

Eutypa Dieback of Sweet Cherry and Occurrence of *Eutypa lata* Perithecia in the Central Valley of California

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ABSTRACT

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Eutypa lata was recovered from cankers on sweet cherry trees in three California counties. *E. lata* ascospores or mycelial isolates from these trees were inoculated into wounded sweet cherry branches or stems, grapevine stems, and apricot branches. With mycelial inoculum, cankers developed on 19 of 30 cherry branches. Inoculation date (April vs. November) did not affect the incidence of cankers or mean canker length, but did affect the extent of xylem necrosis. With ascospore inoculum, infection of pruning wounds ranged from 0 to 100%, depending on pruning date and the time between pruning and inoculation. Of six *E. lata* isolates from three different hosts (sweet cherry, grape, apricot), four caused cankers and/or dieback of potted cherry trees within 14 mo after mycelial inoculations. Canker incidence ranged from 50 to 100% among the four isolates. An isolate from sweet cherry was as virulent to grape and apricot as isolates originating from each of these hosts. The teleomorph of *E. lata* was discovered in several locations in the Central Valley of California, a semiarid region previously believed to be too dry to support formation of the teleomorph. The abundance of this stage of the fungus was determined to be sufficient to provide a large amount of inoculum for infection of grapevines in the Central Valley. A large proportion of the newly discovered perithecia was on sweet cherry; smaller proportions were on grape and apricot. Sprinkler irrigation of orchards and vineyards apparently provided sufficient moisture for development of perithecia.

Additional keywords: disease spread, *Eutypa armeniaca*, host range

Eutypa lata (Pers:Fr.) Tul. & C. Tul. (= *E. armeniaca* Hansf. & M.V. Carter) causes dieback diseases of grape (*Vitis* L. spp.) and apricot (*Prunus armeniaca* L.) (4,15). This fungus also has been reported from other *Prunus* species, including sweet cherry (*Prunus avium* (L.) L.) and at least 50 additional genera of woody dicots (6), usually without tests of pathogenicity. In North America, *E. lata* has been reported only from grape, apricot, chokecherry (*Prunus virginiana* L. var. *demissa* (Nutt.) Torr.), apple (*Malus domestica* Borkh.), and *Ceanothus* L. spp. (6). An isolate of *E. lata* from apricot was reported to be "weakly pathogenic" to sweet cherry (9).

E. lata is usually placed in the family Diatrypaceae, class Pyrenomycetes, of the Ascomycotina. Its teleomorph has perithecia formed in a black, irregular stroma, comprised of both fungal and host tissue, embedded in decorticated wood (11). Isolates causing dieback of *Prunus* spp. and grape were originally classified as *E. armeniaca* Hansf. & M.V. Carter, and one criterion for their identification as such was pathogenicity

to apricot (6,11). There is no evidence for host specificity in this fungus, although there appears to be variation in virulence among isolates (4,5,10,24).

Eutypa dieback of grape is very common in northern California. In some

vineyards in California's Central Valley, over 90% of the vines are infected (8,18). The ascospores of the fungus are the only proven naturally occurring infectious propagules of the disease (3); they infect the hosts through pruning wounds made during the dormant season. Conidia generally are not believed to germinate or infect, but there are conflicting reports (1,4,13). The teleomorph develops very slowly and is not generally found in California in areas with less than 500 mm of annual precipitation (25). Annual precipitation in grape growing areas in the Central Valley of California ranges from 140 to 445 mm, and the teleomorph of the fungus was not reported from commercial plantings in the Central Valley until 1991 (19). The teleomorph was reported from a single, sprinkler-irrigated, backyard grapevine in the Central Valley in 1983 (10). The teleomorph is more common in the coastal and Bay areas of the state, and it has been suggested that these areas provide the majority of inoculum for the Central Valley (23,25).

In 1990, a fungus was collected from a decorticated canker on a sweet cherry tree in a sprinkler-irrigated commercial

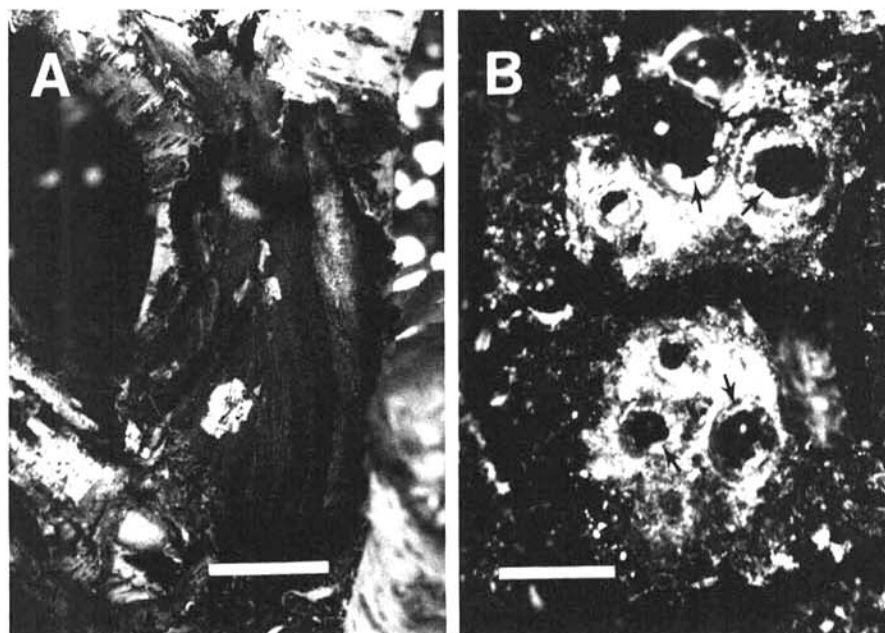


Fig. 1. Stromata of *Eutypa lata* on sweet cherry. (A) Stromata on a large canker. Bar represents 10 cm. (B) Perithecia exposed by tangential section through stroma. Arrows denote perithecial cavities. Bar represents 0.5 cm.

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orchard in San Joaquin County, California, in the Central Valley (19). This fungus had formed a stroma typical of *E. lata*, with embedded perithecia in a random distribution (Fig. 1). The asci were spindle-shaped, long-stipitate, with apical invaginations. Ascospores were allantoid, subhyaline (golden brown in mass), and 9–10 μ m long. The fungus was identified as *E. armeniaca*, according to the keys of Müller and von Arx (17) and Glawe and Rogers (11). *E. armeniaca* is conspecific with *E. lata* (26,27). Occurrence of perithecia was confirmed on 13 of 1,000 trees inspected in this orchard. An additional 35 trees had cankers and rudimentary stromata, but no perithecia. This discovery indicated that *E. lata* may cause cankers of sweet cherries in California, and that *E. lata* stromata within the Central Valley may be important inoculum sources for *Eutypa* dieback of grape in the area.

The objectives of this research were to determine the pathogenicity of *E. lata* from cherry to grape and cherry, to survey for sources of *E. lata* ascospores in orchards and vineyards in the Central Valley, and to investigate the occurrence of perithecia on hosts previously unreported in North America. Preliminary results have been reported (19,20).

MATERIALS AND METHODS

Pathogenicity of *E. lata* from cherry.

Stromata collected from sweet cherry trees in San Joaquin County were soaked in water, and the discharging ascospores were collected on water agar. A culture (CA01) derived from a droplet of these ascospores was used for most inoculations.

In a preliminary experiment, inoculations were performed on 28 June 1990. Twenty first-year cuttings of grape, cv. Chenin blanc, planted in pots in a lathhouse (February 1990) were used in the experiment. A circular plug of bark was removed with a cork borer (8 mm diameter), and a plug of mycelium from the margin of a 7–10 day old culture was inserted into the wound on 10 of the cuttings. The inoculation site was wrapped in laboratory film for 2 wk after inoculation. A sterile plug of potato-dextrose agar (PDA) was placed into the wound on the other 10 cuttings. The same procedure was followed using 20 branches (10 inoculated, 10 noninoculated) each of sweet cherry (cv. Bing) and apricot (cv. Blenheim) trees located in an experimental orchard near Davis, California. The branches were the previous year's growth (1.5–3 cm diameter). An additional 10 apricot branches were inoculated with an *E. lata* isolate from a cankered apricot tree in Contra Costa County, California. The inoculations were left to develop for 2 mo.

A second experiment was conducted involving only grape cuttings. Inoculations were performed on potted dormant

cuttings in February 1991, with 20 cuttings for each of two isolates (CA01 and GC01, a canker isolate from grape [Sacramento County, California]) and a control. The inoculation procedure was

as described above. The cuttings were left for 9 mo in a lathhouse. An additional 10 cuttings were inoculated with the cherry isolate and left until dieback symptoms appeared.

Table 1. Mean lengths of xylem necrosis and frequency of *Eutypa lata* reisolation from sweet cherry (cv. Bing) and apricot (cv. Blenheim) branches and grape (cv. Chenin blanc) cuttings, inoculated April 1990 with *E. lata* from sweet cherry or apricot. Data were collected June 1990. Means are from 10 plants for each treatment

Isolate Host	Inoculated	Noninoculated
Apricot isolate		
Apricot		
Mean length (mm)	68.0 ^a	26.2
Reisolation	10/10	0/10
Cherry isolate		
Apricot		
Mean length	62.4	26.2
Reisolation	9/10	0/10
Cherry		
Mean length	46.4	24.1
Reisolation	10/10	0/10
Grape		
Mean length	28.9	16.8
Reisolation	10/10	0/10

^a Means for inoculated plants were all significantly greater than means for noninoculated plants ($P < 0.0001$), according to *t* tests.

Table 2. Mean lengths of xylem necrosis and frequency of *Eutypa lata* reisolation from grape (cv. Chenin blanc) cuttings, inoculated April 1991 with *E. lata* from cherry or grape. Data were collected November 1991

Isolate	Inoculated	Noninoculated
Cherry		
Mean length (mm)	72.6 ^a	13.6
n ^b	18	17
Reisolation	16/20	0/20
Grape		
Mean length	71.0	13.6
n ^b	17	17
Reisolation	20/20	0/20

^a The effect of inoculation on xylem necrosis was significant ($P < 0.0001$), and the effect of isolate was not significant, according to *F* tests.

^b n = Number of plants used to calculate means. This number varied among treatments because an irrigation malfunction caused death of some plants.

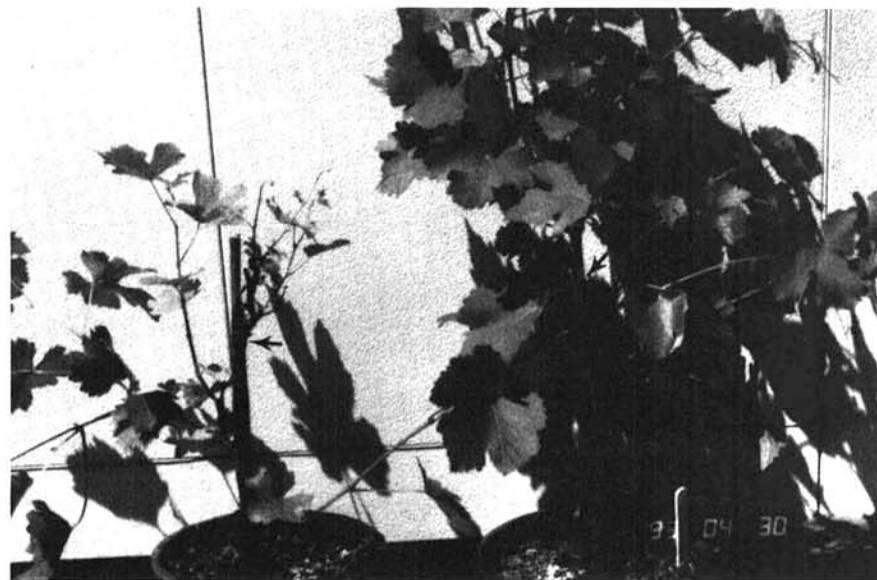


Fig. 2. Shoot symptoms of *Eutypa* dieback on a grapevine inoculated with a *Eutypa lata* isolate from sweet cherry, 26 mo after inoculation. Controls (at right) were wounded but not inoculated. Arrows denote inoculation or wounding sites.

In a third experiment, 45 branches (3–6 cm diameter) were selected in an established cherry orchard (cv. Bing) near Davis. Fifteen were inoculated on 29 April 1991 as described above. For controls, 15 branches were wounded with a cork borer, and sterile PDA plugs were placed in these wounds and wrapped in laboratory film. On 21 November, an additional 15 were inoculated. The inoculations were allowed to develop for 13 mo.

Another experiment was conducted using ascospores as inoculum. This experiment was designed to investigate seasonal differences in wound susceptibility. The experimental design was a split plot, with pruning date as the main plot and inoculation date as subplot. The experiment was conducted in a mature orchard (cv. Bing) near Davis. In each main plot, 80 branches (2–3 cm diameter) were chosen arbitrarily and pruned on the same day. Pruning dates were 29

December 1991, 28 February 1992, and 22 June 1992. For each pruning date, 20 wounds were randomly selected for each of three inoculation dates: 0, 15, and 35 days after pruning. Wounds were lightly misted with sterile distilled water and inoculated with 1,000 ascospores of *E. lata* in a 50- μ l droplet of sterile distilled water. Ascospores were obtained from *E. lata* stromata collected from infected cherry trees in San Joaquin County. Wounds were not covered after inoculation. Twenty wounds were not inoculated. All branch stubs were removed on 2 November 1992.

The virulence of six *E. lata* isolates to sweet cherry was evaluated in a lath-house experiment. Isolates were CA01; CA15, an ascospore isolate from sweet cherry (San Joaquin County); GC13, a canker isolate from grape (Sonoma County); GA17, an ascospore isolate from grape (Napa County); AC01, a canker isolate from apricot (Contra Costa County); and AA02, an ascospore isolate from apricot (Solano County). Seventy dormant trees (cv. Bing, Stockton-Morello rootstock) were planted in pots on 1 February 1992. On 22 February, a wound was made in the stem of each tree with an 8-mm-diameter cork borer, about 3 cm above a lateral branch or spur. Ten trees were inoculated with each isolate by placing a mycelial plug into the wound and wrapping it with laboratory film for 2 wk. Ten trees served as controls. The inoculations were allowed to develop until 30 April 1993 (14 mo).

At the conclusion of each of these experiments, inoculated plants were examined for canker symptoms and the pathogen was reisolated. External canker length (extent of dead cambium) was measured to the nearest millimeter, the bark was removed from the branches (or grape cuttings), and they were split longitudinally. The extent of xylem necrosis was measured to the nearest millimeter, and reisolation of the fungus was performed by aseptically removing wood chips from the margin of the necrotic zone and placing the chips on a semi-selective medium (39 g/L Difco PDA, 100 mg/L streptomycin sulfate, 50 mg/L chlortetracycline hydrochloride, and 5 mg/L dicloran). After 5 days, the plates were inspected for growth of *E. lata*, which can be readily identified by its colony color, morphology, and growth rate (21,24).

Chi-square tests were used to test the association of different isolates with the frequency of cankers or the frequency of reisolation of the pathogen (28). Analysis of variance was performed on the canker length and xylem necrosis length values. Means were compared by orthogonal contrasts or Dunnett's *t* test (28).

Surveys for perithecia. Several Central Valley orchards were selected that were believed to have the highest probability

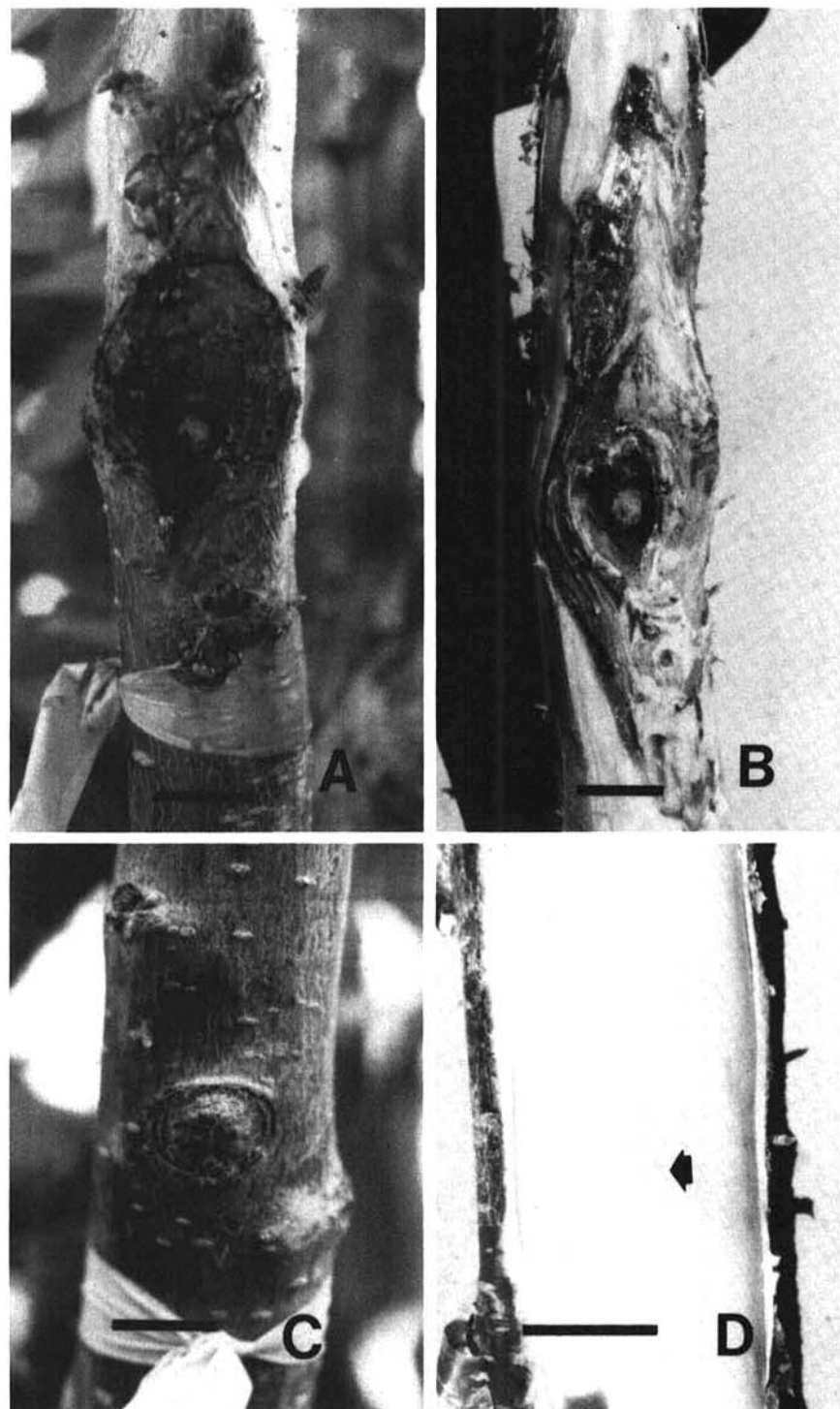


Fig. 3. Canker on limb of sweet cherry tree, 13 mo after inoculation, (A) with bark intact, (B) with bark removed. Bars represent 2 cm. Control limb, wounded but not inoculated, 13 mo after wounding, (C) with bark intact, (D) with bark removed. Arrow denotes wound site. Bars represent 1 cm.

of containing perithecia. These orchards were all mature and sprinkler irrigated. The following orchards were surveyed in 1991 and 1992: four cherry orchards in the vicinity of the original orchard in San Joaquin County, an apple orchard in San Joaquin County, two apricot orchards in Yolo County, and five almond orchards in Yolo County. In addition, home landscapes in Davis were observed during 1990–1992. Twenty-seven vineyards in the Central Valley (Merced, Sacramento, and San Joaquin counties), were also surveyed in 1989–1991. Outside the Central Valley, surveys were conducted in a cherry orchard, a prune orchard, and an almond orchard in Napa County, and in two vineyards in Monterey County. The surveys were designed only to establish the presence of perithecia of *E. lata* in each orchard or vineyard. In order to confirm the field identifications, asci and ascospores were examined in the laboratory.

RESULTS

Pathogenicity of *E. lata* from cherry.

In the preliminary experiment, xylem necrosis developed in each of the inoculated plants and was significantly more extensive than in noninoculated controls. The fungus was reisolated from nearly every inoculated plant (Table 1). There was no significant difference in extent of necroses or frequency of re-isolation between the apricot isolate and the cherry isolate. Externally, inoculated plants did not show symptoms of cankers or dieback, but the inoculated wounds remained open and did not form callus tissue. The wounds on the control plants were completely closed by callus formation.

In the second experiment on grape, similar results were obtained; but due to the longer duration of the experiment, the extent of xylem necrosis was greater. The extent of xylem necrosis was not significantly different between isolates (Table 2). Externally, the inoculated plants displayed a lack of callus formation and slightly sunken cankers around the inoculation site. Three inoculated plants also displayed swelling and cracking of the bark around the inoculation site. Two of these had been inoculated with the cherry isolate, one with the grape isolate. Of the 10 cuttings that were inoculated and left until symptoms appeared, two exhibited typical *Eutypa* dieback symptoms 26 mo after inoculation (Fig. 2). Shoots arising from spurs above the inoculation point were stunted, with small, distorted, chlorotic leaves. Shoots arising from below the inoculation point were normal. *E. lata* was reisolated from both of these plants.

In the third experiment, after 13 mo, external cankers had developed on a majority of the inoculated branches (Table 3). The cankers were similar to those typical of *Eutypa* dieback on

Table 3. Canker development on sweet cherry (cv. Bing) branches inoculated with *Eutypa lata* from cherry on 29 April and 21 November 1991

	Inoculated		Noninoculated
	4/29/91	11/21/91	
Canker incidence	9/15	10/15	0/15
Canker length (mm) ^a			
Range	30–199	65–158	...
Mean	118	105	...
Xylem necrosis (mm) ^b			
Range	50–286	146–655	10–20
Mean	143	296	15
Reisolation	14/15	12/15	0/15
Orthogonal contrasts — xylem necrosis			
	<i>F</i> value	Prob > <i>F</i>	
Inoculated vs. control	50.02	0.0001	
April vs. November	21.03	0.0001	

^a Canker length ranges and means included only those branches with external cankers.

^b Xylem necrosis ranges and means included all branches that received each treatment.



Fig. 4. Profuse gumming from *Eutypa lata* mycelial inoculation on sweet cherry branch, 14 mo after inoculation. g = gum.

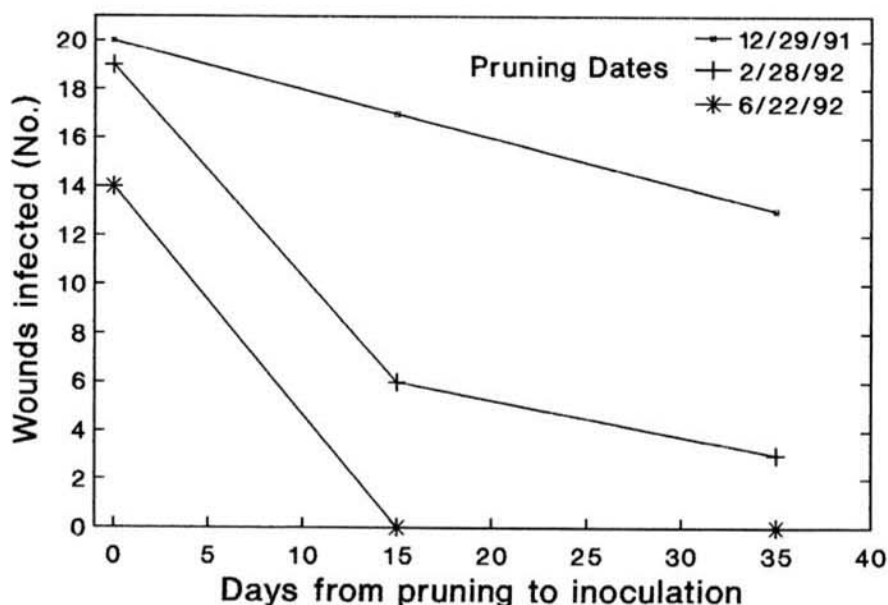


Fig. 5. Effect of pruning date and interval between pruning and inoculation on the frequency of infection of cherry pruning wounds by *Eutypa lata*. Each wound was inoculated with 1,000 ascospores obtained from sweet cherry in San Joaquin County, California. Each point represents the number of infected wound out of 20 inoculated.



Fig. 6. (A) Dieback of sweet cherry (cv. Bing) lateral branch near pruning wound inoculated with *Eutypa lata* ascospores, 11 mo after inoculation. (B) Dieback of sweet cherry (cv. Bing) lateral branch near mycelial inoculation, 6 mo after inoculation. Arrows denote inoculation sites.

Table 4. Canker development on sweet cherry (cv. Bing) trees inoculated in a lathhouse with six isolates of *Eutypa lata* from apricot (AC01, AA02), sweet cherry (CA01, CA15), or grape (GC13, GA17)^a

Isolate	Cankers ^b	Mean canker length (mm) ^c	Mean xylem necrosis (mm) ^d	Dieback ^e	Reisolation ^f
AC01	8/10	121	164	6/10	10/10
AA02	7/10	65	119	5/10	6/10
CA01	6/10	203	181	8/10	8/10
CA15	0/10	...	97	0/10	1/10
GC13	5/10	45	106	4/10	3/10
GA17	0/10	...	32	0/10	1/10
None	0/10	...	25	0/10	0/10

^a Trees were inoculated 22 February 1992, and data were collected 30 April 1993.

^b Isolate had a significant effect on the frequency of cankers (chi-square, $P < 0.001$).

^c Canker length means are included only for those branches with external cankers. Isolate had a significant effect on mean canker length ($P = 0.0002$).

^d Xylem necrosis means are included for all branches that received each treatment. All were significantly greater than the control except GA17 (Dunnnett's t test, $\alpha = 0.05$).

^e The proportion of trees with dieback symptoms is the sum of those observed in November 1992 and April 1993.

^f Isolate had a significant effect on frequency of reisolation (chi-square, $P < 0.001$).

apricot (3). Externally, they appeared as sunken, flattened, or distorted areas of the branch surrounding the inoculation site. The wounds remained open; and in many cases, profuse gumming occurred at the inoculation site and at the canker margins (Fig. 3A and B, Fig. 4). Inoculation date did not have a significant effect on canker length ($P = 0.55$). Internally, necrosis in the xylem tissue of the inoculated branches extended well beyond the margins of the external cankers. Inoculated branches displayed internal xylem necrosis to a greater extent than did the controls. Necrosis varied in length, but in each case extended radially to the pith of the branch. Inoculation date had a significant effect on the extent of xylem necrosis (Table 3). The fungus was reisolated from most of the inoculated branches. The noninoculated wounds on all branches formed callus tissue and were closed within 3 mo after wounding. No gumming occurred surrounding any of the noninoculated wounds (Fig. 3C and D). Controls displayed a zone of internal xylem necrosis only slightly longer than the original wound, and only 2–3 mm in radial width.

Pruning wounds inoculated with ascospores showed seasonal variation in susceptibility, based on the frequency of reisolation of the fungus from inoculated wounds (Fig. 5). Susceptibility was quite high for all three pruning dates when wounds were inoculated the day of pruning, but pruning date had a significant effect ($P < 0.0001$) on susceptibility for the 15- and 35-day inoculations (chi-square test). Branch stubs usually died back to the first spur or lateral branch. This distance was not uniform among branches, so there was no significant effect of inoculation on the extent of xylem necrosis (*data not shown*). Inoculated branch stubs often produced gum at the wound surface. Several inoculated branch stubs had sunken cankers extending several centimeters toward the main branch. Three branch stubs had cankers extending beyond the first lateral branch, resulting in the death of the lateral (Fig. 6A). Two of these were pruned and inoculated on 29 December, and one was inoculated 35 days later. *E. lata* was not reisolated from any non-inoculated branch stubs.

Virulence to sweet cherry varied significantly among the six isolates inoculated in the lathhouse. Frequency of cankers, mean canker length, length of xylem necrosis, and frequency of reisolation were all significantly different among the isolates (Table 4, Fig. 7). Cankers developed on 26 trees inoculated with isolate AC01, AA02, CA01, or GC13. Cankers did not develop on trees inoculated with CA15 or GA17 (Table 4). Wound sites on cankered trees did not close, and the cankers produced large amounts of gum throughout the growing season. By November 1992, dead lateral

branches or spurs were observed on trees inoculated with isolates AC01, AA02, CA01, and GC13 (Fig. 6B, Table 4). At the end of the experiment, three additional trees exhibited dieback (Table 4, Fig. 8); these were killed above the inoculation point. Two were inoculated with CA01 and one with AC01. These trees leafed out in 1993, but quickly wilted and died. This is typical of disease development in apricot (1,3,4). Wounds on the control trees had closed after 3 mo, with no cankers or gumming.

Occurrence of perithecia. Perithecia were detected in several Central Valley locations. In the city of Davis, single apricot trees in two different locations were found to harbor active perithecia and viable ascospores. In addition, the same grapevine with perithecia reported in 1983 (10) still produced viable ascospores.

The four cherry orchards in San Joaquin County all contained trees with perithecia. They occurred on cultivars Bing and Black Tartarian. In the Bing cherry orchard in Napa County, perithecia were detected on six trees of 200 inspected.

On almond, perithecia were detected on only one tree in an orchard in Yolo County. Approximately 150 trees in this orchard were inspected. The cultivar was unknown. No perithecia were detected on almond or prune in Napa County or on apple in San Joaquin County.

Perithecia were detected in both apricot orchards in Yolo County. The cultivar was unknown. Perithecia were found in a sprinkler-irrigated Cabernet Sauvignon vineyard and a furrow-irrigated Chenin blanc vineyard, both in Sacramento County. No perithecia were detected in vineyards in Merced County or in the Salinas Valley (Monterey County).

E. lata was also isolated from three cankers on sweet cherry trees in an experimental orchard in Davis. The cankers were on smaller limbs (2–3 cm diameter) than those discovered elsewhere on sweet cherry. These cankers were associated with pruning wounds; they extended as far as 20 cm from the wound and resulted in the death of several small lateral branches. Gumming occurred at the margins of the cankers. These symptoms were very consistent with the symptoms of *Eutypa* dieback on naturally infected apricot trees, and with those on the artificially inoculated cherry trees. These cankers were not producing perithecia and appeared to be too young to do so.

DISCUSSION

Pathogenicity of *E. lata* to sweet cherry was confirmed by the development of cankers on the inoculated branches after 13 mo, the infection of pruning wounds by ascospores and associated dieback, and cankers on tree stems inoculated with four of the six *E. lata* isolates. In

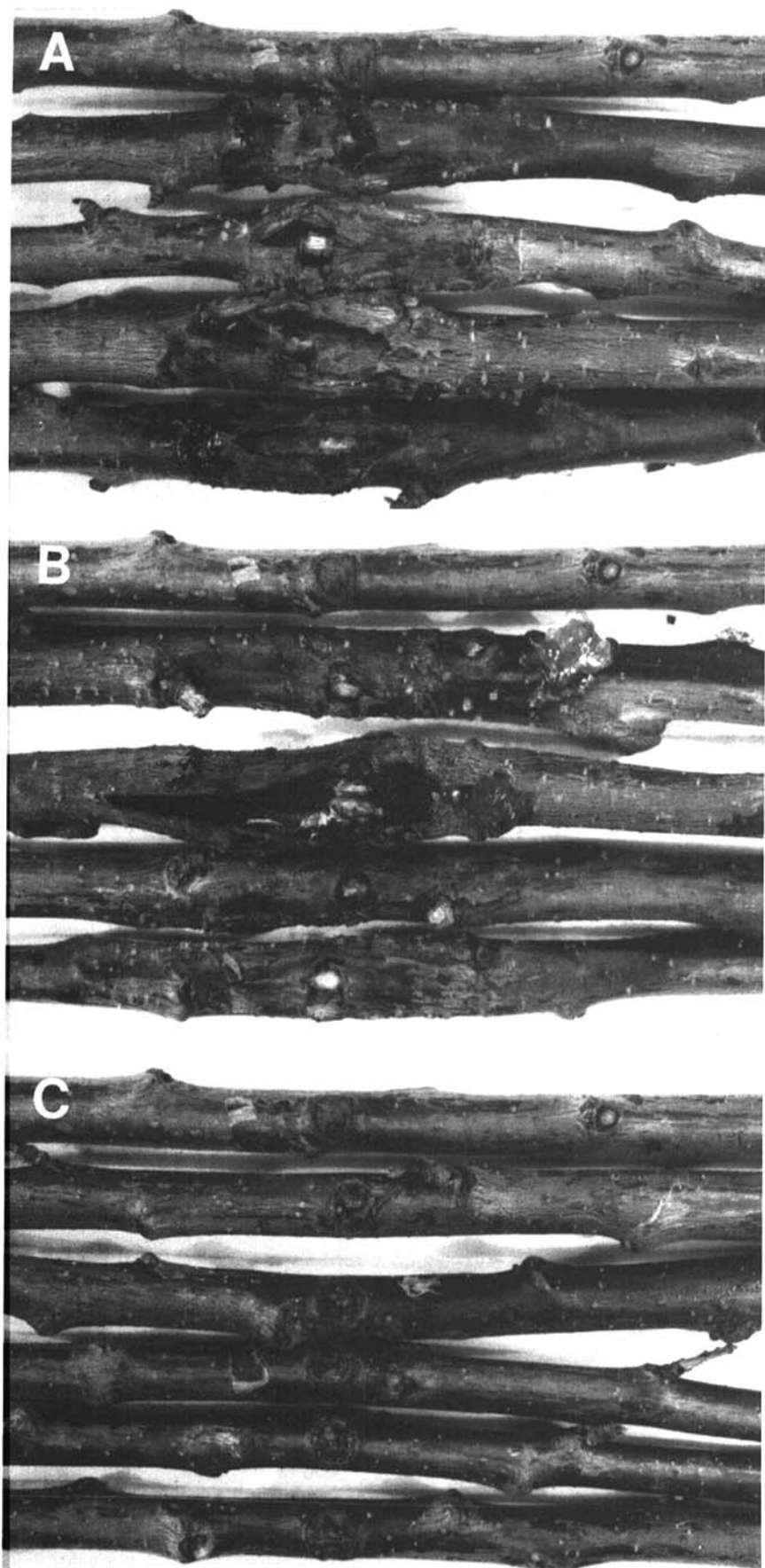


Fig. 7. Effects of inoculation of cherry stems (cv. Bing) with different *Eutypa lata* isolates. (A) Isolate AC01. (B) Isolate CA01. (C) Isolate GA17. Top stem in each panel is noninoculated control.

apricot, dieback does not usually occur until 18 mo or more after infection (3,24). The disease appears to have a similar or somewhat shorter incubation period on cherry; dieback occurred 14 mo (in the lathhouse) and 11 mo (in the field) after inoculation. Under natural conditions, *E. lata* infects pruning wounds during the dormant season, and fall is the most susceptible time for apricot pruning wounds (24). In our experiment with mycelial inoculum, inoculation date did not affect the incidence of cankers or mean canker length, but the fall inoculation resulted in greater xylem necrosis after 13 mo. In the experiment with ascospore inoculation, the earliest pruning date (December) resulted in the greatest frequency of infection and longest duration of susceptibility. An alternative pruning schedule in which pruning is performed in June, after harvest, could minimize infection of cherry trees by *E. lata*. This is also the season during which airborne inoculum is at a minimum (22,23,25).

The variation in virulence among the isolates confirms earlier research (5,10,24). It is interesting that the two isolates which failed to induce cankers were both ascospore isolates. Isolates originating from cankers are presumably able to induce such cankers, but those from ascospores may not always have this ability. This lack of virulence to sweet cherry supports a hypothesis proposed by Carter, who suggested that some isolates of *E. lata* have a restricted host range, but that grape is susceptible to all isolates. Carter did not present data to support this hypothesis (4,5).

Virulence of the cherry isolate, CA01, to grape was confirmed by its identical effect on grape compared to isolate

GC01, and by the expression of typical *Eutypa* dieback symptoms after 26 mo, which is consistent with previously reported incubation periods for this disease (15,16). On apricot, virulence of CA01 was also confirmed by the development of xylem necrosis after 2 mo.

Cankers with *E. lata* stromata were readily found in the selected cherry orchards. These were large cankers originating from pruning wounds on the main branches of the trees. Many trees in these orchards displayed cankers and dieback not associated with *E. lata* stromata. Some of these may also have been caused by *E. lata*, but were not yet sporulating. At this time, we have no quantitative data on the incidence of *E. lata* cankers in cherry, but the disease is present in at least three counties (San Joaquin, Yolo, and Napa).

The occurrence of *E. lata* perithecia in the Central Valley of California is much more common than previously believed, and it appears that the primary sources of inoculum for *Eutypa* dieback in the Central Valley are located within the valley itself. In San Joaquin County, grapes are grown on about 19,000 ha. *Eutypa* dieback is the most serious disease problem in the area. Perithecia have not been detected in any vineyards in this county, but they were detected in a vineyard in adjacent Sacramento County. Cherries occupy about 2,900 ha in San Joaquin County. In some cases, vineyards are interspersed with cherry orchards that harbor *E. lata* perithecia. The evidence presented here indicates that cherries could be a substantial source of inoculum for Central Valley vineyards. Perithecia in home landscapes may contribute additional inoculum.

Fewer *E. lata* perithecia were found on grape, apricot, and almond than on sweet cherry in the Central Valley. However, even a small amount of stromata can produce very large numbers of ascospores (3,14,22), and it seems likely that sprinkler-irrigated apricot orchards and vineyards in the Central Valley also provide inoculum for grapevines in this area. It is possible that perithecia may be capable of developing in the Central Valley without sprinkler irrigation. One of the vineyards reported in this study was furrow irrigated. In Australia, perithecia occur commonly in areas with 350 mm of annual precipitation (3,14). This level of precipitation is exceeded in much of the Valley, including all the locations reported here.

The relative importance of local vs. distant inoculum sources for this disease should be reevaluated. Since each infection must be caused by ascospores, and disease incidence is very high in the Valley, it seems unlikely that a sufficient number of viable ascospores could be dispersed from the Bay Area. Dispersal gradients for *E. lata* have not been established, but spore-trapping studies

on a very similar, closely related pathogen, *Eutypella parasitica*, demonstrated that ascospores could not be detected at a distance greater than 25 m from a source (12). Ascospores of *E. lata* are also adversely affected by ultraviolet radiation and alternate wetting and drying (29). Pruning wounds are relatively small infection courts for the deposition of spores, and the wounds are susceptible only for a limited time (21,24). Single spores are capable of inducing infections, but the infection efficiency of single spores is low (7,24); and our results, along with others (4,5,10,24), suggest that some proportion of the ascospores is not virulent. In addition, the population of apricots in the Bay Area has dropped drastically during the past 20 yr, from approximately 7,000 ha to about 1,800 ha, while incidence of the disease in the Central Valley has increased. Apricot planting in the Central Valley remained steady at 5,000–6,000 ha during that time. Furthermore, the high levels of ascospores that can be trapped in the Central Valley (25) suggest a local source.

Because of the dilution of the spore load over distance (2,12), low probability of contacting a susceptible wound, loss of viability of the spores, somewhat limited duration of susceptibility of wounds, high disease levels and relatively high spore populations trapped in the Central Valley, and the shrinking population of potential inoculum sources in the coastal areas, it seems likely that local inoculum sources are the most important sources for *E. lata* infections in the Central Valley.

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Fig. 8. Dieback of sweet cherry tree (cv. Bing) at right 14 mo after inoculation with *Eutypa lata* mycelium. Tree at right has a canker but no dieback. Both were inoculated with isolate CA01 from sweet cherry. Arrows denote inoculation sites.

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