Growth Retardation of Plantation-Grown Sugar Pine by *Lophodermella arcuata*

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ABSTRACT

Burleigh, J. R., Wood, W., and Sebesta, P. 1982. Growth retardation of plantation-grown sugar pine by *Lophodermella arcuata*. Plant Disease 66:680-682.

Lophodermella arcuata defoliated 20-yr-old saplings of sugar pine (*Pinus lambertiana*) in a 16-ha plantation in California. Only the current season's needles remained on severely infected trees by October of each year. In 1972, when the infestation was first observed, only two adjacent trees were infected; but by 1974 and 1975, the pathogen had infected most trees over areas of 4.2 ha and 12.6 ha, respectively. Disease severity was estimated each year (1974–1977) as the number of fascicles per tree with at least one infected needle (needles brown, hysterothecia visible). There were significant interactions between terminal growth and disease class and between radial growth and disease class. That is, the magnitude of growth was not independent of disease class. Pertinent orthogonal comparisons revealed that radial and terminal growth of diseased trees was significantly less than growth of healthy trees. During years of rapid disease spread (1974–1977), radial and terminal growth of diseased trees decreased annually, while there were no significant between-year differences in annual growth of healthy trees. There appeared to be no terminal growth reduction attributable to drought effects, but average radial growth of healthy trees for 6 yr before the drought was significantly greater than average growth during the 3 yr of the drought.

In 1972, two sugar pine (*Pinus lambertiana*) saplings infected by *Lophodermella arcuata* (Darker) Darker were observed in the Mi-wok district of the Stanislaus National Forest in California. The trees were in a 16-ha plantation planted in 1952 after a major forest fire. Although the disease appeared to be merely a curiosity in 1972, it progressed rapidly and by 1975, the pathogen had infected and defoliated most trees in the plantation.

Although *L. arcuata* was observed in California as early as 1932 (1), it was not viewed as a threat to established stands or seedlings. In 1960, Shaw and Leaphart (4) first reported the capability of *L. arcuata* to severely defoliate white pine, but they gave no evidence of its explosive nature or of its effect on growth. More recent reports, however, have linked *L. arcuata* (5) and other species of *Lophodermella* (3,6) to reduced tree growth and occasional mortality. This paper documents the growth retardation caused by a relatively innocuous needle cast fungus in California that became epidemic when introduced into a plantation of sugar pine of uniform age and relatively uniform susceptibility.

MATERIALS AND METHODS

In November 1976, 10 healthy trees were selected randomly from within a 0.5-ha zone in the plantation previously noted to contain trees infected each year since 1974. In the same zone, 10 diseased trees were selected randomly from among those infected for at least 3 yr (1974–1976). No systematic observations were made in 1973, so some of the trees selected may have been infected in 1973 as well. Trees classed as diseased retained only the current season's needles (needles that emerged the same year observations were made) by October—all needles that emerged the previous year had been infected and cast. Trees classed as healthy were free from disease.

Terminal growth of selected trees was measured between annual whorls with a telescoping rod for the years 1969–1976. All trees selected were the same age, had about the same diameter at breast height (dbh) (17.3 cm healthy and 17.0 cm diseased), and were situated on a south-facing slope. In October 1977, 10 healthy (dbh 21 cm) and 10 diseased trees (dbh 18.9 cm) again were selected randomly from the same zone as in 1976. This second selection was necessary because some diseased trees measured in 1976 died. A core was taken from each tree, and radial growth was measured with a ruler for 1969–1977. Each year from 1974 to 1977, disease severity was estimated on six lateral branches of each tree as the number of fascicles with at least one infected needle. In 1974 and 1975, the boundary of the area enclosing infected trees was surveyed to estimate rate of disease spread.

Disease effects on terminal and radial growth were evaluated by analysis of variance (ANOVA) in 2×8 and 2×9 mixed-model factorial experiments, respectively. Both terminal and radial growth were considered as fixed effects, but disease class was considered as a random effect because it was not determined that all trees were infected the same year. The mixed model is required because the observed differences in growth were affected by random levels of disease class. That is, any test of significance applied to growth effects must consider the growth-disease class interaction. The design was completely random.

Orthogonal coefficients (7) were used to make comparisons among groups of treatments. That is, treatment sums of squares associated with ANOVA of terminal growth and with ANOVA of radial growth were partitioned to give six and eight independent comparisons, respectively, each based on one degree of freedom. Although treatment −1 comparisons were made (15 for terminal growth, 17 for radial growth), only those viewed as pertinent to disease effects are described (Table 1). A major drought in California during 1975–1977 coincided with years of rapid disease spread and therefore prompted comparisons C_3, C_6, C_7, and C_8. Comparisons are independent (orthogonal), because the sum of the

Table 1. Orthogonal comparisons for disease effects on terminal and radial growth of sugar pine

Comparison no.		
Terminal	Radial	Description
C_1	C_1	Was there a response to disease?
C_2	C_2	Was the average growth of healthy trees in group 1 greater than the average growth of healthy trees in group 2 before disease?
C_3	C_3	Was the average growth of trees in group 1 before the drought greater than average growth during the drought?
C_4,C_5	C_4,C_5,C_6	Did disease effects become progressively more severe during years of rapid disease spread?
C_6	C_7,C_8	Was there a difference in growth of healthy trees during drought years?

Accepted for publication 7 October 1981.

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0191-2917/82/08068003/$03.00/0

coefficients in any one line is zero and the sum of cross products between any two lines is zero (Tables 2 and 3). F tests were conducted according to conventional methods.

RESULTS

When we first observed *L. arcuata* in June 1972, it was present on only two trees in a 16-ha plantation. The two trees were severely infected but not defoliated. By 1974, the pathogen had infected and partially defoliated most trees over an area of 4.2 ha (26% of the plantation) and by 1975, it had infected and defoliated most trees over an area of 12.6 ha (75% of the plantation). By 1976, the pathogen had infected native sugar pine saplings at the edge of the plantation.

Average disease severity on diseased trees in the experimental zone rose from 49% (49% of fascicles per tree with at least one infected needle) in 1974 to 100% in 1975. In 1974, only needles formed in 1973 showed symptoms; however, in June 1975, all needles showed symptoms except those formed in 1975. By October 1975, infected needles had been cast, leaving only those formed that year. Severity estimates made in June 1976 and

Table 2. Six orthogonal comparisons of disease effects on terminal growth of sugar pine

Group[a] Treatment	Treatment totals (T_i)	Treatment average	Comparison[b] C_1	C_2	C_3	C_4	C_5	C_6
Group 1								
a_1b_1	454	45.4	+3	+5	+1	0	0	0
a_1b_2	555	55.5	+3	+5	+1	0	0	0
a_1b_3	527	52.7	+3	+5	+1	0	0	0
a_1b_4	500	50.0	+3	+5	+1	0	0	0
a_1b_5	485	48.5	+3	+5	+1	0	0	0
a_1b_6	533	53.3	+3	+5	+1	0	0	0
a_1b_7	521	52.1	+3	+5	−3	0	0	+1
a_1b_8	527	52.7	+3	+5	−3	0	0	−1
Group 2								
a_2b_1	561	56.1	+3	−8	0	0	0	0
a_2b_2	628	62.8	+3	−8	0	0	0	0
a_2b_3	433	43.3	+3	−8	0	0	0	0
a_2b_4	390	39.0	+3	−8	0	0	0	0
a_2b_5	360	36.0	+3	−8	0	0	0	0
a_2b_6	549	54.9	−13	0	0	−2	0	0
a_2b_7	338	33.8	−13	0	0	+1	+1	0
a_2b_8	229	22.9	−13	0	0	+1	+1	0
Q[c]			+4,914	+1,534	−90	−531	+109	−6
Kr[d]			624 (10)	520 (10)	24 (10)	6 (10)	2 (10)	2 (10)
SS[e]			3,870*	453	34	4,699*	594*	2

[a] a_1 = Trees healthy; b_1–b_8 = 1969–1976; a_2 = trees healthy until infected in 1973–1974 (b_6).
[b] See Table 1.
[c] $Q_i = \Sigma C_{1i}T_i$, where C_{1i} = orthogonal coefficients and T_i = treatment totals.
[d] $Kr = \Sigma C_i^2$ (10), where C_i = orthogonal coefficient i, i = 1–16.
[e] $SS = Q^2/Kr$. An asterisk indicates significance at $P = 0.05$.

Table 3. Eight orthogonal comparisons of disease effects on radial growth of sugar pine

Group[a] Treatment	Treatment totals (T_i)	Treatment average	Comparison[b] C_1	C_2	C_3	C_4	C_5	C_6	C_7	C_8
Group 1										
a_1b_1	65.5	6.6	+2	+5	+1	0	0	0	0	0
a_1b_2	67.0	6.7	+2	+5	+1	0	0	0	0	0
a_1b_3	59.0	5.9	+2	+5	+1	0	0	0	0	0
a_1b_4	54.0	5.4	+2	+5	+1	0	0	0	0	0
a_1b_5	52.5	5.3	+2	+5	+1	0	0	0	0	0
a_1b_6	56.0	5.6	+2	+5	+1	0	0	0	0	0
a_1b_7	46.0	4.6	+2	+5	−2	0	0	0	−2	0
a_1b_8	44.5	4.5	+2	+5	−2	0	0	0	+1	−1
a_1b_9	37.0	3.7	+2	+5	−2	0	0	0	+1	+1
Group 2										
a_2b_1	68.5	6.9	+2	−9	0	0	0	0	0	0
a_2b_2	71.5	7.2	+2	−9	0	0	0	0	0	0
a_2b_3	69.5	7.0	+2	−9	0	0	0	0	0	0
a_2b_4	59.5	6.0	+2	−9	0	0	0	0	0	0
a_2b_5	54.0	5.4	+2	−9	0	0	0	0	0	0
a_2b_6	51.5	5.2	−7	0	0	−3	0	0	0	0
a_2b_7	40.0	4.0	−7	0	0	+1	+2	0	0	0
a_2b_8	31.5	3.2	−7	0	0	+1	−1	+1	0	0
a_2b_9	16.0	1.6	−7	0	0	+1	−1	−1	0	0
Q[c]			+636	−500	+99	−67	+33	+16	−11	−8
Kr[d]			252 (10)	630 (10)	18 (10)	12 (10)	6 (10)	2 (10)	6 (10)	2 (10)
SS[e]			161*	40*	54*	37*	18*	12*	2	3

[a] a_1 = Trees healthy; b_1–b_9 = 1969–1977; a_2 = trees healthy until infected in b_6 (1973–1974).
[b] See Table 1.
[c] $Q_i = \Sigma C_{1i}T_i$, where C_{1i} = orthogonal coefficients and T_i = treatment totals.
[d] $Kr = \Sigma C_i^2$ (10), where C_i = orthogonal coefficient i, i = 1–16.
[e] $SS = Q^2/Kr$. An asterisk indicates significance at $P = 0.05$.

Table 4. Analysis of variance (ANOVA) of the effects of infection by *Lophodermella arcuata* on terminal growth of sugar pine

Source of variation	Degrees of freedom	Sums of squares	Mean squares	Observed F	Required F 5%	Required F 1%
Treatments	15	15,635	...	...	...	...
Disease class (A)	1	2,369	2,369	19.4	3.90	6.81
Terminal growth (B)	7	6,824	975	1.06	3.79	6.99
A × B	7	6,442	920	7.5	2.07	2.76
Error	144	17,506	122	...	...	...
Total	159	33,141	...	...	...	...

Table 5. Analysis of variance (ANOVA) of the effects of infection by *Lophodermella arcuata* on radial growth of sugar pine

Source of variation	Degrees of freedom	Sums of squares	Mean squares	Observed F	Required F 5%	Required F 1%
Treatments	17	367	...	...	...	...
Disease class (A)	1	1.6	1.6	0.84	3.90	6.81
Radial growth (B)	8	325	40.6	8.12	3.44	6.03
A × B	8	40.4	5.0	2.63	2.00	2.63
Error	162	305	1.9	...	...	...
Total	179	672	...	...	...	...

in June 1977 again showed that all needles formed during 1975 and 1976, respectively, showed symptoms; by October of each year, all needles that showed symptoms had been cast.

Factorial analysis showed a significant interaction between terminal growth and disease class (Table 4). That is, the growth response to disease class varied among years. Planned F tests showed a significant effect of disease on terminal growth (Table 2, C_1) and a significant difference between growth of diseased trees in 1974, when disease severity was 49%, and growth in 1975 and 1976, when disease severity was 100% (C_4). Growth in 1976 also was significantly less than growth in 1975 (C_5). Growth of trees in group 2 (trees healthy until infected in 1973–1974) before infection (1969–1973) did not differ significantly from growth of trees in group 1 (healthy) (C_1). F tests made to evaluate possible drought effects (C_3, C_6) were not significant.

There also was a significant interaction between radial growth and disease class (Table 5). Significant differences occurred between growth in 1974 and growth in 1975–1977 (Table 3, C_4); between growth in 1975 and growth in 1976–1977 (C_5), and between growth in 1976 and growth in 1977 (C_6). Comparison C_2 showed a significant difference between growth of trees in group 1 and growth of healthy trees in group 2, while C_3 showed that radial growth of healthy trees during the drought was significantly different from growth during the six preceding years. Growth of healthy trees during the drought, however (C_7, C_8), showed no significant between-year differences.

DISCUSSION

L. arcuata has been implicated in reduced tree vigor and growth as a result of repeated consecutive infections (5). Similar events were associated with the growth retardation we observed during 1975–1977. Disease incidence and severity increased each year during our study (1972–1977). Maximum incidence and severity within the experimental zone occurred in 1974 and 1975, respectively; no additional trees were infected after 1974, and disease severity reached 100% infection on year-old needles in 1975, 1976, and 1977. Disease incidence and severity continued to increase on trees outside the zone, however.

L. arcuata exhibited a rapid but not unprecedented rate of disease development for a forest pathogen. An exhaustive study of the epidemiology of *Cronartium ribicola* in California (2) revealed that the most distant infections occurred 85 m from a source but that the limit of significant infection was from 53 to 60 m. By comparison, if we assume that the size of the area infected by *L. arcuata* in 1972 was about 100 m^2, then by 1974 and 1975, the pathogen had infected trees 100 m and 195 m, respectively, from the edge of the source. That rate of spread was about 55 m a year during 1972–1974 and 85 m during 1974–1975. The similarity in effective limit of infection of two pathogens with diverse habits and disease cycles appears unique and suggests that topography and vegetation may exercise major roles in defining the limits of infection by forest pathogens.

Orthogonal comparisons of disease effects on terminal and radial growth showed that diseased trees grew significantly less than healthy trees and that disease effects became progressively more severe each year during the epidemic. Terminal and radial growth in 1974 were significantly different from growth during 1975–1977. Those differences might be linked to the dramatic increase in disease severity between 1974 and 1975. Disease severity reached 100% and remained at that level from 1975 to 1977, yet growth declined significantly each year, which perhaps reflects the effect of consecutive infections.

The severe drought during 1975–1977, when precipitation was 108.0, 60.4, and 55.4 cm per year, respectively, compared with an average of 128.5 cm for 1969–1973, may have caused some of the growth differences between trees classed as healthy and diseased. However, terminal growth of healthy trees (Table 2, C_3) before the drought did not differ significantly from terminal growth during the drought, which indicated that there was no drought effect. On the other hand, radial growth of healthy trees before the drought was significantly greater than radial growth during the drought (Table 3, C_3). Continuation of the drought apparently did not reduce further radial growth, as there were no significant differences between growth in 1975 and growth in 1976–1977 (C_7) or between growth in 1976 and growth in 1977 (C_8).

The evidence, therefore, suggests that *L. arcuata* is capable of severely reducing terminal growth of plantation-grown sugar pine, but the evidence for reduction of radial growth is confounded by the possible effect of low precipitation.

This is not the first report to link infection by *L. arcuata* to severe defoliation and reduced growth (4,5), but it appears to be the first to quantify disease effects on both terminal and radial growth. If our interpretation is correct, the implications are obvious: forest management strategies to enhance productivity cannot ignore the inherent risks associated with cultural and genetic homogeneity. The epidemic potential of relatively benign resident pathogens might be greatly underestimated.

LITERATURE CITED

1. Darker, G. C. 1932. The Hypodermataceae of Conifers. Contrib. Arnold Arbor. I. 131 pp.
2. Kimmey, J. W., and Wagener, W. W. 1961. Spread of white pine blister rust from *ribes* to sugar pine in California. USDA For. Serv. Tech. Bull. 1251. 71 pp.
3. Miller, C. S. 1970. Role of *Lophodermella* species in premature death of pine needles in Scotland. Great Br. For. Comm. Rep. For. Res. 1970:176-178.
4. Shaw, C. G., and Leaphart, C. D. 1960. Two serious foliage diseases of western white pine in the Inland Empire. Plant Dis. Rep. 44:655-659.
5. Smith, R. S., Jr. 1978. Needle diseases. Page 59 in: Diseases of Pacific Coast Conifers. USDA For. Serv. Handb. 521. 206 pp.
6. Staley, J. M., and Bynum, H. H. 1972. A new *Lophodermella* on *Pinus ponderosa* and *P. attenuata*. Mycologia 64:722-726.
7. Steel, R. G. D., and Torrie, J. H. 1960. Principles and Procedures of Statistics. McGraw-Hill, New York. 481 pp.