Time of Infection and Control of Phomopsis Fruit Rot of Grape

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ABSTRACT

Pscheidt, J. W., and Pearson, R. C. 1989. Time of infection and control of Phomopsis fruit rot of grape. Plant Disease. 73:829-833.

Phomopsis viticola, the cause of Phomopsis cane and leaf spot, can incite a fruit rot of grapes in addition to necrotic lesions of the canes and leaves. Fruit rot has occurred in New York during years with unusually high rainfall (>100 mm) during bloom. Inoculations of *Vitis labrusca* 'Concord' clusters at or shortly after bloom using 1×10^7 alpha spores per milliliter resulted in 16–30% fruit rot at harvest. Inoculations at earlier or later growth stages did not result in fruit rot significantly higher than that in nontreated controls. Berries inoculated in the laboratory were progressively less susceptible to infection and colonization as they aged from pea-size to ripe stages of growth (Eichhorn and Lorenz growth stages 30 to 38). Wounding of the fruit was not a prerequisite for infection. Infection and symptom development occurred faster on berries incubated with the pedicel in contact with inoculum than on those with the stylar end in contact with inoculum. Latent infections of fruit and rachis were detected using a paraquat dip technique. Two applications of mancozeb during bloom significantly reduced fruit rot and rachis lesions on cv. Delaware during the 1987 growing season. We suggest that protectant fungicides applied during bloom (Eichhorn and Lorenz growth stage 25) will significantly improve control of fruit and rachis infections.

Phomopsis cane and leaf spot, caused by *Phomopsis viticola* (Sacc.) Sacc., is an economically important disease of grapes in many viticultural regions of the world (3,4,7,15,16,18). The cane and leaf spot phase of this disease has been studied in detail (3,15,16,18), but little information exists on the fruit rot phase. Gregory (8) first described the fruit rot phase, noting that brown necrotic lesions with numerous pycnidia developed on infected berries. Eventually, the entire berry was involved and shriveled into a mummy. These symptoms developed near harvest. Gregory (8) determined that ripe *Vitis labrusca* L. 'Niagara' fruit showed symptoms within 18 days after inoculation. Lal and Arya (10) reported that ripe *V. vinifera* L. 'Thompson Seedless' berries could not be infected unless they were wounded before inoculation.

The objective of this study was to determine when fruit became infected and how to control the fruit rot phase of this disease.

MATERIALS AND METHODS

Historical survey. Extension newsletters from 1958 to 1986 were reviewed to determine which years Phomopsis fruit rot did or did not occur in the western New York grape region. The newsletters, *Vineyard Notes*, were based on the observations of the county extension grape specialist who regularly visited many of the vineyards in this area. The National Weather Service records from Fredonia, New York, were examined to determine if rainfall was related to the occurrence of fruit rot.

Yield loss survey. Unusually high fruit loss occurred in a commercial Concord vineyard heavily infected with Phomopsis cane and leaf spot starting in mid-August 1986. A preliminary survey was initiated to quantify the amount of fruit drop and disease severity. The vineyard had a mechanically pruned, top-wire cordon training system.

Eight sets of three vines were randomly selected within a 1-ha area. A 3 × 6 m sheet of plastic was placed under each set of vines on 24 September 1986. Each week until harvest, the berries that had fallen onto the plastic were gathered and weighed. The plastic was then swept clean of all debris. Disease severity was determined on 2 October 1986 by rating the rachis of 15 clusters on each of three vines using the Barratt-Horsfall rating system. Plots were mechanically harvested on 21 October 1986. Total yield loss was calculated as the ratio between the total crop weight that had fallen onto the plastic and the crop weight obtained by the mechanical harvester.

Another method of estimating yield loss was used on adjacent vines, since berries were randomly distributed within a 0.5-m zone on either side of the trellis after harvest. The number of berries were counted within a square wire frame (930 cm^2) randomly placed on the ground below the canopy. The wire frame was placed under every sixth vine in four 200-m rows. The average number of berries within the wire frame was calculated and expressed as the number of berries lost per hectare for each row. The average berry weight was determined before harvest on 19 September 1986. Total yield loss was calculated as the ratio between the weight of berries on the ground per hectare (berries/ha × grams/berry) and the crop weight obtained by the mechanical harvester.

Inoculation of grape clusters. Hand-pruned umbrella Kniffin trained Concord grapevines were inoculated at Fredonia, New York, during 1986 and 1987 and at Geneva, New York, during 1987. A 6 × 30.5 m sheet of heavy-duty (6-mil) clear polyethylene film was stretched over PVC pipe arches to make tents that covered 16 vines from budbreak to harvest during the 1987 growing season at Fredonia. The plastic sheet protected vines from rainfall and thus reduced contamination from natural inoculum. A greenhouse whitewash solution was sprayed onto the plastic tents to reduce heat buildup during hot, sunny weather. Vines at Geneva were not covered with plastic.

Dormant Concord canes were collected in April 1986 and placed in a moist chamber for 48 hr at room temperature. Cirri from sporulating pycnidia of *P. viticola* were placed on potato-dextrose agar (PDA) and incubated for 1 wk at room temperature. Cultures were maintained on PDA slants and stored at 4 C. Several isolates were transferred from slants to petri dishes of PDA as needed and incubated for 1 wk at room temperature. Dishes were flooded with sterile distilled water to release and suspend most of the spores. The suspension was adjusted with sterile distilled water to a concentration of 1×10^5 alpha spores per milliliter in 1986 and 1×10^7 alpha spores per milliliter in 1987. Spore suspensions were sprayed onto clusters at various stages of development until runoff. Each cluster was enclosed within a plastic bag containing a small piece of moist paper towel. A white paper bag was stapled over the plastic bag to reduce heat buildup from sunlight. Only one cluster per shoot was inoculated. All bags were removed 24 hr after inoculation.

Cluster and grapevine development were evaluated using several methods, including length of shoots, Eichhorn and Lorenz growth stages (E and L) (1), and soluble solids (ss) as measured with a refractometer (Bausch & Lomb). Clusters at Fredonia were inoculated at bloom (11 June, E and L 25) and at

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Accepted for publication 11 April 1989 (submitted for electronic processing).

shatter (25 June, E and L 27) in 1986. Clusters at Fredonia were inoculated at 12.7-cm shoot growth (19 May, E and L 10), bloom (2 June, E and L 25), and *véraison* (19 August, 9% ss, E and L 35) in 1987. Clusters at Geneva were inoculated at bloom (8 June, E and L 25), pea-size (23 June, 10–15 mm diameter, E and L 30), *véraison* (18 August, 9% ss, E and L 35), and 13% ss (9 September) in 1987. Additional clusters were tagged as noninoculated controls. On each date, 30–100 clusters were inoculated or used as controls.

Each cluster was placed in a separate plastic bag at harvest and stored at 1 C until examined. The number of clusters with rachis lesions and the number of infected berries per cluster were determined within 2 wk of harvest.

Reisolation. A total of 100 inoculated berries with symptoms of fruit rot were surface-sterilized and plated on PDA. Berry sections (5 mm^2) were dipped into 0.5% sodium hypochlorite for 2 min, then into sterile distilled water for 2 min, before being placed on PDA. Berry sections were observed after 1 wk of incubation at room temperature.

Inoculation of individual berries. Concord clusters at different stages of development were harvested and transported to the laboratory in 1987. Clusters were surface-sterilized as described above, and individual berries to be inoculated were aseptically cut from the rachis with the pedicel intact. Pea-sized berries (10–15 mm diameter, E and L 30) were obtained from Geneva on 24 June; all other clusters were obtained from under the plastic tents at Fredonia. Clusters were harvested on 17 July at 4% ss (green berries), on 20 August at *véraison* (9% ss, E and L 35), and on 25 September at 15% ss (ripe berries, E and L 38).

Inoculum of *P. viticola* was prepared as described above. A suspension of 1×10^5 alpha spores per milliliter was added to the wells of tissue culture cell wells (Corning Glass Works, Corning, NY). Sterile water was used in one row of wells to serve as a noninoculated control. Surface-sterilized berries were positioned with either the pedicel or the stylar end into the inoculum. The 96-well plates (with 6.4-mm wells) were used for pea-sized berries, and the 24-well plates (with 16-mm wells) were used for all other berries. All plates were enclosed in clear plastic boxes lined with moist paper towels and incubated at room temperature and light throughout the duration of the experiment.

Pea-sized berries and green berries (4% ss) were incubated in constant contact with the inoculum. Another set of green berries, *véraison* berries, and ripe berries were incubated in contact with the inoculum for 24 hr, then transferred to new, dry, sterile cell well plates. A gas chromatograph syringe was used to inject a set of berries from each growth stage with 5 μl of the spore suspension. A small droplet of inoculum remained on the wound after injection and was gradually absorbed over 8 hr. Berries were examined periodically for development of necrosis and pycnidia characteristic of Phomopsis fruit rot.

Detection of latent infections. Symptomless Concord clusters were collected from naturally infected vines with severe shoot infection at Geneva on 11 August 1987. The rachis, pedicel, and berries were green, and the berries had 4% ss. Two berries and portions of the rachis from six clusters were dipped into 95% ethanol for 5–10 sec, 0.5% sodium hypochlorite for 2 min, sterile distilled water for 1–2 min, and a 1:40 dilution of 29.1% paraquat dichloride for 1 min (5). A duplicate set of berries were surface-sterilized but not dipped into the paraquat solution. Berries were placed in a clear plastic box and incubated at room temperature for 2 wk.

Fungicide trials. Concord and Delaware grapevines at different stages of development were sprayed with fungicides to determine if the fruit rot phase of this disease could be controlled. Three field trials were conducted during 1986 and 1987.

In 1986, a hooded boom hydraulic sprayer (100 gal/acre) was used to spray a 1.2-ha block of hand-pruned umbrella Kniffin trained Concord vines near Portland, New York, with captan (Captan 50W) at 1.12 kg a.i./ha. The following treatments were arranged in a randomized complete block design and each block (4 rows × 9 vines) was replicated four times: 1) untreated before bloom and from *véraison* to harvest; 2) captan at 2.5- and 12.7-cm shoot growth, 30 April and 13 May, respectively (prebloom); 3) prebloom (30 April and 13 May) and *véraison* (24 August); 4) prebloom, *véraison*, and 1 and 3 wk before harvest, 24 September and 8 October, respectively (preharvest); 5) prebloom and preharvest; 6) preharvest alone; and 7) *véraison* alone. The entire vineyard was sprayed with folpet 50W (1.12 kg a.i./ha) at 50% bloom (9 June) and postbloom (22 June), with captan (1.12 kg a.i./ha) on 7 July, and with maneb plus zinc (Dithane M-22) (1.6 kg a.i./ha) on 10 August. On 13 October, 30 clusters per plot were harvested. In this and subsequent fungicide trials, the percentage of clusters with rachis lesions and the percentage of berries with fruit rot were determined.

A 1.2-ha block of mechanically pruned, Geneva double-curtain trained Delaware vines near Portland, New York, was sprayed weekly using an air-blast sprayer (100 gal/acre) with captan (1.12 kg a.i./ha) for up to 4 wk before harvest in 1986. The following treatments were arranged in a randomized complete block design and each block (5 rows × 15 vines) was replicated three times: 1) weekly applications initiated on 21 August (four sprays); 2) weekly applications initiated on 28 August (three sprays); 3) two applications on 3 and 9 September; 4) one application on 9 September; and 5) untreated for 5 wk before harvest. The entire vineyard was sprayed with captan (1.12 kg a.i./ha) at 2.5-cm shoot growth (8 May), at bloom (10 June), at postbloom (18 June), and on 7 July, and with maneb plus zinc (1.6 kg a.i./ha) on 15 August. On 17 September, 30 clusters per plot were harvested for disease evaluation.

A similar block of Delaware vines was treated in 1987 using an air-blast sprayer (100 gal/acre). The following treatments were arranged in a completely randomized design where each treatment was applied to a 6 row × 15 vine block: 1) untreated all year; 2) mancozeb (Dithane M-45) (3.6 kg a.i./ha) applied at 2.5- and 12.7-cm shoot growth (8 May and 18 May, respectively); 3) mancozeb applied 1 wk before bloom (1 June) and at bloom (9 June); 4) maneb plus zinc (1.6 kg a.i./ha) applied at *véraison* (4 August); 5) maneb plus zinc salt applied when berries were at 10% ss (11 August); and 6) the grower's schedule of captan (Captan 80W) (2.2 kg a.i./ha) applied at 2.5-, 17.8-, and 30.5-cm shoot growth (29 April, 15 May, and 28 May, respectively), at 10% bloom (4 June), at postbloom (13 June), and on 3 July. On 14 September, 50 clusters per plot were harvested for disease evaluation.

Insecticides and herbicides were applied according to standard practices for the Lake Erie grape region (2). Each vineyard was on a standard 2.75 × 2.44 m spacing between rows and vines, respectively.

RESULTS

Historical survey. Phomopsis fruit rot caused significant losses to New York grape production in 1972 when Hurricane Agnes struck the state during bloom. A review of extension newsletters revealed that the presence or absence of Phomopsis fruit rot was noted in only 5 yr (Table 1). In each of the 3 yr with fruit rot, rainfall was unusually high (>100 mm) during bloom. The severity of cane symptoms was medium in one year and high in the other two.

Yield loss survey. Some crop loss had already occurred 10 days before placement of the plastic in the vineyard. Many rachis lesions had expanded or girdled much of each cluster such that 47.3% of the rachis area was necrotic or dehydrated. Whole clusters (including rachis, subrachis, and cap stems) and portions of clusters had fallen onto the plastic before harvest. Whole shoots girdled by cane lesions were occasionally found on the plastic, sometimes with two or three clusters still attached.

A total loss estimate of 35.7% was

Table 1. Historical survey of Phomopsis fruit rot[y]

Year	Severity of cane symptoms	Fruit rot	Rainfall during bloom (mm)[z]
1972	Medium	+	198.9
1983	Low	−	55.9
1984	High	+	144.3
1985	Low	−	36.8
1986	High	+	139.2

[y]Based on a review of extension newsletters (*Vineyard Notes*) published from 1958 to 1986.
[z]During a 2-wk period from 1 wk before 90% bloom through 1 wk after 90% bloom.

calculated using the plastic tarps. Fruit loss onto the ground started 14 September. About 3.4 t/ha had been lost when local processors began accepting grapes on 1 October. About 6.1 t/ha had been lost by the time of harvest and another 1.1 t/ha was lost during the harvest operation, for a total loss of 7.2 t/ha. A loss of $1,270/ha was calculated using the market price of $176.4/t for Concord grapes. A loss estimate of 37.7% was obtained using the method of counting fallen berries within a wire frame after harvest.

Fruit loss also was attributed to factors other than Phomopsis cane and leaf spot. Downy mildew, caused by *Plasmopara viticola* (Berk. & Curt.) Berl. & de Toni, was estimated as affecting less than 10% of the berries. Berries infected with downy mildew fell from the cluster, and frequent rainstorms (totaling 267 mm rainfall) before harvest disturbed vines and also caused fruit to fall.

Inoculation of grape clusters. The first symptoms of fruit rot and rachis infection appeared on 30 July 1987 prior to *véraison* on clusters inoculated at bloom at Fredonia. Infected fruit showed a brown discoloration that enveloped the stylar or pedicel end of the berry. Berries with advanced symptoms had a rough texture caused by subsurface pycnidia. The percentage of berries with fruit rot on clusters inoculated at bloom increased to 5% by 4 September 1987. Symptoms of fruit rot did not occur until early September at Fredonia in 1986 or at Geneva in 1987. These symptoms first appeared only on a few berries of clusters inoculated at bloom.

Rachis lesions were characterized by sunken black areas that caused the rachis to become brittle and easily broken. Some rachis lesions had girdled portions of the cluster, resulting in the dehydration of berries distal to the infection. Rachis lesions appeared 1 wk before harvest at Fredonia in 1986 and at Geneva in 1987.

Clusters inoculated at bloom had the most fruit rot at harvest (Table 2). Inoculations at 12.7-cm shoot growth, at *véraison* (9% ss), or during ripening (13% ss) did not result in significantly more fruit rot than in noninoculated clusters.

Table 2. Development of fruit rot and rachis lesions after inoculation of Concord clusters at different stages of growth with alpha spores of *Phomopsis viticola*

	Berries with fruit rot (%)			Clusters with rachis infection (%)		
	Fredonia		Geneva	Fredonia		Geneva
Growth stage	1986	1987[w]	1987	1986	1987[w]	1987
Noninoculated control	2.1 b[x]	0.4 b	3.3 c	47[y]	0[y]	58[y]
Shoots 12.7 cm	...[z]	0.5 b	...	...	81	...
90% Bloom	6.4 a	30.1 a	16.3 a	48	97	100
Pea-sized	8.9 a	...	14.0 b	56	...	97
Véraison (9% soluble solids)	...	1.0 b	4.1 c	...	23	66
Ripe (13% soluble solids)	...	...	2.5 c	...	...	56

[w]A 6 × 30.5 m sheet of heavy-duty (6-mil) clear polyethylene film was stretched over PVC pipe arches to cover vines from budbreak to harvest.
[x]Treatment means followed by the same letter are not significantly different according to Fisher's protected LSD procedure at the 5% level.
[y]True population means (no statistical analysis necessary).
[z]Not inoculated.

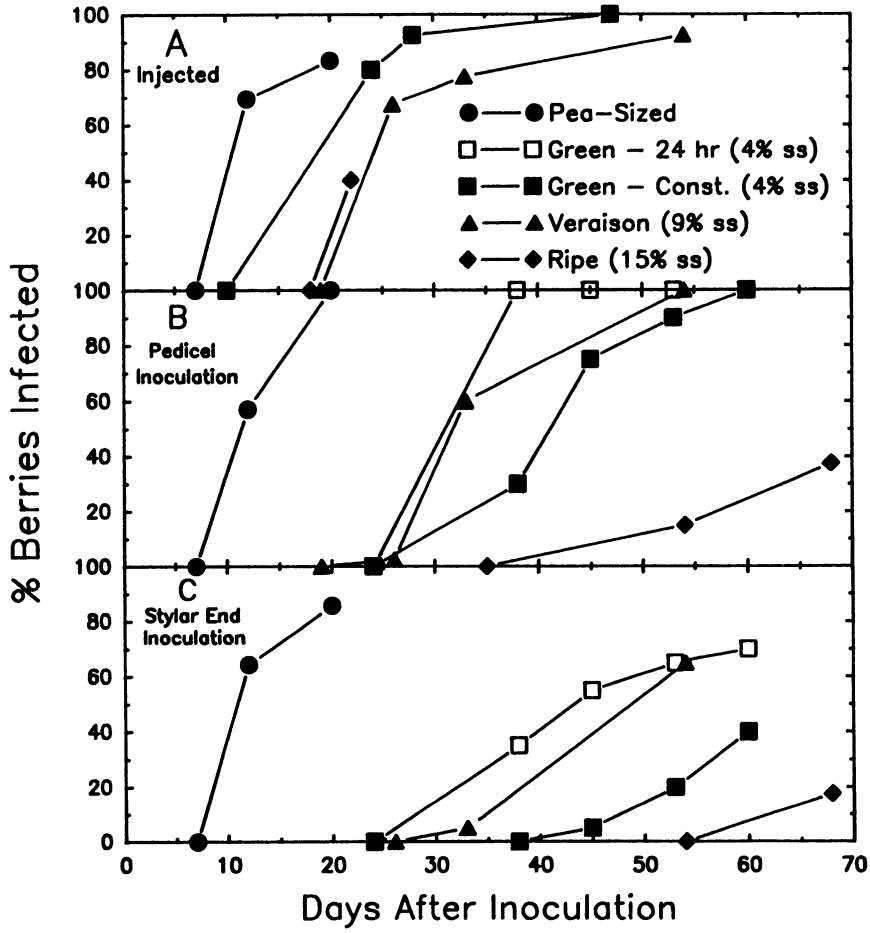

Fig. 1. Phomopsis fruit rot development on berries **(A)** injected with a 5-µl suspension of 1×10^5 alpha spores per milliliter, **(B)** in which the pedicel was submerged in a similar suspension of spores, and **(C)** in which the stylar end was submerged in a similar suspension of spores. Pea-sized and green (green-const.) berries were incubated in constant contact with the inoculum. Another set of green (green-24 hr) berries, *véraison*, and ripe berries were incubated in contact with the inoculum for 24 hr before being transferred to sterile cell well plates for incubation.

Inoculations of pea-sized berries, however, did result in more fruit rot than in noninoculated clusters.

The number of clusters with rachis lesions was highest for clusters inoculated at bloom or pea-sized berries. Clusters inoculated at 12.7-cm shoot growth also had a high incidence of rachis lesions. Since these were true population means, the incidence data were not analyzed statistically.

Reisolation. *P. viticola* was reisolated from 95 of the 100 berries with symptoms of fruit rot. Colony morphology, growth habit, size and structure of pycnidia, and shape and size of alpha spores were identical to the same characters of the isolates used for inoculation. No fungal isolates were recovered from the other five berries, but three of these berries bore pycnidia similar to those of *P. viticola*.

Table 3. Incidence of Phomopsis fruit rot on Concord grapevines in 1986[w]

Fungicide treatment[x]	No. of sprays	Berries with fruit rot (%)	Clusters with rachis infection (%)
Untreated	0	5.1[y]	80 a[z]
PB	2	3.4	40 b
PB, V	3	2.9	43 b
PB, V, PH	5	3.7	43 b
PB, PH	4	2.9	42 b
PH	2	5.6	72 a
V	1	4.0	64 a

[w] Entire vineyard was uniformly sprayed with folpet during bloom.
[x] PB = prebloom (captan applied at 2.5- and 12.7-cm shoot growth), V = *véraison* (captan applied), PH = preharvest (captan applied at 1 and 3 wk before harvest).
[y] Treatment means were not significantly different according to Fisher's protected LSD procedure at the 5% level.
[z] Treatment means followed by the same letter are not significantly different according to Fisher's protected LSD procedure at the 5% level.

Inoculation of individual berries. Each type of berry injected with spores of *P. viticola* developed symptoms of fruit rot within 30 days (Fig. 1A). Berries developed a brown discoloration and then numerous black pycnidia that eventually sporulated. Pea-sized berries and green berries (4% ss) rotted more quickly than *véraison* (9% ss) and ripe (15% ss) berries.

The development of fruit rot symptoms was slowest when berries were inoculated through the pedicel or stylar end, except for pea-sized berries (Fig. 1B and 1C). Most of the pea-sized berries developed symptoms 20 days after inoculation through either the pedicel or stylar end. Green berries (4% ss) incubated with either the pedicel or stylar end in constant contact with the inoculum rotted slower than berries in contact with the inoculum for only 24 hr.

All of the green (4% ss) and *véraison* berries inoculated through the pedicel developed symptoms 50–55 days after inoculation, whereas only 10% of the ripe berries rotted (Fig. 1B). Only 60% of the green (4% ss) and *véraison* berries inoculated through the stylar end developed symptoms 50–55 days after inoculation, whereas none of the ripe berries rotted in this time period (Fig. 1C). In general, ripe berries rotted slower than *véraison* and green (4% ss) berries, all of which rotted slower than pea-sized berries. Berries inoculated through the stylar end rotted slower than berries inoculated through the pedicel.

Detection of latent infection. One week after treatment with paraquat, all berries turned brown and sporulating pycnidia of *P. viticola* were observed on the pedicel of infected berries. Berries not treated with paraquat remained green and showed no evidence of symptom development. Two weeks after the paraquat treatment, infected berries were covered with sporulating pycnidia. Infected untreated berries remained green except for necrotic pedicels.

Fungicide trials. Various combinations of early- and late-season fungicide applications on Concord vines did not result in a significant reduction in fruit rot (Table 3). However, treatments of captan at 2.5- and 12.7-cm shoot growth tended to reduce fruit rot and significantly reduced the number of clusters with rachis lesions.

Phomopsis fruit rot was not significantly reduced on Delaware vines sprayed with up to four late-season applications of captan in 1986 (Fig. 2). Delaware vines treated with mancozeb during bloom had significantly less fruit rot than untreated vines in 1987 (Table 4). Vines treated with mancozeb at 2.5- and 12.7-cm shoot growth or at bloom had significantly fewer clusters with rachis lesions than vines treated with maneb plus zinc salt at *véraison* or at 11.2% ss. The grower's full schedule of six captan (2.2 kg a.i./ha) sprays provided the best control.

Table 4. Control of Phomopsis fruit rot on Delaware grapevines in 1987

Fungicide application[y]	No. of sprays	Berries with fruit rot (%)	Clusters with rachis infection (%)
Untreated	0	10.2 a[z]	82.8 a
Shoots 2.5 and 12.7 cm	2	8.0 ab	69.0 bc
Prebloom and 90% bloom	2	5.1 bc	66.0 c
Véraison (6.4% soluble solids)	1	8.6 ab	82.6 a
Ripe (11.2% soluble solids)	1	8.7 a	79.8 ab
Grower's schedule	6	3.3 c	46.7 d

[y] Mancozeb (3.6 kg a.i./ha) was applied at 2.5- and 12.7-cm shoot growth (8 May and 18 May, respectively), 1 wk before bloom (1 June), and at bloom (9 June). Maneb plus zinc salt (1.6 kg a.i./ha) was applied at *véraison* (4 August) and ripe stages of growth (11 August). The grower's schedule was captan (2.2 kg a.i./ha) applied at 2.5-, 17.8-, and 30.5-cm shoot growth (29 April, 15 May, and 28 May, respectively), 10% bloom (4 June), postbloom (13 June), and 3 July.
[z] Treatment means followed by the same letter(s) are not significantly different according to Fisher's protected LSD procedure at the 5% level.

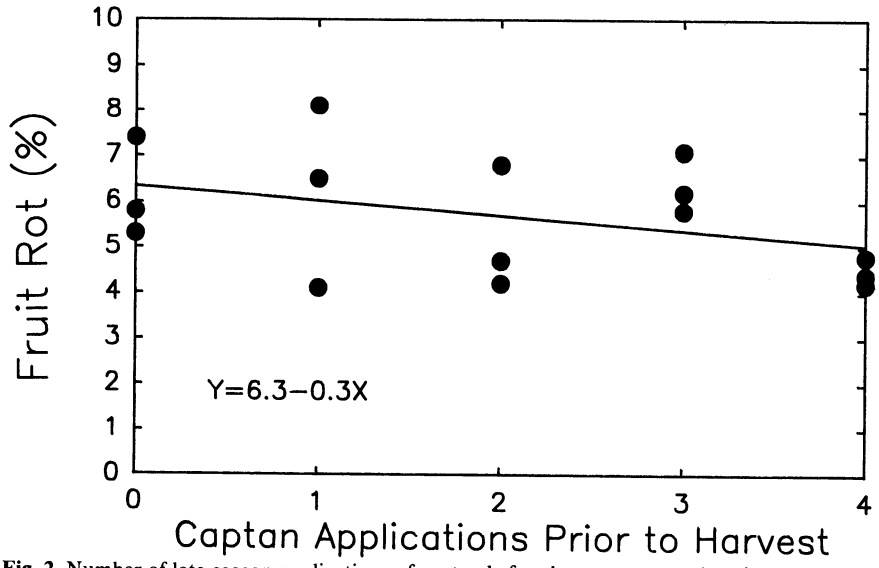

Fig. 2. Number of late-season applications of captan before harvest regressed against percentage of fruit rot caused by *Phomopsis viticola*. The slope is not significantly different from 0 at the 5% level.

DISCUSSION

Phomopsis fruit rot developed from latent infections that occurred at or shortly after bloom. Historically, fruit rot has occurred in years with unusually high rainfall during bloom. Inoculations of Concord clusters at or shortly after bloom resulted in substantial fruit rot, whereas inoculations at later or earlier growth stages did not increase the level of fruit rot over that in controls.

Younger berries (such as pea-sized) were more susceptible to infection and symptom development than older, riper berries. Milholland and Daykin (13) found that small green and mature blueberry fruit were susceptible to infection by *P. vaccinii* Shear; large green fruit, however, were less susceptible. Wounding was not a prerequisite for infection, as Lal and Arya (10) reported. Infection occurred through either the

pedicel or the stylar end of the berry, although symptoms developed faster when the pedicel end was inoculated.

Infections at bloom remain latent, since fruit rot symptoms usually do not develop until close to harvest. Latent fruit and rachis infections were detected by means of a paraquat dip technique. This technique has been used to detect latent infections by other *Phomopsis* species in soybean (5,9) and lupin (6). *Diplodia natalensis* Pole-Evans (*D. viticola* Desm.) and *Botrytis cinerea* Pers., which cause bunch rots of grapes, also produce latent infections of grapes (11,17).

Rachis lesions developed after inoculations at 12.7-cm shoot growth or at bloom. This would suggest that the rachis is susceptible to infection from budbreak through bloom. *P. vaccinii*, the cause of blueberry twig blight, infects twigs through the flower from budbreak through bloom (12). Rachis infections develop into brittle lesions that can cause the entire cluster or portions of the cluster to fall from the vine before harvest. Therefore, rachis infection is the most important phase of this disease.

The yield loss estimates show the importance and impact that this disease can have on Concord grape production. Although Phomopsis fruit rot was not the only factor contributing to yield loss, much of the loss was associated with broken rachis infections of whole clusters and cluster portions. The monetary loss was quite significant. Even if only one-half of the yield loss was due to Phomopsis fruit rot, the monetary loss would have amounted to $635/ha (assuming $176.4/t). The cost of four applications of captan 50W (two early in the season and two at bloom) would have been less than $79/ha.

Fruit rot and rachis infection on Delaware was significantly reduced by application of two fungicide sprays during the bloom period. Additional control was provided with the full grower schedule. Although these data may have other interpretations, they support the hypothesis that fruit infection occurred during bloom and not at more mature stages of growth, as indicated by Gregory (8). Past research has shown that grapevines were most susceptible to the cane and leaf spot phase during the 2.5- and 12.7-cm shoot growth stage (4,14). Protectant fungicide applications during the 2.5- and 12.7-cm growth stage and during bloom should improve control of all phases of this disease and reduce losses caused by the fruit rot phase. Future research should center on inoculum production and disease forecasting to fine-time spray recommendations.

ACKNOWLEDGMENTS

We thank N. Salva, H. Crow, T. Taft, C. Cummings, and D. G. Riegel for technical assistance, and Trenholm Jordan for the use of commercial vineyards. This research was supported by the New York Wine and Grape Foundation.

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