Infection of Pruning and Small Bark Wounds in Almond by *Ceratocystis fimbriata*

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ABSTRACT

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Ceratocystis fimbriata caused cankers at pruning cuts on branches of Nonpareil almond trees inoculated in all months from September through February. Pruning cuts were susceptible when 0–14 days old, and canker length was greatest where fresh wounds were inoculated. Small dead or living twigs of Mission almond trees, broken without visible exposure of the subtending branch cambium, and wounds inflicted with a lancet became infected when inoculated in alternate months throughout the year. Superficial abrasions of the periderm were less susceptible than were full- and half-depth penetrations of the bark. There were no significant differences in percent infection among sites bearing two, four, six, and eight punctures. Wounds became less susceptible to infection with age, and mean canker lengths did not differ. Percent infection of open bark wounds, made by a cork borer, decreased with decreasing inoculum concentration (10^2–10^6 endoconidia per milliliter) and increasing age (0–14 days).

The fungus *Ceratocystis fimbriata* Ellis & Halst. causes a canker disease of stone fruit trees known as Ceratocystis or mallet wound canker (6). Almond trees (*Prunus dulcis* (Mill.) D. Webb) are the most common host, and Mission, Nonpareil, and Ne Plus Ultra are highly susceptible cultivars (5). The cankers are sunken and dark and exude amber-colored gum at the margins. They are perennial, enlarge slowly over several years, and frequently girdle and kill limbs, but the disease seldom destroys the entire tree. Infections are associated with deep wounds that tear and uncover the cambium. Such wounds are made during harvest by mallets that are used to remove nuts by knocking the branches or by mechanical shakers. Improvements in the design and use of harvesting equipment, which have helped eliminate many wounds, have dramatically reduced the incidence of this disease in California (2).

C. fimbriata is transmitted by several insect species (7). The insects become contaminated with fungal spores while visiting infected wounds and, if attracted to fresh bark wounds, deposit the fungus on the exposed, moist cambium. The pathogen invades the cambium and surrounding vascular and cortical tissues and may produce endoconidia, perithecia, and ascospores on the wound surface within 4 or 5 days of infection. Although almond trees are susceptible at any time of the year, infection occurs in fall because harvest and the accompanying wounds happen then and insect vectors are active. The fungus apparently overwinters with certain insects, which may be the source of primary inoculum each season.

Most cankers are located at deep wounds that crush the bark and visibly expose the cambium, but cankers are also found near pruning wounds. The latter are believed to be extensions of previous infections, which had originated in open wounds on the removed branch, not new infections established through the pruning wound. Pruning wounds have been disregarded as potential infection sites because they are of little interest to insects, and pruning usually occurs during winter when few vectors are present. Also, cankers located at pruning wounds often are caused by *Phytophthora syringae* (Kleb.) Kleb. (2). Although some symptoms of the two diseases may be confused, several characteristics differentiate between them. *P. syringae* cankers are annual and expand rapidly during winter and early spring then cease activity, whereas *C. fimbriata* infections are perennial, quiescent during winter, and enlarge during warm months.

Recently, we isolated *C. fimbriata* from small cankers that surrounded twigs or fruit spurs. Often, several small cankers, distributed along a branch, girdled and killed the branch. These small cankers had marginal gumming and were active in summer but did not bear any obvious or visible wounds typical of infections caused by *C. fimbriata*.

The danger of infection by *C. fimbriata* as a consequence of harvest injuries is well recognized. However, injuries made during pruning operations, whether pruning cuts or small inconspicuous wounds, have not been implicated as possible sites of infection. Infection at pruning cuts and the branch death caused by small cankers are relatively infrequent events, but they can produce sufficient enough damage in some orchards to alarm growers. The objectives of the studies reported here were to 1) test the susceptibility of pruning and inconspicuous wounds to infection, 2) examine the effects of wound age and severity on infection, and 3) determine the relationship of wound age and inoculum concentration on infection and canker length.

MATERIALS AND METHODS

Experiments were conducted at the University of California Kearney Agricultural Center in Fresno County, CA. Cultivar Nonpareil almond trees were used for pruning wound experiments; Mission almond trees were hosts for all other studies. Limbs selected for inoculation were 3- to 6-yr-old secondary or tertiary smooth-barked scaffolds, 5–10 cm in diameter.

General. Two isolates of *C. fimbriata* were used. One was obtained from almond and contributed by R. M. Bostock, University of California, Davis (Davis isolate), and the other was cultured from a canker that surrounded a dead twig on a Mission almond tree grown in a commercial orchard in Fresno County, CA (field isolate). The field isolate was used unless otherwise stated. The fungi

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were maintained on acidified potato-dextrose agar (aPDA), and cirri from individual perithecia were transferred to initiate new colonies. Inoculum was prepared as follows. Endoconidia, ascospores, and mycelial fragments from 10- to 14-day-old cultures were collected in 25 ml of sterile deionized water. Approximately 5 ml of this suspension was poured onto each of several aPDA culture plates and allowed to stand for 30 s. The liquid was decanted off, leaving 1–2 ml of the suspension on the plate, and the seeded plates were incubated at 22 ± 1 C for 4–5 days. Endoconidia were collected from these plates in sterile deionized water and the concentration was adjusted to 10^4 endoconidia per milliliter. Except where noted, 0.2 ml of this inoculum was applied to each inoculation site. Percent germination was determined for each experiment by counting the number of germinated endoconidia in three groups of 100 after 24 hr of incubation on aPDA at 22 ± 1 C. Germination ranged from 72 to 94%.

Representative tissue samples from each experiment were cultured to reisolate *C. fimbriata*. Cankers were surface-sterilized in 0.05% sodium hypochlorite for 15 min. After the outer bark was removed from the canker margin with a sterilized knife, pieces of bark that included both necrotic and healthy tissues were excised from the margin and placed onto aPDA culture plates and incubated for 7–10 days at 22 ± 1 C. Identification of the pathogen was based on the presence of endoconidia and, usually, perithecia and ascospores (10).

Experimental design is described separately for each experiment. Disease incidence is reported as percent infection. Data were analyzed by the chi-square test for independence in an $r \times 2$ frequency table where r = number of treatments. If appropriate, a correlation index for linear regression (r') was calculated (9). Canker length data were analyzed by analysis of variance and Duncan's multiple range tests or correlation. Canker length data were not analyzed for treatments with low infection rates because too few cankers were present to allow valid analysis. Uninfected sites were not included in calculation of mean lengths, and wound size was not subtracted from canker lengths.

Small injuries were made by puncturing the bark with a Unilet lancet (Ulster Scientific, Inc., Highland, NY). The lancet blade was approximately 4 mm long and 1 mm in diameter, with a beveled tip. To determine which tissues were reached by the lancet, we estimated bark thickness by measuring four to six bark plugs or the bark of cross sections of removed branches that were the same age and size as those used in the experiment. Because the bark was 3–4 mm thick, full insertion (full depth) of the lancet extended to the cambium or slightly beyond. A shallow wound that reached approximately midway between the surface and the cambium (half depth) was standardized as follows. A bark plug was cut radially to half its depth then the lancet blade was inserted through the slice. When pressed against the branch, the tip of the adjusted lancet pierced the bark without reaching the cambium. Scratch punctures were 1- to 2-mm-long superficial breaks of the periderm made with the lancet tip. Puncture wounds, away from twigs, were located within 1.5- to 2-cm-diameter circles. Open wounds that exposed the cambium were made by removing a 5-mm-diameter bark plug with a cork borer. Inoculation sites were spaced 15–25 cm apart and on all sides of the branch unless otherwise stated. Bark was not surface-sterilized before wounding, but the cork borer was decontaminated between cuts by alcohol dip and flaming, and lancets were stored in vials containing 70% ethyl alcohol between wounding events. Wounds were not covered before or after inoculation unless otherwise stated.

Susceptibility of pruning wounds. Branches were cut radially with a chain saw, leaving a stub 1–1.5 m long, on different days to provide wounds 0, 2, 7, and 14 days old, which were inoculated on the same date each month. In the first experiment (1988–1989), approximately 0.25 ml of inoculum was applied to two opposite quadrants of the circumference of the exposed bark ring on each cut, and the uninoculated quadrants on each cut were used as controls. Only one quadrant of the circumference was inoculated in the second experiment (1989–1990), and controls were other uninoculated branches pruned on each date.

Most almond trees in California are pruned in November, December, and January. In the winter of 1988–1989, we tested the susceptibility of pruning wounds made in November, December, January, and February. The following season, 1989–1990, we did not prune in February but did so in September and October because some growers practice early fall pruning. There were six replications of each treatment in a randomized complete block design with adjacent individual trees as blocks. Canker length was recorded for the first experiment in September 1989 and for the second in August 1990.

Susceptibility of small wounds. Dead and living 1- or 2-yr-old twigs, approximately 5–8 mm in diameter, were injured in either of two ways: they were broken at the base without visibly tearing the bark of the subtending branch or eight full-depth punctures were made in the collar tissue around the twig base. Bark away from twigs was wounded by eight full-depth punctures with the lancet or with the cork borer. All wounds were inoculated immediately. Controls included similarly wounded but uninoculated, and uninjured, inoculated sites.

In July 1988, we compared the ability of both fungal isolates to cause cankers. Dead twigs wounded by breaks or collar punctures were inoculated with each isolate along with the controls described earlier. There were six replications of each treatment, each replication located on a branch using two branches on each of three trees, in a randomized complete block design. Internal canker lengths were measured in October 1988.

A second experiment investigated the susceptibility of bark wounds at different times of the year. The treatments included dead twigs wounded by 1) breaking or 2) eight full-depth punctures of the collar, 3) living twigs wounded by breaking, and bark away from twigs wounded by 4) eight full-depth punctures or 5) the cork borer. Wounds were inoculated immediately, and controls were similar uninjured, inoculated sites. The experiment was performed in September and November 1988 and January, March, May, and July 1989. For each experiment, four branches on each of three trees were used, with each replication assigned to one branch. These 12 replications were divided into two groups of six (two limbs from each tree), each group organized in a randomized complete block design. One group from each inoculation month was sacrificed in October 1989 for measurement of canker length and reisolation of the fungus. The remaining groups were kept to observe further development of symptoms and were evaluated in August 1990.

Effects of wound age and severity on susceptibility. The influence of wound age on susceptibility was investigated by inoculating injuries at various times after wounding. Wound severity was varied in a standardized manner by altering the number and depth of punctures. Wounds were made in bark away from twigs.

The effects on susceptibility of variation in number and depth of punctures were examined independently in September 1988. Puncture depth (full depth) was kept constant and the number of punctures (zero, two, four, six, or eight) varied in one experiment or puncture number (eight) was unchanged for full-depth, half-depth, and scratch puncture wounds. Uninjured, inoculated sites served as controls and all sites were inoculated immediately after wounding. There were six replications (three per branch) of each treatment in a randomized complete block design, and each experiment was located on a different tree. The results were evaluated in November 1988, and the presence of gum and internal necrosis was considered evidence of infection.

The following year, puncture number and depth were varied in the same experiment. Two, four, six, and eight each full-depth, half-depth, and scratch punctures were inoculated immediately after wounding. In a separate experiment,

conducted at the same time, we examined the effect of wound age on susceptibility. Sites bearing eight full-depth or half-depth punctures were inoculated on the same day 0, 4, 8, 24, 48, and 96 hr after wounding. Injured, uninoculated controls for each wound age, depth, and number of punctures were included.

Treatments were replicated six times and arranged in a two-way factorial with split-plot design. A pair of branches, emanating from the same scaffold, served as a replication for main factors (wound age or puncture number), and six main-factor replications were located on two adjacent trees, three replications per tree. Wound depth treatments were randomized in subplots of wound age or number. Both experiments were established in September and December 1989 and evaluated in August 1990.

Inoculum concentration and wound age. Open wounds, made with a cork borer on different days, were 0, 2, 4, 7, 10, and 14 days old simultaneously, and all injuries were inoculated on the same day. The wounds were covered with Lumite Saran (Chicopee Manufacturing Co., Cornelia, GA) between wounding and inoculation to exclude insects. Each wound received 0.2 ml of 10^2, 10^3, 10^4, 10^5, or 10^6 endoconidia per milliliter. Controls were similar wounds of each age treated with 0.2 ml of 0.4% water agar. There were four replications of each treatment arranged in a two-way factorial with a split-plot design. Adjacent individual trees were replications of wound age treatments, the main factor, and all wounds on a branch were the same age. The series of inoculum concentrations was randomized in subplots on each branch. The experiment was conducted in September 1988 and December 1989 and evaluated each following summer.

Survival on bark surface. Fungus survival on the bark surface was tested using inoculum as described earlier or a collection of endoconidia, ascospores, and mycelial fragments gathered by drawing the edge of a small spatula across the surface of 14- to 21-day-old cultures (smear). Endoconidia and ascospores in each of five typical smears were counted to estimate the concentration of propagules in a smear. The averages of these were 8 and 16 $\times$ 10^5 ascospores and endoconidia, respectively. Inoculation sites were identified by drawing 2-cm-diameter circles spaced 25 cm apart along one side of a branch. A companion circle, to be used as an uninoculated control, was drawn for each inoculation circle on the opposite side of the branch. Inoculum was applied to all inoculation circles on 9 November 1989. After application of inocula, eight full-depth punctures were made in the inoculation and companion control circles at the following intervals (days): 0, 2, 7, 14 (November), 30 (December), 61 (January), 103 (February), 141 (March), 165 (April), 190 (May), and 222 (June). In addition, inoculated and companion control sites were wounded during rains on 13 and 20 January. A separate experiment was designed for each inoculum type. Treatments in both experiments were replicated four times, each replication housed on one of four trees, and arranged in a randomized complete block design. Data were not analyzed.

RESULTS

Pruning wounds. Pruning wounds were susceptible to infection in every month tested (Table 1). Mean canker lengths for each month were calculated from combined wound age inoculations, and significant differences among months were inconsistent in both years. The shortest (5.7 cm) and longest (17.7 cm) average lengths resulted from November and December inoculations, respectively, in 1988. Cankers at pruning cuts inoculated immediately were significantly longer than those at older wounds in both years. Uninoculated quadrants in the 1988–1989 experiments died back 1–6 cm. In all cases, canker extension below the inoculated site could be distinguished clearly, however, lateral canker growth made evaluation difficult for uninoculated control quadrants on several limbs. During 1989–1990, average dieback of uninoculated pruned limbs ranged from 2.5 to 6.0 cm, which was significantly less ($P = 0.05$) than average canker extension for every inoculated treatment each month (*data not presented*). *C. fimbriata* was not isolated from any control treatment.

Susceptibility of small wounds. Both isolates of *C. fimbriata* produced cankers at three and five of the six sites wounded by breaking and collar puncture, respectively. Average canker lengths included 9.5 cm (broken twig) and 9.0 cm (collar puncture) for the Davis isolate and 6.7 cm (broken twig) and 8.6 cm (collar puncture) for the field isolate. There were no significant differences ($P = 0.05$) between isolates or wound sites in canker lengths, and infection did not occur at uninjured or uninoculated control sites.

Bark wounds were infected in each of the 6 mo tested, and there were no significant differences in percent infection among months. Cankers formed at all inoculated collar and bark puncture and cork borer wounds in each experiment, and infections were more frequent near broken living than broken dead twigs (Table 2). Control sites were not infected except one uninjured dead twig inoculated in May. Mean canker length (derived from combined data for all months) at cork borer injuries was significantly longer (17.6 cm) than that at collar puncture wounds near dead (9.7 cm) or living twigs (6.5 cm). Too many values were missing in other treatments to allow analysis. Treatment groups

Table 1. Mean canker length (cm) on almond (*Prunus dulcis* 'Nonpareil') with pruning wounds inoculated with *Ceratocystis fimbriata*[y]

	Month inoculated							Wound age at inoculation (days)				
Year	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	LSD	0	2	7	14	LSD
1988–1989	···	···	5.7 a[z]	17.7 b	12.0 ab	12.9 ab	10.5	26.4 a	14.5 b	7.5 b	7.3 b	7.6
1989–1990	6.0 a	6.5 a	10.9 b	9.8 b	10.5 b	···	2.7	12.0 a	8.2 b	7.4 b	7.2 b	1.9

[y] Approximately 0.25 ml of a suspension of 10^4 endoconidia per milliliter was placed onto exposed bark, cambium, and outer wood of one-fourth to one-half the circumference of each pruning cut.

[z] Means across columns followed by the same letter do not differ significantly ($P = 0.05$) according to analysis of variance and Duncan's multiple range tests. There were six replications of each treatment in a randomized complete block design; each mean is the average of all data for that month or wound age.

Table 2. Infection of bark wounds on almond (*Prunus dulcis* 'Mission') by *Ceratocystis fimbriata*

Wound location[x]	Wound type[x]	Infected wounds (%)[y]
Dead twig	Broken	27.8 a[z]
	Collar punctured	100.0 c
Living twig	Broken	63.9 b
Branch, no twig	Bark punctured	100.0 c
	Open wound	100.0 c
$P = 0.05$, LSD = 14.0		

[x] Dead or living twigs broken at base without obvious exposure of branch cambium, Collar of dead twig punctured, to cambium, eight times with a Unilet lancet. Open wound made by removing 5-mm-diameter bark plug with a cork borer. All wounds inoculated immediately with 0.2 ml suspension of 10^4 endoconidia per milliliter.

[y] Combined data from six experiments performed in alternate months, beginning September 1988 and ending July 1989. Six replications of each treatment in a randomized complete block design each month. Evaluated 24 October 1989.

[z] Means followed by the same letter do not differ significantly according to analysis of variance and Duncan's multiple range tests. Analysis performed on arcsine transformed data; actual percentages reported.

maintained for 2 yr to observe symptom development had percentages of infection similar to those reported above. The cankers became more sunken, continued to gum along the margins, and resembled those originally observed in commercial orchards. In three cases, branches were killed.

Wound age and severity. In the 1988 experiments, percent infection increased significantly with increasing number of punctures per wound. Percent infected wounds differed significantly, $r' = 0.886$, and were (number of full-depth punctures is given in brackets): 0.0 [0], 50.0 [2], 83.3 [4], 100.0 [6], and 100.0 [8]. Uninjured or scratch puncture wound sites were not infected but cankers formed at 83.3 and 67.7% (not significantly different) of full- and half-depth puncture wounds, respectively.

Results of experiments performed in September and December 1989 differed somewhat from those just described. Percent infection did not differ significantly among sites wounded with two, four, six, or eight punctures inoculated immediately. Significantly fewer cankers developed at scratch than at full- and half-depth puncture wounds (Fig. 1). Percent infection decreased with increasing wound age in both months, and there was no significant difference between full- and half-depth puncture wound treatments. All control wounds were not infected. There were no significant differences in canker lengths among any wound age, number, or depth. Mean canker length ranged from 5.3 to 8.1 cm.

Inoculum concentration and wound age. Percent infection increased with increasing inoculum concentration and decreased with wound age (Fig. 2). An increase in inoculum concentration was accompanied by an increase in canker length in September. Too few cankers formed in the December experiment to allow statistical analysis of lengths. All control wounds healed and were not infected.

Survival on bark surfaces. Infections occurred at sites inoculated with either inoculum type when wounded immediately or 2, 7, or 14 days later. On these dates, all sites, except one in the 14-day-old treatment, were infected where smear inoculum was used, but there were four, three, two, and one infected injuries at immediate, 2-, 7-, and 14-day-old sites, respectively, where the suspension inoculum was used. One canker developed at a smear inoculum site wounded during rain on 30 January. No cankers were observed at any other inoculated or uninoculated locations.

The fungus was recovered from some but not all representative samples in all experiments.

DISCUSSION

Pruning wounds were susceptible to infection by *C. fimbriata* during fall and winter and remained susceptible for at least 14 days. Fresh wounds appeared to be most at risk because the ultimate canker length was greatest at wound sites inoculated immediately after the cut was made. In contrast with our experience, Bostock and Doster (2) were not able to infect pruning cuts of Nonpareil or Ne Plus Ultra almond trees inoculated with *C. fimbriata* in February. The inoculum they used, mycelial plugs, and the observation period, 3 mo, differed from ours. However, other unidentified factors probably would better resolve this discrepancy.

Small bark wounds were susceptible to infection throughout the year, and the cankers associated with such wounds were similar to those found on the branches of almond trees in commercial orchards. Wound severity, represented in these tests by the number or depth of punctures, was less important than age in determining the success of infection, wounds becoming less susceptible with time. This agrees with previous studies in which open wounds became more resistant to infection with age (1,3,8). Ultimate canker size was affected by wound age and inoculum concentration, but not by time of year when infected. Limb diameter also influences canker length (6).

The differences we found in percent infection and mean canker length among various inoculum concentration, wound age, or wound depth treatments perhaps reflect differences in the wound healing response. Bostock and Doster found that resistance in almond to infection by *C. fimbriata* was related to wound periderm formation (3). Various aspects of wound healing, including deposition of lignosuberized boundary zones, formation of wound periderms, and suberization of cells, are affected by time, temperature, tissue age, and relative humidity (1,4,8). Time of year also may affect infection and disease development (6). *C. fimbriata* grows most rapidly, in culture, at temperatures that may occur in summer and fall (10). Host susceptibility may be affected in summer and fall when trees are under stress from high evaporative and crop load demands. In winter, trees are dormant. These various physiological states of the host, in addition to environmental parameters such as temperature, may influence susceptibility and infection.

The open wounds made by the cork borer in these and other studies (3,6) were meant to simulate wounds made by machinery. The broken twig, scratch, and puncture wounds used here were designed to simulate wounds that may be encountered during the pruning process. These small wounds were adequate infection courts for the pathogen, but the attractiveness and suitability of wounds to insect vectors are equally important. Insects may be observed visiting large bark wounds, such as those made by

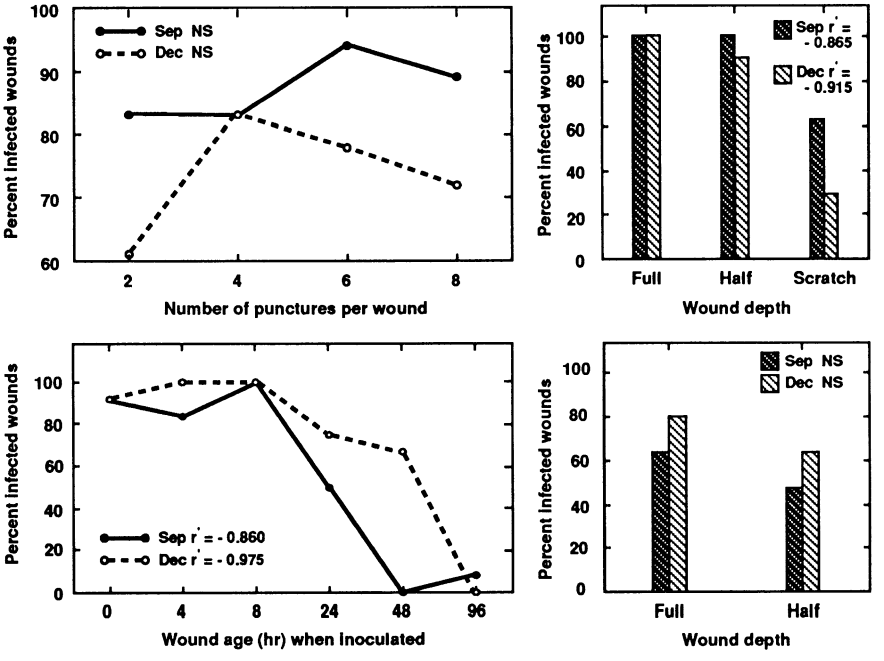

Fig. 1. Effect of age, number, and depth of puncture wounds in Mission almond bark on susceptibility to infection by *Ceratocystis fimbriata*. Wounds made with Unilet lancet within 1.5-cm-diameter circular area on branch. Full-depth and half-depth punctures reached the cambium and cortex, respectively. Scratch wounds were superficial breaks of the periderm. Wounds were inoculated immediately, or after aging, with 0.2 ml of suspension of 10^4 endoconidia per milliliter on 25 September and 20 December 1989; both evaluated 15 August 1990. There were six replications of each treatment in a two-way factorial with split-plot design; wound depth treatments were subplots of puncture number or wound age treatments (main factors). r' = Index for linear regression in an $r \times 2$ frequency table where r = number of treatments, $P = 0.05$ or less. NS = not significant.

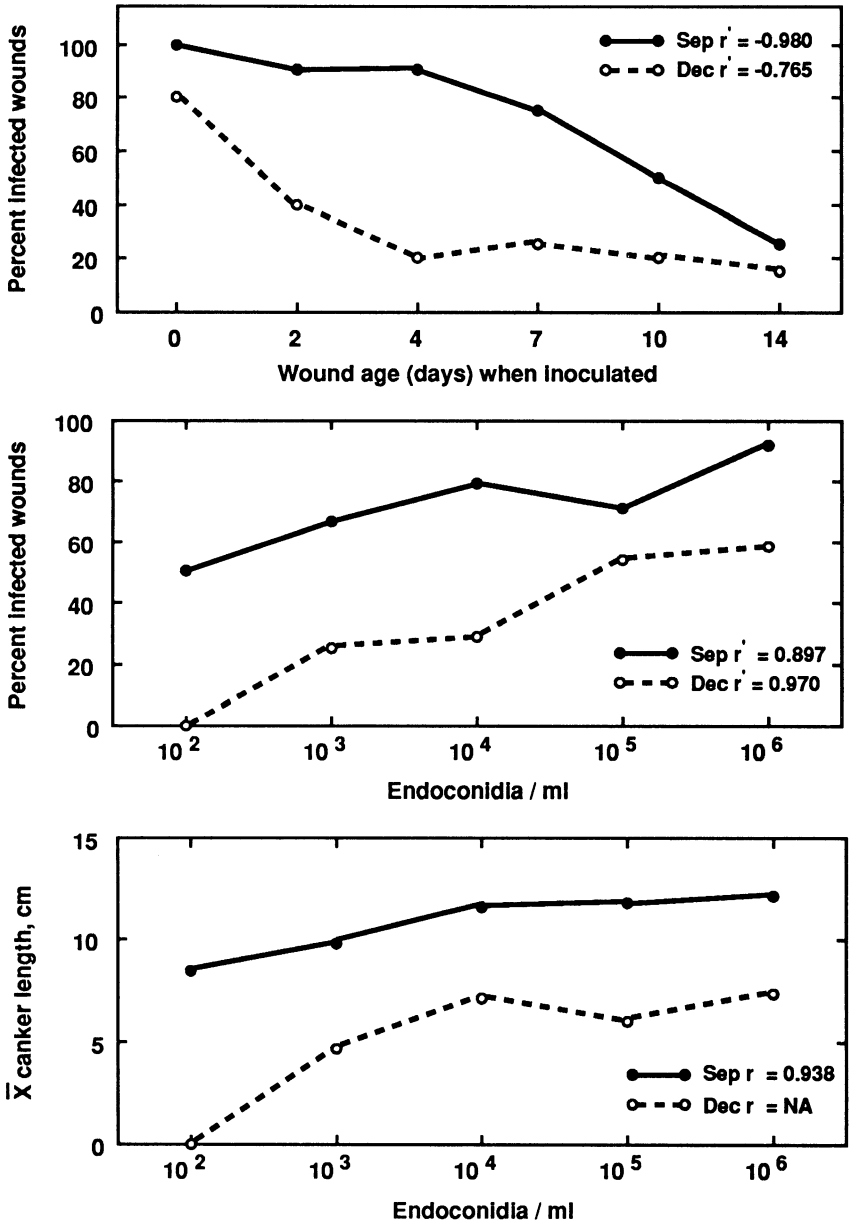

Fig. 2. Effect of inoculum concentration and wound age on susceptibility to infection of almond bark by *Ceratocystis fimbriata*. Bark plugs, 5 mm diameter, removed at intervals with a cork borer. Insects excluded by covering wounds with Lumite Saran between wounding and inoculation. All wounds inoculated with 0.2 ml of suspension of 10^4 endoconidia per milliliter on 30 September 1988 and evaluated on 22 September 1989 or inoculated on 20 December 1989 and evaluated 17 August 1990. There were four replications of each treatment in a two-way factorial with split-plot design; inoculum concentration treatments were subplots of wound age treatments. r' = Index for linear regression in an $r \times 2$ frequency table where r = number of treatments, $P = 0.05$ or less. NA = not available, insufficient number of cankers formed in December experiment to allow statistical analysis.

machinery, shortly after the injury is made (7). Later, they are found infesting the wound, usually under the loosened bark. Thus, the potential for insect transmission is easily recognized. Small, inconspicuous wounds also may attract insects, although this behavior may not be obvious. Insects may further disseminate spores by inadvertently scattering them along the bark during general activity.

Pruning is the most likely orchard practice to result in small bark wounds, although such wounds could occur at any time. The pruning cuts themselves, along with bark abrasions or small breaks near twigs incurred when pruned branches are pulled from the canopy, would provide many potential infection sites. However, pruning usually occurs during late fall and winter when few insects are present or active. Spores on bark surfaces may be moved about by rain and washed into wounds, but this has not been demonstrated. The minimal activity of vectors and random chance of possible rain dispersal may, in part, explain why infection of pruning or small wounds is not more prevalent in nature.

According to our evidence, *C. fimbriata* does not survive easily for long periods when unprotected on bark, but further investigation may prove otherwise. One wound became infected when injury was made during rain, which indicates that some inoculum survived and that rain may play an important role in infection of small wounds. Growers have reported greater incidence of cankers at pruning wounds and along branches in trees pruned just before a rain. (*C. fimbriata* was isolated from these cankers). Rain may act as a disseminating agent as well as maintaining moisture to support germination and infection.

Unfortunately, our research did not identify a time of year at which pruning could be done without risk of infection. At present, the best recommendation to growers would be to avoid bark injuries made by harvesting equipment to prevent establishment of the fungus in the orchard and to prune when there is the least likelihood of rain.

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