

# Phomopsis Cane and Leaf Spot Disease of Grapevine: Effects of Chemical Treatments on Inoculum Level, Disease Severity, and Yield

JIM D. CUCUZZA, Postgraduate Research Plant Pathologist, and MARY ANN SALL, Assistant Professor, Department of Plant Pathology, University of California, Davis 95616

## ABSTRACT

Cucuzza, J. D., and Sall, M. A. 1982. Phomopsis cane and leaf spot disease of grapevine: Effects of chemical treatments on inoculum level, disease severity, and yield. *Plant Disease* 66:794-797.

A commercial vineyard (*Vitis vinifera* cv. Tokay) located in Lodi, CA, was used for a 2-yr study of Phomopsis cane and leaf spot disease of grapevines. Fungal activity was monitored by periodically examining the ability of pycnidia to exude spores when hydrated. Pycnidial levels followed a seasonal pattern, with the maximum (15 active pycnidia per square centimeter) being reached just prior to 100% budbreak. Of the three chemicals used to control *Phomopsis*, dinoseb and sodium arsenite were found to suppress pycnidial activity whereas captan did not. The three chemicals reduced disease severity by different degrees. Pycnidial levels were highly correlated ( $P \leq 0.001$ ) with disease and were used as a measure of inoculum. An increase in disease severity was associated with a reduction in the weight of the fruit per vine ( $r = -0.511$ ,  $P \leq 0.05$ ).

Additional key words: dead arm, epidemiology, *Phomopsis viticola*

Phomopsis cane and leaf spot disease of grapevines, formerly known as grape dead arm, is incited by the pycnidial fungus *Phomopsis viticola* Sacc. (7). It was first reported in California from vineyards near Sacramento in 1935 (2). Since then, the disease has been consistently present in the Central Valley and becomes sporadically severe during wet springs. It causes lesions on young shoots, necrotic spots and tattering on leaves, and occasionally a fruit rot (5,8). The fungus forms two kinds of asexual spores, alpha and beta, of which only the alpha spores cause infection (7). The spores are produced in pycnidia that are found in the outer bark tissue of infected spurs (8). When wetted by spring rains, these spores exude in a cirrus and are splash dispersed onto young shoots and leaves (8). It is reported to take 30 days for visible symptoms to develop after infections occur (8).

The two major methods of controlling the disease are selective pruning of severely infected spurs and chemical treatment (5). In California, chemicals are routinely applied each year either as late-winter, dormant applications or as foliar protectants applied in the early stages of shoot development. Two chemicals commonly used as dormant

treatments are dinoseb and sodium arsenite (4). Captan is the most commonly used foliar protectant for spring application (6).

This study was designed to assess the impact of chemical treatments on the pycnidial activity of *P. viticola* in a field situation, relate pycnidial activity to the level of disease, determine whether chemical treatments have any carry-over effect, and evaluate the effect of disease on yield.

## MATERIALS AND METHODS

**Field plot.** A commercial vineyard (*Vitis vinifera* L. cv. Tokay) located in Lodi, CA, was examined for two seasons (1979, 1980). The vineyard extended over 8.1 ha, was on sandy loam soil, and was furrow irrigated. A block of 4.0 ha was used for experimentation. The vines were 23 yr old, head trained, spur pruned, and own rooted. In years prior to this study, captan had been used for Phomopsis disease control.

**Chemical applications.** Three chemicals and an untreated control, with six replicates each, were arranged in a randomized complete block design; the same plot layout was used in both 1979 and 1980. Each subplot consisted of a single row of 32 vines. The treatments included two materials applied as dormant treatments and one that was a foliar protectant. The dormant treatments were dinoseb (Premerge, Dow Chemical Co., Midland, MI 48640) applied on 28 February 1979 and 29 February 1980 at 0.51 L a.i./100 L of water, 2,570 L/ha; and sodium arsenite (Sodite, Los Angeles Chemical Co., Los Angeles, CA 90280) applied on the same dates at 0.56 L a.i./100 L of water, 2,570 L/ha. Captan (Orthocide 50W, Chevron Chemical Co.,

San Francisco, CA 94105) applied at 100% budbreak (31 March 1979 and 21 March 1980) and again about 2 wk later (10 April 1979 and 8 April 1980) at 0.34 kg a.i./2,570 L of water per hectare was used as the foliar protectant. The chemicals were applied using a commercial air-blast sprayer. Both sides of each treatment row and the two guard rows on both sides of the treatment row were sprayed to avoid interplot interference.

In 1980, two vines in each of the rows treated with dormant chemicals were covered with plastic before application. These vines, which had been treated with sodium arsenite or dinoseb the previous year, represented two subtreatments in the 1980 experiment.

**Monitoring fungal activity using bark stripping.** Within the field plot, the activity of the fungus was periodically monitored by examining the ability of pycnidia to exude spores when hydrated. This ability was assumed to be an indication that the fungus was still physiologically active in the vine.

Strips of outer bark tissue on the current season's spurs were collected throughout the 1979 season (11 January–12 December 1979) and through March of the 1980 season (6 February–31 March 1980). At least six samples were removed from four vines in each subplot.

The bark strips were immersed in distilled water for 1 hr. After removal of excess water, they were placed in a moist chamber and incubated at room temperature (~21°C) under ambient light. Following incubation, counts of active pycnidia (those with a visible cirrus) were made using a dissecting microscope (×15). Because of the difficulty in counting inactive pycnidia (those present but without a visible cirrus), the active pycnidia were expressed on a surface-area basis. Strip surface area was determined with a Quantimet 720 Image Analyzing Computer (Cambridge Instrument Co., Inc., Monsey, NY 10952).

In March 1979, an experiment was done to determine if there were differences in the number of active pycnidia on spurs from the upper and lower halves of the vine. Using strips from untreated vines, eight to 10 strips in each of six replicates were processed as described above. Counts of active pycnidia on each strip were made at 1, 4, 7, 10, and 14 days following hydration.

Based on a portion of an M.S. thesis submitted by the first author.

Accepted for publication 9 November 1981.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

0191-2917/82/09079404/\$03.00/0  
©1982 American Phytopathological Society

**Field observations.** Disease severity in the field plot was evaluated 4 wk after the last spring rain of each season. This allowed for disease development after the last potential infection period. Eight shoots on 10 vines in each treatment row were visually rated for disease on 7 May 1979 and 15 May 1980. The shoot and three leaves proximal to the head of the vine were evaluated using the following indexes: for leaf symptoms, 0 = no infection, 1 = one to several lesions but no distortion of the leaf lamina, 2 = numerous lesions and up to 40% of the lamina tattered or distorted, 3 = as in 2 but  $\geq 40\%$  of the lamina distorted; for shoot symptoms, 0 = no infection, 1 = 0–20% of the shoot showing lesions, 2 = 20–50% of the shoot showing lesions, and 3 =  $\geq 50\%$  of the shoot with coalescing lesions.

In both years, 20 leaves judged to be in the separate index categories were collected, and the number of lesions and surface area of each leaf were measured. The average number of lesions per square centimeter for each group was used as the weighting factor for the leaf index categories. Thus, the weighting factors differed slightly in 1979 and 1980. The shoot index number was used as the weighting factor for the shoots.

Quantitative disease severity (ds)

values were derived using a weighted average of both leaf and shoot symptoms computed from the following equation:

$$ds = \frac{\sum_{i=1}^4 f_i w_i}{\sum_{i=1}^4 w_i}$$

where  $f_i$  is the frequency of leaves in the  $i^{\text{th}}$  category and  $w_i$  is the weighting factor for the respective category.

At the time of normal winery harvest (29 August 1979 and 2 September 1980), the fruit yield for each treatment was determined. All clusters on each vine rated for disease severity were harvested and weighed. The total fruit weight (in kilograms per vine) was analyzed using Duncan's multiple range test after a significant  $F$  test to determine differences in yield between the various treatments.

## RESULTS

**Monitoring fungal activity using bark stripping.** During the 2-wk incubation of hydrated bark strips, there was a rapid increase in the number of active pycnidia for the first 8 days, after which few new cirri appeared (Fig. 1). More active pycnidia were present on strips from the lower half of the vine ( $x = 17.0 \pm 8.0$ ) than from the upper half ( $x = 11.5 \pm 3.4$ ); however, this difference is not statistically

significant. Therefore, sampled bark strips were randomly chosen from spurs throughout the vine.

A period of 5 days of incubation was chosen for subsequent samples because it approached the point where no new active pycnidia appeared and the difference between the number of active pycnidia observed on the upper and lower halves of the vine was minimal. Also, longer incubation led to contamination

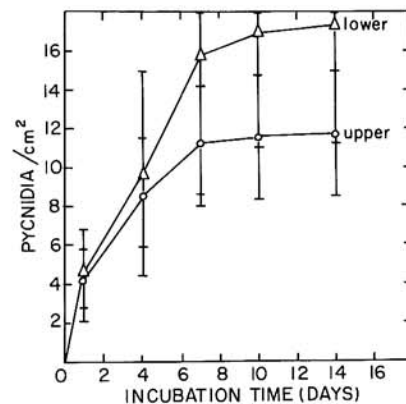


Fig. 1. Number of active pycnidia per square centimeter on bark strips from the upper and lower halves of the vines after incubation in moist chambers. Each point represents the average of six replicates with eight to 10 strips each.

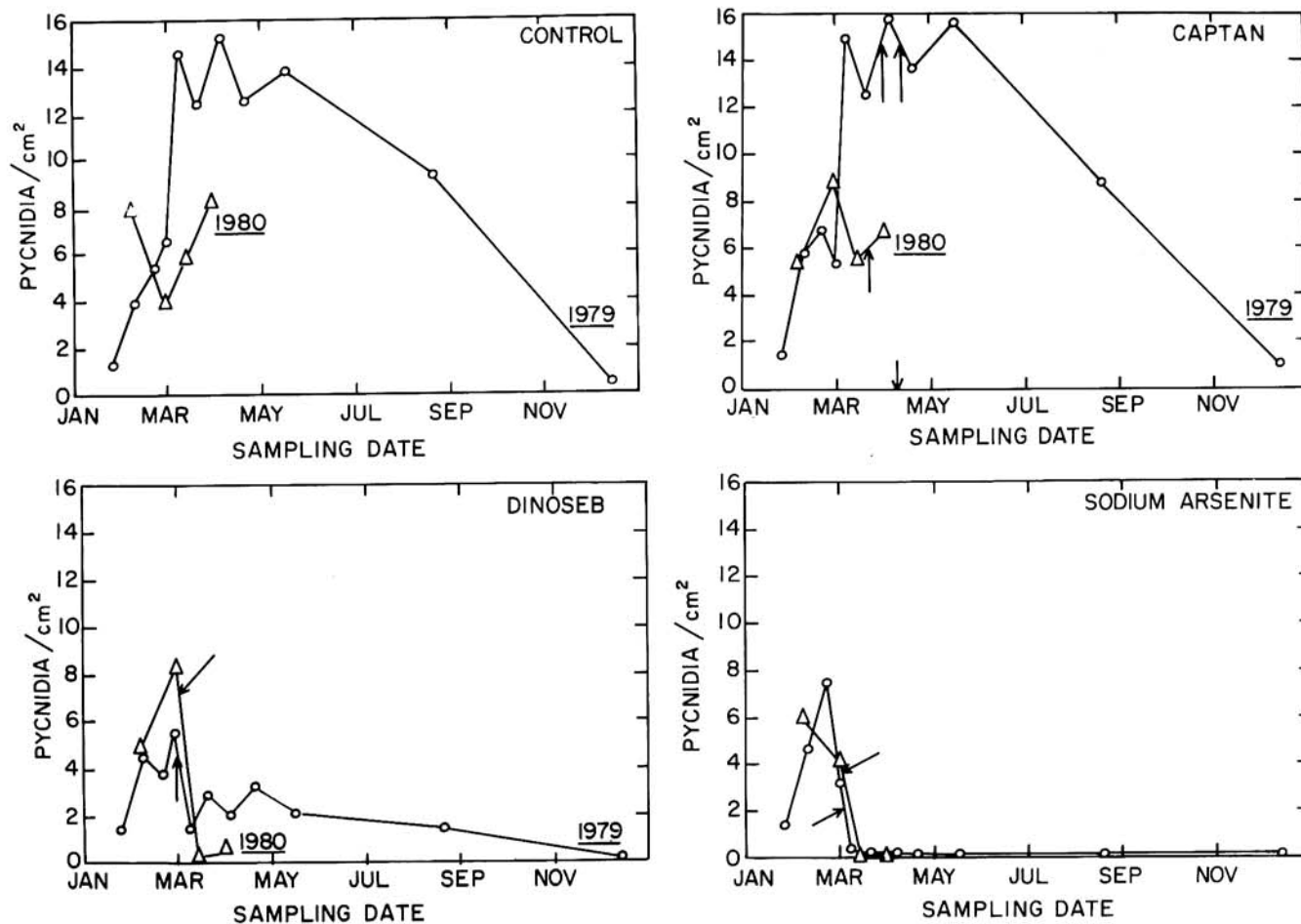


Fig. 2. Effects of chemical treatments on the levels of active pycnidia per square centimeter on bark strips sampled from the field plot in 1979 and through March of 1980. Each point represents the mean of six replicates with five to 10 sample strips in each. Arrows show the date of chemical treatment.

by other fungi, which interfered with counting.

Pycnidial levels in 1979 followed a seasonal pattern (Fig. 2). The levels of active pycnidia increased dramatically early in the season and reached a maximum (ca. 15 pycnidia per square centimeter) just prior to 100% budbreak (31 March 1979). These levels remained high throughout the spring and decreased

as the season progressed. With the onset of winter, the number of active pycnidia was comparable to the level seen at the beginning of the season. A comparison of 1979 and 1980 indicates that in the spring of 1980, pycnidial activity was approximately half of that present at the same time in 1979.

There were significant differences in pycnidial activity in both years following

chemical application (Fig. 2). Dinoseb and sodium arsenite treatments resulted in a substantial decrease (80–100%) in the number of active pycnidia. The reduction in the number of active pycnidia occurred immediately following application, and the level of activity remained low throughout the rest of the season. The pycnidial activity in the captan plots was not statistically different from that in the control plots throughout the season.

The initial samples taken in the 1980 season showed no significant difference in the pycnidial activity in all treatments. Although the dormant treatments were effective eradicants in 1979, the pycnidial activity in these plots increased to a level comparable to the controls during the time prior to treatment in 1980. This indicates that the eradicator effect of these dormant treatments was only effective for one season.

**Field observations.** In both years, chemical treatments differed significantly in their ability to control disease (Table 1). Sodium arsenite caused the greatest reduction in disease severity, followed by dinoseb and captan, when compared with the control plots. In 1980, dinoseb and captan did not differ in disease control, whereas dinoseb was significantly more effective in 1979.

In 1980, there was a significant increase in the level of disease in plots to which dormant treatments were applied in 1979 but omitted in 1980. The disease severity of dinoseb plots treated only in 1979 did not differ significantly from the untreated controls. In plots where sodium arsenite was applied only in 1979, the disease severity was significantly less than in the controls but was significantly higher than that found in any other treatment.

The disease severity in all treatments was higher in 1980 than in 1979. In 1979, the combined leaf and shoot rating averaged 1.1 in the untreated control plots. The maximum possible rating for that year was 4.9. However, in 1980 the average rating was 3.0 out of a maximum possible of 5.6.

Using pycnidial activity as a measure of inoculum, a disease versus inoculum curve was generated (Fig. 3). Correlations between disease severity and pycnidial levels, excluding the captan-treated plots, were made for all available dates after treatment. The pycnidial activity on the date most highly correlated with disease severity was used as the measure of inoculum for the curves (4 April 1979:  $r = 0.873^*$ ,  $P \leq 0.001$ ; 31 March 1980:  $r = 0.860^*$ ,  $P \leq 0.001$ ).

Fruit weight per vine decreased with increasing disease severity (Table 1); however, the differences are not significant at  $P \leq 0.05$  ( $P = 0.09$ ). In 1979, vines treated with captan, dinoseb, and sodium arsenite yielded 3, 6, and 10% more fruit than untreated vines, respectively. When disease severity was higher in the following year, the same treatments

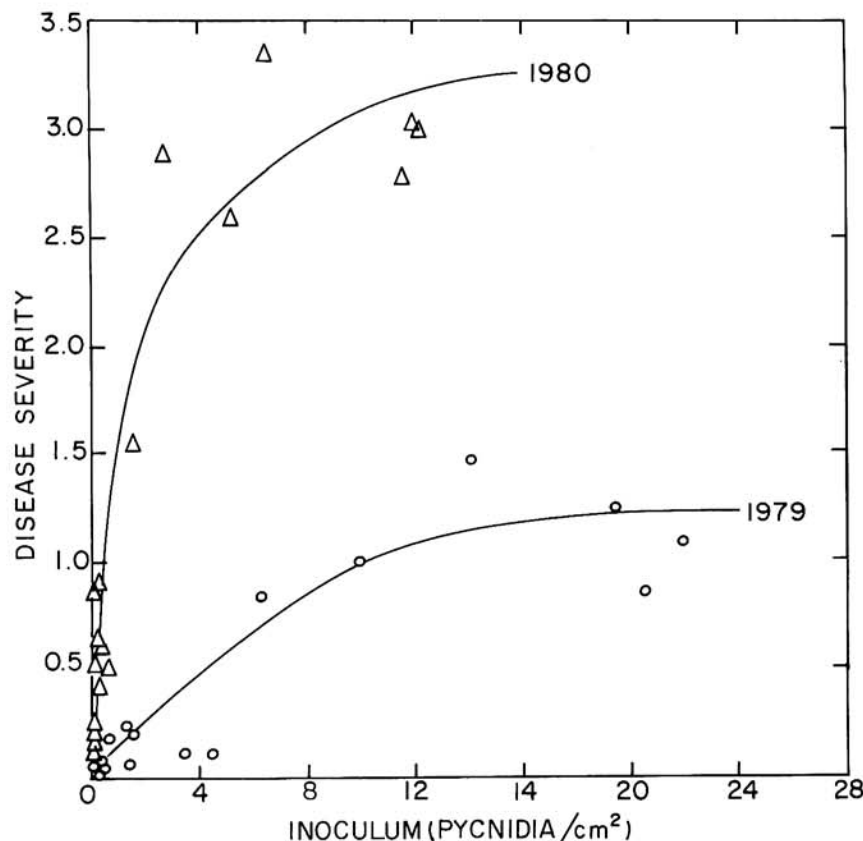
**Table 1.** Disease severity and fruit yield of Tokay grapevines grown near Lodi, CA, and treated for Phomopsis cane and leaf spot disease

Treatment <sup>x</sup>	Disease severity <sup>y</sup>		Fruit/vine (kg)	
	1979	1980	1979	1980
Sodium arsenite	0.1 a	0.3 a	23.9	16.5
Dinoseb	0.2 b	0.8 b	23.2	16.4
Captan	0.9 c	1.1 b	22.4	15.6
Control	1.1 d	3.0 d	21.8	14.4
Sodium arsenite (1979 only)	...	2.3 c	...	...
Dinoseb (1979 only)	...	2.7 cd	...	...
LSD 0.05	(0.12)	(0.41)	...	...
MAX <sup>z</sup>	(4.9)	(5.6)	...	...

<sup>x</sup>Sodium arsenite applied on 28 February 1979 and 29 February 1980 at 0.56 L a.i./100 L of water, 2,570 L/ha; dinoseb applied on 28 February 1979 and 29 February 1980 at 0.51 L a.i./100 L of water, 2,570 L/ha; captan applied on 31 March and 10 April 1979 and on 21 March and 8 April 1980 at 0.34 kg a.i./2,570 L of water per hectare.

<sup>y</sup>Combined leaf and shoot index, where leaf index is weighted by average number of lesions per square centimeter and shoot index is weighted by 0, 1, 2, or 3 for ascending severity; see text for details. Numbers followed by the same letter do not differ significantly according to Duncan's multiple range test ( $P = 0.05$ ).

<sup>z</sup>Maximum possible severity index based on maximum observed number of lesions per square centimeter on leaves in each year plus the highest index of shoot infection.



**Fig. 3.** Disease severity (combined leaf and cane, weighted average, infection rating) as a function of the level of inoculum (active pycnidia per square centimeter) of *Phomopsis viticola* in 1979 and 1980. Data from a field experiment conducted in a commercial vineyard (cv. Tokay) near Lodi, CA.

resulted in an 8, 14, and 15% increase, respectively.

## DISCUSSION

The results of the field study showed differing effects of these chemicals on the activity of *P. viticola*. Captan had no effect on pycnidial activity but did reduce disease severity when applied at the proper time—just after the onset of shoot growth. The dormant treatments, dinoseb and sodium arsenite, were apparently effective eradicates, in that the numbers of active pycnidia per square centimeter were substantially reduced following treatment. These findings corroborate the preliminary study of Hewitt (3) concerning sodium arsenite and the more elaborate work of Gartel (1) with dinoseb. Vines that had not had dormant treatments applied during the second year had high levels of disease; thus, the eradicate effect of these chemicals was not sufficient for a carry-over of disease control into the next season. These results indicate that treatment decisions need to be made on a yearly basis if the disease is to be controlled through the use of chemicals.

Willison et al (9) suggested that the level of disease the previous year is indicative of the potential inoculum for the current season. The present study does not support this theory. Although significantly different levels of disease were related to the chemical treatments in 1979, no significant differences in inoculum levels were found in these plots in 1980. The fact that pycnidial activity increased, even with minimal disease

severity the previous year, was probably the result of the vigorous saprophytic nature of *P. viticola*. The limited infection in 1979 was still enough to allow for colonization of the spurs at some time prior to the spring of 1980.

From the disease versus inoculum curves (Fig. 3), it appears that inoculum was not the sole limiting factor for disease development. Although within each year a reduction in inoculum by chemical treatment decreased the severity of disease, a comparison between years shows that there must have been overriding factors for disease development. In 1980, factors governing infection and disease development provided for more than a twofold increase in the severity of disease with only half the amount of inoculum. Weather records for 1979 and 1980 indicate only slight differences in temperature and humidity. However, the rainfall patterns differed, with 1980 having heavier rains later in the spring season.

Although highly significant differences in disease levels among treatments were found in both years, fruit yields per vine were not statistically different at  $P \leq 0.05$ . However, when all treatments were combined, disease severity was sometimes significantly correlated ( $r = -0.359$  n.s., 1979;  $r = -0.447^*$ , 1980;  $r = -0.511^{**}$ , 1979 + 1980) with yield. Further investigations are needed to determine whether the weakness of the relationship between measures of disease and yield is related to a low potential for damage by this pathogen, our measurement of

disease severity, or inherent variability in fruit weights with inadequate sampling.

The field experiment has shown that treatments applied either during dormancy or after the onset of shoot growth can be effective in reducing the severity of symptoms and possibly yield loss attributable to Phomopsis cane and leaf spot disease. However, no carry-over from one year to the next was observed with either type of treatment.

The results of this project provide managers with a more rational basis for treatment decisions.

## ACKNOWLEDGMENTS

We wish to thank William Moller, Frank Schick, James Kissler, and James Peterson for important contributions to this project.

## LITERATURE CITED

1. Gartel, W. 1974. Untersuchungen über den Einfluss von Fungiziden auf das Ausstossen von Sporen durch die Fruchtkörper des Erregers der Schwarzfleckenkrankheit (*Phomopsis viticola* Sacc.). Weinberg Keller 21:267-280.
2. Hewitt, W. B. 1935. Dead arm disease of grapes in California. Plant Dis. Rep. 19:309-310.
3. Hewitt, W. B. 1947. Sodium arsenite, a promising control of dead-arm of grapes. Phytopathology 37:362.
4. Hewitt, W. B. 1951. Grape dead-arm control. Plant Dis. Rep. 35:142-143.
5. Moller, W. J., Kissler, J. J., and Leavitt, G. M. 1979. Phomopsis cane and leaf spot of grapes. Univ. Calif. Ext. Publ. 21066. 7 pp.
6. Pine, T. S. 1957. The use of captan in the control of the dead-arm disease of grapes. Plant Dis. Rep. 41:822-823.
7. Pine, T. S. 1958. Etiology of the dead-arm disease of grapevines. Phytopathology 48:192-196.
8. Pine, T. S. 1959. Development of the grape dead-arm disease. Phytopathology 49:738-743.
9. Willison, R. S., Chamberlain, G. C., Townshend, J. L., and DeRonde, J. H. 1965. Epidemiology and control of dead-arm of grapes. Can. J. Bot. 43:901-914.