Association of Soil Moisture with Spread of *Ceratocystis wageneri* in Ponderosa Pine Disease Centers

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ABSTRACT

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Soil redox potentials, soil water potentials, and soil temperatures were measured at naturally occurring centers of black stain root disease, caused by *Ceratocystis wageneri*, in *Pinus ponderosa* in the central Sierra Nevada. Soil redox potentials were relatively high, probably because of low soil temperatures during periods of high soil moisture, and no relationship between redox potential and rate of disease spread was apparent. Soil water potentials, particularly in May, were strongly related to disease spread, indicating that disease spread in the field is favored on wetter sites.

Ceratocystis wageneri Goheen et Cobb (anamorph *Verticicladiella wageneri* Kend.) causes black stain root disease of various conifers (2,6,9,12,13) which, in the Sierra Nevada of California, is important primarily on ponderosa pine (*Pinus ponderosa* Laws.). The disease occurs in localized centers that appear to be initiated via single infected trees. In the central Sierra Nevada, infection occurs most often through small rootlets (<5 mm in diameter) of healthy trees that usually do not touch but are within 15 cm of a diseased root and within 20–50 cm of the soil surface (4). Growth of the fungus through soil has been demonstrated (8) and could be the major means of dispersal between diseased and healthy roots (7).

Relatively high soil moisture levels have been shown to enhance infection of inoculated ponderosa pine seedlings (5). Severe mortality caused by the disease in southern California pinyons was associated with years of heavy, well-distributed summer rains (13), and the disease was found most frequently in Colorado on cool, moist sites (9). In the central Sierra Nevada, a high proportion of the largest and most active infection sites occurs in moist, cool areas, and spread of the pathogen appears to be more rapid in gullies and small creek drainages (3,4). The disease is not found in ponderosa pine on drier sites at lower elevations in this area (1,14).

Wilks et al (14) found infection and colonization rates of ponderosa pine seedlings inoculated in the greenhouse to vary with soil redox potential. Maximum infection and colonization generally occurred under slightly to moderately impaired aeration conditions (about +600 to +300 mV at pH 5.9), with lesser amounts under well-aerated and poorly aerated conditions (above about +700 and below 0 mV, respectively).

This study was undertaken to relate site wetness, soil redox potentials, and soil temperatures to rate of prior disease spread in naturally occurring *C. wageneri* centers.

MATERIALS AND METHODS

The study was conducted on the Georgetown Divide, El Dorado County, CA, in the central Sierra Nevada (elevation 1,300–1,400 m) from January 1978 to May 1979. Eighty-eight soil redox/moisture/temperature stations were established in October 1977 along 14 transects across eight naturally occurring mortality centers in ponderosa pine. The transects ran along major axes of spread from the apparent origins of centers or from areas of old (pre-1963) mortality to points beyond existing margins. Disease centers to be studied were chosen from those for which spread maps had been prepared from sequential aerial photographs taken in 1963–1974 (3). Selection was made on the basis of high, uniform ponderosa pine densities, avoidance of sites with extreme soil disturbances from logging or research operations, and a range of spread rates. In some cases, 1976 and 1978 boundaries were added to maps of chosen centers. An example of one of the transects and disease spread plots is shown in Figure 1. Disease centers ranged in size from 0.01 to 25.0 ha, elevations ranged from 1,290 to 1,335 m, and ponderosa pine densities in transect zones ranged from 25 to 200 trees larger than 15 cm dbh per hectare. Soil pH ranged from 5.1 to 5.9.

Enlargement of disease centers was variable over time, both within and between centers. Therefore, increases in disease center areas were calculated on the basis of 4- and 5-yr periods, ie, 1964–1967, 1968–1972, and 1973–1976 in most cases. Normalized spread "rates" (m/yr) were derived from the observed area of new mortality (m^2) during the given period by dividing by the length of the boundary of that zone with the previous margin (m) and by the time interval (yr). The normalization was

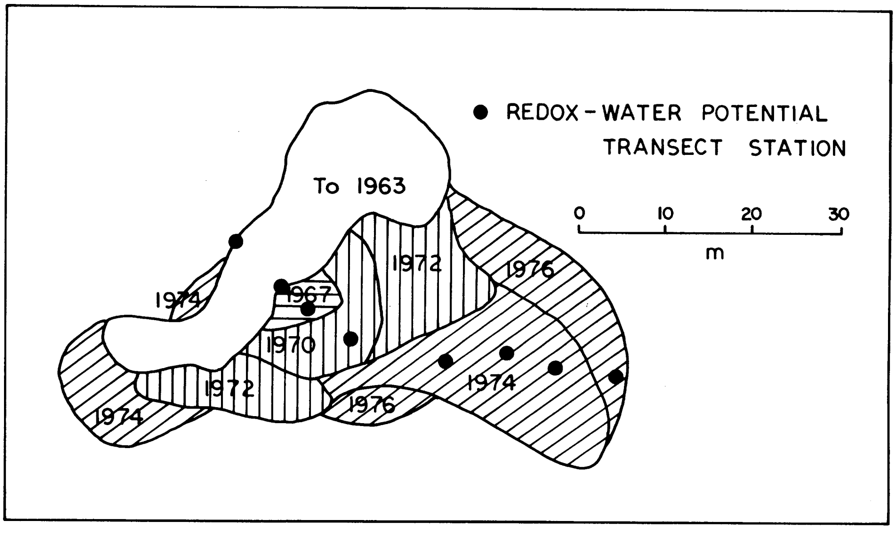

Fig. 1. Sequential spread map of mortality caused by *Ceratocystis wageneri* in ponderosa pine, showing locations of transect stations.

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performed to compare the areas of new mortality associated with disease centers of widely varying size. Stations at boundaries between zones were considered to represent the later (advancing) zone. Stations where mortality had not occurred as of 1978 were not included in the analysis.

Three bright, 0.95-mm-diameter platinum wire electrodes were installed at a depth of 30 cm at each station. Electrodes were inserted horizontally from the bottoms of holes dug to the required depth with trowels in order to disturb soil around the electrodes as little as possible. Small holes 10–15 cm long were bored radially outward from the vertical hole with a 0.95-cm-diameter wood drill. This allowed a snug fit of the 1-cm-diameter electrode body while minimizing compaction during installation. Thermistor probes were installed similarly at selected stations. Trowel holes were refilled horizon by horizon, and electrode jacks were mounted on stakes and protected by plastic bags.

Soil redox potentials were measured with a Photovolt model 126A pH/millivolt meter and saturated calomel electrode. Replicate measurements at each station were generally within 100 mV of each other and were averaged unless one electrode registered very differently and appeared to be malfunctioning. Soil temperatures were measured with a calibrated ohmmeter (YSI 42SC, Yellow Springs Instrument Co., Yellow Springs, OH). Soil water potentials at 30 cm deep and a short distance away from the redox electrodes were measured with a null-type ("quick-draw") tensiometer (Soil Moisture Co., Inc., Santa Barbara, CA). Measurements were made seven times from January through May 1978, once in late July 1978, and once in late May 1979. Not all stations were visited on each occasion during the early part of the study because of winter storms.

RESULTS

Soil water potentials and temperatures appeared to fluctuate rather slowly, and the soil environment could be characterized by three fairly distinct conditions. From January through April (four measurement dates), water potentials and temperatures were low, ranging from +0.002 to −0.014 MPa ($\bar{x} = -0.0059$ MPa, SD = 0.003), and from 2 to 6 C ($\bar{x} = 4.0$, SD = 1.2), respectively. For May of both years (four measurement dates), water potentials varied widely between sites, ranging from +0.001 MPa to beyond the −0.05 MPa range of the tensiometer (for measurable values, $\bar{x} = -0.022$ MPa, SD = 0.0124). Soil temperatures ranged from 8 to 10 C, except for one measurement of 4 C ($\bar{x} = 8.6$, SD = 1.6). In July, essentially all water potentials were beyond the tensiometer range (−0.026 MPa was measured once in an intermittent stream bed), and soil temperatures ranged from 12 to 15 C ($\bar{x} = 13.7$, SD = 0.9).

Water potential, redox potential, and temperature data were each grouped for analysis. Two representative values for each station were calculated by averaging spring measurements made before ("early" values) and after ("late" values) the soil began to dry in early May. The average values were all highly correlated with their individual component data sets ($0.85 < r < 1.0$), with the exception of one group of seven water potential measurements made in late April ($r = 0.67$).

Redox potentials were generally very high and relatively constant ($\bar{x} = +750$ mV, SD = 50) and showed no discernible relationship to rates of disease spread. Soil temperature also showed no independent relationship to disease spread. Water potentials were significantly related to the previously observed spread rates. Figures 2 and 3 show normalized rates of spread of new mortality plotted against representative early (P_E) and late (P_L) water potentials. Six of the 10 stations with high rates of spread were lost because of logging in May 1978 and are therefore not represented in Figure 3. Historical disease spread was significantly related to both P_E and P_L. The relationship between P_E and spread rate is described by the linear regression equation $s = 457\ P_E + 7.53$ ($r = 0.44$, $P = 0.001$), and the May data are described by the equation $s = 1.87 - 0.0382/P_L$ ($r = 0.60$, $P = 0.001$). Regressions of individual components (data sets gathered on given dates) of P_E and P_L against spread rate yielded similar relationships.

DISCUSSION

Redox potentials appeared to have little relation to rates of disease spread. Lowest observed values were in the range of +550 to +650 mV and were detected only rarely. Soil moisture sufficient to seriously impede aeration occurred only in conjunction with soil temperatures of 4 C or lower, which apparently limited microbial respiration sufficiently to prevent development of reducing conditions. Similarly high, uniform redox potentials have been reported for forest soils with comparable water potentials and temperature regimes (10,11).

Although soil moisture measurements were necessarily made in years other than those in which host mortality occurred, they do serve to characterize the relative degree of wetness of the sites. The measurements strongly indicate that disease spread in the field is favored by higher soil moisture conditions. This is particularly evident in the data for May (Fig. 3), when a relatively broad range of soil moisture levels was measured, but is also suggested by the data for winter and early spring (Fig. 2), when all sites were relatively wet.

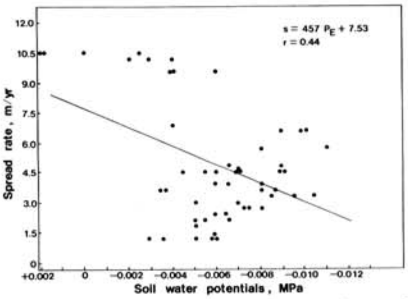

Fig. 2. Scatterplot and regression of *Ceratocystis wageneri* infection center spread rates (m/yr) versus January–April soil water potentials (MPa).

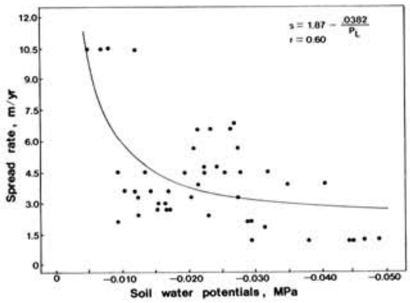

Fig. 3. Scatterplot and regression of *Ceratocystis wageneri* infection center spread rates (m/yr) versus May soil water potentials (MPa).

This study supports results of previous observational and greenhouse work relating disease severity and soil moisture levels and further indicates the possibility of hazard rating based on soil moisture. On the western slopes of the central Sierra Nevada, pure stands of ponderosa pine should be avoided in the zone normally occupied by mixed conifer stands on sites characterized by relatively high, late-season water potentials.

LITERATURE CITED

1. Byler, J. W., Cobb, F. W., Jr., and Rowney, D. L. 1979. An evaluation of black stain root disease on the Georgetown Divide, El Dorado County, California. U.S. For. Serv., S.W. Reg. For. Insect Dis. Manage., San Francisco, Rep. 79-2. 15 pp.
2. Cobb, F. W., Jr., and Platt, W. D. 1967. Pathogenicity of *Verticicladiella wageneri* to Douglas-fir. Phytopathology 57:998-999.
3. Cobb, F. W., Jr., Slaughter, G. W., Rowney, D. L., and DeMars, C. J. 1982. Rate of spread of *Ceratocystis wageneri* in ponderosa pine stands in the central Sierra Nevada. Phytopathology 72:1359-1362.
4. Goheen, D. J. 1976. *Verticicladiella wageneri* on *Pinus ponderosa*: Epidemiology and interrelationships with insects. Ph.D. thesis. University of California, Berkeley. Diss. Abstr. 37(9):4249-B.
5. Goheen, D. J., Cobb, F. W., Jr., and McKibbin, G. N. 1978. Influence of soil moisture on infection of ponderosa pine by *Verticicladiella wageneri*. Phytopathology 68:913-916.
6. Goheen, D. J., and Hansen, E. M. 1978. Black stain root disease in Oregon and Washington. Plant Dis. Rep. 62:1098-1102.
7. Hicks, B. R. 1978. Growth of *Verticicladiella wageneri* through soil and infection of *Pinus ponderosa* as related to selected soil properties. M.S. thesis. University of California, Berkeley.
8. Hicks, B. R., Cobb, F. W., Jr., and Gersper, P. L. 1980. Isolation of *Ceratocystis wageneri* from forest soil with a selective medium. Phytopathology 70:880-883.
9. Landis, T. D., and Helburg, L. B. 1976. Black

stain root disease of pinyon pine in Colorado. Plant Dis. Rep. 60:713-717.

10. McKeague, J. A. 1965. Relationship of water table and Eh to properties of three clay soils in the Ottawa valley. Can. J. Soil Sci. 45:49-62.
11. McKenzie, L. J., and Erickson, A. E. 1954. The use of redox potentials in studies of soil genesis. Soil Sci. Soc. Am. Proc. 18:481-485.
12. Smith, R. S., Jr. 1967. Verticicladiella root disease of pines. Phytopathology 57:935-938.
13. Wagener, W. W., and Mielke, J. L. 1961. A staining fungus root disease of ponderosa, Jeffrey and pinon pines. Plant Dis. Rep. 45:831-835.
14. Wilks, D. S., Gersper, P. L., and Cobb, F. W., Jr. 1983. Relation of soil redox potential to infection of ponderosa pine by *Ceratocystis wageneri*. Phytopathology 73:1120-1125.