L. V. Madden and M. A. Ellis
The Ohio State University, Wooster

G. G. Grove
Washington State University, Wenatchee

K. M. Reynolds
U.S. Forest Service, Anchorage, Alaska

L. L. Wilson
The Ohio State University, Wooster

Epidemiology and Control of Leather Rot of Strawberries

Leather rot of strawberry fruit (*Fragaria* × *ananassa* Duchesne) is caused by *Phytophthora cactorum* (Lebert & Cohn) Schröt. The disease was reported first by Rose in 1924 (17) to occur in several southern states, and he also demonstrated that *P. cactorum* is the causal organism. Later, Rose (18) showed that leather rot is positively associated with rainfall. Since that time, leather rot has been found in several states (9,22,23), but little was published on the disease in the United States until the 1980s.

Leather rot was not reported in Ohio until 1981 and was considered to be of little economic importance. In 1981, however, fruit losses of 20–30% were common in many commercial fields, and some growers experienced a 50% crop loss (2). In a survey of Ohio strawberry fields, all cultivars appeared to be equally susceptible, based on symptoms and percentage of infected fruit (2; Ellis, *unpublished*). Similar surveys in other states have not been reported, and the importance of the disease throughout the United States remains unknown. Leather rot is, however, known to be an important strawberry disease in Europe and parts of Asia (8,20). *P. cactorum* commonly causes a crown rot and wilt of strawberry in Europe that has recently been found for the first time in the United States (21). Crown rot is believed to be caused by a distinct pathotype of *P. cactorum* (20,21).

Because little information about leather rot was available, research was initiated at The Ohio State University in 1982 to study its epidemiology and to develop methods for its control. Research efforts emphasized the influence of environmental factors, such as temperature, rainfall, and wetness duration, on disease cycle components of infection, sporulation, and dispersal. The dynamics of disease increase in time and spread in space were also studied. We wish to summarize here the progress made and the information obtained.

Dr. Madden's address is: Department of Plant Pathology, The Ohio State University, Wooster, OH 44691.

Symptoms

The symptoms of leather rot are easily confused with those of gray mold, caused by *Botrytis cinerea* Pers.:Fr., and of other fruit rots and physiological disorders. *P. cactorum* can infect fruit at any stage of development. On green fruit, diseased areas are usually dark brown but occasionally remain green with a brown margin. As the rot spreads, the entire fruit becomes brown, maintains a rough texture, and appears leathery (Fig. 1). On fully mature fruit, infection may cause little change in color or may cause discoloration ranging from brown to dark purple (Fig. 1). When diseased fruit are sectioned, the vascular tissue to each seed is noticeably darkened. In later stages of decay, mature fruit tend to become tough and leathery. Occasionally, under conditions of high moisture, a fine white growth of mycelium is observed on the surface of infected fruit. Eventually, both infected green and mature fruit dry to form hard, shriveled mummies.

Fruit affected by leather rot have an unpleasant odor and taste, and these are diagnostic symptoms of the disease. In 1982, the smell of leather rot could be detected 0.4 km from a field with a high incidence of disease. The unpleasant taste of leather rot is one of the major concerns facing growers. Even healthy tissue on a slightly rotted fruit has a very bad flavor. Infected, mature fruit with little color change may appear healthy and are picked and may be consumed along with healthy fruit.

Growers have experienced complaints from customers about off-flavored jams and jellies after processing fruit from fields where leather rot was present. In a highly competitive pick-your-own strawberry business, which accounts for most of Ohio's production, growers cannot afford to sell fruit of substandard quality. Other strawberry fruit rots, such as gray mold, have distinct symptoms that consumers can generally recognize and avoid. Although crop loss to other diseases may be severe, pickers can avoid the rotted fruit and harvest the remaining high-quality fruit. When leather rot occurs on mature fruit, symptoms can be subtle and infected berries are easily picked along with healthy fruit. Therefore, the level of tolerance for leather rot in a field is very low. Some growers have

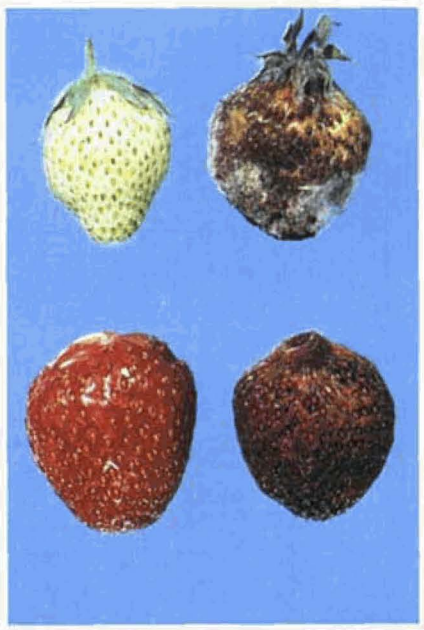

Fig. 1. (Left) Healthy immature (top) and mature (bottom) strawberry fruits compared with (right) discolored, shriveled fruits with leather rot.

closed sections of plantings to pick-your-own customers as soon as leather rot was observed.

Selected Epidemiological Components of Leather Rot

Prior to 1982, the influences of the environment on infection or sporulation by *P. cactorum* were not understood, and the perceived means of overwinter survival was based merely on speculation. Thus, quantitative information on various components of the disease cycle was needed.

Infection. Because free moisture (from dew or rain) and temperature are very important for infection of plants by *Phytophthora* spp., our studies with *P. cactorum* involved a series of attached-fruit inoculations (sporangial suspension, 400/ml) in controlled environment chambers (7). Constant temperatures ranged from 6 to 30 C and wetness durations were between 0 and 5 hours. Five days after each inoculation (at an incubation temperature of 20 C), fruit were evaluated for disease symptoms and *P. cactorum* was isolated from infected fruit to confirm visual assessments. We previously found that the latent period was 5 days at about 20 C.

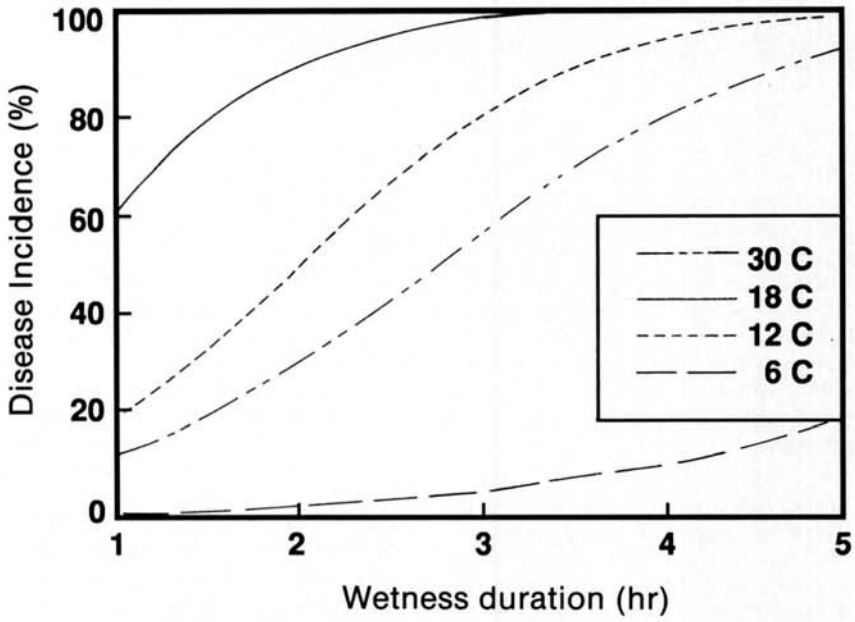

Fig. 2. Predicted percentage of strawberry fruit infected by *Phytophthora cactorum* in relation to hours of wetness at temperatures of 6, 12, 18, and 30 C; predictions for 24 C are virtually identical to those for 18 C. Original data in Grove et al (7).

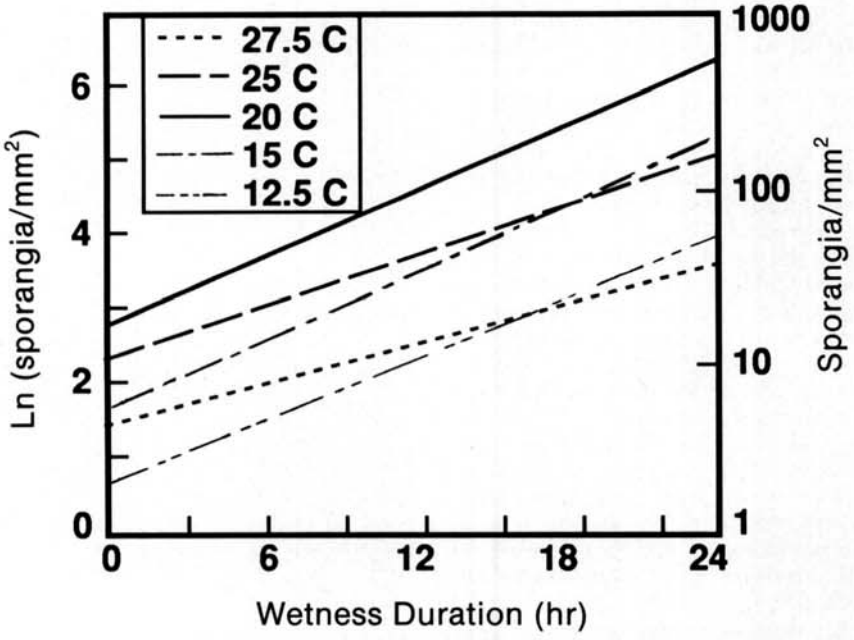

Fig. 3. Predicted logarithm of sporangial production by *Phytophthora cactorum* on strawberry fruit in relation to hours of wetness at 12.5–27.5 C. Original data in Grove et al (6).

Results clearly demonstrated that wetness duration and temperature are significant factors influencing the infection of strawberry fruit by *P. cactorum* (7). Infection occurred only in the presence of free water. Disease incidence (proportion or percentage of diseased fruit) rose with each increase in wetness duration (0–5 hours) at all temperatures tested (7) (Fig. 2). For each wetness duration, disease incidence increased up to the optimum temperature of about 21 C and then declined. Incidence of disease was high even when wetness durations were very short. At temperatures between 17 and 25 C, 2 hours of wetness resulted in greater than 80% of fruit infected.

A multiple regression model was developed to relate disease incidence (Y) to wetness duration (W, hour) and temperature during the wetness period (T, centigrade): $\ln[Y/(1-Y)] = -8 + 0.5\ T + 0.13\ WT - 0.36 \times 10^{-3}\ T^3 - 0.10 \times 10^{-3}\ WT^3$, in which $\ln[Y/(1-Y)]$ is the logit of Y. The derived model met the following criteria: an optimum-type relationship between Y and T (i.e., Y increases to a maximum and then declines) and a positive (monotonically increasing) relationship between Y and W, with constraints that predicted Y cannot be less than 0.0 or greater than 1.0. The constraints are obtained by use of the logit transformation. The equation described 85% of the experimental variability and gave realistic predictions of fruit infection level (Fig. 2). The model also was validated successfully in the field by comparing actual and predicted disease incidence based on the model (7).

Sporulation. Similar experiments were conducted to evaluate the influence of temperature and wetness period on sporangia production. Infected fruit were incubated at temperatures ranging from 10 to 30 C and wetness durations of 0–24 hours. For each temperature and wetness combination, sporangia were collected from the fruit surface and quantified (6).

Sporulation occurred only in the presence of free water. Production of sporangia increased as wetness duration increased from 3 to 24 hours at temperatures between 12.5 and 27.5 C. The most favorable temperatures for production of sporangia were between 15 and 25 C, and the optimum temperature was approximately 20 C (Fig. 3). Sporangia formed on diseased fruit after wetness durations as short as 3 hours at temperatures between 15 and 25 C but required more than 12 and 6 hours of wetness at 12.5 and 27.5 C, respectively. Sporangia were not produced at 10 or 30 C. The following regression model described the sporulation data: $\ln(S) = -5.7 + 0.59\ T + 1.3 \times 10^{-2}\ WT - 4.3 \times 10^{-4}\ T^3 - 1.3 \times 10^{-5}\ WT^3$, in which S represents sporangia per square millimeter of fruit (6). This model, which explained 85% of the

variability, predicts the exponential increase in production of sporangia over time (Fig. 3). Details on model selection and choice of transformation are presented by Grove et al (6).

Overwinter survival. We have observed oospores embedded in tissues of fruit mummies obtained from inoculations in the laboratory as well as naturally infected fruit in the field (2). Oospores are highly effective survival structures that allow *Phytophthora* spp. to survive adverse environmental conditions for relatively long periods of time in plant debris and in soil. However, the role of oospores in the life cycle related to leather rot had not been described. We conducted experiments to determine if mummified fruit could serve as a source of overwintering inoculum of *P. cactorum* in strawberry fields.

Mummies produced in the laboratory were exposed at the soil surface or buried 1 cm beneath the soil inside nylon mesh pouches beginning on 1 July and were retrieved on 15 April the following year. Then, they were observed for the presence of *P. cactorum*.

P. cactorum was recovered from all infected fruit after burial beneath or exposure at the soil surface during 2 years of tests (4). Germinating oospores were observed in all overwintered mummified fruit within 14 days after placement in water. Numerous papillate sporangia, characteristic of *P. cactorum*, were observed microscopically at the distal portions of germ tubes emerging from oospores (Fig. 4). Indirect germination (production of zoospores) from several sporangia was observed also.

Conclusions. Oospores of *P. cactorum* clearly can overwinter in the northern United States, and winter conditions do not limit survival to any measurable degree. Fruit surfaces must be wet for infection to occur, but a high disease level can be obtained with two or fewer hours of wetness over a wide temperature range. Thus, conditions required for infection are not limiting disease development in Ohio. Longer durations of wetness are required for substantial sporulation; variation in sporangia production, therefore, may influence disease progress under Ohio conditions.

Spore Dispersal

Observations as early as 1926 suggested that development of leather rot was associated with rainfall episodes (18). This observation is supported by rainfall patterns in Ohio from the 1980s. In 1981, total rainfall for June in Wooster (when fruit were developing and ripening) was 3.6 cm above the long-term average, and there was a corresponding high incidence of leather rot. Likewise, in 1982, above-average rainfall (+2 cm) coincided with a high incidence of leather rot, although lower than in 1981. In 1983 and 1984, June rainfall was substantially below average (−4 to −6 cm), and leather rot was almost absent in the same fields that had high disease incidence previously. In observations made in a commercial strawberry field from 1985 to 1989, leather rot was found in greater than trace levels only if June rainfall was above average.

Leather rot is common in poorly drained areas and on berries in contact with soil (2). Prior to our work, it was assumed that contact with soil or standing water was required for strawberry fruits to be infected by *P. cactorum*. In this scenario, zoospores in flooded soil swim to fruit and germinate. Although fruit contact with soil or water is highly conducive to infection, it is not required. We have commonly observed infected fruit 30 cm above the soil surface (2). Since many of these were never in contact with soil, dispersal of infective propagules by rain splash was hypothesized as a critical component for development of leather rot epidemics. The hypothesis was supported further by low incidence of leather rot in many years, even though temperature and wetness conditions favored high infection and sporulation. Based on these observations, we initiated a series of experiments in 1983 to study the effect of rainfall on dispersal of *P. cactorum* and the epidemiology of leather rot.

Demonstration of rain splash dispersal. Dispersal of *P. cactorum* propagules by impacting waterdrops was demonstrated by releasing a single stream of individual drops onto an infected strawberry fruit on which sporulation was active. Splash droplets were captured in open petri plates placed at various distances up to 120 cm from the infected fruit (5). The plates contained a selective medium for *Phytophthora* spp. Observations by microscope showed that sporangia mostly, but also some zoospores and mycelial fragments, were readily dispersed by the impact of waterdrops. Dispersal by impact of 4-mm-diameter waterdrops resulted in colony formation on plates up to 120 cm from the inoculum source. There was an exponential gradient in colony numbers over distance (Fig. 5)—90% of all colonies were within 40 cm of the source, half were within 10 cm.

Detailed studies on dispersal from single drop impacts. Based on these results, initial development of methods and procedures to study splash dispersal was emphasized. Equipment was developed to produce single waterdrops of various (and consistent) uniform sizes and velocities at impact, representing the full spectrum of raindrop and canopy drip sizes and velocities (15). Splash events were recorded with high-speed still photography (Fig. 6) or a video camera. After fluorescent staining of sporangia on the surface of infected fruit, splash droplets were captured on water-sensitive paper and the number of sporangia per drop was determined using incident-light microscopy.

The number of droplets formed by splashes and the number of droplets bearing sporangia increased with

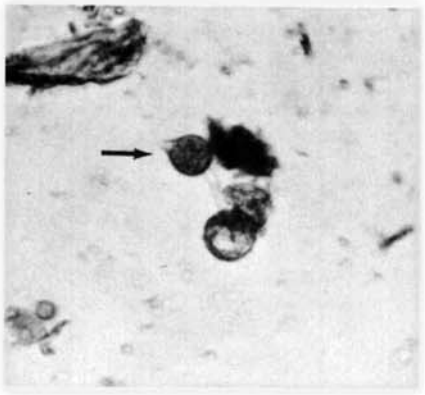

Fig. 4. Germinating oospore from overwintering strawberry fruit mummy infected by *Phytophthora cactorum*; arrow indicates sporangium at distal end of germ tube.

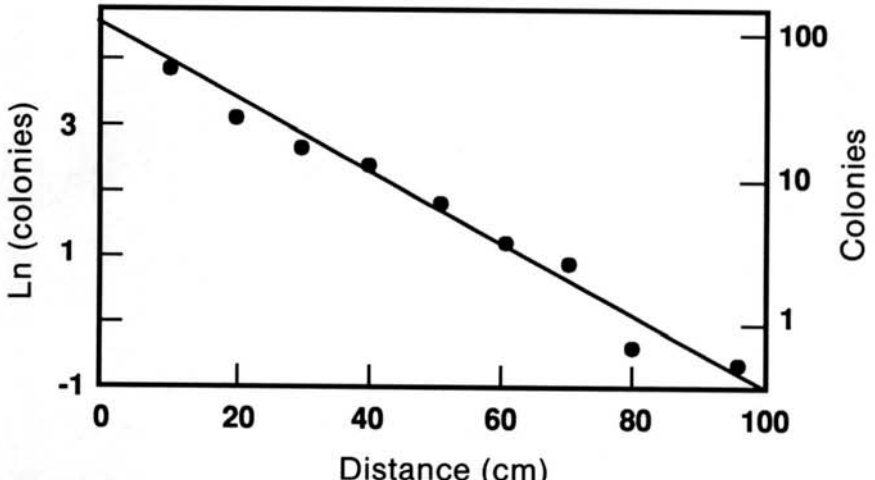

Fig. 5. Dispersal gradient of propagules of *Phytophthora cactorum*. Impact of a stream of 4-mm-diameter waterdrops dispersed propagules, and gradient was assessed as number of colonies formed on a selective medium.

increasing drop size and velocity (16) (Fig. 7). Generally, the number of splashed sporangia also increased as the velocity of impact increased. Travel distance for splash droplets was determined from the study summarized in Figure 7 (i.e., relatively low-velocity impacts) and from studies with healthy fruit targets and impacting drops traveling near terminal velocity. The majority of droplets traveled less than 10 cm from initial splashes, indicating the short-range dispersal by rain splash. The distribution of raindrop sizes during a rain episode can be predicted on the basis of rain intensity (millimeters per hour) (11), eventually making it possible to relate single-impact results to dispersal during a rainstorm.

Rain simulation. Although splash dispersal may be studied by releasing a single stream of waterdrops of uniform size onto a target and calculating resulting dispersal gradients (3), rain in nature consists of drops in a range of sizes. Raindrops also impact over a larger area than just the source of inoculum. To more accurately reproduce natural rainfall, we developed a rain simulator (generator) that uses a spray nozzle mounted on a rotating horizonal boom. Depending on the nozzle size, "rains" generated were very similar to natural rains of 15, 30, or 60 mm per hour (11).

Fig. 6. High-speed, multiple-exposure photograph of a waterdrop impacting on a strawberry fruit infected by *Phytophthora cactorum*. Arrows indicate splash droplets dispersing.

In one series of studies, rains were generated over a 1.2 × 1.2 m area that contained two concentric circular "rows" of potted strawberry plants (Fig. 8) with an inoculum source of infected fruits placed in the center. The potted plants were used to mimic a strawberry canopy in the field. Healthy fruits placed at various locations within the plant canopy served as receptors (or targets) of splash droplets with propagules. After each experiment, fruit were collected, incubated in a mist chamber, and observed for symptom development. *P. cactorum* was disseminated up to 90 cm from the source with as little as 15 minutes of rain (11; Madden, *unpublished*). Disease incidence declined exponentially over distance from the source of inoculum. Fruit placed on the ground had a much higher disease incidence than those elevated 10 cm above the ground. This vertical disease gradient is consistent with field observations.

Additional and more detailed studies have focused on the effects of ground cover and rain intensity on dispersal. Because trajectories of splash droplets generally are very short (≤10 cm), inoculum dispersal over greater distances depends on multiple resplashing of droplets, which in turn should be highly influenced by the type of ground cover. We found that plastic ground covers are conducive to high levels of splash dispersal and disease development (Fig. 9) (10). The level of dispersal was lowest with straw and intermediate with soil and with sand. Differences in surface topography and texture presumably altered trajectories of splash droplets and loss of inoculum through the ground cover. The type of ground cover used (straw vs. plastic mulch) can thus have profound effects on splash dispersal of *P. cactorum* and the epidemiology of leather rot. These results have significant implications because plastic row covers are used extensively in the major strawberry production systems in the United States.

Rain intensity over an experimental area also influences inoculum dispersal in an unexpected manner. For instance, a rain intensity of 30 mm per hour results in less disease than 15 mm per hour (10,11) (Fig. 9). Because raindrop sizes increase with rain intensity and large drops disperse more droplets with sporangia than do small drops (Fig. 7) (3,16), the effect is not caused by initial dispersal from an inoculum source. Apparently, redistribution of droplets by multiple splashings as well as wash-off of sporangia from fruit are responsible for observed results. Use of straw diminished the effect of rain intensity to the point that intensity did not appear to influence disease level (Fig. 9). Ground cover, thus, has a major influence on the epidemiology of leather rot, and its manipulation (i.e., use of straw instead of plastic) should be useful in controlling the disease.

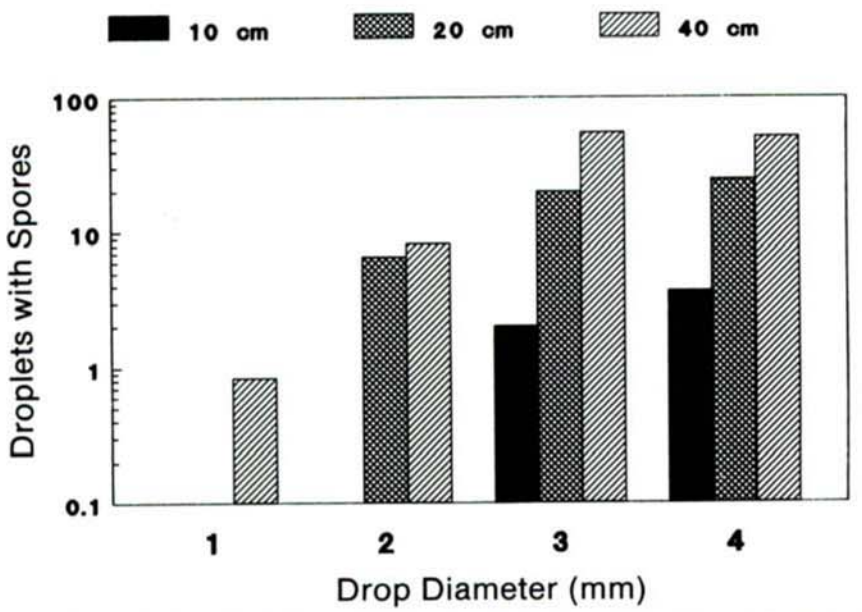

Fig. 7. Number of splash droplets carrying sporangia relative to size and distance of fall of impacting waterdrops onto a strawberry fruit infected by *Phytophthora cactorum*.

Disease Dynamics in the Field

After studying leather rot under controlled conditions, we attempted to characterize leather rot epidemics in the field and thereby confirm the results of our previous work. Epidemic development of strawberry leather rot was monitored in commercial field plots in 1986 and 1987 (12,14). In both years, rainfall was above average during the strawberry growing season. Straw mulch was removed from between the rows in several plots (nonstraw plots), whereas a layer of straw (initially 8 cm thick) was kept between the rows in others (straw plots). The outside rows of the nonstraw plots were also adjacent to straw. Plots were infested with *P. cactorum* by placing five infected strawberry fruits at the end of each plot in the interior aisles. Incidence of diseased fruit (i.e., proportion of tagged cymes with one or more in-

Fig. 8. Experimental setup of strawberry plant canopy consists of two circular "rows" 30 cm apart. Entire 1.2 × 1.2 m area is impacted by a uniform generated rain.

fected fruit) was assessed 5 days after each rain event. Final disease incidence was 60–100% in most of the rows facing soil of the nonstraw plots but less than 10% in straw plots and in the outside rows of nonstraw plots that were adjacent to straw (Fig. 10), as was expected from the rain simulation results. Disease incidence in the nonstraw plots increased over time with apparent infection rates (r) of 0.18–0.25 per day (Madden, *unpublished*). For rows facing straw, r was less than 0.06 per day. Infected fruit were observed at all possible elevations within the canopy.

Regression analyses were calculated to examine the relationship between the change in disease incidence in nonstraw plots and selected weather variables such as rainfall or indices derived from the weather variables. Indices were based on our models for infection (7), sporulation (6), and dispersal (12), as well as a newly derived fruit colonization index (14). Weather variables included consecutive hours of free moisture for the 24-hour period before a rain episode (a measure of estimated sporulation) and total hours of free moisture starting at the beginning of a rain episode (a measure of estimated fruit infection). Temperature variables were also calculated for these periods of free moisture (14). Stepwise regressions using weather variables always yielded predictive models that differed significantly between years, whereas regression models using indices for rainfall, sporulation, and colonization yielded a common model for both years. The model derived from data from both years was: $Z = 2.2X_1 - 15.8X_1X_2 + 7.2X_2X_3$, in which Z = change in logits between successive disease assessments ($i - 1$ and i) [$= \ln(Y_i/(1 - Y_i)) - \ln(Y_{i-1}/(1 - Y_{i-1}))$]; X_1 = rain dispersal index (range of 0 to 1) based on total amount of rain (mm) in an episode (R) [$= 1 - \exp(-0.07R)$]; X_2 = sporulation index calculated from the sporulation equation of Grove et al (6), using temperature and wetness duration from field measurements for the 24 hours before a rain, divided by 640 to obtain a 0–1 range; and X_3 = colonization index equal to the scaled weighted sum of hourly air temperatures in the 72 hours preceding the sporulation period (weights were based on the fraction of each hour with free moisture).

This model explained 65% of the disease incidence variability over 2 years, which is reasonably high considering that data from each plot were not averaged before analysis. The model accounted for fruit colonization and lesion expansion (X_3), sporulation (X_2) immediately before a rain event, and the amount of rainfall (X_1) during a rain event. Because the duration of wetness necessary for infection is very short (7), an infection predictor variable was not significant and was not included in the model. In fact, the infection model indicated nearly 100% infection for all rain events. Inclusion of the X_1X_2 term compensated for the co-occurrence of high values of rain amounts and conduciveness for sporulation. Variables describing rain intensity (mean or maximum) or duration did not significantly improve the predictive ability of the model, indicating that rain intensity and duration do not directly influence disease increase.

Although the regression equation was derived empirically, it is consistent with current knowledge of the epidemiology of leather rot and may be the only practical means of describing or predicting leather rot development until a mechanistic (simulation) model on dispersal is fully developed. At a minimum, the equation can be used to identify periods when rapid increase in disease can occur.

Because there are many potential sources of *P. cactorum* in commercial strawberry fields, pathogen dispersal or disease spread cannot be analyzed by quantifying easily discernible gradients.

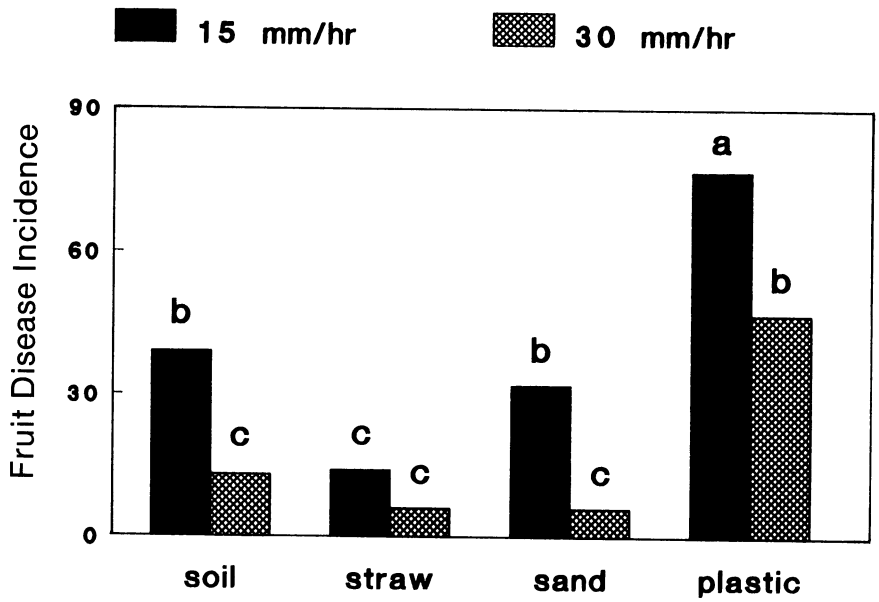

Fig. 9. Effects of ground cover and rain intensity on splash dispersal of *Phytophthora cactorum*. Means represent average for rain durations of 15 and 30 minutes. Bars topped by the same letter are not significantly different ($P = 0.05$).

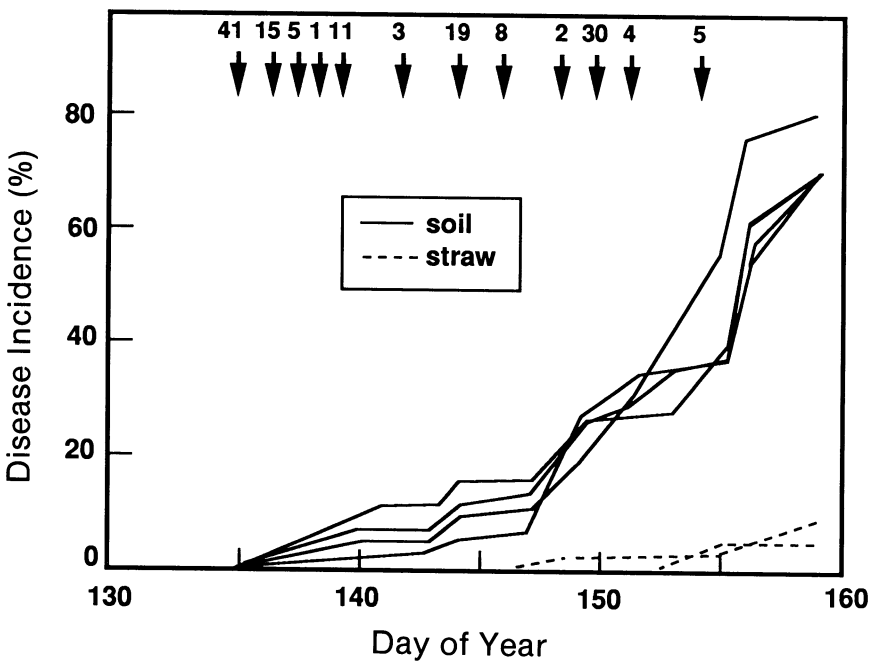

Fig. 10. Temporal progress of strawberry leather rot, caused by *Phytophthora cactorum*, as influenced by straw mulch in a three-row field plot. Values represent percentage of cymes with one or more infected fruits on each side of each of three rows. Inside rows faced soil and had a rapid increase in disease incidence (solid lines). Outside rows faced straw mulch and had a slow increase in disease incidence (dashed lines). Arrows indicate episodes of rain, and numbers are amount (mm) per episode.

However, spread of disease could be quantified with spatiotemporal autocorrelation analysis (13), which is an extension of classical time series analysis. Correlation coefficients support the hypothesis of rain splash dispersal in the epidemiology of leather rot, i.e., correlation of disease level was high between adjacent sampling locations and low between more separated locations. Details are available elsewhere (13).

Life Cycle for *P. cactorum*

Based on the information available, the life cycle for *P. cactorum* can be summarized (Fig. 11). *P. cactorum* survives the winter as oospores that form within mummified fruit. In spring, oospores germinate and produce sporangia. In free water, zoospores generally are produced and liberated. They swim or are splashed to fruit, germinate, and infect either immature or mature fruit. In free moisture (from dew or rain), sporangia are produced on the surface of infected fruit starting about 5 days after infection. Production of sporangia increases exponentially and is highly influenced by temperature (optimum is approximately 20 C). Sporangia are dispersed to other fruit surfaces by splashing of raindrops or overhead irrigation; mycelial fragments and zoospores also can be dispersed in this way. Possibly, zoospores can be released into pools of standing water in the field, which can flow down rows and disseminate propagules to fruit it contacts. Although infection requires free moisture, high levels of disease incidence can occur with as little as 2 hours or less of wetness at 17–25 C. Infected fruit that remain in the field dry up, mummify, and eventually fall to the ground.

Control

Because options for fungicidal control of leather rot are quite limited, cultural controls that provide a less conducive environment for disease development must be emphasized. Current control recommendations are based on field studies and general principles regarding the effect of horticultural practices on strawberry microclimate.

Cultural practices. The first and perhaps most important decision a grower can make to reduce the likelihood of leather rot epidemics is to reduce or eliminate standing water. Planting sites should have excellent water drainage. Increased use of drainage tiles, planting on ridges or raised beds, avoiding cultural operations that result in the formation of ruts between rows, and orienting rows to facilitate runoff of surface water should be beneficial.

The planting site should have good air circulation and be fully exposed to sunlight to reduce periods of free moisture on fruit. Application of supplemental water in the form of irrigation should be timed so that foliage and fruit will dry quickly. Straw mulch is highly beneficial for controlling leather rot (10,14) for at least three reasons: It keeps fruit from contacting the soil where the fungus may reside, it provides a barrier between fruit and freestanding water, and it reduces the splashing of water droplets bearing sporangia.

Because weeds in a strawberry planting can reduce air circulation and prolong wetness periods on fruit, weed control may aid in leather rot control. Excessive fertilizer (especially nitrogen) stimulates very dense foliage that can extend wetness periods. Therefore, proper fertilization based on soil and foliar analysis is recommended. We have not, however, tested the effects of weed control and fertility on leather rot development.

Fungicides. Often cultural practices may be sufficient for effective control of leather rot, but sometimes fungicides will be necessary, especially during wet years. Leather rot is seldom the only fruit rot disease found in strawberry fields. Gray mold, in particular, is very common and can even be found on the same plant with fruit infected by *P. cactorum*. Unfortunately, the most common and efficacious fungicides for control of gray mold (i.e., benomyl, thiophanate-methyl, vinclozolin, and iprodione) are not effective against leather rot. Captan and thiram are broad-spectrum protectant fungicides registered for use on strawberry for gray mold control that, although not highly efficacious, provide some leather rot control. Because these are not registered for leather rot, they can be recommended only for gray mold control. The use of broad-spectrum protectant fungicides in combination with the most efficacious fungicides for gray mold control provide a high level of control of both diseases.

Metalaxyl (Ridomil) and fosetyl-Al (Aliette) are two systemic fungicides that are highly effective for the control of leather rot (1,19; Ellis, *unpublished*). Ridomil was registered for use on strawberry in the United States in October 1990, and Aliette might be registered in the near future. Use of Ridomil, based on weather conditions and the described model, combined with beneficial cultural practices in an integrated control program should result in effective disease control.

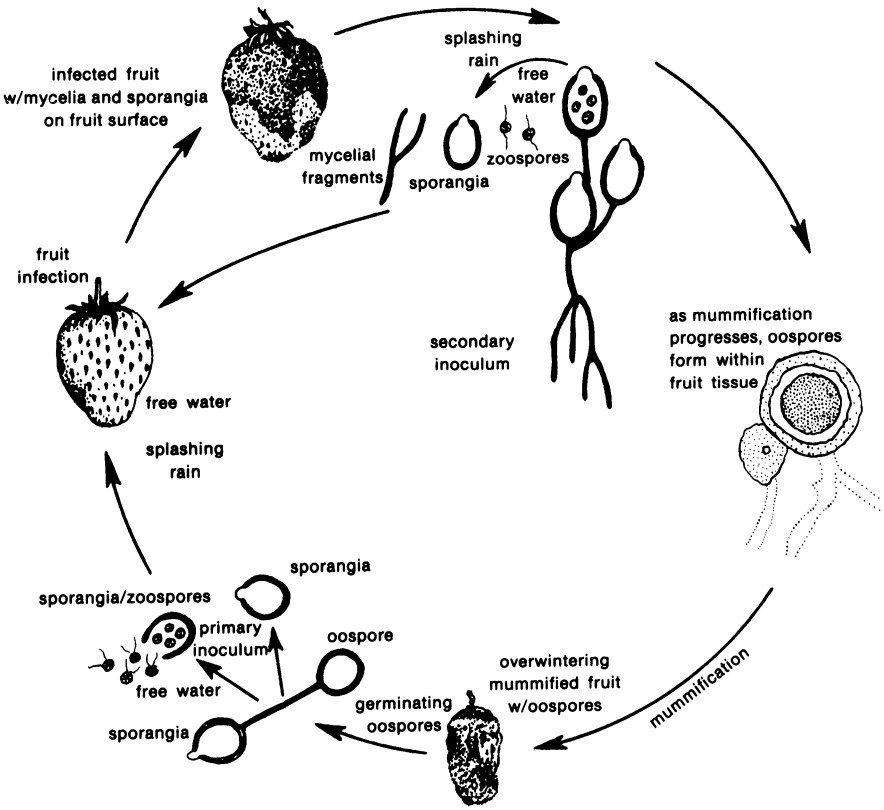

Fig. 11. Life cycle of *Phytophthora cactorum*, the cause of leather rot in strawberries.

Summary and Conclusions

Leather rot remains a sporadic disease in Ohio. Although it may not be possible to find diseased fruit in commercial fields in some years, there can be >50% disease incidence in others. The sporadic nature of the disease and its confusion with other fruit rots probably are reasons why its importance in other parts of the United States is not known.

Leather rot can reach high levels after rain episodes. Rain splash is responsible for propagule dispersal and has a major impact on disease spread. Disease increase can be predicted on the basis of the amount of rain during a rain episode

and the suitability of the environment for sporulation and fruit colonization. It may be possible to use a predictive model to time the applications of fungicides such as Ridomil. During most years, cultural methods should attain adequate control of leather rot. Because it influences splash dispersal and keeps healthy fruit separated from inoculum in the soil, straw covering is highly beneficial in controlling the disease. Still more research is needed to evaluate influences of other cultural practices on disease development.

Acknowledgments

Salaries and research support were provided by state and federal funds appropriated to the Ohio Agricultural Research and Development Center and The Ohio State University; manuscript No. 316-90. Portions of this work were supported by USDA grants 85-CRCR-1-1537 and 87-CRCR-1-2307 as well as the North American Strawberry Growers' Association.

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L. V. Madden

M. A. Ellis

G. G. Grove

K. M. Reynolds

L. L. Wilson

Dr. Madden is an associate professor in th Department of Plant Pathology at The Ohio State University (OSU), Ohio Agric ltural Research and Development Center (OARDC), Wooster. His research invol es the quantitative epidemiology and modeling of fungal and viral plant diseas es. Current research is focusing on pathogen dispersal and disease spread. He received his B.S. degree in 1975, M.S. degree in 1977, and Ph.D. degree in 1980 all from The Pennsylvania State University.

Dr. Ellis is a professor in the Department of Plant Pathology at OSU/OARDC. His research involves the epidemiology and management of diseases of apples, grapes, strawberries, and other fruit crops. He received his B.Sc. degree in 1971 and M.Sc. degree in 1973 from Eastern Illinois University and his Ph.D. degree in 1976 from the University of Illinois. Before coming to Ohio, he served as an assistant professor at the University of Puerto Rico.

Dr. Grove is an assistant plant pathologist at Washington State University, Tree Fruit Research Center, Wenatchee. His research involves the epidemiology and management of diseases of apples and other fruit tree crops. He received his B.S. degree in 1976 and M.S. degree in 1979 from Ohio University (Athens) and his Ph.D. degree from OSU in 1984. He worked as a postdoctoral researcher at the University of California, Davis, before being employed in Washington.

Dr. Reynolds is a research scientist with the U.S. Forest Service, Anchorage, Alaska. His research involves the epidemiology of diseases of forest trees. He received his B.S. degree in 1976 and M.S. degree in 1981 from Oregon State University and his Ph.D. degree in 1984 from North Carolina State University. He worked as a postdoctoral researcher at OSU for 2 years before going to Alaska.

Mr. Wilson is a research assistant in the Department of Plant Pathology at OSU/OARDC. He works on the epidemiology and management of diseases of fruit crops. He received his B.S. degree in 1980 and M.S. degree in 1988 from OSU.