Bioeconomics of Forest Disease: Learning to Manage Emerging Threats

Noam Ross UC Davis REACH IGERT Bridge Proposal September 21, 2012

Advisors: Alan Hastings, Jim Sanchirico, David Rizzo

1. I'm here to talk about my work which takes place at the nexus of forest biology, epidemiology, and economics

I am interested in emerging diseases in forests and what economics can tell us about managing these diseases when our knowledge of them is incomplete.

First I am going to talk a little bit about the consequences of forest disease.

Diseases influence forest structure profoundly, and the introduction of a new disease can rearrange the ecosystem.

Forest diseases transform landscapes



American chestnuts circa 1910

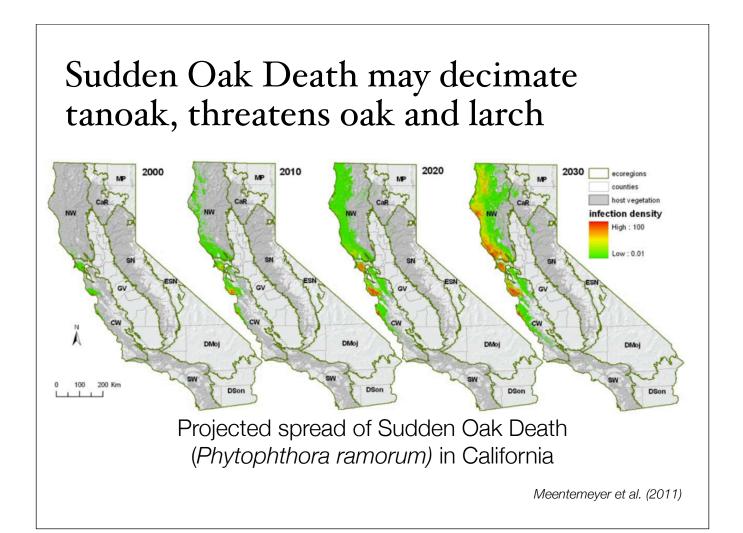
Modern chestnuts reduced to shrubs

A tree killed by chestnut blight

Ellison et al. (2005)

2. Here I'm showing the example of American Chestnut and Chestnut blight. 100 years ago, chestnut was a dominant overstory tree in many eastern forests. The introduction chestnut blight, a fungal disease, decimated the chestnut and removed essentially all adults. Chestnut today is an understory shrub because it sprouts from roots, but can never grow to adulthood. As a result, these forest systems have been restructured. Other trees dominate the overstory, and ecosystem functions like nutrient cycling have been altered. Dutch elms, hemlocks, and many other trees have been reduced in number due to introduced diseases.

Here in California, we have a disease called Sudden Oak Death which was first noticed in the 1990s. It arrived in nursery rhododendrons brought from Asia, and has rapidly spread north and south from the Bay Area. Ten years ago, Dave Rizzo identified the water mold *Phytophthora ramorum* as the disease causing agent.



3. It can reside in many trees, and it kills various oak species, especially tanoak, which could go the way of the chestnut if the disease spreads as projected. It also has the potential to damage economically important oak species that reside on the east coast, and has recently been found to be killing Japanese larch in timber plantations in the UK.

Complexity in *Phytophthora* dynamics makes detection and control difficult

- Variation across and within species
- Low detectability in many species
- Short- and long-distance dispersal
- Host-density thresholds

4. In the past 10 years we've learned a lot about this disease, much of which shows just how difficult controlling it might be.

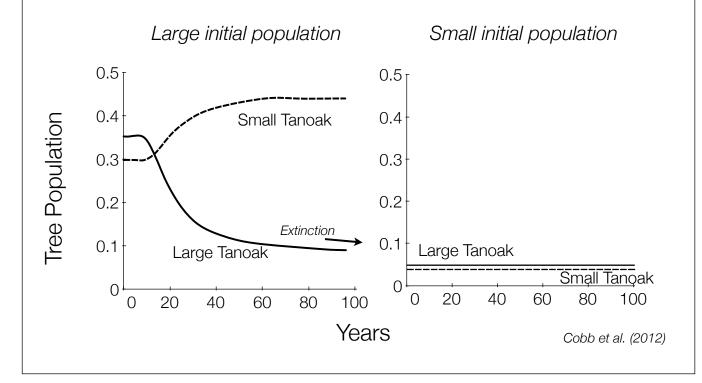
First of all, it kills some species more than others, and produces different amounts of new spores in different species. For instance, it has no averse affects in Bay Laurel, but in produces MANY spores there, and in oak species it can be lethal, but terminal.

Secondly, it can infect some plants without any visible signs for a period, making detection challenging.

It disperses in wind-blown rain, but can also be transported by humans and apparently even fog.

One interesting thing that's been found is that it may have a host density threshold, which has the potential to be a useful tool in management.

Host-density thresholds may inform methods of control



5. Here I show some modeling work by Richard Cobb that indicates this threshold may exist. These two plots show simulations of SOD infection of plots of tanoak with different densities. The axes are time and the tree populations. As you can see, when there are lots of trees, the outbreak the large ones, eventually leading to their extinction, leaving only the small shrubs sprouting from the root systems.

When the population is low and the trees are spaced far apart, though, the infection doesn't take hold, and the population can persist at low levels.

This is something we can potentially add to our toolbox for management

We have a set of imperfect tools for preventing and mitigating outbreaks

- Chemical treatment
- Quarantine and sanitation
- Infectious tree removal
- Pre-emptive removal of high-risk trees
- Species thinning

6. ...and we have several tools, but they are all imperfect and come with costs.

There are chemical treatments for prevention, but they are quite expensive to use over large areas.

Enforcement and education can prevent spread from logging machinery and hikers somewhat.

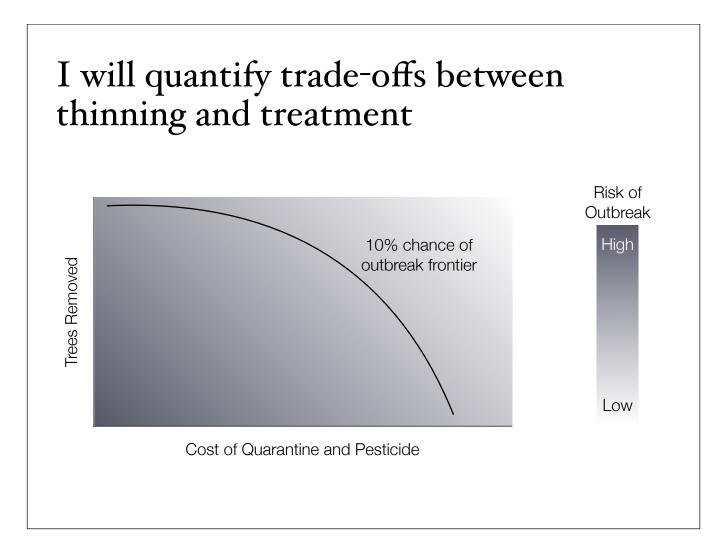
We can remove trees when we see or suspect infection, and even pre-emptively remove ones that could spread disease, even to the point of thinning to get below those density thresholds.



7. But it's important to consider that each of these actions come with trade-offs, and I think its instructive to step back and consider other cases. This image shows cattle being burned in the UK because they had *the potential* to spread foot-and-mouth disease. The UK decided that in many cases it was worth it to destroy these herds to save the cattle industry, even though they were not neccessarily infected but *could* be, and an imperfect but workable vaccine was available. Epidemiological models played a big role in this decision.

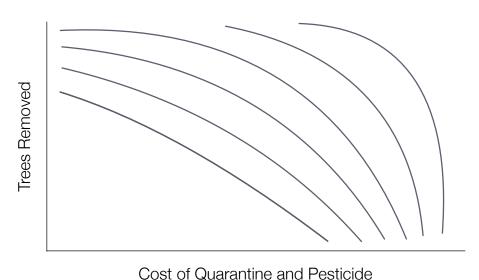
I'm interested in examining this trade-off with sudden oak death, as we have a similar choice with it and other species. Right now, SOD hasn't hit some of the biggest tanoak populations up in Humboldt and Del Norte.

To decide how to protect this population, we have to evaluate the trade-off between removing trees and enacting other protective strategies. Removing trees means losing many of the ecosystem services we're trying to protect, so we want to know how much we gain by doing so.



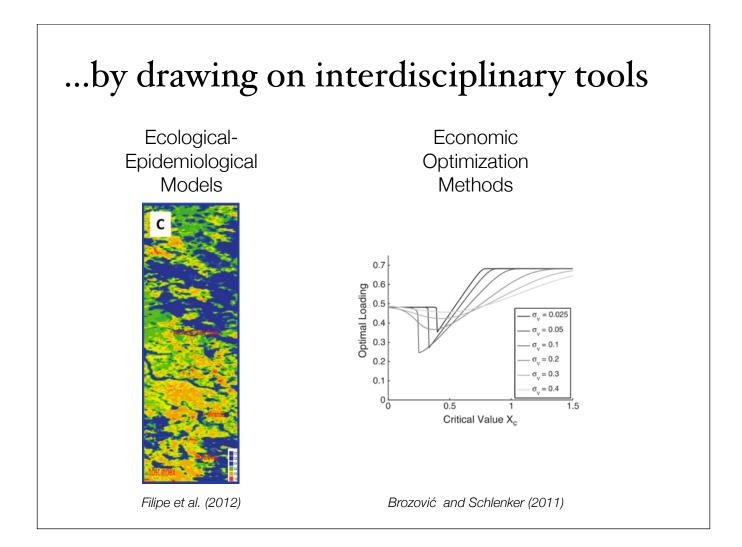
8. This diagram shows a schematic of the relationships that would be important to understand. With a certain level of risk, what's the most efficient balance of these tools that we use. I want to understand the shape of this curve. If we understand it, we can make decisions based on these costs, and the level of services we are willing to sacrifice. It's an interesting way to implicitly value the services of these trees.

...and explore the effects of climate, geography and uncertainty



Oost of Quarantine and resticide

9. Of course, this won't be the same in every location. It depends on how close one is to the advancing disease front, and what the local conditions are that affect disease risk, like climate. What I'm especially interested in evaluating, and what will make this relevant to future outbreaks, is uncertainty. We have limited information on this host-density value and a number of other things. I want to explore how the optimal curve looks as this level of certainty changes.



10. To do this I need the models of my discipline, and the analytical tools of another, which is why its great to have the IGERT umbrella for this project.

Thank you

And thanks to: Alan Hastings, Jim Sanchirico, David Rizzo, Richard Cobb, NSF Reach IGERT

Contact: noamross@ucdavis.edu noamross.net noamross.net noamross

