Northern California Wildfires Likely Exacerbated By Sudden Oak Death

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By Peter Fimrite

A dramatic increase this year in the number of oaks, manzanita and native plants infected by the tree-killing disease known as sudden oak death likely helped spread the massive fires that raged through the North Bay, a UC Berkeley forest ecologist said Thursday.

Dead and dying oak trees make wildfires hotter and cause them to spread more quickly, said Matteo Garbelotto, the director of the UC Berkeley Forest Pathology and Mycology Laboratory.

In a study released Friday, Garbelotto's laboratory found that 37 percent of the trees sampled in fire-ravaged eastern Sonoma County -- prior to the fires -- were infected by sudden oak death, a ten-fold increase compared with two years ago. The areas hardest hit by the disease were Cloverdale, Healdsburg, Santa Rosa and Glen Ellen. Petaluma and Sonoma east of Highway 12 also were hard hit.

The virulent pathogen, which thrives in the aftermath of heavy rains, has infected 13 percent of trees along the California coast, according to samplings this year from 15,000 trees.

Garbelotto said it is the worst outbreak of the disease he has seen since he initiated the testing program in 2007.

Firefighters are still investigating what started the wildfires late on the evening of Oct. 8. They say powerful winds that night in Napa and Sonoma counties propelled the blazes from wildlands into populated areas. The fires were so intense, experts say, that in Santa Rosa they produced fiery tornadoes that flipped cars, uprooted trees and ripped garage doors from their hinges. At least 42 people were killed in the Northern California fires, and recovery teams are still sifting through rubble for remains.

Neither Garbelotto nor fire officials know how much of an impact, if any, sudden oak death had on the fires, but dead oak burns well and keeps burning a long time. Live oak trees are naturally resistant to fire. In fact, Native Americans used to set fires and collect the acorns dropped by oak trees after the area had burned.

"Certainly when I look at the map I see a very obvious overlap with the incidence of sudden oak death and where the fires were," Garbelotto said. The infections "were mostly in forested areas, but there was sudden oak death in almost every one of the big fires."
The Sonoma County Hazard Mitigation Plan, issued in April, warns of increased fuel loads from trees killed by sudden oak death.

Garbelotto's laboratory published a map this week pinpointing infestations, often in urban settings, stretching from Santa Barbara County to the Oregon border. The number of trees infected statewide doubled compared to a year ago and tripled since 2015, according to the map, which used samples collected and tested by 600 scientists and volunteers.

The infection rate in the Bay Area and Northern California is also a concern in future fires, he said. Besides Sonoma, huge increases in infections were found in San Francisco, Santa Cruz, East Bay and in the Peninsula.

"California is more susceptible to larger and hotter fires, specifically because of the large number of dying oaks, which are basically match sticks," he said. "We have these large swaths of forest with a large number of infected oaks and tanoaks. Even when they are not dead, their moisture content is much lower than a healthy tree."

Sudden oak death, discovered in Mill Valley in 1995, has spread rapidly in forests and wildlands along the California coast and in southern Oregon.

It kills big oak trees and the smaller understory tan oaks, which have been ravaged in many areas, including Big Sur, Jack London State Park in Sonoma County and China Camp State Park in Marin County.

More than 100 plants are susceptible to the fungus-like pathogen, known scientifically as Phytophthora ramorum, but California bay laurels are the primary vectors, storing up the spores on their leaves and spreading them in the wind and water to nearby oak trees.
The infection rates outlined in Garbelotto's report were calculated based on tests of leaves from bay trees in 17 counties in Northern California.

It has long been known that the microscopic killer throws out spores mostly during wet years -- the infection rate in California increased tenfold in 2012, a year after heavy rains -- so it wasn't a surprise to scientists that the infection rate went up this year. What was surprising, though, was the movement of the disease into urban areas and places popular with tourists.

Infestations were found on the UC Berkeley campus, Golden Gate Park, at the Point Reyes Visitor Center and in the arboretum on the UC Santa Cruz campus. There were multiple positive tests in the Presidio of San Francisco, which, as a whole, has five times

more infections than it had two years ago, according to the test results.

Martinez and Orinda in Contra Costa County; Oakland, Piedmont and El Cerrito, in Alameda County; Redwood City and Saratoga, in San Mateo County; and the Carmel Valley, in Monterey County, also showed significant infections.

"In all of those areas we had infections close to the towns or in areas where people lived, including Santa Rosa where there were several neighborhoods that I know burned," Garbelotto said.

The other surprise, he said, was how the disease has suddenly attacked manzanita, a native species with 100 different varieties in California. Manzanita was a known host, but not many died until this year when numerous plants from seven different species of manzanita were found dead -- six in the Santa Cruz arboretum and one at the Crystal Springs Reservoir.

"They are all very rare and endangered species, so there are a lot of people scrambling to collect seeds," said Garbelotto, pointing out that dead manzanita are also tinder for wildfires.

"This was unpredictable, but it tells us that we still don't know everything this organism can do," he said. "It only arrived 20 years ago. We know that when it rains a lot it's ready to be aggressive and this is what happened this year."

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