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Your Source For Accurate Hardwood Pricing Information

Sudden Oak Death

Could it Eclipse Chestnut Blight and Dutch Elm Disease?

By now, you've likely heard of Sudden Oak Death (SOD), a disease first noticed by nursery growers in California in 1995. In 2000, scientists identified the cause of Sudden Oak Death as a previously unknown "water mold" called *Phytophthora ramorum* (we'll call it *Pr* for simplicity). *Phytophthora* molds have been around throughout history; one species was responsible for the Potato Blight and Great Famine of Ireland in the late 1840's. SOD has caused widespread dieback of Tanoak, Coast Live Oak and Black Oak in California, and infestations have been discovered in Oregon and Washington. It also is known to cause twig and leaf diseases in Douglas-fir and Redwood.

Until this year, SOD was largely a western U.S. problem, affecting the sale and transport of many nursery plants in California, Oregon and Washington. Late this winter and early this spring, however, infected host plants (rhododendron, mountain laurel, camellias, azaleas) were shipped from a wholesale California nursery to more than 100 nurseries in 13 eastern states. By the time the originating nursery was found to be contaminated with *Pr*, hundreds of potentially infested plants were sold to and planted by eastern homeowners.

While *Pr* has yet to be detected in any eastern forest area, Sudden Oak Death is clearly no longer just a western problem. In fact, the potential ramifications to eastern U.S. and Canadian forests are no less significant than were Chestnut Blight and Dutch Elm Disease in the first half of the 20th century. According to Dr. Kurt Gottschalk, research forester with the Forest Service's Northeastern Research Station in Morgantown, WV, it may be several years before we know whether the recent transport of infected nursery stock allowed *Pr* to get established in eastern forests. In fact, he points out, efforts are still underway to track down all of the infected nursery stock. But, if *Pr* does get established, whether through the recent nursery stock shipments or some other introduction, SOD could radically alter eastern hardwood forests and the forest products industry.

Before looking at Sudden Oak Death and its potential impacts in detail, let's briefly revisit the historic impacts of two other introduced pathogens:

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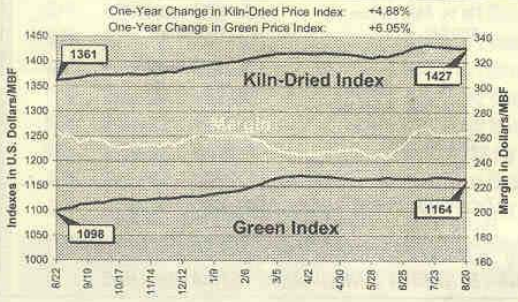
Hardwood Lumber Price Indexes



Indexes represent the average published prices of 14 key hardwood items. The margin is the difference in dollars between the kiln-dried and green indexes, as read from the right-hand axis.

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Editorial

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Chestnut Blight

Chestnut Blight was introduced sometime in the late 1800's, most likely on infected Japanese Chestnut nursery trees that were widely imported for orchards. In 1904, Chestnut Blight cankers were first discovered on American Chestnuts in the Bronx Zoo. By 1940, the fungus was reported throughout the native range of American Chestnut, having progressed roughly 200 miles per decade south from New York to Georgia. By 1950, the entire eastern U.S. population of American Chestnut—the keystone species on some nine million acres—was killed off; only a few survivors and small stump sprouts remained.

Prior to the blight, American Chestnut was a dominant tree of Appalachian forests from Canada to Georgia, accounting for one in four trees in the region. In many forests, American Chestnut was the most numerous and often the largest tree. In 1911, for example, some Pennsylvania counties had 40-50 percent of their timber acreage in Chestnut.

Dutch Elm Disease

Dutch Elm Disease (DED) is actually believed to have originated in the Himalayas or elsewhere in the Orient, but it was first described in Holland in 1921, and the 'Dutch' name stuck. The first U.S. case of DED was discovered in Cleveland in 1930, apparently arriving in either a shipment of logs from France or from wooden crates made from infected lumber. DED spread east quickly, and within two years, was killing trees in New Jersey.

American Elm was the predominant urban street tree in many American cities even as late as the early 1970's. Various sources put the American Elm death toll by the mid-1970's at between 43 million and 77 million trees. A University of California-Berkeley report suggests hundreds of millions of Elms are now dead, and the disease is still spreading west through Manitoba and Saskatchewan.

Sudden Oak Death

In both cases, it took roughly 40 years from the time of their introductions for Chestnut Blight and Dutch Elm Disease to virtually wipe out the entire populations of their host species. While neither tree is extinct in North America, their sprouts and saplings rarely grow to merchantable height before succumbing to their respective diseases. A select few mature, high-valued American Elms are kept alive today with expensive stem injections in prominent places like the Washington, DC Capital Mall, but such measures aren't widely practical. It is not out of the question that Sudden Oak Death could similarly eliminate Red and/or White Oak from productive timberlands by 2035.

In many respects, SOD is showing signs of being potentially much more aggressive and devastating than either of the historic diseases we've discussed, although scientists are still working to understand the fungus-like *Pr*. Nobody knows what will happen when and if it escapes into eastern hardwood forests (we believe the question is more likely *when* than *if*). Remember, scientists have only known about *Pr* for 4 years, and know very little about its behavior outside of the laboratory. What we do know about it and its potential impacts on eastern forests, however, is frightening.

Life Cycle & Hosts

Pr manifests itself as three different types of diseases: leaf spot, twig blight and stem canker. Few host species exhibit all three, but *Pr* requires a foliar host (one which produces leaf spots) in order to produce spores and reproduce. Tanoak produces leaf spots, and thus is a suitable foliar host. The true Red Oaks in California, on the other hand, develop cankers but not leaf spots. As such, Gottschalk calls the Oaks a "dead-end" for the fungus; *Pr* will kill the Red Oak, but it requires an alternate host (such as rhododendron or azalea) to complete its life cycle. Laboratory evidence, however, suggests that *Pr* may behave quite differently in eastern Oaks.

Scientists have already determined that *Pr* can produce leaf spots on Chestnut Oak (a member of the White Oak group widely spread throughout the Appalachian Region), although those spots have not yet been tested for spore production. The spots are significant because White Oaks have not been affected in California. They've also determined that Chestnut Oak (*Quercus prinus*) and White Oak (*Q. alba*) samples artificially inoculated with *Pr* grow cankers at faster rates than either Northern Red Oak (*Q. rubra*) or the California Oaks. Because White Oaks haven't been affected in California, Gottschalk surmises that White Oaks may have better natural defenses to infection in their bark. Nobody knows yet whether eastern White Oaks will show similar natural resistance in the wild. Certainly White Oaks that have

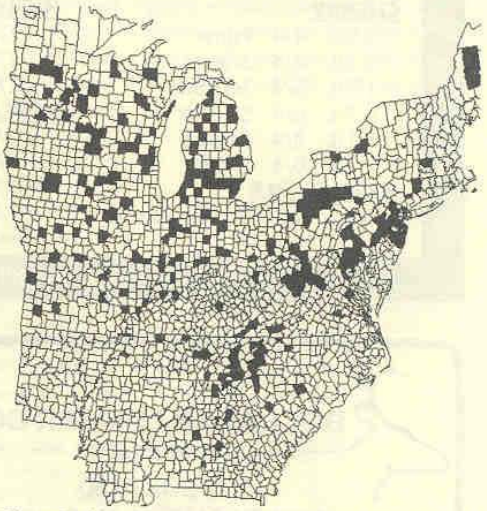

Figure 1. Counties with more than 2,000 board feet of Northern Red Oak (*Quercus alba*) sawtimber per acre of timberland.

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had their bark damaged by logging would be susceptible.

Finally, evidence from a planted specimen in the UK confirms that mature Northern Red Oak trees can be infected in the wild, something only speculated about until recently. European White Oaks and European Beech were also found to produce good *Pr* canker growth in the laboratory.

In summary, we know that Northern Red Oak is highly susceptible to SOD infection in the wild. Despite its regionally misleading name, the species Northern Red Oak (*Quercus rubra*) is a major timber species throughout much of eastern North America. If that species alone were to succumb, we'd lose an enormous volume of the standing timber resource in the eastern United States (Figure 1). Unfortunately, there's a strong possibility that other oaks in the Red Oak family and possibly the White Oak family may also fall prey.

No Effective Treatments

There are no known treatments for trees infected with Sudden Oak Death; most will die within 1 to 3 years. The only way to stop an infestation is to remove and incinerate dead and infested plant material and soil. However, at this point, says Gottschalk, nobody knows how large an area around an infestation needs to be treated to ensure containment.

A phosphate-based pesticide treatment was found to slow canker growth or prevent infection in California, but like Dutch Elm Disease, such treatments applied with plastic syringes and power drills aren't practical on a forest scale. In addition, Matteo Garbelotto, the UC Berkeley pathologist that first identified SOD in 1995 and also developed the pesticide, said that the discovery of a second strain of the disease suggests it may be more resistant to treatment than first thought. In a washingtonpost.com article, he noted, "What happens when something of this size hits in the public health field? You get an international outcry about SARS. But when an environmental crisis of this scope hits, no one pays any attention—it flies under the radar."

Multiple Vectors Encourage Spread

Like Chestnut Blight and DED, SOD benefits from multiple natural and human dispersal mechanisms. SOD won't spread naturally as fast as Chestnut Blight, whose spores were blown long distances by the wind. Rather, the natural movement of SOD spores is primarily through rain splash. Like Dutch Elm Disease, however, natural spread is not the real fear. Human transport is the most likely mechanism for SOD to rapidly be spread long distances. The spread of infested nursery stock is a good example, but dormant SOD spores in the soil can also be picked up and transported on hiking boots, mountain bike tires, etc.

What's Being Done

County, state, federal and international quarantines are in place to stop the spread of SOD to uninfected areas. As vividly demonstrated this year, however, quarantines are only effective if they totally contain the infestation. One of the problems in defining infested areas in California, according to Dr. Gottschalk, is that spores can typically only be detected by standard culture tests from April through early June. This year's infected plants were shipped prior to that detection window. While DNA tests can detect *Pr* year-round, these tests are more expensive and were not widely used.

Massive federal and state efforts are underway to "trace-forward" and "trace-back" all of the infested plants from this year's California shipment. On May 18, Agriculture Secretary Ann Veneman transferred $15.5 million to the Animal and Plant Health Inspection Service (APHIS) to facilitate these surveys and beef up quarantine efforts.

Forest Service Research has also geared up to fight Sudden Oak Death; $1.9 million was committed in FY 2004 and over 30 projects are currently being funded. Much of the research has focused on defining risks and pathways of spread from nurseries to homeowners' yards to forests. At this point, the best hope for controlling SOD is preventing its introduction into eastern forests.

Some Hope?

Amid all this doom and gloom, remember that SOD has not been determined to be loose on the eastern landscape. While the prognosis isn't good, several aspects of *Phytophthora ramorum*, its life cycle and its host species do offer some hope that SOD may not impact all Oak regions equally.

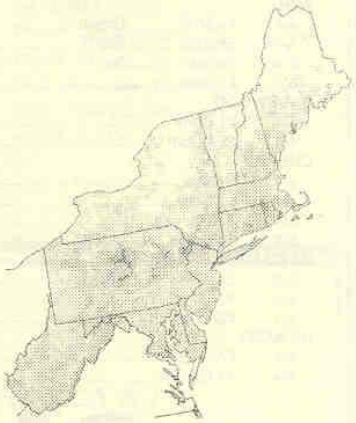

Figure 2. Darker-colored areas more probably contain both overstory and understory hosts (Source: Gottschalk, USFS).

First, SOD leaf spots require 48 hours of continual rain or drizzle (or perhaps fog/high humidity) to trigger spore production. While some of the spores (chlamydospores) are thick-walled and hardy, most of the spores (zoospores) can only survive in a water droplet. If they dry out, they die. In most areas of the East, 48-hour rain events are relatively rare. And, except in the case of spores falling into forest streams, natural transport of these spores would be only a few feet in any direction.

Second, SOD cannot survive extreme heat or cold. The Deep

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South, the Northern-tier states and eastern Canada may be naturally protected by their hot summers or cold winters.

Finally, SOD requires the presence of foliar hosts to reproduce. If SOD is introduced into an area without such hosts, it cannot spread and affect other trees. While all of the host relationships are not yet known, Dr. Kurt Gottschalk has developed a risk map for several northeastern states based solely on the presence of known canker and foliar hosts (Figure 2). Weather factors were not considered, but might further reduce the risk in certain areas. None of this is to say areas in these states outside the high risk zones are in the clear. But, it might mean that certain areas are less prone to infection and tree mortality than others.

Eastern Forests 2035

Based on the historic impacts of major introduced pathogens, the rapid spread of SOD up the Pacific Coast, SOD's broad host range, and the quick kill of SOD cankers, it is very likely that Sudden Oak Death will become established in eastern forests and that Red and White Oak will play a much reduced role in eastern forests 30 years from now.

Even without SOD, Oak regeneration has struggled as many of the natural regenerative tools that Oak relied on (regular fires, clearcuts) have been taken out of play. In the five years between 1992 and 1997, the percentage of eastern hardwood sawtimber in Oaks declined from 40.3 to 39.2 percent. Oak sawtimber volume grew 1.6 percent during the period, while total hardwood sawtimber volume grew 4.5 percent. While we saw this slow species shift coming, SOD could very well speed up the timetable.

Finally, while Chestnut once composed 25 percent of the sawtimber in the Appalachian Region, Oaks presently account for 40 percent of the hardwood sawtimber harvest east of the Great Plains! If the worst case scenario comes to pass, our children and grand children will know an entirely different hardwood forest than we know today.

Information Sources: American Phytopathological Society, The American Chestnut Foundation, Virginia Tech, New Georgia Encyclopedia, UC Berkeley, USDA Forest Service , Kansas State Univ., ELMguard.com, Animal and Plant Health Inspection Service, CA Oak Mortality Task Force, Washington Post. Special thanks to Dr. Kurt Gottschalk, USFS.