Effects of Oxidant Air Pollution on Susceptibility of Pine Roots to *Fomes annosus*

R. L. James, F. W. Cobb, Jr., P. R. Miller, and J. R. Parmeter, Jr.

First, second, and fourth authors: former research assistant, associate professor, and professor, respectively, Department of Plant Pathology, University of California, Berkeley 94720. Third author: Plant Pathologist, Pacific Southwest Forest and Range Experiment Station, Forest Service, U.S. Department of Agriculture, stationed at Riverside, CA. Present address of senior author: Forest Insect and Disease Management, State and Private Forestry, U.S. Forest Service, 11177 W. 8th Ave., Lakewood, CO 80225.

The authors acknowledge J. Bruhn, N. Breisch, G. N. McKibbin, D. Rowney (Plant Pathology Department, University of California, Berkeley), Henry P. Milligan (deceased) (Pacific Southwest Forest and Range Experiment Station, Forest Service, U.S. Department of Agriculture) for assistance in this study, and the helpful leadership of O. C. Taylor, Associate Director, Statewide Air Pollution Research Center, University of California, Riverside.

This project was funded in part by the Environmental Protection Agency (EPA) under Contract 68-03-0273. The content of this article is not to be construed as representing views or policies of the EPA, nor as a concurrence of the Agency with the results presented in this article, and does not represent EPA policy, position, or findings. Mention of trade names or commercial products in this publication does not constitute either endorsement or recommendation for their use.

Accepted for publication 6 December 1979.

ABSTRACT

JAMES, R. L., F. W. COBB, Jr., P. R. MILLER, and J. R. PARMETER, Jr. 1980. Effects of oxidant air pollution on susceptibility of pine roots to *Fomes annosus*. Phytopathology 70:560-563.

Oxidant air pollution injury to foliage of ponderosa and Jeffrey pines increased the susceptibility of roots to infection and colonization by *Fomes annosus* under field and fumigation chamber environments. Roots of ponderosa pine trees severely injured by oxidant air pollution became infected proximal to the inoculation point significantly more often than did roots of healthy trees. Proximal colonization rate of *F. annosus* in inoculated ponderosa pine also was much greater in trees severely affected by air pollution. Exposure of container-grown ponderosa and Jeffrey pine seedlings to ozone in fumigation chambers increased infection and colonization by the pathogen. More seedlings (78% of both species) fumigated with ozone were infected than were nonfumigated seedlings (62 and 53% for ponderosa and Jeffrey pine, respectively). Colonization of host tissue of both species by *F. annosus* was directly related to ozone dose and seedling injury. These results indicate a very substantial, probable effect of oxidant air pollution on the rate of increase of *F. annosus* in coniferous forest stands subjected to chronic injury.

Ozone, a major component of photochemical air pollution, causes chlorotic needle mottle and eventual mortality of ponderosa (*Pinus ponderosa* Laws.) and Jeffrey (*P. jeffreyi* Grev. and Balf.) pines in the San Bernardino Mountains of California (2,15). Chronic ozone injury also appears to predispose pine trees to infestations by bark beetles (3), mainly by affecting natural resistance mechanisms. If resistance of trees to pests is altered by prolonged exposure to air pollution, it is important to elucidate these pollutant-pest interactions.

Fomes annosus (Fr.) Cke. is an important root pathogen in California, including the San Bernardino Mountains in southern California (1). It is often of greatest importance in stands subjected to periodic cutting (6). Such is the case in the San Bernardino Mountains where sanitation-salvage logging is conducted regularly to remove ozone-injured trees.

F. annosus spreads into living pine trees via root contacts and grafts with roots of infected stumps or trees (6,17,19). The mycelium grows directly from the bark of infected roots into that of living roots (16) and penetrates through phloem into the xylem. Thus, host resistance to *F. annosus* is manifested at the point of contact with the infected roots. Roots of suppressed or low vigor trees are more susceptible to infection and colonization by *F. annosus* than those from healthier trees (8,14,16). Since oxidant air pollution can cause major reductions in the health of ponderosa pine (3), root susceptibility to *F. annosus* may be influenced by chronic exposure, thus altering disease development and buildup in affected stands.

To determine effects of oxidant injury on root susceptibility to *F. annosus*, studies were made both in the field with larger trees and under controlled conditions with seedlings.

0031-949X/80/06056004/$03.00/0

MATERIALS AND METHODS

Field inoculation. Ponderosa and Jeffrey pine trees, each species on two sites, were chosen for root inoculations. Trees were dominant or codominant and exhibited a range of oxidant injury. Trees were scored for oxidant injury by using an index (12) based upon needle retention, condition and length in the upper and lower crown, and extent of branch mortality, the score was inversely related to pollution injury. Numbers of trees inoculated by site, species and injury rating are given in Table 1. No Jeffrey pines exhibiting severe air pollution injury were included in these inoculations because of restrictions imposed in selecting study sites. In addition, foliage at the Snow Valley site was severely injured by needle miners. Hence, Jeffrey pine subsequently was eliminated from the field data analysis.

Inoculation procedures were similar to those described by Wallis (21), with slight modification. Inoculum was prepared from Monterey pine (*P. radiata* D. Don) stem sections 8 cm long. They were autoclaved in jars (60 min @ 121 C), inoculated with *F. annosus* conidial suspensions and incubated in the dark at about 24 C for at least 12 wk. The *F. annosus* isolate (SV) used for inoculations at Snow Valley, Holcomb Valley, and Breezy Point was obtained from a colonized Jeffrey pine stump at Snow Valley. Three isolates were used at Camp Paivika: SV, JL (isolated from an infected Jeffrey pine stump near Jenks Lake), and CA (isolated from an infected white fir stump near Camp Angeles). Two roots from each tree were excavated and inoculated at about 50 cm from the root collar. Care was taken to avoid wounding roots during excavation. Ordinarily, one large (10–15 cm in diameter) and one small (2–5 cm in diameter) root were inoculated per tree. Large roots were cut half-way through the root in two places 8 cm apart, the "chip" between cuts was removed, and the inoculum block was inserted. Smaller roots were completely severed in two places 8 cm apart for inoculum block insertion. Inoculation points were washed with distilled water, wrapped in clear plastic, and secured

with masking tape. After inoculation, soil was carefully replaced around inoculation sites.

Small and large roots were analyzed for *F. annosus* infection and colonization 6 and 12 mo after inoculation, respectively. Roots were excavated to the root collar and for at least 1 meter distal to the inoculation point. They were cut from the tree with a hand- or chain saw with the blade swabbed with 95% ethanol before each cut. Roots were taken to the laboratory, split longitudinally with a sterile knife or hatchet blade, wrapped in moistened newspaper, and incubated for 7 days at about 24 C. They were then examined under the dissecting microscope (20–80 ×) for presence and extent of *F. annosus* colonization. In addition to the wrapping technique, wood chips from some roots were plated on a selective medium (9). Isolations onto the medium did not improve the accuracy of the colonization analysis. Because of the colonization pattern, better estimates were obtained with the wrapping technique. Reactions to infections, which were detected by resin accumulations proximal to the points of inoculation, were noted during root analysis.

The field inoculation data were analyzed initially with a "full screen" regression procedure (N. Norick, *personal communication*). This procedure compares a dependent variable to all possible linear combinations of independent variables; thus, the best set of significant independent variables can be chosen for further analysis. In this case, the dependent variables were proximal infection (+ or −) and, for "+" cases only, proximal colonization (millimeters per month). Proximal, rather than distal, colonization was used for assessing tree root susceptibility because the pathogen had to invade unsevered root tissue when moving toward the tree. Distal colonization often involved movement through dead root tissues, especially in roots that were completely severed. The independent variables were inoculum type or site, diameter at breast height (DBH), height, crown class, and 12 components of the tree oxidant injury score.

The dependent variables found to be significant for proximal infection were further analyzed with the chi-square test. The variables for proximal colonization rate were analyzed by analysis of variance tests.

Exposure chamber study. Six-year-old ponderosa and Jeffrey pine seedlings grown from seed collected in the San Bernardino National Forest were kept 3–4 yr prior to the study in greenhouses equipped with activated charcoal filters which removed ambient oxidants. Ponderosa and Jeffrey pine seedlings were grown in 3.8- and 2.9-L containers, respectively, in a soil mix composed of two parts washed plaster sand, one part peat moss, and one part coarse vermiculite (v/v) amended with lime and gypsum. Containers and soil were fumigated with methyl bromide prior to planting. Fertilizer, a 1- or 3-g pellet (depending on container size) of urea-formaldehyde nitrogen, was applied once each year in the early spring.

The single *F. annosus* isolate used in this study was obtained from a wood spore trap exposed to ambient air in the San Bernardino Mountains. Inoculum blocks about 1.7 × 0.5 × 0.5 cm (0.4 cm^3) were prepared from Monterey pine stem sections. These were autoclaved at 121 C for 60 min in glass jars, allowed to cool, inoculated with a conidial suspension of *F. annosus*, and incubated in the dark for 6–10 wk at about 24 C.

Inoculation procedures were similar to those of Kuhlman (11), with the exception that seedlings were not transplanted during the process. Soil was removed from around the root collar and the inoculation site was washed with 95% ethanol. An incision was made into the xylem just above the root collar with a sterile knife. The wound was washed with sterile water, and the inoculum block was inserted. The entire inoculation point was then washed with sterile water and wrapped with masking tape. Uninoculated control seedlings were cut with a sterile knife, washed and wrapped without exposure to inoculum.

The study was designed to test the effects of two chronic ozone dosages as factors predisposing seedlings to *F. annosus* infection and colonization. The dosages are relatively high but not unrealistic. The maximum hourly average dosage of ozone in the field over a year may vary from 0 to 0.35 ppm with a mean of about 0.18 ppm (13). However, seedlings in the field are exposed to pollutants for months or years instead of only several weeks as in this study. Inoculated and uninoculated seedlings were exposed to ozone at 0, 441, and 882 $\mu g/m^3$-hr. In the first treatment (treatment 1), seedlings were inoculated on 27 June at the beginning of fumigation, and fumigation was continued until 23 August (57 days). Thus, total dosages were 0, 3.0×10^5 $\mu g/m^3$ and 6.1×10^5 $\mu g/m^3$. In the second treatment (treatment 2), fumigation began on 27 June, seedlings were inoculated on 2 August, and fumigation was continued through 27 September (92 days). Total dosages were 0, 4.5×10^5 $\mu g/m^3$, and 9.2×10^5 $\mu g/m^3$. In the third treatment (treatment 3), seedlings were not inoculated (checks) but were fumigated for 92 days as in treatment 2. In each treatment, 32 seedlings were exposed to no ozone, 16 seedlings to 441 $\mu g/m^3$ hr ozone, and 16 seedlings to 882 $\mu g/m^3$ hr ozone.

Eight chambers, each 0.9 m × 0.9 m × 1.0 m (0.81 m^3), were used to fumigate seedlings. Chambers were of wood-frame construction with vinyl plastic walls and ceilings which allowed full illumination by sunlight. Refrigerated, charcoal filtered air from a common manifold was introduced at the top of each chamber and escaped from the bottom. Perforated peg board, with larger holes to accommodate seedling stems, formed a floor separating root containers from foliage; this barrier promoted uniform air mixing in the chambers. Ozone was generated by exposing tank oxygen to an electrical discharge and was monitored sequentially in each chamber with a mast ozone analyzer (Mast Development Co., Ames, IA 52803) calibrated with 2% buffered potassium iodide reagent.

Twenty-four seedlings were initially placed in each chamber. They were arranged so that no treatment group was concentrated in any one location. Seedlings were fumigated for 12 hr daily (1000–2200 hours) and watered to soil saturation twice weekly with individual watering tubes to each seedling (Chapin Water-Matic System, Chapin Watermatics Inc., Watertown, NY 13601). Seedlings were monitored for root disease symptoms every 10 days, and a numerical ozone injury rating was determined for each seedling once a month and at the experiment's termination. Both of the two youngest needle whorls (annual stem or branch growth increments that bear needles) were rated on the basis of chlorotic mottle, necrosis, and abscission (0 = none, 1 = very slight, 2 = slight, 3 = moderate, and 4 = severe for each symptom). All numbers were added to arrive at a maximum score of 24.

Seedlings were removed from containers and examined for infection and extent of colonization by *F. annosus* after the specified fumigation intervals. Isolation technique involved either surface sterilization in 10% aqueous sodium hypochlorite or thorough washing with sterile water without surface sterilization. Results obtained by both methods were similar. A selective medium (9) was used for all isolations. Extent of colonization was recorded as the distance to farthest extension up the stem and down the main tap root from the inoculation point.

TABLE 1. Numbers of ponderosa and Jeffrey pine trees per site inoculated with *Fomes annosus* to test effects of oxidant air pollution injury on host susceptibility

	Jeffrey pine		Ponderosa pine	
Air pollution injury[a]	Snow Valley[b]	Holcomb Valley[b]	Breezy Point[b]	Camp Paivika[b]
Very severe (1–8)	0	0	3	2
Severe (9–14)	0	0	6	7
Moderate (15–21)	6	0	3	6
Slight (22–28)	4	2	6	4
Very slight (29–35)	0	2	2	0
No injury (36+)	2	6	0	0
Total	12	10	20	19
Avg. DBH[c] (cm)	38	43	37	30
Avg. Height (m)	12.7	14.8	14.4	11.9

[a] Numerical ratings which follow the categories are based on needle retention, needle length, chlorotic mottling, and branch mortality.
[b] San Bernardino Mountains in southern California.
[c] Trunk diameter at breast height (1.37 m above ground level).

RESULTS

Field inoculations. The "full-screen" analysis of proximal infection of ponderosa pine roots identified two variables that were significantly related to initial infection; these were the inoculum (isolate) and needle retention in the upper crown (NRU), a component of the air pollution injury rating system. The JL isolate infected a significantly greater proportion of inoculated roots of trees in each NRU category than did the other isolates (Table 2) and was the only isolate which infected trees with a NRU of 3 or more yr. For each isolate, the proportion of infected roots was higher for trees with NRU of 2 or 1 yr than that for trees with NRU of 3–4 yr. This difference was highly significant for JL and SV isolates at the Breezy Point plot.

Proximal colonization by the fungus in infected roots also was significantly related to isolate and to NRU of the trees (Table 2). Another variable, needle condition of the first-year needles in the upper crown (NCU1) was found to be almost as significant as NRU. However, since NRU and NCU1 were highly correlated, NCU1 became nonsignificant when included in the equation with NRU. Distal colonization rates showed no distinct trends related to injury.

Differences between rates of colonization for roots of different NRU were examined separately for each fungus isolate and site. For the JL isolate, the colonization rate in roots of trees with NRU of 1 yr was significantly greater than in roots of trees with NRU of 2 or 3 yr. This difference was not significant for SV at Camp Paivika, but the number of roots initially infected was small. For the SV isolate at Breezy Point, the difference between NRU categories was highly significant, even with the small number of infected roots, due to the very rapid colonization rate in trees with NRU of 1 yr.

Exposure chamber study. Generally, the percentage of seedlings that became infected increased with increasing exposure to ozone (Table 3). For Jeffrey pine, there were significant differences ($P = 0.05$) in percentage infection between ozone fumigated and nonfumigated trees. However, there were no significant differences ($P = 0.05$) in infection among treatments for ponderosa pine seedlings.

Product-moment correlations (Jeffrey pine r = 0.27; ponderosa pine r = 0.36; $P = 0.05$) and regression analyses (Jeffrey $P = 0.05$; ponderosa $P = 0.01$) comparing ozone dosage and *F. annosus* colonization showed a significant relationship for both species. The relationship between ozone dosage in the range tested and fungal colonization for both species was linear. Regression line slopes for the two species were not significantly different, indicating that the relationship between pathogen colonization and ozone exposure was approximately the same.

Regression analyses of relationships between ozone injury (instead of dosage) and extent of colonization by *F. annosus* showed statistical significance for both Jeffrey and ponderosa pine ($P = 0.05$) (Fig. 1). Comparison of regression line slopes between species indicated that the slopes were not significantly different. Ponderosa and Jeffrey pine were colonized by the fungus at about the same rate over a similar range of ozone injury. Product-moment correlations (ponderosa pine r = 0.46; Jeffrey pine r = 0.28) were significant ($P = 0.05$) for both species.

Predisposition of ponderosa pine by exposure to ozone for 38 days before inoculation resulted in significantly more colonization by *F. annosus* compared to seedlings inoculated just prior to fumigation ($P = 0.01$).

DISCUSSION

Results of these studies indicate that injury to pines caused by oxidant air pollutants increases the susceptibility of roots of injured trees to *Fomes annosus*. Data from both the greenhouse-ozone fumigation study with seedlings and the field inoculations of large trees support this conclusion. The trees inoculated in the field were undoubtedly exposed to other oxidants as well as to ozone (eg, nitrogen oxides). However, prior studies (12,15) have indicated that ozone alone occurs at dosages high enough to explain most of the injury observed. Hence, we believe that results of the fumigation study under controlled conditions supports the field investigation.

In the field inoculations, two of the three *F. annosus* isolates failed to infect roots of ponderosa pine trees that retained 3 or more years' needles (NRU $\geqslant$ 3 yr), whereas the same isolates infected about 30% of the inoculated roots of trees with 1–2 yr retention. The third isolate infected about 30% of the roots of trees with NRU = 3 yr, and all roots of trees with NRU = 1–2 yr. These differences in isolate virulence also were noted in other studies of sapwood decay and stump susceptibility (10). Such differences

TABLE 2. Proximal root infection and colonization by *Fomes annosus* of ponderosa pine trees with different levels of oxidant air pollution injury (expressed as upper crown needle retention) in the San Bernardino Mountains of southern California

Site	Isolate	Needle retention in upper crown (yr)	Roots inoculated (no.)	Roots with proximal infection (%)	Average proximal colonization (mm/mo)
Camp Paivika	SV	1	6	33	15
		2	7	43	20
		3	8	0	···
Camp Paivika	JL[a,b]	1	6	100	45[c,d]
		2	6	100	13[c]
		3	7	29	6[d]
Camp Paivika	CA	1	2	0	···
		2	2	50	23
		3	5	0	···
Breezy Point	SV[b]	1	10	30	13[c]
		2	14	21	157[c]
		3	12	0	···
		4	4	0	···

[a] Percent roots infected by JL was significantly higher ($P = 0.001$) than % roots infected by the other isolates.

[b] Percent roots infected by JL and SV was significantly higher ($P = 0.003$ and 0.01, respectively) for trees with NRU of 1–2 yr than for trees with 3 or 4 yr NRU.

[c] Within isolate, difference between pair of means is highly significant ($P = 0.01$).

[d] Difference between this pair of means is significant ($P = 0.05$).

TABLE 3. Infection and colonization of ozone fumigated Jeffrey and ponderosa pine seedlings by *Fomes annosus*

Inoculation schedule[x]	Ozone dosage ($\mu g/m^3$)[y]	Jeffrey pine Infection (%)	Jeffrey pine Average colonization (cm)[z]	Ponderosa pine Infection (%)	Ponderosa pine Average colonization[z] (cm)
Concurrent	0	38	1.3 dfh	75	0.5
Postfumigation	0	68	1.0 egi	50	0.8
Mean		53	1.2	62	0.6
Concurrent	441	88	3.4 de	88	0.7
Postfumigation	441	62	1.5 j	75	0.7
Mean		75	2.5	81	0.8
Concurrent	882	75	2.4 fg	75	0.8
Postfumigation	882	88	3.4 hij	75	3.2 k
Mean		81	2.9	75	2.1

[x] Concurrent = inoculation at beginning of fumigation; Postfumigation = inoculation after an initial fumigation of 37 days with fumigation continued an additional 55 days.

[y] Fumigation 12 hr/day for 58 days (concurrent) or for 92 days (postfumigation).

[z] Within columns, values followed by the same letter differ significantly ($P = 0.05$), except for k, which indicates that this value differs significantly from all others in the column. For colonization Duncan's Multiple-Range comparison test was used; for infection the Fisher-Yates Test of Significance in 2 × 2 Contingency Tables was used.

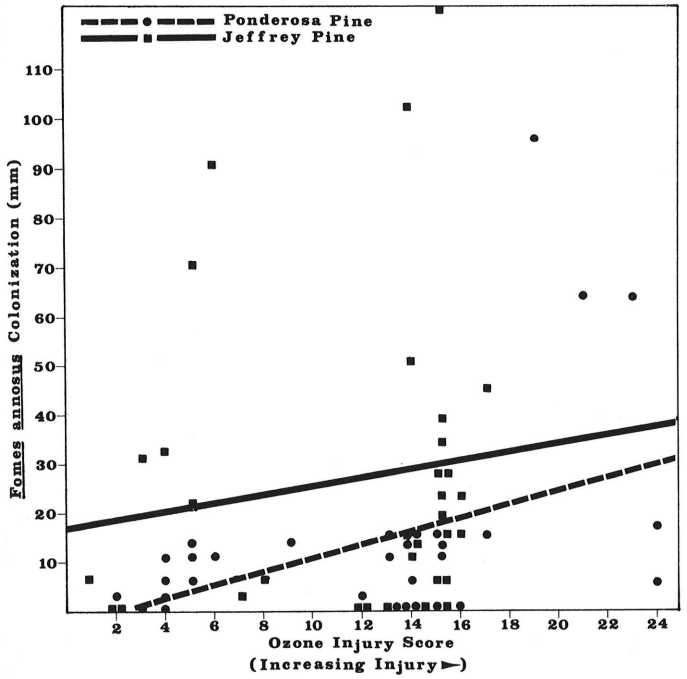

Fig. 1. Relationship between oxidant injury score and *Fomes annosus* colonization of ponderosa and Jeffrey pine seedlings subjected to chronic fumigation with ozone at 441 and 882 $\mu g/m^3$-hr.

might have substantial effects upon pathogen ecology and upon pathogen/host injury interactions.

There were oxidant injury related differences in colonization as well as infection rates. In roots of trees with NRU = 3 yr which became infected, *F. annosus* colonized the xylem tissue at a rate of only about 6 mm per mo. The average for roots of trees with NRU = 1–2 yr was about seven times greater. The implications of such increases in both *F. annosus* infection and rate of colonization associated with oxidant injury are serious. Over a period of several disease cycles, dramatic increases in disease incidence could be expected in forest stands with injured trees.

Although the field inoculations of Jeffrey pine roots were excluded from analyses because an insect infestation occurred in conjunction with oxidant injury, we believe that the results obtained with ponderosa pine would apply to that species as well. Results of the fumigation study indicated that the susceptibility of Jeffrey pine seedlings to *F. annosus* might be affected as much or more by exposure to ozone as that of ponderosa pine.

Distal colonization of severed ponderosa pine roots showed no relation to air pollution injury. Once roots were detached from the host, they became almost uniformly susceptible to *F. annosus*; these results confirm the report by Gibbs (5). In comparison, proximal colonization rates were much less. Slower growth of *F. annosus* in roots still attached to the tree may be due to the continuous supply of food (5) to and resin production (16) in these roots.

Host resin production appears to limit the extent of *F. annosus* colonization in roots (18,20,21). Often there is heavy resinosis which signifies host resistance (7) in the advancing zone of colonization (16). Resin impregnation of wood proximally from the point of inoculation was visibly less in roots from trees severely injured by air pollution. This same characteristic has been found in suppressed and subdominant trees (4) where host vigor, which may be partially expressed as resin production, is inversely related to root susceptibilty to *F. annosus*.

Heretofore, the increase in susceptibility of forest trees to root pathogens such as *F. annosus* and its long-term economic impact have not been considered in assessing the damage caused by oxidant air pollutants. Such increases in susceptibility may far outweigh the direct effects upon the hosts and should be evaluated in deliberations involving air pollution standards.

LITERATURE CITED

1. BEGA, R. V., and R. S. SMITH, Jr. 1966. Distribution of *Fomes annosus* in natural forests in California. Plant Dis. Rep. 50:832-836.
2. COBB, F. W., Jr., and R. W. STARK. 1970. Decline and mortality of smog-injured ponderosa pine. J. For. 68:147-148.
3. COBB, F. W., Jr., D. L. WOOD, R. W. STARK, and J. R. PARMETER, Jr. 1968. Photochemical oxidant injury and bark beetle (Coleoptera: Scolytidae) infestation of ponderosa pine. IV. Theory on the relationships between oxidant injury and bark beetle infestation. Hilgardia 39:141-152.
4. GIBBS, J. N. 1967. The role of host vigour in the susceptibility of pines to *Fomes annosus*. Ann. Bot. 31:803-815.
5. GIBBS, J. N. 1968. Resin and the resistance of conifers to *Fomes annosus*. Ann. Bot. 32:649-665.
6. HODGES, C. S., Jr. 1969. Modes of infection and spread of *Fomes annosus*. Annu. Rev. Phytopathol. 7:247-266.
7. HODGES, C. S., Jr. 1969. Relative susceptibility of loblolly, longleaf and slash pine roots to infection by *Fomes annosus*. (Abstr.) Phytopathology 59:1031.
8. HOWELL, F. C., and W. J. STAMBAUGH. 1972. Rates of pathogenic and saprophytic development of *Fomes annosus* in roots of dominant and suppressed eastern red cedar. Plant Dis. Rep. 56:987-990.
9. HUNT, R. S., and F. W. COBB, Jr. 1971. Selective medium for isolation of wood-rotting basidiomycetes. Can. J. For. Res. 6:159-165.
10. JAMES, R. L., F. W. COBB, Jr., W. W. WILCOX, and D. ROWNEY. 1980. Effects of photochemical oxidant injury of ponderosa and Jeffrey pines on susceptibility of sapwood and freshly-cut stumps to *Fomes annosus*. Phytopathology 70: (In press).
11. KUHLMAN, E. G. 1969. Inoculation of loblolly pine seedlings with *Fomes annosus* in the greenhouse. Can. J. Bot. 47:2079-2082.
12. MILLER, P. R. 1973. Oxidant-induced community change in a mixed conifer forest. Adv. Chem. Ser. 122:101-117.
13. MILLER, P. R., and M. J. ELDERMAN, eds. 1977. Photochemical oxidant air pollutant effects on a mixed conifer forest ecosystem: a progress report, 1976. EPA-600/3-77-104. 339 pp.
14. MILLER, T. 1964. Host factors influencing infection and growth of *Fomes annosus* in roots of *Pinus taeda*. (Abstr.) Phytopathology 54:901.
15. PARMETER, J. R., Jr., and P. R. MILLER. 1968. Studies relating to the cause of decline and death of ponderosa pine in Southern California. Plant Dis. Rep. 52:707-711.
16. RISHBETH, J. 1950. Observations on the biology of *Fomes annosus*, with particular reference to East Anglian pine plantations. III. Natural and experimental infection of pines, and some factors affecting severity of the disease. Ann. Bot. 15:221-246.
17. ROSS, E. W., and C. S. HODGES, Jr. 1964. *Fomes annosus* infections in young slash pine plantations. (Abstr.) Phytopathology 54:904.
18. SCHNURBEIN, Von U. V. 1972. Bedeuntung des Harzflusses fur die infection von Wurzelwunden durch den Wurzelschwamm *Fomes annosus* (Fr.) Cooke. Forstwiss. Centralbl. 91:364-376.
19. TOWERS, B. 1964. Root-contact infection by *Fomes annosus* in a thinned loblolly pine plantation. Plant Dis. Rep. 48:767-769.
20. TOWERS, B. and W. J. STAMBAUGH. 1968. The influence of induced soil moisture stress upon *Fomes annosus* root rot of loblolly pine. Phytopathology 58:269-272.
21. WALLIS, G. W. 1961. Infection of Scots pine roots by *Fomes annosus*. Can. J. Bot. 39:109-121.