Influence of Temperature and Wetness Duration on Infection of Immature Apple and Pear Fruit by *Phytophthora cactorum*

Gary G. Grove and Robin J. Boal

Assistant plant pathologist and research technologist, Washington State University Tree Fruit Research and Extension Center, 1100 N. Western Avenue, Wenatchee, WA 98801.

Plant Pathology New Series Paper 0093. Project 0795, Washington State University Agricultural Research Center, Pullman 99164. We gratefully acknowledge the funding of this project by the Washington State Tree Fruit Research Commission and The Agricultural Research Center, Washington State University.

We would like to express appreciation to Dr. R. A. Spotts for review of the manuscript and to Dr. Richard Alldredge for advice on statistical analyses. The excellent technical assistance of M. Drake is gratefully appreciated.

Accepted for publication 30 July 1991 (submitted for electronic processing).

ABSTRACT

Grove, G. G., and Boal, R. J. 1991. Influence of temperature and wetness duration on infection of immature apple and pear fruit by *Phytophthora cactorum*. Phytopathology 81:1465-1471.

Phytophthora cactorum was recovered from irrigation water from late June to September 1989 and early June to September 1990. Apple (cv. Golden Delicious) and pear (cv. Bartlett) fruit inoculated with a zoospore suspension (10,000/ml) of *P. cactorum* were used to determine the effect of wetness duration and temperature on disease severity and incidence. In controlled environment studies, incidence and severity increased with increased wetness duration (1–12 h) at temperatures between 10 and 30 C on pears and 7 and 30 C on apples. On pears, the loss threshold of one lesion per fruit required wetness durations of 5, 4, and 3 h at 15, 20, and 25–30 C, respectively. At 20–30 C, a ≥3-h wetness duration resulted in 100% infection on pears. On apples, the loss threshold of one lesion per fruit required 11-, 7-, 6-, 5-, and 3-h wetness durations at 10, 15, 20, 25, and 30 C, respectively. Wetness durations of 6–7 and 3–4 h were required for 100% infection at 15–20 and 25–30 C, respectively. Multiple regression equations using temperature and wetness duration as independent variables adequately described disease incidence and severity on both hosts. In orchard studies on pears, infection increased with increased wetness duration (1–20 h) and temperatures up to about 28 C, and then declined slightly at longer wetness durations at 29–31 C. Infection of apples in the field increased with increased wetness duration (1–20 h) up to 20–25 C and then declined. Multiple regression equations using temperature and wetness duration, and temperature, wetness duration, and increasing day of year as independent variables adequately described disease incidence and severity on pears and apples, respectively. The predicted loss threshold of one lesion per fruit on orchard-inoculated pears required wetness durations of 1, 3, 6, and 6–11 h at 25, 20, 15, and 10 C, respectively; on apples the loss threshold ranged from 6 h at 10 and 27.5 C to 3.5 h at 20 C. Susceptibility of pear fruit remained nearly constant from about 60 days after petal fall until harvest; apple fruit susceptibility increased as harvest approached. Results indicate that fruit infection can occur throughout the summer fruit development period and management of the disease may be improved by reducing the duration of overtree irrigation and applying water during cooler periods (i.e., at night).

Additional keywords: irrigation management, *Malus domestica*, *Pyrus communis*, and quantitative epidemiology.

Sprinkler rot of immature pear (*Pyrus communis* L.) fruit, caused by *Phytophthora cactorum* (Lebert & Cohn) Schröt. (3,7,16), has been considered a disease of minor economic importance in the Pacific Northwest (3,7), but has recently resulted in significant losses in the Wenatchee River Valley. Sprinkler rot epidemics occurred on pears in the area in 1989 and 1990. During those years many growers reported fruit losses of 25–30% due to infection by *P. cactorum*. Because the fungus is present in irrigation water (8,10), the disease is most common in orchards with overtree irrigation (3,7), but can also occur with undertree irrigation when the sprinkler nozzle angles are sufficient to wet fruit on the lower tree branches. Sprinkler rot can also occur when contaminated water is used for chemical sprays (3). There are no fungicides currently registered in Washington for the control of this disease. Control is sometimes attained by adding 1 ppm copper to irrigation water, but this can be problematic due to the deleterious effect of copper on the fruit appearance of certain pear cultivars. The only other control recommendation is to keep irrigation water from direct contact with fruit, a practice that is obviously unfeasible in blocks with overtree irrigation. The disease occurs less commonly on apples (*Malus domestica* Borkh.) and is most severe on cv. Golden Delicious (3).

Current irrigation practices in some Washington orchards are apparently promoting disease outbreaks by delivering the pathogen to fruit, and providing wetting conditions that are necessary for fruit penetration. The purposes of this study were to determine the seasonal occurrence of *P. cactorum* in an irrigation canal used to irrigate orchards with histories of sprinkler rot and to determine the effect of temperature and wetness duration on infection of immature apple and pear fruit, and thus ascertain if disease control is attainable by reducing the duration of overtree irrigation.

MATERIALS AND METHODS

Occurrence of *P. cactorum* in irrigation water. An irrigation canal located in the Wenatchee River Valley was used for the study. The canal served as a water source for orchards with histories of sprinkler rot and was surveyed weekly from late June to late August 1989 and early May to late September 1990 for the presence of the *P. cactorum*. Immature pear fruit (cv. Bartlett) were used as baits. Several (3–4) fruits were placed in a No. 4 plastic pot with the pot bottom removed. The pot was enclosed in a nylon mesh, fastened to a rope, and suspended just beneath the water surface for 3 days. Fruits were then retrieved, placed in moist incubation jars, and incubated an additional 3 days at 20 C in continuous light. Tissue was removed from lesion edges and placed on pentachloronitrobenzene-benomyl-neomycin sulfate-chloramphenicol (PBNC) (19) medium and incubated 4 days at 20 C in continuous light. *Phytophthora* spp. were transferred to lima bean agar and identified according to the keys of Newhook et al (14) and Waterhouse (20–22). The pathogenicity of each isolate was determined by inoculating immature pear fruit with mycelia removed from the edges of 7-day-old cultures with a cork borer. A small piece of fruit epidermis was removed with a cork borer, and the fruit was inoculated by placing a mycelial

plug in the wound, which was then covered with petroleum jelly. Inoculated fruit were placed on a moist towel in glass dressing jars and incubated 7 days at 20–22 C in a 16-h photoperiod. After incubation, tissue segments were removed from lesion edges, placed on PBNC medium, and incubated 4 days at 20 C in continuous light. Isolate pathogenicity was confirmed by the presence of lesions and the subsequent reisolation of *P. cactorum* in pure culture.

Inoculum production and inoculation technique. In 1989, controlled environment and field inoculations were done with cultures of *P. cactorum* freshly isolated from infected pear fruit (cv. Bartlett) on PBNC medium. For sporangial production, mycelial plugs 5 mm in diameter were taken from the edges of 7-day-old cultures with a cork borer and transferred to lima bean broth (15,19). Cultures were incubated 5 days at 22 C in continuous light at 2.8 W/m^2. About 1 h before inoculation, zoospore production was induced by pouring off the broth followed by the addition of 5 cm^3 of ice to each culture. Zoospore suspensions were adjusted to 10,000/ml in sterile distilled water after counting with a hemacytometer. Inoculation was accomplished by applying 5 ml of inoculum to each fruit with a small hand-sprayer. Inoculum was applied as uniformly as possible over the entire surface of the fruit. During the winter of 1989–1990, the sporulation capacity of the isolate deteriorated. Therefore, the method for obtaining zoospore inoculum was modified for 1990 field studies on apples and pears, and for controlled environment studies on apples. Mycelial plugs were taken from the edges of 7-day-old cultures as previously described, transferred to lima bean broth, and incubated 10 days at 20–22 C with a 16-h photoperiod. About 12 h before inoculation, the mycelial mats from three cultures were placed into a petri plate containing 25 ml of Chen-Zentmyer salt solution (2,15) and incubated 10 h at 20 C. The salt solution was poured off, and the culture was flooded with cold deionized water. After about 1 h, zoospore concentrations were adjusted to 10,000/ml as described previously. Inoculations were accomplished as described above.

Controlled environment studies. Immature (green) pear (cv. Bartlett) and apple (cv. Golden Delicious) fruits were removed from trees, rinsed twice with deionized water, and inoculated as described above. Studies on pears were conducted with fruit harvested 8–10 wk after petal fall. Apple studies were conducted on fruit harvested 5 wk before harvest. Immediately after inoculation, fruit were individually wrapped in aluminum foil to maintain continuous wetness and placed in incubators set at six constant temperatures between 6 and 30 C. To avoid possible effects of fruit maturation on infection, the completion of each trial of the experiment was expedited by the use of three or two incubators on pears and apples, respectively. The foil wrappings of 10 fruit were removed at periodic intervals (10 fruit per wetness duration) ranging from 1- to 8- or 1- to 11-h periods for pears and apples, respectively. Inoculations for wetness durations of less than 1 h were accomplished by leaving some inoculated fruit unwrapped. Inoculated fruit were incubated in darkness. Temperature and fruit wetness in incubators were continuously monitored with thermistors (Fenwall Electronics, Ashland, MA) and printed-circuit leaf wetness sensors (Wong Labs, Cincinnati, OH) connected to a CR-21 Datalogger (Campbell Scientific, Logan, UT). To determine fruit drying time, leaf wetness sensors were gently misted with sterile distilled water when each group of 10 fruit were unwrapped. After a 24-h drying period (in the same incubator where inoculated fruit were subjected to various wetting treatments), fruit were removed from incubators, sealed in clear polyethylene bags, and incubated at 20–22 C with a 16-h photoperiod for 3 or 6 days for pears and apples, respectively. At the conclusion of the incubation period, disease severity was assessed by visually determining the number of lesions per fruit. Disease incidence was evaluated as the proportion of fruit inoculated that became infected. Isolations were made from representative lesions, as previously described, to verify the presence of *P. cactorum*. The experiment was designed as a two-factor experiment with 10 observations per treatment. The order of temperatures tested, as well as the incubator used for each temperature, was assigned at random. The experiment was conducted three times on pears and twice on apples.

Field studies. Immature (green) pear fruits (cv. Bartlett) and apple fruits (cv. Golden Delicious) ranging in maturity from green to ripe fruits were used in field studies. To study the ontogenetic susceptibility of both hosts to infection, inoculations were done from about 8 wk after petal fall until harvest for pears, and during the 5 wk preceding harvest for apples. For pears, 2- and 4-ha blocks of 45-year-old trees were used in 1989 and 1990, respectively. A 2-ha block of 50-year-old trees was used for studies on apples. All orchards were located at the Tree Fruit Research and Extension Center, Wenatchee, WA. Fruit (20–40) were tagged and inoculated as previously described and immediately wrapped with aluminum foil. Inoculations for wetness durations of less than 1 h were accomplished by leaving some inoculated fruit unwrapped. Some groups of inoculated fruit were incubated at ambient temperatures on uncovered limbs, while other groups were incubated on limbs at temperatures beneath ambient, which were provided by covering limbs with chambers connected to a portable cooling device similar in principle to that previously described (11). Cooled air was provided by a refrigeration unit mounted on a portable frame. The refrigeration unit consisted of a basic refrigeration package with two evaporator coils. An evaporator pressure regulator allowed the temperature of each coil to be set independently. Each coil was connected to two remote incubation chambers with 15.2-cm-diameter rubber tubing about 15 m long. Cooled air was delivered to incubation chambers by high speed fans. The use of two coils allowed the use of two incubation chambers set at one temperature and the second two set at a different temperature. When using the cooling device, each of four limbs bearing fruit to be inoculated were covered with an incubation chamber and cooled for about 1 h before inoculation. Five fruit were unwrapped at periodic intervals ranging from 0–20 h (1989 studies) or 0–10 h (1990 studies). Temperatures and wetness durations after all inoculations were continuously monitored with thermistors and leaf wetness sensors connected to CR-21X Dataloggers. To estimate drying time, leaf wetness sensors were immediately misted with sterile distilled water after unwrapping fruit. After unwrapping, fruit were incubated in the chambers or (for inoculations at ambient temperatures) on uncovered limbs for an additional 24 h. Fruit were then removed from trees and incubated as previously described. At the conclusion of the incubation period, disease severity and incidence were assessed as described above. Isolations were made from representative lesions as described to verify the presence of *P. cactorum*.

Statistical analyses. Estimated drying time for each temperature-wetness inoculation varied from 17 to 67 min with a mean of 31 min. The specific drying time for each inoculation was added to the time that fruit were wrapped to give a total duration of wetness. The effects of wetness duration (W, hours) and the mean temperature (T, C) during the wetness period on disease incidence (I) and severity (S) were evaluated using the regression procedure of Minitab Data Analysis Software (Minitab Inc., State College, PA). All possible combinations of T, W, TW, T^2, T^3, T^2W, T^3W, W^2, TW^2, T^2W^2, and T^3W^2 were regressed on the $\log_{10}$ [(number of lesions per fruit) + 1] for the severity data (S) and on arcsin $\sqrt{I}$ (in which I = the proportion of fruit inoculated that became infected) for the incidence data. To minimize the effects of multicollinearity, temperature and wetness duration were expressed as deviations from their respective means (13) for the analyses. Regression equations were evaluated according to significance of regression coefficients, coefficients of determination (R^2) and R^2 adjusted for degrees of freedom (R_a^2), and pattern and distribution of residuals (6,12,13,23). The analysis of data from controlled environment experiments was done on each trial separately, and then on the pooled data; F tests were conducted to determine if the regression results from each trial were significantly different (6,12,23) and to determine if pooling of the data was warranted. Field data were analyzed as described above, but the data from each year were kept separate due to the changes in inoculum production and different orchard

locations; the day of the year (D) when inoculation was done and fruit surface area (SA, mm^2) were included as independent variables (but not together in the same equation) in addition to the variables listed above.

RESULTS

Occurrence of *P. cactorum* in irrigation water. *P. cactorum* was recovered from irrigation water each week throughout the late June to late August 1989 survey period. In 1990, the fungus was detected first in early June and then again each week from early July to mid-September. Baitings during May and late September were unsuccessful. All isolates were pathogenic to pear fruit in subsequent inoculations.

Controlled environment studies. *Pear disease severity.* Lesions on both hosts first appeared as circular brown lesions 2.5–3.0 cm in diameter. In general, there was an increase in disease severity with increased wetness duration and temperature (Fig. 1A). Infection did not occur at 6 C. Infection required wetness durations of 5, 4, 2, 1, and 2 h at 10, 15, 20, 25, and 30 C, respectively. Between 15 and 30 C, severity increased with increased wetness duration (e.g., at 15 C, values increased from 0.2 lesions per fruit at 4 h to 1.7, 3.9, and 5.3 lesions per fruit at 5, 7, and 9 h, respectively). The loss threshold, described by Arauz and Sutton (1) as the point where one lesion per fruit (and thus fruit loss) occurs, required 5, 4, and 3 h of wetness at 15, 20, and 25–30 C, respectively.

The F test indicated ($P > 0.05$) that the regression results of the three trials were not significantly different. The data were therefore pooled and the equation from the combined data:

$$S = 0.54 + 0.039T + 0.13W + 0.0088TW \quad (1)$$

described disease severity with a coefficient of determination (R^2) of 0.81. The coefficient of determination adjusted for degrees of freedom (R_a^2) and standard error about the regression curve (s) equaled 0.80 and 0.21, respectively. Regression coefficients were significant at $P < 0.05$. Residuals had a random pattern and were normally distributed.

Pear disease incidence. In general, there was an increase in disease incidence with increased temperature and wetness duration

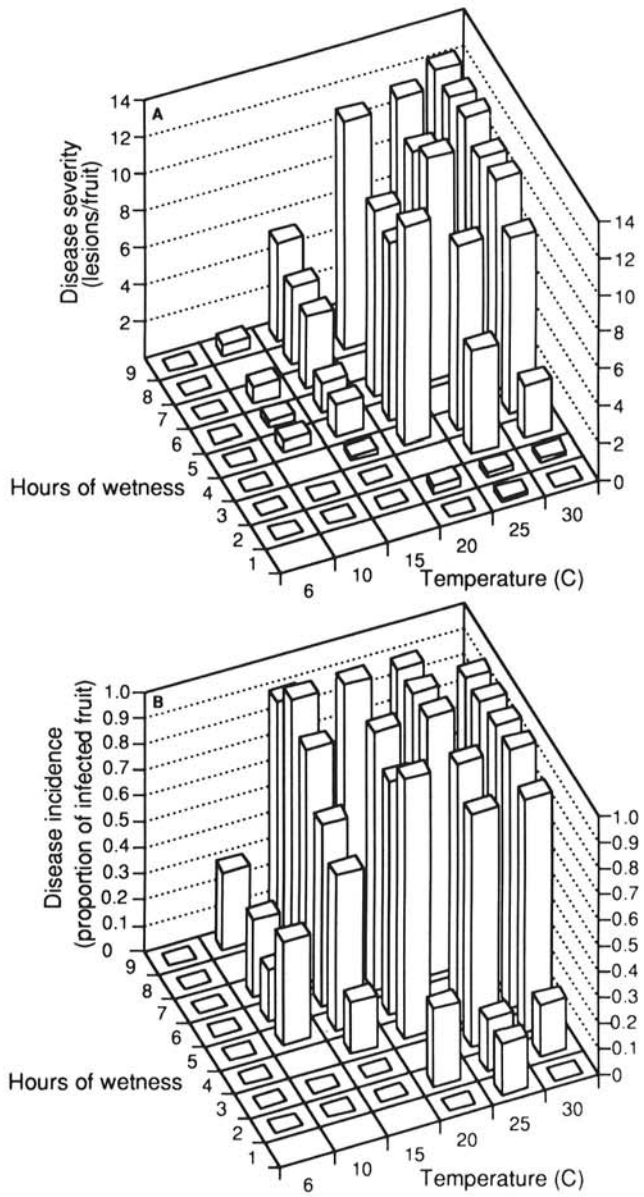

Fig. 1. A, Average disease severity and **B,** incidence on immature pear fruit inoculated with *Phytophthora cactorum* and incubated over a range of wetness durations at six constant temperatures. Temperature and wetness duration are rounded off to the nearest degree centigrade and hour, respectively. Because an F test indicated that the results of the three controlled environment trials were not significantly different, only values from trial 1 are presented.

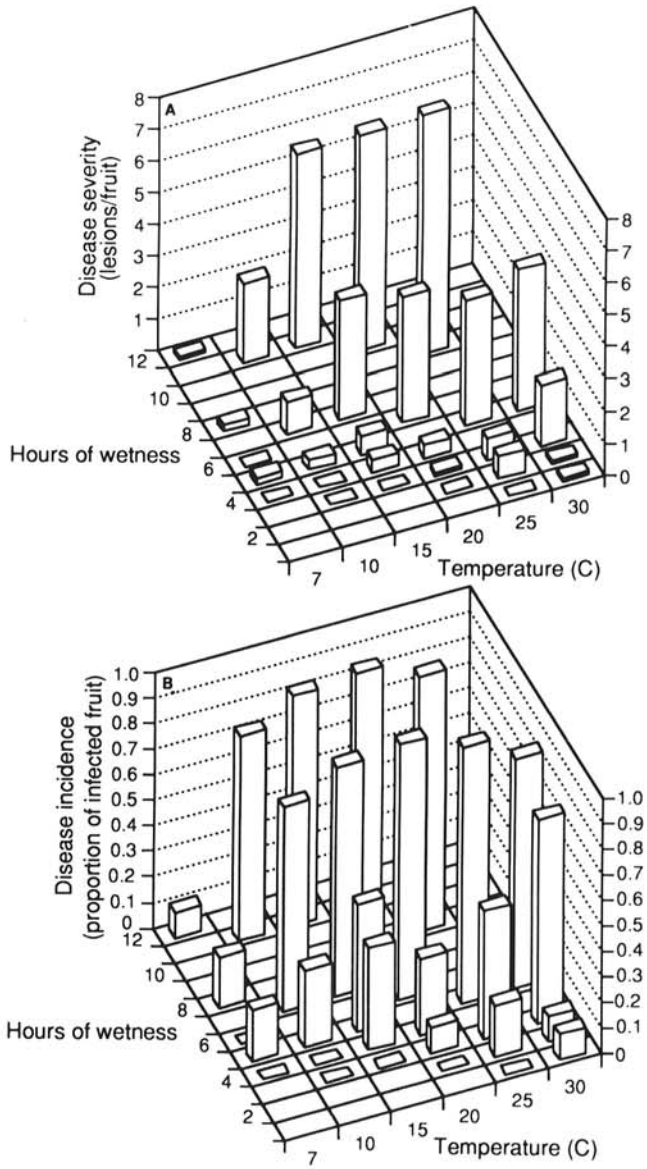

Fig. 2. A, Average disease severity and **B,** incidence on immature apple fruit inoculated with *Phytophthora cactorum* and incubated over a range of wetness durations at six constant temperatures. Temperature and wetness duration are rounded off to the nearest degree centigrade and hour, respectively. Because an F test indicated that the results of the trials were not significantly different, only values from trial 1 are presented.

(Fig. 1B). The 40% maximum infection level observed at 10 C required 6 h. Incidence levels of 100% required 8- and 5-h wetness durations at 15 and 20–30 C, respectively. At 15–30 C, incidence increased with increased wetness duration (e.g., at 25 C incidence values ranged from 20% at 2 h to 90 and 100% at 3 and 4–8 h, respectively).

The F test indicated ($P > 0.05$) the regression results from the three trials were not significantly different. The data were therefore pooled and the equation:

$$I = 1.0 + 0.048T + 0.17W - 0.00044T^2W^2 \quad (2)$$

described disease incidence with R^2, R_a^2, and s values equal to 0.83, 0.82, and 0.28, respectively. Regression coefficients were significant at $P < 0.05$. Residuals had a random pattern and were normally distributed.

Apple disease severity. Results were similar to those observed on pear. At 7 C, infection required 5 h of wetness, resulting in 0.2 lesions per fruit (Fig. 2A). Infection required wetness durations of 5, 4, 3, 2, and 1 h at 10, 15, 20, 25, and 30 C, respectively. At each temperature between 10 and 30 C, severity generally increased with increased wetness duration (e.g., at 25 C severity ranged from 0 at 1 h to 0.7, 3.9, and 7.5 lesions per fruit at 2, 5, and 9 h, respectively). The loss threshold of one lesion per fruit required 11-, 7-, 6-, 5-, and 3-h wetness durations at 10, 15, 20, 25, and 30 C, respectively.

The F test indicated ($P > 0.05$) the regression results from the two trials were not significantly different. The data were therefore pooled and the equation:

$$S = 0.37 + 0.025T + 0.104W - 0.00013T^2W^2 \quad (3)$$

described disease severity with R^2, R_a^2, and s values equal to 0.85, 0.85, and 0.13, respectively. Regression coefficients were significant at $P < 0.05$. Residuals had a random pattern and were normally distributed.

Apple disease incidence. Results were similar to those observed on pear. In general, disease incidence increased with increased wetness duration at all temperatures tested (Fig. 2B). At 7 C, a 5-h wetness duration was required to reach a maximum of 20% infected fruit. At 10–30 C, incidence in general increased with increasing wetness duration (e.g., at 25 C, 0, 20, 50, and 100% infection occurred at wetness duration of 1, 2, 3, and 5 h, respectively). At 10 and 15 C, the 80 and 90% maxima required 7 h. Infection levels of 100% required 6 and 5 h at 20 and 25 C, respectively.

The F test indicated ($P > 0.05$) that the regression results from the two trials were not significantly different. The data were therefore pooled and the equation:

$$I = 0.85 + 0.05T + 0.18W - 0.00024T^2W^2 \quad (4)$$

described disease incidence with R^2, R_a^2, and s values equal to 0.81, 0.80, and 0.26, respectively. Regression coefficients were significant at $P < 0.05$. Residuals had a random pattern and were normally distributed.

Field studies. *Pear disease severity.* In 1989, infection occurred between 9 and 29 C. As temperature decreased, infection required progressively longer wetness durations (e.g., the loss threshold of one lesion per fruit required 2 h at 20–22 C and ≥12 h at 9 C). Infection required about 5-h wetness durations at temperatures <20 C. At 0- to 2-h wetness durations, infection occurred only at temperatures >20 C. Severity values ranged from 0 at 14 C to 2.8 and 7.8 at 20 and 25 C, respectively. Infection occurred over a broader range of temperatures at 5-h wetness durations; values ranged from 0.6 at 11 C to 1.4 and 16.2 at 11, 18, and 27 C, respectively. Results in 1990 were similar to those obtained in 1989. Infection occurred at temperatures between 9 and 30 C and at wetness durations as short as 1 h. Infection occurred at all temperatures tested at wetness durations >7 h. Severity values at wetness durations of 1–2 h ranged from 0 at 30 C to 2.8 at 20 C. At 3- to 4-h wetness durations, severity values ranged from 0 at 12 C to 15.2 and 23.2 at 25 and 27 C, respectively. At wetness durations of 5–6 h, severity increased from 0 at 12 C to 21.6 at 24 C.

Regression analysis indicated that neither day of year (D) nor fruit surface area (SA) had a significant effect ($P > 0.05$) on disease severity during either year of the field studies. The equations:

$$S(1989) = 0.55 + 0.058T + 0.10W - 0.00085T^2W - 0.00058\ TW^2 \quad (5)$$

$$S(1990) = 0.54 + 0.065T + 0.18W - 0.00087T^2W - 0.0054TW^2 \quad (6)$$

described disease severity in 1989 and 1990 with coefficients of determination of 0.75 ($R_a^2 = 0.75$) and 0.77 ($R_a^2 = 0.76$), and s values of 0.24 and 0.25, respectively. All regression coefficients were significant at $P < 0.001$. Residuals had a random pattern and were normally distributed. The F test indicated ($P < 0.05$) that the regression results from the two years of trials were significantly different. The respective equations were used to generate the response surfaces that are presented in Figure 3A and B.

Pear disease incidence. In 1989, incidence at 1- to 2-h wetness durations ranged from 0% at 14 C to 100% at 20 and 25 C, respectively. Infection occurred over a broader range of temperatures at 5-h wetness durations; values ranged from 60%

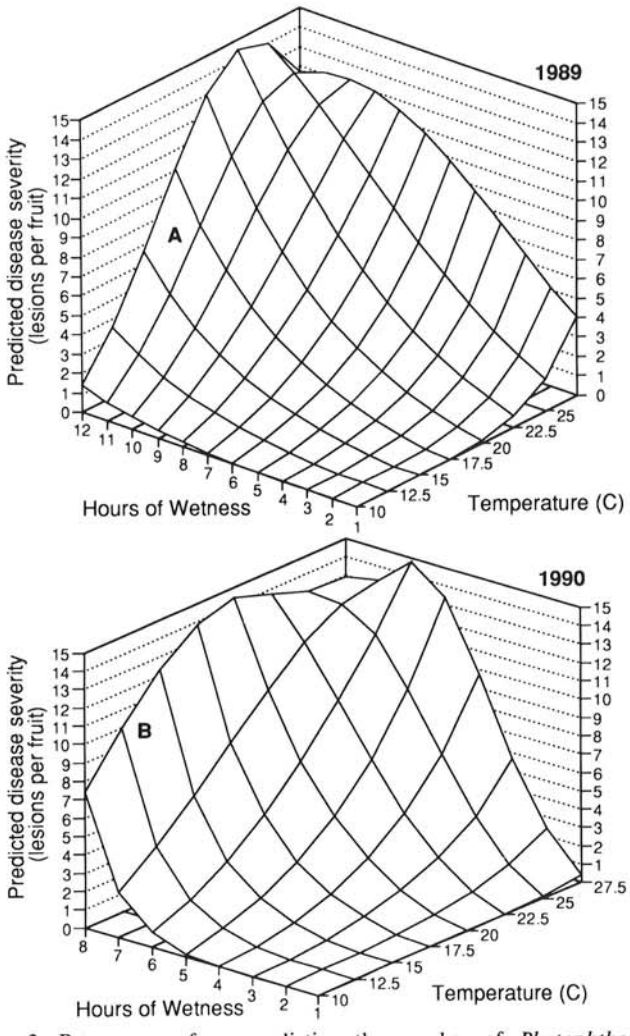

Fig. 3. Response surfaces predicting the number of *Phytophthora cactorum* lesions on immature pear fruit at different wetness durations and temperatures. Surface was generated using equations 5 (1989) and 6 (1990). Predicted lesion numbers were obtained by backtransforming values generated by the equations.

at 11 C to 80% and 100% at 18 and 27 C. At wetness durations >12 h, incidence was >60% at all temperatures >9 C. Results in 1990 were similar to those obtained in 1989. Incidence at 1- to 2-h wetness durations ranged from 0 to 100%; 20% infection occurred at 10 C, while values ranged from 0 to 100% at temperatures ≥20 C. At 5-h wetness durations, 0–60% infection occurred between 12 and 15 C; values of 80–100% occurred at temperatures between 16 and 29 C. At wetness durations of 7–10 h, incidence was 100% between 9 and 27 C.

Regression analysis indicated that neither D nor SA had a significant effect ($P > 0.05$) on disease incidence during either year of the field studies. The equations:

$$I(1989) = 0.93 + 0.10T + 0.12W - 0.00031T^3 - 0.000091T^2W^2 \quad (7)$$

$$I(1990) = 0.92 + 0.11T + 0.19W - 0.00078T^3 - 0.00026T^2W^2 \quad (8)$$

described disease incidence in 1989 and 1990 with coefficients of determination of 0.68 ($R_a^2 = 0.68$) and 0.64 ($R_a^2 = 0.63$), and s values of 0.39 and 0.41, respectively. All regression coefficients were significant at $P < 0.05$. Residuals had a random pattern and were normally distributed. The F test indicated ($P < 0.05$) the regression results from the two years were significantly different. The respective equations were used to generate the response surfaces that are presented in Figure 4A and B.

Apple disease severity. In general disease severity (Fig. 5A,B) increased with increasing wetness duration and increasing day of year. Infection did not occur at wetness durations shorter than 1 h regardless of temperature. At 10–12 C, severity ranged from 0 at 4 h to 0.2 at 6 h. At 19–22 C, severity ranged from 0 at 2 h to 9.4 at 6 h. At 24–26 C, severity ranged from 0 at 1 h to 4.2, 5.2, and 12.6 at 6, 8, and 9 h, respectively.

Increasing D (but not SA) had a significant ($P < 0.001$) effect on disease incidence and severity; regressing severity on D alone resulted in a coefficient of determination of 0.11. The equation:

$$S = -3.64 + 0.11W - 0.00497T^2 + 0.018D - 0.0098W^2 - 0.00033T^2W^2 \quad (9)$$

(in which D = day of year) described apple disease severity with R^2, R_a^2, and s equal to 0.75, 0.73, and 0.19, respectively. All regression coefficients were significant at $P < 0.05$. Residuals had a random pattern and were normally distributed.

Apple disease incidence. At 28–30 C, 100% infection required at least 3 h of wetness. Wetness durations of 3–4 h resulted in disease incidence that ranged from 0 at 11 C to 100% at 26 C. At wetness durations of ≥6 h, incidence was 0 at 33 C and 14 C; incidence was 100% at 19, 21, 23, and 25 C.

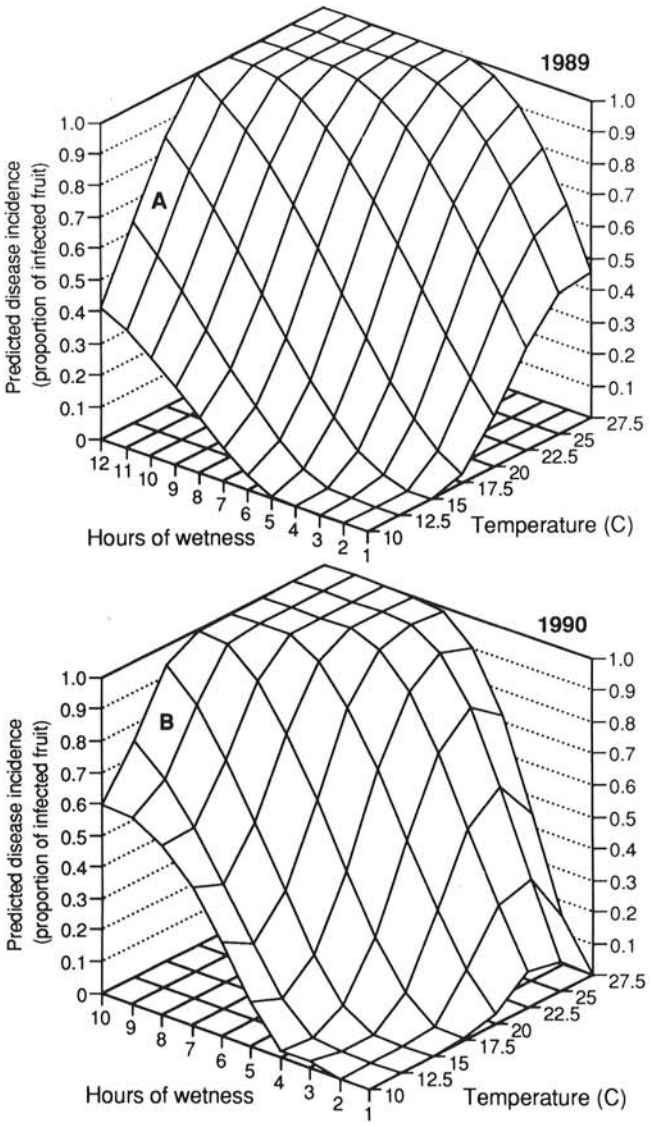

Fig. 4. Response surfaces predicting the proportion of infected pear fruit at different temperatures and wetness durations. Surface was generated using equations 7 (1989) and 8 (1990). The proportions of infected fruit were obtained by backtransforming values generated by the equations.

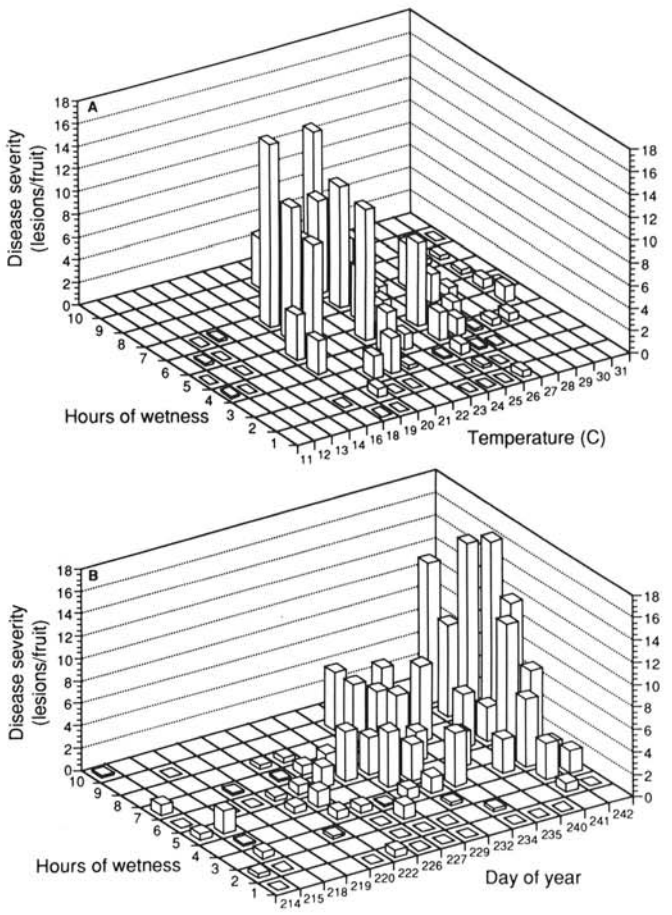

Fig. 5. A, Effect of wetness duration and temperature and **B,** wetness duration and increasing day of year on disease severity (expressed as the number of lesions per fruit) on immature apple fruit inoculated with a zoospore suspension of *Phytophthora cactorum* in the field. Temperature and wetness duration are rounded off to the nearest hour and degree centigrade, respectively. **A,** in cases when two or more inoculations were made at a given temperature and wetness combination, the mean of those severity values is presented. **B,** in cases when two or more inoculations were made at a specific wetness duration on the same day of year, the mean of those values is presented. Inoculations in B were performed over a temperature range of 11–31 C.

Increasing D (but not SA) had a significant ($P < 0.001$) effect on disease incidence; regressing incidence on D alone resulted in a coefficient of determination of 0.21.

The equation:

$$I = -4.6 + 0.47W + 0.018D - 0.032W^2 - 0.00027T^2W^2 \quad (10)$$

described disease incidence with R^2, R_a^2, and s equal to 0.69, 0.67, and 0.37, respectively. All regression coefficients were significant at $P < 0.05$. Residuals had a random pattern and were normally distributed.

DISCUSSION

Temperature and wetness duration are significant environmental factors influencing the infection of immature pear and apple fruit by *P. cactorum*. In controlled environment studies, apple and pear disease incidence and severity increased with increasing temperature up to 30 C. The wetness durations required for apple infection were slightly longer than those required for pear (e.g., at 25 C, the loss threshold of one lesion per fruit required 5 h on apple and 3 h on pear). The differences between hosts could possibly be due to differences in epidermal thickness or the presence of epidermal cracks. In field studies, a slight decrease in pear disease severity was observed at longer wetness durations above 28 C. The difference in temperature response between controlled environment and field studies on pears could be attributable to physiological differences between attached and detached fruit, or the change in temperature during the wetness durations in the field. In controlled environment studies, temperature was relatively constant. Conversely, in field studies temperatures during the wetness durations could rise or fall to a measurable degree depending on the time of day the inoculation was performed and the duration of wetness. Inoculations made under ambient conditions were generally made about 1 h after sunrise; temperatures could easily rise 5–15 C over an 8-h period during a typical summer day in eastern Washington.

Regression equations using temperature and wetness duration as independent variables accounted for more than 80% of the variation in pear disease severity and incidence in controlled environment studies, respectively, and >70% and >60% of the variation in field disease severity and incidence, respectively. The differences between 1989 and 1990 can best be seen in the severity response surfaces generated for each years' field data. The 1990 equation predicted higher disease severity at lower temperatures (i.e., the loss threshold of one lesion per fruit at 10 C was predicted at 11 and 6.5 h in 1989 and 1990, respectively). Conversely, the 1989 equation predicted four lesions per fruit at a 1-h wetness duration at 27.5 C; a severity value of about 0.5 was predicted at this point in 1990. The loss threshold at the temperature mid-range was similar (e.g., at 20 C the loss threshold was predicted at 3.5 and 3 h for 1989 and 1990, respectively). The differences between years could be attributable to several factors: the effect of continuous subculture on the isolate of *P. cactorum* used in the experiments, the different orchard locations, and the effects of the different methods used for inoculum production. Another possible factor that could account for the difference between years and account for some of the unexplained variability in all field studies could again be the change in temperature over the wetness durations.

Regardless of the differences, the predicted loss threshold at the temperatures studied occurred at wetness durations shorter than those provided by standard irrigation sets, which in Washington are seldom shorter than 12 h and average 16–24 h. During the July and August fruit development period, the average daily maximum and minimum temperatures range from 14 to 33 C and 14 to 31 C, respectively. These temperatures are well within the range of temperatures over which infection by *P. cactorum* can occur. Elimination of sprinkler rot by merely reducing the length of irrigation sets is probably not economically feasible, because shortening sets to the wetness durations where infection does not occur would probably fail to provide trees with adequate moisture. However, making the sets as short as possible (while still providing trees with adequate moisture) and applying water during cooler periods (e.g., night) may help reduce losses due to infection by *P. cactorum*. Alternatively, producers could switch from overtree to undertree or trickle irrigation. Where sprinkler rot is a problem in orchards with undertree sprinklers, the water angle could be lowered to minimize fruit contact with water.

As with *P. cactorum* on strawberry fruit (6) and *P. palmivora* on papaya (9), the wetness durations required for infection of apple and pear by *P. cactorum* were exceedingly short (i.e., 1–2 h at temperatures >20 C). Infection of other hosts by *Phytophthora* species has been reported to require wetness durations longer than those required for infection of apple, pear, and strawberry (4,17,18). Interactions between temperature and wetness duration that have been reported for infection of strawberry by *P. cactorum*, and of various other hosts by different *Phytophthora* species (4,5,17,18), although not identical, were also apparent in this study. With strawberry, progressively longer wetness durations were required for infection as temperatures increased or decreased from the 21 C optimum temperature (6). With apple and pear, the wetness durations required for infection decreased with increasing temperature up to 30 C.

Neither increasing day of year nor fruit surface area had a significant effect on pear disease severity. Phenologically, the susceptibility of pear fruit to infection by *P. cactorum* was demonstrated from about 60 days after petal fall until harvest and appeared to remain constant over that period. Apples were susceptible throughout the 30-day experimental period preceding harvest; susceptibility increased as harvest approached. The reason for this is unclear, but could be attributable to a response of the fungus to the fruit ripening process, or a response to physical changes occurring on the fruit surface (e.g., epidermal cracking).

The presence of the fungus in irrigation water during the summer and the infection of immature fruit demonstrated in this study indicate that sprinkler rot outbreaks are possible over a large portion of the summer fruit development period and possibly earlier. Although we failed to detect the fungus in irrigation water during the 1-mo period following petal fall on pear, *P. cactorum* was baited from the same canal as early as pear petal fall (mid-April) in previous studies (8).

The reason for the sudden increase in sprinkler rot incidence and severity in the Wenatchee River Valley is unclear. It is possible that the increase may be attributable to recent grower reluctance (due to toxicological concerns) to use ethylene bis-dithiocarbamate fungicides for the control of pear psylla (*Psylla pyricola* Foerster). Until the 1989 season, many Washington growers applied mancozeb during late spring and summer and may have been protecting developing fruit from infection by *P. cactorum*. Sprinkler rot severity and incidence increased noticeably in 1989 (G. G. Grove, *unpublished*). Inoculations of mancozeb-treated fruit with *P. cactorum* zoospores under disease-conducive conditions in the orchard have been unsuccessful (G. G. Grove, *unpublished*).

Research in progress on the effect of interrupted wetness durations on disease severity and on various irrigation-water treatments will hopefully aid in the control of this increasingly problematic disease.

LITERATURE CITED

1. Arauz, L. F., and Sutton, T. B. 1989. Temperature and wetness duration requirements for apple infection by *Botryosphaeria obtusa*. Phytopathology 77:440-444.
2. Chen, S. W., and Zentmyer, G. A. 1970. Production of sporangia by *Phytophthora cinnamomi* in axenic culture. Mycologia 63:397-402.
3. Covey, R. P., and Harris, D. C. 1990. *Phytophthora* fruit rots of pear and apple. Pages 30-31 in: A Compendium of Apple and Pear Diseases. The American Phytopathological Society, St. Paul, MN. 100 pp.
4. Crosier, W. 1934. Studies in the biology of *Phytophthora infestans* (Mont.) De Bary. N.Y. Agric. Exp. Stn. Mem. 155. 40 pp.
5. Gerlach, W. W. P., Hoitink, H. A. J., and Schmitthenner, A. F.

1976. *Phytophthora citropthora* on *Pieris japonica*: Infection, sporulation, dissemination. Phytopathology 66:302-308.
6. Grove, G. G., Madden, L. V., Ellis, M. A., and Schmitthenner, A. F. 1985. Influence of temperature and wetness duration on infection of immature strawberry fruit by *Phytophthora cactorum*. Phytopathology 75:165-169.
7. Harley, C. P., and Reeves, E. L. 1930. Observations of apple and pear diseases under overtree irrigation. Wash. State Hortic. Assoc. Proc. 26:48-49.
8. Hassan, N. M. M. B. H. N. 1987. Distribution and survival of *Phytophthora cactorum* in irrigated orchard soil in Washington. Ph.D. thesis, Washington State University. 102 pp.
9. Hunter, J. E., and Kunimoto, R. K. 1974. Dispersal of *Phytophthora palmivora* by wind-blown rain. Phytopathology 64:202-206.
10. McIntosh, D. L. 1966. The occurrence of *Phytophthora* spp. in irrigation water in British Columbia. Can. J. Bot. 44:1591-1596.
11. Mellenthin, W. M., and Bonney, D. 1972. A portable limb enclosure for temperature modification of tree fruits. HortScience 7:134-136.
12. Neter, J., and Wasserman, W. 1974. Applied Linear Statistical Models. 1st ed. Richard D. Irwin, Inc., Homewood, IL. 842 pp.
13. Neter, J., Wasserman, W., and Kutner, M. H. 1985. Applied Linear Statistical Models. 2nd ed. Richard D. Irwin, Inc., Homewood, IL. 1127 pp.
14. Newhook, F. J., Waterhouse, G. M., and Stamps, D. J. 1978. No. 143 in: Tabular key to the species of *Phytophthora* de Bary. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 20 pp.
15. Ribeiro, O. K. 1987. A Source Book of the Genus *Phytophthora*. Strauss and Kramer, A. R. Gartner Verlag K.G.l, Fl-9490. 417 pp.
16. Rose, D. H., and Lindegren, C. C. 1925. Phytophthora rot of pears and apples. J. Agric. Res. 30:463-468.
17. Rotem, J., Cohen, Y., and Putter, J. 1971. Relativity of limiting and optimum inoculum loads, wetting durations, and temperatures for infection by *Phytophthora infestans*. Phytopathology 61:275-278.
18. Schlub, R. L. 1983. Epidemiology of *Phytophthora capsici* on bell pepper. J. Agric. Sci. (Camb.) 100:7-11.
19. Schmitthenner, A. F. 1973. Isolation and identification methods for *Phytophthora* and *Pythium*. Woody Ornamental Disease Workshop, University of Missouri, Columbia. 128 pp.
20. Waterhouse, G. M. 1963. No. 92 in: Key to the species of *Phytophthora* de Bary. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 22 pp.
21. Waterhouse, G. M., and Blackwell, E. M. 1954. No. 57 in: Key to the species of *Phytophthora* recorded in the British Isles. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England. 9 pp.
22. Waterhouse, G. M. , and Waterson, J. M. 1966. *Phytophthora cactorum*. No. 111 in: Descriptions of Pathogenic Fungi and Bacteria. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, Surrey, England.
23. Wilson, L. L., Madden, L. V., and Ellis, M. A. 1990. Influence of temperature and wetness duration on infection of immature and mature strawberry fruit by *Colletotrichum acutatum*. Phytopathology 80:111-116.

SUSTAINING ASSOCIATES

AGDIA INCORPORATED, Elkhart, IN

AGRI-DIAGNOSTICS ASSOCIATES, Moorestown, NJ

AGRICULTURE CANADA, Vineland Station, Ontario

AGRIGENETICS COMPANY, Madison, WI

ALF. CHRISTIANSON SEED CO., Mt. Vernon, WA

AMERICAN CYANAMID CO., Agriculture Center, Princeton, NJ

ASGROW SEED COMPANY, San Juan Bautista, CA

ATOCHEM NORTH AMERICA, Philadelphia, PA

BASF CORPORATION, Research Triangle Park, NC

BUCKMAN LABORATORIES, Memphis, TN

BUSCH AGRIC. RESOURCES INC., Ft. Collins, CO

CALGENE, INC., Davis, CA

CHEVRON CHEMICAL CO., San Ramon, CA

CIBA-GEIGY CORPORATION, Agric. Div., Greensboro, NC

CONVIRON, Asheville, NC

DEKALB PLANT GENETICS, DeKalb, IL

DEL MONTE FOODS USA, Walnut Creek, CA

DNA PLANT TECHNOLOGIES INC., Oakland, CA

DOW CHEMICAL USA, Midland, MI

E. I. DUPONT DE NEMOURS & CO., INC., Agric. Chem. Dept., Wilmington, DE

FERRY MORSE SEED CO., San Juan Bautista, CA

FUNK SEEDS INTERNATIONAL, INC., Bloomington, IL

GEORGE J. BALL INC., West Chicago, IL

GREAT LAKES CHEMICAL CORPORATION, West Lafayette, IN

GRIFFIN CORPORATION, Valdosta, GA

GUSTAFSON, INC., Des Moines, IA

HARRIS MORAN SEED CO., Hayward, CA

H. J. HEINZ CO., Bowling Green, OH

HOECHST ROUSSEL AGRI. VET. CO., Somerville, NJ

ICI AMERICAS, INC., Richmond, CA

ILLINOIS CROP IMPROVEMENT ASSOCIATION, Champaign, IL

ILLINOIS FOUNDATION SEEDS, INC., Champaign, IL

ISK BIOTECH CORPORATION, Mentor, OH

ISTITUTO DI FITOVIROLOGIA, Torino, Italy

JANSSEN PHARMACEUTICA, Piscataway, NJ

LANDIS INTERNATIONAL, Valdosta, GA

LOXTON RESEARCH CENTRE, Loxton, Australia

MAHARASHTRA HYBRID SEEDS CO., Bombay, Maharashtra, India

MERCK & CO., INC., Rahway, NJ

MOBAY CORPORATION, Kansas City, MO

MONSANTO CO., St. Louis, MO

NOR-AM CHEMICAL CO., Wilmington, DE

NORTHFIELD LAB—DEPT. OF AGRICULTURE, Adelaide, Australia

PEST PROS, INC., Plainfield, WI

PIONEER HI-BRED INTERNATIONAL INC., Johnston, IA

RHONE-POULENC AG COMPANY, Research Triangle Park, NC

RICERCA, INC., Painesville, OH

RJR NABISCO INC., Winston-Salem, NC

ROGERS N K SEED COMPANY, Woodland, CA

ROGERS N K SEED COMPANY, Nampa, ID

ROHM & HAAS CO., Philadelphia, PA

ROTHAMSTED EXPERIMENT STATION, Herts, England

SAKATA SEED AMERICA, INC., Salinas, CA

SANDOZ CROP PROTECTION CORP., Des Plaines, IL

O. M. SCOTT & SONS, Marysville, OH

TRICAL INC., Hollister, CA

UNIROYAL CHEMICAL COMPANY, Bethany, CT

UNOCAL CHEMICALS, West Sacramento, CA

USDA FOREST SERVICE, Ogden, UT