Occurrence of Fire Blight of Pears in Relation to Weather and Epiphytic Populations of Erwinia amylovora

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ABSTRACT

Applications of bactericides are often made in California pear orchards long before *Erwinia amylovora* is present in flowers, and apparently before such measures are warranted. Monitoring pear flowers for the occurrence and population of *E. amylovora* in 1973, revealed that the bacterium did not colonize flowers during early or full-bloom periods, but subsequently colonized secondary (rattail) flowers in most orchards. In some orchards as many as 18 applications of bactericides were made, even though bacteria were not detected in the flowers. *E. amylovora* multiplied in healthy pear flowers, and was detected in flowers 14 days prior to disease occurrence in some orchards. Although over 50% of the healthy flowers in some orchards were infested with approx. 10^6 cells of *E. amylovora* per flower, subsequent disease incidence was only 1-3 infections per tree. Where populations of fire-blight bacteria in healthy blossoms were low or absent, the incidence of disease was of no economic importance. Precipitation was not requisite for colonization of flowers by *E. amylovora,* nor for disease development. Fire blight bacteria were on the surface and interior of overwintered cankers, but were not detected in healthy buds from healthy or diseased trees. *Erwinia amylovora* was detected on leaves, but only after fire blight was common in orchards. Populations of *E. amylovora* were present on flowers of the alternate hosts, *Malus pumila* and *Pyracantha* spp., even though the disease was not observed. The susceptibility of pear flowers to disease varied from year to year, during the season, and among flowers of a cluster. Accordingly, the capacity of *E. amylovora* to multiply in flowers varied greatly regardless of inoculum concentration. Monitoring epiphytic populations of *E. amylovora* is now used to improve the timing of bactericide applications for fire blight control.

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Additional key words: epidemiology, forecasting, resistance.

The occurrence of fire blight epiphytotics is sporadic and largely unpredictable. Forecasting epiphytotics has been attempted by several investigators who made correlations between the climatic factors thought to influence growth of the bacterium and subsequent disease incidence. Mills (8) made a statistical comparison of 37 years of New York weather data in relation to fire blight incidence, and reported that control was more effective if the following criteria were met before application of streptomycin during bloom: the maximum temperature exceeded 18 C, precipitation was forecast and the relative humidity (RH) was over 70%. Even though the inoculum level was not known, this method was based on the supposition that it was safer to apply chemicals than to have a severe outbreak of fire blight. Powell (10, 11) advanced the concept that pre-bloom environmental conditions influenced multiplication of the pathogen, and the necessity to apply a chemical control could be predicted by observing climatic changes. He suggested that flower-blight control measures were precluded when conditions during pre-bloom were unfavorable for bacterial growth.

Although Mills' and Powell's predictive approaches were ably conceived, disease prediction based on weather data alone has generally been of limited value because the interaction of inoculum potential, host susceptibility, and environment in epiphytotic development is complex. Furthermore, methods to monitor the population of *E. amylovora* as related to environmental conditions have not been available until recently (7).

Without methodology to accurately predict disease occurrence, many pear growers in California have routinely applied streptomycin or copper compounds to trees, beginning at 5% bloom with additional applications every 5 days until at least 30 days after full bloom (1). The time period involved, including the secondary (rattail) bloom, may last up to 3 months, with as many as 18 applications. This is an expensive operation costing approx. $6 per acre each time the chemical is applied. In some years, the expense was worthwhile and growers obtained significant financial benefits compared to those who did not apply bactericides. In other years or locations, however, fire blight was insignificant even with the most careless grower. Therefore, the efficacy of control would be improved, as well as providing a financial saving to the grower, if sprays were applied only when necessary.

We examined the possibility of improving the timing of bactericide applications, and predicting the occurrence of disease, by monitoring epiphytic populations of *E. amylovora* on pear flowers throughout the blossoming period, recording environmental conditions, and investigating differences in host susceptibility.

MATERIALS AND METHODS.—A selective medium with sorbitol (MSS) as the carbon source (7) and 0.167 g/liter cobaltous chloride as a reducing agent was used for isolation of *E. amylovora* from flowers and other plant parts. Although the medium was not exclusively selective for *E. amylovora,* most colonies were identified directly on the medium because of distinctive colony

characteristics. Identification of *E. amylovora* by use of the medium alone was 95% reliable, but representative colony types were selected for verification. Single colonies (1 mm in diameter) were transferred to 0.02 ml of sterile distilled water (SDW) and a loopful of the suspension was then streaked onto King's B (5) medium to further examine colony color and appearance. A 0.01-ml aliquot of the suspension was also injected into the intercellular spaces of leaves of *Nicotiana tabacum* L. 'Turkish' for the hypersensitive reaction (HR) (6). Pathogenicity was determined by inoculating excised green Bartlett pear fruits with a turbid drop of the culture in question. Colonies of *E. amylovora* could be identified on MSS within 48-72 hours after the flowers were sampled; verification by the HR or pathogenicity tests required an additional 8 to 16 or 48 to 72 hours, respectively.

Mass flower sampling.—To determine the seasonal occurrence of *E. amylovora* as an epiphyte, 15 Bartlett pear orchards in Butte, Contra Costa, El Dorado, Glenn, Lake, Mendocino, Napa, Sacramento, and Yolo counties were monitored regularly during the spring and summer. These orchards are located in the different climatological regions of the major pear-producing areas of California. Two-hundred flowers were randomly selected from approximately 2-hectare (5-acre) areas about every 4 days, beginning at 1% bloom. Flowers were collected in plastic bags, transported in an ice chest and processed within 3 hours. The sample size was reduced during secondary (rattail) bloom because of the limited number

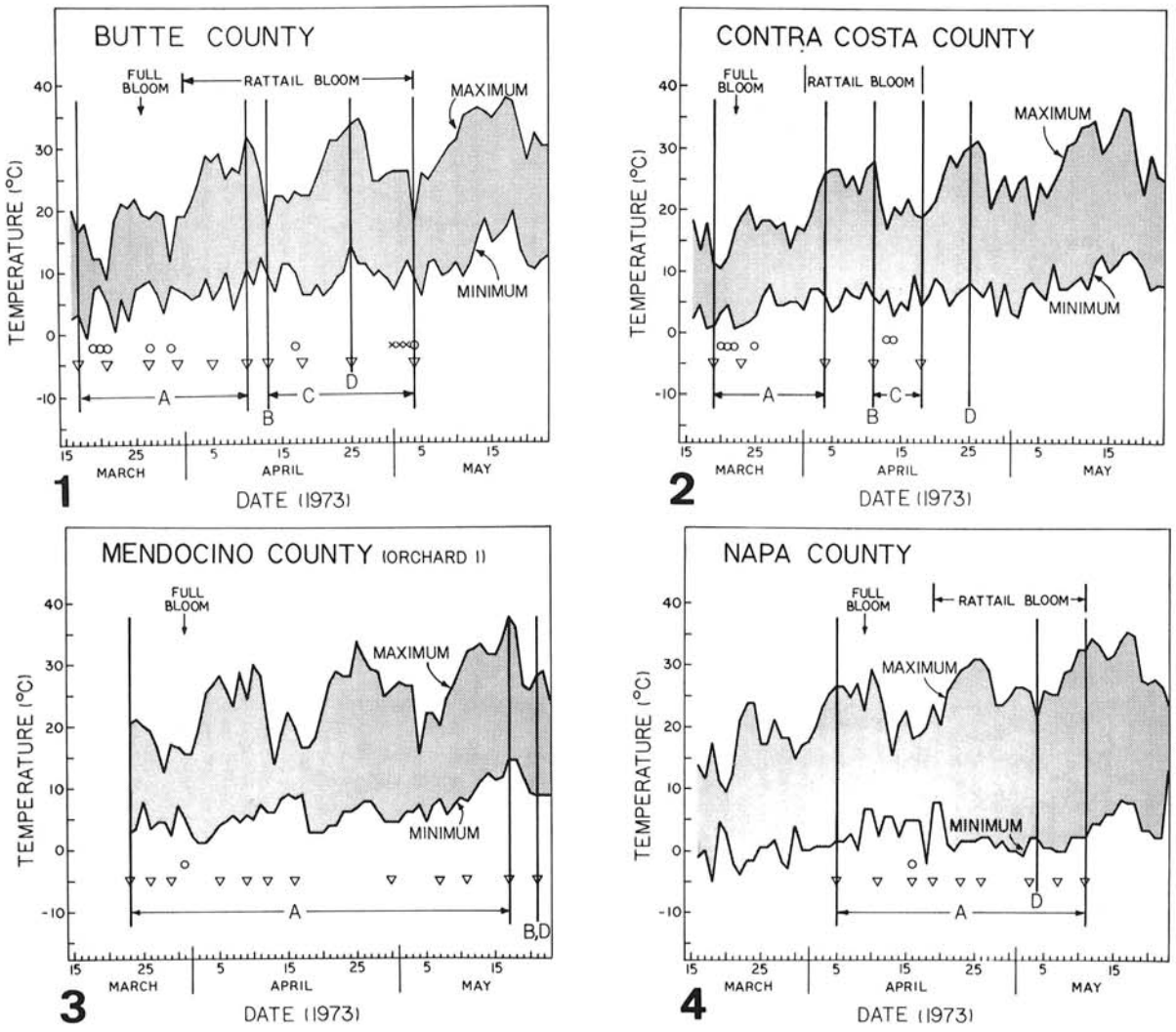

Fig. 1-4. Occurrence of epiphytic populations of *Erwinia amylovora* in healthy Bartlett pear flowers and subsequent disease occurrence. *Erwinia amylovora* bacteria were not found (A) in flowers until rattail bloom (B). Populations in subsequent samples remained high (C) until infections were first noted (D). Precipitation = 0; irrigation = x; monitoring dates = ▽. **1)** Butte County orchard. 3.3×10^5 bacteria/flower detected on 13 April (B) and the first infections noted on 25 April (D) with 3.2 infections/tree. **2)** Contra Costa County orchard. 5.0×10^4 bacteria/flower detected on 11 April (B) and the first disease noted on 25 April (D) with 1.4 infections/tree. **3)** Mendocino County orchard No. 1. Fifteen bacteria per flower detected on 21 May and only one infection in 8 acres found on 21 May. **4)** Napa County orchard. Bacteria were not detected and only 20 infections in 9 acres were found after 4 May.

of flowers. The flowers were washed in 0.5 ml of tap water (TW) per flower and aliquots of 0.1 ml of the undiluted wash and a 10^{-2} dilution were spread evenly on MSS with a bent glass rod and incubated at 29 C. Identification of *E. amylovora* colonies was done at the Berkeley laboratory.

Individual flower sampling.—After the mass flower sample revealed that *E. amylovora* was present in an orchard, individual flowers were then sampled to determine the percent flowers that were infested and the number of bacterial cells per flower. Individual healthy flowers were collected by inserting a fully opened flower into a sterile test tube and detaching it from the tree by sliding the plastic cap over the tube. Tubes were kept at 15-20 C and returned to the laboratory for processing. The flowers sampled in this study were taken from areas where bactericides had not been applied. Each flower was washed and shaken on a rotary tube shaker for 20 seconds in 10 ml TW; a 0.01-ml drop of the flower wash was then dispensed to plates of MSS with an Eppendorf pipette (Brinkman Instruments, New York) and spread with a bent glass rod.

Leaf, bud, terminal shoot and canker samples.—The surfaces of overwintered fire blight cankers collected 3 weeks prior to bloom (15 February) from commercial pear orchards were swabbed with a moist cotton Q-tip® (Johnson and Johnson, New Brunswick, N. J.) and the Q-tip streaked on MSS. These cankers were then treated for 5 minutes in 0.5% sodium hypochlorite and a piece of the internal bark tissue near the canker margin was ground in TW and streaked on MSS. Fire blight cankers were similarly sampled in January, 1974, during a rainstorm, and also after 10 days of dry weather.

Apparently healthy buds, terminal shoots, and leaves from both healthy and diseased trees were sampled during 1971-1973 to determine the extent of bacterial dissemination, and possible origins of inoculum. The terminal 3-cm portion of shoots were excised from the branch and individually washed for 1 minute in 1 ml DW. Buds were treated for 3 minutes in 0.5% sodium hypochlorite while still attached to a 3-cm piece of twig, rinsed in SDW, and allowed to dry. The buds were then excised from the twig and ground with mortar and pestle in 1.0 ml SDW. In April, 1973, healthy leaves from trees with and without disease were washed and vibrated for 1 minute in 10 ml of SDW. Two-hundred healthy flowers were collected at the time leaf and bud samples were taken to compare populations of *E. amylovora* in flowers with populations from leaf and bud samples. These flowers were treated as described for mass flower sampling. Aliquots of 0.1 ml from each sample described above were spread with a bent glass rod on MSS.

Malus pumila Mill. and Pyracantha spp. Roem. samples.—*Malus pumila* (apple) and *Pyracantha* spp. were monitored for epiphytic populations of *E. amylovora* on flowers because they are common hosts of fire blight, and may serve as inoculum sources. Flowers from apple and pear trees in a mixed orchard were concurrently sampled using the techniques previously described for sampling individual flowers.

Flowers of *Pyracantha* plants from seven climatically distinct locations were sampled for epiphytic populations of *E. amylovora* between 11 and 29 May. Clusters of approximately 50 flowers were washed and vibrated in 20 ml of DW in 125-ml flasks for 15 seconds, 0.1-ml aliquots were spread on MSS, and the plates were incubated at 29 C. The number of *E. amylovora* colonies was noted and representative colonies were verified with the HR or pathogenicity tests. Some of the *Pyracantha* plants were within a few meters of pear orchards, while others were several kilometers from commercial pear orchards.

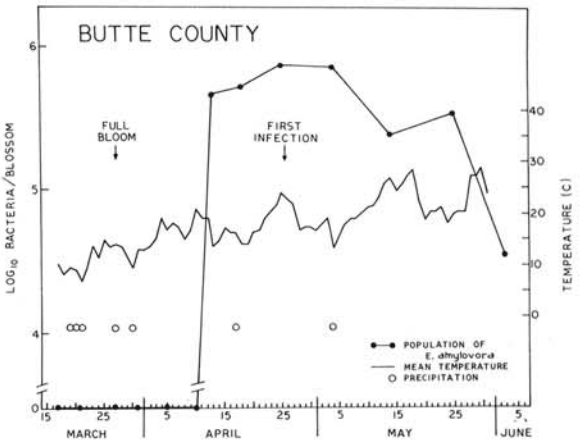

Fig. 5. Relationship between climatological changes and the epiphytic population of *Erwinia amylovora* on Bartlett pear flowers in Butte County.

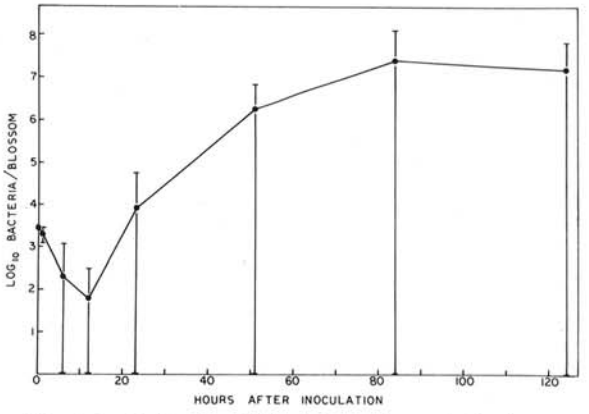

Fig. 6. Multiplication of *Erwinia amylovora* in Bartlett pear flowers. Each point is a population mean of the bacterium in 10 flowers. The vertical lines indicate the population range in 10 flowers.

Susceptibility of pear flowers.—Bartlett pear twigs with closed flower buds were collected in February of 1972 and 1973 and either forced into bloom or stored at 5 C for use later in the year. Flowers were forced by placing cuttings with one to three flower buds in 250 ml of water at 26 C. After emergence, the open flowers were inoculated on the receptacle with suspensions of *E. amylovora* ranging from 4 to 10^4 cells per flower in 0.01 ml sterile TW. Fresh-cut twigs with flowers from the field were also used when available. The inoculated flower clusters were incubated at 26 C at 90% RH and observed after 5 days for oozing of bacteria or other typical symptoms and signs of the disease.

TABLE 1. Susceptibility of inoculated Bartlett pear flowers to infection by *Erwinia amylovora*[a]

Sample date	Isolate	Inoculum concentration (cells/flower)	Diseased flowers (no.)
2-1-72	FB-6	45	0/25
	FB-6	2,600	0/25
	FB-42	40	0/25
	FB-42	362	0/25
2-9-72	FB-6	1,492	3/20
	FB-6	10,000	4/20
	FB-42	1,042	2/20
	FB-42	10,000	4/20
3-8-72	FB-2	500	5/30
	FB-2	2,000	6/30
	FB-6	500	8/30
	FB-6	1,000	16/30
3-13-72	FB-42	150	0/10
	FB-42	2,000	15/30
3-15-72	FB9+26	6,000	0/25
3-10-73	FB-26	293	0/50
3-15-73	FB-26	392	0/50
3-16-73	FB-26	312	0/50
3-28-73	FB-26	3,000	5/133
4-17-73	FB-26	150	28/57
3-6-74	FB-58	2,500	1/93
3-23-74	FB-58	2,500	0/128
3-27-74	FB-58	2,500	0/190

[a]Clusters of flowers on twigs placed in water were inoculated with varying dosages of *E. amylovora* in 0.01 ml tap water and incubated at 26 C and at approx. 90% RH.

TABLE 2. Number of Bartlett pear flowers in a cluster in which *Erwinia amylovora* was not recovered after inoculation[a]

Cluster	Number flowers without bacteria/number flowers sampled
1	0/6
2	3/6
3	5/6
4	2/6
5	0/6
6	3/6
7	1/6
8	1/6
9	3/6
10	4/6

[a]*Erwinia amylovora* at 2.7×10^3 cells/0.01 ml of tap water was placed on the receptacle of each flower.

The growth characteristics of *E. amylovora* in pear flowers was determined by inoculating forced flowers (collected in February 1973) with 2.6×10^3 cells of *E. amylovora* per flower in 0.01 ml sterile TW. Changes in the population of *E. amylovora* were determined by removing flowers from the clusters up to 96 hours after inoculation, washing them individually in 10 ml TW and plating 0.1 ml of the wash on plates of MSS. Remaining flowers were observed after 5 days for oozing or other typical fire blight symptoms.

RESULTS.—*Seasonal occurrence of E. amylovora as an epiphyte.*—The mass flower sampling revealed that *E. amylovora* was present in healthy pear flowers up to 14 days prior to finding any disease in orchards (Fig. 1, 2). The bacteria were not found in flowers early in the season nor during full bloom in any orchard, but were detected in most orchards late in the season during secondary (rattail) bloom. After the initial isolation of *E. amylovora* from flowers, bacteria were detected in subsequent samples until the end of flowering even though maximum temperatures reached 37 C (Fig. 1). Every isolate tested of *E. amylovora* from healthy flowers was pathogenic on excised pear fruit and HR-positive.

The flowers in the Butte orchard were not infested with *E. amylovora* until 13 April, when only rattail flowers were present. An average of 3.3×10^5 bacteria per flower were detected (Fig. 1, 5) and disease was observed 12 days later with an incidence of 3.2 strikes per tree. (A strike is defined as a blighted twig emanating from an infected flower or leaf.) Similar results were obtained in the Contra Costa orchard, with the bacteria first detected on 11 April (5.0×10^4 cells/flower) and disease occurring on 25 April, with an incidence of 1.4 strikes per tree (Fig. 2).

In several orchards, the population of bacteria was less than 10^3 per flower, and the incidence of disease was either none or insignificant (Fig. 3, 4). For example, in the Mendocino No. 1 orchard the population of *E. amylovora* never exceeded 15 cells per flower, and only one strike was located during the summer in the 3.24-hectare (8-acre) orchard (Fig. 3). However, when populations averaged 10^5 to 10^6 per flower, the incidence of disease was usually more than one strike per tree (Fig. 1, 2).

The establishment of a relationship between temperature and primary colonization of flowers was not attempted since relatively few orchards and only one year's data was included in the tests. An evaluation of the relationship of temperature to colonization will not be made until data are accumulated for three seasons to account for variation.

The first detection of *E. amylovora* in flowers was not always associated with precipitation. In the Butte County orchard (Fig. 1), the bacterium was detected 4 days after rain while in the Mendocino No. 1 orchard (Fig. 3), the closest rain occurred 52 days preceding the first detection. Initial disease observations were also not correlated with precipitation patterns. However, dew frequently formed on trees during periods of high humidity and cool nights.

Epiphytic populations on individual flowers.—Whereas mass flower sampling of *E. amylovora* in the Butte and Contra Costa orchards revealed 7.6×10^5 and 3.2×10^5 cells per flower, respectively, individual flowers were infested with an average population of 1.5×10^6 cells per infested flower in the Butte orchard and 1.2×10^6 cells per infested flower in the Contra Costa orchard. Fifty-two percent of the flowers collected at the Butte orchard were infested, whereas 79% of those from the Contra Costa orchard had epiphytic populations of *E. amylovora*. The disease incidence at the time individual flowers were collected was 6.4 and 3.2 strikes per tree at the Butte and Contra Costa orchards, respectively.

Epiphytic populations on cankers, leaves, buds, and terminal shoots.—In February, 1973, *E. amylovora* was

found on the surface of 33% (10/30) of the cankers prior to any evidence of ooze, and was also isolated from the interior of 57% (17/30) of the same cankers. The pathogen was detected on both the surface and interior of 15% (3/20) of the cankers collected during a rainstorm in 1974. However, *E. amylovora* was isolated from the exterior of only 3% (2/67), and the interior of 13% (8/67), of the cankers sampled after 10 days of dry weather. *Erwinia amylovora* was not detected in 262 healthy buds from trees with disease, nor from 587 buds from trees showing no evidence of disease during the years 1971-1973. It also was not found in 1973 in 90 surface-sterilized buds taken from healthy branches on trees with a disease incidence of less than one strike per tree. Terminal shoots of 48 branches were also found to be free of *E. amylovora*. The pathogen also was not detected on the surface of 256 leaves taken from healthy trees or trees with less than five strikes per tree. The mass flower monitoring of the same trees from which leaves and buds were taken in spring, 1973, showed that *E. amylovora* was present in flowers at populations greater than 3.5×10^5 cells per flower. *Erwinia amylovora* was frequently detected later in the season on healthy leaves from orchards with a disease incidence greater than five strikes per tree.

Epiphytic populations on alternate hosts.—Apple flowers, but not pear flowers, contained epiphytic populations of *E. amylovora* on April 18 in an orchard with both apple and pear trees. Populations of the bacterium ranged from 3.0×10^3 to 1.6×10^6 cells per apple flower and 80% of the flowers were infested. Eight days later, on 26 April, the mass flower sampling revealed a low epiphytic population of *E. amylovora* (2.5×10^3 cells/flower) in pear flowers from the same orchard. The disease was not observed in any of the apple trees and only one strike was found on the pear trees.

Pyracantha plants from all seven sampled locations were colonized with epiphytic populations of *E. amylovora* in flowers, even though no disease was observed on any *Pyracantha* plants in the areas sampled during the 1973 season. Populations of *E. amylovora* in the 50 flower samples averaged from 2.0×10^4 to 5.7×10^5 cells per flower and were frequently over 10^5 cells per individual flower. Some of the *Pyracantha* plants infested with *E. amylovora* were within a few meters of pear orchards, but the pathogen was also present on plants in residential areas and on the Berkeley campus which are several kilometers from commercial pear orchards.

Susceptibility of pear flowers.—Pear flowers in general appeared to be fairly resistant to infection by *E. amylovora* when tested with inocula containing cell concentrations up to 6×10^3 cells per flower (Table 1). There were, however, considerable differences in the susceptibility of flowers collected from 1972, 1973, and 1974 with no obvious reason to account for the variations. Flowers even varied in susceptibility during the seasons. An interesting relationship existed between flower susceptibility and the occurrence of the disease in the field in 1973. The flowers during the season were resistant to fire blight infection until rattail flowers developed. This also coincided with the first observation of disease in the field.

Experiments following the multiplication rates of *E. amylovora* in flowers after inoculation showed that the bacterium did not multiply in many flowers. The variability even existed among flowers of a cluster (Table 2). This experiment was repeated three times with similar results with flowers from different orchards. Multiplication occurred in every flower in a few clusters. However, the bacterium did not survive in 37% of the flowers, suggesting the possibility that a toxic principle may exist. In flowers where *E. amylovora* multiplied, there was an initial decrease in cell count or lag phase followed by a rapid increase thereafter (Fig. 6). Even when populations reached levels of over 10^7 cells per flower (40% of the inoculated flowers), only 1/60 flowers showed symptoms of disease. It was common throughout the experiments to note drops of water turbid with bacteria in the receptacles of apparently healthy flowers.

DISCUSSION.—The fire blight monitoring program demonstrated that during the spring of 1973, bactericides were generally applied far in advance, and perhaps unnecessarily, before detection of epiphytic populations of *E. amylovora* in flowers. In some orchards, the bacteria were not detected in flowers throughout the entire season. However, in most orchards the bacterium was detected in flowers from 3 days to 2 weeks in advance of disease appearance. The only exceptions were in several orchards where the epiphytic population was very low or below the level of detection of our assay. Accordingly, the incidence of disease in these orchards was none or of minimal economic importance (Fig. 3, 4).

Our data suggest that bactericides might be applied immediately after the pathogen is detected to limit further epiphytic multiplication which can occur rapidly under favorable conditions (Fig. 5) and more importantly to protect newly-opened flowers from colonization. This aspect of protecting newly-opened flowers is critical since the bactericides now available are not eradicants, and are most effective when applied prior to infection. Furthermore, bactericides might have to be applied more frequently than the currently recommended 5-day intervals, considering the rapid rate of multiplication and dissemination of fire blight bacteria under optimal conditions.

The susceptibility of flowers is highly erratic, varying from year to year, during the season, and even among flowers of a cluster. That inoculated flowers did not become diseased by subjecting them to various temperature and humidity regimes in exploratory research, suggested that the inherent susceptibility of flowers transcends the importance of weather conditions in limiting infections. The susceptibility of flowers to infection may be influenced by biotic and abiotic factors during the previous year, the dormant period, as well as the current growing season.

There was also considerable variability in the capacity of *E. amylovora* to multiply in flowers. Even when multiplication occurred, disease was infrequent. The ability of *E. amylovora* to multiply in many flowers, and the rapid death of inoculated cells raises the question of whether or not a toxic principle may be present in the flower under certain conditions. The susceptibility of pear flowers to *E. amylovora* in California is probably "the exception rather than the rule" since laboratory and field studies indicate that relatively few flowers become diseased, even when inoculum is present and

environmental conditions appear favorable for infection. For example, in 1973, over 50% of the rattail flowers in the Butte and Contra Costa orchards were infested with populations of 10^6 cells per flower, yet only one-to-three strikes occurred per tree. In 1973, it was noteworthy that laboratory tests showed that primary flowers in one location were resistant to *E. amylovora,* whereas rattail flowers were susceptible; rattail flowers became infected in the field coincident with flower infection in the laboratory.

Several investigators have reported that rain is not necessary for fire-blight infection (12, 13). Our monitoring data of 1973 indicate that dissemination of *E. amylovora* may also occur without rain. Accordingly, the first detection of *E. amylovora* in flowers occurred as long as 52, and as soon as 4 days after rain storms. However, there is still no question that rain and dew enhance both dissemination and infection.

The effect of temperature on colonization and disease occurrence is also inconclusive from this year's data. However, there were enough examples to indicate that Mills' (8) criteria that disease often follows a maximum temperature of 18 C and precipitation does not apply in California; maximum temperatures of 18 C concurrent with precipitation frequently occurred very early in the flowering period, but neither were flowers colonized, nor did disease occur, until several weeks later.

During some years, *E. amylovora* may survive in a large percentage of cankers (e.g. 50%), while in other years only a small proportion of the cankers (e.g. 3%) may be sources of inoculum. We also noted that *E. amylovora* was present on the surface of overwintered cankers and that rainfall increased surface contamination by *E. amylovora.* Investigators have suggested that the fire-blight pathogen overwinters on hosts other than pear or apple, and that the primary inoculum originates from these sources (3, 4). However, our research indicates that primary inoculum is readily available on overwintering pear cankers although alternate hosts may also serve as additional sources. *Pyracantha,* which begins flowering in late March, could be a significant source of inoculum from the middle of the pear bloom period. By late May, every *Pyracantha* plant sampled had high populations of *E. amylovora* on flowers and young fruits.

Healthy buds have also been suggested as an overwintering site for fire-blight bacteria in several geographical areas (2, 9), yet *E. amylovora* was not detected in any pear buds during the winters or early springs of 1971 to 1973 in California orchards. However, in the spring of 1974 in California, *E. amylovora* was isolated from one of 120 apparently healthy buds taken within 30 cm of a fire blight canker (T. J. Burr, *personal communication*). Climatic differences between regions presumably influence the capacity of *E. amylovora* to colonize buds. Certain environmental conditions are apparently important for survival in healthy buds, and such conditions may not frequently occur in the pear growing regions of California.

Erwinia amylovora has been reported as an epiphyte on leaves (7) and other plant parts. However, we found that the fire-blight bacteria were present on shoot tips and pear leaves only after disease incidence in orchards was significant. The ability of these bacteria to survive as epiphytes on plant parts was not examined.

Control recommendations and forecasts of disease occurrence can now be made by the determination of populations of *E. amylovora* on pear flowers combined with a careful consideration of environmental conditions, and perhaps the inherent susceptibility of plant tissue. Ultimately, the time-consuming and relatively expensive monitoring of flowers might be eliminated; disease prediction and control guidelines could then be based upon a computer simulation of fire blight epiphytotics.

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