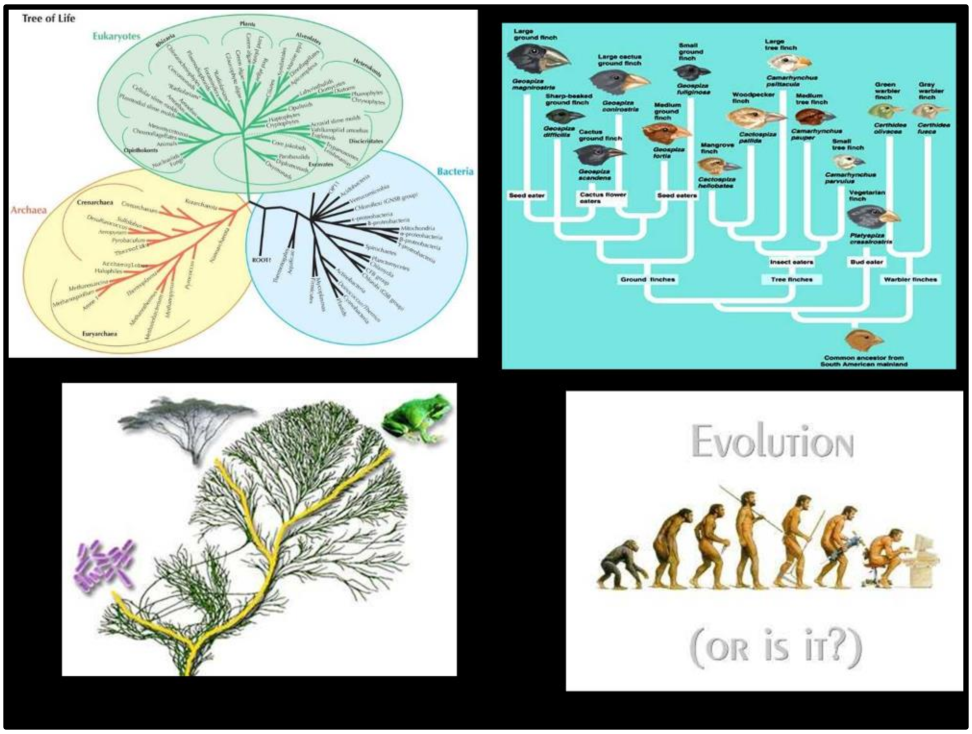


BIG QUESTION TWO: *ABUNDANCE and DISTERIBUTION*. What factors determine abundances and distributions of particular species? Ranges of species? Are there patterns? Predictions?

(Different types of ecosystems or *ecological communities* are 'assembled' as a result of factors that regulate species distributions and abundances. We intuitively interpret these assemblages to tell us things about they area/habitat; we are all applied ecologists…



BIG QUESTION THREE: *BIOLOGICAL PRODUCTIVITY*. What factors control the rate of overall ecosystem processes – particularly the rates at which new organic material (biomass) is produced and consumed? What do patterns in rates of ecosystem production suggest?



BIG QUESTION FOUR: : *EVOLUTIONARY EFFECTS.* What are the linkages between ecological and evolutionary processes and patterns? How is the diversity of life derived over evolutionary time?