the study areas and control plots. They found about 45 times more of these pupae and moth egg masses in plots with fewer mice. Using freeze-dried pupae attached to wax, which picked up tooth marks, they were able to confirm that mice were eating the pupae in the control plots.

After firming up that link, the researchers simulated a masting. With help from local Girl Scouts, they spread 3500 kilograms (nearly 4 tons) of acorns on their experimental plots. Mouse populations skyrocketed. "Our data show that the key trigger [for moth outbreaks] is this relationship between the acorns and the mice," Jones says.

Along the way, the researchers kept an eye on ticks, because white-footed mice are a reservoir of the Lyme disease spirochete, which they transmit to tick larvae. More mice wouldn't necessarily mean more ticks: Ticks of reproductive age live on deer, not mice. But the summer after the masting, the team found far more tick larvae—an eightfold rise—in the acorn-rich plots compared to other plots. The acorns had apparently attracted tick-bearing deer and boosted mouse numbers as well, Jones says. And the adult ticks, in turn, had spawned more offspring, which infested more mice: The mice in the acorn-rich plots bore 40% more tick larvae than those in other plots.

More acorns, more mice, more deer, more ticks: It adds up to a larger Lyme disease risk, the researchers argue. "It suggests that you may be able to warn people when the risk of Lyme disease is high," Ostfeld says. But the study also highlights the challenge of managing ecosystems, because in this case trying to cut down on Lyme disease by, say, chemically suppressing acorn production could send gypsy moth numbers soaring. "Once we start tinkering with nature, we could get in a wonderful mess," Dobson says.

Fish and other epidemiologists, however, interpret the study more cautiously. They point out that a high larval tick count the summer after a masting may not necessarily mean more infected juvenile ticks a year later. Indeed, Jones's group members didn't measure infection rates on their plots last summer. "The question is still open," says Joseph Piesman, who heads the Lyme disease vector branch at the Centers for Disease Control and Prevention in Fort Collins, Colorado. Many other factors, such as rainfall and competing parasites, also affect the abundance of ticks carrying the Lyme disease spirochete, Piesman says. So nailing down any acorn link, he adds, may take at least a decade of observing mastings and tick outbreaks.

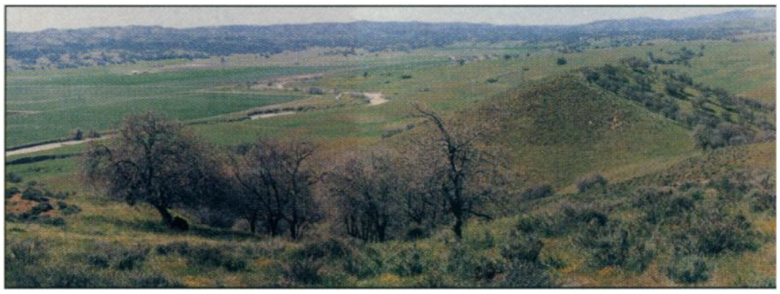
Most experts agree, however, that the work underscores ecology's importance in studying vector-borne diseases. "The entire genetic sequence of the organism wouldn't tell you this," Dobson says.

–Jocelyn Kaiser

SEISMOLOGY

A Slow Start for Earthquakes

If seismologists had their way, every earthquake would have a prelude—days or weeks of preparations along the fault that was about to break. Quake prediction would then be a matter of watching for the right signals. Theoretical and lab studies have suggested that faults should give off such warning signs as they edge toward rupture, but no one has yet found them. Now, researchers using a seemingly roundabout method—testing for the effects of tides on quake timing—offer the strongest evidence yet that some faults do start to slip, rapidly concentrating stress, for hours or days before the full-blown rupture. "The good news is that something must be happening before earthquakes," says seismologist Thomas Heaton of the California Institute of Technology in Pasadena. However, there's no guarantee of successful prediction, he cautions. "The bad news is that it may be so small it's useless."



R. A. KERR

Tidal test-bed. Earth tides don't affect the timing of small quakes on the San Andreas fault, buried beneath these hills near Parkfield.

Seismologist John Vidale of the University of California, Los Angeles, and his colleagues coaxed this bit of good news from the timing of more than 13,000 small to moderate quakes on California's San Andreas fault, near the town of Parkfield, and on one of the great fault's branches, the Calaveras. The ultimate driver of earthquakes on these faults is the slow march of the tectonic plates to either side, which continually adds stress at about 0.1 millibar per hour, building over the years toward the 1 bar to 100 bars needed to rupture a fault. But the gravitational tugs of the moon and sun, which raise tides in the earth just as they do in the ocean, also vary the stress—much faster than tectonics does. As the tides wax and wane, they alternately increase and relieve stress on faults at a rate of several millibars per hour.

If the steady buildup of stress from tectonics and the rapid variations from tides were the only factors involved, Vidale's team reasoned, the tides should sometimes trigger quakes on faults already close to the breaking point. The effect would be subtle—most seismologists long ago rejected schemes to predict earthquakes from tides. Nevertheless, the seismic events should be more common when the tidal pull is strongest, for example during full and new moons. They could occur randomly with respect to tides only if some third process rapidly loads stress onto faults just before quakes, overwhelming the tidal effects.

To test for any trace of tidal influence, Vidale and colleagues included only quakes on straight segments of the two faults, which amounted to 13,042 earthquakes of magnitude 1 to 6. The large number of events together with the known fault orientations—which allowed the tidal stresses to be calculated reliably—gave the study unprecedented sensitivity. Still, they found no correlation between the two phenomena. Of the 13,042 quakes, only 95 more occurred when tidal stresses favored fault failure than when they discouraged failure—not a significant result. Some other short-term stress source must have overwhelmed the tidal effects. "The lack of a tidal correlation argues that there is some preparation process over the days before an earthquake," says Vidale. During the final hours before such an event, stress must build many times faster than it does during a tidal cycle—at least 150 millibars per hour, he adds.

Can seismologists catch this preparatory movement in action and so predict earthquakes? No one knows yet. In theory and lab experiments, the stress-inducing process is a slow but accelerating slip on a small patch of fault. That slip causes stress to build up faster and faster around the edge of the patch, until a larger area of the surrounding fault ruptures in an earthquake. "What we don't know is the size" of the patch, says theoretician and experimentalist James Dieterich of the U.S. Geological Survey in Menlo Park, California. Estimates range from a patch a few hundred meters across before a magnitude 5 quake, which might be detectable by strainmeters buried near the surface, or one only a few meters in size, in which case detection would be hopeless.

Researchers may get an answer from the next magnitude 6 quake to hit the heavily instrumented Parkfield area (*Science*, 19 February 1993, p. 1120) or from a proposed project to monitor a small patch of the fault at Parkfield that regularly fails in frequent magnitude 1 quakes. The answer could make or break earthquake prediction forever.

–Richard A. Kerr