Inhibition of Mycelial Growth of *Monilinia* Species and Suppression and Control of Brown Rot Blossom Blight of Almond with Iprodione and E-0858

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ABSTRACT

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The fungicide iprodione and the heterocyclic anilide E-0858 were compared with benomyl for in vitro inhibition of mycelial growth of *Monilinia laxa* and *M. fructicola*, for suppression of anther infection of blossoms in the laboratory, and for control of brown rot blossom and twig blight in the field. All 100 isolates of *M. laxa* obtained from an orchard in Solano County, California, were sensitive to benomyl (no growth at 1.0 μg a.i./ml), whereas all 100 isolates from an orchard in Fresno County, California, were resistant to benomyl (growth at 1.0 μg a.i./ml). Effective concentrations (EC_{50}) of benomyl in Czapek's medium for inhibition of mycelial growth of *M. laxa* or *M. fructicola* averaged 0.09 μg/ml for sensitive isolates and 0.77 μg/ml for resistant isolates. Both iprodione and E-0858 were active against benomyl-sensitive and benomyl-resistant isolates of *M. laxa* and *M. fructicola*. Iprodione incorporated into PDA or Czapek's medium was inhibitory to both *M. laxa* and *M. fructicola*, with EC_{50} values between 0.52 and 0.75 μg a.i./ml. EC_{50} values of E-0858 incorporated in Czapek's medium were 0.30–0.36 μg a.i./ml for *M. laxa* and 0.89–0.98 μg a.i./ml for *M. fructicola*, whereas EC_{50} values of E-0858 in potato-dextrose agar were >450 μg a.i./ml for inhibition of either *M. laxa* or *M. fructicola* mycelia. In laboratory studies, anther infection was suppressed when open blossoms were sprayed with E-0858 or iprodione within 24 hr after inoculation with a benomyl-sensitive isolate of *M. laxa*. In field studies conducted in almond orchards with benomyl-sensitive or benomyl-resistant populations of *M. laxa*, applications of E-0858 or iprodione at pink bud (closed blossoms) and full bloom (opened blossoms) effectively reduced brown rot twig blight. Mycelial inhibition and suppression of disease development contribute to the high efficacy of iprodione and E-0858 in control of brown rot blossom and twig blight of almond.

Additional keywords: benzimidazoles, dicarboximides, *Prunus dulcis*, pyridines

Brown rot blossom and twig blight, caused by *Monilinia laxa* (Aderhold & Ruhland) Honey and *M. fructicola* (G. Wint.) Honey, is a devastating disease of almond (*Prunus dulcis* (Mill.) D. Webb) and other stone fruit crops and causes severe crop losses annually (19). In California, both *M. fructicola* and *M. laxa* have been reported as pathogens of almond blossoms, but *M. laxa* is more common (8). Because the stigma, anthers, and petals are the most susceptible tissues of almond blossoms (7), timing of fungicide applications in relation to blossom development is critical. In California, depending on weather conditions and fungicides used, blossom blight of almond is prevented with one to three applications of fungicides.

The use of protectant or nonsystemic fungicides such as captan for control of blossom diseases of stone fruit crops requires a minimum of two applications to provide continuous protection of susceptible blossom tissues. In almond, a pink bud (closed blossom) spray and a full bloom (opened blossom) spray of nonsystemic fungicides provide protection of petals and internal floral tissues, respectively. With the introduction of benomyl, however, one application of this fungicide at pink bud reduced almond blossom blight by 92%, and the fungicide was translocated systemically into the nonexposed internal blossom tissues (pistils and stamens) (12). Furthermore, Ogawa et al (10) demonstrated that a single application of benomyl was equivalent to two applications of a protectant fungicide. After its registration, benomyl was used almost exclusively because of its high level of efficacy in controlling blossom blight. In an effort to reduce the development of fungicide-resistant populations, benomyl was used in combination with protectant fungicides. However, benomyl-resistant populations of *M. laxa* and *M. fructicola* have become widespread throughout California (6,9,16) and other stone fruit production areas (3,17,18,20,22). Resistant populations remained stable for several years after benomyl was withdrawn from disease control programs (1,2). Thus, in some locations, the efficacy of benomyl for brown rot control has greatly diminished and alternative fungicides are used for blossom blight control.

With the establishment of benomyl-resistant populations of *Monilinia* spp. in orchards, efficacy of alternative fungicides and optimum timing for these fungicides in commercial orchards required study. Osorio et al (11,12) characterized the local systemic action of iprodione and a new class of fungicides, represented by E-0858 (ICIA-0858, SC-0858), in almond blossoms and found that both fungicides were translocated from exposed outer to enclosed inner parts of blossoms and fruit. The objectives of this study were to determine: 1) the effect of iprodione and the experimental anilide E-0858 on mycelial growth of *Monilinia* spp., 2) their efficacy in suppressing blossom infections caused by *M. laxa* in the laboratory, and 3) their efficacy in controlling brown rot twig blight in almond orchards with and without benomyl-resistant populations of *M. laxa*.

MATERIALS AND METHODS

Fungal isolates. One hundred brown-rot blighted twigs with sporodochia were collected from Drake almond trees in Solano County in 1987 and from Ruby almond trees in Fresno County in 1988. The samples were individually collected in plastic bags, and one sporodochium per twig was plated on lactic acid potato-dextrose agar (LAPDA). This medium was made by adding 2 ml of 25% lactic acid to cooled, autoclaved Difco PDA. Cultures of *M. laxa* were obtained for each isolation. Sensitivity of each isolate to benomyl was assessed by transferring 4-mm-diameter agar disks, taken from the edge of 3-day-old colonies grown on LAPDA, onto petri dishes with PDA amended with benomyl at 1 μg a.i./ml. Three single-plate replications were used for each isolate. Two representative isolates, one from the Solano orchard (S1-87) and the other from the Fresno orchard (F1-88), were selected and used in the studies described below. The isolates were stored on PDA slants at 5 C and grown on PDA at 23 C in the dark for production of mycelium and on oatmeal agar at 23 C under fluorescent light for production of conidia. Benomyl-sensitive (MUK-1) and benomyl-resistant (489-81) isolates of *M. fructicola* were obtained from the culture collection at the University of California, Davis, for comparative studies.

Assays of mycelial growth inhibition. The inhibition of mycelial growth was

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determined by placing 4-mm-diameter agar disks, taken from the edge of actively growing 3-day-old colonies on PDA, onto petri dishes containing PDA or Czapek's medium amended with 0, 0.1, 0.3, 0.5, 1.0, 1.5, or 2.0 μg/ml of iprodione (Rovral 50WP) or E-0858 50WP (*N*-[5-(2-methoxypyridinyl)]-cyclopropane-carboxamide, M_r = 192, Zeneca AgProducts, Wilmington, DE). Benomyl (Benlate 50WP) was incorporated in Czapek's medium at 0, 0.05, 0.08, 0.1, 0.3, 0.5, 1.0, or 1.5 μg a.i./ml. Additionally, PDA medium was amended with 0, 1, 10, 100, or 1,000 μg a.i./ml of E-0858. Media amended with iprodione or E-0858 were made by adding suspensions of the fungicides in sterile water to autoclaved agar media cooled to 50 C; benomyl was added to media before autoclaving. Radial growth was assessed after incubation in the dark for 4 days at 23 C by measuring the distance from the edge of the inoculum plug to the advancing margin of the colony. The percent inhibition of mycelial growth was determined by comparing radial growth measurements of treated cultures with fungicide-free medium (check). Linear regression analysis was performed on mean values, and the $\log_{10}$ of the fungicide concentration was regressed on the probit of percent mycelial inhibition to obtain EC_{50} values for each compound (15). Each treatment was replicated five times in each of two experiments.

Laboratory assay of disease suppression. Drake almond shoots with closed blossoms were excised from 5- and 6-yr-old trees. The cut ends were placed in water and held at 25 C until the blossoms were open. Fully opened blossoms were detached from the shoots, placed in plastic containers (100 × 235 × 315 mm) containing sterile moist sand, inoculated by spraying them with an aqueous conidial suspension (1.2×10^4 conidia per milliliter) of a benomyl-sensitive isolate of *M. laxa* (S1-87), and exposed to wetness periods of 1, 12, 24, or 36 hr at 20 C. Blossoms were sprayed with E-0858, iprodione, or benomyl at 300 μg/ml following each wetness period and incubated for 3 days at 20 C, RH >95%. Blossoms not treated with fungicides were sprayed with sterile water and functioned as controls. Disease was evaluated on a severity scale of 0–4, where 0 = no visible symptoms, 1 = 1–25%, 2 = 26–50%, 3 = 51–75%, and 4 = 76–100% anthers/blossoms infected. The experiment was repeated once, and each treatment was replicated four times (10 blossoms per replication). Data were analyzed using general linear model and regression analysis procedures of SAS (15).

Field evaluation of fungicides. Field experiments were conducted during 1988 and 1989 in a 5-yr-old Drake almond orchard in Solano County and a 12-yr-old Ruby almond orchard in Fresno County. Treatments of E-0858, iprodione, or benomyl, each at 1.12 kg a.i./ha, were applied to closed blossoms (pink bud), to fully opened blossoms (full bloom), or at both stages. Nonsprayed trees served as controls. The fungicides were mixed in water and applied with a handgun sprayer at a pressure of 1,694 kPa and volume of 3,141 L/ha. Each treatment was replicated five times (single-tree replications) in a randomized complete block design. Disease severity was based on the percentage of blighted twigs 3 wk after the full bloom application. For this evaluation, 100 randomly selected twigs from each quadrant of a tree were examined for disease symptoms. Values from the four quadrants were averaged to obtain the percentage of diseased twigs per tree. Meteorological data were recorded during the test periods in each orchard using a Campbell 21X micrologger (Campbell Scientific Inc., Logan, UT), except for 1988 at the Fresno site, when data were obtained from the Fresno airport weather station located about 10 km northwest of the orchard. For each year and orchard, data were analyzed using a two-way factorial analysis of variance for fungicide and stage of bloom (or time fungicides were applied). General linear model and orthogonal contrasts of main effects of treatments (check vs. fungicides) were performed for each year and orchard (15).

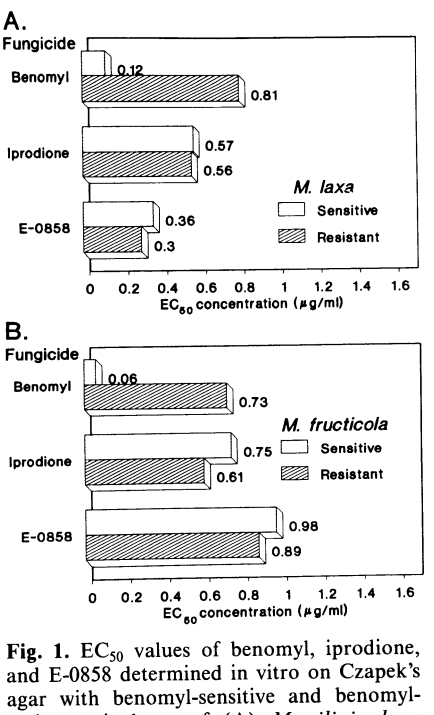

Fig. 1. EC_{50} values of benomyl, iprodione, and E-0858 determined in vitro on Czapek's agar with benomyl-sensitive and benomyl-resistant isolates of **(A)** *Monilinia laxa* (benomyl-sensitive isolate S1-87/benomyl-resistant isolate F1-88) and **(B)** *M. fructicola* (benomyl-sensitive isolate Muk-1/benomyl-resistant isolate 249-81). Values were determined by solving the linear regression equation of $\log_{10}$ fungicide concentration on probit of mycelial inhibition for 50% reduction of growth.

RESULTS

Inhibition of mycelial growth. All isolates of *M. laxa* obtained from the Solano orchard were sensitive to benomyl, whereas all of those from the Fresno orchard were resistant to 1 μg/ml of benomyl. All regression models of the $\log_{10}$ of fungicide concentration on probit of mycelial inhibition in Czapek's medium or PDA for each fungicide evaluated were significant ($P < 0.05$). All regression equations had R^2 values >0.95 except the model for benomyl using the benomyl-sensitive isolate of *M. laxa* (S1-87), where the R^2 value was 0.75. On Czapek's medium, EC_{50} values of benomyl-resistant isolates of *M. fructicola* and *M. laxa* were approximately 10 times greater than those of benomyl-sensitive isolates, whereas benomyl-sensitive and benomyl-resistant isolates of *M. laxa* and *M. fructicola* were similar in their sensitivity to iprodione and E-0858 (Fig. 1).

On PDA, the EC_{50} values of iprodione were similar to those obtained for the fungicide on Czapek's medium (Figs. 1 and 2). However, E-0858 incorporated into PDA was ineffective at low concentrations. EC_{50} values of E-0858 in PDA were >450 μg a.i./ml for both *Monilinia* spp. (Fig. 2).

Disease suppression. Symptoms of anther infection were observed under laboratory conditions after incubation for 24–28 hr. After 3 days, E-0858, iprodione, and benomyl at 300 μg/ml suppressed almond blossom blight development when applied less than 36 hr after inoculation. Models for regressions of disease severity on wetness period for fungicide and nonfungicide treatment times were significant ($P < 0.05$). Disease severity increased linearly with duration of wetness period and the delay of fungicide application in all fungicide treat-

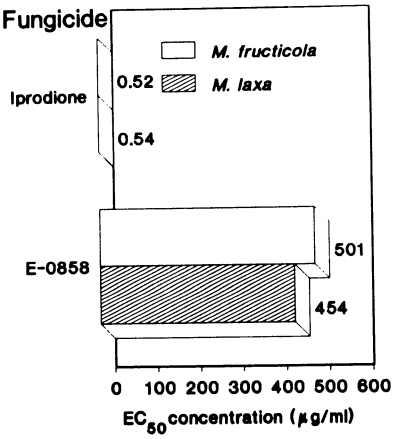

Fig. 2. EC_{50} values of iprodione and E-0858 determined in vitro on PDA agar with benomyl-sensitive isolates of *Monilinia laxa* (isolate S1-87) and *M. fructicola* (isolate Muk-1). Values were determined by solving the linear regression equation of $\log_{10}$ fungicide concentration on probit of mycelial inhibition for 50% reduction of growth.

ments (Fig. 3). Blossoms treated with fungicides after inoculation had lower disease indexes than blossoms not treated with fungicides, as indicated by significantly lower ($P < 0.05$) midpoint and Y-intercept values and significantly higher slopes (Fig. 3). No significant differences ($P > 0.05$) were observed between the slopes and midpoint values of the regressions for blossoms treated with fungicides.

Rainfall and temperatures during field studies. At the Solano site, rainfall was low (<1 mm) and the average daily temperature was 15 C during bloom in 1988 (Fig. 4A); in 1989, rainfall was 67 mm and the average daily temperature was 12 C during a 10-day period of bloom (Fig. 4B). At the Fresno site, rainfall was 22 mm and the average daily temperature was 14 C during bloom in 1988 (Fig. 5A); in 1989, rain fell on 5 days between 3 and 17 March, for a total of 7 mm, and the average daily temperature during this period was 15 C (Fig. 5B).

Fungicide efficacy. At the Solano site in 1988 and 1989, orthogonal contrasts indicated that twig blight was significantly ($P < 0.01$) reduced by each fungicide treatment compared with no treatment. No significant difference ($P > 0.10$) was observed among fungicides (Table 1) or the interaction of fungicides and stage of bloom (or time of fungicide application). However, a significant difference ($P < 0.01$) was observed for stage of bloom when fungicides were applied. In 1988, no differences were observed among pink bud, full bloom, and pink bud + full bloom sprays (Fig. 6A). During the wet bloom period in 1989, the two-spray treatment (pink bud + full bloom) significantly decreased percent twig blight for the benomyl, E-0858, and iprodione treatments from the single full bloom spray. Additionally, for E-0858, disease was significantly less with the pink bud spray than with the full bloom spray (Fig. 6B). A single application at pink bud or full bloom of either of the fungicides decreased twig blight from 43% in the nontreated trees to 7.4, 9.9, and 16.3% for benomyl, E-0858, and iprodione treatments, respectively.

At the Fresno site, where only benomyl-resistant isolates of *M. laxa* were detected, orthogonal contrasts indicated that percent twig blight for each fungicide treatment (including benomyl) was significantly lower ($P < 0.01$) than for the nontreated trees in 1988 and 1989. For both years, significant differences ($P < 0.05$) were observed for fungicides and stage of bloom when fungicides were applied, but no significant differences ($P > 0.05$) were observed for the interaction of fungicide and stage of bloom. In 1988 and 1989, benomyl significantly ($P < 0.05$) reduced the incidence of twig blight compared with the nontreated trees (Table 1). Twig blight was significantly less with iprodione and E-0858 treatments than with the benomyl treatment in 1988 or 1989 (Table 1); no differences were observed between iprodione and E-0858.

In 1988 and 1989, percent twig blight with the two-application (pink bud + full bloom) treatments was similar and consistently lower than with the single pink bud or full bloom application for iprodione and E-0858 (Fig. 7). Efficacy of the full bloom application of iprodione or E-0858 in 1988 and the pink bud application of E-0858 in 1989 was not significantly different from the two application treatment (pink bud + full bloom) in these years (Fig. 7). Regardless of time of application, twig blight incidence was 4.5 and 2.4% in 1988 and 9.1 and 6.5% in 1989 for iprodione and E-0858, respectively. No significant differences, however, were observed between time of application of benomyl, although in 1989 the pink bud + full bloom treatment was numerically lower than the single applications of benomyl (Fig. 7).

DISCUSSION

Our previous and current studies indicate that the EC_{50} values of E-0858 and iprodione for inhibition of mycelial growth on Czapek's medium of both benomyl-sensitive and benomyl-resistant isolates are similar for each *Monilinia*

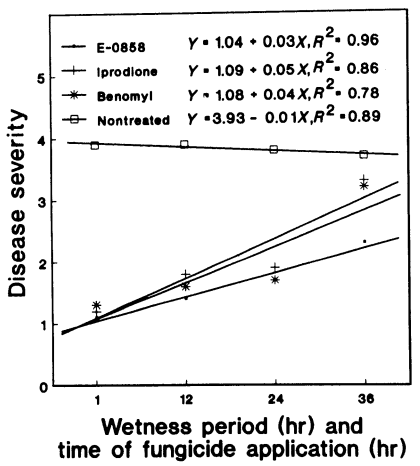

Fig. 3. Regression of disease severity of anther infection on wetness period and time of fungicide application. For each equation, Y = disease severity and X = hours of wetness prior to fungicide application. Blossoms were inoculated with a conidial suspension (1×10^4 conidia per milliliter) of a benomyl-sensitive isolate of *Monilinia laxa* (S1-87), incubated for 3 days (20 C, >95% RH), and evaluated for disease on a scale of 0–4, where 0 = no visible symptom, 1 = 1–25%, 2 = 26–50%, 3 = 51–75%, and 4 = 76–100% of the anthers of each blossom infected. Values are the means of four replications (10 blossoms per replication).

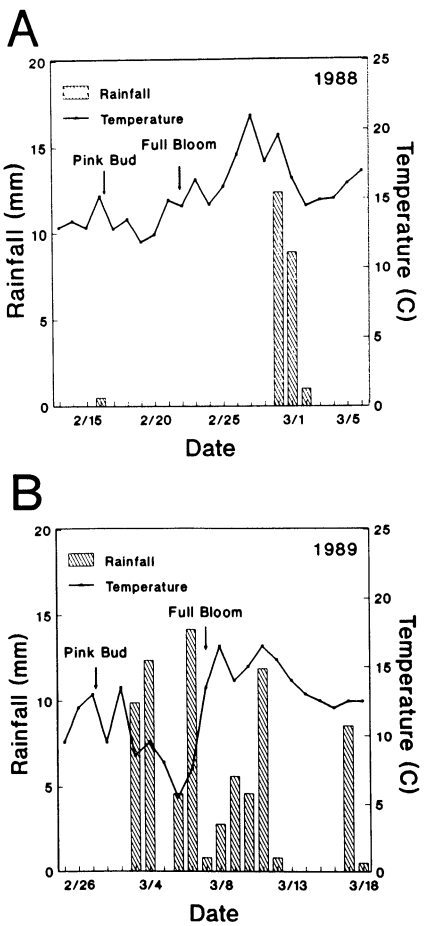

Fig. 4. Rainfall, average daily temperatures, and stage of bloom when fungicides were applied to Drake almond trees in the Solano site during **(A)** 1988 and **(B)** 1989.

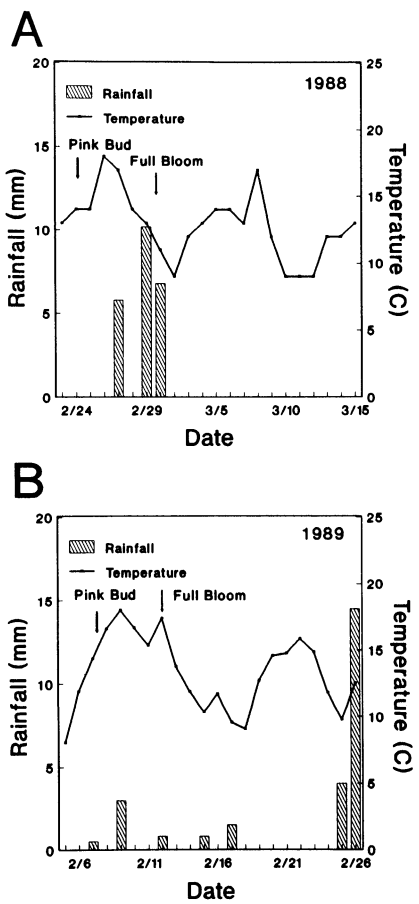

Fig. 5. Rainfall, average daily temperatures, and stage of bloom when fungicides were applied to Ruby almond trees in the Fresno site during **(A)** 1988 and **(B)** 1989.

species (13). The EC_{50} values that we obtained for iprodione in PDA and Czapek's media are similar to those reported by Katan and Shabi (4) for *M. laxa* using PDA. However, when incorporated in PDA, E-0858 was not fungitoxic at low concentrations and had EC_{50} values >450 μg a.i./ml for both species of *Monilinia*. Matheron and Matejka (5) also found that E-0858 (SC-0858) provided control of *Sclerotinia sclerotiorum* comparable to that of iprodione under field conditions. In their in vitro tests with PDA, however, EC_{50} values were 158 and 0.42 μg a.i./ml for E-0858 and iprodione, respectively (5). These data indicate that E-0858 is active against species of *Monilinia* in Czapek's medium but not in PDA. Osorio et al (11) indicated that the solubility of E-0858 in water is >6,000 μg/ml, compared with 13 μg/ml for iprodione. Perhaps PDA provides an alternative biochemical pathway that prevents E-0858 from inhibiting mycelial growth of the fungi evaluated. The ineffectiveness of E-0858 in PDA, however, has not been explained. In screening potential compounds as fungicides, in vitro assays that utilize only one type of medium could eliminate potentially active compounds. Therefore, screening should be done on several types of media and should include in vivo or field tests.

Laboratory tests revealed that E-0858, iprodione, and benomyl were capable of effectively suppressing the development of anther infection in almond blossoms up to 24 hr after inoculation. These results suggest that field applications of these fungicides after an infection period and before symptom development may control blossom blight. Because low temperatures during bloom delay the development of blossom blight (7), the period of field application of these fungicides could possibly be extended from that observed in our laboratory tests conducted at 20 C. In postinoculation tests of potted sour cherry plants, Wilcox (21) demonstrated that sterol biosynthesis inhibitors and dicarboximides (e.g., iprodione) suppressed brown rot blossom blight up to 48 hr after inoculation at 20 C.

Benomyl-resistant populations of *M. laxa* and *M. fructicola* have been reported in California and other areas of North America (1,3,6,9,16,22). The levels of resistance (1–4 μg a.i./ml) in isolates from California are relatively low compared to the levels (300–1,000 μg a.i./ml) reported in isolates from South Carolina (22) and Michigan (3). In orchards with high populations of isolates resistant at 1–4 μg/ml of benomyl, disease control with benomyl or carbendazim has been effective (1,16). In our study, we confirmed that benomyl can reduce twig blight of almond in orchards with benomyl-resistant populations (1 μg a.i./ml) of *M. laxa*. However, the reduction in the incidence of twig blight from

Table 1. Efficacy of benomyl, iprodione, and E-0858 for control of brown rot twig blight of almond in California orchards with benomyl-sensitive and benomyl-resistant populations of *Monilinia laxa*

County	Sensitivity of *M. laxa* to benomyl[x]	Fungicide[y]	Percent twig blight[z] 1988	1989
Solano	Sensitive	Benomyl (Benlate 50WP)	0.6 a	4.2 a
		Iprodione (Rovral 50WP)	1.1 a	11.6 a
		E-0858 50WP	1.0 a	5.8 a
		None	4.3 b	43.7 b
Fresno	Resistant	Benomyl	7.6 b	14.7 b
		Iprodione	2.6 a	5.6 a
		E-0858	1.3 a	3.3 a
		None	12.1 c	21.7 c

[x]All 100 isolates obtained from the Solano orchard did not grow on media containing 1 μg/ml of benomyl (benomyl-sensitive), whereas all 100 isolates from the Fresno orchard grew on media containing 1 μg/ml of benomyl (benomyl-resistant).

[y]Fungicides were applied with a hand-gun sprayer (1,694 kPa and 3,141 L/ha) at a rate of 1.2 kg a.i./ha.

[z]Average percent twig blight was determined 3 wk after full bloom based on percentage of 400 (100 per quadrant) blighted twigs per tree for five single-tree replications for each treatment. For each year and site, values followed by the same letter are not significantly different ($P > 0.05$) based on general linear model and least significant difference mean separation procedures.

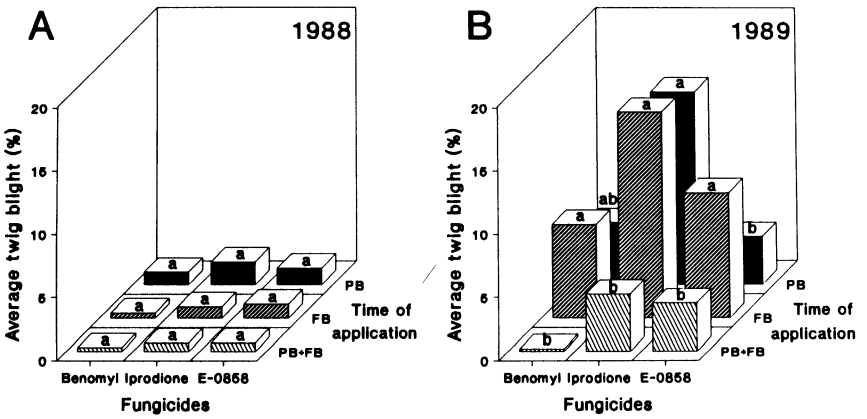

Fig. 6. Influence of time of application of benomyl (Benlate 50WP), iprodione (Rovral 50WP), or E-0858 50WP in reducing average percent twig blight caused by *Monilinia laxa* in the Solano site in **(A)** 1988 and **(B)** 1989. For each fungicide, bars with the same letters are not significantly different ($P > 0.05$) based on analysis of variance and least significant difference mean separation procedures. Fungicides were applied at a rate of 1.2 kg a.i./ha at pink bud (PB) or full bloom (FB) or at both stages (PB+FB) using a handgun sprayer (1,694 kPa and 3,141 L/ha).

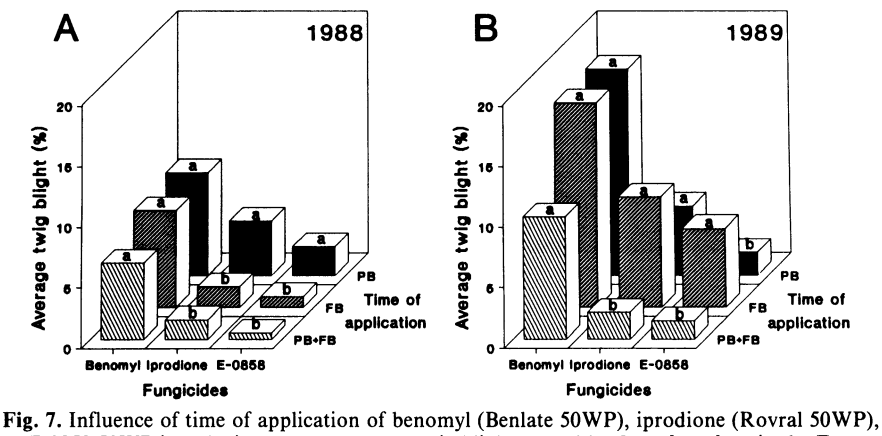

Fig. 7. Influence of time of application of benomyl (Benlate 50WP), iprodione (Rovral 50WP), or E-0858 50WP in reducing average percent twig blight caused by *Monilinia laxa* in the Fresno site in **(A)** 1988 and **(B)** 1989. For each fungicide, bars with the same letters are not significantly different ($P > 0.05$) based on analysis of variance and least significant difference mean separation procedures. Fungicides were applied at a rate of 1.2 kg a.i./ha at pink bud (PB) or full bloom (FB) or at both stages (PB+FB) using a handgun sprayer (1,694 kPa and 3,141 L/ha).

12.1% (nontreated) to 7.6% (benomyl-treated) in 1988 and from 21.7 to 14.7% in 1989 is not sufficient economically, considering the availability and efficacy of iprodione.

In our field experiments, single applications of iprodione and E-0858 applied to closed blossoms (pink bud) significantly reduced disease incidence compared to no treatment. Depending on the orchard and time of rainfall in relation to bloom, a single application may be as effective as a two-spray program for iprodione or E-0858, although E-0858 was consistently better than iprodione. Furthermore, this information supports other research (12) that these compounds are translocated from exposed outer blossom tissues (petals and sepals) to enclosed inner blossom tissues (stamens and pistils). Iprodione or E-0858, when applied to closed blossoms under field conditions, inhibits growth of *M. laxa* in blossom tissue, as demonstrated with benomyl (14), and is highly effective in controlling almond blossom and twig blight in orchards with benomyl-sensitive or benomyl-resistant populations of *M. laxa*.

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