

Annual Report 1998*Prepared for the Almond Board of California*

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Project No. 95-JA1: **Management of Almond Anthracnose in California:**
 Detection and Identification of the Causal Pathogen,
 Epidemiology, and New Management Practices.

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SUMMARY

In 1998, a major disease outbreak occurred in April and May. Although rainfall occurred early in the season, low temperatures delayed the onset of the anthracnose epidemic. Disease incidence was correlated with rainfall and warmer temperatures. The importance of mummified fruit as inoculum sources for new spring infections was confirmed. In both central and northern regions of the state, both genotypes of the pathogen *C. acutatum* could be found, often occurring within the same orchard or even on the same fruit. Genotype P, however, was isolated predominately from diseased tissue. Natural disease incidence that was observed for the first time in an almond variety trial indicated that the almond varieties Wood Colony, Sonora, 13-1, 2-43W, and 1-102W had more anthracnose fruit infections as compared to Nonpareil and several other varieties. In this evaluation, anthracnose leaf infections were also observed and their incidence differed from the occurrence of fruit infection among the varieties. Non-wound inoculation studies with detached fruit correlated with field observations for most varieties. Wound inoculation studies using detached fruit indicated that most varieties were very susceptible to anthracnose infection. In 1997 and 1998, wound-inoculation studies with attached nuts in the field showed greater differences among varieties but results did not correlate with field observations on the natural incidence.

In postharvest studies, kernel colonization by the fungal pathogen occurred after 7-20 days at kernel moisture levels higher than 13.1%. No growth of the fungus was observed below kernel moisture contents of 10.1% after 3 months. These results indicate that postharvest damage to almond kernels was probably the result of preharvest epidemics that were not managed. Additional studies over longer periods of time (e.g. 6-12 months) are currently being conducted.

Fungicides evaluated in field trials included: Abound (azoxystrobin), Indar (fenbuconazole), Rally (myclobutanil), Break (propiconazole), Elite (tebuconazole), Captan (captan), Bravo (chlorothalonil), Elevate (fenhexamid), and Flint (a numbered strobilurin compound). No fungicide program eradicated the disease. Six applications (in approximately 2-week intervals) of either Abound, Flint, Elite, Break, or Bravo resulted in the lowest fruit disease incidence in single-fungicide programs. Elevate and Indar were ineffective at the rates evaluated. Rotation programs with Abound, Break or Elite with Bravo or Captan were very effective in reducing fruit anthracnose incidence. In these studies, fungicide rotation programs also significantly increased crop yield in a commercial orchard with a high incidence of disease. Multiple applications on regular intervals during conducive environments from late February to

May were necessary for successful disease management. In addition, a pruning program to remove previous year's dead and diseased wood improved the efficacy of fungicide treatments. Programs based on rotations of Break, Abound, and Captan will be presented and discussed. Preliminary studies evaluating temperature and wetness relationships for disease development were initiated and results may help to improve timing of fungicide applications in an anthracnose management program.

INTRODUCTION

In recent years, almond anthracnose has become a widespread and serious problem in the major almond growing regions in California. Symptoms of the disease occur on blossoms, developing and maturing fruit, and on spurs and branches with often extensive leaf yellowing and dieback throughout the spring and summer, if conducive conditions persist.

Previously, the fungal pathogen was characterized and identified as *Colletotrichum acutatum* rather than *C. gloeosporioides* as has been reported from Israel. Two distinct sub-populations could be identified among the isolates of *C. acutatum* from almond that differed in their cultural pigmentation (pink vs. gray), pathogenicity on wound-inoculated almond fruit, sensitivity to benomyl at low conidial concentrations, and in their DNA fingerprints. In 1997, the disease was also found to cause postharvest problems on the 1996 crop. Preliminary studies to determine the cause of dieback symptoms in trees associated with almond anthracnose indicated that phytotoxic compounds secreted by the fungus may be responsible for leaf yellowing and branch dieback. Thus, the disease can cause direct pre- and postharvest losses of the almond crop by infecting fruit and kernels, as well as potentially indirect losses by causing shoot dieback and tree decline.

OBJECTIVES

- I. Epidemiology
 - A. Continue to survey isolates from major almond growing regions within California
 - B. Determination of overwintering mechanisms
 - C. Evaluation of host susceptibility
- II. Identification of *Colletotrichum* spp.
 - A. Cultural studies on morphology
 - B. Molecular characterization
 - C. Detection of *Colletotrichum* species in plant tissue using molecular probes
- III. Preliminary management strategies
 - A. *In vitro* sensitivity of fungal isolates to selected fungicides
 - B. Fungicide efficacy studies
 - C. Orchard sanitation and anthracnose management

MATERIALS AND METHODS

Fungal isolations. Fungi were isolated from selected symptomatic tissues of almond including fruit, leaves, peduncles, spurs, and branches. For fungal isolations, infected tissue was surface-sterilized with 400 mg/liter hypochlorous acid (HOCl) for 1 min and plated onto potato dextrose agar (PDA) amended with rifampicin (20 mg/liter) and ampicillin (130 mg/liter) to inhibit bacterial growth. Cultures were maintained on PDA (up to 1 year) or cryogenically.

Molecular characterization of *Colletotrichum* spp. from almond. Fungal DNA was isolated using approximately 5 mm³ of mycelium. The population substructure was investigated

using the simple repeat primers (CAG)₅ and (GACA)₄ and the random primers OPX-12 and OPY-11 in PCR amplification experiments. Amplification reactions were carried out in 25- μ l volumes containing 1 μ l DNA extract, 14.3 μ l sterile distilled water, 2.5 μ l 10x reaction buffer (500 mM KCl, 100 mM Tris-HCl pH 8.3, 1% Triton X-100), 2 μ l dNTP stock (2.5 mM each dNTP), 4 μ l of 10 mM MgCl₂, 1 μ l primer (10 μ M) and 1 unit [primers (CAG)₅ and (GACA)₄] or 1.5 units (primers OPX-12 and OPY-11) *Taq* DNA polymerase. Amplifications were performed in a thermal cycler programmed for the following cycling conditions: initial denaturation at 94°C for 30 s; 35 amplification cycles consisting of 30 s at 94°C, 30 s annealing and 1 min extension at 72°C; final extension at 72°C for 7 min. The annealing temperature was 60°C for primer (CAG)₅, 48°C for primer (GACA)₄, and 35°C for primers OPX-12 and OPY-11. Reaction products were separated by electrophoresis in 1.5% agarose using 0.5 x Tris-borate-EDTA buffer at 10-15 V/cm.

Population structure of the almond anthracnose pathogen within selected orchards in two counties. In the summer of 1998, the frequency of occurrence of the two sub-populations of the anthracnose pathogen was investigated within four orchards in Butte Co., two orchards in Stanislaus Co. and one orchard in Merced Co. Fifty to 90 isolates were obtained from 100 mature fruit with anthracnose symptoms collected from 38-46 trees/orchard (1-4 fruit/tree). Sub-populations as determined by cultural pigmentation (pink vs. gray) were verified for approximately 20% of the isolates by RAPD analysis. The distribution of both genotypes was then mapped for each orchard.

Temperature optima for growth of the two *Colletotrichum* spp. sub-populations from almond. To determine the growth rate at selected temperatures, agar plugs (5 mm in diameter) obtained from the margin of 5- to 7-day-old cultures were placed on the surface of PDA in 10-cm petri dishes. Cultures were incubated at 3, 10, 15, 20, 25, 32, or 36°C. Growth rates were recorded daily for 5 days and radial growth rates were calculated as the 5-day mean daily growth (mm/day) for each culture and temperature. Data for each temperature were statistically analyzed using analysis of variance, and multiple comparisons of means were performed using least significant difference (LSD) procedures of SAS 6.11. (SAS Institute, Cary, NC).

Effect of kernel moisture content on growth of *C. acutatum*. In order to define environmental conditions that lead to postharvest kernel damage, almond kernels that were inoculated in the laboratory either with conidial suspensions or agar disks of the pink cultural type were incubated for up to 3 months at selected relative humidities using salt or sugar solutions. For this, almond kernels (cultivar Carmel) were split in two halves along the sutures and were sterilized by autoclaving for 20 min. Kernels were then placed on sterile wire racks in plastic boxes containing 200 ml of one of the following solutions: saturated KNO₃, saturated K₂HPO₄, saturated KCl, saturated NaCl, or 6 molal sucrose. These solutions served to obtain and maintain different levels of humidity within the boxes and therefore different kernel moisture contents. The boxes were sealed with silicon sealant or plastic tape and the kernels were allowed to equilibrate for 7 days at 20-24°C. The kernels were then inoculated with either 10 μ l of a spore suspension (10⁶ spores/ml) or with mycelial agar plugs (5 mm diameter, 2 mm thick) of an isolate of *C. acutatum*. Three boxes were prepared for each solution and each box contained three replications for each inoculation method with 10 kernel halves each and three replications for measuring kernel moisture contents with 20 kernel halves each. The boxes were incubated at 20-24°C for up to 3 months. Kernels were checked weekly for fungal growth. When fungal growth was observed, aliquots of the non-inoculated kernels were ground using a food processor and passed through a 12-mesh sieve. The moisture content of the ground almond kernels was determined using a moisture balance (Mettler LP16). Kernels of treatments that did not show any fungal growth after 3 months of incubation were processed for determining their moisture content. To test the viability of the fungus in these latter treatments, the dried-up agar

Evaluation of almond varieties for anthracnose resistance. Inoculation experiments using selected almond varieties were either conducted in the field or in the laboratory using detached fruit. In addition, the natural disease incidence was evaluated in an almond variety trial in Butte Co. For field inoculations, almond fruit were wounded, branches were sprayed with conidial suspensions (10^5 spores/ml) of *C. acutatum* genotype G or P, and branches were then bagged overnight. Disease incidence on leaves and severity on fruit were evaluated after 4-5 weeks. For laboratory inoculations, almond fruit were collected from orchards in Fresno or Bakersfield. Fruit were wound-inoculated with 20 μ l of 10^5 spores/ml of almond anthracnose genotype P or G and incubated for 14 days at 20C, >95% RH. For disease evaluations the outer lesion diameters were measured. For spray-inoculations of non-wounded fruit, fruit were sprayed with 10^5 spores/ml of almond anthracnose genotype P. Fruit were incubated for 14 days at 20C, >95% RH, and lesions were then evaluated and rated on a scale from 0 (no lesions) to 2 (many lesions).

Field and laboratory evaluation of fungicides and evaluation of cultural practices for the development of management strategies. For laboratory evaluations of experimental compounds for control of almond anthracnose, almond fruit (cultivars Butte or Wood Colony) were harvested, surface sterilized, air-dried, wounded, and treated with fungicides using an air-nozzle sprayer. After air-drying, the fruit were inoculated with 20 μ l of a conidial suspension (10^5 spores/ml) of almond anthracnose genotype P into the wounds. The outer lesion diameters were recorded after 14 days of incubation at 20 C, >95% RH. In field trials the following compounds were evaluated in selected spray programs using rotations and tank mixes: Abound (azoxystrobin), Indar (fenbuconazole), Rally (myclobutanil), Break (propiconazole), Elite (tebuconazole), Captan (captan), Bravo (chlorothalonil), Elevate (fenhexamid), and Flint (a numbered strobilurin compound). To evaluate the effect of pruning, trees in a commercial orchard were pruned in 1997 to remove all dead twigs and branches larger than 1 cm in diameter and trees were treated with six applications of Captan.

RESULTS AND DISCUSSION

Epidemiology and characterization of Colletotrichum spp. isolates. A major disease outbreak occurred in mid to late spring (April-June) during the 1998 growing season when the warm and unusual wet spring and early summer weather due to El Niño caused highly conducive conditions for disease development. Although disease levels had been very low in 1997, the fungus still could be isolated from mummified fruit, stressing the importance of fruit mummies as inoculum sources for new spring infections. Although early spring rainfall occurred, the disease was not observed until April, indicating that temperature-wetness relationships are critical for disease development. Overall, isolates were obtained from sunken orangish fruit lesions, irregular necrotic or watery lesions of leaves, peduncles of diseased fruit, and other woody tissues of spurs and branches with dieback symptoms. Generally, isolations from fruit showing sunken, orangish lesions were highly successful with recovery rates of 95.6% (Fig. 1). High recovery rates (81%) were also obtained with peduncles, whereas isolations from leaf and woody tissue were significantly less ($P < 0.05$) successful (11.6%, 13.6%, and 6.3% recovery rates for spurs, leaves and wood, respectively) regardless of extensive branch dieback and foliar chlorosis (Fig. 1). Isolations from tissues other than fruit indicate that the amount of tissue infected by the pathogen is more extensive than previously considered. The low recovery rates from leaves and woody tissues could mean that the pathogen often does not invade these types of tissues. Isolations from branches, however, may have been obscured by extensive growth of secondary organisms. Considering leaf tissues, isolations generally were not obscured by secondary organisms, except by the omnipresent species of *Aureobasidium*. Leaf isolations were more successful early (spring) and late (fall) in the season

and we speculate that this is because at these times the fungus is able to infect leaf tissue due to favorable weather conditions. During the summer, however, host responses to secondary metabolites could be responsible for yellowing leaves and branch dieback. We previously have been able to demonstrate the *in vitro* production of phytotoxic compounds by the almond anthracnose fungus. Therefore it is conceivable that some of the disease symptoms on leaves and branches are caused by toxins that are transported from infected blossoms and fruit into the peripheral parts of the tree. Phytotoxic compounds have been described from other species of *Colletotrichum* and their involvement in symptom development has been suggested by a number of authors. Our research on the phytotoxic compounds of *C. acutatum* from almond is ongoing and we hope to characterize the active substances.

The disease also occurred in orchards that were symptomless in 1997. Both anthracnose sub-populations were identified, often occurring within the same orchard or even on the same fruit. Overall, genotype P was isolated predominantly from diseased almond tissue. The spatial distribution of the two sub-populations was determined within four orchards in Butte Co., two orchards in Stanislaus Co., and one orchard in Merced Co. In all orchards except one orchard in Stanislaus Co. both genotypes were found, however, the proportion of both genotypes differed significantly between orchards (Fig. 2). The proportion of isolates with the pink pigmentation within the population was 97.6%, 83%, 60.7%, and 15% in the Butte Co. orchards, and 100%, 94.3%, and 83.3% in the two Stanislaus Co. orchards and the Merced Co. orchard, respectively. Thus, both sub-populations were found in all orchards except in one orchard in Stanislaus Co. and the relative abundance of the two types varied between orchards. For approximately 20% of the isolates, the genotype was also determined by RAPD analysis. In these studies all isolates with gray colonies had banding patterns specific for this gray type, whereas all isolates with pink colonies had banding patterns specific for the pink type. All members of each genotype were found to be identical.

We continued to characterize both anthracnose sub-populations and we determined their temperature optima for growth in the laboratory (Fig. 3). Optimal growth rates for both sub-populations were observed at 25°C with a radial growth of 3.5 mm ± 0.1 mm/day for the gray type and 3.6 mm ± 0.2 mm/day for the pink sub-population. At 20°C both sub-populations grew at a rate of 3.0 mm ± 0.1 mm/day. At 10, 15 and 30°C significant differences ($P < 0.05$) in growth were evident between the two sub-populations. The gray cultures grew faster at 10 and 15°C as compared to the pink cultures (0.86 and 0.24 mm and 1.7 and 1.27 mm, for 10 and 15°C, respectively). At 32°C, however, growth of the gray cultures almost completely stopped (0.5 mm daily growth), whereas the pink cultures still grew at a rate of 1.6 mm ± 0.2 mm/day. At 36°C both, pink and gray cultures, did not show any growth during the experimental period of 5 days. Consequently, genotype P may be better adapted to warmer environments, whereas genotype G grows better in cooler weather conditions.

Postharvest damage of almond kernels in storage. Postharvest damage of almond kernels in storage was first reported in 1997. The pathogen was isolated from processed kernels in storage, predominantly from kernels with internal brown or purplish discolorations. Recovery rates from shriveled or gummy kernels were much lower. In order to define environmental conditions that lead to postharvest kernel damage, almond kernels that were inoculated in the laboratory either with spores or mycelium of the fungus were incubated for up to three months at selected humidities. At kernel moisture levels higher than 13.1% kernel colonization by the fungus occurred after 7-20 days following either inoculation method, and most kernels in each treatment were colonized (Fig. 4). No growth of the fungus was observed at or below a kernel moisture content of 10.1%. The fungal mycelium, however, was still viable at a kernel moisture content of as low as 4.3% after the same time period. This latter moisture content was the lowest value obtained with any of the treatments in the experiments. These results indicate that postharvest

damage to almond kernels was probably the result of preharvest epidemics that were not managed. Since the moisture content of almond kernels in storage is kept between 5% and no more than 10%, new infections are unlikely to occur in storage. We cannot rule out, however, that fungal growth may occur from pre-existing infections over long periods (up to 1 year) in storage. Additional studies are in progress.

Evaluation of almond varieties for anthracnose resistance. Natural disease incidence of anthracnose was evaluated in an almond variety trial in Butte Co. Varieties evaluated were Rosetta, Wood Colony, Nonpareil, Sonora, Padre, Butte, Price, Carmel, 2-19E, 13-1, 2-43W, 1-102W, Aldrich, and Mission. Of these almond varieties, Wood Colony, Sonora, 13-1, 2-43W, and 1-102W had the highest incidence of anthracnose fruit infections as compared to Nonpareil and the other varieties (Fig. 5). Anthracnose has not been observed before in this five-year old orchard. Anthracnose leaf infections were also observed among varieties, however, the incidence differed among the varieties as compared to the incidence of fruit infection. Non-wound inoculation studies with detached fruit correlated with field observations from most varieties. Wood Colony, Butte, and 13-1 had higher severity ratings than Nonpareil, Padre, and Carmel (Fig. 6). Wound inoculation studies using detached fruit indicated that most varieties were very susceptible to anthracnose infection (Fig. 7). In 1997 and 1998, wound-inoculation studies with attached nuts in the field showed greater differences among varieties but results did not correlate with field observations on the natural incidence. Thompson, NePlus Ultra, Nonpareil, Sonora, Peerless, and Merced had the highest disease severity in these wound-inoculation studies. Thus, identification of resistance among varieties will be dependent on non-wound inoculation techniques and natural incidence studies.

Field and laboratory evaluation of fungicides and evaluation of cultural practices for the development of management strategies. Alternative fungicides in addition to the currently registered compounds need to be identified in order to design rotation programs using fungicides with different modes of action. These rotation programs are more likely to reduce the potential of development of resistant pathogen populations. In continued laboratory-screening studies using fungicide-treated almond fruit, disease severity was lowest using Abound (azoxystrobin), Medallion (fludioxonil) and fluazinam as compared to Flint (a strobilurin compound), cyprodonil and fenhexamid at the rates evaluated.

Fungicides evaluated in field trials included: Abound (azoxystrobin), Rally (myclobutanil), Elite (tebuconazole), Break (propiconazole), Indar (fenbuconazole), Bravo (chlorothalonil), Elevate (fenhexamid) and Flint. In single fungicide/six application programs, Abound, Flint, Break, Elite and Bravo were the most efficacious compounds (Fig. 8). None of the fungicides, however, eradicated the disease. In additional field trials evaluating rotations and tank mixes of Break, Elite, Rally, and Abound with either Captan or Bravo, all treatments were effective in reducing the incidence as compared to the non-treated check (Fig. 9). Abound-Captan rotations had the lowest disease incidence. In other commercial fungicide programs, mixtures or rotations of Captan and Break, as well as repeated Captan applications were also very effective when six applications were applied from late February (bloom) through May (Fig. 10). Brown rot programs of Rally and Captan during bloom and an anthracnose program of Break and Break/Captan or Ziram in late spring significantly increased yield of healthy kernels as compared to non-treated trees in a commercial orchard (Fig. 11).

The effect of pruning on the incidence of anthracnose was evaluated in a commercial orchard. When trees were pruned in 1997 to remove all dead twigs and branches larger than 1 cm in diameter and trees were treated with an six applications of Captan, disease incidence was significantly reduced as compared to non-pruned trees (Fig. 12).

A suggested guideline for management of almond anthracnose and other spring time foliar fungal diseases of almond in California is shown in the attached Table. The number of

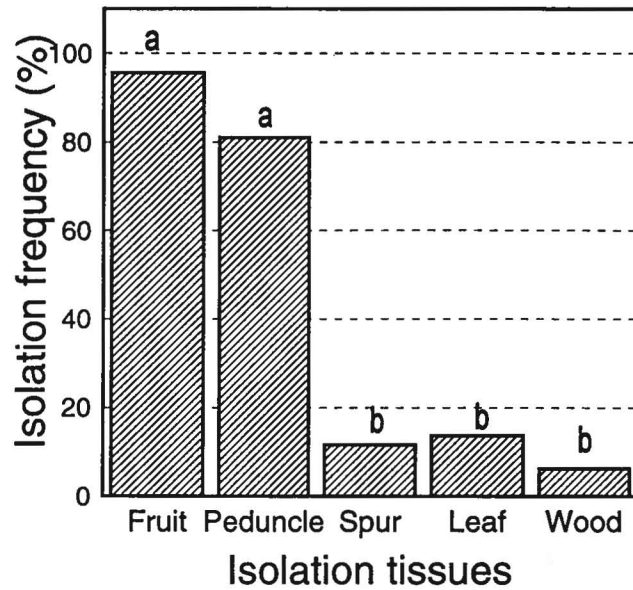
petal fall and mature fruit applications depends on environmental conditions (warm, wet weather). Under conducive conditions, applications for anthracnose should be made in 10-12 day intervals.

In 1999, we expect to have a Section-18 emergency registration of propiconazole (Break 3.6EC) for management of almond anthracnose. This will be available for use from bloom to 90 days PHI. Additionally, with the captan label being revised for use only up to five-weeks-after petal fall, we expect to have a Section 24C, Special Local Needs registration of Abound for management of anthracnose and Alternaria leaf spot by mid-April 1999. Thus, after the 5-wk after petal fall deadline for most fungicide applications on almond in California, we will have two materials Abound and Break that we could use in rotation programs to manage disease and to reduce the risk of the development of resistant populations of target organisms to any one fungicide (See Brown Rot Report for overview of fungicides for management of almond diseases).

Guide to management of anthracnose with fungicides:

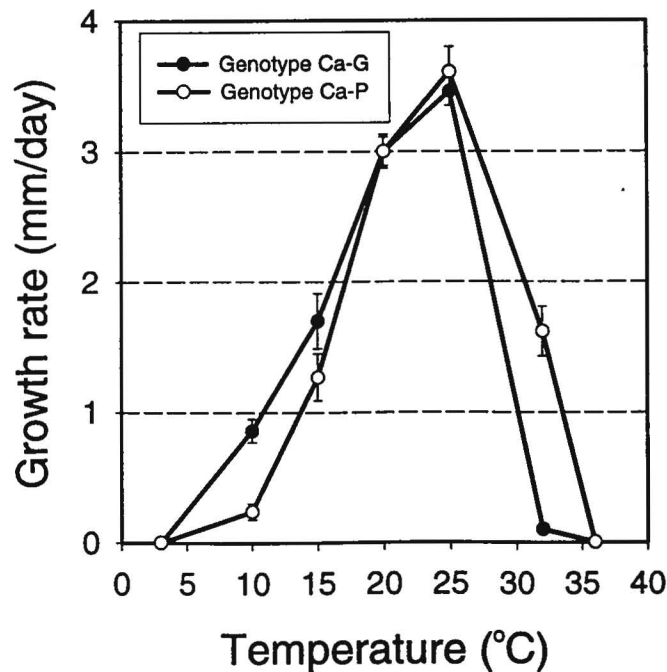
- 1) No fungicide program will eradicate the disease.
- 2) Multiple applications of fungicides are required during conducive wet, warm periods during the spring.
- 3) Preventative and protective programs are the best way to manage the disease based on early detection (Do not wait until you have a major problem).
- 4) Rotations or mixtures of different classes of fungicides (Break, Abound, Captan represent three different fungicides classes) reduces the risk for resistant populations of the fungal pathogen to develop.
- 5) Pruning dead and diseased wood in the summer and fall seasons improves fungicide efficacy in the following spring season.

Fig. 1: Isolation frequency of *Colletotrichum acutatum* from selected tissues of almond



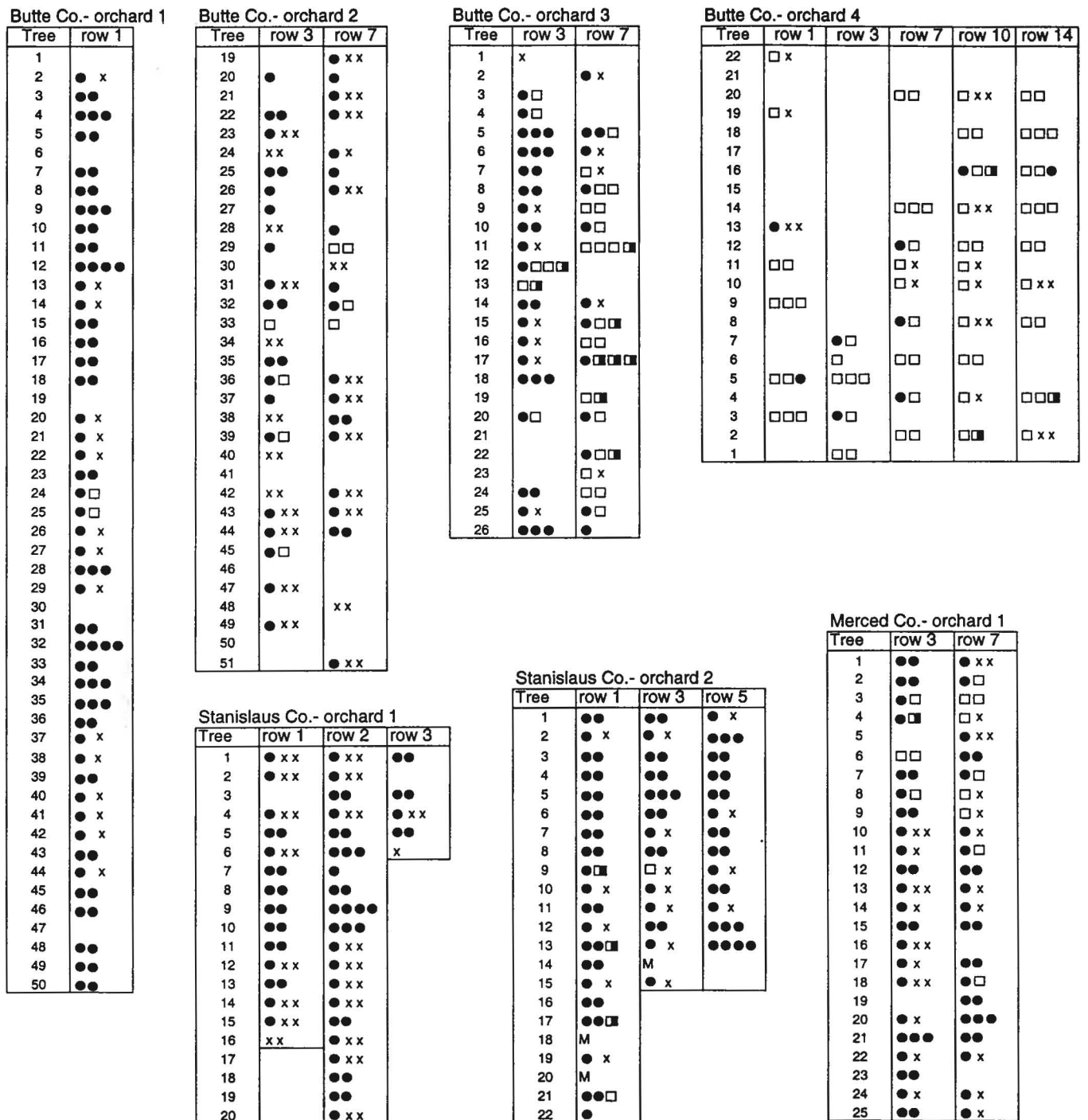
Infected tissue was surface-sterilized for 1 min with 400 mg/L hypochlorous acid and plated out on PDA amended with rifampicin (20 mg/L) and ampicillin (130 mg/L). The isolation frequency is based on the number of tissue pieces plated out.

Fig. 2: Temperature-growth relationships of almond *C. acutatum* genotypes P (pink colonies) and G (gray colonies)



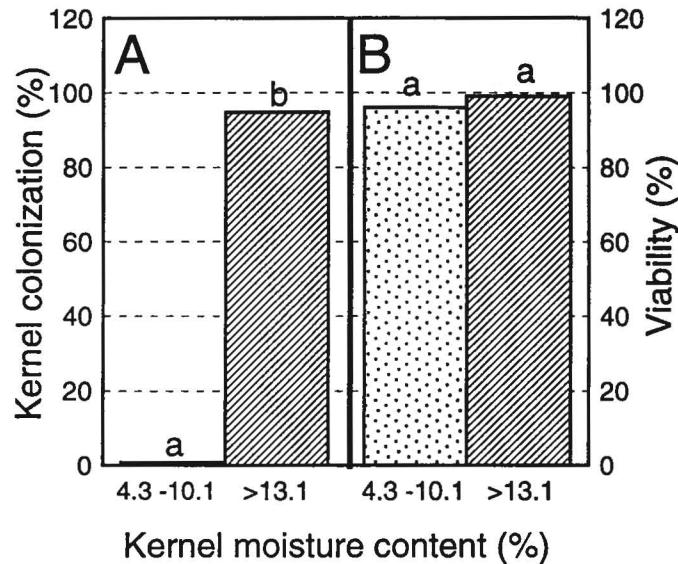
Each data point represents the average growth rate/day (mm) on PDA of seven isolates of each of the two genotypes. Growth rate was recorded over a 5-day period at each temperature.

Fig. 3: Frequency of occurrence of the two *C. acutatum* sub-populations within seven almond orchards in California



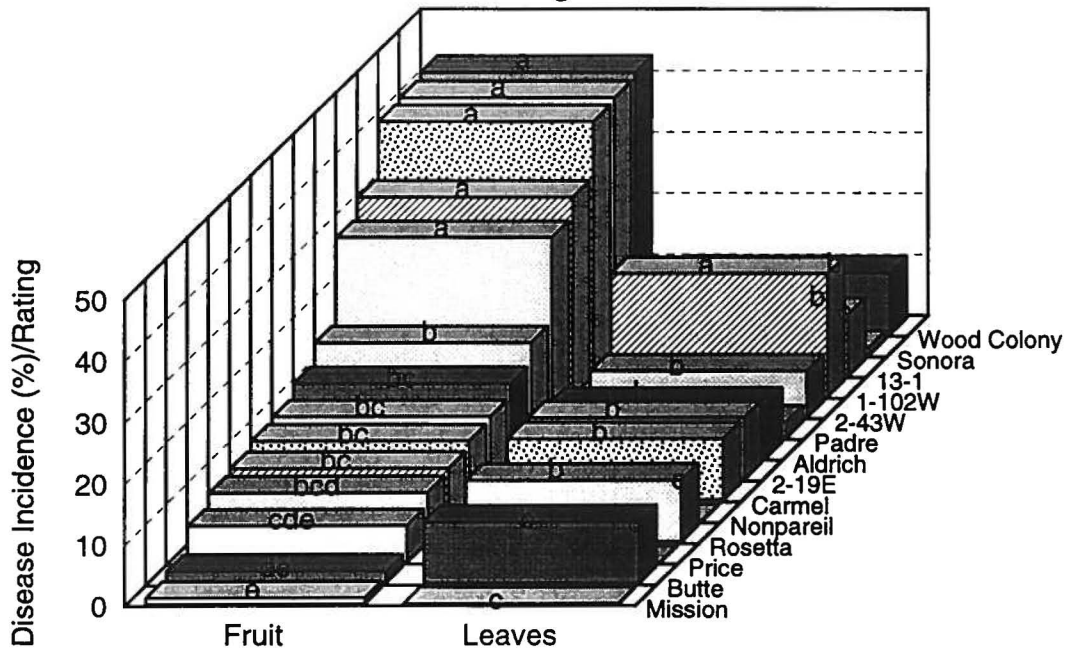
Fifty to ninety isolates were obtained from 100 mature fruit with anthracnose symptoms collected from 38-46 trees/orchard (1-4 fruit/tree). Subpopulations as determined by cultural pigmentation (pink vs. gray) were verified for approximately 20% of the isolates by RAPD analysis.

Fig. 4: Colonization of almond kernels by *C. acutatum* and viability of inoculum at different kernel moisture levels
 - Laboratory studies -



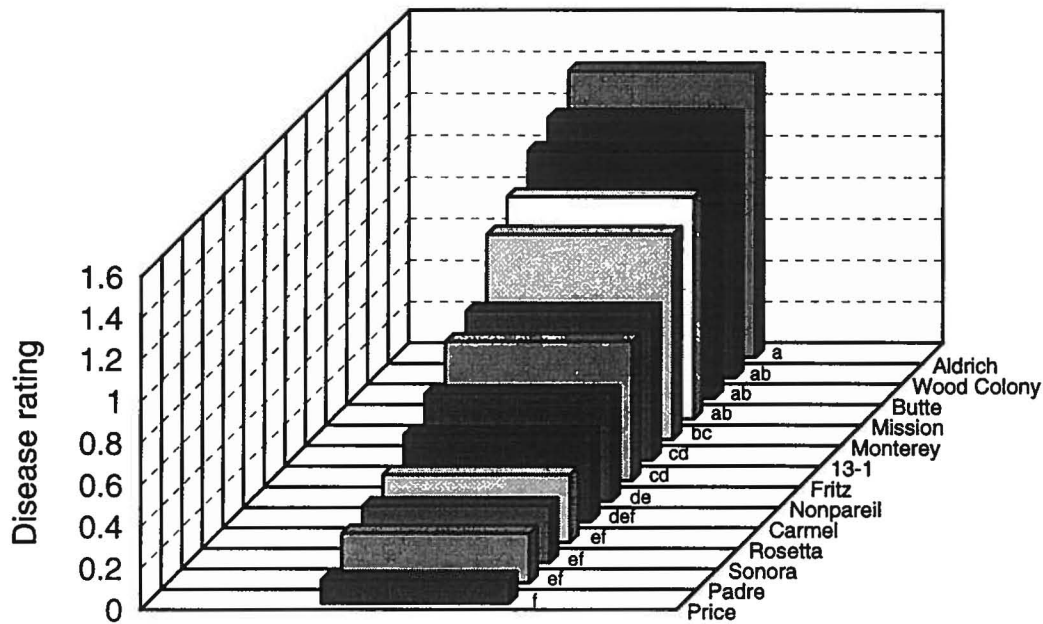
Almond kernel halves were incubated in plastic boxes above selected salt or sugar solutions to obtain and maintain different humidities. Kernels were incubated for up to 3 months and moisture contents were determined when fungal growth was observed or at the end of the experimental period.

Fig. 5: Natural incidence of anthracnose on fruit and leaves of almond varieties at Chico State Farm variety trial
 - 1998 Growing Season -



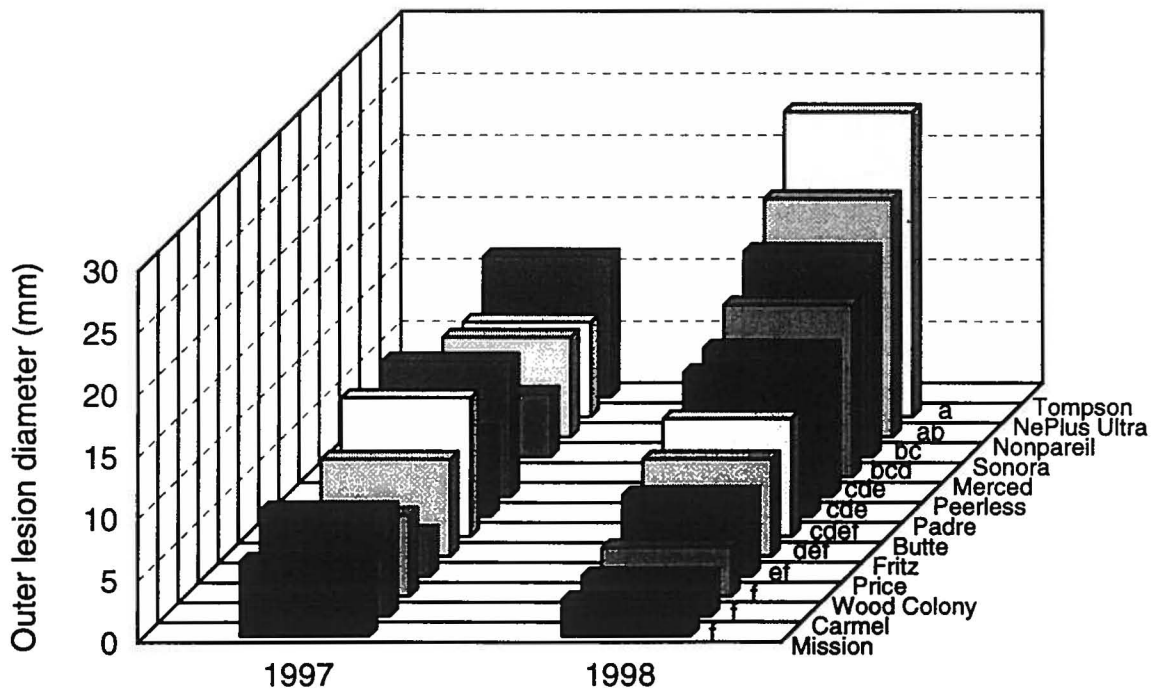
Fruit and leaf symptoms were evaluated on four single-tree replications for each variety.

Fig. 6: Spray-inoculation of almond fruit of selected varieties with *C. acutatum* in the laboratory



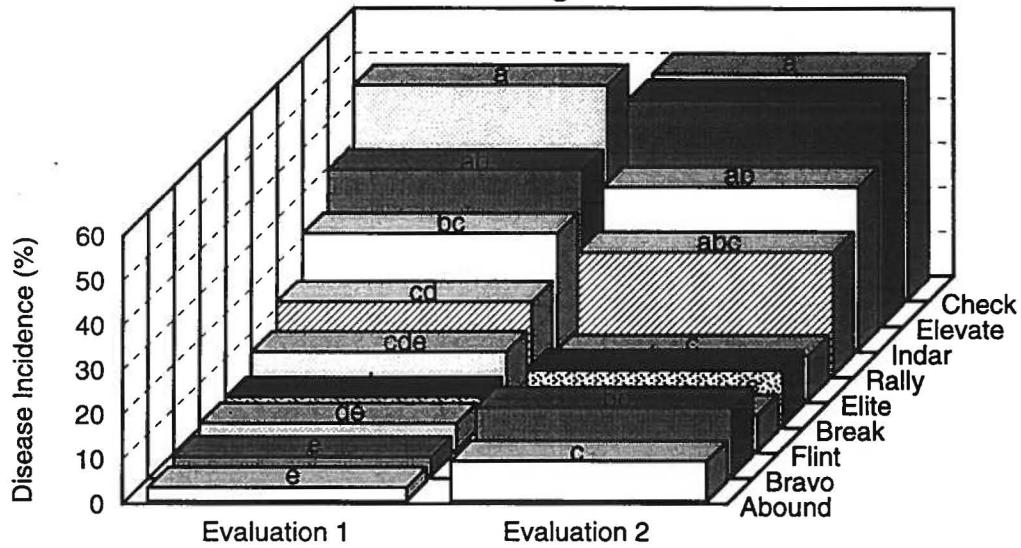
Almond fruit were harvested from an orchard in Kern Co. and were spray-inoculated onto non-wounded fruit with 10^5 spores/ml of *C. acutatum* genotype P. Fruit were then incubated at RH >95%, 20C for 14 days. Lesions were evaluated and rated on a scale from 0 (no lesions) to 2 (many lesions, extending into mesocarp and kernel).

Fig. 7: Wound-inoculation of almond fruit of selected varieties with *C. acutatum* in the field



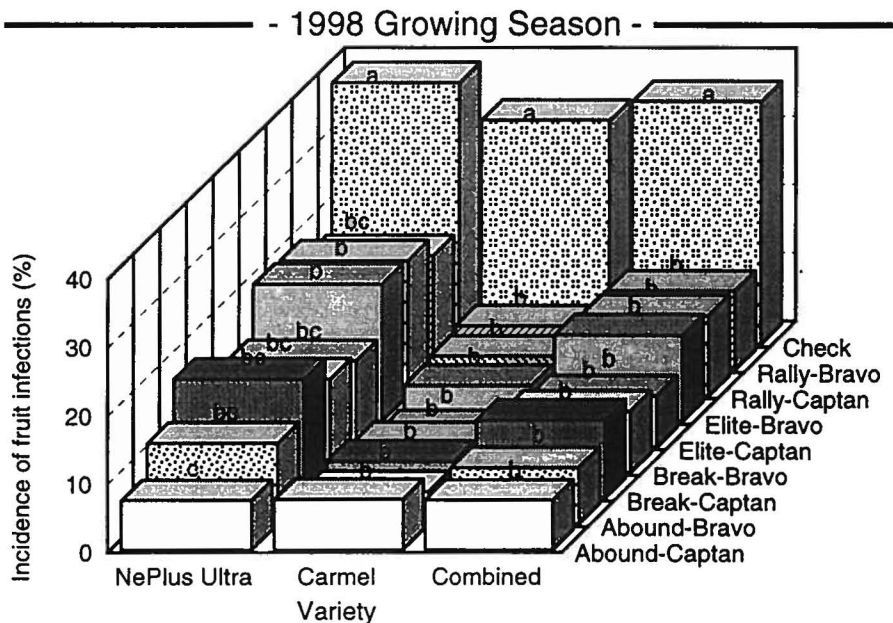
Wounded almond fruit were spray-inoculated with 10^5 spores/ml of almond anthracnose genotype P. Branches were bagged up overnight. The outer lesion diameters were measured after 4-5 weeks. In 1997 the experiment was conducted at two locations, whereas in 1998 only one location was done.

Fig. 8: Efficacy of fungicides for management of anthracnose on Price almond in Butte Co.
 - 1998 Growing Season -



Evaluation 1 was done after 5 applications, and evaluation 2 after 6 applications of each fungicide. Treatments were applied using a back-pack sprayer (100 gal/A).

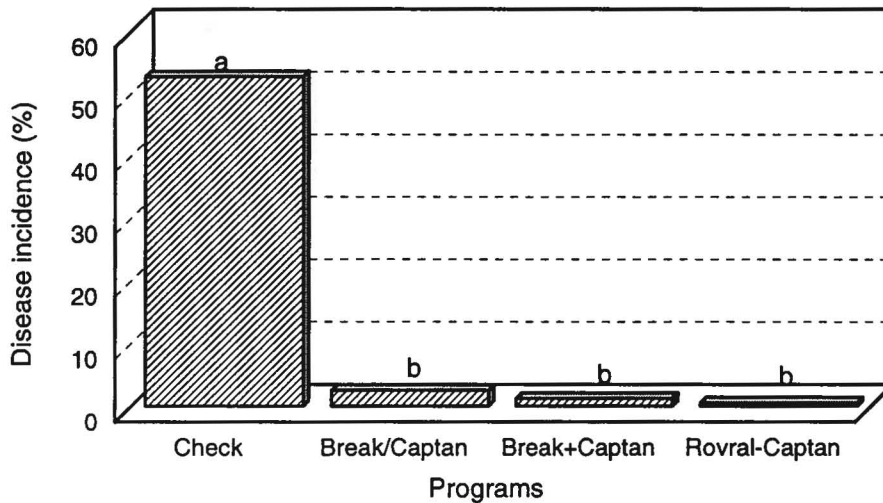
Fig. 9: Efficacy of fungicides for management of anthracnose on Carmel and NePlus Ultra almond in Merced Co.
 - 1998 Growing Season -



Evaluation of each cultivar was done after 5 applications of each fungicide program beginning at pink bud. Treatments were applied using an air-blast sprayer (100 gal/A). The program was: 2 single-site, 1 multi-site, and 2 single-site mode of action fungicide applications for both varieties except the second application was a mixture for the Carmel variety.

Fig. 10: Efficacy of commercial fungicide programs for management of anthracnose on Price almond

- 1998 Growing Season -

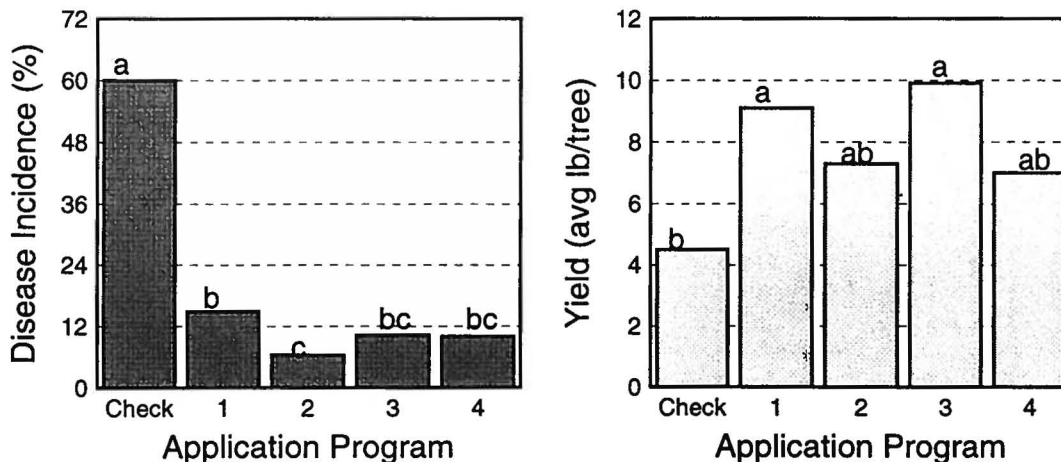


Treatments were applied from pink bud to mature fruit (Feb. - May) for a total of seven applications that were applied every two weeks using an air-blast sprayer (100 gal/A):

- 1) One fungicide: Rovral (PB), Captan - 8 lb (every application after)
- 2) Alternation: Break - 4 oz/ Captan - 8 lb
- 3) Mixture: Break - 2 oz+Captan - 4 lb

Fig. 11: Efficacy of Fungicide Application Programs for Management of Almond Anthracnose in Merced Almonds with High Levels of Disease

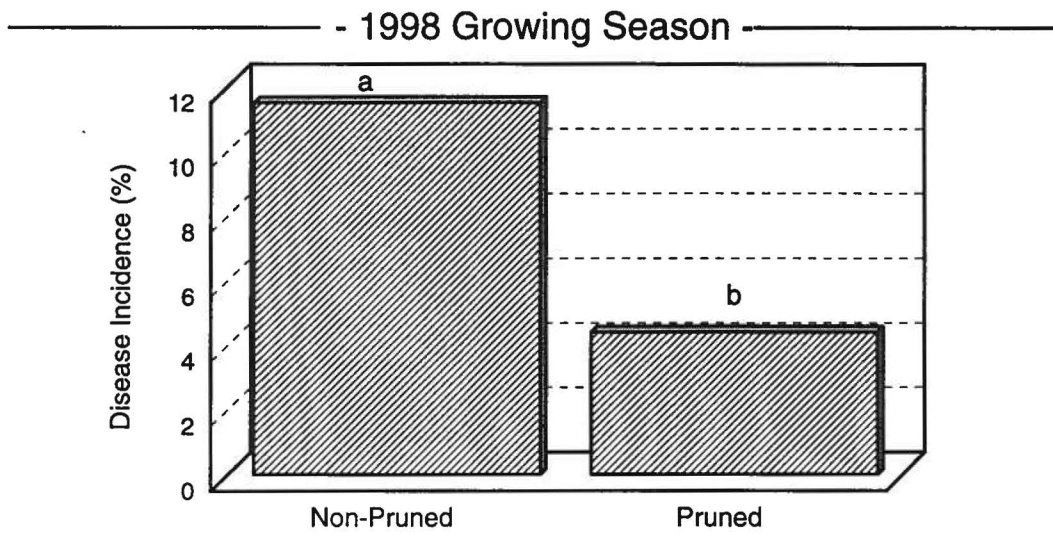
1998 Growing Season



Treatments were applied at full bloom, petal fall, 5-wk after petal fall, mature fruit and were applied using an air-blast sprayer (100 gal/A):

- 1) Rally 4 oz/Captan 5 lb; Rally/Captan; Break 4 oz/Ziram 6 lb; Break
- 2) Rally 8 oz/Captan 10 lb; Rally/Captan; Break/Ziram; Break
- 3) Rally 8 oz; Captan 10 lb; Break/Ziram; Break
- 4) Break, Break/Ziram; Captan 10 lb; Break

Fig. 12: Effect of pruning on the incidence of anthracnose in a commercial Price almond orchard



- * - Price almond trees were treated with Rovral at pink bud followed by six applications of captan (8 lb/A) at two week intervals (Feb.-May).
- ** - In 1997, trees were pruned to remove all dead twigs and branches larger than 1 cm in diameter.