

## Powdery Mildew of Tomato: The Effect of Planting Date and Triadimefon on Disease Onset, Progress, Incidence, and Severity

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### ABSTRACT

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Powdery mildew epidemics of tomato, caused by the pathogen *Leveillula taurica* (= *Oidiopsis taurica*), were monitored in experimental field plots. Disease onset, incidence, severity, and disease progress were measured in relationship to host growth during several successive planting dates in both 1984 and 1985. Disease was assessed by counting individual lesions on randomly sampled leaves and whole plants. Canopy defoliation was measured by counting the number of necrotic leaves on individual plants. Disease onset occurred at an earlier stage of crop development with each successive planting date; the data indicate that disease onset was not related to the physiological age of the crop nor any specific meteorological conditions. In general, disease distribution on a single plant was very aggregated, with considerably more lesions observed on older leaves than

younger leaves for all sample dates. In the field, both immature and mature host tissue was susceptible to infection by *L. taurica*. When whole plants were the sampling unit, individual plants within a treatment from throughout the experimental plot had a similar disease incidence and severity for any given sample date. Disease progress curves fit both a logistic and Gompertz model reasonably well ( $r^2$  values ranged from 0.60 to 0.99). The fungicide triadimefon was effective at controlling powdery mildew epidemics. Overall, powdery mildew caused a significant increase in defoliation over triadimefon treatments in the late plantings. However, even when powdery mildew disease was severe, it did not cause any reduction in yield of two susceptible fresh market tomato cultivars harvested at the "mature green" stage of development.

Powdery mildew of tomato (*Lycopersicon esculentum* Mill.), caused by the pathogen *Leveillula taurica* (Lév.) Arn. (= *Oidiopsis taurica* (Lév.) Salmon), was first reported in the United States in 1978 (13). Since this report, *L. taurica* has been found in all of the major tomato-growing regions of California (9). Although *L. taurica* has a broad host range in the geographical regions where it occurs (11,16,19,21), including California (9), its primary economic host in the state is tomato. Approximately 100,000 ha of tomatoes are grown in California annually, and the introduction of this pathogen poses a potentially serious threat to the tomato industry. However, the impact of this disease on tomato production is not known. Furthermore, there are few published accounts of the impact this disease has on tomato production worldwide (16).

Powdery mildew has been reported to be more important on mature tomatoes, and several workers have reported that older tissue may be more susceptible to infection (10,14,16,17). However, many of the variables that affect powdery mildew of tomato are poorly understood, with epidemics often being sporadic and unpredictable. As a result, the early stages of an epidemic often go unnoticed. Moreover, most of the reports on the biology of this pathogen have been based on work done in the greenhouse or growth chamber (15-17). As pointed out by Palti (16), very little has been published on the epidemiology of *L. taurica* under field conditions. Consequently, there is a paucity of data on the factors that influence the epidemiology of this disease. The objective of this investigation was to quantify several

epidemiological parameters of this somewhat unusual powdery mildew under field conditions.

Field observations in California (6) suggested that the level of powdery mildew of tomato may be correlated with both host age and planting date. Therefore, disease onset, incidence, severity, and disease progress were measured in relation to host growth and development for several successive planting dates in 1984 and 1985. Field inoculations were conducted to determine the susceptibility of mature and immature tomato foliage. The impact of powdery mildew on tomato yields also was assessed.

### MATERIALS AND METHODS

**Field plots.** A field plot, located at the University of California West Side Field Station (Fresno County, CA), was established in 1984 and 1985. Two fresh market tomato cultivars, Royal Flush and Jackpot, were used throughout the study. These two cultivars were widely used commercially in California during the 1984 and 1985 seasons and are susceptible to powdery mildew (6). Tomatoes were either direct seeded (in 1984) or 6-wk-old seedlings transplanted (in 1985) onto beds with 1.5 m centers. Transplanted seedlings were spaced 0.4 m apart, and direct seeded tomatoes were thinned to a 0.4-m spacing. All rows received a preplant fertilizer (NO<sub>3</sub>-N, 160 kg/ha; P<sub>2</sub>O<sub>5</sub>-P, 146 kg/ha) and a preplant herbicide (devinol, 1.2 kg/ha).

There were three planting dates in 1984 and four in 1985 for each of the two cultivars. Tomatoes were seeded on 8 March, 5 May, and 25 July in 1984. After emergence, plants were thinned on 3 May, 8 June, and 20 August for each of the three successive plantings. In 1985, seedlings were transplanted on 2 April, 16 May,

2 July, and 30 July. All planting date treatments were furrow irrigated every 8–14 days after plants were established.

Each cultivar from each planting date was split into a fungicide-sprayed and unsprayed treatment. Fungicide-treated plants received one application of triadimefon (Bayleton 50WP) approximately 45 days after thinning or after transplanting. Triadimefon was applied with a 4-L hand-held pressure sprayer at a rate of 0.14 kg/ha. The material was applied with 2 L of water per 16 m of row. Each treatment was replicated three times, and the plot was set up in a completely randomized split-plot design. Each subplot measured 16 × 6 m (four rows); there were approximately 200 plants in each subplot and 10,000 plants in the entire plot. Only the center two rows in each subplot were sampled. A border row was left between each main plot and two rows were left between each block.

Russet mites (*Aculops lycopersici* Masee) caused some damage to tomato foliage during the 1984 season. In 1985, the insecticide cyhexatin (Plictran 50W) was used to control russet mites. All treatments from all four planting dates received one application of cyhexatin approximately 50 days after transplanting. Cyhexatin was applied at a rate of 1.1 kg/ha with a low pressure orchard sprayer. Cyhexatin was used because it was effective at controlling the russet mite population and had no measurable effect on powdery mildew of tomato (Correll, unpublished).

**Weather data.** A USDA/ARS CIMIS (18) weather station, located 0.5 km from the West Side Field Station plot, was used for collecting weather data. Crop development was expressed as the cumulative degree days after transplanting or thinning (23,24). Crop development was expressed in degree days to provide a physiological comparison between planting dates. Disease progress was expressed in days as well as degree days to provide a more direct comparison between disease and physiological age of the crop for the different planting dates. Degree days were calculated during the growing season using a computer-based subroutine as follows: temperatures were recorded every hour and degree days accumulated per day: degree days/day = ( $\Sigma$  temperature (in C) per hr/24) – 10 (Eq. 1).

Photosynthesis of tomato slows down considerably at temperatures exceeding 32 C (23,24). Therefore, any temperature greater than 32 C was recorded as 32 C. Degree day accumulation began the day after transplanting or at the time the plants were thinned in the direct-seeded plot (1984).

**Disease progress.** Preliminary work in 1983, in both commercial and experimental field plots, indicated that there were no quantitative differences in disease development between the two cultivars, Royal Flush and Jackpot (6). As a result, to reduce the sampling volume, disease progress data were only collected from the cultivar Royal Flush.

Disease severity was measured in both 1984 and 1985 by collecting 25 individual leaves at random from the middle portion of the tomato canopy (i.e., neither terminal nor older necrotic leaves were sampled) from each replication or 75 leaves per treatment. The number of individual lesions incited by *L. taurica* was recorded along with the number of leaflets per leaf. Lesions caused by *L. taurica* were positively identified by the presence of conidia and/or conidiophores. Leaf samples were collected every 7–14 days, and disease severity was recorded as the mean number of lesions per leaflet. Lesions per leaflet were obtained by dividing the number of lesions per leaf by the number of leaflets per leaf.

A disease gradient is often observed on tomato plants with powdery mildew; that is, disease severity is usually greater on the older leaves of an individual plant. Therefore, in addition to individual leaf samples, whole plant samples also were collected. Whole plant samples of cultivar Royal Flush were used for measuring both disease development and plant growth parameters from the unsprayed treatment from each planting date in 1985. Between two and five whole plants per replication were sampled every 7–14 days. The number of leaves, leaflets, and necrotic leaves per plant as well as fresh fruit weight were measured. Disease severity on whole plants was recorded as the mean number of lesions per leaflet per plant by dividing the number of lesions per plant by the number of leaflets per plant. Disease incidence per

plant was recorded as the proportion of infected leaflets (i.e., evidence of sporulation and/or symptoms) per plant.

**Harvest data.** In California, fresh market and processing tomatoes are grown as distinct crops in terms of their production methods. Typically, the fruit of fresh market tomatoes are manually harvested at a "mature green" stage approximately 75–95 days after seedlings are transplanted. This is in contrast to processing tomatoes, which are mechanically harvested when the crop is at a "mature red" fruit stage. Therefore, fresh market tomatoes are generally harvested at an earlier physiological age than are processing tomatoes.

In 1985, tomatoes from each planting date were harvested when the crop was at the "mature green" stage. The harvest time for each of the four planting dates was picked on the basis of the physiological maturity of the crop. Five plants were harvested from each treatment in each of three replications. Fresh fruit weight and the number of necrotic leaves per plant were recorded at harvest. Leaf area per plant also was measured on two plants from each treatment in each of the three replications. Leaf area has been correlated ( $r^2 = 0.87$ ) with leaf length and width (6) and was estimated by measuring the length and width of each leaf on a plant: Leaf area (cm<sup>2</sup>) = (LW × 0.615) – 5.15 (Eq. 2), where LW = leaf length × leaf width (in centimeters). Leaf width was measured at the widest point.

After fruit from a planting date treatment was harvested, the remaining plants were not incorporated into the soil but allowed to remain in the field. Thus, plants from all four planting dates were present throughout the duration of each experiment.

**Inoculation experiment.** A field plot also was established at the San Jose Field Station (Santa Clara County, CA) in 1985. This location was selected because of the absence of surrounding tomato acreage and thus a low probability of background inoculum of *L. taurica*. Six-week-old tomato seedlings (Royal Flush) were transplanted on 16 May and 9 July 1985 using agronomic practices similar to those previously described. On 25 July, six leaves were tagged on each of six randomly selected plants from the earlier planting; three older fully expanded mature leaves on mature plants and three newly emerging immature terminal leaves on mature plants were tagged. Plants from the earlier planting had green fruit (about 3,000 g per plant or approximately 65% of maximum fruit load) at the time of inoculation. Three newly emerging leaves also were tagged on each of 10 randomly selected immature plants from the later planting. The younger plants were blooming, had no fruit, and had approximately 15 leaves at the time of inoculation.

Inoculum for the field inoculation study was collected from a commercial tomato field in Fresno County on 24 July. Tomato leaves, which showed heavy sporulation of *L. taurica*, were collected from mature tomatoes at 0800 hours and stored at approximately 15 C in plastic bags until they were used as the inoculum source for tests at the San Jose Field Station. All tagged leaves were then inoculated on 25 July at 1800 hours as follows: two to five heavily infected leaves were held about 20 cm above each tagged leaf and rapidly shaken for 10–20 sec. Tagged leaves were positioned so that dislodged conidia would land on all the leaflets of a given leaf. Viability of the conidia was confirmed by collecting conidia on glass microscope slides placed adjacent to several of the tagged leaves. The slides were left in the canopy for 24 hr, after which time conidia were examined for germination. Uninoculated leaves were tagged on plants in nearby rows to verify the absence of background (natural) inoculum.

Infection and latent period (time from inoculation until sporulation) were determined on mature leaves (fully expanded) of mature plants, immature leaves (newly emerged) of mature plants, and on immature leaves of immature plants.

Disease and growth parameters were measured on all tagged leaves. Leaf length, width, leaflets per leaf, number of leaflets infected, lesions per leaflet, and number of necrotic leaflets per leaf were recorded. Leaf area was estimated by measuring leaf length and width as previously described. Measurements were taken 0, 4, 7, 11, 15, and 26 days after inoculation. In addition, inoculated leaves were examined for conidia and conidiophore production at

each sample date with a 20× hand lens.

Temperature and relative humidity were continuously monitored using a calibrated hygrothermograph placed in a white, ventilated weather shelter placed at canopy height (40 cm). Degree days were calculated as previously described.

**Air sampling.** Limited attempts to monitor airborne spores of *L. taurica* in 1983 and 1984 with a roto-rod spore sampler were unsuccessful. No spores were recovered on rods when the air was sampled over several 24-hr periods in tomato fields with a high incidence of powdery mildew. Air was also sampled in a commercial processing tomato field with a hand-held field-portable spore sampler (8). In this particular field, plants had a very high incidence of powdery mildew and were approximately 1 wk from harvest. Air samples were taken between 1200 and 1400 hours and 2100 and 2300 hours on 23 and 29 July. Approximately 1.5 L of air was sampled 1 m above the tomato canopy before and after the canopy was disturbed. The air was sampled in 10–20 locations throughout the field. The tomato canopy was disturbed by shaking plants approximately 3 m upwind from where the air was being sampled. This was done to simulate canopy disturbance as would occur during commercial mechanical harvesting operations. The wind speed during the collection period was less than 2 m s<sup>-1</sup> except for the night sample taken on 29 July when wind speed was estimated to be between 2 and 7 m s<sup>-1</sup>.

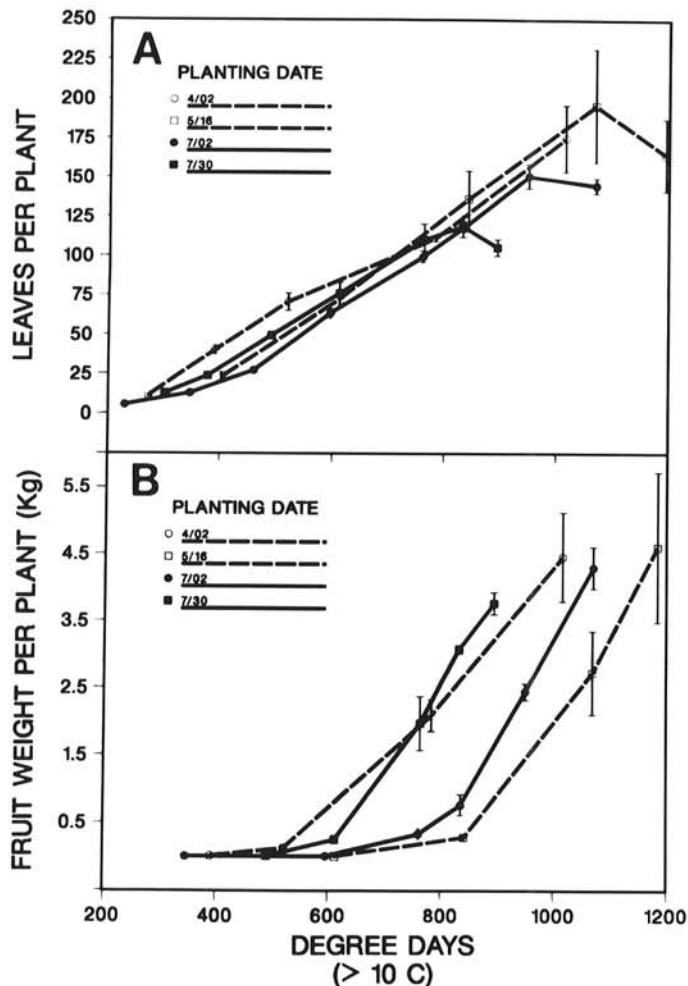


Fig. 1. Growth parameters of plants in the unsprayed, Royal Flush treatment. **A**, Total number of leaves per plant (including necrotic leaves) for four planting dates in 1985. Each data point is the mean of two to five plants from each of three replications. Vertical bars represent the standard error of the mean. **B**, Fresh fruit weight per plant (in kilograms) for four planting dates in 1985. Each data point is the mean fruit weight from two to five plants from each of three replications. Vertical bars represent the standard error of the mean.

## RESULTS

**Crop development.** In 1984, crop development within a planting date treatment was not uniform. Soil compaction during the season led to uneven seed emergence, poor canopy development (particularly in the last planting), and a very uneven canopy development within each of the planting dates; the result was that individual plants from the same planting date would often differ markedly in their physiological maturity. Russet mite damage in 1984 also may have had an adverse effect on canopy development. In 1985, canopy development was much more uniform within a planting date treatment.

In 1985, crop development was compared for the four planting dates of the unsprayed Royal Flush treatment. The rate of leaf development per plant was similar for all four planting dates (Fig. 1A). However, the maximum number of leaves per plant reached at harvest varied between planting dates. The rate of fruit development also was similar for the four planting dates even though the beginning of fruit set occurred at different times (Fig. 1B). Fruit set occurred earlier in the first (2 April) and the last (30 July) plantings than in the second (16 May) and third (2 July) plantings. The delay in fruit set for the second and third planting dates may have been due to hot temperatures (several days > 38 C) during the initial flowering period. Rudich et al (20) and Wilson et al (24) have observed that high temperatures may cause both flowers and fruit of tomato to abort.

**Disease onset.** Disease onset, as measured by individual leaf samples, occurred at an earlier stage of crop development for each successive planting date in both 1984 and 1985. Disease was first observed in the plot on 5 July (day 186) in 1984 and 16 July (day

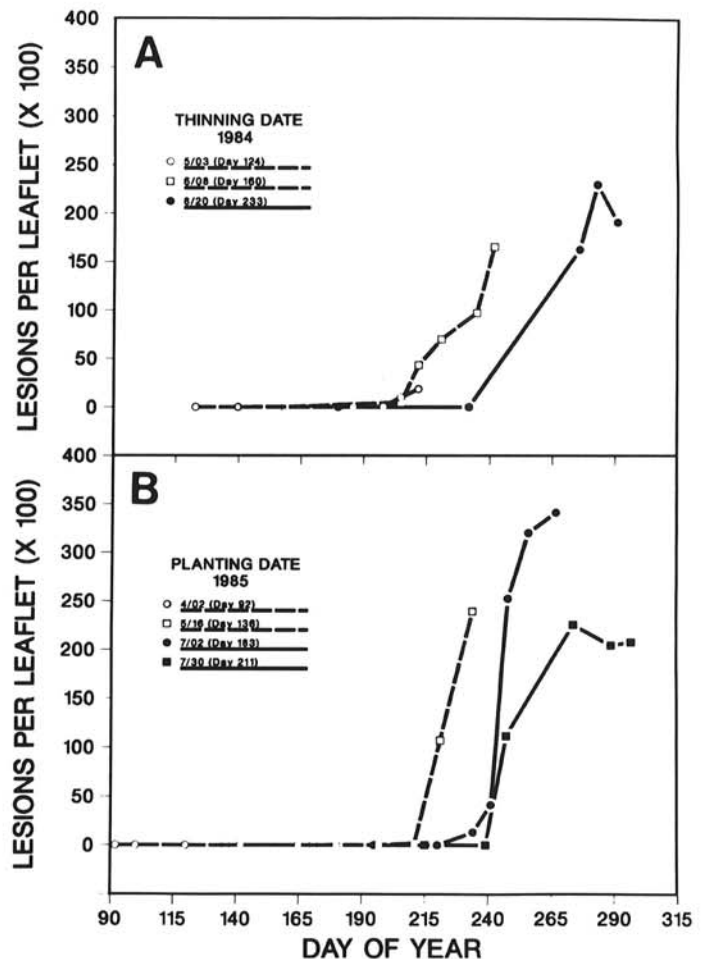


Fig. 2. Disease progress of epidemics in the unsprayed Royal Flush treatment in 1984, **A**, and in 1985, **B**, plotted against the day of the year. January 1 = Day 1. Each data point is the mean disease severity (lesions per leaflet) of 25 leaves from each of three replications.

197) in 1985 (Fig. 2). In 1984, disease onset occurred approximately 1,000 (5 July), 700 (25 July), and 350 (18 August) degree days after thinning for the first, second, and third planting dates, respectively (Fig. 3A). In 1985, disease onset occurred approximately 1,100 (16 July), 600 (8 August), and 400 (26 August) degree days after transplanting for the second, third, and fourth planting dates, respectively (Fig. 3B). No disease was observed on plants from the first planting date in 1985. Disease onset occurred when the crop was at the "mature green" fruit stage, fruit set, and bloom (no fruit) stage for the early, middle, and late plantings, respectively, in both years.

Disease also was measured by sampling whole plants in 1985. Disease onset, as measured by whole plant samples, closely corresponds with disease onset measured by individual leaf samples (Fig. 4). However, lesions were observed on whole plants somewhat sooner than on individual leaf samples. For example, no lesions were observed at 595 and 301 degree days in the third and fourth planting dates, respectively, for the individual leaf samples (Fig. 4); whole plant samples taken at these times had a mean disease severity of 0.0062 and 0.0039 lesions per leaflet per plant.

**Disease incidence and severity.** Disease progress was monitored by measuring disease incidence on whole plants and disease severity on both whole plants and randomly collected leaves. All three methods yielded similar results. In 1985, the mean disease incidence per plant (percentage of leaflets infected) at the time of harvest was 2.6, 73.7, and 40.8% for the second, third, and fourth planting dates, respectively (Fig. 5). In the third planting date treatment, disease incidence increased very rapidly between 835 (27 August) and 1,070 (17 September) degree days. Disease incidence was higher in the fourth planting date treatment throughout crop

development even though disease incidence only reached 40.8% by harvest. For any given sample date, disease incidence was similar on plants within a treatment throughout the experimental plot; i.e., the standard error of the mean of disease incidence for most sample dates was relatively small (Fig. 5).

The highest disease severity, as measured by randomly collected individual leaf samples was observed in the third planting date in both years reaching a mean of 2.4 and 3.3 lesions per leaflet in 1984 and 1985, respectively (Fig. 3). Disease severity in the last planting date in both 1984 and 1985 started to decrease by the middle of October. In general, disease severity based on individual leaf samples was considerably more variable among the replicated plots (i.e., larger standard error of the mean) in 1984 than in 1985.

Disease incidence and severity, based on whole plant samples, was also highest in the third planting date in 1985. The mean number of lesions per plant at harvest for the four successive planting dates were 0.0, 49, 4,477, and 1,170 lesions per plant at harvest for the four planting dates, respectively.

The apparent infection rate was calculated using both a logistic

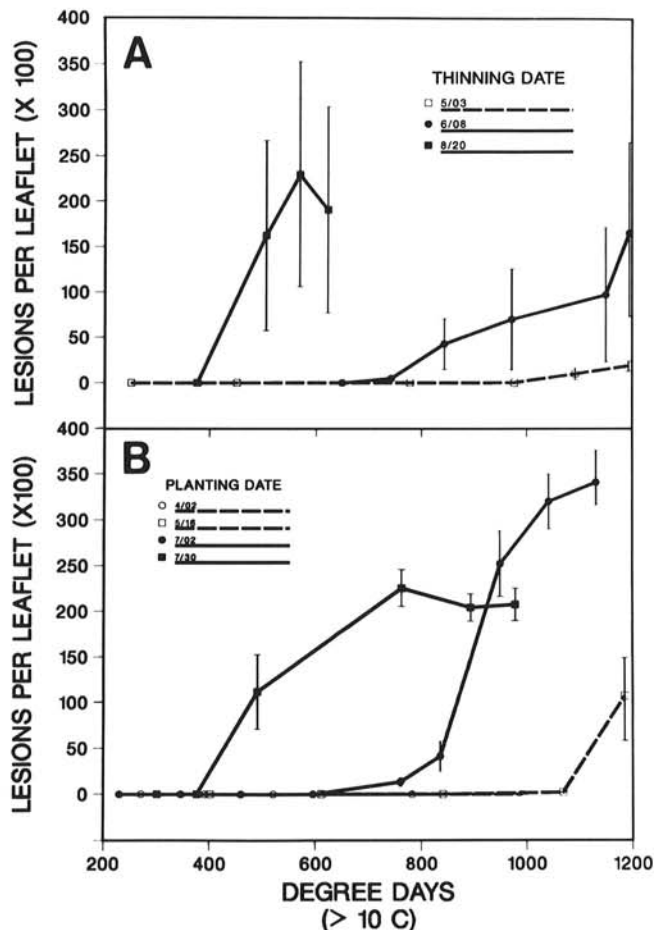


Fig. 3. Disease progress of epidemics in the unsprayed Royal Flush treatment plotted against degree days in 1984, A, and 1985, B. Each data point is the mean disease severity (lesions per leaflet) of 25 leaves from each of three replications. Vertical bars represent the standard deviation.

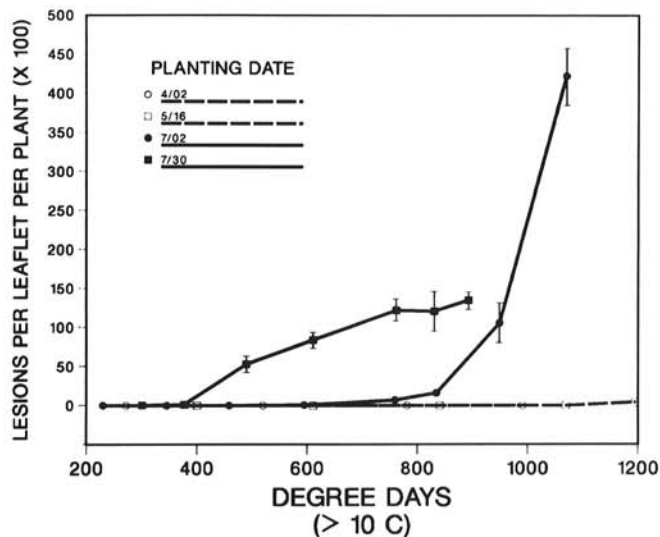


Fig. 4. Disease severity based on whole plant samples from the unsprayed Royal Flush treatment in 1985. Each data point is the mean disease severity of two to five plants from each of three replications. Vertical bars represent the standard error of the mean.

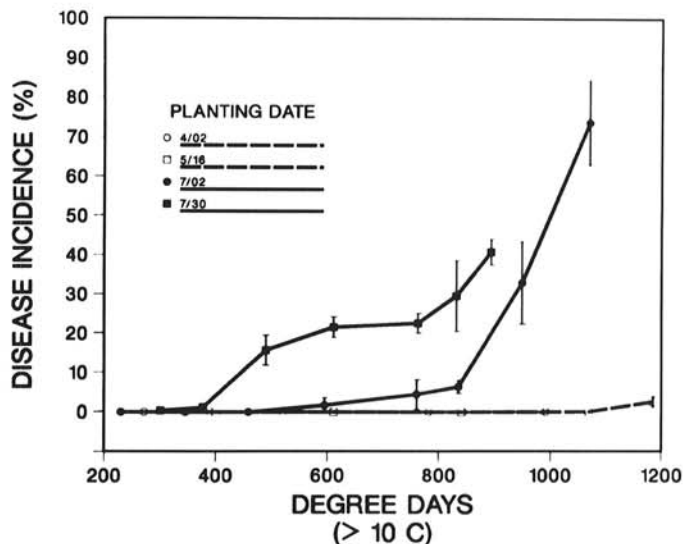


Fig. 5. Disease incidence per plant from the unsprayed Royal Flush treatment in 1985. Each data point is the mean disease incidence of two to five plants from each of three replications. Vertical bars represent the standard error of the mean.

(22) and Gompertz (3) growth model. The rate parameter, 'r' (logistic) or 'k' (Gompertz), was calculated for disease severity of both individual leaf samples and whole plant samples, disease incidence, and necrotic leaves for epidemics when at least four observations per epidemic were available. Calculated values for the rate parameter for disease severity ranged between 0.0062 and 0.0137 lesions per leaflet and 0.0097 and 0.0149 lesions per leaflet per plant per degree day (Table 1). The coefficient of determination ( $r^2$ ) for disease severity ranged between 0.60 and 0.99 (Table 1). The highest infection rate was observed in the first planting date in 1984 (thinning date, 3 May) and the third planting date in 1985 (planting date, 2 July).

**Fungicide applications.** Triadimefon was effective in controlling powdery mildew epidemics. One application of triadimefon reduced the mean disease severity (lesions per leaflet) at harvest to less than 0.50 lesions per leaflet in 1984 and 1985 (data from 1985, Royal Flush cultivar shown, Fig. 6). In the third planting date in 1985, disease severity (lesions per leaflet) decreased rapidly after application of the fungicide.

Triadimefon also reduced the rate parameter for both disease severity (lesions per leaflet) and necrotic leaves in the third and fourth planting dates (Table 1).

**Necrotic leaves.** Symptoms of powdery mildew infection initially appear as small (<0.5 cm), light green to yellow lesions on tomato leaves. The lesions expand into bright yellow chlorotic lesions that may become necrotic. Often, multiple lesions per leaf can cause an entire leaf to become necrotic. Although uninfected tomato leaves senesce as the plant matures, powdery mildew accelerates the rate of leaf necrosis, and this represents a major component of disease severity.

The number of necrotic leaves per plant were counted in the Royal Flush unsprayed treatment for each planting date to quantify defoliation. Leaf senescence started approximately 400 degree days after transplanting in the first and second planting dates in 1985 when disease was absent. The mean number of necrotic leaves per plant at harvest was 16 (approximately 10% of the total canopy) and 21 (approximately 10% of the total canopy) for the first and second planting dates, respectively. The number of necrotic leaves per plant increased rapidly at approximately 750 and 400 degree days for the third and fourth planting dates (Fig. 7) corresponding to the appearance of disease. At harvest, the mean number of necrotic leaves per plant was 47 (33% of the total canopy) and 34 (34% of the total canopy) in the third and fourth

planting dates, respectively. The mean estimated leaf areas per plant in the unsprayed Royal Flush treatment at harvest were  $1.43 \pm 0.18$ ,  $1.50 \pm 0.49$ ,  $0.98 \pm 0.38$ , and  $0.89 \pm 0.11 \text{ m}^2$  in the four successive plantings, respectively.

**Yield and leaf necrosis at harvest.** All plants were harvested between 893 and 1,196 degree days after transplanting (Table 2). Analysis of variance of fruit weight and necrotic leaves indicated a significant effect ( $P = 0.0001$ ) of both planting date and fungicide treatment on necrotic leaves but no treatment effect on fruit weight.

The mean total fresh fruit weight per plant was approximately 4.3 kg for all treatments. There were no significant differences ( $P = 0.05$ ) in fruit weight per plant among all the treatments (Table 3). However, there were significant differences ( $P = 0.05$ ) in the number of necrotic leaves per plant between planting dates and

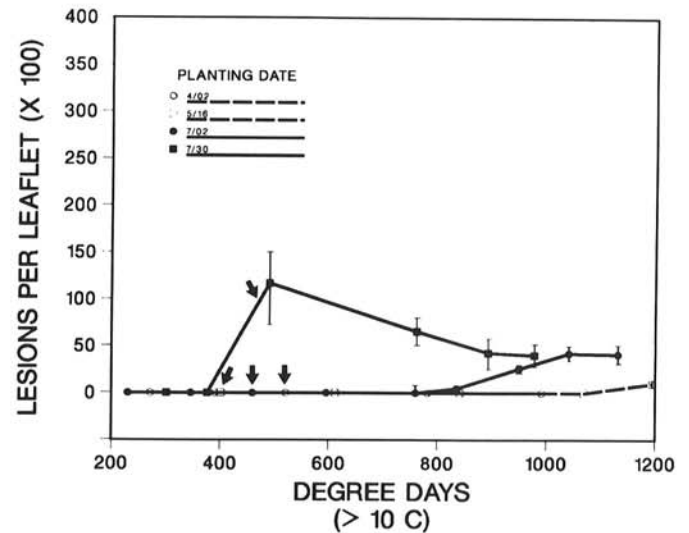


Fig. 6. The effect of triadimefon on disease severity based on individual leaf samples from the fungicide sprayed Royal Flush treatment in 1985. Each data point is the mean disease severity (lesions per leaflet) of 25 leaves from each of three replications. Vertical bars represent the standard deviation. Arrows indicate the time of fungicide application (one application of fungicide per planting date).

TABLE 1. The infection rate parameter and the coefficient of determination for powdery mildew epidemics of tomato at West Side Field Station

Disease assessment method <sup>a</sup>	Year	Planting date	Observations	Model <sup>b</sup>			
				Logistic		Gompertz	
				Rate <sup>c</sup>	R <sup>2</sup>	Rate <sup>c</sup>	R <sup>2</sup>
No fungicide							
LPL	1984	5/03 <sup>d</sup>	4	0.0137	0.89	0.0022	0.87
	1984	6/08 <sup>d</sup>	5	0.0062	0.81	0.0019	0.89
	1985	7/02	6	0.0116	0.87	0.0035	0.92
	1985	7/30	4	0.0114	0.60	0.0028	0.68
LPLPP	1985	7/02	5	0.0149	0.99	0.0044	0.92
	1985	7/30	6	0.0097	0.78	0.0023	0.84
DI	1985	7/02	5	0.0109	0.94	0.0054	0.85
	1985	7/30	5	0.0070	0.82	0.0026	0.88
NECLVS	1985	4/02	4	0.0079	0.82	0.0019	0.89
	1985	5/16	5	0.0056	0.96	0.0017	0.96
	1985	7/02	4	0.0078	0.97	0.0033	0.99
	1985	7/30	5	0.0060	0.97	0.0023	0.99
Triadimefon treatment							
LPL	1985	7/02	6	0.0104	0.83	0.0020	0.86
NECLVS	1985	7/02	4	0.0058	0.94	0.0021	0.97
	1985	7/30	4	0.0035	0.98	0.0011	0.98

<sup>a</sup> LPL = Lesions per leaflet; LPLPP = lesions per leaflet per plant; DI = disease incidence (proportion of leaflets infected per plant); NECLVS = necrotic leaves per plant. All disease progress data collected from cultivar Royal Flush.

<sup>b</sup> Logistic growth model,  $r = (\text{logit}(y_2) - \text{logit}(y_1)) / (t_2 - t_1)$  where  $\text{logit}(y) = \ln(y / (1 - y))$ ; and Gompertz growth model,  $k = ((\text{gompit}(y_2) - \text{gompit}(y_1)) / (t_2 - t_1))$ , where  $\text{gompit}(y) = -\ln(-\ln(y))$ ;  $t$  = time in degree days;  $y$  = proportion of disease. For  $x = \text{LPL}$  or  $\text{LPLPP}$ ,  $y = x / 10$ . For  $x = \text{NECLVS}$ ,  $y = x / 150$ .

<sup>c</sup> Rate for the logistic model is the apparent infection rate ( $r$ ) of Vanderplank (22), and for the Gompertz model (3) it is the parameter of  $k$ .

<sup>d</sup> Thinning dates.

between fungicide treatments (Table 3). Significant differences in the number of necrotic leaves per plant were observed between the sprayed and unsprayed treatments for the third and fourth planting dates but not the first and second planting dates.

**Field inoculations at San Jose Field Station.** The temperature and relative humidity during the first 48-hr period following the field inoculation fluctuated daily. Night temperatures were  $17 \pm 2$  C with day temperatures reaching 27 C; relative humidity during the first two nights was  $> 85\%$  RH for over 10 hr reaching a high of 95–98% RH each night. Daytime relative humidity reached a low of 55% RH both days. Daily maximum and minimum temperatures over the remaining 24 days of the observation period were  $24 \pm 4$  and  $12 \pm 2$  C, respectively. The highest daily maximum temperature during this period was 31 C. The accumulated degree days at 4, 7, 11, 15, and 26 days after inoculation were 39, 67, 109, 147, and 266, respectively.

The mature leaves on the mature plants were fully expanded when inoculated and had a mean leaf area of approximately 220 cm<sup>2</sup> and approximately 20 leaflets per leaf (Table 4). Immature leaves on mature plants had a mean of nine leaflets per leaf, which increased to 12 leaflets per leaf after 26 days, while mean leaf area increased from 2 to 95 cm<sup>2</sup> in the same period. The largest increase in leaf area and number of leaflets per leaf occurred on the immature leaves on immature plants; mean leaflets per leaf increased from 8 to 14 during the experimental period while leaf area increased from 23 to 138 cm<sup>2</sup>. No indication of infection (symptoms or sporulation) was evident on any leaflets 67 degree days after inoculation. Symptoms (light green-yellow lesions with sparse sporulation) were evident on 4.4, 1.2, and 1.3% of the leaflets from the mature leaves on mature plants, immature leaves

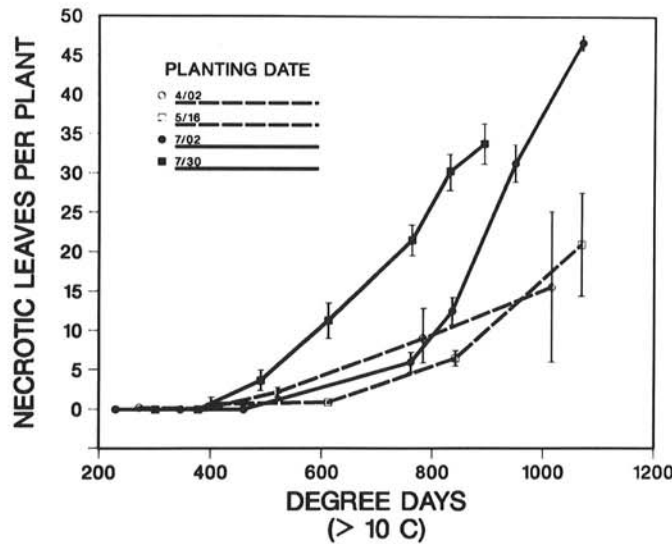


Fig. 7. Number of necrotic leaves (i.e., more than 80% of the leaflets per leaf were necrotic) per plant from the unsprayed Royal Flush treatment. Each data point is the mean of two to five plants from each of three replications. Vertical bars represent the standard deviation.

TABLE 2. Planting and harvest dates for tomatoes at the the West Side Field Station in 1985

Planting date	Harvest <sup>a</sup> date	Elapsed days	Elapsed <sup>b</sup> degree days	Mean <sup>c</sup> degree days
April 2	July 2	91	991	10.9
May 16	August 9	85	1,196	14.1
July 2	September 17	77	1,070	13.9
July 30	October 16	78	893	11.4

<sup>a</sup> Harvest date was picked when crop was at the "mature green" stage of development.

<sup>b</sup> Degree days per day calculated as follows:  $(\sum \text{temperature (C) per hr} / 24) - 10$ .

<sup>c</sup> Mean degree days accumulated per day.

mature plant, and immature leaves-immature plant treatments, respectively, after 109 degree days (11 days) (Fig. 8A). Symptoms developed rapidly over the next 4 days. After 147 degree days (15 days), 95, 56, and 68% of the leaflets in the mature leaves-mature plant, immature leaves-mature plant, and immature leaves-immature plant treatments, respectively, had developed symptoms. When disease incidence per leaf was corrected for host growth (i.e., the percentage of those leaflets that were present at the time of inoculation that developed symptoms) over 95% of the leaflets from all three treatments had developed symptoms 147 degree days (15 days) after inoculation (Fig. 8B).

**Spore samples.** The mean number of conidia collected above the undisturbed tomato canopy was less than 0.6 conidia per liter of air sampled for both the day and night samples on 23 July and 29 July.

TABLE 3. The effect of planting date and triadimefon on fresh fruit weight and leaf necrosis caused by *Leveillula taurica* on two tomato cultivars at West Side Field Station in 1985

Planting date	Cultivar	Fungicide <sup>w</sup> treatment	Necrotic leaves <sup>x</sup>	Fruit <sup>y</sup> weight (kg)
4/02	Royal Flush	Sprayed (S)	13.4 a <sup>z</sup>	3.99 a
	Royal Flush	Unsprayed (U)	15.1 a	4.60 a
	Jackpot	S	16.2 a	4.42 a
5/16	Royal Flush	S	20.9 b	4.53 a
	Royal Flush	U	21.7 b	4.49 a
	Jackpot	S	20.9 b	4.20 a
7/02	Royal Flush	S	25.1 c	4.37 a
	Royal Flush	U	43.2 e	4.12 a
	Jackpot	S	24.3 c	4.23 a
7/30	Royal Flush	S	15.1 a	4.38 a
	Royal Flush	U	36.3 d	3.96 a
	Jackpot	S	16.4 a	4.40 a
	Jackpot	U	36.6 d	4.41 a

<sup>w</sup> Treated plots received one application of triadimefon (Bayleton 50 WP) at 0.14 kg/ha approximately 45 days after transplanting.

<sup>x</sup> Number of necrotic leaves per plant at harvest. The number is the mean of five plants from each of three replications.

<sup>y</sup> Fresh fruit weight (in kilograms) per plant at harvest. The number is the mean of five plants from each of three replications.

<sup>z</sup> Variables followed by the same letter are not significantly different ( $P = 0.05$ ).

TABLE 4. Growth parameters of leaves artificially inoculated with conidia of *Leveillula taurica* at the San Jose Field Station

Treatment <sup>a</sup>		Elapsed days	Leaflets per leaf	Leaf area <sup>b</sup> per leaf	Necrotic leaflets per leaf
Leaves	Plants				
Mature	Mature	0	19.2	214.5	0.0
		4	21.1	216.1	0.0
		11	19.3	222.6	0.0
		15	20.9	220.0	1.0
		26	20.9	214.8	12.4
Immature	Mature	0	8.6	20.9	0.0
		4	11.2	46.5	0.0
		7	10.2	62.8	0.0
		11	9.9	79.1	0.0
		15	12.1	86.9	0.1
Immature	Immature	0	7.9	23.4	0.0
		4	10.2	58.0	0.0
		7	11.8	89.0	0.0
		11	12.6	126.9	0.0
		15	13.6	132.1	0.0
		26	13.8	138.2	4.6

<sup>a</sup> The cultivar used was Royal Flush. Mature leaves were fully expanded. Immature leaves were newly emerging terminal leaves. Mature plants had approximately 3,000 g of green fruit; immature plants had no fruit.

<sup>b</sup> Leaf area was estimated (6): Leaf area =  $(LW \times 0.615) - 5.15$ , where  $LW$  = leaf length  $\times$  leaf width (in centimeters).

However, a large number of spores were trapped above the disturbed canopy; mean day and night conidia counts were 106 and 119 per liter, respectively, on 23 July. On 29 July, day and night conidia counts were 117 and 128 conidia per liter, respectively.

## DISCUSSION

Powdery mildew of tomato has been considered to be a disease of mature plants (16,17). Our results, however, show that disease onset occurred at an earlier stage of crop development with each successive planting in field plots in both 1984 and 1985. Disease was observed on plants as early as the bloom and prebloom stage in the last planting date in both years. These data indicate that the tomato foliage is susceptible to infection at different stages of crop development. Furthermore, we have been able to show that both mature (fully expanded) and immature (newly emerging) leaves on mature and immature plants are susceptible to infection by *L. taurica* under field conditions under a relatively high inoculum pressure. More than 95% of inoculated leaflets, from all age treatments, became infected and developed symptoms within 147 degree days (15 days) after inoculation. We have also been able to consistently infect both young and old tomato leaves in the greenhouse (9). Latent period, or the time from infection to sporulation, was relatively long for *L. taurica*, and there was apparently no difference in latent period between the different age tissue. However, there could be quantitative differences in susceptibility, and the length of latent period, between mature and immature tissue under a lower inoculum pressure (1).

The stage of crop development at which disease onset occurred was apparently related to the available inoculum and not the physiological age of the crop. Tomatoes were planted or thinned as early as March in 1984 and 1985, and, although apparently susceptible tomato foliage was present in the field from March through June, no disease was detected until July. Disease onset, due to naturally occurring inoculum, was first observed in July of both years (Fig. 2). The importance of available inoculum is more easily recognized when disease progress is plotted as a function of the day of the year (Fig. 2) as opposed to degree days (Fig. 3). The epidemics in 1984 and 1985 can be viewed as single epidemics if allowance for plant size and sample variability are considered. All epidemics were initiated in a relatively narrow time frame (between day 186 and 240, Fig. 2).

In addition, disease onset apparently was not associated with any specific meteorological conditions. There was no significant rainfall during this period in either year. Day and night

temperatures were approximately  $32 \pm 5$  and  $15 \pm 7$  C, respectively, in both 1984 and 1985. Short periods of hot weather did occur from June through September with maximum daytime temperatures reaching 40–45 C. Relative humidity varied with location, cropping situation, and temperature, but, in general, relative humidity was >85% at night and <55% during the day.

Primary inoculum involved in initiating epidemics could have originated from any of several possible sources. A number of weed and crop hosts commonly found in Fresno County are susceptible to *L. taurica* and, thus, may have been the source of primary inoculum (5,7,9). However, inoculum may also have originated from surrounding tomato acreage. In Fresno County, where the West Side Field Station plot was located, approximately 30,000 ha of processing tomatoes are grown annually. Tomatoes are planted early (March–April), and approximately 70% are harvested by the end of July. These early plantings may allow the buildup of inoculum of *L. taurica* on a region-wide basis. At the beginning of the tomato harvest, powdery mildew can be found in most of these fields although disease incidence varies considerably (Correll, unpublished). We have shown that disturbing the tomato canopy, as would occur during mechanical harvesting operations, could liberate a large number of mildew conidia if plants had a high incidence of disease. A similar phenomenon has been described by Berger (2), in which celery harvesting operations substantially increase the number of airborne spores of *Cercospora apii*. Thus, tomato harvesting operations may be responsible for a large increase in airborne inoculum, which could result in a higher inoculum pressure in the vicinity of harvested fields.

The apparent infection rate for powdery mildew of tomato calculated by the logistic growth model was 0.0062–0.0137 (lesions per leaflet or lesions per leaflet per plant/degree day). Similar apparent infection rates have been observed in commercial tomato fields in California (6). The logistic growth model accounted for 60–99% of the variation of disease increase. In general, the Gompertz growth model gave a better statistical fit to disease data than did the logistic model. Disease progress was similar whether disease was measured by severity (lesions per leaflet or lesions per leaflet per plant) or incidence. Epidemics in both years slowed down considerably by the middle of October. Although the calculated infection rates are useful for interpreting disease progress, an inherent problem associated with the calculation of the infection rate is the limited number of observations that can be made after the relatively late onset of this disease.

Conidial germination, germ tube elongation, and leaf colonization are favored by cool to moderate temperatures (<30 C) under laboratory and greenhouse conditions (6,9), but in the field powdery mildew reached the highest disease incidence and severity during the warmest part of the summer. This paradox is somewhat difficult to explain. It may be attributable, in part, to the fact that temperatures in the canopy may be somewhat lower than ambient temperatures. Also, once a leaf has become infected, higher temperatures may accelerate symptom development (chlorotic and necrotic tissue) once a colony has become established (6). A similar situation apparently occurs with epidemics of *Pseudoperonospora cubensis* on cucumbers (4).

The assessment of disease development based on the number of necrotic leaves per plant is not as precise as counting individual lesions, but it does provide information on an important component of disease severity. Powdery mildew accelerates the death of leaves to a higher rate than what can be attributed to natural leaf senescence. A distinct increase in leaf necrosis was observed shortly after disease onset in the third and fourth planting dates in 1985 (Fig. 7).

Disease onset occurred relatively early, at fruit set and bloom stages, in the third and fourth planting date treatments, respectively, in 1985. Although inoculum pressure may have been higher in our experimental field plot (due to inoculum from within the plot from earlier planted tomatoes) than occurs in most commercial tomato fields, the disease levels observed were similar to those observed in many commercial tomato fields (6). At these disease levels, tomato powdery mildew did not cause any yield reduction of either of the cultivars Royal Flush or Jackpot relative

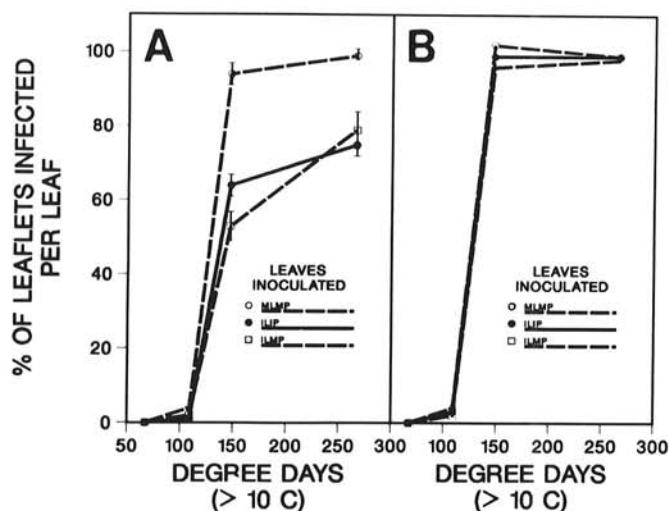


Fig. 8. Leaf inoculations of cultivar Royal Flush at the San Jose Field Station. MLMP = mature leaves, mature plants, ILIP immature leaves, immature plants, ILMP = immature leaves, mature plants. A, Percent of leaflets infected per leaf for different age leaves. B, Percent of leaflets infected per leaf when corrected for leaf growth. Each data point is the mean of three leaves from each of 6–10 replications.

to the triadimefon treatments. Powdery mildew did, however, cause a significant increase in defoliation of the canopy in the third and fourth planting dates. Powdery mildew caused a 75–125% increase in the number of necrotic leaves over triadimefon controls. This degree of defoliation represented 30–40% of the total tomato canopy. We have observed a similar degree of defoliation in commercial fields where disease severity was often higher (6). In the absence of disease, defoliation of plants due to natural leaf senescence at harvest represents 5–10% of the canopy.

Based on the early disease development in the third and fourth planting date treatments, infected tomato tissue can substantially increase the inoculum pressure on tomatoes planted later in the season. The observed late development of powdery mildew in commercially grown tomatoes (6) suggests that inoculum is limiting. Consequently, tomatoes planted adjacent to a mature tomato crop would be at a particular risk of developing a high level of powdery mildew.

We have not been able to demonstrate any significant reduction in fresh fruit weight per plant of fresh market tomatoes due to powdery mildew. These data are in contrast to what has been reported by Jones and Thomson (12) in Utah, where yield reductions of up to 40% have been reported. Processing tomatoes were used in their study that were harvested at the "mature red" stage of development (approximately 82–95 days after transplanting), so a direct comparison of physiological development is not possible. However, such a large reduction in yield is surprising in light of the relatively low disease severity (lesions per plant) they reported (fewer than 500 lesions per plant); tomatoes in the third and fourth planting dates of the current study had a mean of 4,477 and 1,170 lesions per plant, respectively, at harvest. We have also observed similarly high disease severity values in commercial fresh market tomato fields in the San Joaquin Valley of California with no effect on yield (6).

The fact that powdery mildew can substantially increase defoliation may predispose fruit to insect damage, sunburning, and/or reduced quality. However, we did not observe any such damage in our fresh market tomato field plots. As previously mentioned, processing tomatoes are generally harvested at a much later physiological age than are fresh market tomatoes. Thus, a high level of powdery mildew could have a substantial impact on the fruit quality of processing tomatoes.

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