

FOREST HEALTH IMPACTS OF THE LOSS OF AMERICAN CHESTNUT

Steve Oak
USDA Forest Service, Forest Health Protection,
P.O. Box 2680, Asheville, NC 28802 USA (soak@fs.fed.us)

INTRODUCTION

I consider this meeting and these topics – the restoration of American chestnut and trying to return chestnut to an important ecosystem component in southern Appalachia or in the Appalachians of the Eastern United States – to be very important. My life has been consumed recently with sudden oak death. I leave tomorrow to train the last of three groups of people involved in an expanded early detection survey for sudden oak death. I open that training by helping people visualize what it might have been like in the days, weeks and months after the initial discovery of chestnut blight in the Bronx Zoo and it kind of brings the message home. We don't know if that is what is going to happen with sudden oak death, but it does represent one of the possible outcomes on a continuum from innocuous or no impact to a chestnut blight type of scenario.

FOREST HEALTH

“Health and integrity are not inherent properties of an ecosystem and are not supported by either empirical evidence or ecological theory.” (Wicklum and Davies 1995)

I will start with a bit of discussion on forest health. This would be a short talk if this was the definition that we accepted – that essentially there is no such thing as forest health. Health and integrity are not inherent properties of an ecosystem and are not supported by either empirical evidence or ecological theory. I could stop there, but I won't.

A draft Forest Service Policy was set forth in 1996 and '97. It is still a draft because when you try to bring diverse groups of people together and come to consensus, you end up with chaos, usually. This is still a draft policy, but there are elements that I want you to think about as we walk through the talk.

Forest health is measured at a landscape scale. We are not talking about tree health or even stand health, but forest health – consider it on a landscape scale. The notion of forest health carries with it the idea of ecological integrity and that forest components and relationships are all present, functioning, and self-renewing. And you can imagine what the elimination of chestnut as a functioning ecosystem component did in the early part of the 20th century – how was that affected, that ecological integrity component? Forest health also has a human dimension – the idea that forests should provide for human values, uses, products, and services. And those values etc. are fluid; they change with our ideas about why forests are important.

It's appropriate that the previous presentation was about forest history and what was originally here. Ten thousand years ago the forest composition was quite different from what it is today. That pushes back the perspective from what it was like at European settlement and when the first native people were in this area to the idea that these forests are nothing if not ever-changing as a result of the way that people interact with the forest. I will start with this supposition that southern Appalachian forest landscapes are unprecedented in history. There's never been anything like what you see here today. And the forest that will result in decades hence from what is there today will be like nothing else that has ever existed in the

past. The components of these ecosystems were already in place, I've read, about 58 million years ago, ebbing and flowing with ice sheets and fire. But it really wasn't until the last 10,000 years or so, or maybe even more recently than that, that we've had forests that resemble in structure and composition what was present at European settlement. So why has there never been anything like what we see here today? Of course one important thing, and perhaps the most significant element, was the introduction of the chestnut blight, with ground zero at the Bronx Zoo in 1904.

PROGRESSION OF THE CHESTNUT BLIGHT

It may be something of a fallacy to think of the chestnut blight moving through the eastern hardwood



Figure 1. Chestnut blight distribution in 1909 (Metcalf and Collins 1909). ● = Bedford County, VA.

forests in a wave, nothing in front of this wave and devastation behind it. But I draw your attention to a little spot of infection in Bedford County, Virginia (circle in Figure 1), four or five years after discovery of the blight in New York, well in front of the general advance. There is no way that occurred from a continuous spread, and I suggest that this and many other infection sites were the result of subsequent introductions or movement of infected material either prior to or after the discovery in New York.

In 1911, the infestation in Bedford County, Virginia, was well ahead, or outside of, what might be referred to as the advancing front (Figure 2). Maps from literature published in the 1920's about the progress of the blight through the southern Appalachian assessment area show infection in Greenville County, South Carolina/Henderson County North Carolina, in 1926 (Figure 3). It was known that at the border between Polk, which is the county immediately to the East of Henderson and Greenville County, there was an infestation dated back to 1912, based

on the regular increments and dating of cankers at that location. So, there in 1912, and in 1908 in Bedford County, shows that it was not a continuous spread, not an even wave running through the system.

The blight wasn't the only thing going on (in the woods) at that time. There was heavy duty forest utilization. What I try to point out to people, is that what forms the structure of today's forest is a result of not just the chestnut blight, because land use practices and events immediately prior to and just after the chestnut blight were very important as well. Some of those were fire and heavy utilization, and then with regard to fire, not just the presence of fire, but then the almost complete absence of fire following the Weeks Act and the formation of the National Forest and Cooperative Forest Fire Control Programs in the states. So you went from a heavy disturbance regime, introducing chestnut blight on top of that, and then ceasing most heavy disturbance activities.

Table 1 summarizes the pre-1900 and current conditions of the southern Appalachian forests. The southern Appalachian forests before 1900 were dominated by American chestnut in many places. Whether this was an artifact of disturbance by native people or early European activities is less relevant than what was there and being impacted at the time. But anywhere from a quarter to a third, depending on the inventory that you read from the period, in this core Appalachian area of North Carolina-Tennessee-North Georgia-Virginia, had sparse understories, large, widely spaced overstories, and a high level of disturbance from farming, logging, and fire. When fire regimes were altered, and with oaks already an important part of the forest, oaks were positioned to take the newly available space that was

made available with the loss of chestnuts. So now we have dense understories, dense overstories of somewhat smaller diameter trees, and relatively low disturbance regime as compared with the historical past. And then there was the introduction of the gypsy moth, a non-native defoliator, fires suppression programs and a growing human population. These are the backdrops against which we interpret forest health changes.

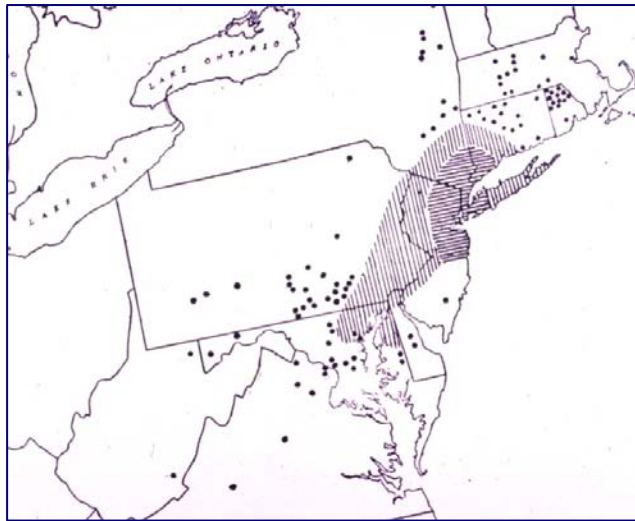


Figure 2. Chestnut blight distribution in 1911 (Metcalf 1912).

Figure 3. Chestnut blight epidemic in the southern Appalachians in 1926.

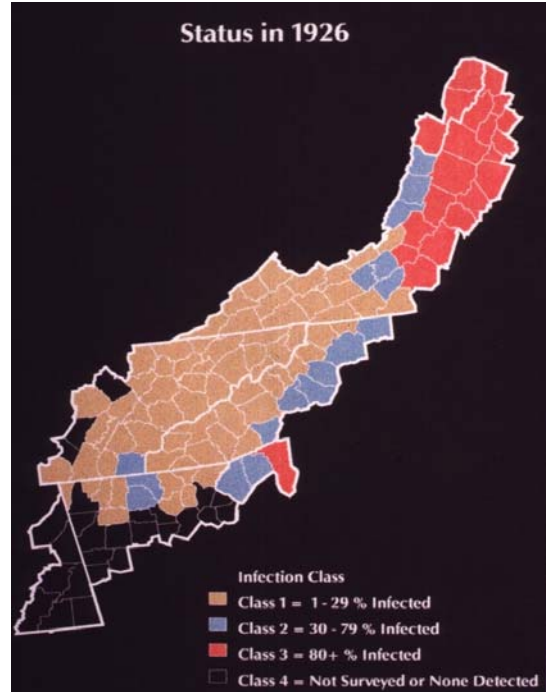


Table 1. Composition, structure, and disturbance profiles of southern Appalachian forests, pre 1900's vs. present day.

Pre-1900	Current
American chestnut	Aging oak cohort
Sparse understory	Dense understory
Large overstory	Dense overstory
High disturbance	Low disturbance
Farming	Gypsy moth
Logging	Fire suppression
Fire	Human population

FOREST HEALTH IMPACTS OF THE LOSS OF AMERICAN CHESTNUT

This is a quote from Smith (1976) from the 'Changes in Eastern Forests' article in *Perspectives in Forest Entomology*:

"We are perhaps entitled to speculate that our chronic and alarming problems with the gypsy moth and other oak defoliators in the eastern or Appalachian portions of the mixed deciduous forest could be as evil a consequence of the chestnut blight as the loss of chestnut itself."

Oak decline is a disease that I will be discussing as a major forest impact of chestnut blight. Again, these oaks came in as a relatively even-age cohort after the loss of chestnut. They have grown up pretty much without disturbance since. People who drive the parkway up on the ridge above you here look out at the landscape and think that it has always looked like this. It is wonderful that we have this preserved area, but in fact this landscape is probably less than 100 years in the making.

What is oak decline?

The symptoms of oak decline are a progressive dieback from the top down and outside in, on dominant and codominant oaks trees that have proved their competitive metal over the decades. Again, decline is progressive from the standpoint that it may take years or even decades to progress from those initial symptoms to more advanced symptoms. In late stage symptoms you have epicormic sprouts coming off the main stem. There can be a gradation of twig condition, from twigs that still have buds on them, and are very recently dead, to branches that have dieback. But these are signs of a progressive dieback, taking years or even decades, progressing to mortality in susceptible trees. The species in the red oak group are more susceptible to oak decline mortality than those in the white oak group.

According to Sinclair (1965), oak decline etiology begins with factors that predispose the tree to decline (predisposing factors):

- Soil depth and texture
- Species composition
- Competition
- Physiologic age
- Topography
- Climate trends, past events
- Air pollution

These are longstanding conditions that predispose trees to effects that we will discuss. But one in particular, physiologic age, is different from chronological age. An 80-year-old tree is not an 80-year-old tree; it depends on where it is growing. An 80-year-old tree on a poor quality site, or a low productivity site, such a site index of 60, is more mature physiologically than that same age tree growing on a more productive site, say with a site index of 80. And we use this in modeling work to predict where oak decline is likely to be a problem.

The second group of factors are the inciting factors (Sinclair 1965):

- Defoliation
- Drought
- Frost
- Stand disturbance
- Air pollution

These factors are relatively short term, occurring at a point in time or a period of time that can be identified with the inciting event. And defoliation, spring defoliation in particular, is an important factor here. What happens with spring defoliation is that the carbohydrate chemistry of the tree is altered. Food is stored in roots as starch. In times of stress, such as when the crown is removed, the tree has to mobilize that starch into sugars.

Finally, there are the contributing factor (Sinclair 1965), such as:

- Root pathogens
 - Armillaria root disease
- Canker pathogens
 - Hypoxylon
 - Shoot cankers
- Boring insects
 - 2-lined chestnut borer
 - Red oak borer

Root diseases, for example, can take advantage of a tree weakened by inciting factors through recognizing chemical changes in the roots and then switching from a saprophytic to a pathogenic relationship with the tree. These include *Armillaria* root disease, and in particular *Armillaria mellea*.

Using FIA data points of various dissections of forest type, the oak forest type is the most common one in the East, of course, and the message is that “there sure is a lot of oak out there” (Figure 4). When plots are displayed that are ‘vulnerable’, meaning that they have a relatively high basal area of oak, these are really saw timber and pole timber stands that have a high concentration of oak (using size as a surrogate for age). Vulnerable plots are concentrated in the Appalachian Mountains, the Blue Ridge in Virginia, the Eastern and Western Highland Rims in Tennessee, and the Ozark Mountains in Arkansas. “Affected” stands in Figure 4 are those in which oak decline symptoms are actually present, and these reveal a pattern. There is about 3.6 million acres of oak forest type in the 12 southern states of this region, about 10 percent of the total in the East. Oak and oak decline are especially abundant in the southern Appalachians, where chestnut would have been concentrated.

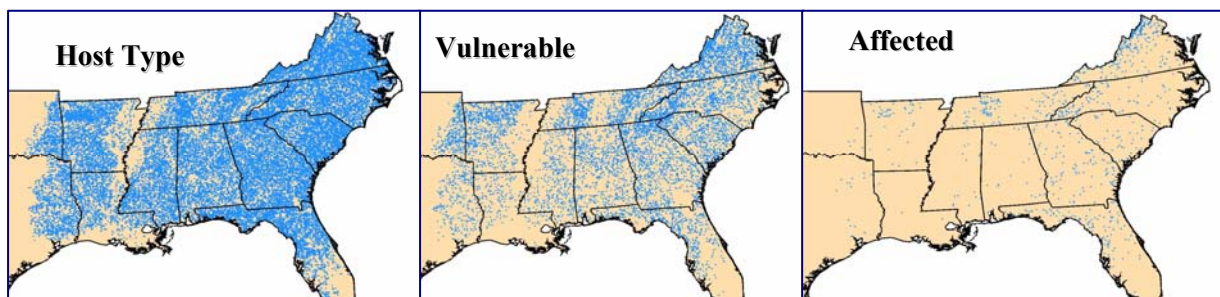


Figure 4. FIA oak decline analysis for the USDA Forest Service’s Southern Region, 1984-1989 inventory cycle.

We mentioned earlier that defoliators have an important impact. The fall canker worm is a common defoliator, but probably made more serious by the loss of chestnut and its replacement with oak, as their favorite food is oak leaves. There was an outbreak of fall canker worm on some 10,000 acres on the Blue Ridge Parkway a couple of years ago that resulted in some tree mortality. This is an example of why spring defoliation is important in the oak decline scenario and in general tree health. Oaks produce an instant crown in the spring. If something comes along and removes those leaves in the first few weeks, then the tree has to make a decision about replacing that foliage, and starch needs to be converted to sugars from the roots. Spring defoliation stimulates a refoliation before the starch can be replenished, and

then you get the root diseases coming in. And the lesson also is that compounding stresses such as defoliation in combination with drought unhappily occur together frequently. Nitrogen content in leaves go up in drought periods, which makes it more palatable to insects, a positively-reinforcing loop. When predisposed oaks of advanced age are defoliated in the spring, combined with drought, disaster is waiting.

Another added element is the gypsy moth, a non-native defoliator. The male has feathery antennae and the sex pheromone is from the female, which doesn't fly. Unhappily, the gypsy moth prefers oak species as host; they love to eat oak leaves. Among the more resistant species is the dearly departed American chestnut, and there is another array of hosts that are also relatively immune (Table 2.). Some other immune hosts are species that we do not need necessarily need more of. The bottom line is that the replacement of chestnut, a relatively resistant host to the gypsy moth, with the much more preferred oak again has forest health implications, especially in the oak decline scenario.

Table 2. Tree host preferences for gypsy moth.

Gypsy Moth Preferred Hosts	Gypsy Moth Resistant Hosts	Gypsy Moth Immune Hosts
Oak species	American chestnut and beech	Ash
Basswood	Cottonwood and sourwood	Fir
Sweetgum	Sweet and yellow birch	Grape and holly
Serviceberry	Hemlock and pines	Black locust
Hornbeam, hop-hornbeam	Blackgum and buckeye	Sycamore
Willow	Walnuts and hickories	Yellow-poplar
Apple	Black cherry and elms	Striped maple
Aspen	Cucumbertree and sassafras	Dogwood
Gray, paper, and river birches	Red and sugar maple	Mountain-laurel

Outbreak frequency, severity, and periodicity tend to be different between native and non-native defoliators, and this has forest health implications. Outbreaks of non-natives tend to be more severe and have shorter return intervals than outbreaks of native species.

So to summarize the chestnut blight-oak decline-gypsy moth interactions, we have an introduced pathogen superimposed on an altered forest due to the loss of chestnut and replacement with oak. That is an oversimplification; oaks weren't the only species to come in, but they were a very significant component to replace chestnuts. But when you impose the interacting factors of oak decline, gypsy moth, forest composition, and existing composition, oaks will decrease as a result of oak decline. This is somewhat site specific. Sometimes oaks replace themselves, but often they don't. The usual case is that there is incomplete oak replacement. So when a forest has 40-60 percent oak prior to these disturbances, you may end up with 20-25 percent remaining afterwards. Nobody projects that oaks will be lost completely; you couldn't get rid of them if you wanted to. There is an increase in the taller, mid-story species, and it is the same scenario as what happened when oaks were positioned to take newly available space when the chestnut went out. You get shade tolerant mid-story species as a result of going decades without disturbance (no fire, no cutting, or very little anyway). You have a build-up in the mid-story of shade tolerant species like red maple, blackgum, and sourwood. Of course, this does not matter if all you want is something green out there. But if you place differential value on different species, then this could be a bad result, especially with regard to wildlife habitat components.

Evidence of forest composition change

Unpublished data from the USDA Forest Service’s Forest Inventory and Analysis (FIA) unit was assembled by Bob Anderson, recently retired. This was a study of a cluster of counties in northern Virginia where gypsy moth, oak decline, and dogwood anthracnose have come together over a number of decades. Between 1977-92, approximately three inventory cycles, there was a major change in trees 17 inches and larger in diameter at breast height (Figure 5). The bottom line is that the large-tree component increased dramatically, especially for eastern white pine but other species, also. That is a positive change. But the picture is very different at the other end of the size spectrum. In the trees 1-5 inches in diameter, which are going to be the next forest, over the same period of time, eastern hemlock showed a fairly robust increase but all other species declined. At the bottom, with the most negative changes, were the oak species. So the next forest is probably going to have a smaller oak component.

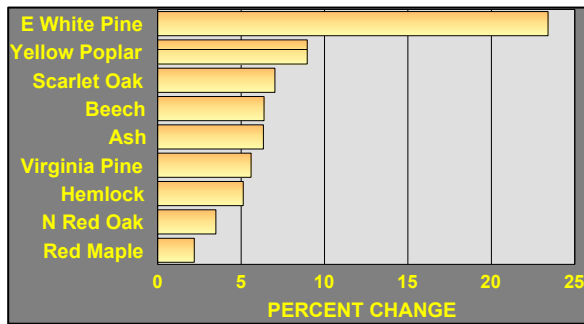


Figure 5. Forest composition in northern VA, change in trees 17+” d.b.h., 1977-92.

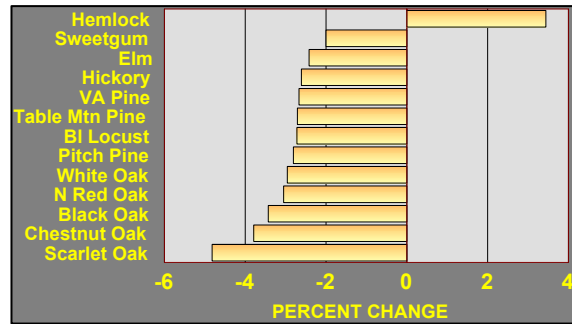


Figure 6. Forest composition in northern VA, change in trees 1.0-4.9” d.b.h., 1977-92.

All of these changes have consequences for wildlife habitat. The mast quality and quantity is reduced, and this has consequences not only for food for mast-loving wildlife, but also in oak regeneration opportunities. This must be put in the context of oak as an incomplete surrogate for chestnut, and what chestnut provided in decades past. We have an increase in small openings, not such a bad thing in some contexts, depending on which wildlife species you are talking about and the landscape you are dealing with. There will be a change in species composition, both in the abundance and diversity of oaks, since the red oak group is more susceptible to the decline mortality than is the white oak group. Reduced canopy density, an increase in denning sites for types of wildlife, and structural changes from dead and downed wood, standing snags and so forth could be a good thing. But how many dead snags do you need in a landscape before they can no longer be exploited by the available wildlife populations? We tried to model what the effect of oak decline would be on acorn production. If all standing trees were alive, healthy, and producing an average amount of mast per year, the annual mast production would be somewhere on the order of 280 pounds per acre. But, of course, many of those trees aren’t alive. A real stand was modeled in Virginia, on the Deerfield Ranger District on what is still the GW Jefferson National Forest. Mast production from the dead oak was, of course, zero, and some trees had partial crown dieback and partial reduction in their mast-production capacity. Instead of 280 pounds per acre, the stand was producing 168 pounds on average. Projecting the current pace of decline, knowing that red oaks decline faster than white oaks, we predict that within 10 years of this inventory there will be only 115 pounds per acre. Again, superimpose this on the context of a chestnut forest prior to its loss and replacement with oak. We don’t have an accurate number for the mast production of chestnut historically on this kind of a site, but it might have been measured in tons per acre rather than hundreds of pounds per acre.

Sudden oak death and chestnut

How does sudden oak death, or the potential of sudden oak death, fit into this? I tell people that there is a wide spectrum of possible outcomes with sudden oak death, from a chestnut blight type of scenario to innocuous. Sudden oak death was confined to the West Coast (and Europe) until March of this year. It wasn't in the East until the disease (caused by *Phytophthora ramorum*) was shipped on nursery stock to virtually all of the states plus Puerto Rico and the Virgin Islands. However, introduction does not necessarily mean establishment. So what does sudden oak death look like? The diagnostic symptom is a



Figure 7. *Phytophthora ramorum* diseases – bleeding stem canker, shoot dieback, and leaf blight (clockwise from left).

bleeding stem canker on oak, but there are a lot of agents that cause cankers on oak stems. So bleeding cankers are not strictly speaking diagnostic, but a good clue. The bleeding is a running, wine or burgundy colored ooze (Figure 7). Underneath the bleeding spot are irregular lesions. On other species, *P. ramorum* infection may cause only shoot dieback (madrone) or leaf blight (California-laurel). It has been said that sudden oak

death is neither sudden, doesn't affect only oaks, and doesn't always result in death. So maybe that is not a good name. But it has crept into the common usage. You would have to say at the low end of the symptom scale that it might be 5 or 10 years from the infection to mortality. We haven't been looking long enough to know if some trees can recover. It doesn't appear so.

Prior to March 2004, the distribution of *P. ramorum* in North America was thought to be confined to the West Coast, to 12 central coastal California counties plus Curry County, Oregon, just north of the California border and a couple of hundred miles north of the most northerly known site in California. We tried models to guide our survey efforts, to have a risk-based survey and to focus our resources in places where we were most likely to find this disease. We looked at climatic variables where the disease exists on the West Coast, and combined those with distributions of known potential hosts. As Figure 8 shows, there appears to be a heavy risk of sudden oak death in the southern Appalachians.

On March 10, 2004, it became known or confirmed that *P. ramorum* pathogen was present in the Monrovia nursery in Los Angeles, California. Shipments of nursery stock from Monrovia, and another nursery called Specialty Products, to eastern destinations may have contained infected material. *P. ramorum* has been confirmed in nursery stock sent to Maryland, Virginia, North Carolina, Tennessee, Georgia, Florida, Louisiana, and Texas. Testing is continuing. Just because states are not known to have the shipments yet, does not mean it's not there. It is just that the testing is still underway in many of those places.

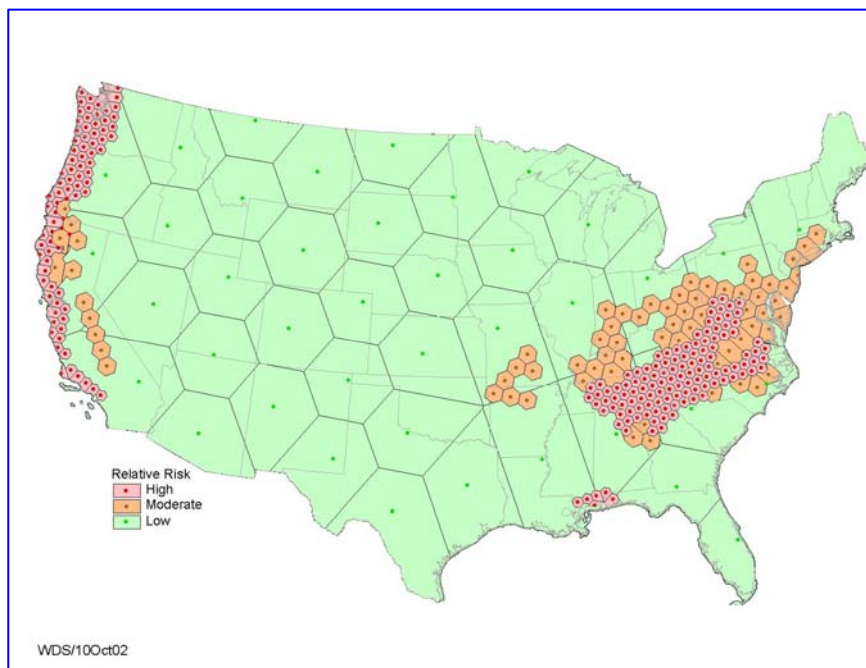


Figure 8. Preliminary sudden oak death risk/hazard map.

LITERATURE CITED

- Metcalf, H. 1912. The chestnut bark disease. P. 363-372 in Yearbook of the department of agriculture for 1912, Washington, D.C.
- Metcalf, H., and J.F. Collins. 1909. The present status of the chestnut bark disease. USDA Bull. 141 part 5, Washington, D.C., p. 45-53.
- Smith, D.M. 1976. Changes in eastern forests since 1600 and possible effects. P. 1-20 in Perspectives in Forest Entomology, Anderson, J.F., and H.K. Kaya (eds.). Academic Press, New York.
- Wicklum, D., and R.W. Davies. 1995. Ecosystem health and integrity? Can. J. Bot. 73:997-1000.