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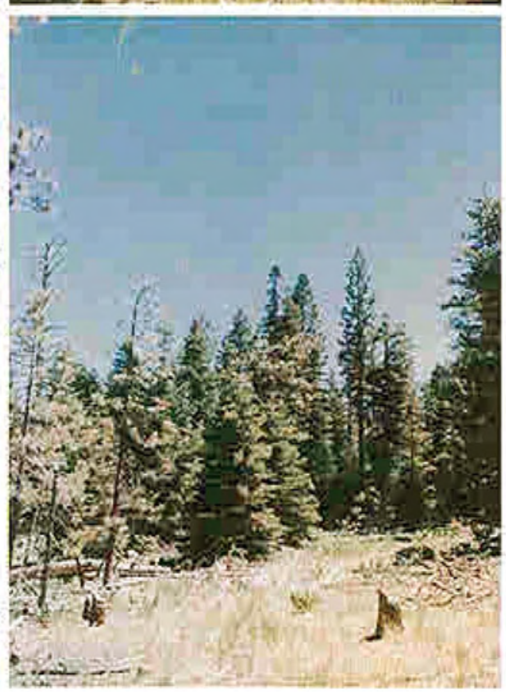
Elytroderma Disease Reduces Growth and Vigor, Increases Mortality of Jeffrey Pines at Lake Tahoe Basin, California

Robert F. Scharpf Robert V. Bega



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Cover: Portion of plot 1 photographed at various intervals after the outbreak of Elytroderma disease *(upper left)* at Lake Tahoe Basin, California. *Upper right*: May 23, 1972—a year after the outbreak; *lower left*: June 15, 1973—both trees in the foreground now dead; *lower right*: June 24, 1976—nearly all Jeffrey pines now dead and removed, and only a few white firs, in center, remain alive.

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1981. **Elytroderma disease reduces growth and vigor, increases mortality of Jeffrey pines at Lake Tahoe Basin, California.** Res. Paper PSW-155, 6 p., illus. Pacific Southwest Forest and Range Exp. Stn., Forest Serv., U.S. Dep. Agric., Berkeley, Calif.

Retrieval Terms: Pinus jeffreyi, Pinus ponderosa, Pinus contorta, parasitic fungi, *Elytroderma deformans*, Lake Tahoe Basin, forest protection, mortality rate.

The fungus *Elytroderma deformans* (Weir) Darker is responsible for the most serious needle disease of pines in Western North America. Ponderosa pine (*Pinus ponderosa* Dougl. ex Laws.), Jeffrey pine (*P. jeffreyi* Grev. & Balf.), and Sierra lodgepole pine (*P. contorta* ssp. *murrayana* [Balf.] Critchfield) are the trees most frequently infected, but some other pine species are also attacked. The fungus usually occurs at low, endemic levels in the forest but in some years on some sites the disease spreads and builds up to epidemic proportions. During and for several years after these epidemic outbreaks, loss of vigor, growth reduction, and tree mortality may continue. The extent to which this damage takes place in Jeffrey pine stands is not well understood.

After a severe outbreak of the disease at Lake Tahoe Basin, California, in 1971, six plots ranging in size from about 0.2 to 1.0 acre were established in or near the disease area. Trees were measured and their general condition assessed at the beginning of the study. Most trees on the plots were of pole size (mean diameter-at-breast height [d.b.h. 8.9 inches), but some saplings and sawtimber sizes were present also. Data on initial distribution and intensity of disease were recorded. Tree mortality was recorded at yearly intervals, and at 2- and 6-year intervals after the outbreak the surviving trees were examined, their condition noted, and disease distribution and intensity reevaluated.

In 1971, about one-half of the trees were heavily infected and about one-half moderately or lightly infected. No uninfected Jeffrey pines were observed. Nearly all trees had the disease distributed through 50 percent or more of their crowns. Severity of infection was not related to stand basal area, tree diameter, or crown class. On the average, trees with heavy infection were of poor vigor.

Host and disease conditions changed somewhat during the 7 years. About one-half of the trees remained unchanged in vigor, or disease intensity, or both. Of the remaining trees, most became more heavily infected and decreased in vigor. Average annual radial growth of test trees was slow—about 1.0 mm per year—before the disease outbreak. After infection, mean annual radial growth was reduced, and growth showed a significant downward trend with an increase in infection intensity. Heavily infected trees were growing on the average of 0.7 mm per year.

Mortality was heavy on some plots and was greatest among severely infected trees. In some cases mortality was associated with Jeffrey pine beetle attack. Of the original 607 trees, 192 or nearly one-third died by 1978.

Our study suggests that in managing Jeffrey pine stands to control disease losses, high-risk sites should be identified on the basis of past or current knowledge about epidemic outbreaks. On these sites, management of nonsusceptible species should be considered. Increasing tree vigor through thinning or by other means will probably not affect the occurrence of epidemics but may help reduce tree mortality resulting from Elytroderma disease and associated insect attack.

Jeffrey pine (*Pinus jeffreyi* Grev. & Balf.) in northern California is often infected with the fungus *Elytroderma deformans* (Wier) Darker that causes a needle and twig disease of pines. Forest managers are concerned with growth loss and mortality that result from this disease and how to control it.

Little is known about the effects of Elytroderma disease on Jeffrey pines, but scientists have observed and studied the disease on ponderosa pine (*P. ponderosa* Dougl. ex Laws.) (Childs 1968; Childs and others 1971; Lightle 1954; Roth 1959; Waters 1957, 1962). Wier (1916) discovered this disease on ponderosa pine in the West, and since then several severe outbreaks have occurred and timber losses estimated to be enormous. Wagener and others (1949), and Childs (1968) reported that in Oregon and Washington, salvage cuttings of ponderosa pine were necessary to utilize millions of board feet of dead and dying timber. In addition, volume was lost in trees reduced in growth rate and in dead trees inaccessible to salvage.

Certain aspects of the biology and spread of the disease are known. Elytroderma disease is prevalent in the Western United States and Canada but only appears in epidemic outbreaks occasionally. After an outbreak, the disease persists in infected trees resulting in damage for many years. The disease often occurs repeatedly on the same sites. Ponderosa pine is the tree most commonly attacked, but Jeffrey pine and other pine species are also infected (Bynum and Miller 1964). Both growth loss and mortality result in infected trees. The extent to which Elytroderma disease spreads, builds up, and damages Jeffrey pines is not well understood.

Although widespread in California, Elytroderma disease is common on Jeffrey pines only in localized areas within the State. Lassen National Park and the Lake Tahoe Basin, for example, have a history of Elytroderma disease on Jeffrey pines (Lightle 1955, Wagener and others 1949). In the Lake Tahoe Basin, endemic levels of the disease have been observed for 40 years or more. Reports suggest that at least one outbreak took place in 1949-1950.[1] Since that time, no other outbreak of the disease has been reported in the Basin except for the one discussed in this paper.

The present outbreak of Elytroderma disease in the South Lake Tahoe area was first reported in 1969.[2] In the Eldorado National Forest, pines were heavily infected with Elytroderma disease from Tallac Village to Emerald Bay, north along the west shore of Lake Tahoe and in the vicinity of the present area of study. By June 1970, the disease was causing considerable discoloration of pines in many areas—particularly in the valley bottom but it was estimated that little, if any, tree mortality would occur unless heavy infection continued in succeeding years.[3]

By spring 1971, more and more Jeffrey pines on the Eldorado National Forest were dying, causing widespread concern among visitors to the Lake Tahoe Basin.[4] Consequently, the Pacific Southwest Forest and Range Experiment Station was asked by the California Region, Forest Service, U.S. Department of Agriculture, to investigate. The study was started in June, a progress report issued in November 1972,[5] and a further report issued in 1974.[6]

This paper reports on the incidence, rate of spread, and buildup of Elytroderma disease in Jeffrey pine stands after the epidemic outbreak of 1971, and discusses the effects of the disease on vigor, growth, and mortality of Jeffrey pines in the study area. The report illustrates how a single outbreak of Elytroderma disease can damage and kill Jeffrey pines over a period of years in certain conifer stands in the Lake Tahoe Basin, California.

METHODS

After completing a field survey with National Forest personnel, we selected plots on the basis of degree of infection, as judged by the severity of current foliage symptoms in the stand. Selection was not random, and was to include as much variation as possible among plots in levels of infection. Six plots containing about 100 Jeffrey pines each were chosen in areas judged light or heavy in disease intensity.

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[1]Personal communication from Willis W. Wagener, California Forest and Range Experiment Station, to J. M. Miller, Bureau of Entomology and Plant Quarantine, Berkeley, Calif., May 7, 1951.

[2]Personal communication from Douglas R. Miller, California Region, Forest Service, U.S. Department of Agriculture, to Forest Supervisor, Eldorado National Forest, August 4, 1969.

[3]Personal communication from Douglas R. Miller to Forest Supervisor, Eldorado National Forest, July 6, 1970.

[4]Personal communication from James M. Olson, District Ranger, Lake Valley Ranger District, to Forest Supervisor, Eldorado National Forest, May 20, 1971.

[5]Scharpf, R. F., and R. V. Bega. One-year progress report on the impact of Elytroderma disease on Jeffrey and ponderosa pines at South Lake Tahoe, November 13, 1972. (Unpublished report on file, Pacific Southwest Forest and Range Experiment Station, Berkeley, Calif.)

[6]Scharpf, R. F., and R. V. Bega. Third year progress report on the impact of Elytroderma disease on Jeffrey and ponderosa pines at Lake Tahoe, June 24, 1974. (Unpublished report on file at Pacific Southwest Forest and Range Experiment Station, Berkeley, Calif.)

Plots were irregular in shape and ranged in size from about 0.2 acre to slightly more than 1 acre. The plots consisted mostly of Jeffrey pines but a few trees of other species, such as white fir, *Abies concolor* (Gord & Glend. Lindl. ex Hildbr.) and Sierra lodgepole pine (*P. contorta* ssp. *murrayana* [Balf.] Critchfield) were also present. Per acre basal areas of both host and nonhost trees were measured on all plots. All plots were within a few hundred yards of one another so that differences in topography, stand composition, and microclimatic conditions were kept to a minimum.

Only Jeffrey pines 0.5 inch or larger at 4.5 feet above the ground (d.b.h.) were included in the study. Test trees were measured for: d.b.h. to the nearest 0.1 inch; tree height to the nearest foot; crown class: (1) dominant, (2) codominant-intermediate, (3) suppressed; tree vigor: (1) good—trees with full crowns and normal needle complement (live crown ratios of 70 percent or more; 3 or more years of needle retention), (2) average—trees with somewhat less than full crowns, or less than normal needle complement, or both, (3) poor—trees with poor live crown ratios, or with sparse foliage, or both.

In addition to tree measurements, data on distribution and intensity of Elytroderma disease were recorded for test trees. Disease distribution was assessed by visually dividing the living crown into four parts (lower, lower-mid, upper-mid, and upper crown) and determining presence or absence of the disease in each portion. Proportion of the crown infected was used as a measure of disease distribution within the tree. Disease intensity was estimated on the percentage of foliage showing disease symptoms: (0) no infection; (1) light—from 1 to 10 percent of the foliage showing symptoms; (2) moderate—from 11 to 50 percent of the foliage showing symptoms; and (3) heavy—51 percent or more of the foliage showing symptoms.

Permanent photo points were set up on several plots to record visual changes in tree condition, disease distribution, and disease intensity. Trees were photographed in color at least once, sometimes twice each year, from June 1971 to October 1977. Mortality usually was recorded twice a year, once in late spring and again in early fall from 1971 through summer 1978.

At 2- and 6-year intervals after the start of the study, tree vigor, disease distribution, and disease intensity were again recorded.

RESULTS

Host and Disease–1971

At the beginning of disease outbreak, d.b.h. of the test trees ranged from about 1 to 40 inches. Mean d.b.h. was 8.9 inches with a standard deviation of 4.9. Most trees were in the pole size class but some saplings and sawtimber sizes were present, also. Basal areas ranged from about 53 to 225 square feet per acre among the plots. More than 95 percent of the basal area on all plots was in Jeffrey pines.

The various crown and vigor classes were fairly well represented in the study:

Crown class			Vigor class		
Dominant	Codominant-intermediate	Suppressed	Good	Average	Poor
Percent			*Percent*		
43	37	20	29	40	31

Distribution and intensity of the disease, as recorded at the beginning of the study, were:

Disease distribution (proportion of crown affected)					Disease intensity			
None	¼	½	¾	All	None	Light	Moderate	Heavy
Percent					*Percent*			
0	1	1	22	76	0	28	25	47

None of the Jeffrey pines on any of the plots was free from Elytroderma infection. In addition, the disease most often occurred throughout the crown of the trees, even when the infection was rated light. This pattern strongly suggests a windborne mechanism of spore dispersal resulting in a more or less random pattern of infection in trees. Also, the apparent random distribution of infection in trees of all sizes studied indicated that specific microclimatic conditions or unusual weather in the area did not limit the disease to any portion of the live crown or to trees of any particular size class. More than 80 percent of all trees had some infection within 6 feet of their tops.

Disease intensity varied among test trees. About one-half (47 percent) were heavily infected, one-fourth (25 percent) moderately infected, and about one-fourth (28 percent) lightly infected. Tree size (d.b.h.) had no noticeable effect on disease intensity in that the percentage of the trees at each rated level of infection was about the same for trees of different size class (*table 1*). Similarly, disease intensity did not appear to be related to tree crown class. The proportions of trees at each rated level of infection was nearly the same for trees of different crown classes, but intensity of infection differed among trees of the three vigor classes (*table 1*).

In general, trees of good vigor were lightly to moderately infected, whereas trees of poor vigor were heavily infected. According to Lightle (1954), Elytroderma disease causes de-

Table 1—*Infection ratings of Jeffrey pines, by sizes, crown, and vigor class, Lake Tahoe Basin, California, 1971*

Class	Infection rating: Light	Moderate	Heavy
Size (d.b.h.) (inches)		*Percent*	
0.5 to 6.0	27	26	47
6.1 to 12.0	27	27	46
> 12.0	34	24	42
Crown			
Dominant	29	25	46
Codominant-intermediate	31	27	42
Suppressed	19	29	52
Vigor			
Good	61	28	11
Average	23	34	43
Poor	5	13	82

Table 2—*Disease intensity of Jeffrey pines, by plot and total basal area, Lake Tahoe Basin, California, 1971*

Plot	Total basal area[1]	Disease intensity		
		Light	Moderate	Heavy
	sq. ft.		*Percent*	
1	99	16	15	69
2	112	9	24	67
3	207	29	31	40
4	196	9	20	71
5	53	59	31	10
6	224	50	33	17

[1] A X^2 value of 2.50 and significance probability > 0.25 demonstrated independence of disease intensity and basal area where basal area was classified as either low or high.

foliation and a marked reduction in the length of infected needles of ponderosa pines. It is not surprising, therefore, that with the rating system we used for vigor in this study, Jeffrey pines with thin crowns and short needles were lower in vigor than trees with a normal foliage complement.

Taken on a plot basis, disease intensity varied considerably but was not related to stand basal area. Plots 1, 2, and 4 contained a higher proportion of trees in the heavily infected class than did plots 3, 5, and 6 (*table 2*).

In summary, infection was fairly well distributed throughout the crowns of test trees of all sizes and live crown ratio classes in 1971. Similarly, levels of infestation were fairly proportional among trees of different size classes and live crown ratio classes with about one-half of the trees showing heavy infection, one-fourth showing light infection, and one-fourth showing moderate infection. Heavily infected trees rated lower in vigor than did light and moderately infected trees.

Disease Changes–1971 to 1977

1973

Trees were examined in June 1973 to assess changes in disease intensity, disease distribution, and tree vigor for a 2-year period. Of the 553 living trees, 62 percent had the same intensity rating in 1973 as in 1971; 30 percent rated one class higher (light to moderate or moderate to heavy infection) and 8 percent rated one class lower (heavy to moderate or moderate to light). No trees rated more than one class higher or lower than they did in 1971. About two-thirds of the trees, therefore, did not change in infection rating during the 2-year period. Of the remaining one-third, most increased in infection rating and a few decreased.

Of the trees living in 1973, 56 percent were heavily infected, 25 percent moderately infected, and 19 percent lightly infected. The disease ratings for all trees surviving in 1973, therefore, were similar to those made at the beginning of the outbreak in 1971. What apparently occurred during the 2-year period was that some heavily infected trees died and the infection ratings in the remaining trees increased slightly to keep the proportion of trees in each rating class similar to that observed 2 years previously.

Analysis of disease distribution in the crowns of test trees suggested that the fungus was somewhat more widely distributed through the crowns in 1973 than in 1971. Of the 553 trees examined in 1973, 22 percent showed an increase in disease distribution, 2 percent showed a decrease, and 75 percent showed no change. The disease had increased in distribution but not necessarily in intensity in some trees for the 2-year period.

Tree vigor changed noticeably from 1971 to 1973. Forty percent of the trees decreased in vigor, and only 5 percent increased in vigor. Vigor remained unchanged in about one-half (55 percent) of the trees. Of the 553 trees living in 1973, 55 percent were of poor vigor, 26 percent of average vigor, and 19 percent of good vigor. The overall vigor of the plot trees, therefore, had diminished substantially in 2 years.

1977

Changes in disease intensity, disease distribution, tree vigor, and tree growth were investigated in test trees in 1977, 6 years after initiation of the study. Of the 440 trees that were alive in 1977, about one-half (55 percent) showed no change, 38 percent increased, and 7 percent decreased in disease intensity rating. These results are similar to those obtained in 1973 and indicate that no dramatic change in disease intensity occurred in the test trees since 1971. The proportion of trees with different ratings of infection intensity was nearly the same in 1977 as it was in 1971 and 1973. In 1977, 55 percent of the surviving trees were heavily infected, 25 percent moderately infected, and 20 percent lightly infected or free from disease. A few trees, free from disease in 1977, had been rated lightly infected in 1971 and in 1973.

From 1971 to 1977, the proportion of living trees with light, moderate, or heavy levels of infection had remained fairly constant. Some increase in disease intensity had occurred in test trees over time, however, because of the 167 trees that died during the 6 years, about 90 percent were heavily infected. For intensity ratings to have remained constant over time, the disease must have intensified in a substantial proportion of the remaining trees.

In tree crowns in 1977, the disease was less widely distributed than it was in 1971. Of the 440 surviving trees in 1977, 12 percent showed an increase in disease distribution, 24 percent showed a decrease, and 64 percent showed no change. What appears to have happened in some trees is that the new shoot and top growth that developed since 1971 was free of disease, thereby reducing the proportion of the live crown affected by Elytroderma.

Host vigor continued to decline among the test trees. Vigor in 57 percent of the remaining trees declined at least one rating since 1971, in 4 percent increased, and in 39 percent remained unchanged. When one also considers that about three-fourths of the trees that died after 6 years were of poor vigor, it is easy to see that, overall, stand vigor declined dramatically. Of the remaining live trees, 54 percent were rated poor vigor, 34 percent moderate, and only 12 percent good. For the stands as a whole, therefore, not only had the level of stocking dropped noticeably after 6 years as a result of the Elytroderma outbreak,

Table 3—*Mean annual radial growth of Jeffrey pines 5 years before and 6 years after the outbreak of Elytroderma disease in 1971*

Disease rating	Trees	Before 1971	After 1971
		mm	
Light	76	1.2	1.1
Moderate	100	1.1	0.9
Heavy	196	1.0	0.7

but the average vigor of the surviving trees had also dropped markedly.

Radial growth rate of living trees was measured from increment cores taken at d.b.h. 6 years after the disease outbreak. Growth was measured for the 5 years before the outbreak and for 6 years afterwards (*table 3*). A two-way analysis of variance with a split plot on time indicated no interaction among variables. On the average, trees grew less per year after the outbreak than before ($F = 4.04$; $\propto 1$), and mean growth after infection showed a downward trend with an increase in disease intensity rating ($F = 5.01$; $\propto 5$). In general, most plot trees were growing slowly even before the disease outbreak, as indicated by an average radial growth rate of only about 1 mm per year. Elytroderma disease, therefore, reduced radial growth rate in trees already growing slowly. Reduction of radial growth in trees that died during the study was not determined.

Tree Mortality–1971 to 1978

Tree mortality was heavy during the 7-year study (*table 4*). Of the original 609 test trees, 192 died. Mortality, however, was not evenly divided among the plots. Plots 1 and 2 accounted for 118 of the dead trees; therefore, out of an initial 203 trees on plots 1 and 2, 58 percent died during the study. Only about 5 percent of the trees died on plots 5 and 6, however, and for all plots combined, about 32 percent of the trees died.

Of the trees that died, 169 (88 percent) were heavily infected with Elytroderma disease at the start of the study. Plots with the greatest numbers of heavily infected trees had the greatest mortality. In addition, heaviest mortality (67 percent) occurred among trees of poor vigor, and 32 percent occurred among trees of moderate vigor. Only 1 percent mortality occurred among trees with good vigor at the beginning of the study.

Table 4—*Tree mortality at intervals after the outbreak of Elytroderma disease in 1971*

Month and year	Living trees	Dead trees	
June 1971	607	—	—
June 1972	581	26	[1]4.3
June 1973	553	28	4.8
June 1974	522	29	5.2
June 1976	447	[2]74	14.2
June 1977	439	10	2.2
June 1978	433	6	1.4
Sept. 1978	414	19	4.4
		Total	Pct.
		192	[3]31.6

[1]Percentage of living trees.
[2]Mortality during a 2-year period. No data recorded in 1975.
[3]On the basis of 607 trees at the beginning of study.

In general, mortality was fairly evenly divided among trees of different crown class. Of the dead trees recorded, 36 percent were dominant, 36 percent codominant or intermediate, and 28 percent suppressed. Mortality was not limited to any d.b.h. class, but was well distributed among trees of all diameters. Mean d.b.h. of the dead trees was 8.9 inches, exactly the same as the mean d.b.h. of all test trees.

Mortality of Jeffrey pines in the study area was not caused entirely by Elytroderma disease. In a biological evaluation in 1973 of about 800 acres in the general area of disease infestation, pest control specialists of the Forest Service, U.S. Department of Agriculture, reported several variables involved in tree mortality.[7] Probable cause of death of trees surveyed and sampled at random in the evaluation was recorded as: Elytroderma disease alone—11 percent; Elytroderma disease and Jeffrey pine beetle—44 percent; Jeffrey pine beetle alone—11 percent; other pests and unknown—34 percent.

In our study, we attributed nearly all of the mortality in Jeffrey pine to Elytroderma disease or to Elytroderma disease and Jeffrey pine beetle. As our data show, a large proportion of the trees that died were heavily infected with Elytroderma and were of poor vigor. Many of these trees were also invaded by Jeffrey pine beetle. Although a random sample was not taken on all plots, the 8 trees that died on plots 1 and 2 between 1972 and 1978 that were not heavily infected by the disease and were of good to average vigor were also heavily attacked by Jeffrey pine beetle. We concluded that these less severely diseased trees in the outbreak area were probably killed by high populations of bark beetles that were attracted to or had built up in the severely diseased stands. We saw little evidence that other diseases or insects were responsible for the tree mortality we recorded, although *Fomes annosus* was observed in the general area of Elytroderma outbreak.[2] Our observations as well as observations by local forest managers indicated that, outside the general area of disease outbreak, Jeffrey pines in the Tahoe Basin were not experiencing above-normal levels of bark beetle attack and mortality during the study.

We had expected that the severe drought of 1976-1978 would further weaken the trees and result in increased levels of mortality, particularly among the remaining trees of poor vigor and heavy infection. Results of the study showed this not to be true. The heaviest mortality recorded occurred between June 1974 and June 1976. We believe that this mortality was caused by continued stress from Elytroderma disease and associated bark beetle activity. Mortality during the prolonged drought in California, summer 1976 to summer 1978, was actually less than that recorded during the pre-drought years.

Although 7 years have passed since the outbreak of the Elytroderma disease at Lake Tahoe Basin, a substantial portion of the remaining trees continue to die on some of the study plots.

[7]Pierce, John R., and Michael D. Srago. Biological evaluation—pest conditions in the Taylor Creek drainage between Fallen Leaf Lake and Lake Tahoe. March 5, 1974. (Unpublished report on file, Pacific Southwest Region, Forest Service, U.S. Department of Agriculture, San Francisco, Calif.)

Table 5—*Plot size, live trees, and change in per acre basal area for Jeffrey pines on the study plots in 1971 and 1978*

Plot	Plot size	1971 Live trees	1971 Per acre basal area	1978 Live trees	1978 Per acre basal area	Change in basal area, 1971-1978
	acre		*sq. ft.*		*sq. ft.*	*sq. ft.*
1	0.87	101	96	52	70	−26
2	.60	102	112	35	34	−78
3	.22	98	201	77	174	−27
4	.26	107	194	62	126	−68
5	1.07	99	53	95	52	− 1
6	.21	100	218	94	222	+ 4

This cumulative mortality has resulted in heavy reduction in levels of stocking and basal area on some plots (*table 5, cover*).

No relationship was found between the original level of stocking or basal area of Jeffrey pines and the severity of mortality on the plots during the study. Tree mortality resulted in an appreciable reduction of basal area on plot 2 and substantial reductions on plots 1, 3, and 4. On these plots, increased basal area from 7 years of growth in residual pines did not compensate for the high loss in basal area from tree mortality. On plot 5, basal area lost from mortality was almost equal to basal area gained through growth in plot trees. Only plot 6 showed an increase in basal area. On this plot, the increase in basal area from growth was just slightly greater than that lost through mortality after 7 years. On both plots 5 and 6, mortality occurred entirely among trees less than 6 inches d.b.h. As was reported earlier, growth rates of almost all plot trees were poor.

DISCUSSION

Mortality and growth reduction of Jeffrey pine continued during a 6-year period after an epidemic outbreak of Elytroderma disease in 1971 at South Lake Tahoe, California. Results of the study indicate that no additional outbreaks of the disease occurred from 1971 through 1977. Why, then, did the effects of an epidemic outbreak that occurred in only 1 year, persist for several years?

It is known from past research that *Elytroderma deformans* initially infects needles of pines, but over time, the fungus grows into twigs and branches. Once the fungus reaches the branch tissues, it grows systemically within the phloem region of the branch and persists for years, often invading the growing tip or buds of the branches (Childs 1968). Each succeeding year, therefore, newly produced needles are infected by the fungus living within the growing tips of the infected branches.

The proportion of the needle infections that become systemic branch infections is not well known. Our observation and photographs of some trees for several years suggest that many of the initial infections on needles result in systemic infections. Systemic infection also explains the fairly constant levels of the disease in stands for several years after an epidemic outbreak, even though climate and other variables do not favor new needle infection. Childs (1968) pointed out for ponderosa pine, and we found it true for Jeffrey pines, that epidemic outbreaks need not occur frequently for tree damage and mortality to result.

Some variables involved in mortality in this study were similar to those found by Childs (1968) in the Pacific Northwest:

- Mortality occurred among trees of all size classes.
- Mortality was highest in trees of poor vigor and with heaviest infection.
- Mortality was the result of not only Elytroderma disease but was associated with bark beetle attack.

Childs (1968) considered certain root disease fungi to be the agents which, when associated with Elytroderma, resulted in tree death. Childs and others (1971) reported that severely infected ponderosa pines are seldom attacked by bark beetles. Instead, they are more likely to die as a direct result of Elytroderma disease. Pines killed by bark beetles, according to them, are more likely to be the ones with about one-fourth to three-fourths of the needles on twigs infected. We observed, and Pierce and Srago[7] recorded, Jeffrey pine beetle as a principal agent in association with Elytroderma disease and tree death.

We noticed, as did Childs and others (1971) and Hunt and Childs (1957)[8] that local outbreaks of Elytroderma disease are generally restricted to certain areas. These areas are often sheltered situations such as bottoms of draws, meadows, forests along lake shores, and forested slopes subject to heavy dew or condensation from persistent fogs. Undoubtedly, epidemic outbreaks of the disease require certain weather conditions and, likely, specific climatic variables for heavy infection to occur. Unfortunately, we do not yet know precisely what variable(s) are involved in these infrequent but periodic outbreaks. Further study is needed to better understand the climate or other variables involved in the epidemiology of Elytroderma disease.

Knowledge about the occurrence of past outbreaks and surveys of present outbreaks allow the forest manager to better delineate sites on which outbreaks are apt to occur again. In the management of these high-risk sites it is necessary for the manager to realize that susceptible species will periodically suffer heavy mortality and growth loss from Elytroderma disease. In such areas, management may call for retaining or regenerating tree species not susceptible to the disease.

One observation we made during the study was that Sierra lodgepole pine throughout the area was almost uninfected by Elytroderma disease, even though the Rocky Mountain-intermountain race, *P. contorta* ssp. *latifolia* (Engelm.) Critchfield is reported to be a principal host of the disease in British Columbia, Canada. In a strip survey through a stand of mixed lodgepole and Jeffrey pines, we found only 5 lightly infected lodgepole pines out of 71 trees examined even though

[8]Hunt, John H., and T. W. Childs. 1957. Ponderosa pine needle blight in eastern Oregon during 1955 and 1956. (Unpublished report on file at Pacific Northwest Forest and Range Experiment Station, Corvallis, Oreg.)

40 Jeffrey pines examined in the survey were moderately to heavily infected. For reasons not determined in the study, lodgepole pines intermixed with Jeffrey pines in severely infected stands were nearly free from disease. It appears from our general observations throughout the area and from this strip survey that lodgepole pine, along with other resistant trees, can be considered as an alternative species for planting, management, or both, on high-risk sites.

Air pollution (smog) has been reported to be causing damage to trees in the Lake Tahoe Basin.[4] We saw no evidence of this on trees in the Elytroderma disease areas we examined. In fact, from 1971 to 1977, both concentrations and duration of ozone levels were considered below those required to cause visual symptoms of smog injury to pines in the Lake Tahoe Basin.[9]

The findings of our study that we consider most valuable for aiding forest managers in reducing Elytroderma disease are:

- Elytroderma disease occurs in epidemic proportions infrequently but periodically in certain stands of Jeffrey pine in the Lake Tahoe Basin. Mortality and growth losses during and for years after epidemics can be severe, particularly among heavily infected trees.
- High-risk areas should be defined by surveys shortly after disease outbreaks. Managers should biologically evaluate the possibility that heavy mortality and growth losses will disrupt current management plans or prevent reaching management goals.
- Level of stocking seems to have no influence on the incidence and severity of disease, but overstocked stands often contain many trees of poor vigor. Thinning or other means to increase stand vigor before an outbreak will probably not prevent other outbreaks from occurring but may help to reduce tree mortality resulting from the disease. Trees of all size and age classes appear equally susceptible to infection and damage from the disease; therefore, planting or favoring existing nonsusceptible tree species is the best way to control the disease in high-risk areas.
- Weakening of trees by the disease also predisposes the stand to attack by bark beetles, which further increases mortality and damage.

[9]Personal communication from Paul R. Miller, Pacific Southwest Forest and Range Experiment Station, Riverside, Calif., 1978.

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A disease of Jeffrey pines (*Pinus jeffreyi* Grev. and Balf.) at Lake Tahoe Basin, California, caused by Elytroderma disease (*Elytroderma deformans*) was studied for 7 years after a severe outbreak of the fungus in 1971. Among 607 Jeffrey pines on six plots, about one-half were heavily infected and about one-half were moderately or lightly infected in 1971. No uninfected trees were observed. During the 7-year study, about one-half of the trees remained unchanged in vigor, disease intensity, or both, and about one-half decreased in vigor, became more heavily infected, or both. Of the original 607 trees studied, nearly one-third died before 1978. Average radial growth of surviving trees was less per year after the outbreak than before, and heavily infected trees were growing more slowly than lightly infected trees. Intensity of the disease, however, was not related to stand basal area.

Retrieval Terms: Pinus jeffreyi, Pinus ponderosa, Pinus contorta, parasitic fungi, *Elytroderma deformans*, Lake Tahoe Basin, forest protection, mortality rate.