IN: Morrison, D. J. (ed.). Proceedings of the Seventh International Conference on Root and Butt Rots. Vernon and Victoria, British Columbia, Canada. August 9-16, 1988. IUFRO Working Party S2.06.01 Forestry Canada, Pacific Forestry Centre, Victoria, B. C., Canada. pp. 513-520.

INTERACTIONS BETWEEN PHOTOCHEMICAL AIR POLLUTION AND *HETEROBASIDION ANNOSUM* IN A MIXED CONIFER FOREST ECOSYSTEM

R. L. James

Timber, Cooperative Forestry and Pest Management, USDA Forest Service, Northern Region, Coeur d'Alene, Idaho 83814

F. W. Cobb, Jr.

Department of Plant Pathology, University of California, Berkeley, CA 94720

SUMMARY

Within the mixed conifer forest ecosystem of the San Bernardino Mountains in southern California, photochemical air pollution significantly affects the epidemiology of the root pathogen *Heterobasidion annosum*. Most aspects of the disease cycle of *H. annosum* in ponderosa and Jeffrey pine are enhanced by oxidant pollution. An analytical model, prepared using data from several experimental studies, predicts that tree losses from annosum root disease are 6.5 times greater in stands severely injured by air pollution as compared with stands only moderately or slightly injured. The proposed model requires validation under natural forest conditions.

INTRODUCTION

Photochemical air pollutants cause chlorotic decline of ponderosa (*Pinus ponderosa* Laws.) and Jeffrey (*Pinus jeffreyi* Grev. and Balf.) pines in the San Bernardino Mountains of southern California (Parmeter and Miller 1968). Severely affected trees are usually killed by biotic agents such as bark beetles (Cobb and Stark 1970) and root pathogens, e.g. *Heterobasidion annosum* (Fr.) Bref. (= *Fomes annosus* (Fr.) Cke.) (James 1977) which is widely distributed throughout the San Bernardino Mountains.

As part of a large interdisciplinary project to determine pollutant effects of forest ecosystems, studies were conducted to evaluate possible effects of photochemical air pollution on crucial portions of the typical disease cycle of *H. annosum* within a mixed conifer ecosystem in the San Bernardino Mountains. Studies of ponderosa and Jeffrey pines stump infection and rates of stump and sapwood colonization (James et al. 1980b) indicated that twice as much surface area is

colonized by the fungus in stumps from severely air pollution-injured trees as compared with trees with slight or no injury. Also, rate of vertical colonization of stumps by H. annosum was almost 50% greater in stumps of pollution-injured Jeffrey pine and about 29% greater in stumps of injured ponderosa pine. In studies of root susceptibility to infection (James et al. 1980a), pine trees injured by photochemical air pollution were infected to a greater extent (about 30%) than trees not injured. Rate of H. annosum root colonization toward the stem was seven times greater in trees with severe air pollution injury. Although studies of the effects of ozone on conidial production, germination, and growth in culture of H. annosum showed a substantial effect (James et al. 1982), environmental conditions and incidence of spore dispersal (James and Cobb 1984) suggested that these effects may be minimal in the field. Also, the capacity (or virulence) of the fungus to infect pine trees is apparently not affected by exposure to photochemical air pollution (James and Cobb 1982). These results were used to develop a predictive model for use in forest ecosystems where both root diseases and air pollution cause serious damage. The end product of this model is the rate of mortality resulting from H. annosum infection as mediated by oxidant air pollution.

QUANTIFICATION OF AIR POLLUTION EFFECTS

To illustrate quantitative effects of oxidants on spread of H. annosum, two hypothetical mixed conifer stands will be compared. Both stands include ponderosa or Jeffrey pine, incense cedar (Libocedrus decurrens Torr.), white fir (Abies concolor (Gord. & Glend.) Lindl.), Douglas-fir (Pseudotsuga menziesii (Mirb.) Franco), and sugar pine (Pinus lambertiana Dougl.). Initial incidence of H. annosum root disease and tree and site conditions are identical in both stands. The only major difference between the stands is the level of oxidant air pollution injury present. Stand "A" exhibits slight to moderate air pollution damage with an average upper crown needle retention of 4 years (Miller 1973). Stand "B" is severely damaged by air pollution with an overall upper crown needle retention of 2 years. Comparison of H. annosum disease development between Stands "A" and "B" will be made using equations and data from several studies noted above.

Both stands contain 1,000 ponderosa and Jeffrey pine trees mixed with other conifer species. One hundred pine trees are selectively cut in each stand. No data are available on natural pine stump infection by H. annosum in the San Bernardino Mountains, but on pine in other areas (Rishbeth 1950; Stambaugh et al. 1962) the rate varies from about 36 to 96 percent. We will assume that initial inoculum is not a limiting factor and that 75 percent of the freshly-cut stumps become infected in stands with slight to moderate air pollution injury level. We would expect a 5-25 percent increase in stump

infection in severely injured stands (James et al. 1980b); however, to be conservative we propose to use a 5% increase. We recognize that after a few H. annosum generations there will likely be much more inoculum in the pollution-injured stands than in noninjured stands.

The expected number of lateral stump roots infected for each stand can be calculated from regression equations (James et al. 1980b). In this analysis, the regression equations representing the greatest colonization ratio for ponderosa pine will be used to more closely simulate natural conditions where the more virulent isolates are likely to cause disease.

Stand "A" (Y = Average percent surface colonization; X = Average upper crown needle retention)

Y = 113.5 - 19.6 X; where X = 4, Y = 35.1%

Since this percentage is directly related to the probability of stump lateral root infection (Meredith 1960), the number of infected stump roots would become:

(35.1%) (6) = 2.1 roots/stump NOTE: Six is the average number of lateral roots per tree.

Stand "B": Y = 113.5 - 19.6 X; where X = 2, Y = 74.3%

The number of infected stump roots:

(74.3%) (6) = 4.5 roots/stump

The total number of infected stump roots for each stand (# stumps infected X # roots infected/stump):

Stand "A": # stumps infected = 75

roots infected/stump = 2.1

Total # infected stump roots = 158

Stand "B": # stumps infected = 80

roots infected/stump = 4.5

Total # infected stump roots = 360

Average percentage of root contacts for both stands would approximate 20 percent (D. J. Goheen, personal communication). Therefore, total number of infected stump roots which could theoretically transmit the fungus to contacted live tree roots would be: # roots infected X average % root contact.

Stand "A": 158 roots X 20% = 32 roots

Stand "B": 360 roots X 20% = 72 roots

If all contacted live tree roots became infected, and each was on a different tree, about 32 infected trees in Stand "A" and 72 trees in Stand "B" would become infected. However, based on our experience with root inoculations (James et al. 1980a), not all contacted live tree roots would likely become infected. Therefore, if data from our root inoculations are used, we could expect 13.8 percent of the contacted roots in Stand "A" and 47.2 percent of such roots in Stand "B" to become infected.

Thus, for Stand "A", the number would be:

(13.8%) (32) = 4 infected roots

For Stand "B", the number would be:

(47.2%) (72) = 34 infected roots

If each of these infected roots is on a different tree, there are four infected trees in Stand "A" compared to 34 infected trees in Stand "B".

To estimate number of potential tree deaths during a certain time period, colonization rates by the pathogen must be considered. Fungal development within infected stumps is different than in infected trees (James et al. 1980a; James et al. 1980b). For the stump colonization portion of the cycle (James et al. 1980b), the expected rate would be:

Stand "A": $Y = 41.8 - 0.316 X$; where $X = 4$ (average upper crown needle retention), $Y = 22$ mm/month

Stand "B": $Y = 41.8 - 0.316 X$; where $X = 2$, $Y = 39$ mm/month

For the root colonization portion of the cycle (James et al. 1980a), the expected rate would be:

Stand "A": 6 mm/month

Stand "B": 41 mm/month

If the average distance required for the fungus to move between trees is 2 meters (this assumes equal spacing among trees and equal lengths of roots from the root collar to the point of contact with adjacent roots), time required for fungal spread from tree to tree is:

Stand "A": Stump roots Live tree roots

$$\frac{1{,}000 \text{ mm}}{22\text{mm/month}} + \frac{1{,}000 \text{ mm}}{6 \text{ mm/month}} =$$

45 months + 167 months = 212 mos. or 17.7 yr.

Overall rate of spread:

$$\frac{2{,}000 \text{ mm}}{\frac{1{,}000}{22} + \frac{1{,}000}{6}} = 9.4 \text{ mm/month}$$

Stand "B": Stump roots Live tree roots

$$\frac{1{,}000 \text{ mm}}{39 \text{ mm/month}} + \frac{1{,}000 \text{ mm}}{41 \text{ mm/month}} =$$

26 months + 24 months = 50 mos. or 4.2 yr.

Overall rate of spread:

$$\frac{2{,}000 \text{ mm}}{\frac{1{,}000}{39} + \frac{1{,}000}{41}} = 40.0 \text{ mm/month}$$

The calculated rates of spread in stands are much less than would be expected in nature. For example, in many nonstressed pine stands, H. annosum colonizes at least 1/2 meter of root tissue annually (Cobb, unpublished; Felix et al. 1974; Hodges 1974). The colonization rate probably accelerates as the trees age and the fungus advances through roots. However, if we assume that colonization follows a typical linear acceleration rate, the total time for spread from tree to tree could be calculated as follows:

Total time:

$$\frac{\text{Length of spread}}{\frac{\text{Initial colon. rate} + \text{Final colon. rate}}{2}}$$

Stand "A":

Final colon. rate = 1 meter/year (Felix et al. 1974: Hodges 1974)

$$\frac{1{,}000 \text{ mm}}{12} = 83.8 \text{ mm/month}$$

Total time:

$$\frac{2{,}000 \text{ mm}}{\frac{9.4 \text{ mm/month} + 83.8 \text{ mm/month}}{2}}$$

= 42.9 months (3.6 years)

Stand "B":

If we assume that Stand B has the same percent change frominitial colonization rate to final colonization rate as Stand A, the following final colonization rate would be expected:

Rate of change: $\frac{83.8}{9.4} = 8.9$

Final colonization rate:

8.9 X 40.0 mm/month = 356.0 mm/month

Total time:

$$\frac{2{,}000 \text{ mm}}{\frac{40.0 \text{ mm/month} + 356.0 \text{ mm/month}}{2}}$$

= 10.1 months (0.8 years)

If we assume 50 years to harvest in both stands, the following computations represent expected number of tree deaths from infection by H. annosum during this time. We also assume that a tree dies after the fungus colonizes its roots from the point of contact to the root collar. Actually, some additional time may be required for cambial girdling.

Stand "A":

Total trees killed = $\sum_{i=1}^{14} (Xi)(0.138)$

Xi = number of trees infected at the beginning of each disease cycle.

X_1 = 4 (see page 4).

cycles = $\frac{50 \text{ years}}{3.6 \text{ years}} = 14$

Total trees killed = 17 in 50 years

Stand "B":

Total trees killed = $\sum_{i=1}^{62} (Xi)(0.472)$

X_1 = 34 (see page 4).

cycles = $\frac{50 \text{ years}}{0.8 \text{ years}} = 62$

Total trees killed = 110 in 50 years

This model therefore predicts the death of 17 and 110 trees in Stands "A" and "B", respectively, in 50 years.

CONCLUSIONS

Our model predicts that 6.5 times as many trees are killed by H. annosum within the same time interval in stands severely impacted by oxidant air pollution as in stands with moderate or slight injury. Field testing is required to evaluate validity of the model.

Accuracy of the model can be improved by additional studies on other aspects of the disease cycle. For example, information on oxidant effects of basidiospore production, release, and germination would improve our knowledge of stump infection probabilities ass well as inoculum buildup over time. Theoretically, the fungus might develop tolerance to gaseous pollutants which would render it more fit in environments subjected to prolonged oxidant exposures. Another important aspect of the disease cycle which merits attention is direct and indirect effects of oxidants on microorganisms antagonistic to H. annosum, particularly stump colonizing fungi like Trichoderma spp. Since abundance and aggressiveness of these antagonists greatly affect pathogenic activities of H. annosum (Meredith 1959; Rishbeth 1959), their effects as mediated by air pollution should be evaluated.

We conclude that photochemical air pollution has substantial effects on H. annosum epidemiology in the direction of increased disease losses. Therefore, such effects should be considered when managing forests where both annosum root disease and air pollution are damaging.

LITERATURE CITED

Cobb, F. W., Jr. and R. W. Stark. 1970. Decline and mortality of smog-injured ponderosa pine. J. For. 68, 147-148.

Felix, L. S., J. R. Parmeter, Jr., and B. Uhrenholdt. 1974. Fomes annosus as a factor in the management of recreational forests. In: Kuhlman, E. G. (ed.). Proceedings Fourth Int. Conf. on Fomes annosus. Athens, GA. 1973. Southeastern For. Exp. Station, USDA Forest Service. pp. 2-7.

Hodges, C. S., Jr. 1974. Relative susceptibility of slash, loblolly and longleaf pines to infection by Fomes annosus. In: Kuhlman, E. G. (ed.). Proceedings Fourth Int. Conf. on Fomes annosus. Athens, GA. 1973. Southeastern For. Exp. Station, USDA Forest Service. pp. 86-91.

James, R. L. 1977. The effects of photochemical air pollution

on the epidemiology of Fomes annosus. Ph.D. Dissertation. University of California, Berkeley. 200 pp.

James, R. L. and F. W. Cobb, Jr. 1982. Variability in virulence of Heterobasidion annosum isolates from ponderosa and Jeffrey pine in areas of high and low photochemical air pollution. Plant Disease 66, 835-837.

James, R. L. and F. W. Cobb, Jr. 1984. Spore deposition by Heterobasidion annosum in forests of California. Plant Disease 68, 246-248.

James, R. L., F. W. Cobb, Jr. and J. R. Parmeter, Jr. 1982. Effects of ozone on sporulation, spore germination, and growth of Fomes annosus. Phytopathology 72, 1205-1208.

James, R. L., F. W. Cobb, Jr., P. R. Miller and J. R. Parmeter, Jr. 1980a. Effects of oxidant air pollution on susceptibility of pine roots to Fomes annosus. Phytopathology 70:560-563.

James, R. L., F. W. Cobb, Jr., W. W. Wilcox and D. L. Rowney. 1980b. Effects of photochemical oxidant injury of ponderosa and Jeffrey pines on susceptibility of sapwood and freshly cut stumps to Fomes annosus. Phytopathology 70, 704-708.

Meredith, D. S. 1959. The infection of pine stumps by Fomes annosus and other fungi. Ann. Bot. 23, 455-476

Meredith, D. S. 1960. Further observations on fungi inhabiting pine stumps. Ann. Bot. 24, 63-78.

Miller, P. R. 1973. Oxidant-induced community change in a mixed-conifer forest. Adv. Chem. Sci. 122, 107-117.

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. Rept. 52, 707-711.

Rishbeth, J. 1950. Observations on the biology of Fomes annosus with particular reference to East Anglian pine plantations. II. Spore production, stump infection, and saprophytic activity in stumps. Ann. Bot. 15, 1-22.

Rishbeth, J. 1959. Dispersal of Fomes annosus Fr. and Peniophora gigantea (Fr.) Massee. Trans. Br. Mycol. Soc. 42, 243-260.

Stambaugh, W. J., F. W. Cobb, Jr., R. A. Schmidt and F. C. Krieger. 1962. Seasonal inoculum dispersal and white pine stump invasion by Fomes annosus. Plant Dis. Rept. 46, 194-198.