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Project Title: Update on Low Temperature Fumigation

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Project 89-S9 - Tree and Crop Research

Project Leader: Mr. Preston L. Hartsell
USDA, Horticultural Crops Research Laboratory
2021 South Peach Avenue
Fresno, CA 93727
(209) 453-3090

Project Consultant: Dr. William L. Stanley
8545 Carmel Valley Road
Carmel, CA 93923
(408) 625-3063

Project Personnel: Jane C. Tebbets, John K. Ostrom

Objectives: (1) Determine minimum lethal fumigation conditions of phosphine for navel orangeworm larvae, pupae and eggs. (2) Determine depletion of organic bromide from shelled and inshell almonds treated with methyl bromide at different temperatures and rates under varying aeration conditions in three container types and evaluate effectiveness of methyl bromide fumigation on eggs of navel orangeworm (NOW) Amyelois transitella (Walker) and Indianmeal moth (IMM) Plodia interpunctella (Hübner) when shelled almonds in cartons with liners are fumigated at different temperatures and rates.

Interpretive Summary:

1. Phosphine/Navel Orangeworm Fumigation Study

Fumigation tests were conducted for control of NOW eggs, the most resistant stage, in inshell almonds at 60 70, 80 or 90°F using a constant dosage of 30g/1000 ft^3 phosphine gas generated from aluminum phosphide pellets. Phosphine fumigation at 60°F for 8 days resulted in a mean egg mortality of 99.8% with an ending mean gas concentration of 33 ppm. Treatment at 70°F for 4 days resulted in 100% mortality with an end gas concentration of 62 ppm. Fumigation at 80°F for 3 days or 90°F for 2 days resulted in 100% mortality of NOW eggs, with an ending gas concentration of 48 ppm in each case. In our tests we have found that the length of the exposure period in relation to treatment temperature are more critical elements than dosage rate in obtaining complete kill with phosphine gas. However, it is essential that the phosphine gas is held by the fumigation structure for the length of the exposure period. Therefore, as a guideline for efficacious phosphine treatment of NOW in almonds, we recommend that at the end of the respective exposure times for each treatment temperature, the phosphine concentration be no less than 50 ppm in any commercial fumigation.

Fumigation temp. °F	Minimum exposure	Recommended ending phosphine concentration for efficacy
60-69	8-9 days	50 ppm
70-79	4 days	50 ppm
80-90	3 days	50 ppm
90+	2 days	50 ppm

Experimental Procedure for Objective 1:

Only the NOW egg stage was tested since earlier studies have shown this stage is the most resistant to phosphine (Table 1). A minimum of 400 eggs were used per fumigation replication. Screen vials containing the eggs were placed amongst the almonds which were contained within the fumigation chambers. The almond load was approximately 53% within the 28L fiberglass chamber. Air circulation was used throughout the exposure and aertion periods. Temperatures were controlled at $\pm$ 2.0°F.

To determine the length of exposure time required for 100% mortality at each temperature, several individual fumigations were started at the same time. This procedure allowed mortality to be determined every 24 hours. The exposure period at each temperature that provided the highest mortality was then replicated 3 times (Table 2). The phosphine concentrations that were obtained from these replications are shown in Table 3.

As this study progressed through several approaches to reach our goal, the data continued to indicate that the insect response (mortality) to phosphine was more dependent on exposure time than on concentration. For instance, in some of preliminary tests the ending phosphine concentration were very low, 6.3 ppm after 3 days at 60°F. 1.5 ppm after 3 days at 80°F, and 6.4 ppm at the end of 2 days at 90°F. Yet in each test we obtained 100% mortality of NOW eggs. The Australians, especially R. G. Winks, have shown that phosphine takes longer to kill at high concentrations than at low concentrations. This is because high concentrations can lead to insects becoming narcotized. Dr. Winks further states that insects detoxify significant amounts of phosphine, therefore longer exposure periods are requred to absorb a lethal dose. In summation, dosage is increased by increasing time, i.e., increase exposure time = decrease the end point, increase concentration = increase the end point (Winks, R. G., 1986. The Significance of Response Times in the Detection and Measurement of Fumigant Resistance in Insects with Special Reference to Phosphine. Pestic. Sci. 17: 165-174).

The table in the abstract which concludes our findings and recommendations shows ending exposure time concentrations of 50 ppm. It should be noted that these final tests were conducted under ideal conditions and therefore represent ideal concentrations. Actual commercial fumigation concentration readings will probably vary somewhat from this.

The ending concentrations are intended as guidelines when only one concentration reading is taken during exposure. It is more advisable to make concentration readings throughout the exposure period (at least every 24 hours) to be assured of actual phosphine generation (from source) and containment. Figure 1 is an example of concentration over time from a dosage of 30 g/m^3 (pellets) on inshell almonds. Maximum concentrations obtained commercially will probably not be this high.

In conclusion, the dosage schedule listed on the labels of the various sources of phosphine are adequate for NOW control. Exposure time as it relates to fumigation (commodity) temperature is the most critical variable in obtaining adequate control of NOW. Fumigation structures must be adequately sealed to contain phosphine as it generates from the source material and concentration readings should be made to ascertain phosphine is being held by the fumigation structure.

2. Depletion of Organic Bromide Residues from Almonds (Data tables at end)

Inshell and shelled Nonpareil almonds were fumigated and stored at three different temperatures with methyl bromide using both new and old almond fumigation schedules. Residues were compared for the length of time required to reach minimum detectability. Bin, carton, and bulk fumigation, each with different sorptive properties, were tested.

The lowest temperature schedule treatments yielded the slowest desorption rates. The highest temperature schedule treatments yielded the fastest desorption rates. For example, shelled almonds fumigated at $50^{\circ}F$ in bins using the new schedule, required approximately 28 days to reach the reliable minimum detectability level of 0.005 ppm organic bromide, whereas approximately only 5 days were required to reach the same level when fumigated at $80^{\circ}F$.

Treatments in bins had slower desorption rates than treatments in cartons which, in turn, had slower desorption rates than treatments in bulk when the new fumigation schedules were used. For example, shelled almonds fumigated at $50^{\circ}F$ in bins required approximately 28 days to reach the 0.005 ppm level, whereas treatments in cartons required approximately 25 days and treatments in bulk required approximately 20 days.

Treatments conducted using the old fumigation schedule exhibited a reversal of the aformentioned pattern. Here, treatments in bins required the least amount of time to reach 0.005 ppm followed by treatments in bulk. Treatments in cartons required the most time for the residues to reach the 0.005 ppm level. For example, shelled almonds fumigated at $50^{\circ}F$ in cartons required approximately 70 days to reach 0.005 ppm, whereas treatments in bulk required approximately 63 days and treatments in bins required approximately 50 days.

This study also evaluated the effectiveness of methyl bromide fumigation on the eggs of NOW and IMM when shelled almonds in cartons with commercial 25-pound liners were fumigated with both new and old schedules. This liner was determined to be a low density polyethylene film with a thickness of 1.6 mil. Insect mortality data indicated that the old fumigation schedule (56 g/m^3 of fumigant at $70^{\circ}F$) afforded no advantage

Table 1. Susceptibility of NOW eggs, larvae or pupae to phosphine fumigation when treated naked for 24 h at 70°F.

PH_3 dosage (ppm)	NOW % mortality		
	Eggs	Larvae	Pupae
30	45.4	68.7	49.3
60	48.5	70.7	70.0
120	49.6	100.0	88.0
250	51.1	100.0	98.7
500	44.4	100.0	100.0

Table 2. Mortality results of NOW eggs, 0-24 h old, treated in an inshell almond load (53%) with 30g/1000 ft^3 PH_3 gas from aluminum phosphide pellets at 60, 70, 80 or 90°F for 8, 4, 3 or 2 days, respectively.

	NOW % mortality[1]			
Exposure temp. °F	Total no. of eggs treated	No. of eggs hatched	No. of eggs not hatched	Percent mortality[2]
60	1293	2	1291	99.8
70	1317	0	1317	100.0
80	1390	0	1390	100.0
90	1292	0	1292	100.0

[1]Data represent total numbers from 3 replicated fumigations.

[2]Percent mortality for treated eggs is corrected for natural mortality in untreated eggs.

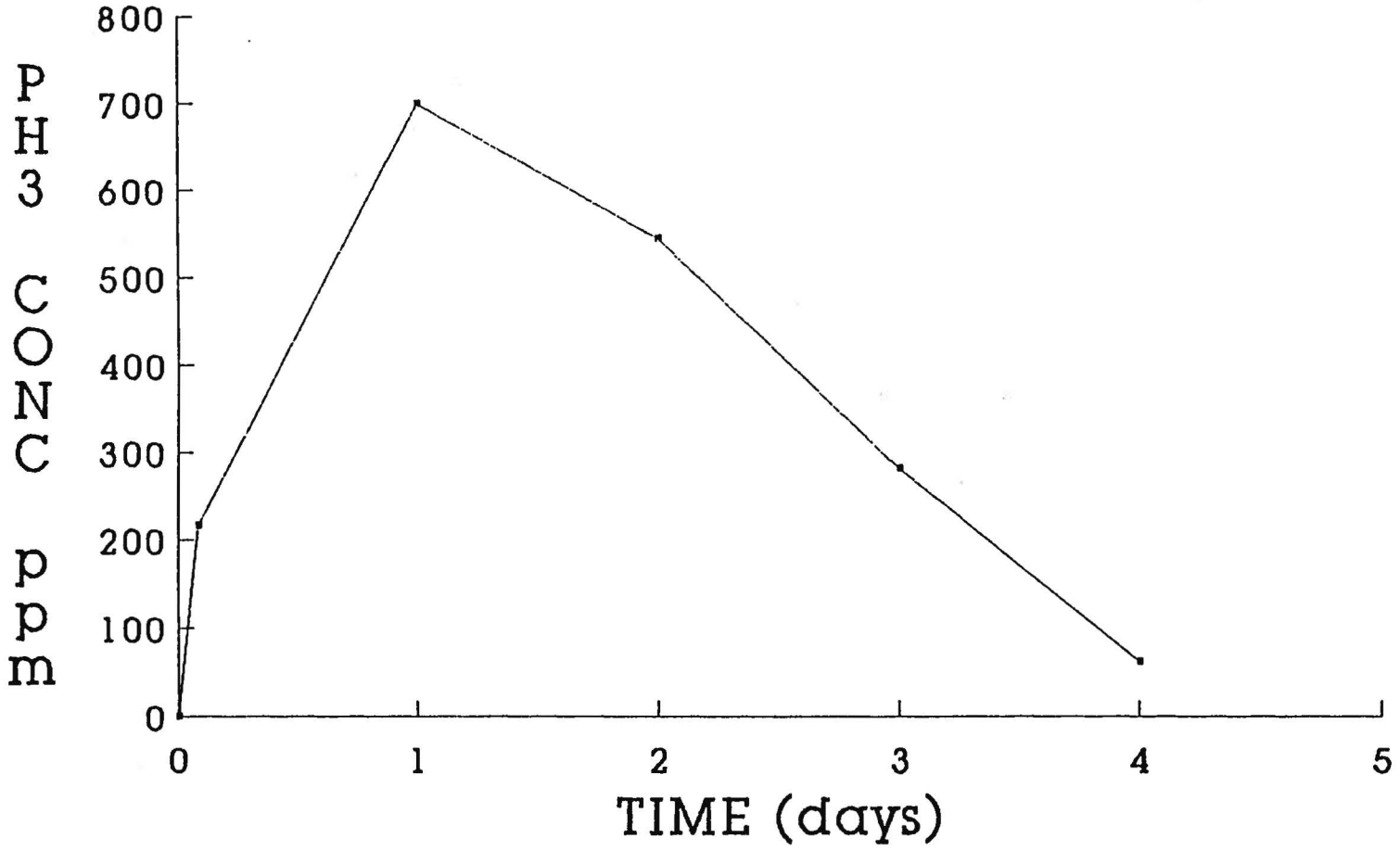
Table 3. Phosphine gas concentrations during fumigations of NOW eggs, 0-24 h old, treated in an inshell almond load (53%) with 30g/1000 ft^3 PH_3 gas from aluminum phosphide pellets at 60, 70, 80 or 90°F for 8, 4, 3 or 2 days, respectively.

	PH_3 concentration (ppm)[1]								
	Gas sampling times								
Exposure temp. °F	2 h	24 h	48 h	72 h	96 h	5 days	6 days	7 days	8 days
60	363.8 ± 108.3	716.9 ± 132.3	478.7 ± 42.9	294.1 ± 47.4	168.8 ± 38.2	101.2 ± 30.8	65.0 ± 21.6	46.0 ± 14.6	32.6 ± 11.1
70	215.9 ± 70.1	700.3 ± 138.6	545.2 ± 145.0	282.1 ± 78.7	62.2 ± 33.7	-	-	-	-
80	362.2 ± 22.5	318.5 ± 32.8	115.3 ± 3.8	47.5 ± 11.9	-	-	-	-	-
90	138.5 ± 16.9	424.0 ± 93.7	48.4 ± 11.9	-	-	-	-	-	

[1]Data represent mean and standard deviation from 3 replicated fumigations.

Figure 1

TYPICAL PH3 CONCENTRATION CURVE FROM AlP FUMIGATION OF ALMONDS



over the new schedule (16 g/m^3 of fumigant for 24 h at 70°F). Both dosage schedules resulted in 100% mortality in the NOW eggs and 99.9% in the IMM eggs. The old schedule, however, resulted in higher residues and a slower fumigant desorption rate than the new schedule. The old schedule treatments required approximately 40 days to reach the 0.005 ppm level, whereas the new schedule treatments required approximately 30 days.

Experimental Procedure for Objective 2:

This research was conducted from September 1987 to October 1988 at the United States Department of Agriculture, Agricultural Research Service, Horticultural Crops Research Laboratory in Fresno, California. The study was conducted by John Ostrom and was used to fulfill the requirements of a thesis project for the Master's Degree in Agriculture at California State University, Fresno.

The commodity to be fumigated in this study consisted of inshell and shelled almonds, Prunus dulcis (Mill.) D. A. Webb, c.v. paper-shell 'Nonpareil'. The nuts came from three separate lots sent from the Almond Board of California in Sacramento at three separate times throughout the course of this study. The first lot was received in August, 1987; the second in Febraury, 1988; and the third in July, 1988. Upon arrival the nuts were all stored under the same conditions of 50°F and 40 to 60% relative humidity.

The insects to be fumigated in this study consisted of the egg stage of NOW and IMM. The eggs were chosen because they are the most resistant stage and hardest to kill. These insects were reared and their eggs collected at the USDA, ARS, Horticultural Crops Research Laboratory.

Experimental Design

This study consisted of two separate experiments (A and B). Experiment A was designed to compare organic bromide residue levels, desorption rates, and half lives for inshell and shelled almonds fumigated with methyl bromide using the new and old schedules at different temperatures, each with its corresponding scheduled dosage. The schedules used in Experiment A consisted of (a) the revised almond fumigation schedules developed by Hartsell et al.[1] which utilizes a fumigant dosage of 16 g/m^3 applied for varying exposure periods dependent on fumigation temperature and (b) the old fumigation schedule which utilizes a fumigant dosage of 56 g/m^3 applied for 24 hours (E.P.A. Reg. No. 8536-15-aa). Residue levels were measured for the length of time required to reach minimum reliable detectability of organic bromide (approximately 0.005 ppm). Three different containers, each with different sorptive qualities, were tested with shelled almonds. Inshell almonds were tested in only two container types. Experiment A consisted of 26 fumigation treatments with three replications per treatment, set up in such a manner that they were analyzed as a series of randomized complete block designs. Tables 1 and 2 show the treatment schedules and desorption parameters for the almonds in Experiment A. A control replication, which received no fumigation, but which was stored under conditions identical to the treated replications, was also included in each treatment in order to assure that all residues recorded were due to the fumigation treatment.

Experiment B was designed to evaluate the effectivness of methyl bromide fumigation on NOW and IMM eggs when they were treated along with shelled almonds in closed liners within cartons using both new and old schedules. Organic bromide residues on the shelled almonds from these insect tests were determined and then correlated with insect mortality. Residue desorption rates and half-lives were compared as well. Experiment B consisted of two fumigation treatments, with three replications per treatment, set up in a randomized complete block design. Table 3 shows treatment schedules and desorption parameters for almonds fumigated with insect eggs in Experiment B. A control replication, identical to that run in Experiment A, was also included for the same reason.

The same nuts were used repeatedly in this study (Experiment A and Experiment B). However, treated nuts were not reused until their organic methyl bromide content was below detectable limits.

Container Description

Experiment A utilized three different container types; open wire baskets which simulated a bulk treatment, wooden bins which simulated a commercial bin treatment, and cardboard cartons without liners or packing material. The open steel wire baskets measured 27.9 cm by 27.9 cm by 22.9 cm in order to fit into the fumigation chamber and to be easily manipulated in and out of the chamber. These open wire baskets were considered to simulate bulk treatments since at the concentrations used in fumigation practice, methyl bromide has no effect on most metals, and the baskets displaced practically no volume in the chambers.

The wooden bins used were made from 0.95 cm (3/8 inch) thick C-DX grade plywood and held together with small galvinized nails. The bin dimensions were 27.9 cm by 27.9 cm by 22.9 cm. These bins were open at the top and had three slots, 22.9 cm long, 0.3 cm wide and 6.4 cm apart, cut into each of the four sides. The slots allowed these bins to better simulate a commercial bin. The bottoms of the bins were left intact.

The cardboard cartons (Tharco) were again 27.9 cm and 27.9 cm by 22.9 cm. These cartons were used because they were found to have intermediate percentage sorption levels of methyl bromide compared to two commercial almond cartons (Monte Cristo and Blue Diamond). The cartons were closed at the top and bottom with masking tape. Tape was placed along the entire junction of the top and bottom flaps and tape was used instead of glue because the cartons had to be periodically opened for sampling.

Experiment B utilized the carboard cartons and the almonds were enclosed in a commercial 25-pound carton liner. This liner was determined to be a low density polyethylene film with no additives by running a sample on a Fourier Transform Infrared Spectrometer (Mattson, model Polaris). The unknown sample determination was made by cross reference of it percentage transmission spectra with an infrared spectra atlas. The thickness of this liner was found to be 1.575 mil $\pm$ 0.070 by mechanical means using a thickness measuring device (Ames, model 252).

Fumigation Procedure

In each fumigation treatment for Experiment A and Experiment B, the almonds were tempered for 12-18 hours at the appropriate temperature and in the appropriate container corresponding to each treatment in the schedule.

All fumigations were conducted in modified 29.3L fiberglass chambers. The chambers were equipped with air circulation fans which operated continuously during both the exposure period and the chamber aeration period. The fans distributed the fumigant evenly throughout the chamber atmosphere. Each chamber was equipped with a side port valve for introduction and withdrawal of methyl bromide. The chambers were all checked for performance before use in accordance with USDA, APHIS specifications.

The six chambers used in this study were situated in a temperature controlled walk-in box. This box was equipped with an outside air exchange fan for safety to the operator. The fan was allowed to operate whenever the walk-in box was entered by the operator and during the chamber aeration period to better simulate a commercial aeration. Due to the use of this fan and to the limits of the temperature control apparatus, fumigation temperatures were accurate within two degrees of the prescribed setting. Relative humidity ranged from 40 to 60% for all treatments. Temperature and relative humidity were monitored on a recording hygrothermograph. The walk-in box also contained connections to a vacuum pump system which was used to remove the fumigant from the chambers.

The estimated load factor for all treatments based on volume was 50 to 60%. On a weight basis, 21 lbs. of shelled almonds and 14-15 lbs. of inshell almonds were loaded per container. These weights represent the maximum amount of almonds which would fit in each type of container and still allow easy manipulation in and out of the chamber.

All fumigations were conducted at normal atmospheric pressure. The calculated quantity of pure methyl bromide gas for each dosage at its corresponding temperature was drawn from a small gas cylinder (Matheson lecture bottle) and then injected into the chamber. Procedures used for manipulating gaseous methyl bromide as well as a detailed discussion of gas cylinder apparatus are described by Hartsell et al.[2] and Tebbets et al.[5] Concentrations of methyl bromide within the chambers during fumigation were monitored by gas chromatography.

Test Insects

NOW and IMM eggs treated in Experiment B were placed in plastic vials with screens at both ends. The screened holes allowed direct passage of the fumigant into the vials.

The age of the NOW eggs was 0-1 day. They were laid on filter paper and the paper was cut into strips. The eggs were counted under a dissecting microscope at 1.3x power. Each vial contained two to three filter paper strips such that the total number of eggs in each vial equaled approximately 200. The vials were half filled with NOW larval diet media. This allowed the maintenance of an optimal humidity level needed for

maximal egg hatch as well as deterring larvae from early hatched eggs from cannibalizing unhatched ones by providing an alternate food source.

The IMM eggs were also 0-1 day of age. They were laid loosely and placed in a petri dish. These eggs are extremely small, but were counted under a dissecting microscope at 1.3x power. Approximately 350 IMM eggs were placed, with the use of a small brush, on a small filter paper boat with the sides folded so as to keep them from spilling. One filter paper boat was then placed in each vial which also containted larval diet media.

One vial each of the NOW and IMM eggs was placed upright in the middle of the shelled almonds which were enclosed in a liner within a carton. Liners were closed with a single piece of masking tape approximately 8 cm in length. Cartons were subsequently closed with tape and then fumigated at the two schedules specified in Table 3. Control eggs in vials were all placed together in a carton of untreated almonds which was placed at 70°F in a separate location so as to not be contaminated by methyl bromide during the aeration process. Insect eggs were not tempered along with the almonds because the protocol called for eggs of 0-1 day old (most resistant). The eggs were added to the shelled almonds just prior to fumigation.

Aeration and Storage Procedures

After fumigation, chambers were evacuated by use of a vacuum pump. Air was then allowed back in to satisfy the vacuum at which time the chamber doors were opened. The almonds were left in the chamber with each fan and the walk-in box air exchange fan running for the duration of the chamber aeration. Chamber aeration was for the same length of time as the exposure period. After such time, the containers of almonds were removed from the chambers and placed on the floor of the walk-in box. The chamber fans and walk-in box air exchange fan were then turned off. Treatment samples were stored here at the same temperature at which they were fumigated with the exception of the 70°F treatments in Experiment A. These treatments were stored outdoors in the shade under ambient conditions. Ambient temperature for the duration of almond storage in each treatment was calculated by averaging daily high and low temperatures and then taking their mean over the entire storage period. Ambient relative humidity varied widely depending on climatic conditions. These treatments were stored in an area where wind velocity was kept at a minimum. To assure uniformity in results, the test protocol called for all ambient storage tests to be run during the same period when average daily (24 hour) temperature was approximately 70°F. This was not accomplished due to large climatic changes and will be reported on in the results section of this report.

Conversely, treatments in Experiment B were stored at a constant 70°F temperature in the walk-in box. Vials containing the insects were removed upon completion of the chamber aeration period. They were immediately opened and the filter paper containing both NOW and IMM eggs removed. Treated and control eggs on filter paper were placed on larval diet media enclosed in petri dishes with lids. These dishes were placed in an 80°F incubation room at 60% relative humidity. Eggs were allowed to hatch for 5 days before mortality rates were determined.

Residue Procedure

Random almond samples, each of at least 50 g, were taken from the center of each container for the purpose of determining organic bromide residues. Samples were taken as close to the following schedule as possible; at the end of chamber aeration, after 12 hours aeration, and 1, 2, 5, 7, 9, 13 days and approximately every 5 days thereafter. However, when organic bromide residues for each treatment averaged approximately 0.005 ppm, analysis was discontinued. Samples were placed in the freezer when immediate analysis was not possible. Analysis was completed on frozen samples within 2 weeks.

Organic bromide residue analysis was conducted using modifications of the procedure developed by King et al.[3]. This procedure utilizes a rapid headspace assay which is quantified by comparison to a standard of known concentration using a gas chromatograph equipped with an electron capture detector.

Extremes in residue levels, the initial sampling time residues and the final sampling time residues before the levels dropped below the reliable detectability limit of 0.005 ppm, with standard deviations, are reported in tabular form for Experiments A and B (Tables 4, 5, 6, 7 and 8). The first reading below the limit is reported as well. Linear regression was used to determine the rates of desorption and half lives of the organic bromide residues from fumigated almond treatments. Desorption rates were determined from the slopes of the regression lines. Half lives were determined by using the regression lines to calculate the period of time it took for the residue levels to drop in half. Both data types are reported in tabular form (Tables 9, 10 and 11). In order to make the data linear, a log transformation was performed on the residue values. The data was fitted to the equation $\log C = mt + b$, where C is the organic bromide residue in ppm, m is the slope, t is the time in days, and b is the intercept. Actual linear regressions, with their corresponding coefficients of determination (r^2) and equations, along with all the raw data, are depicted in graphical form, comparing treatments in Experiment A by temperature and container type (Figures 1-16). Linear regressions for Experiment B are also depicted in the same manner (Figure 17). The x-axis denotes the time in days and the y-axis denotes the log of the residue values. In a few cases, residue means of just slightly lower than 0.005 ppm are included to give a more accurate regression line. An explanation of this follows in the results section of this report. These figures allow a visual comparison of the treatments involved in this study. However, no regression comparison by container type was made for either ambient aerated inshell or shelled treatments since the temperature was not constant between them.

Insect Mortality

Mortality rates were determined 5 days after treatment. Mortality was based on maturation to the next stage of development (i.e., eggs to larvae). Mortality rates were determined as a percentage mortality. Abbott's formula was used to correct percentage mortality values in order to compensate for observed mortality in untreated controls. These data are presented in tabular form for NOW and IMM as the mean of percentage

mortality with standard deviation for each treatment (Tables 12 and 13). Analysis of variance was performed on the data and means were separated using Duncan's multiple range test at the 0.05 level.

Results of Objective 2:

Residues

The reliable detectability limit of organic bromide was found to be approximately 0.005 ppm. Although the gas chromatograph could easily determine the residue levels at the 0.001 ppm level, in most cases, background from extracted almond samples precluded any work below 0.005 ppm. At levels just below 0.005 ppm, a characteristic plateau was observed in the residues from most samples.

Background irregularities caused the reliable limit to be established at approximately 0.005 ppm. In a few instances, residue means slightly lower than 0.005 ppm were included to give a more accurate regression line. This was done only in the cases where the characteristic background was at a lower level and a clearly dicernible resolved peak was obtained from the GC, and where the previous sampling residue was well above 0.005 ppm.

The figures are comparisions of organic bromide residue desorption by use of linear regression. Actual residue data along with regression lines, equations, coefficients of determination (r^2), and storage temperatures or container types are shown in each figure. The x-axis depicts time in days and the y-axis depicts the log of the organic bromide residue. Thus, a value of zero on the y-axis denotes a residue of 1 ppm organic bromide and a value of -2.30 denotes 0.005 ppm. Therefore, all regression lines end at -2.30.

Figures 1, 2, and 3 represent comparisons of the residue data on the basis of fumigation and storage temperture for shelled almonds fumigated in each of the three container types with methyl bromide at a dosage of 16 g/m^3 (new schedules). All three figures indicate that the highest fumigation and storage temperature treatments (80°F) resulted in the shortest time required to reach the minimal detectability limit of 0.005 ppm for a given container type. These figures also indicate that the lowest fumigation and storage temperature treatments (50°F) resulted in the longest time required to reach the 0.005 ppm limit. In each case, the treatments fumigated and stored at 50°F had higher average initial residues than the 80°F treatments (Table 4).

The regression line for ambient storage treatments in bulk (Figure 1) is practically identical to that of the 80°F treatments indicating that approximately the same amount of time was required to reach to the 0.005 ppm level. Both lines have high coefficients of determination. Ambient storage treatments in bins (Figure 2) demonstrate that an intermediate amount of time, somewhere between the 50°F and 80°F storage treatments, was required to reach 0.005 ppm. This regression line is distinctly separate from the other two lines. Ambient storage treatments in cartons (Figure 3) demonstrate only a slight increase of a few days from the 80°F treatments in the time require to reach 0.005 ppm. These two regression lines are similar, but the ambient storage line has a low coefficient of determination (81.1%).

The ambient storage temperature data are variable because these tests were not all run at the same time or when the avereage (24 h) temperature was 70°F as specified in the protocol. To follow the protocol exactly was physically impossible due to the volume of treatments involved and to rapid changes in climatic conditions. These data are reported, however, so that a common industry practice can be examined.

Standard deviations reported in parentheses following each of these ambient storage temperatures do not adequately reflect the range of temperatures to which almonds were exposed. Reported deviations only indicate the range in means of daily high and low temperatures used to calculate mean ambient storage temperature over the entire storage duration. These deviations do not directly take into account the actual extremes in temperatures. This must be considered when comparing ambient storage treatments to treatments at a constant storage temperature (50°F and 80°F). Comparisons cannot be made at all between ambient storage treatments in different container types since they were not all run at the same time under identical conditions.

Figures 4 and 5 represent comparisons of the residue data on the basis of container type for shelled almonds fumigated with methyl bromide at a dosage of 16 g/m^3 (new schedule) at 50°F and 80°F and stored at these same two temperatures. Both of these figures show three widely separated regression lines, one for each container type. However, these two figures indicate very different residue desorption phenomena.

Figure 4 clearly indicates that bulk treatments stored at 50°F required the fewest days to reach the 0.005 ppm residue level. Bin treatments required the most days to reach minimal limit, whereas carton treatments required a number of days between that of the other two treatments. Carton and bulk treatments had very similar average initial residues, whereas bin treatments had average initial residues of less than half those of the other two treatments. Even though bin treatments started with residue levels far less than carton and bulk treatments, they had the slowest desorption rates. Carton treatments provided the next slowest desorption rate followed by bulk treatments. Bulk treatments did not provide any material that could act as a barrier to the off-gasing fumigant and subsequently allowed the fastest desorption.

Figure 5 clearly demonstrates a totally different desorption phenomenon from that in Figure 4. These treatments were all fumigated and stored at 80°F. Bin treatments required the fewest number of days necessary to reach the 0.005 ppm level. Bulk treatments required the next fewest numbers of days, whereas carton treatments required the most number of days to reach the detectability limit. These treatments were all fumigated and stored at 80°F. Again, bin treatments, due to the sorptive quality of the wood, had the lowest average initial residue (Table 4). Carton and bulk treatments had similar average initial residue levels, with cartons slightly higher (Table 4). Both were approximately three times higher than the levels in the bin treatments. Thus, at 80°F fumigation and storage, it seems that the amount of initial residue was the determining factor in the time required for residues to reach the minial level. Conversely, at 50°F fumigation and storage, the amount of initial residue was secondary to the barrier ability of the container type as the factor

in determining the time required to reach the minimal level. Both factors are dependent on container type. The difference is that the container type determined the time needed to reach the 0.005 ppm limit in the 50°F fumigation and storge by its ability to let the fumigant off the commodity, but in the 80°F fumigation and storage by its ability to let the fumigant into the commodity.

Table 4 reports extremes in organic bromide residues from new schedule shelled almond treatments in Experiment A. These are the same treatments that are graphically depicted in Figures 1, 2, 3, 4 and 5. Whereas the figures give a good visual representation of the data along with the regression lines, the table gives a numerical representation. This is useful when looking for the actual range in days that it took for a certain treatment to reach the minimal residue limit and for determining initial average residues.

Figures 6 and 7 represent comparisons of the residue data on the basis of fumigation and storage temperature for inshell almonds fumigated in baskets and bins with methyl bromide at a dosage of 24 g/m^3 (new schedules). Both figures indicate that treatments with the highest fumigation and storage temperature (80°F) resulted in the shortest time required to reach the 0.005 ppm limit. This is similar to the data in Figures 1, 2 and 3. However, bulk treatments with the lowest fumigation and storage temperature (50°F) in Figure 6 did not yield the longest time required to reach the minimal limit as expected. Here we see that ambient storage treatments took a few days longer to reach the 0.005 ppm level than the 50°F fumigation and storage treatments. This variation may be due to the fact that the huge variability in ambient temperature somehow affected desorption of the fumigant.

Figure 7 shows that treatments with the lowest fumigation and storage temperature (50°F) resulted in the longest time required to reach the 0.005 ppm level. The highest fumigation and storage temperature treatments (80°F) required the least time to reach the minimal detectability limit. Ambient storage treatments required an amount of time between the 50°F and 80°F treatments.

Figures 8 and 9 represent comparisons of the residue data on the basis of container type for inshell almonds fumigated with methyl bromide at a dosage of 24 g/m^3 (new schedules) at 50°F amd 80°F and stored at these same two temperatures.

Figure 8 shows the two treatments at 50°F fumigation and storage to be very similar. Both lines have fairly high coefficients of determination. Bin treatments started at a lower average initial residue level than bulk treatments (Table 5) and again resulted in a slightly longer requirement of time to reach the minimum detectability limit. However, this was not nearly as convincing as in the case of shelled almonds under the same circumstances (Figure 4). The two regression lines in Figure 8 do not appear to be significantly different. Thus, a statement cannot be made as to the effect of container type under these conditions.

Figure 9 indicates quite a substantial difference in desorption phenomena of bulk and bin treatments at 80°F fumigation and storage.

Unlike the data in Figure 5, bin treatments started with a higher average initial residue (Table 5) which seems odd in itself taking into account the sorptive quality of wooden bins. This higher initial residue is not reflected in the regression line, due to its somewhat low coefficient of determination (85.0%), and is most likely due to sampling variation. Thus, it appears here that bin treatments required a longer time to reach the 0.005 ppm limit because they started with higher residues and the desorbing fumigant had to contend with the barrier presented by the wooden bins.

Table 5 reports extremes in organic bromide residues from new schedule inshell almond treatments in Experiment A. These are the same treatments that are graphically depicted in Figures 6, 7, 8, and 9.

Figures 10, 11 and 12 represent comparisons of organic bromide residues on the basis of fumigation and storage temperatures for shelled almonds fumigated in baskets, bins and cartons with methyl bromide at a dosage of 56 g/m^3 for 24 h (old schedule). All three figures demonstrate that the highest fumigation and storage temperature treatments (80°F or 70° fumigation with ambient storage) resulted in the shortest time required to reach the 0.005 ppm level for a given container type. The lowest fumigation and storage temperature treatments (50°F) resulted in the longest time required to reach the 0.005 ppm level. This pattern is similar to the new schedule fumigation treatments except for the fact that the old schedule treatments required many more days in general to reach the detectability limit.

Figure 13 represents a comparison of organic bromide residues on the basis of container type for shelled almonds fumigated in baskets, bins and cartons with methyl bromide at a dosage of 56 g/m^3 for 24 h (old schedule) at 50°F. This figure indicates that the least amount of time required to reach the 0.005 ppm level occurred in treatments in bins and the greatest amount of time occurred in treatments in cartons. Treatments in baskets required an intermediate amount of time. This pattern contradicted the pattern seen for shelled almonds at 50°F using the new schedule dosages (Figure 4) where bin treatments were found to require the most time and bulk treatments the least. This very well could be a reflection in the large difference in dosage, i.e., the actual physical movement of large or small quantities of gas molecules in and out of the nuts, with or without any containment.

Table 6 depicts the data in Figures 10, 11, 12, and 13 numerically. These data again indicate a quicker depletion of organic bromide in the highest fumigation and storage temperature treatments (70°F fumigation and ambient storage or 80°F fumigation and storage in the case of the carton treatments). The 50°F treatments not only required more time to reach the 0.005 ppm level but also had the largest initial residues.

On a container type comparison basis, only the 50°F treatments can be examined since the ambient storage temperatures were not uniform. These treatments in all three container types had very similar initial residue levels. However, bin treatments reached the 0.005 ppm level well before either of the other two treatments. This is somewhat puzzling since the opposite was noted for the same treatments using the new fumigation schedules (Figure 4 and Table 4). One would expect the bins treatments to

require more time to reach 0.005 ppm than the bulk treatments since the initial residues for both treatments were very similar (Table 6). Logically the bin should provide a barrier to the off-gasing fumigant. However, the data collected are contrary to this logic. This irregularity may in fact reflect more accurately the desorption phenomena at higher fumigant dosages, but is more likely a variation encountered in any fumigation and residue procedure. Contamination of samples is also possible when working at these extremely low residue levels and may also explain some of the observed variation.

Figures 14 and 15 represent comparisons of organic bromide residues on the basis of fumigation and storage temperature for inshell almonds fumigated in baskets and bins with methyl bromide at a dosage of 56 g/m^3 for 24 h (old schedule). Figure 14 again demonstrates that for treatments in bulk, the higher fumigation and storage temperature treatments (70°F fumigation and ambient storage) required fewer days to reach 0.005 ppm organic bromide than did the lower temperature treatments (50°F fumigation and storage).

Conversely, Figure 15 demonstrates a longer time requirement for the higher fumigation and ambient storage temperature treatments in bins (70°F fumigation and ambient storage) to reach the 0.005 ppm level than for the lower treatments (50°F fumigation and storage). This is contrary to what was observed in every other case. However, note the poor coefficient of determination for the ambient storage regression line (78.9%).

Figure 16 represents a comparison of organic bromide residues on the basis of container type for inshell almonds fumigated in baskets and bins with methyl bromide at a dosage of 56 g/m^3 for 24 h (old schedule) at 50°F. This figure indicates that bin treatments required less time than bulk treatments to reach 0.005 ppm.

Table 7 depicts the data in Figures 14, 15 and 16 numerically. The data show the contradiction in residue desorption on a fumigation and storage temperature basis between bulk and bin treatments. The difference in the times required to reach 0.005 ppm are not very large and might be due to natural variation or possible contamination in the nut samples.

On a container type comparison basis, bulk and bin treatments at 50°F fumigation and storage were very similar in the time required to reach 0.005 ppm. Bin treatments required slightly fewer days.

Figure 17 represents a comparison of the residue data for shelled almonds fumigated at two dosages of methyl bromide at 70°F in cartons with liners in Experiment B. The regression line depicting the treatments using 56 g/m^3 for a 24 h period does not fit the data too well. This line has a poor coefficient of determination (80.9%). It seems as if the log transformation failed to make these data linear. The two treatments appear more similar than they really are due to the poor fit of this one regression line. Table 8 demontrates these same data numerically. It is evident from Table 8 that the the 56 g/m^3 treatments had extremely higher average initial residues and that they required a substantially longer periods of time to reach the 0.005 ppm level.

It is difficult and confusing to draw conclusions from all these residue data, since they were examined both on the basis of fumigation and storage temperature and container type. It appears that on a constant container type basis, the 50°F schedule treatments required the most time to reach the 0.005 ppm level compared to the 80°F schedule treatments. This was true for both inshell and shelled almonds using the new and old fumigation schedule dosages.

On a constant storage temperature basis for both inshell and shelled almonds fumigated with the new schedule dosages (16 g/m^3 or 24 g/m^3), bin treatments required the most time to reach the 0.005 ppm level for the 50°F schedules. Bulk treatments required the least time to reach the same level and carton treatments required an intermediate value. This pattern held for the 80°F schedules for inshell almonds. However, a reversal of this pattern occurred in the 80°F schedule for shelled almonds. Here bin treatments required the least time to reach the 0.005 ppm level and carton treatments required the most time.

This trend was reversed for almonds fumigated with the old schedule (56 g/m^3 for 24 h). Here the bin treatments required the least amount of time to reach 0.005 ppm followed by the bulk treatments. The carton treatments, when conducted, required the greatest amount of time to reach 0.005 ppm.

Tables 9 and 10 report organic bromide desorption rates and the half lives for inshell and shelled almonds in Experiment A. Table 11 reports these same values for shelled almonds fumigated in liners within cartons in Experiment B. These two descriptors were both calculated using the equations for regression lines in Figure 1 through 17. Desorption rates are simply the slopes of each regression line. They are reported as log ppm/day and are negative values, evidence of the fact that residues decreased over time. The more negative the value, the faster the desorption rate. Half lives were calculated from regression equations using an arbitrary time frame. Half life is defined as the time necessary for a residue to drop in half. Since this value was calculated from the regression line and reflects the desorption rate, the time frame used to measure this decrease had no bearing on the half life.

Table 9 indicates that both inshell and shelled almonds fumigated with the new schedules had the fastest desorption rates in treatments with the highest fumigation and storage temperature (80°F). This comparison assumes a constant container type. Likewise, both almond types had the slowest desorption rate in treatments with the lowest fumigation and storage temperature (50°F) in every case except one. This irregularity surfaced in the treatments with inshell almonds fumigated at 70°F in baskets and stored at ambient temperatures. These treatments yielded desorption rates slower than the corresponding treatments fumigated and stored at 50°F. These same ambient storage treatments presented an anomaly in Figure 6. This irregularity can most likely be attributed to extremes in temperatures (per 24 h), thus causing higher residues and a longer time requirement to reach the 0.005 ppm level responsible for determining a regression line with less slope.

Table 9 shows that half life values for the comparisons mentioned above made on the basis of fumigation and storage temperature, reflect the same pattern as the desorption rate data. Treatments at a lower fumigation and storage temperture (50°F) required more days than the higher temperature treatments (80°F) for their residue levels to drop in half. Again, variation existed in half life data for the same treatments that were previously mentioned and explained.

When a comparison of desorption rates and half lives is made on the basis of container type for inshell almond treatments, it is clear to see that, at a constant fumigation and storage temperature, 50°F or 80°F, the desorption rate is faster and the half life shorter in bulk treatments than in bin treatments. Carton treatments resulted in values between the other two treatments.

However, the 80°F fumigation and storage treatments for shelled almonds did not follow this pattern. Here we see that bin treatments had the fastest desorption rate and the shortest half life. Carton treatments had the slowest desorption rate and the longest half life. Bulk treatments showed intermediate values between the other two treatments. This variation in desorption rate and half life patterns of bin treatments is most likely due to the fact that the compartively low initial residues and the short time required to reach the 0.005 ppm level recorded for this treatment (Table 4) resulted in a regression line of greater slope. This regression line also has a fairly low coefficient of determination of 89.5% (Figure 5).

Table 10 shows basically the same pattern in desorption rates and half lives for almonds fumigated with the old schedules as for the new schedule treatments (Table 9). In most instances, treatments at the higher fumigation and storage temperature had corresponding faster desorption rates and shorter half lives. However, a few exceptions exist. The inshell bin treatment at 70°F fumigation and ambient storage had a slower desorption rate and longer half life than the same treatment at 50°F fumigation and storage. The shelled bin treatments at both temperatures also show a reversal of the pattern, but these values are very similar. Since these values were computed using the regression lines from the aforementioned figures, the possible causes for these variations have been explained in the discussion of the figures.

When a comparison of desorption rates and half lives is made on the basis of container type for inshell almonds at 50°F fumigation and storage, the data show that bin treatments had the fastest desorption rates and shortest half lives, whereas carton treatments had the slowest desorption rates and longest half lives. Bulk treatments had intermediate values between the other two treatments.

Table 11 compares desorption rates and half lives for treatments fumigated at two dosages in Experiment B. Table 11 shows that the desorption rate is faster and the half life shorter in treatments with a lower dosage of fumigant applied for a shorter time (new schedule) than treatments with a higher dosage for a longer time (old schedule). This seems logical, but a greater difference in the two treatments was expected. However, since these data were calculated using the regression lines in

Figure 17, desorption rates and half life values for 56 g/m^3 dosage treatments (old schedule) are suspect due to the poor coefficient of determination of this regression line 80.9%.

These residue values, desorption rates, and half lives are only useful as a guideline for fumigation procedures. These results are not meant to be used as an absolute predictive tool. The data are only applicable to similar conditions maintained in this study. In order to use residue data in a more accurate manner, it is necessary to establish mathematical models to which the data fit under repeatable conditions. Sell et al. were able to derive a mathematical model of the desorption of methyl bromide from cherries (4). Their model gave good estimates of residue concentrations over a wide range of load factors, pulp temperatures, and aeration periods, and was used to predict the length of time of aeration period required to reduce the residue to a proposed tolerance of 0.1 ppm. It has not been determined at this time whether or not the data reported in this report could be made to fit such a model.

Variation between almonds can also account for some of the observed variation in the residue data. The amount of damage and disease certainly affects residue levels. Almonds also vary in oil and moisture content even when stored and treated under identical conditions. In the case of inshell almonds, shell breakage and the presence of hulls affect fumigation and residue phenomena. Where containment is used, type, age (bins), moisture content, painted surface or not, even the type and condition of pallets used for transport, may effect consequential residue levels. Fumigation as reflected by subsequent residue analysis is an inexact discipline, but the sources of variation have to be dealt with as best as possible in order to allow some understanding of these procedures.

Insect Mortality

Tables 12 and 13 report mortality data for the eggs of NOW and IMM. It is evident in both tables that the two treatments were equally effective in killing the eggs under these circumstances.

The treatments using a lower dosage of fumigant for a shorter exposure period (new schedule) proved to be efficacious in control of both insects. Thus, no advantage was gained by using a higher dosage of methyl bromide for a longer exposure period (old schedule) under the conditions employed in this study. In fact, the higher dosage proved to be a disadvantage when residue data are taken into account. These treatments resulted in a slower desorption rate and longer half life of fumigant residues compared to the lower dosage treatments (Table 11). This higher dosage also resulted in a longer time to reach the 0.005 ppm residue level (Table 8). It must be noted that these comparisons are only valid for the type of commercial liner used in this study. This low density polyethylene liner obviously allowed adequate penetration of the fumigant needed to kill the insect eggs in the lower dosage (new schedule) treatments. However, no correlation can be made for the use of a different liner type.

Conclusions

This study demonstrated that the fumigation schedules and container types used in the treatment of these almonds significantly affected both fumigation and residue desorption phenomena. Almonds which were fumigated at the lowest temperature using both new and old fumigation schedules resulted in the longest requirement of time necessary for organic bromide residues to reach the minimum detectability limit of 0.005 ppm. Conversely, the highest temperature treatments using both fumigation schedules resulted in the shortest requirement of time necessay for residues to reach the 0.005 ppm level.

In most instances, treatments conducted in wooden bins using the new fumigation schedules required the greatest amount of time to reach the 0.005 ppm level. Conversely, most of these same treatments in bulk (open wire baskets) required the least amount of time to reach the 0.005 ppm level. Treatments conducted in carboard cartons yielded intermediate values between treatments in the other two container types.

Treatments conducted using the old fumigation schedules exhibited a reversal of the aforementioned pattern. Here, treatments in wooden bins required the least amount of time to reach 0.005 ppm followed by treatments in bulk. Treatments in cartons required the most time for the residues to reach the 0.005 ppm level.

Treatments in liners with the lower dosage for a shorter exposure time (new schedule) proved to be efficacious in the control of NOW and IMM eggs. Both dosage schedules resulted in an equal mortality of the eggs. The lower dosage (new schedule) proved more advantageous because of its resultant lower residues and faster desorption rate.

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Table 1

Treatment schedules and desorption parameters for 'Nonpareil' almonds fumigated using the new schedules.

Product description	Container type	Fumigation temp.(°F)	Dosage (g/m^3)	Exposure time (h)	Chamber aeration (h)	Storage temp.(°F)
Inshell	Open basket[z]	50	24	12	12	50
Inshell	Open basket	70	24	6	6	Ambient
Inshell	Open basket	80	24	4	4	80
Inshell	Wooden bin[y]	50	24	12	12	50
Inshell	Wooden bin	70	24	6	6	Ambient
Inshell	Wooden bin	80	24	4	4	80
Shelled	Open basket	50	16	12	12	50
Shelled	Open basket	70	16	6	6	Ambient
Shelled	Open basket	80	16	4	4	80
Shelled	Wooden bin	50	16	12	12	50
Shelled	Wooden bin	70	16	6	6	Ambient
Shelled	Wooden bin	80	16	4	4	80
Shelled	Carton[x]	50	16	12	12	50
Shelled	Carton	70	16	6	6	Ambient
Shelled	Carton	80	16	4	4	80

[z]Open baskets simulated commercial bulk treatment.

[y]Small wooden bins simulated commercial bin treatments.

[x]Cardboard cartons without liners or packing material.

Table 2

Treatment schedules and desorption parameters for 'Nonpareil' almonds fumigated using the old schedule.

Product description	Container type	Fumigation temp.(^{o}F)	Dosage (g/m^3)	Exposure time (h)	Chamber aeration (h)	Storage temp.(^{o}F)
Inshell	Open basket[z]	50	56	24	24	50
Inshell	Open basket	70	56	24	24	Ambient
Inshell	Wooden bin[y]	50	56	24	24	50
Inshell	Wooden bin	70	56	24	24	Ambient
Shelled	Open basket	50	56	24	24	50
Shelled	Open basket	70	56	24	24	Ambient
Shelled	Wooden bin	50	56	24	24	50
Shelled	Wooden bin	70	56	24	24	Ambient
Shelled	Carton[x]	50	56	24	24	50
Shelled	Carton	70	56	24	24	Ambient
Shelled	Carton	80	56	24	24	80

[z]Open baskets simulated commercial bulk treatments.

[y]Small wooden bins simulated commercial bin treatments.

[x]Cardboard cartons without liners or packing material.

Table 3

Treatment schedules and desorption parameters for 'Nonpareil' almonds fumigated using both new and old schedules along with eggs of navel orangeworm (NOW), Ameylois transitella (Walker) and Indianmeal moth (IMM), Plodia interpunctella (Hübner).

Product description	Container type	Fumigation temp.(°F)	Dosage (g/m^3)	Exposure time (h)	Chamber aeration (h)	Storage temp.(°F)
Shelled	Carton[z]	70	16	6	6	70
Shelled	Carton	70	56	24	24	70

[z]Cardboard cartons with commercial liners.

Table 4

Extremes in organic bromide residues in shelled almonds fumigated with methyl bromide using the new schedules (16 g/m^3 dosage).

Container type	Fumigation temp.[z] (°F)	Exposure time (h)	Storage temp.[z] (°F)	Post-fumigation sample time (days)	Organic bromide[yx] (ppm)
Basket	50	12	50	0.5	22.784 ± 1.253
				20	0.006 ± 0.007
				23	<0.005
	70	6	71.2 ± 1.3[w]	0.25	5.470 ± 1.700
				7	0.032 ± 0.026
				10	<0.005
	80	4	80	0.167	8.412 ± 1.594
				9	0.007 ± 0.004
				12	<0.005
Bin	50	12	50	0.5	10.569 ± 0.854
				23	0.029 ± 0.009
				28	<0.005
	70	6	72.9 ± 2.8[w]	0.25	4.915 ± 0.606
				18	0.015 ± 0.005
				23	<0.005
	80	4	80	0.167	3.984 ± 0.971
				2	0.056 ± 0.034
				5	<0.005
Carton[v]	50	12	50	0.05	25.908 ± 5.518
				23	0.016 ± 0.018
				28	<0.005
	70	6	79.3 ± 5.8[w]	0.25	17.233 ± 3.887
				19	0.011 ± 0.003
				23	<0.005
	80	4	80	0.167	11.354 ± 1.397
				13	0.021 ± 0.009
				18	<0.005

[z]All fumigation temperatures and the 50°F and 80°F storage temperatures were ± 2°F.
[y]All control samples had <0.005 ppm organic bromide.
[x]Values denote means of three replications ± standard deviations.
[w]Value denotes mean ambient temperature for duration of storage ± standard deviation.
[v]

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Extremes in organic bromide residues in inshell almonds fumigated with methyl bromide using the new schedules (24 g/m^3 dosage).

Container type	Fumigation temp.[z] (°F)	Exposure time (h)	Storage temp.[z] (°F)	Post-fumigation sample time (days)	Organic bromide[yx] (ppm)
Basket	50	12	50	0.5	17.918 ± 0.504
				23	0.014 ± 0.009
				28	<0.005
	70	6	74.1 ± 7.3[w]	0.25	3.382 ± 0.537
				28	0.013 ± 0.005
				33	<0.005
	80	4	80	0.167	5.433 ± 1.174
				2	0.118 ± 0.022
				5	<0.005
Bin	50	12	50	0.5	12.434 ± 0.632
				23	0.031 ± 0.035
				28	<0.005
	70	6	72.8 ± 2.8[w]	0.25	5.185 ± 1.358
				13	0.006 ± 0.003
				18	<0.005
	80	4	80	0.167	9.585 ± 0.270
				7	0.023 ± 0.021
				9	<0.005

[z]All fumigation temperatures and the 50°F and 80°F storage temperatures were ± 2°F.

[y]All control samples had <0.005 ppm organic bromide.

[x]Values denote means of three replications ± standard deviation.

[w]Value denotes mean ambient temperature for duration of storage ± standard deviation.

Table 6

Extremes in organic bromide residues in shelled almonds fumigated with methyl bromide using the old schedule (56 g/m^3 dosage).

Container type	Fumigation temp.[z] (°F)	Exposure time (h)	Storage temp.[z] (°F)	Post-fumigation sample-time (days)	Organic bromide[yx] (ppm)
Basket	50	24	50	1	52.538 ± 4.162
				58	0.011 ± 0.013
				63	<0.005
	70	24	81.8 ± 5.8[w]	1	25.250 ± 4.782
				37	0.005 ± 0.000
				42	<0.005
Bin	50	24	50	1	49.554 ± 2.268
				40	0.034 ± 0.020
				50	<0.005
	70	24	82.4 ± 5.5[w]	1	29.934 ± 5.934
				45	0.006 ± 0.002
				47	<0.005
Carton[v]	50	24	50	1	50.803 ± 5.687
				66	0.011 ± 0.004
				70	<0.005
	70	24	80.3 ± 4.1[w]	1	24.491 ± 2.221
				29	0.005 ± 0.001
				31	<0.005
	80	24	80	1	7.575 ± 0.476
				16	0.011 ± 0.010
				19	<0.005

[z]All fumigation temperatures and 50°F and 80°F storage temperatures were ± 2°F.
[y]All control samples had <0.005 ppm organic bromide.
[x]Values denote means of three replications ± standard deviations.
[w]Value denotes mean mean ambient temperature for duration of storage ± standard deviation.
[v]Carton without liner.

Table 7

Extremes in organic bromide residues in inshell almonds fumigated with methyl bromide using the old schedule (56 g/m^3 dosage).

Container type	Fumigation temp.[z] (°F)	Exposure time (h)	Storage temp.[z] (°F)	Post-fumigation sample-time (days)	Organic bromide[yx] (ppm)
Basket	50	24	50	1	19.852 ± 4.423
				32	0.006 ± 0.004
				36	<0.005
	70	24	81.7 ± 7.4[w]	1	55.102 ± 1.357
				28	0.012 ± 0.002
				33	<0.005
Bin	50	24	50	1	21.576 ± 1.715
				23	0.030 ± 0.016
				28	<0.005
	70	24	79.5 ± 7.8[w]	1	37.307 ± 4.423
				36	0.005 ± 0.003
				39	<0.005

[z]All fumigation temperatures and 50°F and 80°F storge temperatures were ± 2°F.

[y]All control samples had <0.005 ppm organic bromide.

[x]Values denote means of three replications ± standard deviations.

[w]Values denote mean ambient temperature for duration of storage ± standard deviation.

Table 8

Extremes in organic bromide residues in shelled almonds fumigated with methyl bromide using new and old schedules at 70°F in cartons with commercial liners and stored at 70°F.

Dosage (g/m^3)	Exposure time (h)	Post-fumigation sample time (days)	Organic bromide[yx] (ppm)
16	6	0.26	9.639 ± 1.326
		29	0.007 ± 0.003
		33	<0.005
56	24	1	19.822 ± 5.056
		39	0.007 ± 0.003
		45	<0.005

[z]These treatments included navel orangeworm and Indianmeal moth eggs in plastic vials buried in the shelled almonds.

[y]All control samples had <0.005 ppm organic bromide residue

[x]Values denote means of three replication ± standard deviation.

Table 9

Organic bromide desorption rates and half-lives for inshell and shelled almonds fumigated using the new schedules.

Product description	Container type	Fumigation temp.[z] (°F)	Storage temp.[z] (°F)	Desorption rate[y] (log ppm/day)	Half-life[x] (days)
Inshell	Basket	50	50	-0.141	2.135
		70	74.1 ± 7.3[w]	-0.099	3.028
		80	80	-0.650	0.463
	Bin	50	50	-0.128	2.352
		70	72.8 ± 2.8[w]	-0.188	1.601
		80	80	-0.392	0.768
Shelled	Basket	50	50	-0.195	1.544
		70	71.2 ± 1.3[w]	-0.322	0.935
		80	80	-0.362	0.832
	Bin	50	50	-0.113	2.664
		70	72.9 ± 2.8[w]	-0.130	2.316
		80	80	-0.603	0.499
	Carton[v]	50	50	-0.152	1.980
		70	79.3 ± 5.8[w]	-0.176	1.710
		80	80	-0.230	1.309

[z]All fumigation temperatures and 50°F and 80°F storage temperatures were ± 2°F.

[y]Values represent slope terms from the linear regression equations.

[x]Values were computed using linear regression equations. Values represent time required for organic bromide residues to drop in half.

[w]Value denotes mean ambient temperature for duration of stoage ± standard deviation.

[v]Carton without liner.

Table 10

Organic bromide desorption rates and half-lives for inshell and shelled almonds fumigated using the old schedule ($56 g/m^3$ for 24 h).

Product description	Container type	Fumigation temp.[z] (°F)	Storage temp.[z] (°F)	Desorption rate[y] (log ppm/day)	Half-life[x] (days)
Inshell	Basket	50	50	-0.126	2.389
		70	81.7 ± 7.4[w]	-0.142	2.120
	Bin	50	50	-0.137	2.197
		70	79.5 ± 7.8[w]	-0.107	2.813
Shelled	Basket	50	50	-0.066	4.596
		70	81.8 ± 5.8[w]	-0.087	3.456
	Bin	50	50	-0.088	3.436
		70	82.4 ± 5.5[w]	-0.081	3.698
	Carton[v]	50	50	-0.058	5.181
		70	80.3 ± 4.1[w]	-0.120	2.509
		80	80	-0.171	1.760

[z]All fumigation temperatures and 50°F amd 80°F storage tempertures were ± 2°F.

[y]Values represent slope terms from linear regression equations.

[x]Values were computed using linear regression equations. Values represent time required for organic bromide residues to drop in half.

[w]Value denotes mean ambient temperature for duration of storage ± standard deviation.

[v]Carton without liner.

Table 11

Organic bromide desorption rates and half-lives for shelled almonds fumigated with methyl bromide using new and old schedules at 70°F in cartons with commercial liners and stored at 70°F.

Dosage (g/m^3)	Exposure (h)	Desorption rate[y] (log ppm/day)	Half-life[x] (days)
16	6	-0.117	2.573
56	24	-0.097	3.116

[z]These treatments included navel orangeworm and Indianmeal moth eggs in plastic vials buried in the shelled almonds.

[y]Values represent slope terms from linear regression equations.

[x]Values were computed using linear regression equations. Values represent time required for organic bromide residues to drop in half.

Table 12

Percentage mortality for eggs of navel orangeworm (NOW), Amyelois transitella (Walker) fumigated with methyl bromide using new and old schedules at 70°F in the presence of shelled almonds enclosed in liners within cartons.

Dosage (g/m^3)	Exposure time (h)	Number of eggs per replication[z]	Mortality[zy] %
Control	—	206.0 ± 4.0	7.8 ± 0.2a
16	6	202.0 ± 4.4	100.0 ± 0.0b
56	24	213.7 ± 8.7	100.0 ± 0.0b

[z]Values denote means of three replications ± standard deviations. When applicable, means separated using Duncan's multiple range test at the 0.05 level.

[y]Values corrected using Abbott's formula to compensate for observed mortality in untreated controls.

Table 11

Organic bromide desorption rates and half-lives for shelled almonds fumigated with methyl bromide using new and old schedules at 70°F in cartons with commercial liners and stored at 70°F.

Dosage (g/m^3)	Exposure (h)	Desorption rate[y] (log ppm/day)	Half-life[x] (days)
16	6	-0.117	2.573
56	24	-0.097	3.116

[z]These treatments included navel orangeworm and Indianmeal moth eggs in plastic vials buried in the shelled almonds.

[y]Values represent slope terms from linear regression equations.

[x]Values were computed using linear regression equations. Values represent time required for organic bromide residues to drop in half.

Table 10

anic bromide desorption rates and half-lives for inshell and shelled almonds fumigated using the old schedule (56g/m^3 for 24 h).

Product description	Container type	Fumigation temp.[z] (^oF)	Storage temp.[z] (^oF)	Desorption rate[y] (log ppm/day)	Half-life[x] (days)
Inshell	Basket	50	50	-0.126	2.389
		70	81.7 ± 7.4[w]	-0.142	2.120
	Bin	50	50	-0.137	2.197
		70	79.5 ± 7.8[w]	-0.107	2.813
Shelled	Basket	50	50	-0.066	4.596
		70	81.8 ± 5.8[w]	-0.087	3.456
	Bin	50	50	-0.088	3.436
		70	82.4 ± 5.5[w]	-0.081	3.698
	Carton[v]	50	50	-0.058	5.181
		70	80.3 ± 4.1[w]	-0.120	2.509
		80	80	-0.171	1.760

[z]All fumigation temperatures and 50^oF amd 80^oF storage tempertures were ± 2^oF.

[y]Values represent slope terms from linear regression equations.

[x]Values were computed using linear regression equations. Values represent time required for organic bromide residues to drop in half.

[w]Value denotes mean ambient temperature for duration of storage ± standard deviation.

[v]Carton without liner.

Table 9

Organic bromide desorption rates and half-lives for inshell and shelled almonds fumigated using the new schedules.

Product description	Container type	Fumigation temp.[z] (°F)	Storage temp.[z] (°F)	Desorption rate[y] (log ppm/day)	Half-life[x] (days)
Inshell	Basket	50	50	-0.141	2.135
		70	74.1 ± 7.3[w]	-0.099	3.028
		80	80	-0.650	0.463
	Bin	50	50	-0.128	2.352
		70	72.8 ± 2.8[w]	-0.188	1.601
		80	80	-0.392	0.768
Shelled	Basket	50	50	-0.195	1.544
		70	71.2 ± 1.3[w]	-0.322	0.935
		80	80	-0.362	0.832
	Bin	50	50	-0.113	2.664
		70	72.9 ± 2.8[w]	-0.130	2.316
		80	80	-0.603	0.499
	Carton[v]	50	50	-0.152	1.980
		70	79.3 ± 5.8[w]	-0.176	1.710
		80	80	-0.230	1.309

[z]All fumigation temperatures and 50°F and 80°F storage temperatures were ± 2°F.

[y]Values represent slope terms from the linear regression equations.

[x]Values were computed using linear regression equations. Values represent time required for organic bromide residues to drop in half.

[w]Value denotes mean ambient temperature for duration of stoage ± standard deviation.

[v]Carton without liner.

Table 13

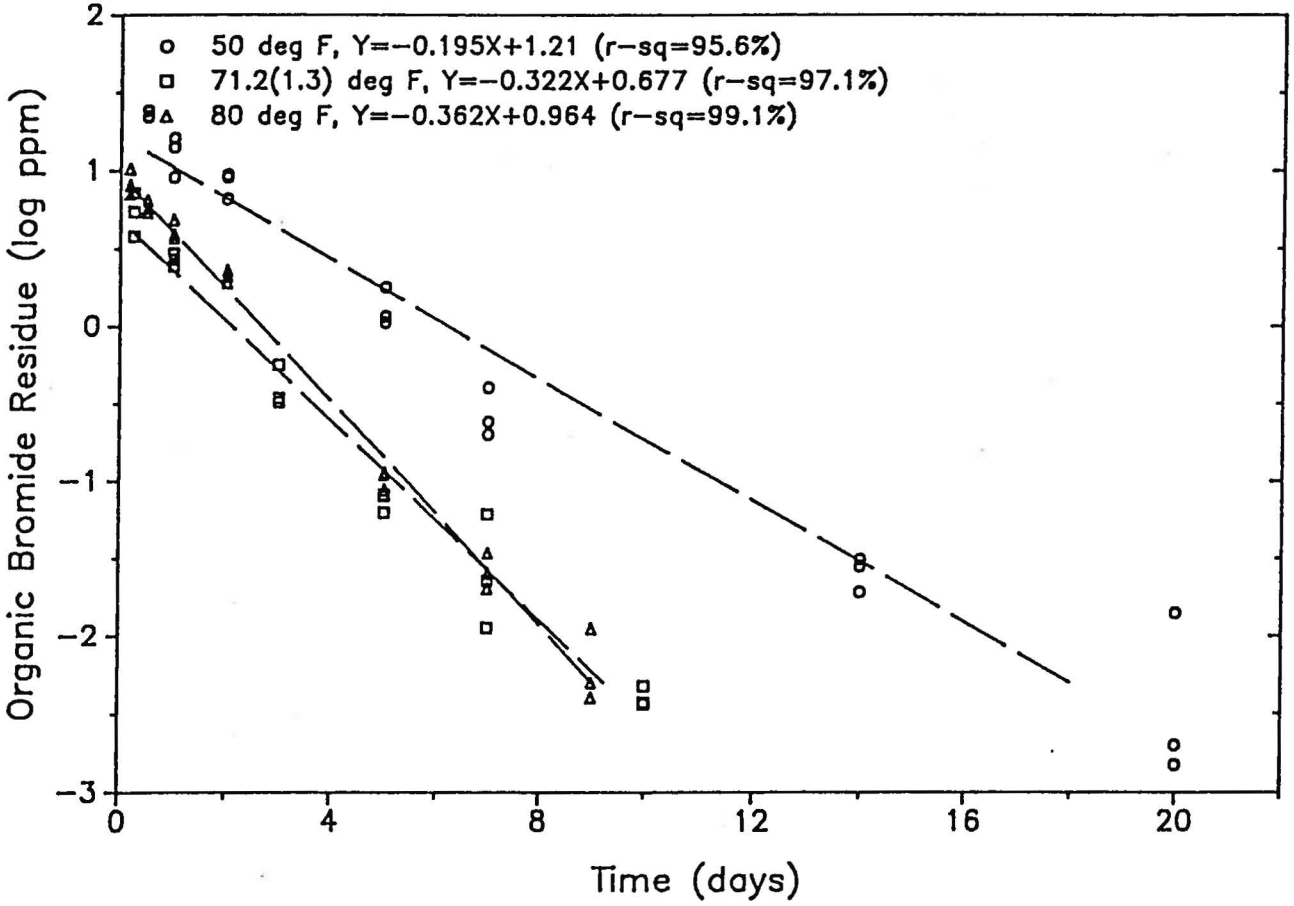
Percentage mortality for eggs of Indianmeal moth (IMM), Plodia interpunctella (Hübner) fumigated with methyl bromide using new and old schedules at 70°F in the presence of shelled almonds enclosed in liners within cartons.

Dosage (g/m^3)	Exposure time (h)	Number of eggs per replication[z]	Mortality[zy] %
Control	—	358.7 ± 8.0	3.9 ± 0.3a
16	6	353.3 ± 10.5	99.9 ± 0.2b
56	24	360.3 ± 12.0	99.9 ± 0.2b

[z]Values denote means of three replications ± standard deviations. When applicable, means separated using Duncan's multiple range test at the 0.05 level.

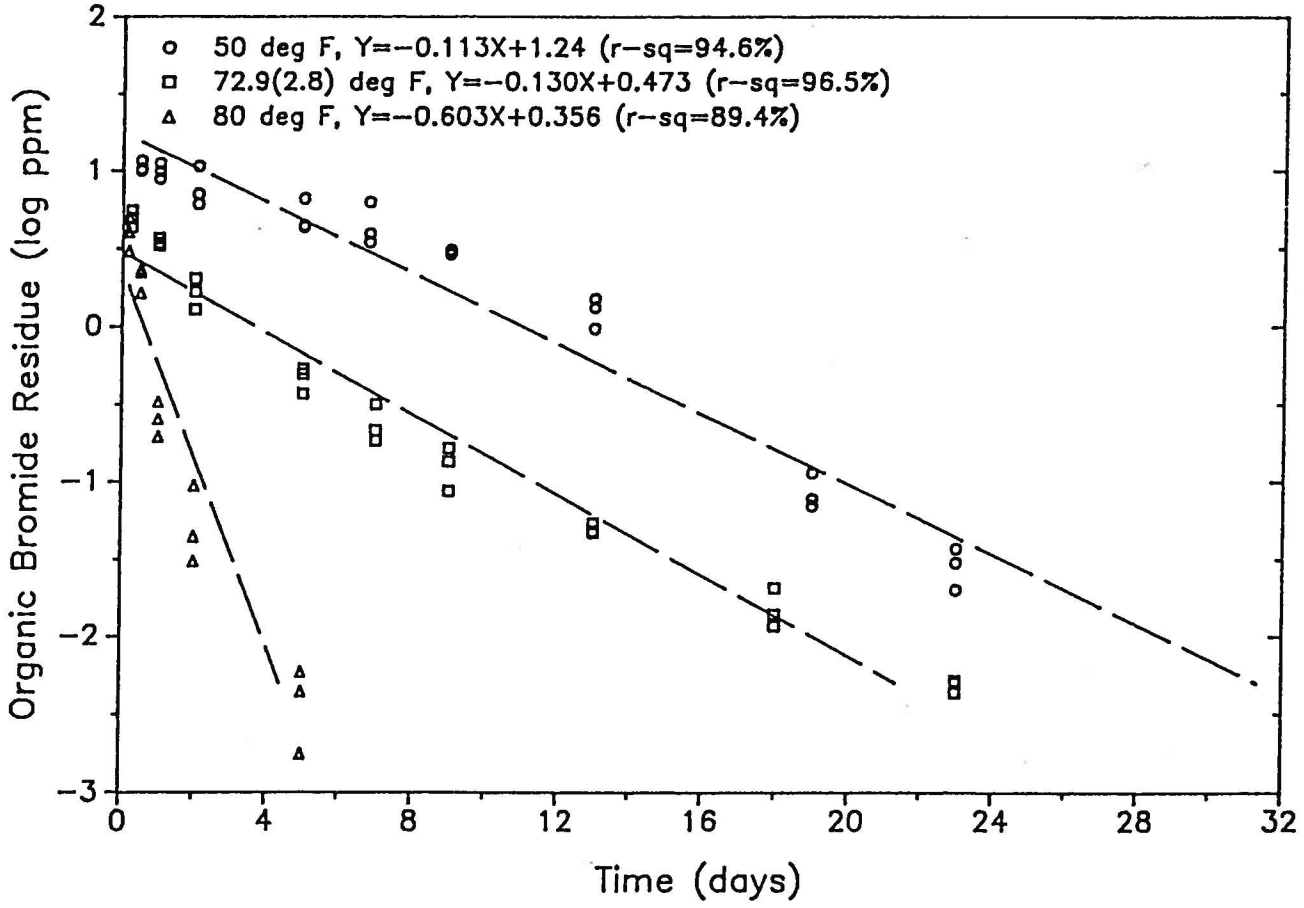
[y]Values corrected using Abbott's formula to compensate for observed mortality in untreated controls.

Figure 1



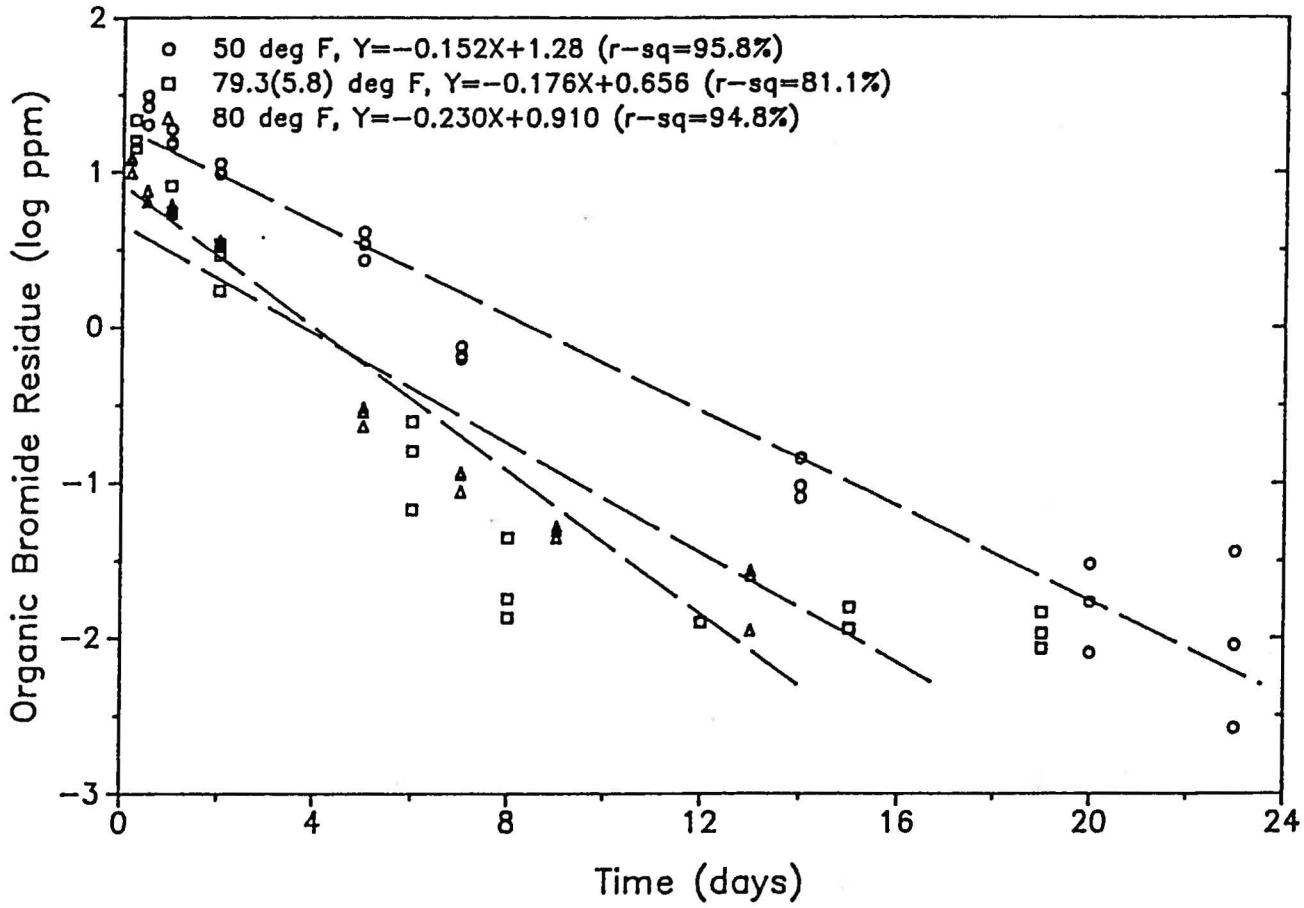
Comparison of organic bromide residues on the basis of fumigation and storage temperature for shelled almonds fumigated in open wire baskets at 50°F for 12h, 70°F for 6h, and 80°F for 4h with methyl bromide at a dosage of 16g/m^3 (new schedules) and stored at 50°F, ambient temperature, and 80°F.

Figure 2



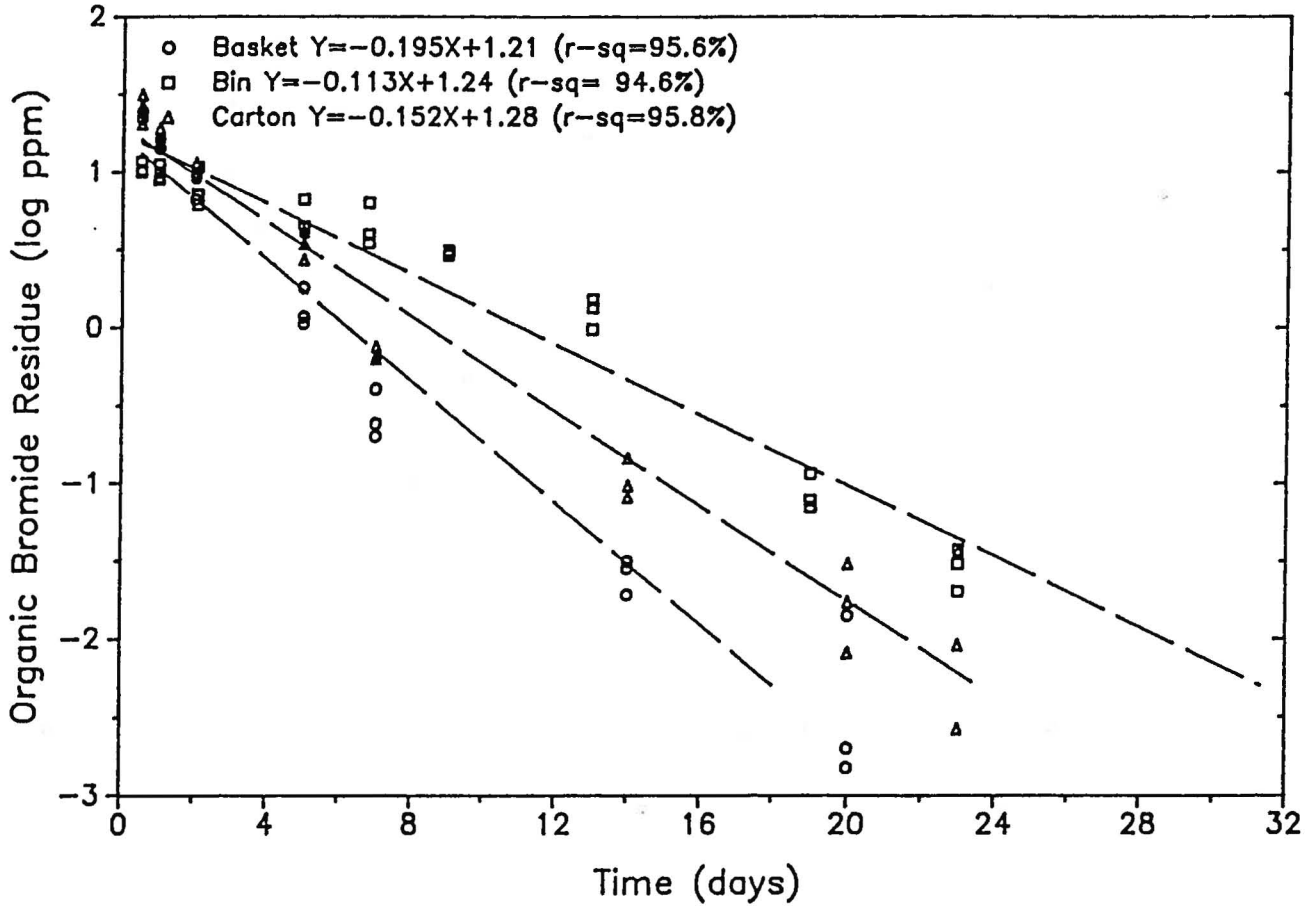
Comparison of organic bromide residues on the basis of fumigation and storage temperature for shelled almonds fumigated in wooden bins at 50°F for 12h, 70°F for 6h, and 80°F for 4h with methyl bromide at a dosage of 16g/m^3 (new schedules) and stored at 50°F, ambient temperature, and 80°F.

Figure 3



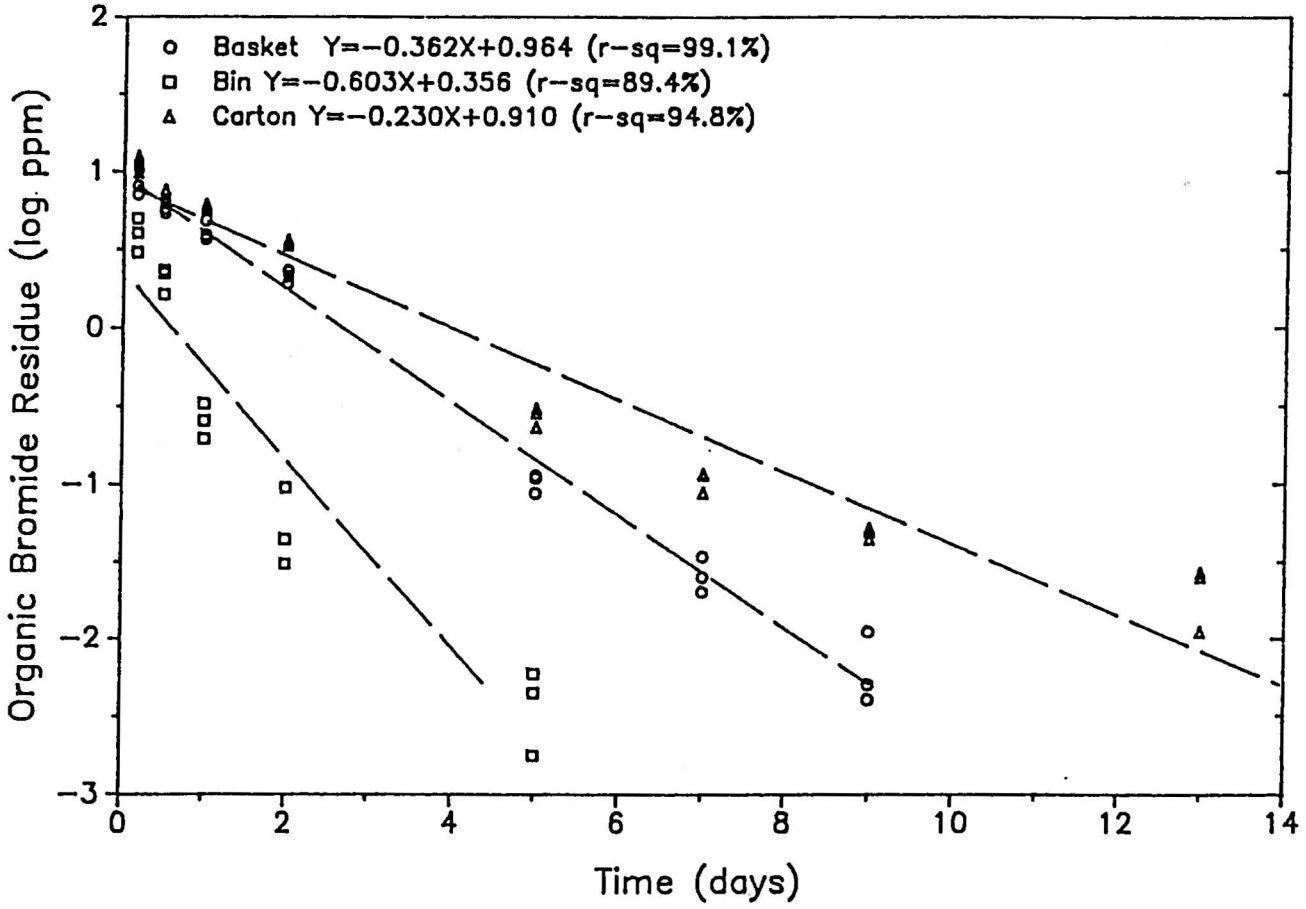
Comparison of organic bromide residues on the basis of fumigation and storage temperature for shelled almonds fumigated in cardboard cartons without liners at 50°F for 12h, 70°F for 6h, and 80°F for 4h with methyl bromide at a dosage of 16g/m^3 (new schedules) and stored at 50°F, ambient temperature, and 80°F.

Figure 4



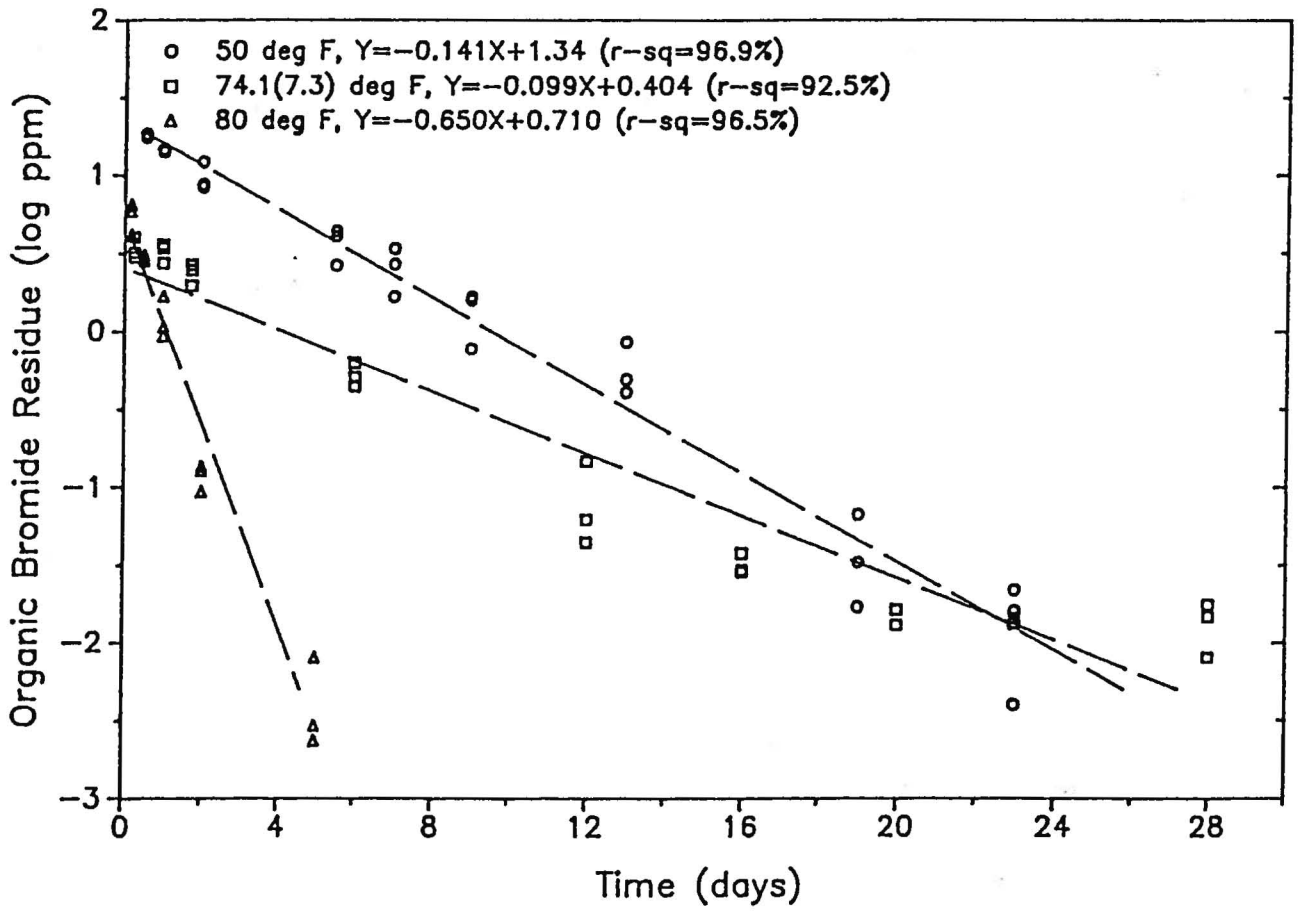
Comparison of organic bromide residues on the basis of container type for shelled almonds fumigated in open wire baskets, wooden bins, and cardboard cartons without liners at 50°F for 12h with methyl bromide at a dosage of 16g/m^3 (new schedule) and stored at 50°F.

Figure 5



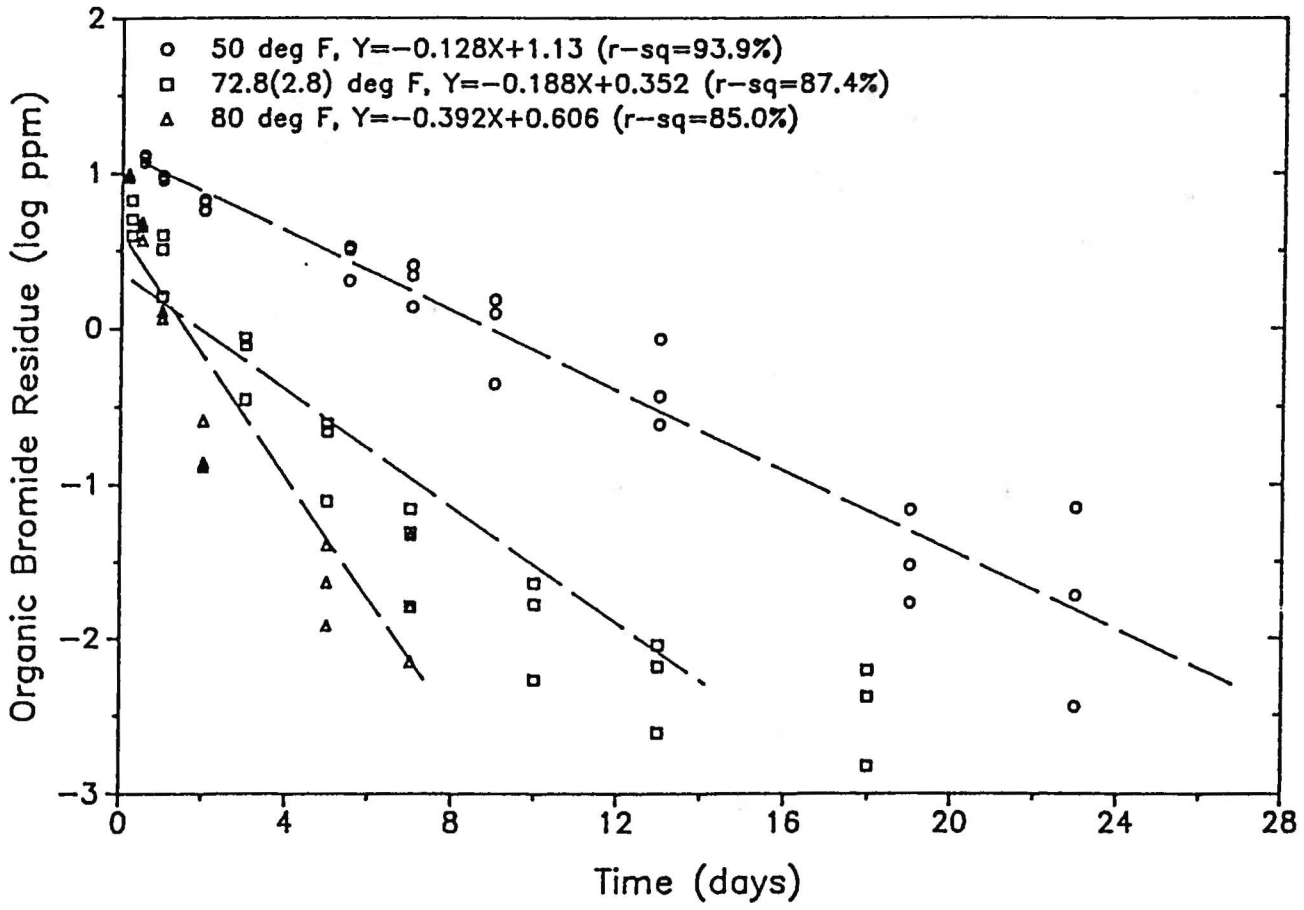
Comparison of organic bromide residues on the basis of container type for shelled almonds fumigated in open wire baskets, wooden bins, and cardboard cartons without liners at 80°F for 4h with methyl bromide at a dosage of 16g/m^3 (new schedule) and stored at 80°F.

Figure 6



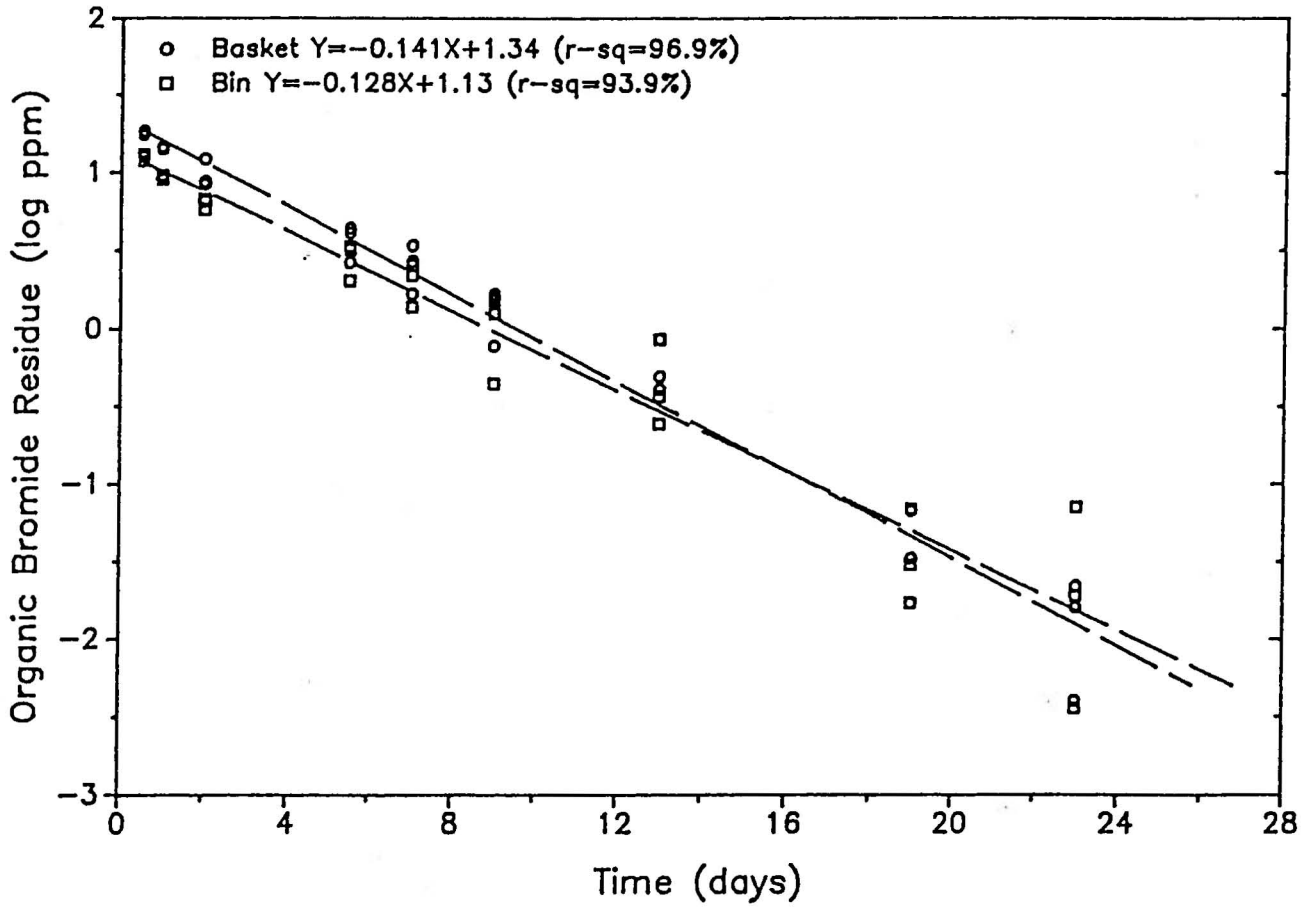
Comparison of organic bromide residues on the basis of fumigation and storage temperature for inshell almonds fumigated in open wire baskets at 50°F for 12h, 70°F for 6h, and 80°F for 4h with methyl bromide at a dosage of 24g/m^3 (new schedules) and stored at 50°F, ambient temperature, and 80°F.

Figure 7



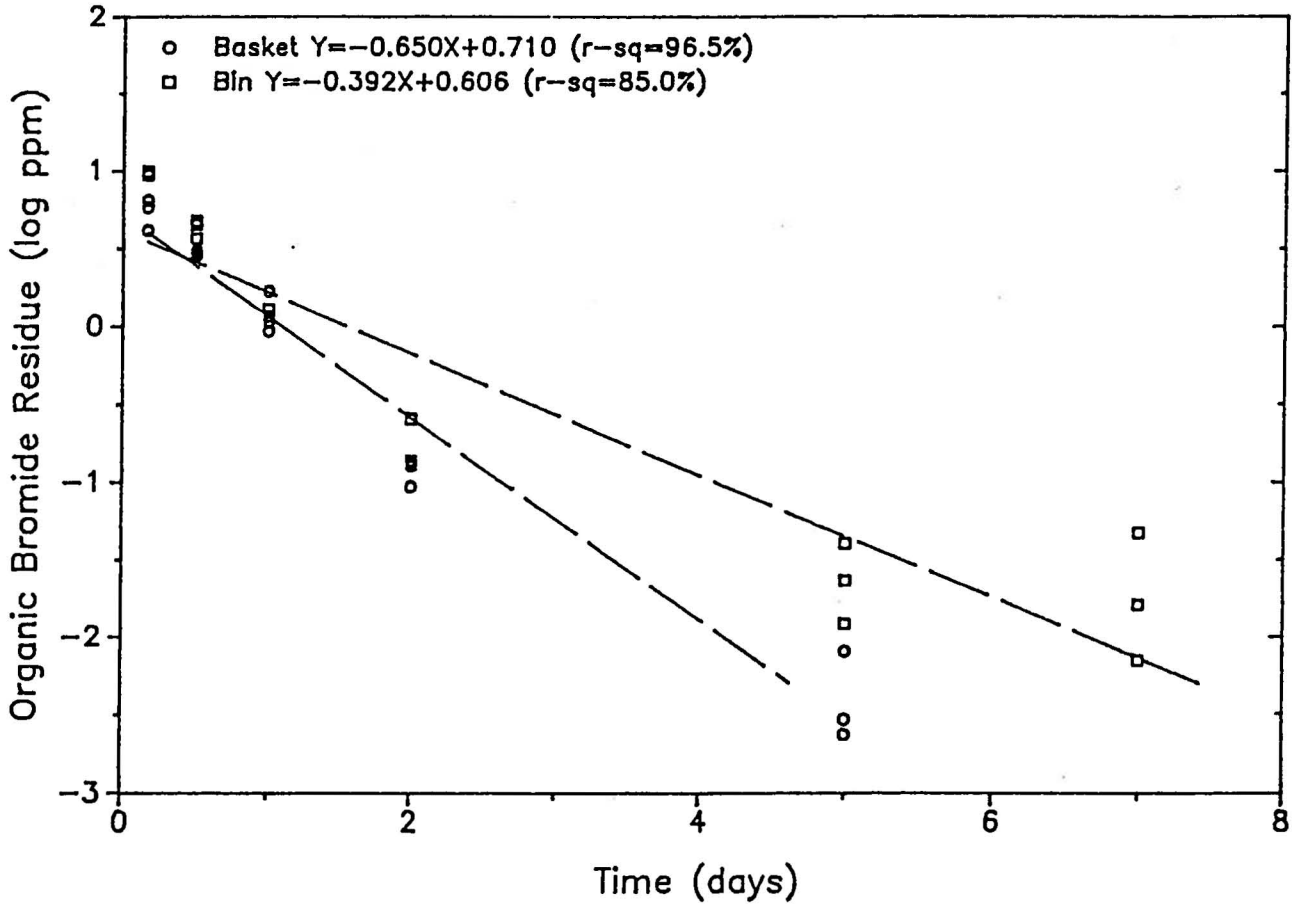
Comparison of organic bromide residues on the basis of fumigation and storage temperature for inshell almonds fumigated in wooden bins at 50°F for 12h, 70°F for 6h, and 80°F for 4h with methyl bromide at a dosage of 24g/m^3 (new schedules) and stored at 50°F, ambient temperature, and 80°F.

Figure 8



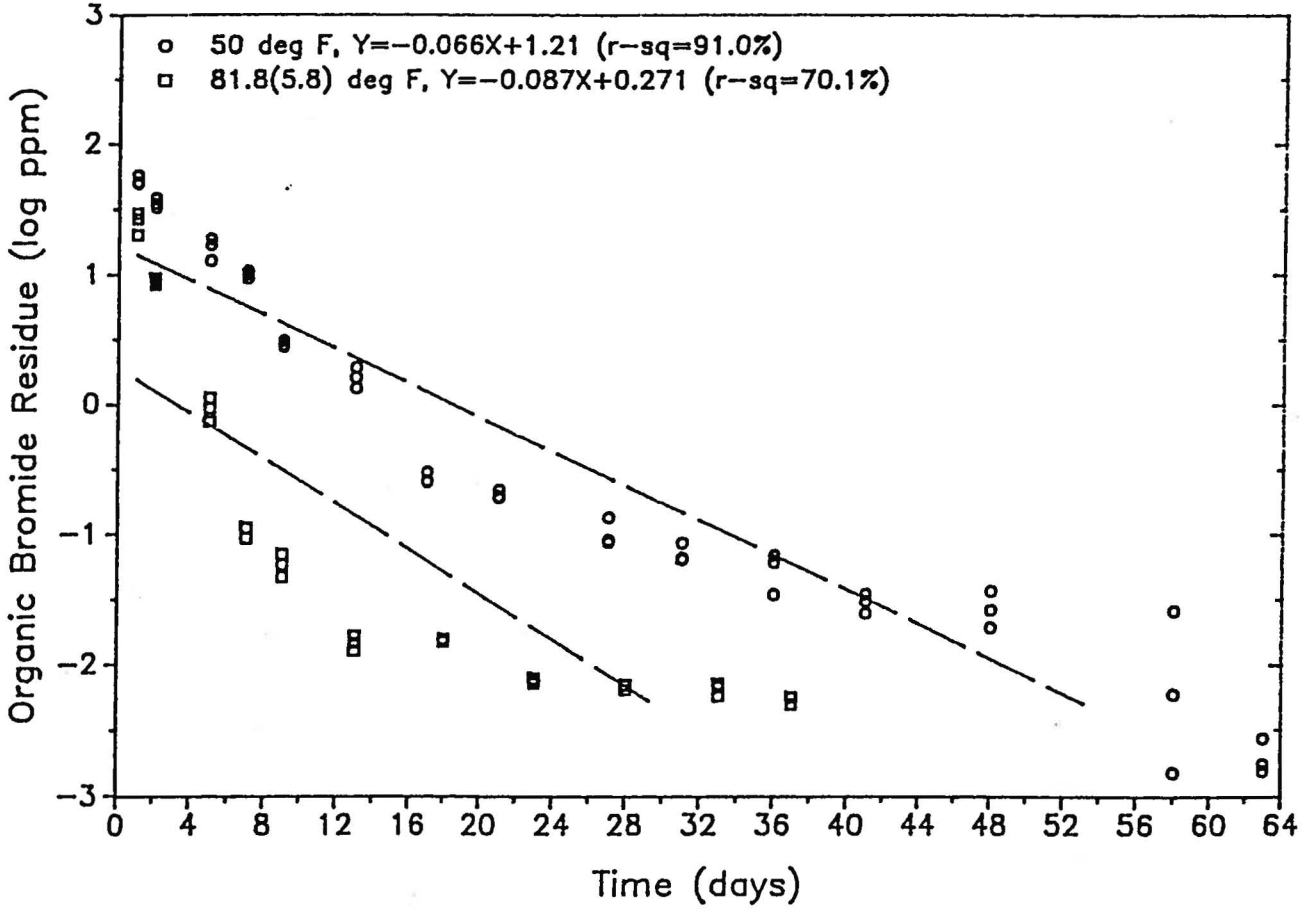
Comparison of organic bromide residues on the basis of container type for inshell almonds fumigated in open wire baskets and wooden bins at 50°F for 12h with methyl bromide at a dosage of 24g/m^3 (new schedule) and stored at 50°F.

Figure 9



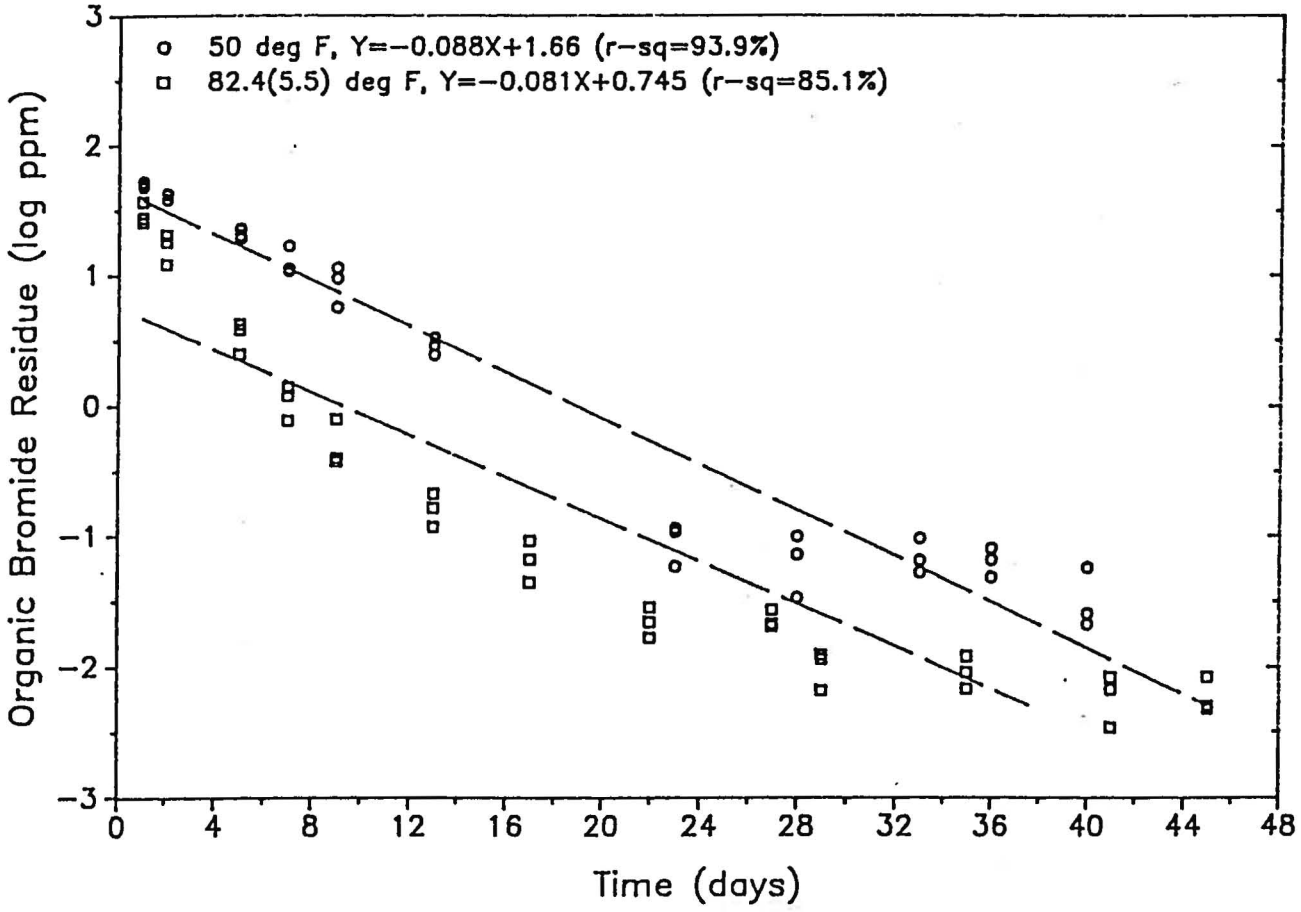
Comparison of organic bromide residues on the basis of container type for inshell almonds fumigated in open wire baskets and wooden bins at 80°F for 4h with methyl bromide at a dosage of 24g/m^3 (new schedule) and stored at 80°F.

Figure 10



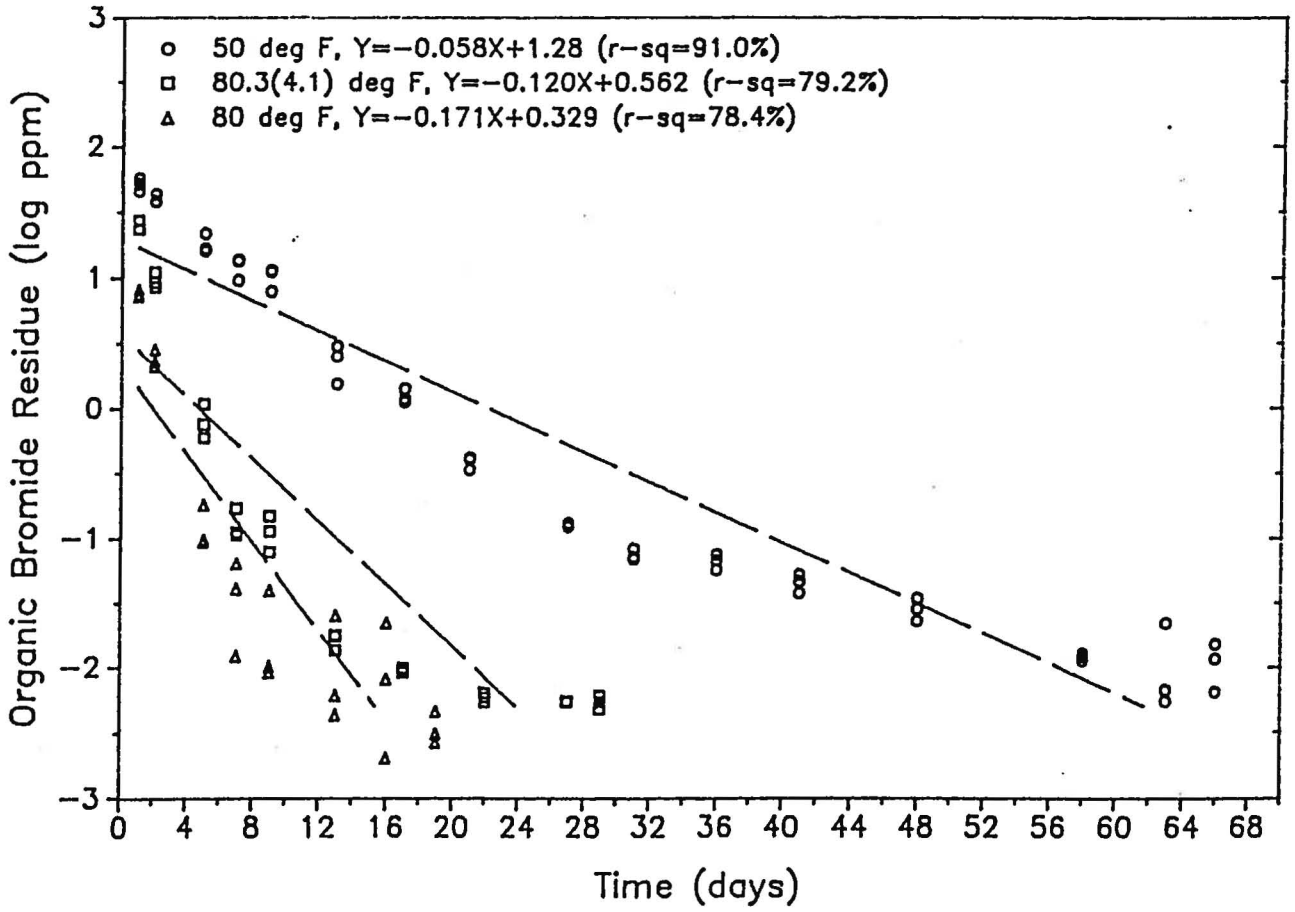
Comparison of organic bromide residues on the basis of fumigation and storage temperature for shelled almonds fumigated in open wire baskets at 50°F and 70°F with methyl bromide at a dosage of 56g/m^3 for 24h (old schedule) and stored at 50°F and ambient temperature.

Figure 11



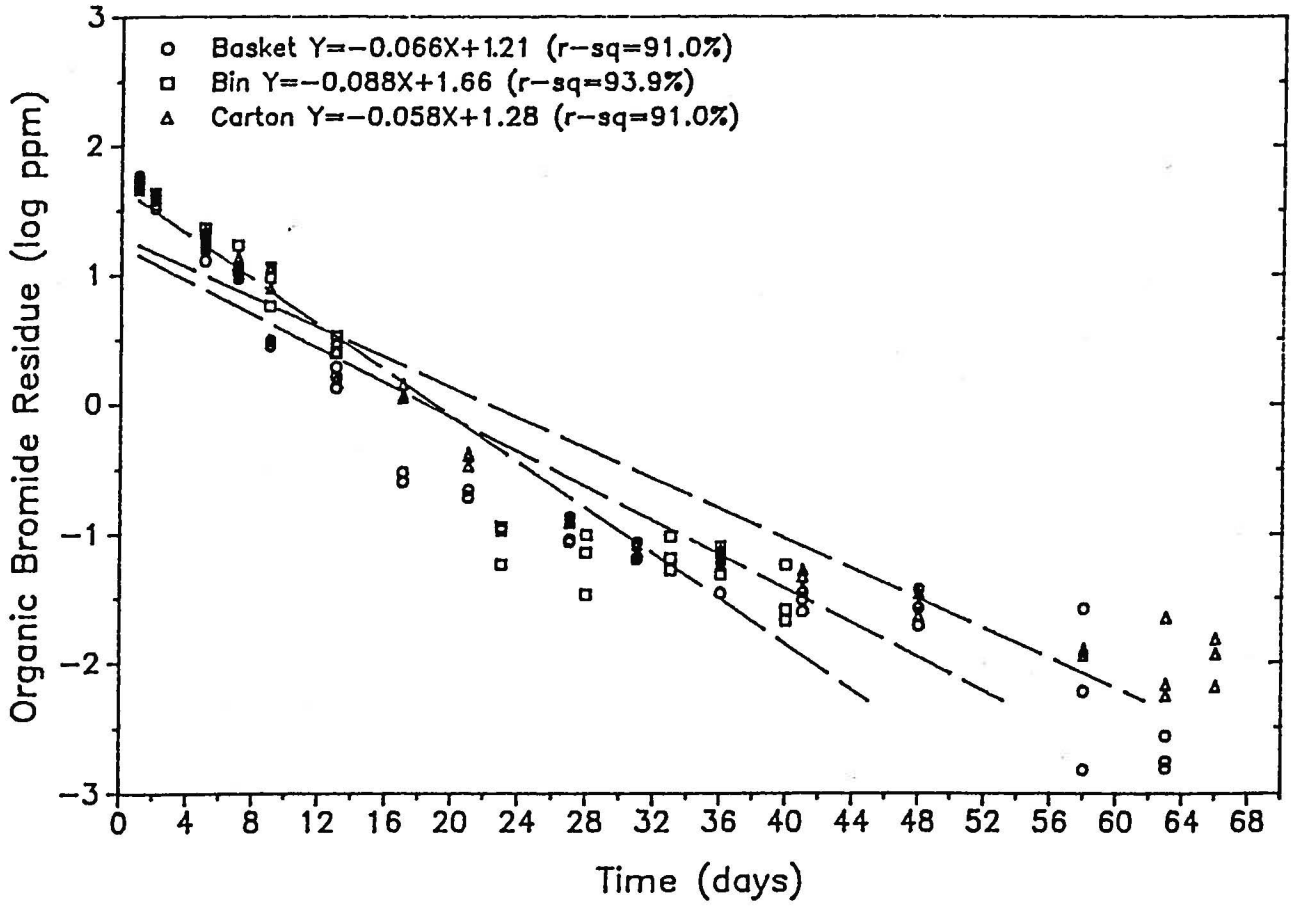
Comparison of organic bromide residues on the basis of fumigation and storage temperature for shelled almonds fumigated in wooden bins at 50°F and 70°F with methyl bromide at a dosage of 56g/m^3 for 24h (old schedule) and stored at 50°F and ambient temperature.

Figure 12



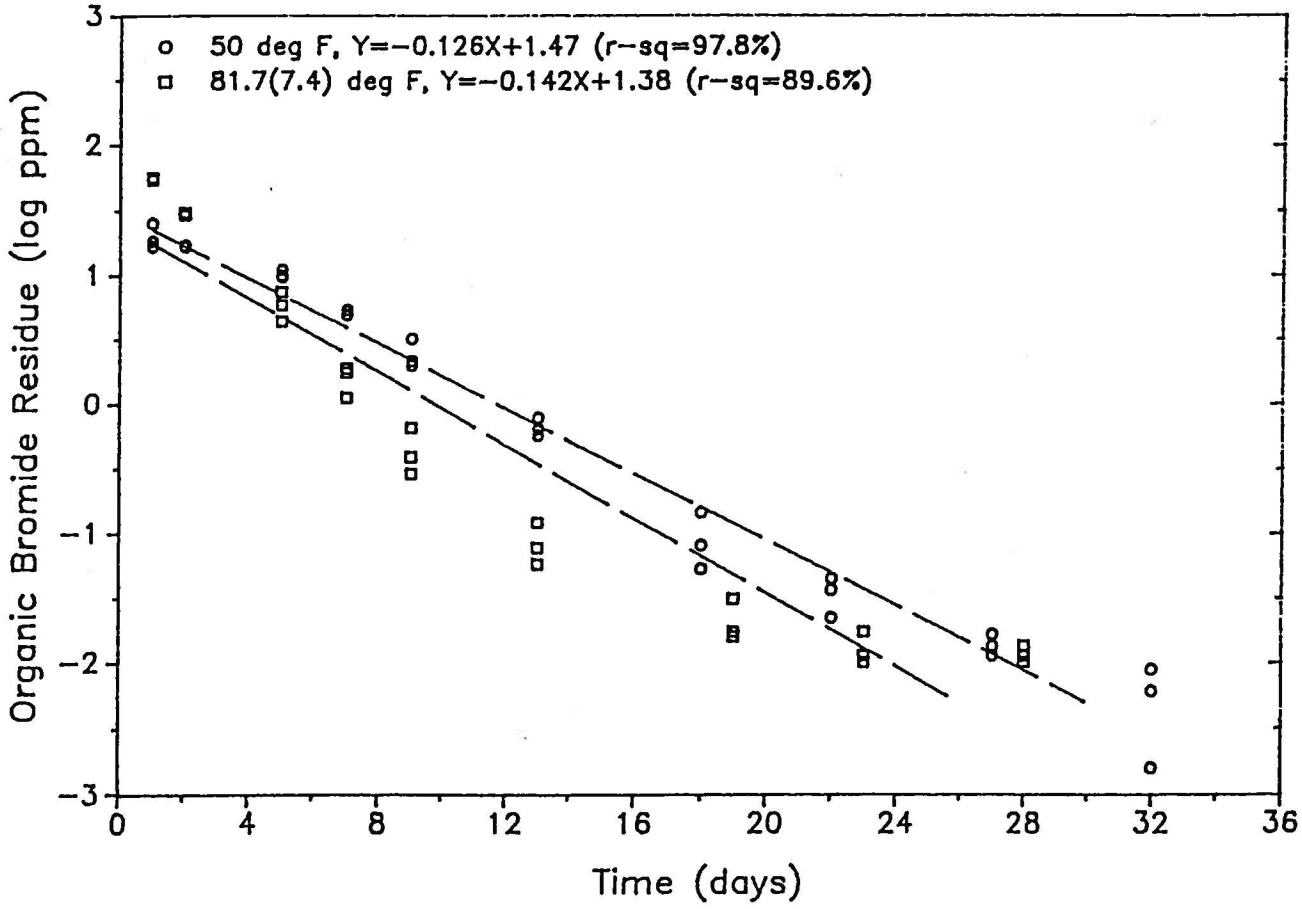
Comparison of organic bromide residues on the basis of fumigation and storage temperature for shelled almonds fumigated in cardboard cartons without liners at 50°F, 70°F, and 80°F with methyl bromide at a dosage of 56g/m^3 for 24h (old schedule) and stored at 50°F, ambient temperature, and 80°F.

Figure 13



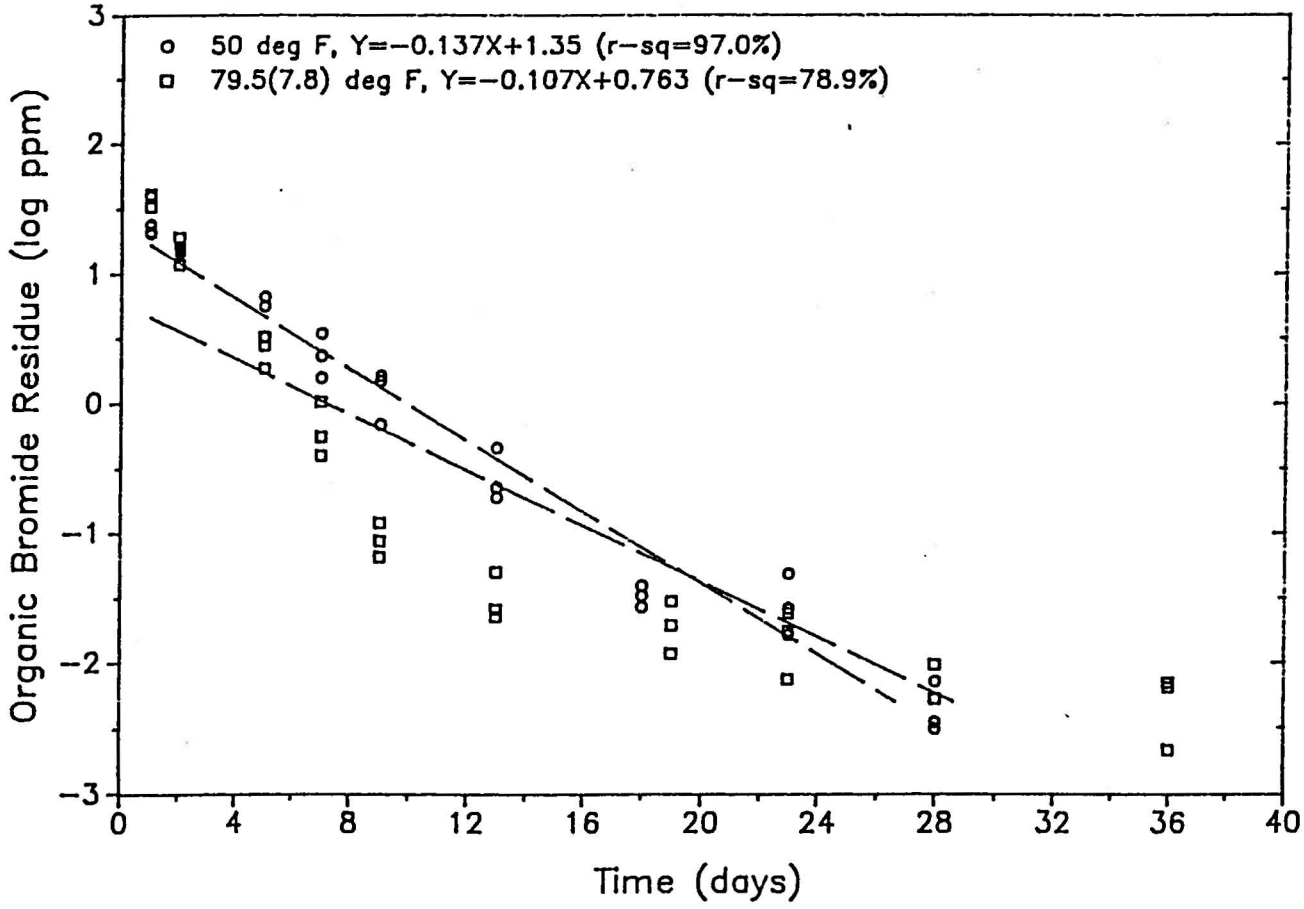
Comparison of organic bromide residues on the basis of container type for shelled almonds fumigated in open wire baskets, wooden bins, and cardboard cartons without liners at 50°F with methyl bromide at a dosage of 56g/m^3 for 24h (old schedule) and stored at 50°F.

Figure 14



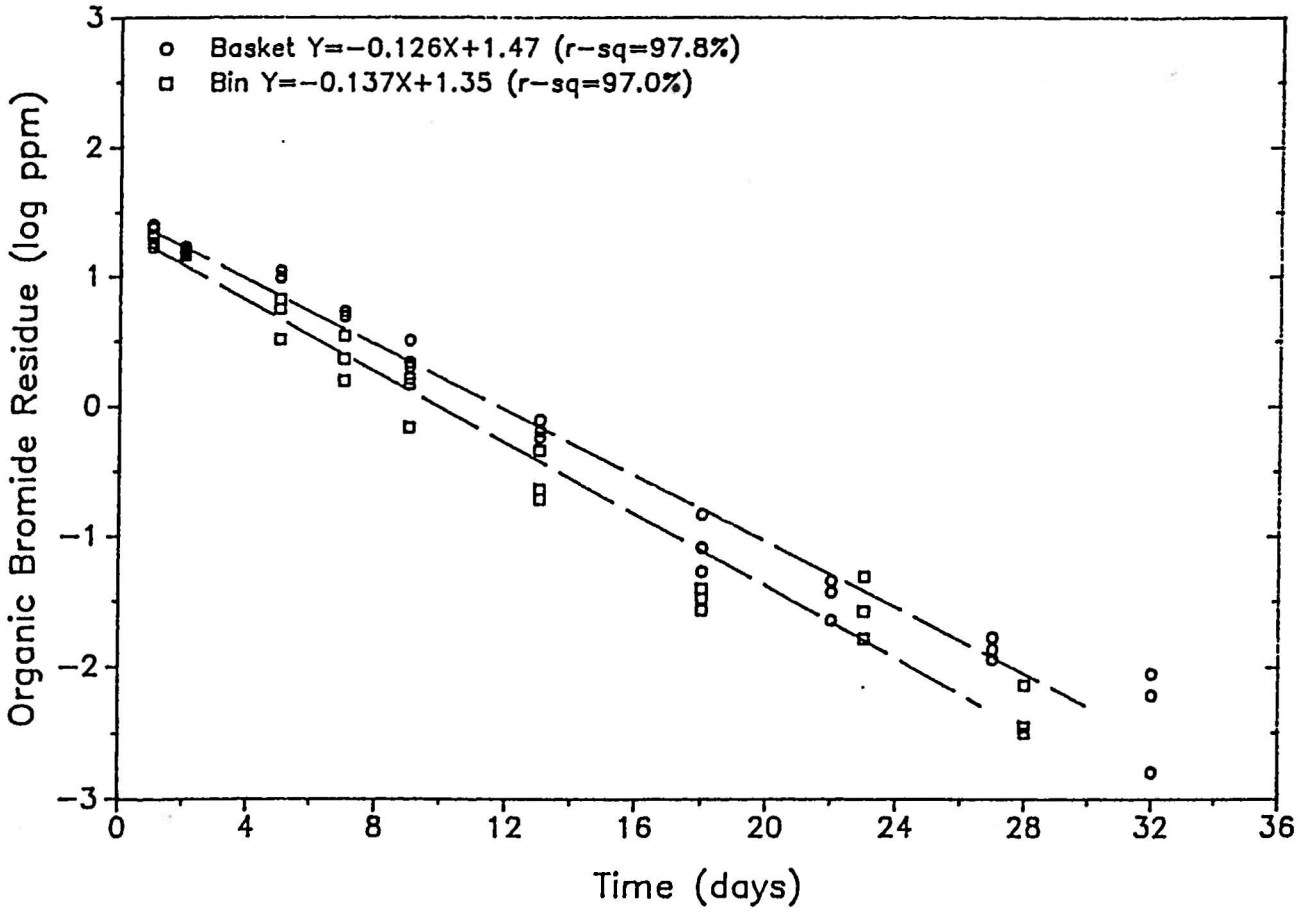
Comparison of organic bromide residues on the basis of fumigation and storage temperature for inshell almonds fumigated in open wire baskets at 50°F and 70°F with methyl bromide at a dosage of 56g/m^3 for 24h (old schedule) and stored at 50°F and ambient temperature.

Figure 15



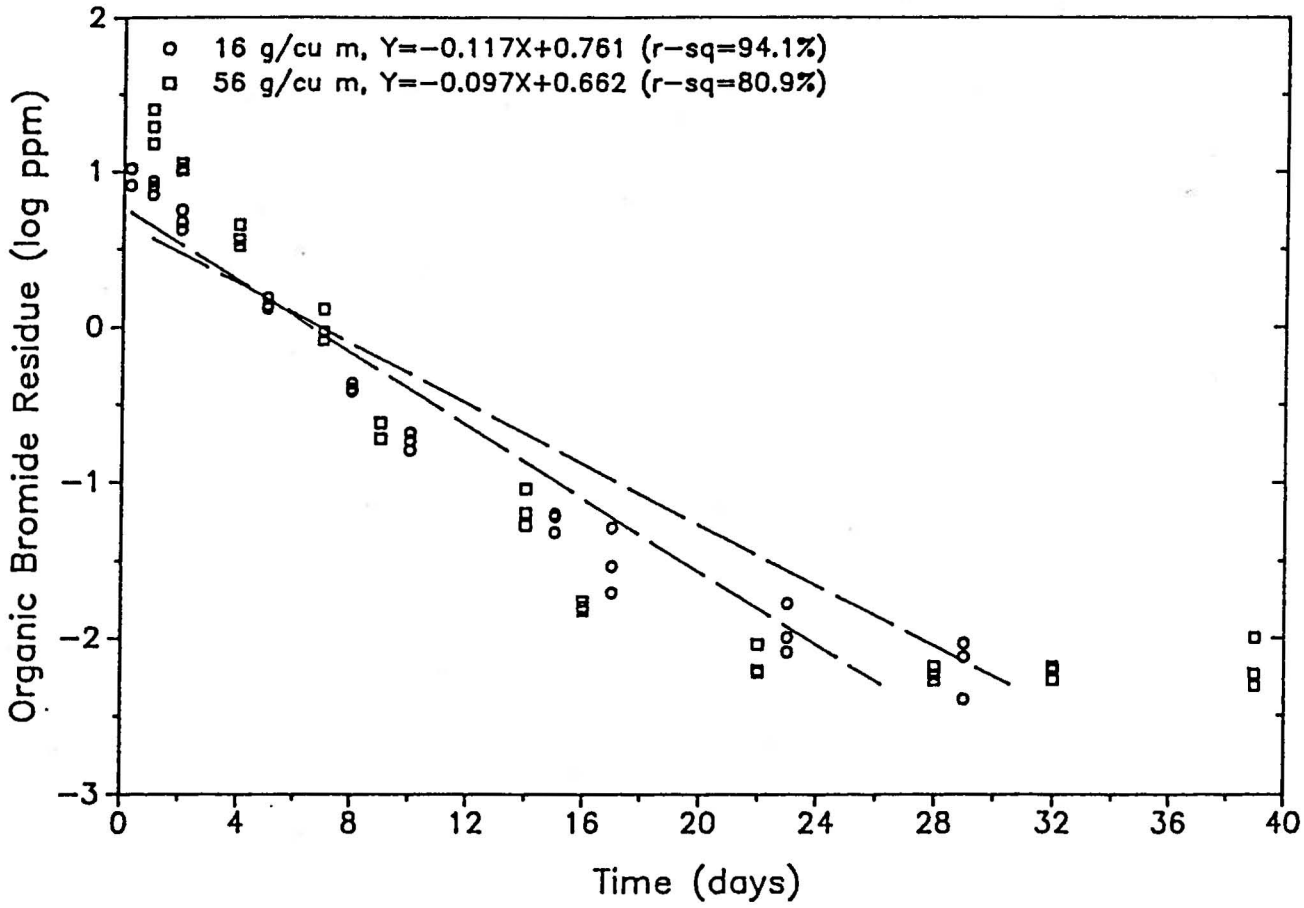
Comparison of organic bromide residues on the basis of fumigation and storage temperature for inshell almonds fumigated in wooden bins at 50°F and 70°F with methyl bromide at a dosage of 56g/m^3 for 24h (old schedule) and stored at 50°F and ambient temperature.

Figure 16



Comparison of organic bromide residues on the basis of container type for inshell almonds fumigated in open wire baskets and wooden bins at 50°F with methyl bromide at a dosage of 56g/m^3 for 24h (old schedule) and stored at 50°F.

Figure 17



Comparison of organic bromide residues in shelled almonds fumigated at 70°F with methyl bromide at a dosage of 16g/m^3 for 6h or 56g/m^3 for 24h (new and old schedules) in cardboard cartons with commercial liners and stored at 70°F.

Rf #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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[1] Department of Plant Pathology, University of California, Davis, CA 95616

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.

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)	*Stigminia 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

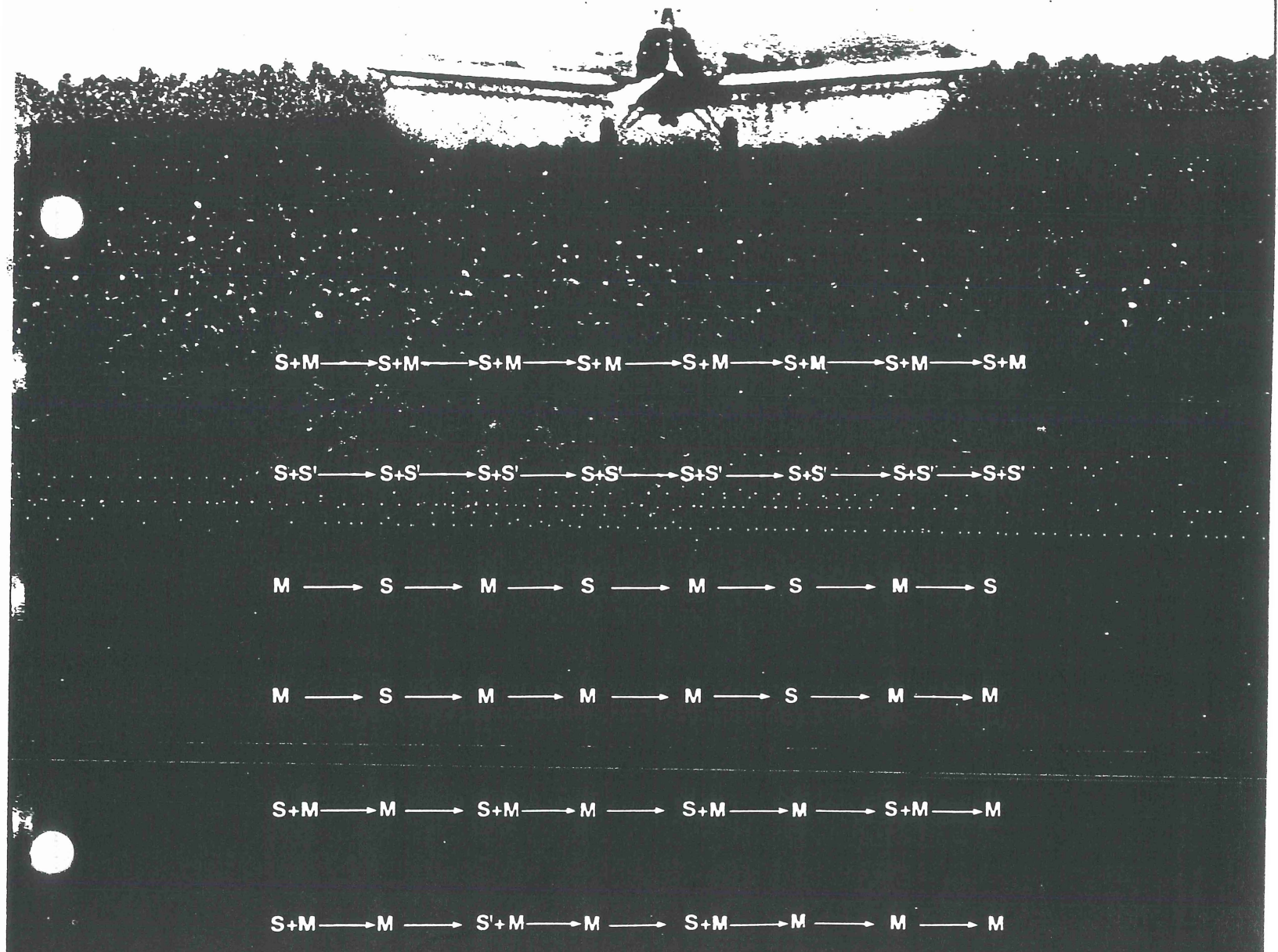
Common name	Pathogen or cause
Anthracnose	*Gloeosporium inconspicuum* Cavara = *Cylindrosporella inconspicua* (Cavara) Arx
	G. ulmicola Miles
Bacterial wetwood	*Bacillus megaterium* de Bary
	Enterobacter cloacae (Jordan) Hormaeche & Edwards = *Erwinia nimipressuralis* (Carter) Dye
	Pseudomonas fluorescens 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)

Fungicide Resistance in North America

Charles J. Delp, Editor

Associate Subject Matter Editors:
Bryan R. Delp, Thomas M. Fort III,
H. Vincent Morton, and Constance M. Smith



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 20 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 cichoracerarum* 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 25 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		Cost d/
			Systemic b/	Contact c/	(X $1,000)
Apricots	13	9,308	2	3	2,792
Nectarines	60	9,308	3	6	5,585
Peaches					
Processing	38	14,522	3	4	5,821
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.

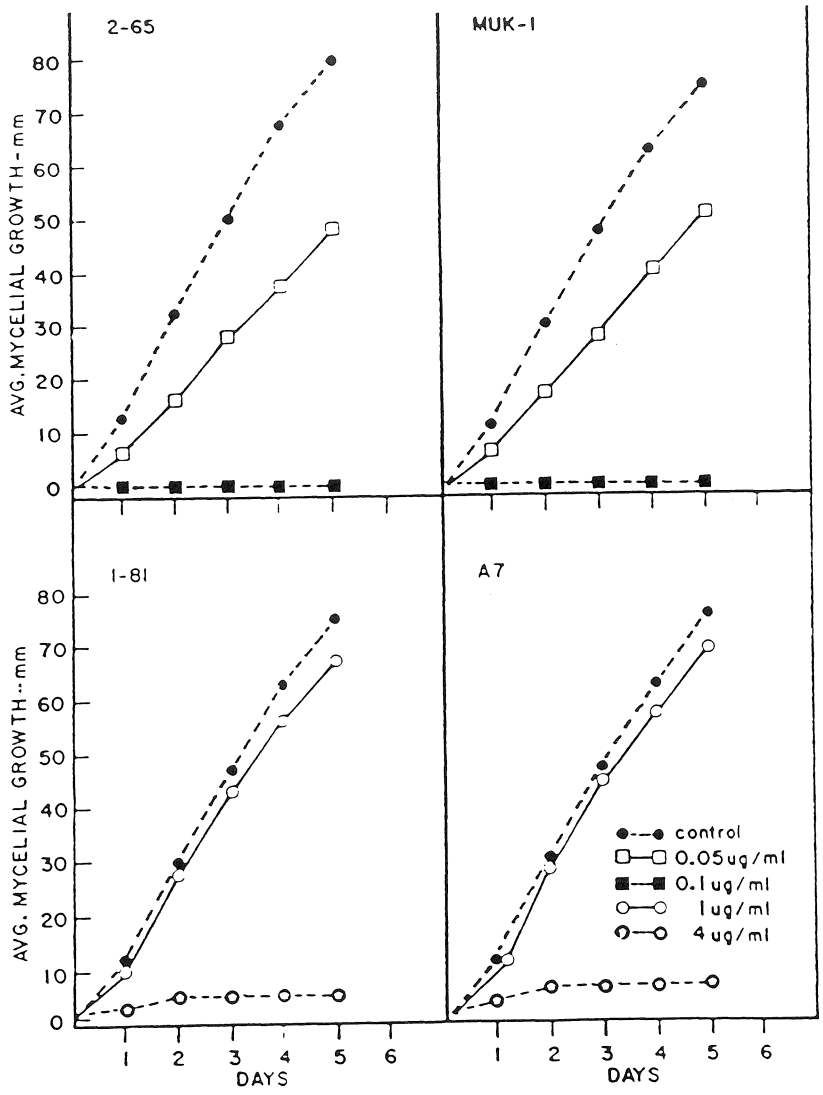
In the same peach orchard (Lockford, CA), Adaskaveg et al (1987) found that isolates of *M. fructicola* resistant to benomyl remained stable at 35% in the absence of benomyl treatments for an eight-year period. The orchard had been sprayed with a sterol biosynthesis inhibitor, triforine, for four years 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 model 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 (Dekhuijzen et al. 1983). 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 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.

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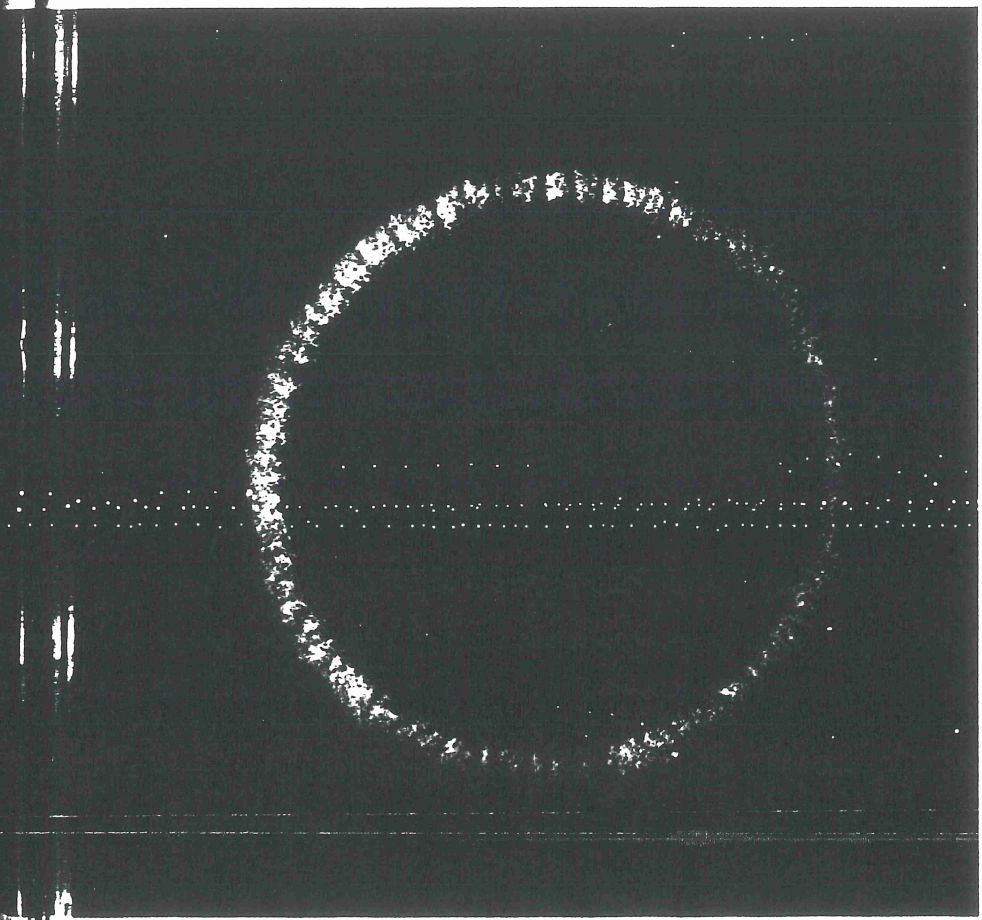

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Effect of sterol biosynthesis inhibitors on diseases of stone fruits and grapes in California

J. M. Ogawa, W. D. Gubler and B. T. Manji
Department of Plant Pathology, University of California, Davis, CA 95616, USA

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

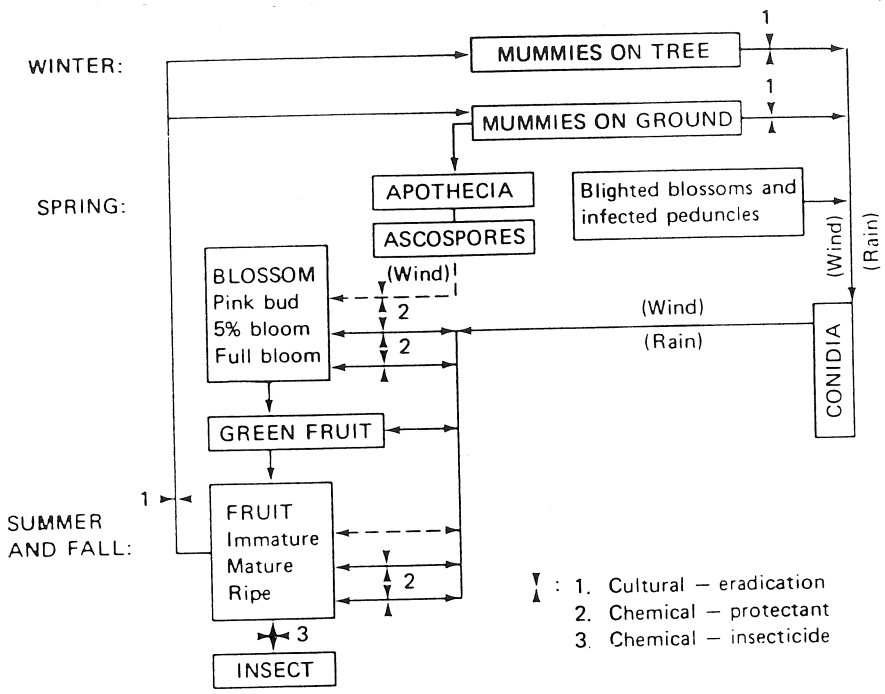

Fig. 1 — Disease cycle of *M. fructicola* on peaches and nectarines.

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 (sepais 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

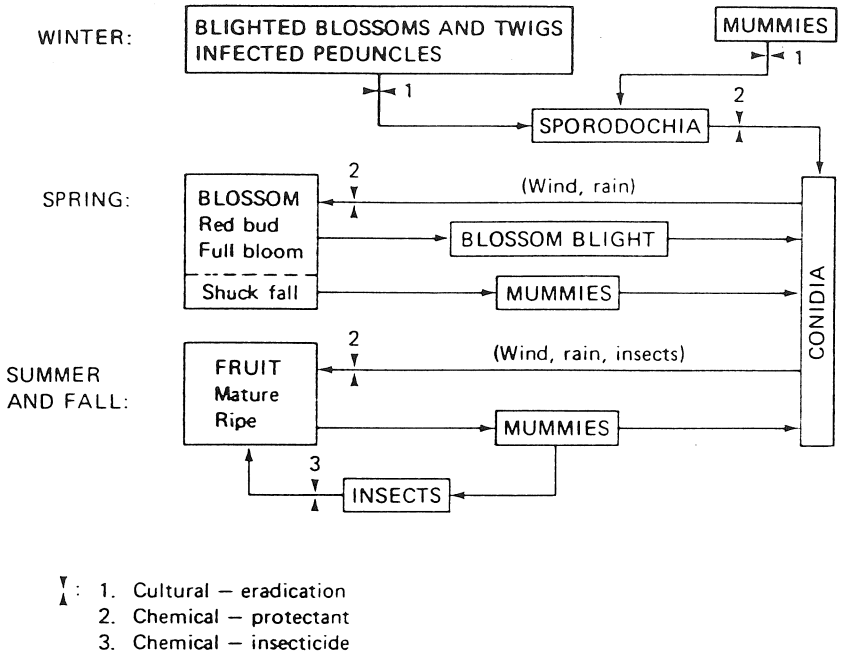

Fig. 2 — Disease cycle of *M. laxa* on apricots.

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					
6.0					79			
4.0	79							
Apricot								80 (2)
16.0							50	
10.0								
8.0				91			45	
5.0	67							
4.0				87				
2.5	27							
Prune								97
6.0					97			
4.5								
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)

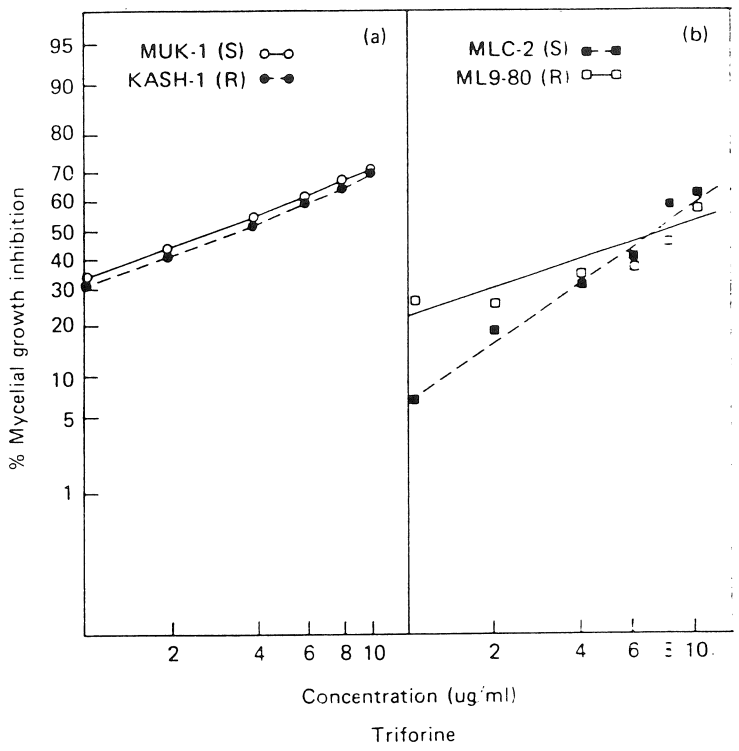

Fig. 3 — Dosage response curve of benomyl-sensitive *M. fructicola* (MUK-1) and *M. laxa* (MLC-1) compared with benomyl-resistant *M. fructocola* (KASH-1) and *M. laxa* (ML9-80) on PDA medium amended with triforine.

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

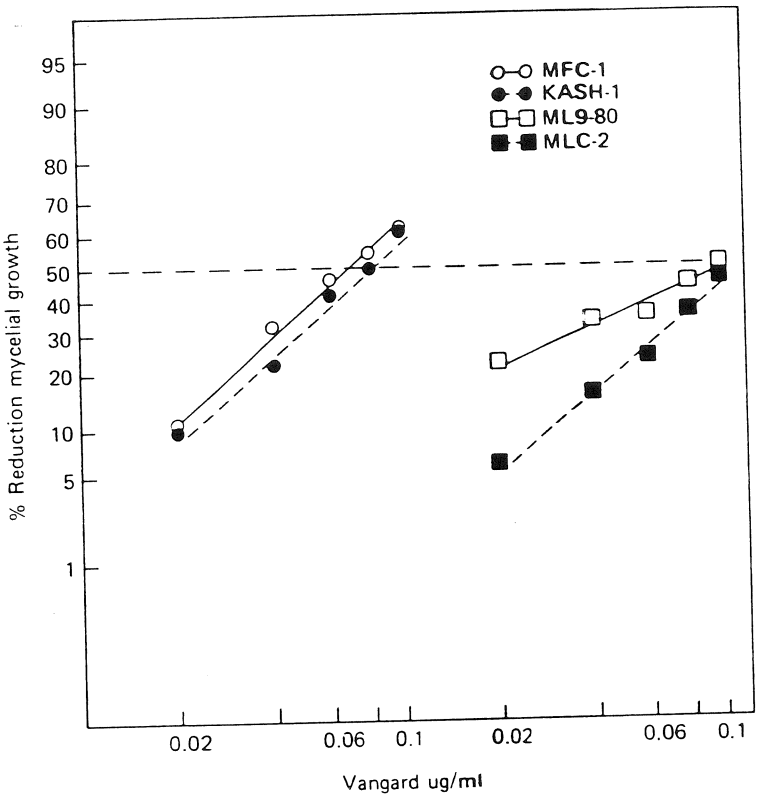

Fig. 4 — Dosage response curve of benomyl-sensitive *M. fructicola* (MFC-1) and *M. laxa* (MLC-2) compared with benomyl-resistant *M. fructicola* (KASH-1) and *M. laxa* (ML9-80) on PDA medium amended with etaconazole.

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 — 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]:		Pyrimidine[b]				Imidazole[b]:	
	Funginex		EL 228		EL 222		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					100 (2)
6.0							95	
4.0								
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 sprayer: 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

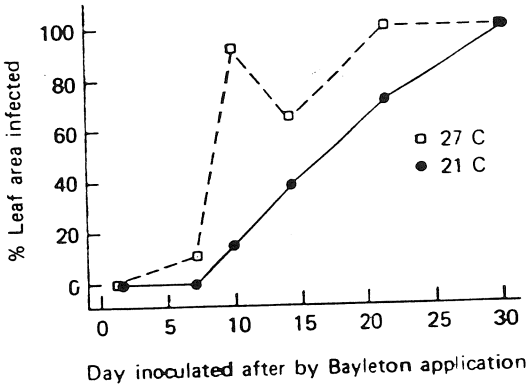

Fig. 5 — Effect of Bayleton 50W residual (150 g/ha) on colony of *U. necor* established on carignane seedlings when inoculated at intervals following treatment.

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

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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