Blueberry Fruit Rot Caused by *Phomopsis vaccinii*

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ABSTRACT

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Phomopsis vaccinii caused a fruit rot of blueberry at harvest in North Carolina. The fungus apparently penetrates blueberry fruit at all stages of development and remains latent until maturation. Fruit infected with *Phomopsis* are typically soft and often split resulting in leakage of juice. The infected fleshy tissue is reddish brown and mushy. Blueberry cultivars Croatan, Harrison, Murphy, and Wolcott were all susceptible to Phomopsis fruit rot with Harrison being the most susceptible.

Phomopsis vaccinii Shear causes a serious canker and dieback disease of blueberry (*Vaccinium corymbosum* L.) in Michigan (4) and a blighting of 1-yr-old woody blueberry stems with flower buds in North Carolina (2). Twig blights caused by *P. vaccinii* on susceptible cultivars can result in fruit losses of 2 or 3 pints per bush (2). In addition to fruit loss by twig blight lesions, the fungus has recently been isolated from soft decayed fruit at harvest in several North Carolina blueberry plantings. An average of 15.2% defective fruit of blueberries was found in consumer samples obtained from Greater New York supermarkets during 1978 and 1979 (1). Phomopsis fruit rot accounted for 0.5% of this loss. The purpose of this study was to evaluate *P. vaccinii* as a preharvest fruit-rotting organism and determine the periods of infection.

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MATERIALS AND METHODS

Field isolations. In 1981, 500 berries of the cultivar Harrison were collected at random from several bushes each week from 23 April (small green berries) to 2 June (mature fruit). The berries were surface-disinfested in 0.525% sodium hypochlorite for 2 min, rinsed in sterile distilled water, and placed separately on a wire screen inside a plastic container (325 × 210 × 55 mm) lined with a wet paper towel about 24 hr after collection. The containers and berries were placed at 25 C and the number and type of rot was determined after 2 wk.

Pathogenicity. Greenhouse and field inoculations were conducted in 1982 with *P. vaccinii* isolate PV-1 and the inoculum was prepared as described previously (2).

Three-year-old dormant Murphy plants set in 25-cm-diameter pots containing a peat:sand (1:1, v/v) mixture were placed on a greenhouse bench at 20–30 C until plants were at the stage of berry development for inoculation. A concentration of about 1×10^6 conidia per milliliter was sprayed onto one fruit cluster of each plant and one cluster was sprayed with distilled water as a control. The berries were inoculated at the small green fruit (5–7 mm diam.) and mature fruit stages. Inoculated and uninoculated fruit clusters were covered with two layers of wet cheesecloth, a plastic bag, and a paper bag. The cheesecloth and bags were removed after 48 hr and plants were maintained on a greenhouse bench. Treatments were replicated five times. Disease readings were taken 5 and 1 wk after inoculating the small green fruit and mature fruit, respectively. Isolations from both soft and underdeveloped shriveled berries were made on potato-dextrose agar (PDA) and the fungi identified.

Four-year-old plants of the cultivars Croatan, Harrison, Murphy, and Wolcott on a research farm near Castle Hayne, NC, were inoculated as described. Inoculations were made on 28 April (small green berries 5–7 mm diam.), 11 May (large green berries 10–12 mm diam.), and 1 June (mature fruit 15 mm diam.). One cluster of berries per plant was inoculated and one cluster was sprayed with distilled water as the control. Treatments were replicated four times. Inoculated and uninoculated fruit were harvested on 9 June 1982 and the number of decayed fruit determined. The

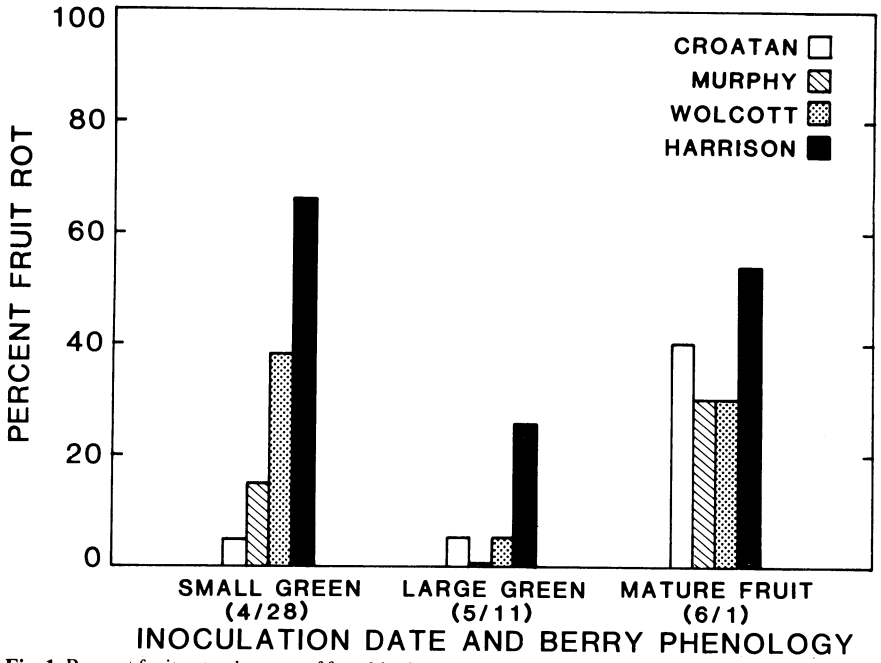

Fig. 1. Percent fruit rot at harvest of four blueberry cultivars inoculated with *Phomopsis vaccinii* at three stages of berry development. Means of four replicates.

number of fruit per cluster at harvest ranged from five to 35 with a mean of 18. Isolations were made on PDA from 60 decayed fruit (about 15/cultivar) and the fungi identified.

RESULTS AND DISCUSSION

Field isolations. In 1981, *P. vaccinii* was isolated from 13, 16, 9, 16, 35, 37, and 50% of the Harrison berries collected on 23 and 28 April, 5, 12, 19, and 27 May, and 2 June, respectively. Previous studies (2) indicated that conidia of *P. vaccinii* are present in blueberry fields in North Carolina from late February to early August.

Pathogenicity. Greenhouse inoculation of small green fruit and mature fruit of the cultivar Murphy with *P. vaccinii* resulted in soft decayed fruit at maturity. Forty-eight percent of the 52 fruits inoculated at the small green fruit stage were either decayed or underdeveloped and shriveled. None of the berries sprayed with distilled water alone were decayed. Twenty-five of 69 (36%) berries inoculated at maturity developed rot after 1 wk. Forty-three of 50 isolations from the soft and shriveled fruit yielded *P. vaccinii*. Blueberry fruit infected with *P. vaccinii* were very soft and often split resulting in leakage of juice. The fleshy tissue of infected fruit was reddish brown and mushy compared with the firm white fleshy tissue of uninfected fruit. The small shriveled berries appeared to develop from infection of the pedicels. *P. vaccinii* was isolated from 12 of 14 shriveled berries plated on PDA.

Field inoculations with *P. vaccinii* resulted in fruit rot similar to the greenhouse test. Harrison was the most susceptible cultivar tested, with 67, 25, and 53% rot when inoculated at the small green fruit, large green fruit, and mature fruit stages, respectively (Fig. 1). Berries inoculated at the large green stage (11 May 1982) had the least amount of Phomopsis fruit rot of all cultivars tested.

Fruit rot at harvest for uninoculated controls was 1% or less for all cultivars sprayed with distilled water at the small and large green fruit stages but averaged 15% for the mature fruit. *P. vaccinii, Colletotrichum gloeosporioides*, and *Alternaria* sp. were isolated from these decayed fruit. The higher percentage of fruit rot occurring in uninoculated fruit at maturity may be due to the increased time of exposure to natural infection. Isolations from 60 field-inoculated fruit resulted in 90% recovery of *P. vaccinii*.

Results of field isolations and greenhouse and field inoculations indicated that *P. vaccinii* is capable of causing fruit rot of blueberry and that infection occurs throughout the growing season at different stages of berry development. Because rot symptoms are not observed until harvest, the fungus apparently penetrates the developing fruit and remains latent until maturation. Phomopsis fruit rot and anthracnose fruit rot caused by *C. gloeosporioides* are the two most important preharvest fruit rots of blueberry in North Carolina. *Phomopsis* causes a very soft rot at harvest, often splitting the fruit and causing leakage of juice. Anthracnose fruit rot is somewhat firmer with the infected area slightly sunken and formation of acervuli that exude salmon-colored masses of conidia on the fruit surface (3). Harrison is one of the major blueberry cultivars grown in North Carolina and is severely affected by both fruit rots as well as Phomopsis twig blight.

LITERATURE CITED

1. Cappellini, R. A., Ceponis, M. J., and Koslow, G. 1982. Nature and extent of losses in consumer-grade samples of blueberries in Greater New York, 1978–1980. HortScience 17:55-56.
2. Milholland, R. D. 1982. Blueberry twig blight caused by *Phomopsis vaccinii*. Plant Dis. 66:1034-1036.
3. Stretch, A. W. 1967. Occurrence and control of *Glomerella cingulata* on highbush blueberry. Plant Dis. Rep. 51:401-404.
4. Weingartner, D. P., and Klos, E. J. 1975. Etiology and symptomatology of canker and dieback diseases of highbush blueberries caused by *Bodronia* (*Fusicoccum*) *cassandrae* and *Diaporthe* (*Phomopsis*) *vaccinii*. Phytopathology 65:105-110.