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Project No. 88-T13 - Almond Disease
Shot hole, Brown rot, Botrytis Green Fruit Rot, and Leaf Rust

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Abstract

Springtime blossom and leaf diseases were almost nonexistent except in those orchards with previous high inoculum buildup of the brown rot and shot hole pathogens. The exception was the prevalence of the green fruit rot caused by Botrytis. Although the summer was dry and hot, unusually high incidence of leaf rust (Tranzchelia) and scab (Cladosporium) were noted in both the Sacramento and San Joaquin Valleys causing early defoliation. Almond hull rot was also common. Wood rots caused by a complex of basidiomycetous fungi were found to be widespread in occurrence as evidenced by fruiting structures especially on older trees.

On the project on forecasting of the shot hole disease, the critical points in the epidemiology of the pathogen (Stigmina carpophila) was outlined as: 1) the overwintering inoculum is produced on leaves (necrotic lesions with sporodochia) in the fall-winter months; 2) leaf infections are favored by moisture periods of over 10 hrs; 3) infections on leaves form necrotic tissue which drop out (shot holing symptom) at high temperatures (22C); and under low temperatures (8C) and free moisture the necrotic lesions tend to remain in place and form sporodochia. These data indicate that if leaves are removed (by possibly using a zinc sulphate spray) before the fungus produces sporodochia, the inoculum for spring infection could be significantly reduced. Thus during the following spring the primary infections would be minimal and that the shot hole control spray(s) would be applied only after the emergence of the fruiting structure (sporodochia) on the leaves. Such disease management strategy would require a shot hole spray only when necessary. Research conducted in 1988 on trees with little to no shot hole disease or trees defoliated with zinc sulphate in fall 1987 showed essentially no disease on trees which had not been sprayed for shot hole control. Whereas, an orchard with high incidence of shot hole infections and sporodochia on leaves during fall 1987 developed high incidence of shot hole affecting both leaves and fruit during the 1988 season. For the 1988-89 season, test plots comparing fall defoliated and nondefoliated trees have been set up in Butte, Merced, and Kern counties and the requirements for spraying fungicides during spring 1989 will be forecasted using the weather monitoring equipment and observations by the farm advisors and growers on the shot hole infection and sporodochial development.

For brown rot blossom blight caused by benomyl-resistant M. laxa, both iprodione and E-0858 (SC-0858) provided effective control when applied at the green bud stage of bloom (stigma starting to protrude) on cultivbar Ruby. Application of the radio-active parent E-0858 fungicide showed translocation of the labelled compound from the sepals and petals into the anthers and stigma. This compound further suppressed disease developement on the cultivar Drake even when applied 24 hr after inoculation and incubation. Because of the widespread occurrence of the leaf rust and scab with early defoliation of trees during the 1988 season, test plots are planned for the 1989 season comparing efficacy of sulfur, mancozeb, and chlorothalonil.

Data obtained by Dr. Adaskaveg on the identification of the wood rot fungi on almonds will be published in a research paper and in the book by Ogawa and English on Diseases of Fruit and Nut Crops Produced in Temperature Zones of California to be published by the University of California, Division of Agriculture and Natural Resources during summer 1989.

Introduction: The low incidences of foliage and blossom diseases during the last few years have provided and excellent opportunity to develop the basics of plant disease management in that the most devastating almond diseases, shot hole, brown rot, and Botrytis green fruit rot were important only in few orchards where our test plots were located. Further studies on almond scab and leaf rust will be possible as the disease is more widespread but not at an epidemic proportion. Thus our research efforts have been geared towards the objectives of: 1) defining the parameters required to reduce the use of fungicides by using disease forecasting methods for control of the shot hole disease; 2) developing data on new alternative fungicides for controlling benzimidazole-resistant Monilinia, 3) determining the species of the common wood rot organisms, and 4) completing the information required for publication of two manuscripts on the epidemiology of the shot hole disease.

Materials and Methods:

1. Shot hole disease forecasting: Techniques are included in the manuscript which has been accepted with revisions for publication in Plant Disease and Phytopathology (Ref. 7,8,10)

2. Shot hole disease management: Fungicides tested were a single dormant copper and captan spray followed by captan, ziram or iprodione. One test plot was located in Fresno County and two in Merced County. All treatments were made with a handgun sprayer except the dormant copper and captan sprays. Disease evaluations were made by collecting leaves and making isolations or observing the lesions for presence of Stigmina sporodochia. Both plots in Merced County were also harvested to detect any trends in yield differences.

3. Alternative fungicides for control of benomyl-resistant Monilinia: Carbon^{14} labelled experimental fungicide E-0858 and registered iprodione were applied on almond blossom obtained from nonsprayed trees on the University of California campus. Proprietary fungicide formulations of E-0858 and iprodione were were compared to that of benomyl on an almond orchard in Fresno County by spraying during various blossoming period.

4. Survey of wood decay organisms: Observations and collections were made throughout the almond growing districts of California. Techniques used for identification are included in manuscripts by Adaskaveg et al (1,2).

Results and Discussion:

1. Shot hole disease forecasting: Abstract for the manuscript for Plant Disease reads as follows: "A portable, self-contained, moisture generator controlled by a micrologger was constructed for the study of shot hole disease of almond in the field. The system produced localized conditions of free water on selected plant tissues and recorded environmental parameters during infection periods of Stigmina carpophila. Programmed misting at 2.5 and 5 min intervals with a 4 sec duration resulted in conditions conducive for infection. The greatest number of lesions per leaf occurred with intermittent misting for a 16-hr period compared to 0, 6, 8, 10, 12, and 14-hr treatments after inoculation with 300-400 conidia/leaf. Fewer lesions developed when intervals between misting were increased to 10, 20, 30, or 60 min. Misting durations longer than 5 sec caused excessive water runoff from leaves which reduced inoculum. Infections were also obtained when two different types of electrical conductance leaf wetness sensors were used to monitor and govern moisture regimes." The MGEMS equipment could become a very useful tool to study epidemiological requirements for disease induction as well as for screening fungicide efficacies.

2. Shot hole disease management: In an orchard with abundant shot hole lesions on leaves in fall 1987 (Table 1), data from trees sprayed only at the popcorn stage of bloom showed that all treatments significantly reduced incidence of shot hole lesions on the leaves. Yet the bloom sprays provided significantly better disease control than the dormant copper or captan treatments alone and cover sprays on trees with dormant captan or copper sprays provided no additional benefits. Examination of fruits for the shot hole disease after the petal fall stage of bloom (Table 2), showed that sprays with copper or captan applied on January 14 showed only slight reductions in shot hole disease control on the fruit. Yet when a popcorn or petal fall cover spray was applied, significant reductions in disease were noticed except for the single spray of captan at popcorn on the plot receiving no dormant treatment. Two spray applications, at popcorn and petal fall, with ziram or captan did not provide better control than a single spray at popcorn or petal fall. Furthermore, dormant copper or captan spray treatment plus popcorn and/or petal fall spray with captan, ziram, or iprodione provided no additional benefits. Iprodione treatments were found equivalent to that of captan or ziram statistically but iprodione figures on disease incidence were slightly higher than those of ziram or captan.
Further analayses of the fruit data on shot hole was made to determine specific differences between dormant treatments with that of ziram and captan sprays (Table 2A). Data revealed that compared to the nonsprayed control trees, two sprays at popcorn and petal fall with ziram or captan provided equavalent control as that of a single ziram spray at popcorn and a single popcorn spray of captan provided very poor results. Similar trends with the captan popcorn cover spray was shown in treatments with dormant copper and captan. Again, no significant differences were noted between the ziram and captan treatments with two sprays and the popcorn spray with ziram.

In an orchard with traces of shot hole lesions with sporodochia in fall 1987 (Tables 3), dormant copper spray followed by a popcorn alone or a popcorn plus petal fall spray of iprodione provided variable results. On the plot without dormant spray, no differences were noted between the nonsprayed and iprodione sprays applied at popcorn and petal fall, although better control was shown with a single popcorn spray. On the plot receiving dormant copper treatment, no differences were noted between it and the iprodione cover sprays at popcorn or popcorn and petal fall. Yield data trends were not observed between nonsprayed and sprayed trees.

In another orchard where the shot hole incidence was nondetectrable on leaves in the fall (Table 4) dormant copper sprays followed by captan or ziram sprays at popcorn alone or at popcorn plus petal fall showed variable results. The nonsprayed trees showed the least amount of shot hole lesions when compared with fungicide spray treatments and the kernel weight data also was nonsignificant between treatments harvested.

Three experimental plots were set up in November 1988 to determine if fall defoliation with zinc sulphate would reduce shot hole fungus populations to the point where spring sprays could be delayed until disease incidence is monitored. Plots are located in Kern, Merced, and Butte counties. Weekly monitoring for incidence of shot hole disease will be made starting from the time leaves emerge.

3. Alternative fungicides for control of benomyl-resistant Monilinia: Comparisons in efficacy of registered fungicides, benomyl and iprodione were made with an experimental fungicide E-0858 (Stauffer Chemical Company, now ICI of England) in field plots established in an almond orchard with benomyl -resistant M. laxa (Fresno County on Ruby almond cv) and without benomyl-resistant isolates (Yolo County on Drake Cv) (Table 5). The fungicides tested were benomyl 50W, iprodione 50W, and an experimental fungicide formulation E-0858 50W at 8 oz per 100 gal spray applied with a handgun sprayer using 3-4 gal on each of five single tree replications. Fungicides were sprayed at the phenological blossom stages of early pink bud, full bloom, and pink bud plus full bloom. Disease control was evaluated 3-weeks after full bloom by obtaining the percentage of blossoms blighted out of 400 counted per tree. As expected, benomyl spray at pink bud failed to control the disease while the full bloom treatment provided some control. Both E-0858 and iprodione provided significantly better control than the nonsprayed control trees. The pink bud spray of iprodione was not as effective as that of E-0858 which may indicate more systemic movement of the active compound than that of iprodione.

The results on Drake cv showed that under low disease pressure, differences in efficacy between fungicides were not shown regardless of the fungicides applied or the numbers of applications (Table 6). Fungicide treatments did reduce percent blight when compared to that of the nonsprayed control trees.

The systemicity of the fungicide E-0858 was examined by applying C^{14} labelled E-0858 on the unopened blossoms of the Thompson cv (Table 7). Translocation of the active compound or its breakdown metabolite to stamens and pistil was shown when the mixture of proprietary and labelled E-0858 were applied to sepals or petals. For the 1989 season, the systemicity of

iprodione will be compared to that of E-0858.

4. Survey of wood decay organisms: Predominant fungal genera and their incidence in almond orchards in California include Armillaria (0.8%), Ganoderma (3.1%), Laetiporus (1.6%), Oxyporus (4.0%), Perenniporia (0.6%), Phellinus (1.0%), Stereum (0.4%), and Trametes (1.6%).

Publications

1. Adaskaveg, J. E. and J. M. Ogawa. 1988. Survey of wood decay fungi and their role in the decline of fruit and nut trees in California. Proceedings, 4th Workshop on Stone Fruit Tree Decline. University of California, September 25-28, 1988. pp. 55-61. (Final version in press, draft enclosed)

2. Adaskaveg, J. E. and J. M. Ogawa. 1988. Survey of wood decay fungi and their effects in California fruit and nut orchards. Proceedings on Deciduous Tree Fruit (Apple) Disease Workshop. A symposium held August 16, 1988, at the Hokke club in Hirosaki, Japan. pp. 10-11. (MS available)

3. Ogawa, J. M., B. T. Manji, J. E. Adaskaveg, and T. J. Michailides. 1988. Population dynamics of benzimidazole-resistant Monilinia species on stone fruit trees in California. In: Fungicide Resistance in North America. Ed. C. J. Delp. APS Press, St. Paul, Minnesota. pp. 36-39. (MS enclosed)

4. Ogawa, J. M., B. t. Manji, J. E. Adaskaveg, and R. M. Sonoda. 1988. Fungi and bacteria resistant to pesticides and their management in California. Proceedings on Deciduous Tree Fruit (Apple) Disease Workshop. A symposium held August 16, 1988 at the Hokke Club in Hirosaki, Japan. pp. 24-25. (MS available)

5. Ogawa, J. M. 1988. Collator of Common names for Plant Diseases -Almond [Prunus dulcis (Mill.) D. A. Webb]. Plant Disease 72:567. (MS enclosed)

6. Ogawa, J. M. and W. H. English. 1989. Diseases of Temperate-Zone Tree Fruit and Nut Crops in California. University of California, Division of Agriculture and Natural Resources. Textbook in press to be published in 1989.

7. Ogawa, J. M., W. D. Gubler, and B. T. Manji. 1988. Effect of sterol biosynthesis inhibitors on diseases of stone fruits and grapes in California. IN: Sterol Biosynthesis Inhibitors, Pharmaceutical and Agrochemical Aspects. Ed. D. Berg and M. Plempel. Ellis Horwood Ltd., Chichester (England), 1988. pp. 262-278. (M. S.Enclosed)

8. Shaw, D. A. 1988. M.S. Thesis: Influence of moisture and temperature on infection and development of shot hole disease of almond caused by Stigmina carpophila. Department of Plant Pathology, University of California, Davis, CA 95616. (Available)

9. Shaw, D. A., J. E. Adaskaveg, and J. M. Ogawa. 1988. Influence of moisture and temperature on shot hole disease of almond leaves. Phytopathology (Abstr.): In press.

10. Shaw, D. A., J. E. Adaskaveg, and J. M. Ogawa. 1988. A field moisture generator for studying shot hole of almond caused by Stigmina carpophila. Phytopathology (Abstr.): In Press.

Table 1. Percentage of almond leaves with shot hole disease after various dormant treatments and blossom sprays

Chemical Treatment[1]	Blossom Application[2]		Percentage of Leaves Diseased[3]		
	Popcorn	Petal Fall	Dormant Treatment[4]		
	Nonsprayed	Copper	Nonsprayed	Copper	Captan
Control	-	-	17.83 a	7.83 a	6.17 a
Ziram	+	-	1.17 b	1.33 b	0.83 b
Captan	+	-	2.17 b	1.50 b	1.33 b
Rovral	+	-	3.17 b	1.83 b	1.67 b

[1]Blossom application of ziram and captan were applied at 2 lb/100 gal spray using handgun sprayer delivering 4.8 gal/tree.

[2]Blossom sprays was applied at popcorn stage (5% bloom; February 22, 1988) and petal fall sprays were not made.

[3]One hundred leaves were randomly collected on March 11, 1988 and evaluated for shot hole lesions. Data were anlayzed using Analyses of Variance (ANOVA) and Duncan's Multipe Range Test (DMRT). Means followed by the same letter were not significantly differen ($P = 0.05$). A significant interaction between dormant and blossom applications of fungicide was observed (0.05).

[4]Dormant applications of copper (Kocide 101) and captan 50W (Stauffer Chemical Company) were applied at 2 lb/100 gal spray on January 14, 1988 using a hand gun sprayer delivering 4.8 gal/tree.

Table 2. Comparisons in control of shot hole disease of almonds between dormant spray treatments with and without blossom sprays.

Chemical Treatments			
	Blossom Spray[2]		Percent Fruit[3]
Dormant Spray[1]	Popcorn	Petal Fall	with Disease
None	None	None	33.7 a
None	Ziram	None	6.8 cd
None	None	Ziram	0.8 cd
None	Ziram	Ziram	0.3 d
None	Captan	None	22.2 b
None	None	Captan	0.3 d
None	Captan	Captan	0.2 d
None	Iprodione	Iprodione	1.6 cd
Captan	None	None	21.3 b
Captan	Ziram	None	5.3 cd
Captan	None	Ziram	--
Captan	Ziram	Ziram	0.4 d
Captan	Captan	None	6.7 cd
Captan	None	Captan	--
Captan	Captan	Captan	0.2 d
Captan	Iprodione	Iprodione	2.2 cd
Copper	None	None	21.1 b
Copper	Ziram	None	5.5 cd
Copper	None	Ziram	--
Copper	Ziram	Ziram	0.1 d
Copper	Captan	None	8.4 c
Copper	None	Captan	--
Copper	Captan	Captan	0.2 d
Copper	Iprodione	Iprodione	1.2 cd

[1] Dormant applications of copper (Kocide 101) and captan 50W (Stauffer Chemical Company were applied at 2 lb/100 gal spray on January 14, 1988 using a hand gun sprayer delivering 4.8 gal/tree.

[2] Blossom applications of ziram and captan were applied at 2 lb/100 gal spray using hand-gun sprayer delivering 4.8 gal/tree. Sprays were applied at popcorn stage (5% bloom; February 22, 1988) and petal fall (99% petal fall with 3-5 opened leaves/terminal branch; March 12, 1988.

[3] Twenty fruit were randomly collected on April 28, 1988 from six single tree replications and evaluated for shot hole disease. Data were analyzed using ANOVA and DMRT. Means followed by the same letter were not significantly differe P = 0.05). A singificant interaction between dormant and blossom applications of fungicides was observed (0.05).

Table 2A. Percentage of almond fruit with shot hole disease after various dormant treatments and blossom sprays.

Chemical[1] Treatment	Time of Spray Application[2] Popcorn	Petal fall	Percentage of Fruit with Disease[3] Dormant Treatments[4] Nonsprayed	Copper	Captan
Control	-	-	33.7 a	21.1 a	21.3 a
Ziram	+	-	6.8 c	5.5 bc	5.3 bc
Ziram	+	+	0.3 c	0.1 c	0.4 c
Captan	+	-	22.2 b	8.4 b	6.7 b
Captan	+	+	0.2 c	0.2 c	0.2 c

[1]Blossom applications of ziram and captan were applied at 2 lb/100 gal spray using a handgun sprayer delivering 4.8 gal/tree.

[2]Blossom sprays were applied at popcorn stage (5% bloom; February 22, 1988) and petal fall (99% petal fall with 3-5 opened leaves/terminal branch; March 12, 1988.

[3]Twenty almond fruit were randomly collected on April 28, 1988 from six single tree replications and evaluataed for shot hole disease. Data were analyzed using ANOVA and DMRT. Means followed by the same letter were not significantly different (P = 0.05). A significant interaction between dormant and blossom applications of fungicide was observed (0.05)

[4]Dormant applications of copper (Kocide 101) and captan 50W (Stauffer Chemical Company) were applied at 2 lb/100 gal spray on January 14, 1988 using a handgun sprayer delivering 4.8 gal/tree.

Table 3. Percentage of almond leaves with shot hole disease after dormant copper treatment and iprodione blossom sprays.

Chemical Treatments[1]	Blossom Spray Applications[2]		Percentage of Diseased Leaves[3]	
	Popcorn	Petal Fall	Dormant Treatment[4]	
			Nonsprayed	Copper
Control	+	-	7.33 a	5.42 a
Iprodione	+	-	3.94 b	4.35 a
Iprodione	+	+	5.18 ab	5.65 a

[1]Spray treatment during blossom applied with handgun sprayer using 6.5 gal/tree on 6 single tree replications.

[2]Blossom sprays applied at popcorn and petal fall stage of bloom on February 10, and February 21, 1988, respectively.

[3]Percentage of leaves infected with shot hole based on randomly collected leaves of 360 to 835 per tree. Data were analysed using ANOVA and DMRT. Means followed by the same letter were not significantly different (P= 0.05). A significant interaction between dormant and blossom application of fungicides was observed (0.05).

Yield data comparisons between dormant copper and nonsprayed treatments with and without two blossom sprays of iprodione provided no significant differences. Average kernel weight between treatments ranged from 46.8, 43.0, 41.2 to 41.3 lb/tree.

4Dormant spray of copper applied with airblast sprayer at 100 gals/acre on January 21, 1987.

Table 4. Percentage of shot hole disease on leaves after dormant copper and nonsprayed treatment with and without ziram and captan blossom sprays

Chemical Treatment[1]	Blossom Spray Applications[2]		Percentage of Diseased Leaves[3]	
	Popcorn	Petal Fall	Dormant[4]	
			Nonsprayed	Copper
Control	-	-	6.37 b	8.61 a
Ziram	+	-	6.77 b	10.00 a
Ziram	+	+	7.53 b	8.25 a
Captan	+	-	8.46 ab	10.73 a
Captan	+	+	10.64 a	10.21 a

[1]Blossom sprays were applied with a handgun sprayer at popcorn and petal stage of bloom at 7.5 and 8.0 gal/tree, respectively on five single tree replications.

[2]Blossom spray applications were made using 2 lb/100 of ziram 75% and captan 50W at popcorn (February 18) and petal fall (February 21), 1988).

[3]Leaf samples were collected on March 15, 1988. Data were analyzed using ANOVA and DMRT. Means followed by the same letter were not significantly different (P = 0.05). Significant interactions between dormant and blossom applications of fungicide was not observed.

Yield data collected at harvest on September 27, 1988 between dormant copper and nonsprayed with and without two applications of captan or ziram provide no significant differences with analyzed using ANOVA and DMRT. Average kernel weight ranged from 46.7, 50.1, 49.4, 53.1 to 47.5 lb/tree.

[4]Dormant spray of 53% tribasic copper WP was applied with an airblast sprayeer at 20 lb/acre on January 14, 1988.

Table 5. Efficacy of one and two blossom spray applications of E-0858 and iprodione, and benomyl on control of blossom blight (M. laxa with benomyl-resistant populations) on almond cv Ruby

Chemical Treatment and Concentration (ug ai/ml)		Percent Blossom Blight2		
		Pink Bud	Full Bloom	Pink Bud plus Full Bloom
E-0858	300	13.80 a	7.06 a	3.15 a
Iprodione	300	23.50 b	10.47 a	5.06 a
Benomyl	300	32.20 bc	24.87 b	26.41 b
Nonsprayed	--	40.11 c	40.11 c	40.11 c

[1]Chemicals used were proprietary formulations of 50% E-0858 (ICI Americas Inc.), 50% iprodione (Rhone Poulenc Ag. Co.), and 50% benomyl (E. I. du Pont de Nemours and Co., Inc.) applied as spray with a handgun at the rate of 3 to 4 gal/tree on each of five single tree replications at pink bud (2/25/88) and full bloom (3/2/88) and pink bud plus full bloom. Means followed by the same letter are not significantly different (P = 0.05)

[2]Blossom blight was evaluated by counting 400 blossom per tree. Data was analyzed by ANOVA and DMRT. Means followed by the same letter are not significantly different (P = 0.05).

Table 6. Efficacy of a one and two spray applications of E-0858, iprodione, and benomyl in control of blossom blight (M. laxa) on almond cv Drake.

Chemical Treatment1	Concentration (ug ai/ml)	Percent Blossom Blight2		
		Pink Bud	Full Bloom	Pink Bud plus Full Bloom
Benomyl	300	1.0 a	0.4 a	0.3 a
E-0858	300	1.3 a	1.1 a	0.7 a
Iprodione	300	1.8 a	0.7 a	0.9 a
Control	--	4.3 b	4.3 b	4.3 b

1Chemicals used were proprietary formulations of 50% benomyl (E. I. du Pont de Nemours and Co., Inc.), 50% E-0858 (ICI Americas Inc.), and 50% iprodione (Rhone-Poulenc Ag. Co.). Sprays were applied with a hand gun at the rate of 3 TO 4 gal/tree on each of 5 single tree replications at pink bud (2/17/88), full bloom 2/22/88) and pink bud plus full bloom.

2Disease data were obtained by examination of 100 blossom/replication. Data were analyses using ANOVA and DMRT. Means followed by the same letter are not significantly different (P = 0.05).

Table 7. Percent of E-0858 radioactivity (DPM) in different parts of almond blossoms cv Thompson[1]

	Petal Treatment			Sepal Treatment		
	Days after treatment					
Blossom part	1	2	3	1	2	3
Pistil	0.03	0.04	0.26	0.38	0.37	0.86
Stamen	0.13	0.28	0.83	3.03	3.15	4.37
Sepals	0.69	1.19	3.60	89.12	91.53	87.44
Petals	99.15	98.47	95.31	7.47	4.95	7.33

[1]Values are averages of four replications. Samples were combusted and radioactivity measured in a scintillation counter.

Rpt #1

Wood Decay Fungi and Their Role in the Decline of Fruit and Nut Trees in California

J.E. Adaskaveg and J.M. Ogawa[1]

ABSTRACT

In the fall and winter seasons of 1986-87 and 1987-88, surveys in commercial fruit and nut orchards were conducted in ten counties throughout the Sacramento and San Joaquin Valleys of California. Orchards assessed for wood decay were generally ≥ 15 yr old and included: almond, peach and nectarine, apricot, plum and prune, fig, and walnut. Fungal species collected as fruiting bodies and their incidence differed between crops and orchards surveyed. Fruiting bodies of wood decay fungi were also collected from the hosts previously mentioned as well as from cherry, pistachio, and olive, in orchards not surveyed for wood decay. Thirty-three species of fungi were collected from 23 genera. The majority of the fungi collected caused or were associated with white wood rots; whereas three genera caused brown wood rots; and the decay of one genus was undetermined. Wood decay and fruiting bodies were primarily associated with wounds on trunks and scaffold branches. Trees with wood decay were commonly associated with orchards showing a decline in shoot growth, limb breakage, and decayed root systems. Several species collected in this survey have been implicated as pathogens of various fruit tree species by other researchers.

Wood decay disorders occur in commercial fruit and nut trees throughout California. The fungi causing these disorders are primarily in the Basidiomycotina. Information available on these fungi in fruit orchards is limited to mycological descriptions (Overholts 1953; Gilbertson and Ryvarden 1986, 1987) and scattered reports of incidence on various hosts (Anonymous 1961; Shaw 1973; French 1987). Detailed surveys of wood decay fungi on apple trees have been conducted in Washington (Dilley and Covey 1980; Helton and Dilbeck 1984) and Minnesota (Eide and Christensen 1940; Bergdahl and French 1985). To date no specific studies or surveys of wood decay fungi have been published on stone fruit trees in California.

The purpose of this study was to determine: i) species of wood decay fungi found on selected stone fruit trees, ii) incidence of these species and wood decay in surveyed orchards, and iii) association of tree wounds and decay fungi on surveyed trees.

MATERIALS AND METHODS

Twenty-nine, 15-yr old orchards in California under commercial production were selected in 10 counties in both the San Joaquin and Sacramento Valleys. Numbers of trees, orchards surveyed, and crop varieties were (crop/no. of orchards/total trees/ varieties): almond/15/2688/Carmel,

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Drake, Merced, Mission, NePlus Ultra, Nonpareil, and Thompson; apricot/2/210/Blenheim, and Perfection; fig/2/50/Calimyrna; nectarine and peach/4/408/Flamekist (nectarine), Loadel, Starn, and Fay Elberta; plum and prune/3/300/Friar and French, respectively; and walnut/3/133/English on native Black. Fruiting bodies, type of decay, and wounds associated with specific tree portions were determined for each tree surveyed.

Fruiting bodies of wood decay fungi were also collected from the hosts previously mentioned as well as from cherry, pistachio, and olive, in orchards not surveyed for wood decay. Fruiting bodies collected were identified using macro- and microscopic characteristics (Gilbertson and Ryvarden 1986, 1987; Juelich and Stalpers 1980). Fungi were cultured on 2% malt extract agar and identified (Nobles 1948, 1965; Stalpers 1978).

RESULTS AND DISCUSSION

Wood decay within the orchards ranged from 21-92% with almond having 25%, peach and nectarine 36%, apricot 21%, plum and prune 36%, fig 92%, and walnut 34% decay. Table 1 indicates the incidence of decay fungi collected as fruiting bodies from each crop surveyed. Predominate fungal genera found on *Prunus* sp. were *Oxyporus*, *Ganoderma*, *Laetiporus*, *Trametes*, *Fomitopsis*, *Armillaria*, *Phellinus*, and *Perenniporia*. Common genera on walnut were *Armillaria* and *Pleurotus*, while on fig only species in the genus *Inonotus* were found.

Thirty-three species of fungi were collected from the following genera: *Armillaria*, *Ceriporia*, *Coprinus*, *Fomitopsis*, *Ganoderma*, *Hyphoderma*, *Hyphodontia*, *Inonotus*, *Laetiporus*, *Lenzites*, *Oxyporus*, *Peniophora*, *Perenniporia*, *Phanerochaete*, *Phlebia*, *Phellinus*, *Pholiota*, *Pleurotus*, *Schizophyllum*, *Schizopora*, *Sistotrema*, *Stereum*, and *Trametes*. Three genera, *Coprinus*, *Fomitopsis*, and *Laetiporus*, caused brown wood rots, decay by the *Pholiota* species was undetermined, while the remaining genera were associated with or caused white wood rots. Species collected or reported in California on stone fruit trees are presented in Table 2.

Basidiocarps and decayed wood were commonly associated with tree wounds created by: mechanical harvesters, canopy support methods, pruning, and sunburn. Limb breakage during fruit production and uprooted trees during wind storms were damages primarily associated with wood decay in scaffold branches and roots of infected trees, respectively. In some cases, wood decay of specific portions of infected trees was limited to certain genera of fungi. For example, species of *Perenniporia*, *Schizophyllum*, *Stereum*, and *Trametes*, were commonly found on scaffold branches associated with pruning and sunburned wounds. Species in the genera Armillaria, *Ganoderma*, and *Oxyporus* were primarily collected from roots and lower portions of trees in association with trunk injuries. Other fungi, such as those in the genera *Laetiporus* and *Phellinus*, caused decay in roots, trunks, and scaffold branches of trees.

Two of the eight most common fungal genera, *Laetiporus* and *Fomitopsis*, collected in surveyed orchards caused brown wood rots. Generally, fungi that cause brown wood rots cause a greater reduction in wood strength and weight loss than fungi that cause white wood rots in the same time period. The high incidence and destructive nature of species in these two genera suggests that

these species may play a major role in the decline of fruit and nut trees in California.

The majority of fungi collected caused white wood rots. The role of these fungi in the decline of fruit and nut trees is not well established, except for species of *Armillaria* which are known root rot pathogens of fruit trees (Raabe 1967; Wilbur et al. 1972; and Proffer et al. 1987) and *Chondrostereum purpureum*, the causal organism of silver leaf disease of fruit trees (Setliff 1973). The other genera of fungi in high incidence in surveyed orchards that may contribute to declining orchards are *Ganoderma*, *Trametes*, and *Oxyporus*. Bergdahl and French (1985) indicated that *Oxyporus latemarginatus* (= *Irpex tulipiferae*), Trametes versicolor (= *Coriolus versicolor*), and *Schizophyllum commune* could cause decline of 3 yr old apple trees in less than optimal growing sites in Minnesota. Pathogenicity of *Trametes versicolor* on young apple trees (2-3 leaf stage) in Washington has also been reported (Covey et al. 1981). Dilley and Covey (1981) further associated dieback symptoms with *T. versicolor* on mature apple trees in Washington, while in Australia this fungus is also known to cause a serious disease of mature apple trees (Darbyshire et al. 1974; Kile and Wade 1974, 1975; and Kile 1976). The significance of wood decay fungi in California needs to be further evaluated and management strategies designed to limit their introduction and spread in newly established (2-3 years) and older commercial orchards.

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Table 1. Predominant fungal genera and their incidence in fruit and nut tree orchards in California.[1]

Orchards Surveyed[2]					
Almond	Apricot	Fig	Peach and Nectarine	Plum and Prune	Walnut
Armillaria (0.8%)	*Laetiporus* (4.3%)	*Inonotus* (18.3%)	*Armillaria* (0.8%)	*Fomitopsis* (4.3%)	*Armillaria* (1.5%)
Ganoderma (3.1%)	*Oxyporus* (5.7%)		*Ceriporia* (0.2%)	*Oxyporus* (0.7%)	*Laetiporus* (0.7%)
Laetiporus (1.6%)	*Phellinus* (0.9%)		*Ganoderma* (24.3%)	*Perenniporia* (0.7%)	*Pleurotus* (3.0%)
Oxyporus (4.0%)	*Perenniporia* (1.0%)		*Oxyporus* (0.7%)	*Phellinus* (2.7%)	
Perenniporia (0.6%)	*Trametes* (6.7%)		*Phellinus* (1.2%)	*Stereum* (1.0%)	
Phellinus (1.0%)			*Pholiota* (0.2%)	*Trametes* (0.3%)	
Stereum (0.4%)			*Schizophyllum* (0.9%)		
Trametes (1.6%)			*Trametes* (1.7%)		

1 – Predominant fungal genera collected as basidiocarps on living trees in commercial production and their incidence based on total trees surveyed for each crop.

2 – Orchards surveyed: almond (Carmel, Drake, Merced, Mission, NePlus Ultra, Nonpareil, and Thompson); apricot (Blenheim and Perfection); fig (Calimyrna); nectarine (Flamekist); peach (Loadel, Starn, and Fay Elberta); plum (Friar); prune (French); and walnut (English grafted on California Black).

Table 2. Common Wood Decay Fungi of Selected Fruit and Nut Tree Species in California.

Fungus	Host[a]	HA[b]	Decay[c]	Source[d]
Abortiporus biennis (Bull.:Fr.) Sing.	2,3	1,2	W	L
Armillaria spp.	1-10	1,(2)	W	L
Armillaria mellea Fr.	5,9	1,(2)	W	A
Ceriporia spissa (Schw.: Fr.) Rajch.	9	2	W	A
Chondrostereum purpureum (Pers.:Fr.) Pouz.	6,8,9	1,2	W	L
Coprinus spp.	11	1,2	B	A
Daedalea quercina Fr.	2	(1),2	B	L
Daedaleopsis confragosa (Bolt.: Fr.) Schroet.	2	(1),2	W	L
Fomitopsis cajanderi (Karst.) Kotl. et Pouz.	8	(1),2	B	A
Ganoderma annularis (Fr.) Gilbn.	5,9	1,(2)	W	A
G. applanatum (Pers.) Pat.	9	1,(2)	W	A
G. brownii (Murr.) Gilbn.	5,9	1,2	W	A
G. lucidum (W.Curt.:Fr.) Karst.	5,7,9,11	1,2	W	A
Hyphoderma puberum (Fr.) Wallr.	5	2	W	A
Hyphodontia aspera (Fr.) J. Erikss.	5	2	W	A
Inonotus cuticularis (Bull.:Fr.) Karst.	1	1	W	A
I. rickii (Pat.) Reid	1	1	W	A
Irpex lacteus (Fr.:Fr.) Fr.	7,11	(1),2	W	L
Laetiporus sulphureus (Bull.:Fr.) Murr.	2,5,11	1,(2)	B	A
Lenzites betulina (Fr.) Fr.	5,7	(1),2	W	A
Oxyporus corticola (Fr.) Ryv.	9,11	2	W	A
O. latemarginatus (Dur. & Mont. ex. Mont.) Donk	7	1,2	W	A
O. similis (Bres.) Ryv.	5,9	1,2	W	A

Peniophora albobadia (Schw.:Fr.) Boidin	5	2	W	A
Perenniporia medulla-panis (Jacq.: Fr.) Donk	11	1	W	A
Phanerochaete velutina (Fr.) Karst.	9	2	W	A
Phlebia rufa (Fr.) M.P. Christ.	5	2	W	A
Phellinus ferruginosus (Schard.: Fr.) Bourd. et Galz.	11	(1),2	W	L
P. gilvus (Schw.) Pat.	5,9,11	1,2	W	A
P. igniarius (L.: Fr.) Quél.	11	1	W	L
P. pomaceus (Pers.: S.F. Gray) Maire	5	2	W	A
P. robustus (Karst.) Bourd. & Galz.	5,8	1	W	A
P. texanus (Murr.) A. Ames	8	1	W	A
Pholiota sp.	11	1	NS	A
Pleurotus ostreatus (Fr.) Kummer	2,4	1,2	W	A
Pycnoporus cinnabarinus (Jacq.: Fr.) Karst.	11	2	W	L
Schizophyllum commune Fr.	1,2,4-7	1,2	W	A
Schizopora flavipora (Cke.) Ryv.	5	2	W	A
Sistotrema brinkmannii (Bres.) J. Erikss.	9	2	W	A
Stereum hirsutum (Willd.: Fr.) S.F. Gray	5,8,9	1,2	W	A
Trametes hirsuta (Wulf.:Fr.) Pilát	6,9,11	1,2	W	A
T. versicolor (L.: Fr.) Pilát	3,5-9	1,2	W	A

[a]- Hosts included: (1) *Ficus carica* L. (Fig); (2) *Juglans* spp. (Walnut); (3) *Olea* spp. (Olive); (4) *Pistacia vera* L. (Pistachio); (5) *Prunus dulcis* (Mill.) W.A. Webb (Almond) (6) *P. armeniaca* L. (Apricot); (7) *P. avium* L. (cherry); (8) *P. domestica* L. and *P. americana* L. (Prune, Plum); (9) *P. persica* (L.) Batsch. (Peach); (10) *P. salicina* Lindl. (Japanese Plum); and (11) *Prunus* species. Host numbers separated by semicolons correspond to occurrence by state.

[b]- Host association (HA): 1- Living trees; (1)- Possibly living trees; 2- Dead wood; (2)- Possibly dead wood; 3- Not specified.

[c]- Wood Decay: W = White wood rot; B = Brown wood rot; NS = not specified.

[d]- Information obtained from author (A) or from literature (L) listed in reference section of this paper.

Fungicide Resistance in North America

Charles J. Delp, Editor

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Bryan R. Delp, Thomas M. Fort III,
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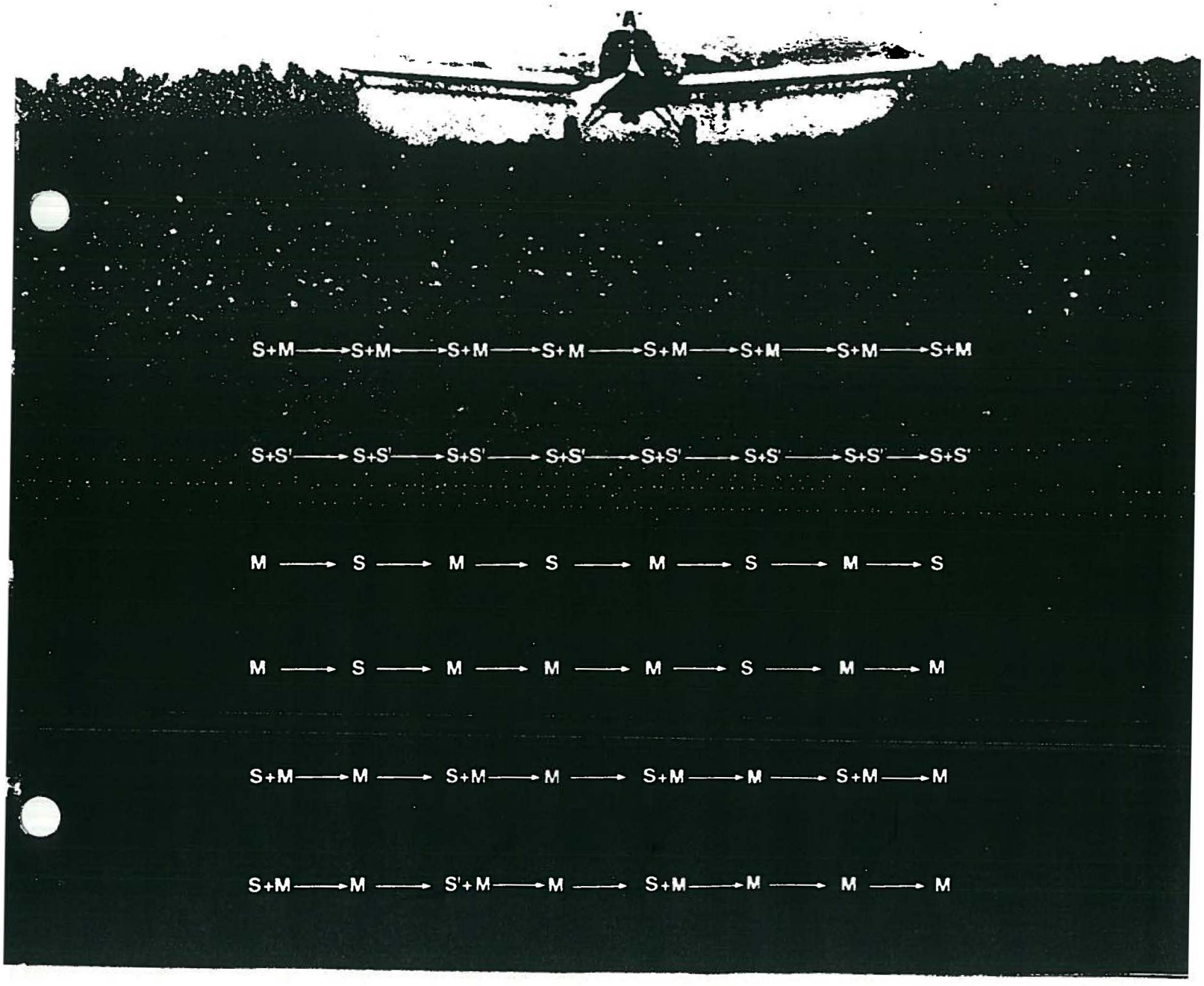


13. POPULATION DYNAMICS OF BENZIMIDAZOLE-RESISTANT MONILINIA SPECIES ON STONE FRUIT TREES IN CALIFORNIA

J. M. Ogawa, B. T. Manji,
J. E. Adaskaveg, and T. J. Michailides

Benomyl was introduced for experimentation in 1967 by E.I. du Pont de Nemours, Inc. and was registered in the United States for control of brown rot in 1972. With its specific mode of action, high activity against *Monilinia* species, and local systemic activity in host tissue (Ogawa et al, 1973a,b), benomyl was widely adopted for control of the brown rot disease on stone fruits and almonds.

In 1967, before experimental or field applications of benomyl were made in California, in vitro studies were made to establish the baseline sensitivity of *Monilinia* species to benomyl (Ogawa et al. 1968). These studies showed that mycelial growth of both *Monilinia fructicola* and *M. laxa* was completely inhibited on potato-dextrose agar (PDA) amended with 0.1 µg/ml benomyl. These results led to extensive field tests on sweet cherries, apricots, almonds, peaches, and prunes to determine effectiveness of benomyl in control of blossom blight, fruit rot and postharvest fruit decay. Results indicated that a single spray of benomyl was equivalent to two spray applications of other fungicides tested in control of brown rot blossom blight. Benomyl, when sprayed on peach fruit, showed activity for 2.0 days after peaches were inoculated with spores of *M. fructicola* and incubated in the laboratory. In addition, fruit dips in a mixture of benomyl and DCNA provided excellent disease control and suppressed established infections of postharvest fruit decays caused by *M. fructicola* and *Rhizopus stolonifer*.

USAGE OF BENOMYL

Control of brown rot caused by both *M. fructicola* and *M. laxa* became dependent on benomyl after its registration in 1972. Benomyl replaced other protectant fungicides such as captan, maneb, dichlone, and coppers for blossom blight control. The importance of fungicides in controlling brown rot in California stone fruit and almond orchards is indicated in Table 1. The number of fungicide applications per season varies with fungicide and crop. In California, one to two applications are made during bloom, followed by two to three preharvest applications, except on apricots, prunes, and almonds, where preharvest sprays are not applied. The number of preharvest applications on fresh market nectarines and peaches is based on the number of times the fruit is harvested.

In 1973, environmental conditions in California were conducive to blossom blight caused by *Monilinia* species and resulted in varied disease control. Prompted by a report by Schroeder and Provvidenti (1969) of benomyl resistance in *Erysiphe cichoracearum* on curcurbits, a survey was made of 73 orchards that reported repeated applications of benomyl, with some showing severe blossom blight. Sensitivity of isolates to benomyl was tested by measuring mycelial growth on PDA amended with 0.1 and 1.0 µg/ml of benomyl. None of the isolates of *M. fructicola* or *M. laxa* exhibited measurable mycelial growth on PDA amended with 1.0 µg/ml benomyl. At the 0.1 µg/ml benomyl level, no growth occurred on the second day, but growth started soon after and within 2-3 days covered the plate, which differed from earlier reports where the fungus failed to grow during the five-day incubation period (Ogawa, 1982; Tate et al, 1974). In 1974, our conclusion was that resistant populations were absent from California orchards that were sprayed repeatedly with benomyl. Poor control in some orchards was probably related to environmental conditions which prevented proper application of benomyl sprays.

Detection of benomyl-resistant *M. fructicola*

The first report of *M. fructicola* resistant to benomyl was by Wharton (1976) in Australia, followed by a report by Jones and Ehret (1976) in Michigan. Resistance levels reported ranged from 100 to 1,000 µg/ml benomyl (Jones, 1983). In New York, Szkolnik and Gilpatrick (1977) reported that 9% of the benomyl-resistant isolates showed profuse mycelial growth in medium amended with 50 µg/ml benomyl, while 53% showed profuse mycelial growth at 10 µg/ml benomyl. In the summer of 1977, Ravetto first detected isolates of *M. fructicola* resistant to benomyl on fruits collected in a peach orchard in the northern San Joaquin Valley (Lockford, CA). This orchard had been sprayed repeatedly over the years, first with benomyl, then with combination sprays of benomyl plus captan in attempts to reduce the severe crop losses from the brown rot disease. Ravetto's isolates of *M. fructicola* were found to be resistant to benomyl at 1.0 µg/ml and not at the high levels previously reported from Australia and Michigan. The levels of resistance were determined by comparing mycelial growth of isolates never exposed to benomyl with that of isolates collected from benomyl-sprayed orchards on PDA amended with benomyl (Manji et al, 1982). Benomyl-sensitive isolates failed to grow at 1 µg/ml benomyl and had a slight reduction in mycelial growth at 0.05 µg/ml benomyl, while the mycelial growth of benomyl-resistant isolates collected in 1977 from the same orchard was essentially identical on medium amended with 1.0 µg/ml benomyl as on unamended medium. Mycelial growth of resistant isolates was reduced at 4 µg/ml (Figure 1). Since that time, surveys of orchards in the San Joaquin Valley have indicated an increase in number of orchards with benomyl-resistant populations of *M. fructicola*. In 1978, 70 orchards (10 isolates from each) were sampled and four additional orchards were found with isolates of *M. fructicola* resistant to benomyl: in 1979, resistant isolates were detected in six orchards; and in 1980, 38 orchards. The use of benomyl was re-evaluated, and benomyl usage was discontinued in orchards where high populations of *M. fructicola* were resistant to benomyl.

Population dynamics of benomyl-resistant isolates in blossom infections

Under conditions of extremely high disease pressure, control of blossom blight with benomyl was less effective in commercial orchards with populations of *M. fructicola* resistant to benomyl at 1–4 µg/ml (Rough et al, 1979). In a Loadel cling peach orchard with 22% of the population of *M. fructicola* resistant to benomyl, blossom blight was 42%

TABLE 1

Crops, cultivars, production area, average number of spray applications, and estimated cost of treatments for brown rot control in California orchards

Crop	No. of cultivars	Production area (ha)a/	No. fungicide applications/year		Costd (X $1,000)
			Systemicb/	Contactc/	
Apricots	13	9,308	2	3	2,792
Nectarines	60	9,308	3	6	5,585
Peaches					
Processing	38	14,522	3	4	5,521
Fresh market	69	12,801	3	6	7,681
Plums	52	13,760	1	2	2,752
Prunes	11	34,131	1	2	6,826
Sweet cherries	8	4,800	2	4	1,920
Almonds	40	171,995	1	2	34,399
TOTAL		270,665			67,776

a/ For production year 1986.
b/ Estimated for benzimidazole fungicides.
c/ Estimated for sterol biosynthesis inhibiting and dicarboximide fungicides.
d/ Cost figures derived by multiplying production area, average number of contact fungicide treatments, and application cost of fungicide treatments, estimated at $200 per hectare.

in the benomyl-sprayed plot and 77% in the unsprayed plot. The benomyl-resistant population increased from 22% to 80% in the benomyl-sprayed plot, while in the unsprayed plot resistance increased to 40% (Szkolnik et al, 1978).

In further experiments in the same Loadel cling peach orchard, under conditions of high disease pressure and a moderate population of *M. fructicola* resistant to benomyl (36% as determined from mummified fruits), benomyl applications of 1.1 and 2.2 kg a.i./ha reduced blossom blight to 56% and 43%, respectively, of that of the unsprayed trees (Sonoda et al, 1983). The difference in blighted blossoms between the benomyl-sprayed trees and the unsprayed trees may be accounted for by hypothesizing that benomyl controlled only the benomyl-sensitive isolates. In another study on nectarines near Parlier, CA, blossom blight was effectively controlled with benomyl in an orchard with low disease pressure and a low resistant population (20%). However, the percentage of benomyl-resistant isolates increased from 20% to almost 90% after a single benomyl application. This increase in benomyl-resistant isolates could have an effect on the control of preharvest fruit rot with benomyl. Conclusions from these studies are that benomyl sprays applied during bloom effectively prevented infections caused by benomyl-sensitive isolates but not those of the benomyl-resistant isolates.

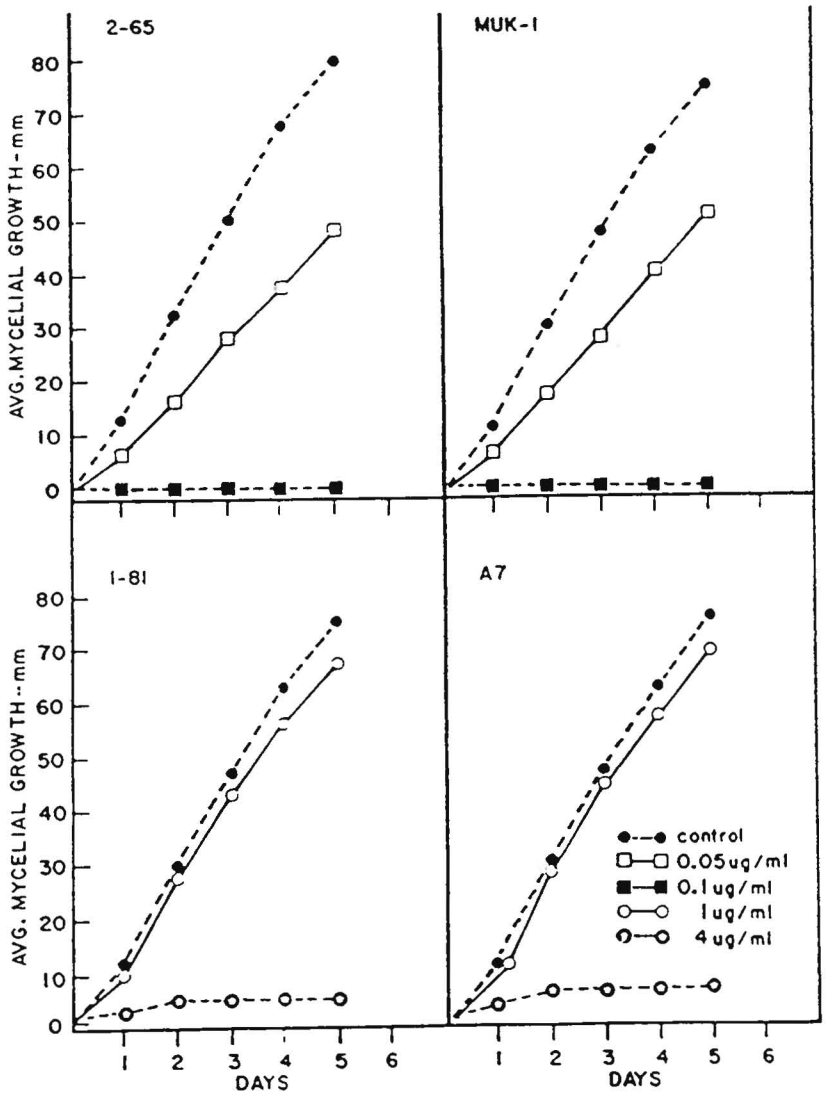
In the same peach orchard (Lockford, CA), Adaskaveg et al (1987) found that isolates of *M. fructicola* resistant to benomyl remained stable at 35% in the absence of benomyl treatments for an eight-year period. The orchard had been sprayed with a sterol biosynthesis inhibitor, triforine, for four years and with a dicarboximide, iprodione, for another four years. The nectarine orchard (Parlier, CA) was sprayed with a combination of benomyl plus captan during the previous six years, and the percent of benomyl-resistant isolates had increased from 20 to 55%.

Population dynamics of benomyl-resistant isolates in fruit infections

The amount of inoculum and proportion of resistant to sensitive isolates of *M. fructicola* on diseased fruit could determine the inoculum status for the following spring. Primary inocula for blossom infection are conidia from sporulating mummies found on the tree and on the ground, as well as ascospores from apothecia (Shabi and Ogawa, 1981). Apothecia are rarely found in California orchards except under prolonged wet soil conditions during spring bloom.

Comparative pathogenicity of resistant and sensitive isolates is one parameter determining parasitic fitness. Jones and Ehret (1976) in Michigan compared resistant and sensitive isolates of *M. fructicola* and found that they were similar in virulence. Penrose et al (1979) in Australia co-inoculated peach fruit with one resistant and one sensitive isolate using spore suspensions with differing proportions of the two isolates. In most cases, the isolate inoculated in the larger proportion predominated. However, mixtures of resistant and sensitive isolates were present in many of the resulting lesions. They concluded that the two isolates were about equally virulent. Sonoda and Ogawa (unpublished) co-inoculated resistant and sensitive conidial suspensions of *M. fructicola* onto injured peach fruit in the laboratory and found them to coexist in some lesions but not in others. In pairings of equal proportions (resistant at 1-3 µg/ml benomyl), the sensitive isolates predominated in 83% of the peach lesions. However, as the proportion of the sensitive or resistant isolates increased, the isolate in higher propor-

FIGURE 1



Comparisons in sensitivity of benomyl-sensitive (2-65 and MUK-1) and benomyl-resistant (1-81 and A7) *Monilinia fructicola* isolates on the basis of mycelial growth on PDA medium amended with various concentrations of benomyl.

tion became dominant in the resulting lesions except in oneinoculation. Lesions caused by individual sensitive isolates were larger than those caused by individual resistant isolates (Sonoda and Ogawa, 1982). In these studies, resistant and sensitive isolates were both pathogenic, however, the sensitive isolates were more virulent.

Zehr (1982) determined the level of resistance of isolates of *M. fructicola* in South Carolina as 500-1,000 µg/ml benomyl. Parasitic fitness of these isolates was determined by introducing benomyl-resistant isolates in benomyl-sprayed and non-sprayed peach orchards and observing their spread and overwintering ability. Resistant isolates became the predominant population only in trees sprayed with benomyl and then failed to overwinter. In contrast, California isolates resistant at lower levels (1-4 µg/ml) remained stable after eight years in the absence of benomyl, indicating their equal ability to survive compared with sensitive isolates (Adaskaveg et al, 1987). Further studies are needed to determine whether levels of resistance affect the survival of *Monilinia* species under varied environmental conditions.

Population dynamics of *M. laxa* causing blossom blight

Populations of *M. laxa* resistant to benomyl were not detected before 1980 in surveys conducted in almond, apricot, and prune orchards sprayed with benomyl where populations of *M. laxa* predominated. Crop losses were not reported except from orchards in Merced County, where severe apricot blossom blight occurred. This high disease incidence could be attributed to rains during blossoming, which prevented the proper application of benomyl. Eight years after the first application of benomyl, resistant isolates of *M. laxa* were detected (Ogawa et al, 1984). The level of resistance was 1 µg/ml, similar to those reported for *M. fructicola*. Isolates of *M. laxa* resistant to benomyl produced smaller cankers than sensitive isolates on almond shoots. Furthermore, two of the resistant isolates produced only a few conidia on PDA, and three were incapable of establishing colonies on benomyl-free medium.

In a 1982-1983 survey of *M. laxa* and *M. fructicola* in prune and apricot orchards in California, *M. fructicola* was the dominant species and only isolates of *M. fructicola*

were found to be resistant to benomyl (Michailides et al. 1987). A shift in population had occurred from previous studies, which indicated *M. laxa* as the predominant species on these crops. The reasons for this shift are not fully understood. Possibly, under continued use of benomyl, populations of *M. fructicola* became dominant with the development of resistance. The nondetection of isolates of *M. laxa* resistant to benomyl may be due to the single application of benomyl during bloom controlling the sensitive population of *M. laxa* or to the reduced parasitic fitness of resistant populations of *M. laxa*. Cañez and Ogawa (1982) found that isolates of *M. laxa* from apricot resistant to benomyl were less parasitically fit. However, in one almond orchard sprayed repeatedly with benomyl alone and later in combination with captan, 75% of the isolates of *M. laxa* from blighted blossoms were resistant. This resistant population was established. These resistant isolates showed reduction in rate of germ tube elongation, pathogenicity, and sporodochial production on twigs but good fitness in virulence when compared to the benomyl-sensitive isolate (Cañez, 1986). A resistant isolate from apricots showed reduced fitness only in germ tube elongation and sporodochial development. The slight reductions in parasitic fitness of the almond, benomyl-resistant isolate could not be measured in inoculation of twigs and measurement of canker development in the two years of experimentation in field test plots. In 1986, three years after the exclusive use of iprodione in this almond orchard, the percent of resistant isolates (82%) had not been reduced. Interestingly, under extremely low disease pressure, protective sprays of benomyl or iprodione provided effective and equivalent disease control (Adaskaveg et al, 1987).

Fungicide management studies for orchards with resistant populations of *M. laxa* showed that under high disease pressure, a mixture of benomyl plus iprodione provided more effective control than a mixture of benomyl plus captan. Under low disease pressure, differences between treatments were not shown (Cañez, 1986).

DISCUSSION

Contributions have been made on how to delay the occurrence or prevent increases of resistant populations since the advent of benzimidazole resistance in plant pathogens. Delp (1980) proposed a combination treatment of benomyl with another fungicide with a different mode of action to manage the development of resistant fungal populations. Kable and Jeffery (1980) also noted from their models that fungicide mixtures are more advantageous than alternations. Skylakakis (1981) qualified that chemical mixtures are optimal when infection rates of the resistant subpopulation are low. Thus the efficacy of the at-risk fungicide decreases as the resistant subpopulation increases. Further the delaying effect of mixtures increases as the efficacy of the companion fungicide increases. Treatments with systemic fungicides either alone or in alternation, or in a mixture with a protectant fungicide, reduced disease severity (Levy et al. 1983) but increased the frequency of resistant populations, with some exceptions (D. Shtienberg et al. 1988). Ritchie (1983) reported, from the previous models, that the use of an active systemic fungicide increased the frequency of selection of resistant subpopulations. Furthermore, the three models predicted that a mixture of benomyl with an unrelated fungicide would be more effective in reducing the rate of occurrence of a resistant subpopulation than the use of benomyl alone or in alternation. Ritchie further noted that the larger the population to which the selective agent is applied, the greater the probability of selecting a resistant subpopulation. He concluded that benomyl should not be used when a brown rot epidemic in the orchard is severe, and if mixtures are essential, they should be used in multiple applications.

Management strategies must include a monitoring system because it is unlikely that resistance will occur in all orchards (Ogawa et al. 1979, 1983; Ogawa, 1985). We need to consider the varying degrees of resistance to the at-risk fungicide, the role of the at-risk fungicide when used under low versus high disease pressure, and the presence of more than one species of pathogen being controlled with the same treatment. Further, we need to study the specific management strategies required for various stone fruit crops under an arid climate where rainfall and disease epidemics are limited compared to the more humid and high-rainfall temperate regions. To prevent the development of resistant fungal populations, management studies of fungicide usage require the determination of the minimal number of applications of the at-risk fungicide and the effectiveness of alternating several fungicides or mixtures of fungicides when multiple applications are necessary.

Acknowledgments

We thank R. M. Sonoda, V. M. Cañez, K. G. Tate, and E. Shabi for their role in the research presented; E.I. Zehr for providing his research data included in this paper; and the cooperating staff of E.I. du Pont de Nemours, Inc.

Common Names for Plant Diseases

In 1978 The American Phytopathological Society established a committee to develop listings of APS approved names for plant pathogens and the diseases they incite. These names are then considered the preferred names for use in APS journals and other publications. The Committee on Standardization of Common Names for Plant Diseases published lists of preferred names for 35 commodities in 1985 (Plant Disease 69:649-676).

The following eight lists are presented for reference. They were previously edited by committee members and taxonomists and published for comment in *Phytopathology News*. To achieve long-term uniformity in nomenclatural standards, the committee has adopted the taxonomic system being prepared for the USDA's second edition of *Agricultural Handbook 165 (Index of Plant Diseases, 1970)*. It is expected that the lists will not be revised for at least five years so that stability in use of common names will be achieved.

The committee thanks the collators of each list and those who have been involved in many days of editorial process.

Richard W. Smiley, *Chairman, Committee on Standardization of Common Names for Plant Diseases*

Almond (*Prunus dulcis*) (Mill.) D. A. Webb)

J. M. Ogawa, Primary Collator

Common name	Pathogen or cause
Almond bull mission	Genetic (nontransmissible)
Almond corky growth (on kernels)	Cause unknown
Almond corky spot	Cause unknown (nontransmissible)
Almond foamy canker	Cause unknown
Almond leaf scorch	Unidentified rickettsialike bacterium
Almond noninfectious bud failure	Genetic (nontransmissible)
Almond virus (bud failure)	Prunus ring spot virus—calico strain
Almond yellow bud mosaic	Tomato ring spot virus—yellow bud mosaic strain
Armillaria crown and root rot	*Armillaria mellea* (Vahl: Fr.) P. Kumm. (anamorph: *Rhizomorpha subcorticalis* Pers.)
Bacterial canker and blast	*Pseudomonas syringae* pv. *syringae* van Hall
Band canker	*Botryosphaeria dothidea* (Moug.: Fr.) Ces. & de Not.
Brown rot blossom and blight	*Monilinia fructicola* (Winter) Honey
	M. laxa (Aderhold & Ruhland) Honey
Ceratocystis canker	*Ceratocystis fimbriata* Ellis & Halst.
Crown gall	*Agrobacterium tumefaciens* (Smith & Townsend) Conn
Dagger nematode	*Xiphinema* spp.
Green fruit rot	*Botrytis cinerea* Pers.: Fr. (teleomorph: *Botryotinia fuckeliana* (de Bary) Whetzel)
Hull rot	*Monilinia fructicola* (Winter) Honey
	M. laxa (Aderhold & Ruhland) Honey
	Rhizopus stolonifer (Ehrenb.: Fr.) Vuill.
Leaf blight	*Hendersonia rubi* West.
Leaf rust	*Tranzschelia discolor* (Fuckel) Tranz. & Litv. f. sp. *dulcis*
Phytophthora crown and root rot	*Phytophthora* spp.
Powdery mildew	*Podosphaera tridactyla* (Wallroth) de Bary
	Sphaerotheca pannosa (Wallroth: Fr.) Lev.
Ring nematode	*Criconemella* spp.
Root knot	*Meloidogyne* spp.
Root lesion	*Pratylenchus* spp.
Scab	*Cladosporium carpophilum* Thuem. (teleomorph: *Venturia carpophila* E. E. Fisher)
Shothole (= Coryneum blight)	*Stigmina carpophila* (Lev.) Ellis = *Coryneum beyerinckii* Oud.
Verticillium wilt	*Verticillium dahliae* Kleb.
Wood rots	*Ganoderma brownii* (Murrill) Gilbn.
	G. lucidum (Curtis: Fr.) P. Karst.
	Laetiporus sulphureus (Bull.: Fr.) Murrill
	Perenniporia spp.
	Schizophyllum commune Fr.
	Stereum spp.
	Trametes hirsuta (Wulfen: Fr.) Quel.
	T. versicolor (L.: Fr.) Pilat

Elm (*Ulmus spp.*)

R. Jay Stipes and Richard J. Campana, Primary Collators

Common name	Pathogen or cause
Anthracnose	*Gloeosporium inconspicuum* Cavara = *Cylindrosporella inconspicua* (Cavara) Arx
	G. ulmicola Miles
Bacterial wetwood	*Bacillus megaterium* de Bary
	Enterobacter cloacae (Jordan) Hormaeche & Edwards = *Erwinia nimipressuralis* (Carter) Dye
	Pseudomonas fluorescens Migula
Black spot	*Gnomonia ulmea* (Schw.: Fr.) Thuem. = *Stegophora ulmea* (Schw.: Fr.) Syd & P. Syd. (anamorph: *Gloeosporium ulmicola* Miles)
Botryodiplodia canker	*Botryodiplodia hydodermia* (Sacc.) Petr. in Petr. & Syd. = *Sphaeropsis ulmicola* Ellis & Everh.
	B. malorum (Berk.) Petr. & Syd. (teleomorph: *Physalospora mutila* N. E. Stevens
Botryosphaeria canker	*Botryosphaeria dothidea* (Moug.: Fr.) Ces. & de Not. = *B. ribis* Gross. & Duggar (anamorph: *Dothiorella gregaria* Sacc.)
British tar spot	*Dothidella ulmi* (Duval: Fr.) Theiss. & Syd.
Chalara root rot	*Chalara thielavioides* (Peyronel) Nag Raj & Kendrick.
Coniothyrium canker	*Coniothyrium* spp.
Cytospora canker	*Cytospora ambiens* Sacc. (teleomorph: *Valsa ambiens* (Pers.: Fr.) Fr.)
	C. chrysosperma (Pers.: Fr.) Fr. (teleomorph: *V. sordida* Nits.)
	C. nivea (Hoffm.) Sacc. (teleomorph: *Valsa* spp.)
Cytosporina canker	*Cytosporina ludibunda* Sacc.
Damping-off, Fusarium	*Fusarium* spp.
Damping-off, Pythium	*Pythium ultimum* Trow
Damping-off, Rhizoctonia	*Rhizoctonia solani* Kühn (teleomorph: *Thanatephorus cucumeris* (Frank) Donk)
Decay (xylem)	*Coriolus versicolor* (L.: Fr.) Quel.
	Flammulina velutipes (Fr.) P. Karst.
	Ganoderma applanatum (Pers.) Pat. = *Fomes applanatus* (Pers.) Gill.
	Phellinus spp. = *Fomes* spp.
	Pleurotus spp.
	Polyporus squamosus Micheli ex Fr.
	Other basidiomycetes
Discoloration (xylem)	Bacteria, Ascomycetes, Deuteromycetes
Dothiorella canker and wilt	*Dothiorella ulmi* Verrall & May
Dutch elm disease	*Ophiostoma ulmi* (Buisman) Nannf. in Melin & Nannf. = *Ceratocystis ulmi* (Buisman) C. Moreau (anamorphs:

(continued)

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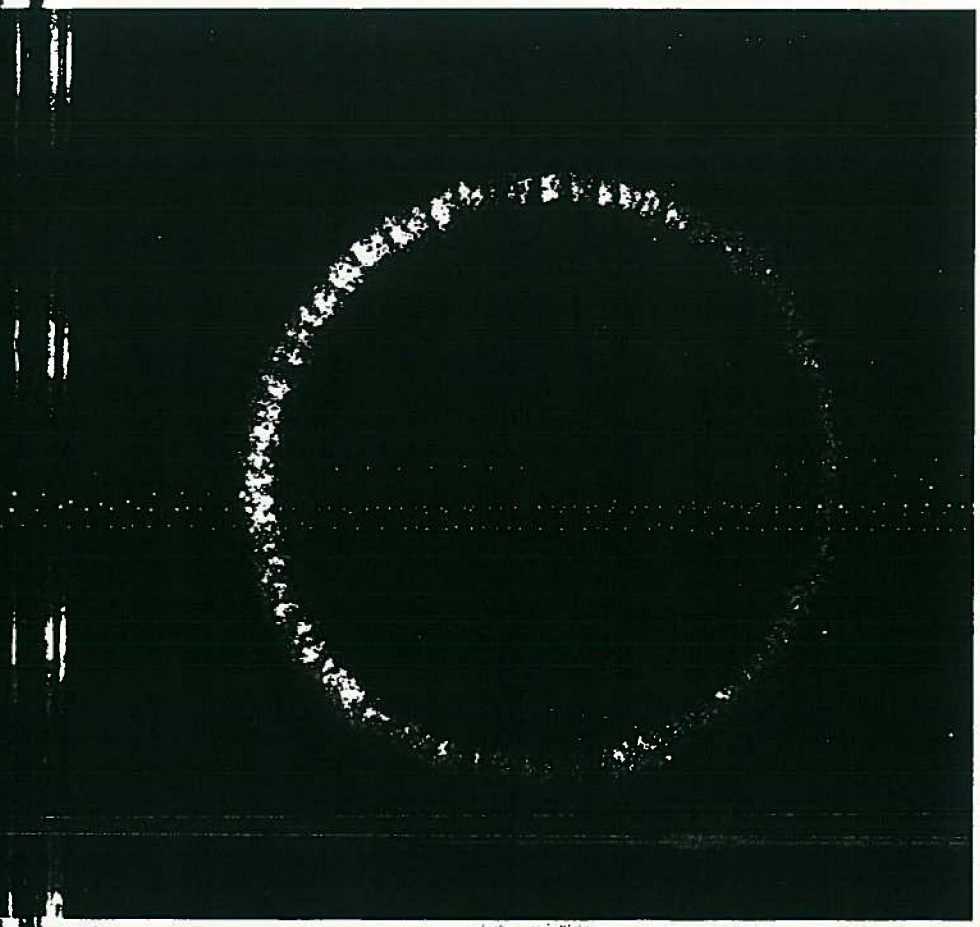

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Effect of sterol biosynthesis inhibitors on diseases of stone fruits and grapes in California

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1. ABSTRACT

In California, fungal diseases controllable with sterol biosynthesis inhibiting (SBI) fungicides on stone fruits are brown rot caused by *Monilinia fructicola* and *M. laxa*, shot hole caused by *Stigmina carpophila*, and the powdery mildews caused by *Podosphaera oxycanthae (P. clandestina)* and *Sphaerotheca pannosa*. On grapes in addition to powdery mildew caused by *Uncinula necator* the SBIs are effective against black rot of grapes caused by *Guignardia bidwellii* in the midwest and eastern

US and Europe. Since 1974, 13 SBI compounds including formulations of triazoles, piperazines, pyrimidines and imidazoles have been tested for control of blossom blights, foliage and twig infections, preharvest fruit rots, and post-harvest decays. Of the SBI compounds only the piperazine derivative triforine is currently registered for use on stone fruits and a triazole derivative, triadimefon, for grapes. Triforine received an Experimental Use Permit in 1978, Special Local Need registration for California in 1979, and registration in 1980 for use as an alternative to control benomyl-resistant isolates of *Monilinia* detected in California in 1977. On stone fruits, limited tests with SBIs shows control of the powdery mildews but its efficiency for control of the shot hole disease caused by *Stigmina carpophila* has not been confirmed. Data on SBI activity against *Botrytis* and *Rhizopus* have been limited. However, imazalil is effective as a post-harvest treatment to control *Botrytis* decay of fresh market tomatoes; reports on BAS 469 OOF indicate activity against *Botrytis* of red peppers and Folicur on *Botrytis* of grapes. Triadimefon is the only SBI compound registered for control of grape powdery mildew in California. One of the first SBI compounds to be extensively tested was the pyrimidine EL 271, which encountered registration problems. In 1982 the registration of triadimefon brought to the grape industry immediate benefits of better disease control than sulphur dust but possibly somewhat longer-term negative results. Reduced effectiveness was experienced whereby growers reverted again to using sulphur. Other benefits were the reduction in the number of applications required for control during the season with the possibility of delaying the initial treatment to take advantage of its eradicatory effect. The negative result was the selection of *Uncinula necator* isolates by 1985 which were only sensitive to tridimefon if treated 4 days after inoculation. Therefore effective powdery mildew control on grapes now requires triadimefon treatment every two to two and a half weeks based on the sensitivity of isolates instead of the original three to four week interval. In addition, triadimefon was found to be very effective in eradication of established mildew colonies. Powdery mildews on sweet cherry, peach, and plum can also be effectively controlled with triadimefon (not registered). In the eastern US triadimefon has allowed more flexibility on control of black rot diseases on grapes by taking advantage of its 'kick-back' action for up to 96 h. Also, the SBIs were found to be effective in control of cherry leaf spot disease caused by *Coccomyces hiemalis* and peach scab caused by *Venturia carpophila*. In California, further experiments are planned to test the SBIs against leaf rust on stone fruit caused by *Tranzchelia discolor* and almond scab caused by *Venturia carpophila*.

2. INTRODUCTION

Important fungal diseases on perennial stone fruits and grapes are controlled with fungicides. Little to moderate advancements have been made on other methods of disease control such as host resistance and cultural controls [7,21]. Thus the topic of fungicides for control of diseases of stone fruits and grapes strikes a historical note in line with the introduciton of sulphurs, coppers, dithiocarbamates, captan, benzimidazoles and dicarboximides to the current discussion on SBIs. Each of these classes of fungicide has played or plays a direct role in disease management systems and without them quality stone fruit and grape crops cannot be produced for current

markets. Trade names (Table 1) are used in our discussion as proprietary formulations were tested in field plots.

This presentation highlights our research on the triazole fungicides for control of brown rot on stone fruits and powdery mildew on grapes.

2.1 Stone fruit crops and their diseases

Stone fruit crops such as sweet cherries, apricots, nectarines, peaches, plums and prunes as well as the nut crop almonds are cultivated throughout the temperate zones of the world. In California these crops are grown primarily in the arid San Joaquin and Sacramento valleys with some in the coastal valleys. Current estimates show over 270 000 ha (Table 2) with the greatest area planted in almonds. This arid region has an average rainfall of 150 mm in the Southern San Joaquin Valley to 560 mm in the northern Sacramento Valley during the late fall through winter months with possible showers during bloom in February and March and essentially no rains during the summer months. Harvest season starts in May with the sweet cherries, peaches and nectarines followed by apricots and plums in June while the prune and almond harvests begin in August. Irrigation with drip, sprinkler and furrow appear to have little effect on disease development except increased incidence of almond shot hole and scab disease from the high-angle sprinklers. During the summer, dew occurs when the temperature drops during the night to the mid-50°F level (13°C) in July and August but the relation between persistence of dew and infections has not been clearly established. Thus the greatest concern is rain during May, June, July and August which triggers brown rot decay of fruits.

3. BROWN ROT OF STONE FRUITS

A brief background on the brown rot disease of stone fruits and almonds in California is given to provide the necessary picture on the complexity in developing control measures. The disease is caused by two closely related fungi, *Monilinia fructicola* (Wint.) Honey and *M. laxa* (Aderh. & Ruhl.) Honey. In California, the two species are somewhat selective in their host range with *M. fructicola* the principal species causing fruit rot of nectarines, peaches, prunes, plums and sweet cherries and hull rot of almonds.

M. laxa is the primary species causing blossom blight of apricots, almonds, prunes and plums, while *M. fructicola* is the primary species causing blossom blight on peaches and nectarines, and occasionally on apricots and almonds. Both species attack these stone fruits causing blossom blight and fruit rot and their population ratio has varied over the years [5]. Disease cycles for *M. fructicola* and *M. laxa* are shown in Figs. 1 and 2 [10]. Data collected over a 12-year period have been summarized elsewhere [11] (see Tables 2, 3 and 6).

The blossom blight phase of the brown rot disease may not seem to be important in terms of crop loss but spores produced on blighted blossoms serve as the major inoculum source for later fruit infections. For *M. fructicola*, the inoculum sources for the blossom blight are the conidia produced on overwintering fruit mummies on the tree, the previous year's blighted blossoms and infected fruit peduncles, and ascospores produced on fruit mummies partially buried on the orchard floor. For *M. laxa*, however, conidia produced on blighted blossoms and twigs as well as fruit

Table 1 — List of SBI and other fungicide formulations field tested on stone fruit and grapes in California

Company	Name		Year first tested
	Common	Trade or experimental	
Triazole derivatives			
Mobay	Bitertanol	Bay KWG 0599 25W	1978
		Baycor 25W, 50W	1979
	HWG 1608	HWG 1608 22.5% DP	1985
		Folicur	
	Triadimefon	Bay 6447 25W	1975
		Bayleton 50W	1977
Ciba–Geigy	Propiconazole	Tilt 3.6 EC	1985
		Orbit, CGA 65250	
	Etaconazole	CGA 64251 10W	1978
		Vangard 10W	1981
	Penconazole	Topas, CGA 71818	1984
Du Pont	DPX H6573	DPX 40% EC	1984
		Nustar	
Rohm & Haas	Myclobutanil	Systhane, RH3816, Ralley	1986
Chevron	Diniconazole	Xe 77, Spotless 25W	1986
Piperazine derivative			
E. M. Industries	Triforine	Cela 20% EC	1974
		Cela 50W	1978
		Cela 80F	1978
		T10225 50W	1979
		T10236 50W	1979
		Funginex 1.6 EC	1980
		Funginex 50W	1981
Pyrimidine derivatives			
Eli Lilly	Fenarimol	EL 222 12.5% EC	1976
		Rubigan 1.0 EC	1982
	Nuarimol	EL 228 9.46% EC	1976
		Trimidal 0.75 EC	1982
Imidazole derivatives			
Janssen	Imazalil	Imazalil 69.3% EC	1980
		Fungaflor	
Nor-Am	Prochloraz	Prochloraz 50W	1980
		BTS 40542, Sportak	
UniRoyal	Triflumizole	Procure 50% WP	1984
		A815	
Other formulations			
Diamond Shamrock	Chlorothalonil	Bravo 500	1975
		Bravo 6F	
Stauffer	Captan	Captan 50W	
Rhone-Poulenc	Iprodione	Rovral 50W	1975
		RP26019 50W	
		Rhodia 26019 50W	
ICI (Stauffer)		SC-0858	1986
Stauffer	Sulphur	Wettable Sulfur	
Nor-Am	DCNA	Botran 75W	

Table 2 — Crops, cultivars, production area, average number of spray applications and estimated cost of treatments for brown rot control in orchards

Crop	Cultivars grown	Production area (Ha)[a]	Number of fungicide applications per year		Cost[b] ($×1000)
			Systemic	Contact	
Apricots	13	9308	2	3	2792
Nectarines	60	9308	3	6	5585
Peaches					
Processing	38	14522	3	4	5821
Fresh market	69	12801	3	6	7681
Plums	52	13760	1	2	2752
Prunes	11	34131	1	2	6826
Sweet cherries	8	4800	2	4	1920
Almonds	40	171995	1	2	34399
Total		270665			67776

[a] 1986 figures.
[b] Cost figures derived by multiplication of production area, average number of treatments and application cost of fungicide treatment estimated at $100 and $50 per hectare.

mummies are the only sources of inoculum. Apothecia of *M. laxa* have not been observed in California. Sanitation programmes of mummy removal and pruning blighted twigs do reduce inoculum but are not sufficient for effective disease control. Eradicant-type treatment such as the application of calcium cyanamide to the orchard floor to prevent apothecial development has not been successful. Monocalcium arsenite on apricots and sodium pentachlorophenolate for apricots and almonds applied during full dormancy of the tree were extremely effective in reducing *M. laxa* inoculum for blossom infections but these compounds are no longer registered for use. Benomyl fungicide when combined with spray oil applied before sporodochial emergence [15] showed benefits in suppressing *M. laxa* sporodochial formation. SBI compounds have not been reported to suppress sporodochia. Without eradicant fungicides, blossom blight control with protectant fungicides relies on protection of susceptible blossom parts as they open which may take a week during warm temperatures (21°C) or could be prolonged to as much as two weeks or more during cool temperatures (13–18°C). Stone fruit blossoms are susceptible to *Monilinia* infections but the susceptibility of floral parts vary according to the crop. With peaches and nectarines, anther and filament infections are most common. Thus the partially systemic benzimidazole fungicides required one spray application just before or during anther emergence for protection of the stamens. With SBI compounds with no systemicity, two sprays are required to assure protection, the first just as the blossoms open (5% bloom) and another at around 80% or before the next infection period (rains). On almonds, in addition to stamens and stigma, the

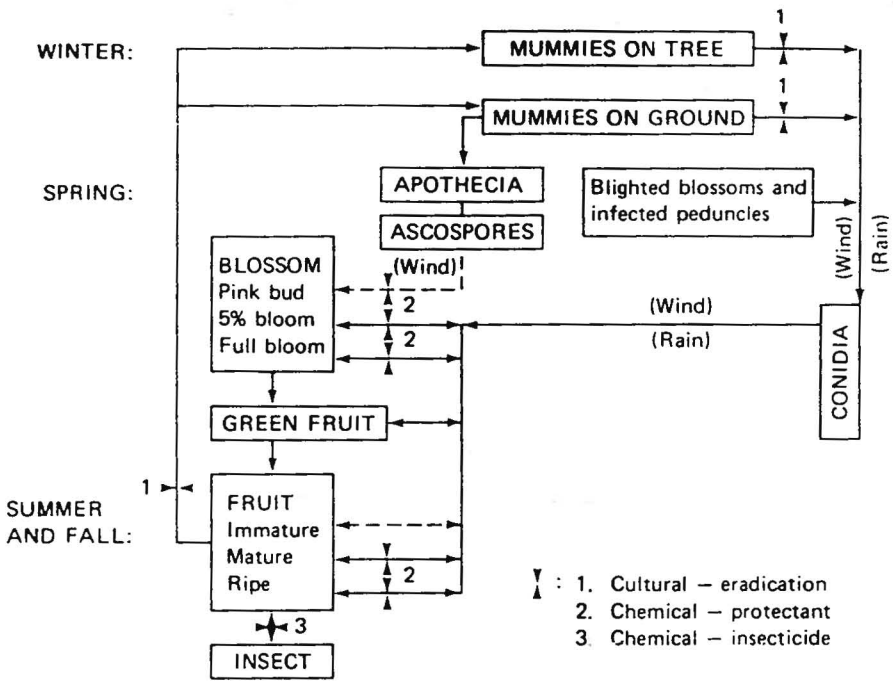

Fig. 1 — Disease cycle of *M. fructicola* on peaches and nectarines.

petals are also susceptible so, with a benzimidazole, a single spray as petals begin to show (pink bud) could afford adequate protection. Again, the SBI compounds require two applications, the first at pink bud, and the second when stigma and stamens emerge. On apricots and prunes all flower parts including the sepals are susceptible and require treatments starting at the red or green bud stage (sepals showing). On plum and sweet cherry blossoms the first treatment is applied a little later when the petals are showing for blossoms infected early tend to fall off as the fungus moves slowly down the long fruit peduncles and seldon causes flower cluster blight. Timely benzimidazole (benomyl and thiophanate methyl) treatments can reduce blossom blight as well as the number of conidia produced on diseased parts. With triforine (Funginex 1.6 EC) and iprodione (Rovral 50W) blossom blight control has been satisfactory but it lacks the residual activity of benomyl. An imidazole, prochloraz, provided excellent field control of blossom blight [3] but has not been registered for use.

4. TEST FOR BASELINE SENSITIVITIES TO TRIFORINE AND ETACONAZOLE

With the possible development of resistance to fungicides in the *Monilinia* species, baseline sensitivities of *M. fructicola* and *M. laxa* to triforine and the etaconazole

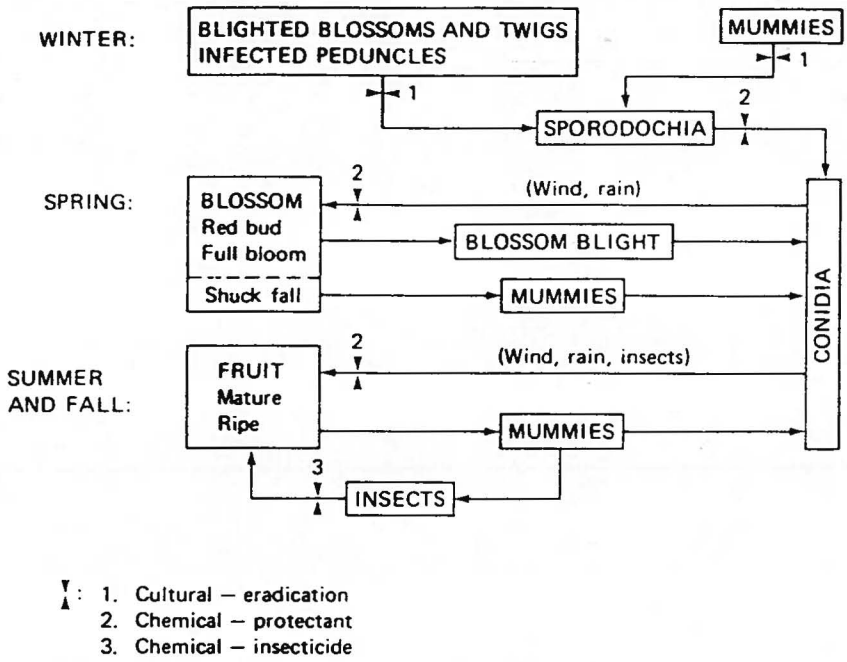

Fig. 2 — Disease cycle of *M. laxa* on apricots.

were established. Conidial germination and mycelial growth studies were made on Difco potato–dextrose agar (PDA) amended with the fungicide. For triforine (*N,N'*-[1,4-piperazinediylbis(2,2,2-trichloroethylidene)]bis(formamide)), conidial germination was 93% or greater for *M. fructicola* and 78–85% for *M. laxa* on medium amended with 10 μg/ml active ingredient. Germination of both species was reduced to 2% or less with concentrations greater than 50 μg/ml. ED_{50} values for mycelial growth inhibition were as follows: 2.9 μg/ml for *M. fructicola* isolate MUK-1 (sensitive to benomyl), 3.2 μg/ml for KASH-1 (resistant to benomyl), 7.4 μg/ml for *M. laxa* isolate MLC-2 (sensitive to benomyl) and 9.1 μg/ml for ML9-80 (resistant to benomyl) (Fig. 3). For etaconazole (1-[(2-(2,4-dichlorophenyl)-4-ethyl-1,3-dioxolan-2yl) methyl]-1H-1,2,4-triazole)condial germinations were 92.5% for *M. fructicola* and 95.6% for *M. laxa* on medium amended with 100 μg/ml and 0% for both on medium containing 500 μg/ml. ED_{50} values for mycelial growth inhibition were 0.08 μg/ml and 0.1 μg/ml active ingredient for *M. fructicola* and *M. laxa*, respectively (Fig. 4).

Conidia used for the germination study were harvested from 9-day old colonies of *M. fructicola* grown on PDA and from 9-day old colonies of *M. laxa* grown on oatmeal agar. Conidia were suspended in sterile glass distilled water and adjusted to 1×10^5 conidia per millilitre. 50 μl drops of conidial suspension, replicated three times for each reading, were placed onto fungicidal-amended or non-amended PDA plates. 100 conidia were examined for each replicate and were considered germinated when the germ tube length equaled or exceeded the length of the conidia.

Table 3 — Average per cent control of brown rot blossom blight of stone fruits with triazole derivatives and Rovral[a]

Concentration (oz 100 gal)	Triazole compounds[b]							
	Bayleton	Folicur	Baycor	Orbit	Vangard	Nustar	Systhane	Rovral
Peaches								
24.0			41 (2)					100
16.0			75 (4)	100				
8.0	89 (3)		70 (3)					83
6.0	71		98 (2)		97 (3)			
4.5	68 (2)		88					
4.0	90 (3)				82 (7)			85 (3)
3.75						90		
3.0	86 (2)		82 (2)					
2.0	58 (2)			94				
1.0				98				
Nectarines								
16.0			83					
8.0	86 (2)		92					
6.0	100		100		96			100
4.0	89		82		83			
3.75						96		
3.0	83 (2)		86					
1.0				88				
Plums								
16.0			55					
8.0			40					
5.0					79			
4.0	79							
Apricot								
16.0								80 (2)
10.0							50	
8.0				91				
5.0	67						45	
4.0				87				
2.5	27							
Prune								
6.0								97
4.5					97			
Almond								
16.0			79					
8.0	80							
6.0			60		62 (2)			
4.0	85				81 (2)			

[a] Per cent control based on comparison with non-treated plot; values in parentheses are the numbers of field tests made if more than 1.
[b] Proprietary compounds: Bayleton 50W; Baycor 25W; Folicur 22.5% DP; Orbit 3.6 EC; Vangard 10W; Nustar 40% EC and 20% DF; Systhane and Spotless 25W.

For mycelial growth studies, 27 benomyl-sensitive and five benomyl-resistant *M. fructicola* and 27 benomyl-sensitive and one benomyl-resistant *M. laxa* were tested on proprietary triforine (Funginex 1.6 EC, EM Laboratories Inc., Hawthorne, NY)

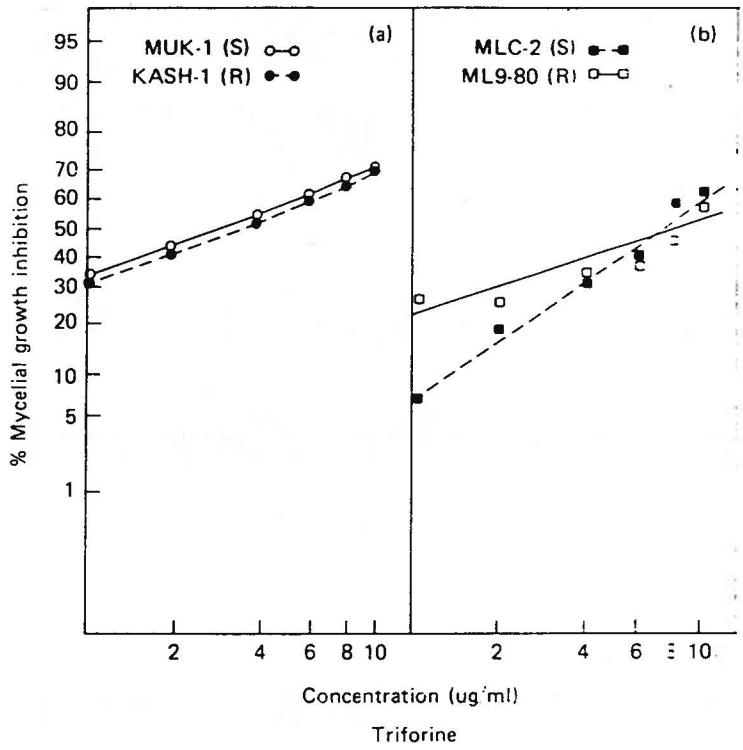

Fig. 3 — Dosage response curve of benomyl-sensitive *M. fructicola* (MUK-1) and *M. laxa* (MLC-1) compared with benomyl-resistant *M. fructocola* (KASH-1) and *M laxa* (ML9-80) on PDA medium amended with triforine.

or proprietary etaconazole (Vangard 10W, CGA 64251 10W, Ciba–Geigy Corp., Greensboro, NC) diluted in sterile distilled water and added to cooling PDA. 4 mm disks of 5-day old colonies grown on PDA were transferred to fungicide-amended or non-amended PDA. Plates were left at room temperature (21 ± 1°C) and colony diameters for the five replications measured daily for each concentration. Mycelial growth inhibition was calculated as per cent inhibition relative to growth on fungicide-free medium. Linear regression equations were fitted to the data by using logarithms for each concentration. Probits of percentage growth inhibition were used to determine ED_{50} values. Dosage response curves were plotted on logarithmic-probability paper.

Baseline sensitivity studies are necessary for other SBI compounds on pathogens such as *M. fructicola*, *M. laxa*, *Sphaerotheca pannosa* and *Podosphaera oxycanthae* before their use on commercial crops.

4.1 Fungicide testing on stone fruit blossoms

Since 1974, triazole derivatives have been compared with the dicarboximide Rovral in 78 field trials (Table 3) [9]. Four triazole compounds (Bayleton, Baycor, Orbit and

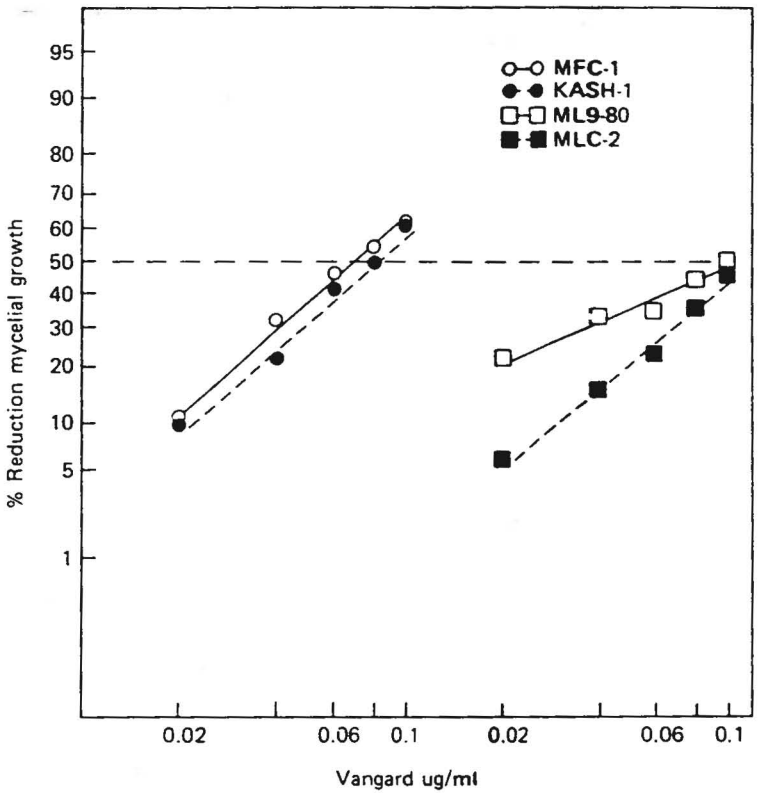

Fig. 4 — Dosage response curve of benomyl-sensitive *M. fructicola* (MFC-1) and *M. laxa* (MLC-2) compared with benomyl-resistant *M. fructicola* (KASH-1) and *M. laxa* (ML9-80) on PDA medium amended with etaconazole.

Vangard) were found effective in control of stone fruit blossom blight, especially brown rot caused by *M. fructicola*. The concentration required per 100 gal of proprietary material with Bayleton is 6–8 oz, Baycor 4–6 oz, Orbit, 1–2 oz and Vangard 6 oz. The results were more variable on control of *M. laxa* blossom blight of apricots and almonds. Preliminary trials on apricots with Systhane at 10 oz/100 gal were not outstanding. Tests on almonds (*M. laxa*) showed that the amount of active ingredient required with DPX H6573 was less than that for Funginex or Rovral (Table 4). An example of field data obtained for blossom blight control is shown for Fay Elberta peaches (Table 5). Direct comparisons with the standards, Rovral 50W and Benlate 50W plus captan 50W, show that the SBI compounds tested are equally effective with no significant separations.

4.2 Fungicide testing for preharvest brown rot control

Effective blossom blight control is essential to prevent quiescent infections of developing fruit during the blossoming period. Furthermore, blighted blossoms are a source of inoculum throughout the summer months. Rains during the last month

Table 4 — Evaluation of SBI fungicides for control of brown rot (*M. laxa*) on Blenheim apricots (1984 season)

Treatment[a]	Concentration		Average number of blighted twigs per tree[b]
	(/100 gal)	(g a.i./ha)	
Rovral 50W	4.0 oz	561	10.7 X
DPX H6573 40% EC	2.4 fl oz	282	12.7 X
Funginex 1.6 EC	12 fl oz	672	14.0 X
DPX H6573 40% EC	1.2 fl oz	141	24.0 Y
Non-sprayed	—	—	38.3 Z

[a] Two blossom sprays: February 25, 1% bloom; March 5, full bloom.
[b] Numbers followed by the same letter are not significantly different, $P = 0.05$.

Table 5 — Evaluation of fungicides for control of blossom brown rot (*M. fructicola*) of Fay Elberta peaches (1983 season)

Treatment[a]	Concentration		Average number of blossom blight per tree[b]
	(/100 gal)	(g a.i./ha)	
Rovral 50W	4 oz	561	0.5 X
Benelate 50W	6 oz	840	
+			0.8 X
Captan 50W	24 oz	3363	
Rubigan 1 EC	8.5 fl oz	311	1.5 X
Funginex 1.6 EC	12 fl oz	672	1.8 X
Bayleton 50W	3 oz	420	1.8 X
Vangard 10W	4 oz	111	3.2 X
Rubigan 1 EC	4.25 fl oz	156	3.3 X
Funginex 1.6 EC	8 fl oz	447	4.0 XY
Bravo 500	24 fl oz	3509	8.5 Y
Non-sprayed	—	—	23.8 Z

[a] Two blossom spray applied with hand-gun sprayer, 3.8 gal/tree at early pink (February 14) and full bloom (March 2).
[b] Average of 400 blossoms counted on each of six trees. Disease read on April 6. Numbers followed by the same letter are not significantly different, $P = 0.05$.

before harvest have triggered fruit rot epidemics and protective SBI treatments applied before the rain effectively reduce disease. Post-infection treatment with liquid lime-sulphur suppressed decay of processing peach while SBIs provided some benefits. As a protective treatment, benzimidazole compounds were effective for a

longer period than the SBIs or dicarboximides on peaches, nectarines and sweet cherries. Effective disease control has not been observed from preharvest treatments on apricots, prunes and almonds.

Since 1974, 111 evaluations have been made on triazole derivatives and all the compounds (Table 6) are active against fruit brown rot caused by *M. fructicola*. Dosages (about 8 oz/100 gal for Bayleton, Folicur, Baycor, Orbit and Vangard and 2 oz/100 gal for Nustar) are similar to those required for blossom blight control. Examples of specific data supporting the summary are presented in Tables 7–9. On peaches, Bayleton was comparable with Funginex, Vangard and Rubigan as well as Benlate plus captan. On nectarines, DPX H6573 (Nustar) and Funginex were comparable with and significantly better than Systhane, Ronilan, Spotless and Rovral. On plums, DPX H6573 (Nustar) and HWG 1608 (Folicur) performed better than Funginex, Rovral or Tilt.

Derivatives of pyrimidines (EL 228 and EL 222) and an imidazole (Prochloraz) are compared with a piperazine (Funginex) in Table 10. For blossom blight, pyrimidines appear less effective at the dosages tested while prochloraz was consistently better even at 4–6 oz/100 gal. Prochloraz is not being considered for fruit rot control because it cause off-flavours. Sufficient efficacy has not been determined for the pyrimidines.

4.3 Fungicide testing for post-harvest decay control

Post-harvesting disease control is essential in fresh market, perishable stone fruits [12]. *M. fructicola* and *Botrytis cinerea* as well as *Penicillium expansum* are controlled effectively by fungicide sprays in combination with waxes. DCNA (Botran 75W) applied immediately following the washing and defuzzing of fruit controls *Rhizopus stolonifer*.

Triforine (50% wettable powder) applied in a water suspension spray before fruit waxing has been effective against *Monilinia* sp. In water the activity of triforine decreases quickly so the suspension spray is prepared just before application. Studies on brown rot of nectarine fruit show equivalent control among the standard Benlate 50W plus Botran 75W plus Funginex 50W, Stauffer Chemical Company's (now ICI, England) SC 0858 and Rovral 50W (Table 11).

5. POWDERY MILDEW OF STONE FRUITS

Climates suitable for stone fruit production also favours disease from powdery mildew fungi. The life cycle of the pathogens as well as its importance on various stone fruits and cultivars vary. The important pathogens are *Sphaerotheca pannosa* on peaches, nectarines and apricots, *Podosphaera oxycanthae* (*P. clandestina*) on sweet cherries, and the mildew species on Red and Black Beaut plums which have not been identified. Sulphur fungicides are commonly used to control mildew on susceptible cultivars of peaches and nectarines and more recently on Red Beaut and Black Beaut plums. Wettable sulphur is used on sweet cherries for mildew control. On apricots, mildew is controlled by removing the host plant (roses) as well as fungicide sprays. On almonds and prunes, mildew is not a problem.

Sulphur sprays and dusts are used most commonly as a protectant against fruit infections. The first application is made after bloom and additional treatments are

Table 6 — Average per cent control of brown rot fruit rot of stone fruits controlled with triazole derivatives and Rovral[a]

Concentration (oz/100 gal)	Triazole compounds[b]							
	Bayleton	Folicur	Baycor	Orbit	Vangard	Nustar	Systhane	Rovral
Peaches								
32.0			56 (2)					
24.0					68 (3)			
16.0			32 (3)					63
15.0 DF						72		
8.0	93 (5)		58 (3)	75				
7.1		94						
6.0			81 (2)	70 (2)				
6.0	85							
4.5	76		55					
4.0	72 (2)		35 (2)	69	69 (7)			61 (3)
3.6		85						
3.0	79 (3)		51 (2)					
2.5						92 (2)		
2.0				87				
1.25						92 (2)		
1.0				70				
Nectarines								
16.0			63					49 (2)
15.0 DF						83 (2)		
10.0							56	
8.0	90 (3)		68 (2)					
6.0	84		88		74 (2)			50
4.0	76		49	86	60 (2)			68 (2)
3.0	62 (2)		65					
2.5						98 (2)		
2.0				60				
1.0				36				
Plums								
16.0			90 (2)					
15.0 DF						71		
8.0	77 (2)		78 (2)					
6.0			98 (2)		47 (2)			
4.0			41 (2)		54 (4)			61
3.6		97						
3.0			55					
2.5						86 (2)		
2.0				21				
1.25						54 (2)		
1.8		98						

[a] Per cent control based on comparison with non-treated plot: values in parentheses are the numbers of field tests made if more than 1.
[b] Spotless or diniconazole at 6.4 oz/100 gal in water gave on nectarines 46% reduction of fruit rot.

Table 7 — Efficacy of preharvest fungicide sprays in reducing post-harvest decay of Fay Elberta peach fruit (1983 season)

Treatment[a]	Concentration		Per cent brown rot[b]
	(/100 gal)	(g a.i./ha)	
Bayleton 50W	3 oz	420	16.0 V
Funginex 1.6 EC	12 fl oz	672	19.2 VW
Vangard 10W	4 oz	111	22.0 VW
Rubigan 1 EC	8.5 fl oz	311	22.0 VW
Benlate 50W	6 oz	840	
+			33.2 YWX
Captan 50W	24 oz	3363	
Rovral 50W	4 oz	561	
+			38.8 XY
oil	32 fl oz		
Non-sprayed	—		67.2 Z

[a] Two blossom spray (February 24 and March 2) and two preharvest (July 12 and July 26) sprays applied with hand-gun sprayer. 4 gal/tree. Harvested August 2.
[b] Per cent disease figures are averages of 40 fruit replicated five times. Disease evaluation made after 4 days at 20°C. 85% RH. Numbers followed by the same letter are not significantly different, $P = 0.05$.

Table 8 — Efficacy of preharvest fungicide sprays in reducing post-harvest brown rot (*M. fructicola*) on nectarines (1986 season)

Treatment[a]	Concentration		Per cent brown rot on fruit[b]
	(/100 gal)	(g a.i./ha)	
Funginex 1.6 EC	12 fl oz	672	16.4 W
DPX H6573 20%	3.8 oz	210	23.2 W
Systhane 40W	2.5 oz	279	
+			42.4 X
Triton CS-7	2 fl oz		
Ronilan 50W	8 oz	1121	48.8 X
Spotless 25W	1.6 oz	112	
+			51.2 X
X-77	8 fl oz		
Rovral 50W	4 oz	561	61.2 XY
Non-sprayed	—		95.6 Z

[a] Two spray applications (June 5 and June 17) except for dicarboximides Funginex and Rovral which were sprayed also at full bloom (February 27). Harvest June 18. Handgun sprays at 4 gal/tree.
[b] Five single tree replications. 50 fruit harvested and incubated from each replication and incubated at 20°C, 90% RH for 4 days. Numbers followed by the same letter are not significantly different, $P = 0.05$.

Table 9 — Efficacy of preharvest fungicide sprays in reducing post-harvest decay of Casselman plum fruit (1985 season)

Treatments[a]	Concentration		Per cent brown rot on fruit[b]
	(/100 gal)	(g a.i./ha)	
HWG 1608 22.5% DP	4.0 oz	252	0.6 W
HWG 1608 22.5% DP	8.0 oz	504	1.1 W
DPX H6573 40% EC	2.4 fl oz	282	5.3 WX
DPX H6753 40% EC	1.2 fl oz	141	8.8 WX
Funginex 1.6 EC	9.0 fl oz	504	8.8 WX
Rovral 50W	4.0 oz	561	12.2 XY
Tilt 3.6 EC	2.0 fl oz	252	14.5 XY
Tilt 3.6 EC	1.0 fl oz	126	23.9 Y
Non-sprayed	—		37.3 Z

[a] Two blossom (5% bloom on February 27 and full bloom on March 5) and two preharvest (July 17 and August 7) sprays applied with hand-gun sprayer at 4 gal/tree. Harvested on August 14 and evaluated after 9 days incubation at 20°C and 90% RH. Fruit were inoculated with *M. fructicola* spore suspension before incubation.

[b] Per cent disease based on averages of 150–175 fruit replicated three times. Numbers followed by the same letter are not significantly different, $P = 0.05$.

based on disease severity. SBI compounds have been shown to provide equivalent protection with suggestions of eradicant action. If SBI compounds can be applied during the blossoming period for control of both mildew and brown rot, this would be advantageous. Results of experiments on Bing cherries and Black Beaut plums are presented in Table 12 and 13.

6. SHOT HOLE OF STONE FRUITS

Some SBI compounds appear to have some activity on shot hole of stone fruits caused by *Stigmina carpophila* but are less effective than the standard fungicides used, such as copper, captan and ziram [21]. On Drake almonds, SBI compounds were less effective than chlorothalonil which is not commercially used solely for control of shot hole (Table 14).

7. SBIs ON OTHER STONE FRUIT DISEASES

Peach scab caused by *Venturia carpophila* and cherry leaf spot caused by *Coccomyces hiemalis* are both effectively controlled by SBI compounds in the eastern US [6]. In California, fungicide tests are planned with SBI compounds for control of almond scab and cherry leaf spot if the disease occurs or can be induced, and also leaf rust on almonds, prunes and peaches caused by different forma specialis of *Tranzschelia discolor*.

Table 10 stone

Concentration (oz/100 gal)	
Peach	
48.0	
36.0	
13.5	
12.0	
10.2	
9.0	
8.0	
6.0	
4.0	
2.0	
Nectarines	
48.0	
12.0	
06.0	
04.0	
Plums	
9.0	
6.0	
4.0	
Apricots	
10.2	
6.0	
1.8	
Prunes	
12.0	
6.0	
Almonds	
10.2	
8.0	
6.0	
4.0	
Sweet cherrie	
12.0	
8.0	
6.0	

[a] Per cent co
field tests ma
[b] Proprietary
50W.

Table 10 — Average per cent control of brown rot blossom blight and fruit rot of stone fruits controlled with piperazine, pyrimidine and imidazole derivatives

Concentration (oz/100 gal)	Piperazine[b]: Funginex		Pyrimidine[b] EL 228		EL 222		Imidazole[b]: Prochloraz	
	BB	FR	BB	FR	BB	FR	BB	FR
Peach								
48.0	100	61						
36.0	86 (3)	96						
13.5			72					
12.0	77 (3)	74 (3)						
10.2					51	86		
9.0	77	77 (4)						
8.0		64					98	
6.0							92 (2)	76 (2)
4.0							96 (2)	93
2.0							99	
Nectarines								
48.0		83						
12.0	86	81 (2)						
06.0							88 (2)	94 (2)
04.0							96	
Plums								
9.0			88					
6.0								100 (2)
4.0							95	
Apricots								
10.2					78			
6.0							85	
1.8	64							
Prunes								
12.0	88							
6.0							98	
Almonds								
10.2					55			
8.0	66						87	
6.0							100	
4.0							83	
Sweet cherries								
12.0		67						
8.0	65	78 (2)						
6.0								94

[a] Per cent control based on comparison with non-treated plot; values in parentheses are the numbers of field tests made if more than 1.
[b] Proprietary formulations are Funginex 18% EC, EL 222 12.5% EC, EL 228 9.46% EC and Prochloraz 50W.

Table 11 — Efficacy of post-harvest fungicide sprays in reducing decay of Fairlane nectarine fruit

Treatment[a]	Concentration (μg/ml)	Per cent brown rot[b]
Benlate 50W + Botran 75W + Funginex 50W	600 + 1800 + 600	6.0 X
SC 0858 50W	600	7.0 X
Rovral 50W	600	10.0 X
Guazatine 40 EC	200	20.0 Y
Non-sprayed	—	51.0 Z

[a] Fungicide applied with a small commercial post-harvest treater. Water soluble peach wax was used on all treatments.
[b] Healthy and disease percentage are averages of 25 fruit replicated four times. Disease evaluations made after 4 days at 20°C, 90% RH. Numbers followed by the same letters are not significantly different, $P = 0.05$.

Table 12 — Efficacy of fungicides in protection and eradication of powdery mildew of Bing cherry on leaves and fruit (1977 season)

Treatment[a]	Concentration		Diseased[b]	
	(/100 gal)	(g a.i./ha)	Leaves (/300 shoots	Fruit (/100 fruit)
Rubigan 1.25% EC	3.4 fl oz	124	11.0 Y	6
Bayleton 50W	4.0 oz	561	11.3 Y	6
Wettable Sulfur 92%	5.0 lb	20615	15.3 Y	5
Control	—	—	57.3 Z	31

[a] Three large branches sprayed on each of three trees sprayed twice. First spray applied on April 4 before mildew symptoms and the second on April 20 at time few leaves showed signs of mildew.
[b] Data collected before harvest when fruit still green. Numbers followed by the same letter are not significantly different, $P = 0.05$.

8. POWDERY MILDEW ON GRAPES

Powdery mildew caused by *Uncinula necator* (Schw.) Burr, also called *Oidium* in Europe and South America, is possibly the most common fungal pathogen of grapevines worldwide. In California, powdery mildew has a large economic impact on grape production, both in terms of economic loss due to disease as well a dollars

Table 13 — Efficacy of fungicides for control of powdery mildew on Black Beaut plum fruit (1984 season)

Treatment[a]	Concentration		Per cent fruit with mildew[b]
	(/100 gal)	(g a.i./ha)	
Trimidal 0.75 EC	3.8 fl oz	100	0.08 Y
Bayleton 50W	3.0 oz	420	0.08 Y
Funginex 1.6 EC	12.0 fl oz	671	0.08 Y
Wettable Sulfur 92%	5.0 lb	20615	0.17 Y
Non-sprayed	—	—	0.92 Z

[a] Two hand-spray applications with the first on April 2 and second on April 9 on each of six trees.
[b] Evaluations made on May 9. Numbers followed by the same letter are not significantly different. $P = 0.0$.

Table 14 — Evaluation of fungicide for control of shot hole disease of Drake almond (1983 season)

Treatment[a]	Concentration		Per cent healthy leaves[b]
	(/100 gal)	(g a.i./ha)	
Captan 50W	24 oz	3363	
+			69.8 V
Benlate 50W	6 oz	840	
Bravo 500	24 fl oz	3509	55.3 W
Vangard 10W	4 oz	111	34.2 X
Funginex 1.6 EC	8 fl oz	447	26.2 Y
Rubigan 1 EC	4.2 fl oz	156	21.4 Y
Non-sprayed	—	—	10.4 Z

[a] Two blossom sprays applied with hand-gun sprayers 6 gal/tree at 1% bloom (February 20) and early petal fall (March 4). For Bravo the third spray was applied March 15.
[b] Evaluations made by cutting 10 shoots (length 8–10 in) from each of six single-tree replications on March 18. Numbers followed by the same letter are not significantly different, $P = 0.05$.

spent in control programs. The powdery mildew pathogen attacks all succulent or green portions of the grapevine. One source of primary inoculum for powdery mildew epidemics has been shown to be overwintering mycelium in infected dormant buds in many production areas of the world including California [5], Western Europe [6] and South Africa [10]. In California, shoots from infected buds may show disease symptoms and signs of the fungus soon after bud break. Symptoms include stunting of shoots and leaves and leaf distortion. White web-like fungal growth soon occurs on

the affected leaves and conidial production which occurs in 10–14 days allow for rapid secondary spread within the affected grapevine canopy.

Although the pathogen also commonly produces its perfect stage in many viticulture areas it has only recently been shown that cleistothecia are the primary source of overwintering on grapevine in New York [14].

Pearson and Gadoury [14] have found no evidence of overwintering bud infection in New York vineyards. Their work showed that ascospores were released from cleistothecia beginning in mid-to-late April and continued for 6–10 weeks with initial release coinciding approximately with bud burst. Initial ascosporic infections were found to occur on basal leaves in close proximity to cordons and the head of the vine.

Secondary inoculum in the form of conidia is produced in 5–7 days under optimal temperatures of 23–30°C [12]. Because of the relatively rapid inoculum build-up of *U. necator* it is important that disease control strategies take into account the early-season infection capabilities of the pathogen as well as the rather explosive epidemic threat that ensues from secondary inoculum.

8.1 Fungicide testing for grape powdery mildew control

In California [13], and elsewhere [1, 2, 17], grapevine powdery mildew has been controlled by repeated sulphur or Bayleton applications based solely on the phenology of the grapevine with little regard to infection by primary inoculum. This type of control strategy is necessary because of the general lack of information regarding infection periods resulting in the onset of disease. However, recent efforts to correlate initial disease occurrence with environmental parameters suggest that infection takes place in the coastal production areas of California soon after spring rains with colony growth occurring in 7–10 days (Gubler, Stapleton and Chellemi, unpublished). Similar results have been observed in New York [14]. These findings should aid in the development of a potentially more successful control strategy based on conditions affecting initial infection.

Prior to 1982, control programmes consisted primarily of protectant sulphur applications. Economic control in a severe powdery mildew year required growers to apply wettable sulphur or dusting sulphur on a 7–10 day interval with immediate re-application following rain or sprinkler irrigation. In cases where powdery mildew was not adequately controlled using a protectant sulphur program, an eradication spray, consisting of high water volume, a wetting agent (sodium lauryl sulphate) and wettable sulphur, was used. In 1982, triadimefon (Bayleton) was introduced into California vineyards. The impact of Bayleton on the grape industry had immediate beneficial results but possible longer-term negative results. Spray initiation was moved back from 10–15 cm shoot growth to 45–60 cm shoot growth, application intervals were lengthened from 7–10 days to 21–28 days and three to four applications per year resulted in economic control. The recommended spray schedule was 150–300 g/ha applied at 45–60 cm shoot length, bloom and pea-sized berry. Excellent results were achieved using these recommendations until 1985. In that year and again in 1986 the recommended programme did not provide economic mildew control and 1986 was one of the most severe powdery mildew epidemic years on record in California. Much of the lack of control using Bayleton could be attributed to lax efforts by growers, i.e. waiting to spray until the disease was established (although the product was considered an eradicant), decreased fungicidal rates, poor coverage

as a result of equipment failures or excessive tractor speed or longer intervals between applications.

Research initiated in 1986 resulted in information regarding what the spray schedule should be in California under conditions conductive for rapid pathogen population build-up. Under conditions that favour a completion of the disease cycle in 5–7 days, i.e. 23–30°C and 75–80% RH, the spray interval had to be shortened from 21 days to 12–17 days depending on the particular mildew isolate tested.

Data shown in Table 15 indicates that, for this particular isolate, one could expect

Table 15 — Infection of deteched Chenin blanc leaves inoculated with *U. necator* 1, 8, 10, 14, 17, 19, 21 or 24 days after application of Bayleton

Bayleton concentration (g/ha)	Per cent of leaves showing mildew colonies for incubation on the following numbers of days after Bayleton application							
1	8	1	0	14	17	19	21	24
0	100	14	14	29	80	57	57	33
300	0	0	0	0	67	33	43	17
562	0	0	0	0	17	71	43	29

to get 14–17 days of protection against *U. necator* infection when using Bayleton at 300–562 g/ha. However, when an isolate obtained from a vineyard in which Bayleton usage afforded little protection was tested, results showed that the 300 g/ha rate of Bayleton afforded protection for only 6–7 days (Fig. 5). The data suggest that now

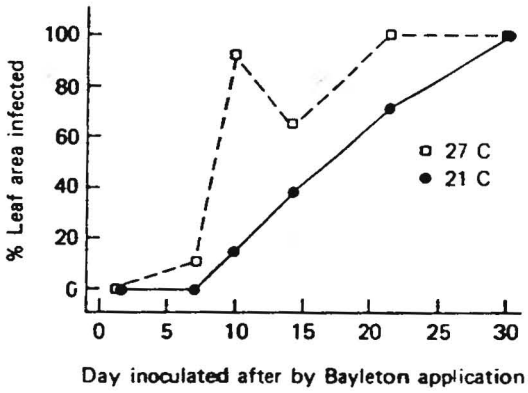

Fig. 5 — Effect of Bayleton 50W residual (150 g/ha) on colony of *U. necor* established on carignane seedlings when inoculated at intervals following treatment.

there are differences in isolates with regard to their ability to colonize leaves after Bayleton usage. Whether these differences existed prior to 1985–1986 is unknown. The information partially explains the problems California growers encountered in controlling powdery mildew in 1985 and 1986 since most growers were using a 21 day or longer spray interval. The effect of temperatures was also investigated. Tests at 21 and 27°C (Fig. 5) indicate that *U. necator* is capable of more rapid colonization at 27°C than at 21°C. These results are in close agreement with those of Delp [2].

Research conducted to test the eradicant capabilities of Bayleton showed that post-inoculation application of 300 g/ha controlled *U. necator* up to 4 days after inoculation. Applications made 5 days after inoculation allowed *U. necator* to become established and applications made 7 and 10 days after inoculation resulted in no significant control (Table 16). The results of these tests suggest that if a spray

Table 16 — Effect of Bayleton 50W as an eradicant when applied at intervals following inoculation with *U. necator*

Bayleton treatment[b]	Powdery mildew evaluation[a]			
	Day 10	Day 14	Day 21	Day 31
Day 3	2	0	0	1
Day 5	5	4	3	3
Day 7	5	3	6	5
Day 10	9	8	10	10
Check	10	10	10	10

[a] Leaves were evaluated for sporulating colonies at 10, 14, 21 and 31 days after inoculation. The number of leaves with sporulating powdery mildew colonies is shown: 10 leaves were evaluated in each case.
[b] Bayleton was applied at 80 ppm (300 g/ha) in the equivalent of 1900 l/ha 3, 5, 7 and 10 days after inoculation with *U. necator*.

interval is stretched 4–5 days beyond the normal 14–17 day interval it would not be practical to expect economic control using Bayleton.

Tests conducted to determine whether resistant isolates occur in California shows that all isolates tested could be controlled using protectant sprays of 150–300 g/ha. However, when exposed to sublethal rates of 1–20 ppm active triadimefon, variation in isolate sensitivity to Bayleton was observed. ED_{50} values (Table 17) showed a range of sensitivity from 5.6 to 19.2 ppm for California isolates while New York and Canadian isolates showed ED_{95} values of 4.3 ppm and 3.2 ppm respectively.

Powdery mildew control trials have been established in several locations in California for several years. The primary purpose of these trials is to evaluate new fungicides. Since 1982 there have been several SBI fungicides tested. All appear to have extremely good activity against powdery mildew. In the 1985 field trial located in Monterey County, California (Table 18), mildew pressure was heavy and most compounds gave satisfactory control of powdery mildew. Bayleton used at 300 g/ha applied on a 21 day schedule did not provide economic control. In this production area, powdery mildew is a serious threat to the industry each year. The primary

Isolate

206-1
243-4
291-5
M-1
BBRR
225-5
225-3
206-3
243-3
312
234
314
313
BBRR
316
243-1
300
NY 1
CAN 1

Table 18

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[b] Figures re

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Table 17 — Bayleton ED_{50} values for 19 isolates of *U. necator*

Isolate	Location	ED_{95} (ppm a.i.)
206-1	Kern County	19.5
243-4	Santa Barbara County	15.3
291-5	Sonoma County	15.3
M-1	Tulare County	14.8
BBRR	Napa County	14.4
225-5	Fresno County	11.4
225-3	Fresno County	11.4
206-3	Kern County	10.6
243-3	Santa Barbara County	9.4
312	Sonoma County	9.0
234	Kern County	7.7
314	Sonoma County	7.5
313	Somoma County	7.2
BBRR	Napa County	7.1
316	Sonoma County	6.6
243-1	Santa Barbara County	5.6
300	Yolo County	5.6
NY 1	New York	4.3
CAN 1	Canada	3.2

Table 18 — Control of grapevine powdery mildew, var. Chardonnay, using ergosterol biosynthesis inhibiting fungicides (Monterey County, 1985)

Treatment[a]	Formulation	Rate	Per cent incidence[b]
Control			82.1 A
Bayleton	50W	285 g/ha	34.1 B
Procedure	50W	285 g/ha	5.5 C
Rally	40W	213 g/ha	5.2 C
Rally	40W	213 g/ha	3.9 C
Procedure	50W	426 g/ha	0.8 C
Topas	1 EC	320 g/ha	0.3 C

[a] Treatments made on a 21-day schedule. First application made at 45 cm shoot growth.
[b] Figures represent average per cent disease incidence fom 25 clusters in each of four replications.

reason is that temperatures are mild and fog and high relative humidities exist during the morning and late evening–night hours and the growing season is extended owing to the slow maturing of fruit. The data obtained from this field trial partially support

data obtained from greenhouse and laboratory studies that, under conditions optimum for pathogen reproduction, spray application intervals beyond 17 days allow for disease build-up between applications.

A similar field trial was established in 1987 in Yolo County, California. Environmental conditions in this area are not optimum for the pathogen in that daily maximum temperatures during June, July and August generally range from 90 to 105°F and relative humidities range between 20% and 40%. However, even in this location, powdery mildew has caused serious losses over the years. Results of this trial (Table 19) show that mildew pressure was relatively high in 1987. All materials

Table 19 — Control of powdery mildew on grapevine, var. Chenin blanc, using ergosterol biosynthesis inhibiting fungicides (Yolo County, 1986)

Treatment[a]	Formulation	Rate	Per cent incidence[b]	Average percent severity[c]
Control	—	—	83.8 A	48.3 A
Spotless	25W	60 g/ha	0.8 B	0.0 B
Spotless	25W	30 g/ha	0.4 B	0.0 B
Procure	50W	300 g/ha	0.4 B	0.0 B
Nustar	20 DF	188 g/ha	0.4 B	0.0 B
Bayleton	50W	300 g/ha	0.0 B	0.0 B
Rally	40W	225 g/ha	0.0 B	0.0 B
Rally	60 DF	150 g/ha	0.0 B	0.0 B

[a] Treatments were made using a 21 day application interval. First application made at 10 cm shoot growth.
[b] Figures represent average per cent disease incidence from 50 clusters in each of four replications.
[c] Figures represent average portion of each grape cluster infected with powdery mildew. Four replications, 50 clusters/replication.

were sprayed on a 21 day spray schedule and all treatments resulted in excellent mildew control including Bayleton.

There could be several reasons why Bayleton, which appeared to be in trouble in 1986, performed effectively in 1987. Disease onset in the non-sprayed control vines was delayed until 5 weeks after spray initiation, disease increase was slowed as a result of high temperatures, and isolates obtained from this vineyard have shown a high sensitivity to Bayleton.

Powdery mildew control strategies in California must take into account the conditions favouring or responsible for initial infection and the effects of environmental parameters on disease increase. These areas are currently being investigated in relation to isolate sensitivity to Bayleton.

9. BLACK ROT

The impact of azoles on black rot of grape caused by *Guignardia bidwellii* (Ellis) Viala & Ravaz has been one of allowing more flexibility in control programmes

Black rot is one of the most economically destructive grape diseases in the midwestern and northeastern US [4]. The disease is favoured by warm, humid weather and frequent rainfall is necessary for disease build-up and spread, thus eliminating its occurrence in California. The introduction of Bayleton has allowed for its use as a postinfection control of black rot for up to 96 h after the initiation of the infection period [4].

Prior to the introduction of triadimefon for use against black rot, carbamates or captan were commonly used in protectant spray programmes. Spray initiation began at 2.5 cm shoot growth and continued at 10 and 20 cm shoot growth prebloom then 2 postbloom sprays; the first at 7–10 days following the prebloom spray, the second 10–14 days after the first postbloom spray. Recommendations called for additional protective sprays as needed in July and August [22].

In 1977, Spotts [18] identified the environmental parameters necessary for grape leaf infection by *G. bidwellii*. These findings enabled researchers to predict infection periods. Because triadimefon has curative action against the black rot pathogen if used within 96 h after an infection period, spray control programme can be delayed until an infection period has occurred [4]. Ellis *et al.* [4] found that, in both 1983 and 1984, four fewer applications were made to obtain an equal level of disease control using triadimefon in a curative programme compared with ferbam used in a protective programme.

10. DISCUSSION

SBI compounds were introduced in the early 1970s and additional analogues continue to be introduced for field testing on control of stone fruit and grape diseases. Under California's climatic conditions where disease pressure is less, fewer treatments are required during blossoming and again at preharvest to control effectively the brown rot pathogens with SBI compounds. Climatic conditions are more favourable for powdery mildews and repeated applications of SBI compounds are required for disease control. Some benefits have been shown for control of the shot hole disease but not comparable with those currently being found because the SBI compounds appear to have less residual activity. For brown rot disease control the SBIs, in general, require more applications than the benzimidazoles and behave similarly to our contact fungicides such as captan at lower dosages, but at higher dosages the SBIs are very effective. The eradicant action of SBI compounds has made them the preferred material for powdery mildews. SBI formulations when used at higher dosages cost more than the dicarboximide fungicides at this time. This price disadvantage prohibits their development and use on stone fruit crops and product development is being pursued on crops requiring lower dosage. In addition to cost, the chemical industry has been somewhat reluctant to recommend higher dosages for fear of plant growth regulator effects and in some instances phytotoxicity.

California's stone fruit and nut industry has greatly benefited from the registration of triforine because the fungicide was used to control benomyl-resistant brown rot organisms (*M. fructicola* and *M. laxa*). For blossom blight control, the dicarboximides (iprodione and vinclozolin) have entered the marketplace which was exclusive to triforine for a few years. The success of SBIs in this market will depend on cost and control of other diseases such as powdery mildews, shot hole (*Stigmina*) and gray

mould (*Botrytis*). Other SBI compounds are being considered on grapes to counter the reduced effectiveness of triadimefon. This alternative may be feasible if the mechanisms of action between groups of SBI compounds are significantly different. SBIs or other compounds need to be registered as future alternatives with the possible development of dicarboximide-resistant *Monilinia*. For the control of preharvest fruit rot, the SBI compounds including triforine have performed as well as or better than the dicarboximides. Again with the possible development of dicarboximide resistance in pathogens and the fact that benzimidazole-resistant isolates continue to survive competitively in the fields not sprayed with benzimidazoles, the SBI compounds certainly would be important in brown rot disease management programmes. For post-harvest decay control, the SBI compound triforine is very effective in control of brown rot and is currently registered for use in treatment of fresh market peaches, plums and nectarines. Triadimefon is very effective on fruit rot and benefits could be even greater than those of the current post-harvest treatment. The dicarboximides are good candidates for post-harvest use because of their activity against *Botrytis* and *Rhizopus* which are major post-harvest decay pathogens.

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