Humans fostering forest-destroying disease

August 15 2007

Enjoying your August vacation? Well, (as they say in the summer movies) there's a killer in the woods. Its strike has been consistently quiet, sudden, and deadly. Unknowingly, we have all been playing into its hands… But put down that rock -- you personally are not in any danger. It's the woods themselves that are getting axed and you may be an accomplice.

Melodrama aside, the threat is very serious – the killer is an invasive, forest-destroying plant disease known as Sudden Oak Death. Caused by an (apparently) non-native water mold (Phytophthora ramorum), the disease affects a broad range of woody plants, and is particularly lethal to our native oaks. In the last few years, it has infected and killed large stands of western oaks with alarming suddenness (hence the name). From its initial California appearance sometime in the mid-1990's, the disease has been spreading rapidly, changing the landscape as it goes.

"People tend to not care about plants and forests as much as we do about humans and animals, but sudden oak death could be a bird flu of the plant world waiting to happen," said Ross Meentemeyer, a landscape ecologist at the University of North Carolina at Charlotte. "This may be even worse than chestnut blight in its impact on our forests, since it is affecting multiple keystone species."

Since the same plant pathogen has also been found in forests of Europe, it is suspected (but not yet proven) that the sudden oak death pathogen was introduced by humans, probably from Asia. What has been shown by recent research is that human activities are amplifying the disease's

impact and spread.

A recent article published by Meentemeyer and colleagues in the *Journal of Ecology* showed that "pathogen inoculum load" (the actual amount of infectious pathogen present) is greater in forests with high landscape connectivity and high abundance of host species. In a follow-up study forthcoming in Ecological Applications, Meentemeyer and his colleagues report that years of fire suppression and other land-use practices have altered structure and composition of forests in way that may facilitate spread of the disease. California's oak woodlands have grown larger and denser over the past six decades. Counter-intuitively, this has made them more susceptible to being wiped out by the rapid onset of sudden oak death.

"More connected forests have more disease," said Meentemeyer. "Smaller and more isolated forests have less disease. Being smaller and more isolated doesn't necessarily prevent disease, but it occurs at smaller levels in those areas."

This finding connects with research presented in the article forthcoming in Ecological Applications. Meentemeyer and colleagues discuss findings that further suggest humans have been causing landscape changes that are encouraging the spread of the disease. The study examines detailed aerial photography records for an area of northern California and shows that oak woodlands have increased in area by 25% over a 58-year period, while grasslands and chaparral shrublands (non-host vegetation types) have both significantly decreased.

The researchers suspect that the reason larger and denser patches of forest encourage the spread of the disease involves the role played by another California woodland tree—Bay laurel. Bay laurel is the primary carrier for the disease, since it acquires a non-lethal but highly contagious leaf infection. Bay laurel seems to be increasing in abundance

in the changing forests and to make matters worse denser forests create cooler and moister understory microclimates that encourage the spread of the bay laurel leaf infection, which in turn can lead to the fatal infection of the oaks.

The effect of larger, cooler forests “explained significant variation in infection level of P. ramorum,” the article argues. “We conclude that enlargement of woodlands and closure of canopy gaps, likely due to years of fire suppression, facilitated the establishment of P. ramorum by increasing contagion of hosts and enhancing forest microclimate conditions.”

Unhealthy forest management may be encouraging the spread of the disease, but it is perhaps not the only way humans are furthering sudden oak death’s deadly march. In other research using Geographic Information System analysis, Meentemeyer and his colleague Hall Cushman at Sonoma State University found evidence that humans may be spreading the disease directly through the unintentional transportation of small amounts of infected soil.

“There is some compelling evidence that humans could be moving the disease in infested soil,” Meentemeyer said. “We have found evidence for human involvement at three different scales of analysis. First of all, the pathogen is much more likely to occur along hiking and biking trails, where humans travel. Second, using some of our landscape and regional data, we have shown that highly visited state and county parks have more disease than private ranches and lands that have very limited visitation.

“We have also found on an even broader scale across the state of California that forests surrounded by high human population densities are more likely to be infected. In this analysis, we carefully controlled for the effect of climate and other environmental factors known to cause disease.”

The research has alarming implications, as California's much-loved woodlands are already approximately 10 percent infected and the remainder, the models suggest, are highly vulnerable.

"Only a fraction of the high risk forests are currently affected, so there is a lot of potential for more to happen," Meentemeyer said.

"This is why we have also been working on more dynamic models that simulate spread through time. We've been using this work to guide management activities for early detection surveys at sites that are likely to be infected. Once the disease is present, the only way you can control it is by detecting early it at locations where it is isolated and small – before it becomes epidemic.

"Over the past 4 years I have directed an early detection program in which we have been sampling over a thousand sites all over high-risk habitats in California, guided by our computer risk models," he noted.

Whether the disease is at risk to spread beyond its current range, particularly to eastern North America's oak-rich forests, is still unclear.

"Eastern forest trees like red oak and pin oak have been shown to be susceptible in the lab," Meentemeyer warns. "There are models that show what areas might be at risk based on climate matching with California, but we can't validate the models yet. The disease is not there, so we don't know what conditions in nature are favorable or not… we have very different weather conditions and very different plant species in these forests. The Eastern forests are certainly at risk, but we don't know to what degree at this time."

Source: University of North Carolina at Charlotte