Blueberry Twig Blight Caused by *Phomopsis vaccinii*

R. D. MILHOLLAND, Professor, Department of Plant Pathology, North Carolina State University, Raleigh 27650

ABSTRACT

Milholland, R. D. 1982. Blueberry twig blight caused by *Phomopsis vaccinii*. Plant Disease 66: 1034-1036.

In North Carolina, *Phomopsis vaccinii* causes a blighting of 1-yr-old blueberry twigs with flower buds, resulting in reduced fruit production. The primary mode of infection was found to be through flower buds from budbreak through bloom. The fungus apparently entered the stem through the vascular tissue and progressed down the stem 50–150 mm. The entire stem was not killed. Systemic invasion also occurred when the fungus infected the leaf margins and progressed down the petiole into the stem. Infection of unwounded succulent stems resulted in small, raised lesions that failed to develop further. Rain-dispersed conidia of *P. vaccinii* were collected in traps throughout the growing season, the largest number being caught from blossom budbreak through bloom.

Blueberry (*Vaccinium corymbosum* L.) twig blight caused by *Phomopsis vaccinii* was reported by Stevens (2) in 1924 and described by Wilcox (4,5) in 1939 and 1940. Wilcox (4) demonstrated the pathogenicity of *Phomopsis* sp. isolated from blueberry twigs and indicated that only young, succulent twigs are blighted and that localized lesions develop when woody tissue is inoculated. Weingartner and Klos (3) proposed the name "Phomopsis canker and dieback" instead of "twig blight" to describe the disease as it occurs in Michigan. In North Carolina, "twig blight" is a more descriptive term for this disease because cankers are rarely found and blighting of 1-yr-old woody stems with flower buds is the predominant symptom (Fig. 1).

In a recent survey, Phomopsis twig blight was found in 14 of 15 blueberry plantings in North Carolina. Disease incidence of individual plantings ranged from 1 to 50%, with a mean percentage of twig blight of 15 and 9 for Murphy and Croatan cultivars, respectively (Milholland, *unpublished*). The purpose of this study was to determine the mode of infection and periods of spore dispersal by *P. vaccinii* as it occurs in North Carolina blueberry plantings.

Use of trade names in this paper does not imply endorsement of the products named nor criticism of similar ones not mentioned.

Paper 8084 of the Journal Series of the North Carolina Agricultural Research Service, Raleigh 27650.

Accepted for publication 1 March 1982.

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0191-2917/82/11103403/$03.00/0

MATERIALS AND METHODS

Inoculum. *P. vaccinii* isolate PV-1 was isolated from an infected blueberry twig at the Horticultural Crops Research Station, Castle Hayne, NC, and isolate PV-2 from a blueberry stem canker in Michigan. The PV-2 isolate was provided by D. C. Ramsdell, Michigan State University.

Conidial inoculum was produced on oatmeal agar by flooding a 14-day-old culture with 20 ml of distilled water, scraping the surface with a razor blade, and filtering the suspension through cheesecloth. Concentrations were adjusted with a hemacytometer (American Optical, Scientific Instrument Division, Buffalo, NY 14215) to $1–3 \times 10^6$ conidia per milliliter.

Greenhouse tests. Two-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 development for inoculation. Approximately 0.5 ml of a conidial suspension of *P. vaccinii* isolates PV-1 and PV-2 was placed in the terminal flower buds at budbreak of 10 stems each of the cultivar Murphy. Control and inoculated plants were placed in a 95–100% relative humidity moisture chamber at 25–30 C for 72 hr and then transferred to a greenhouse bench. The number and length of twig blight lesions were recorded 1 mo after inoculation.

A second test included inoculations at tight bud, budbreak, and bloom stages. A conidial suspension of the PV-1 isolate was sprayed with a DeVilbiss atomizer to one bud or flower cluster on each of three stems of a 2-yr-old Murphy plant. A double layer of wet cheesecloth was placed over the inoculated bud or flower cluster, which was then covered with a plastic bag and sealed with a paper bag. The control plants were sprayed with distilled water. The cheesecloth and bags were removed after 72 hr, and the plants were maintained on a greenhouse bench at 20–30 C for 1 mo. Treatments were replicated four times.

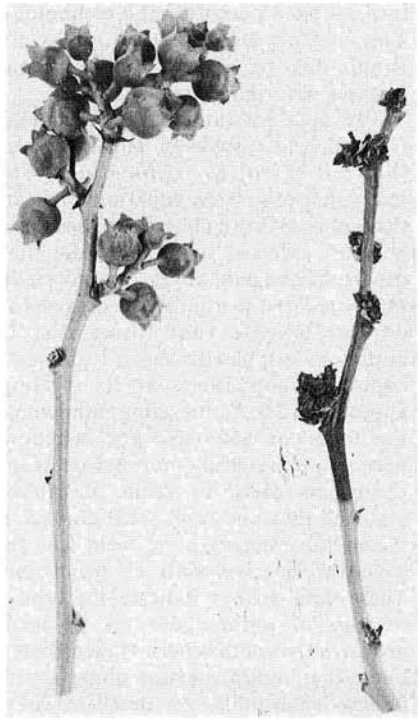

Fig. 1. Blueberry twig blight caused by *Phomopsis vaccinii*. Necrosis of 1-yr-old infected stem and flower buds **(right)** as compared with uninfected stem with developing fruit **(left)**.

Succulent unwounded stems of 1-yr-old Murphy plants were also sprayed with a conidial suspension of isolate PV-1. Three inoculated and three uninoculated plants with three to four stems each were placed in the moist chamber for 72 hr and then transferred to a greenhouse bench. Disease readings were taken 1 mo after inoculation.

Field tests. Fifteen-year-old Murphy plants located on a research farm near Castle Hayne, NC, were used in all field tests. In 1980, the terminal flower bud on each stem inoculated was sprayed with a conidial suspension of isolate PV-1. Two stems of each plant were inoculated, and two stems were sprayed with distilled water at budbreak on 27 March 1980. Inoculated and uninoculated buds were covered with two layers of wet cheesecloth, a plastic bag, and a paper bag. The cheesecloth and plastic bag were removed after 72 hr, and the paper bag was replaced over the stem. The treatments were replicated 10 times. Number of twig blight lesions, length of lesions, and number of buds killed per twig were recorded on 8 May 1980.

In 1981, field inoculations were made at tight bud (24 February), budbreak (24 March), and bloom (7 April). All stems inoculated or used as controls were covered with a paper bag on 3 February to reduce the amount of natural infection. Inoculations were made as previously described for the 1980 test. Five buds (one per stem) per plant were inoculated at each stage of bud development. Distilled water was sprayed on five additional buds for controls. Treatments were replicated four times.

Conidial dispersal. Twigs of the cultivar Murphy infected with *P. vaccinii* the previous year (overwintered lesions) were excised from the plants on 21 February 1980 and cut into 2- to 3-cm pieces. Ten pieces were placed into each of three spore traps that consisted of mesh-lined plastic funnels supported over 1-L plastic bottles. Ten milliliters of a 5% cupric sulfate solution was added to each bottle to prevent conidial germination. Spore traps were inserted into 10-cm-diameter plastic pots and placed in the ground between blueberry bushes. Three additional traps containing stem pieces with twig blight lesions from current-season infections were placed in the field on 10 May 1980. Weekly counts of conidia collected in traps from rainwater runoff from both overwintered and current-season lesions were made with a hemacytometer and then adjusted to a 7-day period.

Trapping of conidia in 1981 was similar to the procedures used in 1980. On 3 February 1981, spore traps were placed within the plant canopy below twig lesions infected the previous year. No spores were trapped for the first 3 wk despite the fact that mature spores were observed in pycnidia. Traps containing excised stem pieces with overwintered lesions or with current-season infections were then set in the field on 17 March and 14 May, respectively.

RESULTS

Greenhouse tests. Inoculations with both isolates of *P. vaccinii* at budbreak resulted in typical twig blight lesions. Seventy percent of the stems inoculated with each isolate developed twig blight symptoms after 2 wk. Mean length of lesions after 1 mo for stems inoculated with PV-1 and PV-2 was 56 and 50 mm, respectively. No disease was observed on uninoculated controls. Isolations from the twig blight lesions yielded cultures typical of the inoculum.

The percentage of inoculated stems that developed twig blight 1 mo after inoculation at the tight bud, budbreak, and bloom stages was 0, 66, and 41, respectively. Length of lesions ranged from 10 to 80 mm. One to four flower buds per stem were killed. No twig blight lesions developed on the uninoculated controls.

Small, raised lesions (1–2 mm diameter) developed on succulent unwounded stems 14 days after inoculation, but the lesions did not develop further. However, large, brown stem lesions did develop where the fungus had infected the leaf petiole and progressed into the stem through the vascular tissue. The lesions averaged 30 mm in length 1 mo after inoculation.

Field tests. Sixty percent of the stems inoculated (12 of 20) at budbreak showed twig blight symptoms on 8 May 1980. Length of lesions ranged from 10 to 160 mm (mean: 78 mm). The number of buds killed per twig varied from 1 to 12 (mean: 6). One of the 20 uninoculated controls was infected with Phomopsis twig blight.

In 1981, no visible symptoms of Phomopsis twig blight were observed from natural infections in the field at the tight bud stage (24 February) or at blossom budbreak (17–24 March). Small, brown stem lesions at the base of the flower buds were first noted on the cultivar Murphy on 31 March. Inoculations at budbreak and bloom resulted in 95–100% of the twigs being infected, whereas only 10% of the buds inoculated at the tight bud stage developed twig blight (Fig. 2). The length of lesions ranged from 10 to 170 mm (mean: 67 mm). Some of the bags were torn during the period 17 February to 5 May, which would account for some of the uninoculated controls being naturally infected with *P. vaccinii*. Isolations from inoculated and uninoculated twigs with twig blight yielded *P. vaccinii*.

Conidial dispersal in rainwater. Conidia from overwintered twig blight lesions on excised stem pieces were trapped from 28 February until 5 August 1980 (Fig. 3). The greatest number of conidia (6.4×10^7) were collected the week of 15 April (full bloom); concentrations then decreased to 7.3×10^4 by 17 June (harvest). Conidia from current-season infections were trapped from 27 May until 29 July 1980. The maximum number of conidia trapped (4.9×10^6) occurred during the week of 27 May. No conidia were collected from overwintered or current-season lesions after 12 August 1980.

The weekly rainfall from 1 March through August fluctuated between a low of 5 mm during the third week of April to a high of 165 mm the last week in July but did not always correlate with the levels of conidia trapped. A total of 725 mm was recorded for the 6-mo period.

Results of trapping conidia in rainwater

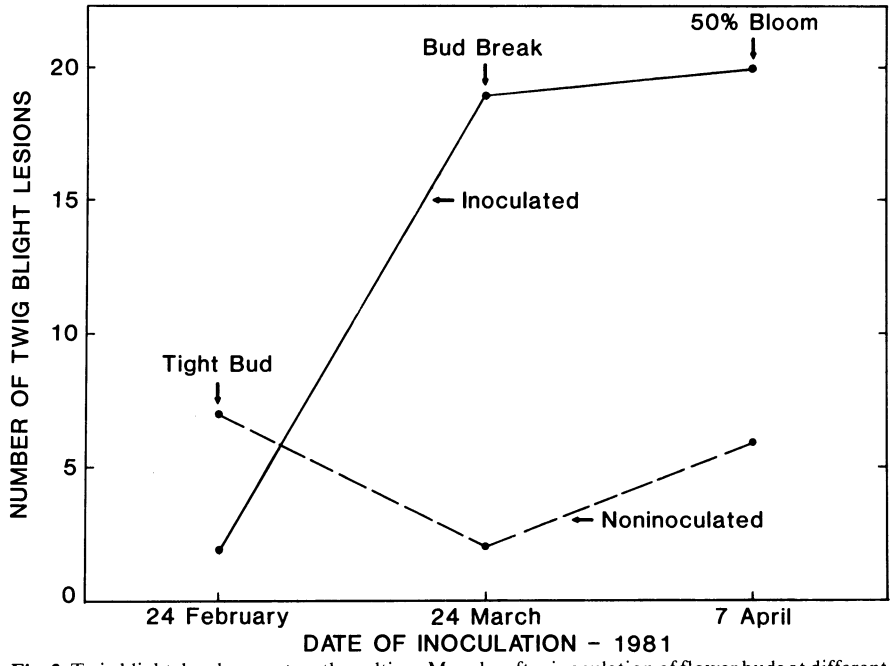

Fig. 2. Twig blight development on the cultivar Murphy after inoculation of flower buds at different stages of development with *Phomopsis vaccinii*. Mean length of lesions was 67 mm.

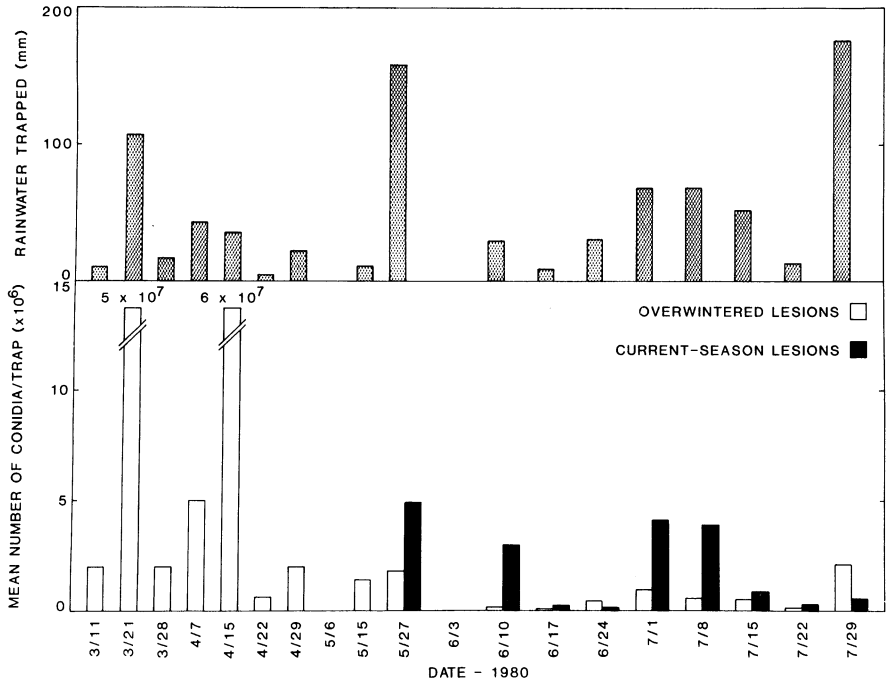

Fig. 3. Mean number of conidia of *Phomopsis vaccinii* collected per week in rainwater traps from overwintered and current-season twig blight lesions in 1980.

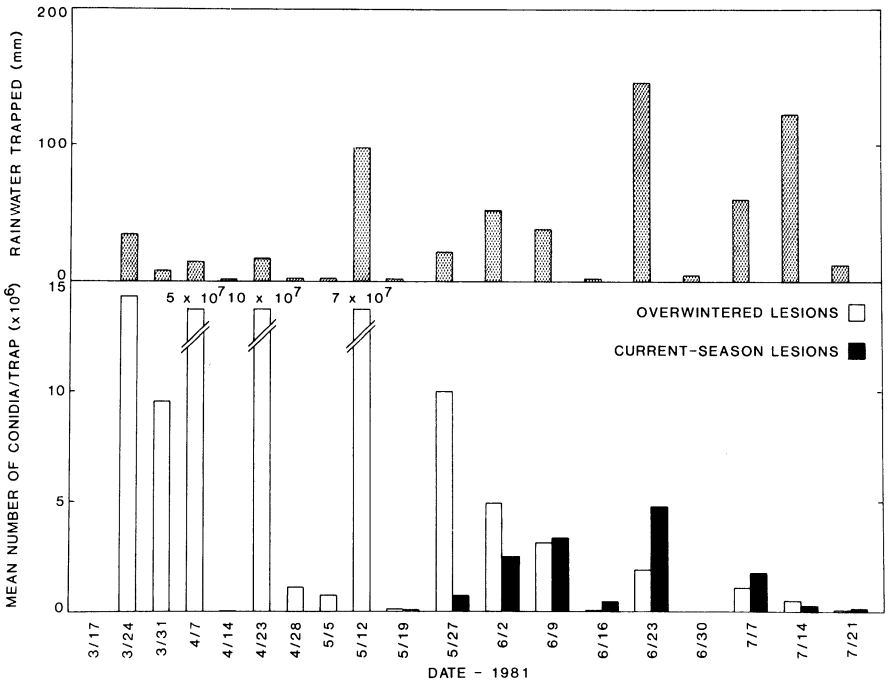

Fig. 4. Mean number of conidia of *Phomopsis vaccinii* collected per week in rainwater traps from overwintered and current-season twig blight lesions in 1981.

runoff showed a similar trend in 1981 as in the previous year. A substantial number of conidia were trapped throughout the early part of the growing season, with 1.4×10^7 at blossom budbreak (17–24 March) and 10×10^7 at late petal fall (23 April) (Fig. 4). The number of conidia trapped during the ripening period (16 June) decreased to 7.7×10^4. Conidia from current-season infections were trapped from 19 May through July. No conidia were collected from these lesions after July despite an abundance of rainfall in August.

DISCUSSION

Inoculations with *P. vaccinii* in greenhouse and field tests indicated that blueberry twig blight developed primarily from infections of flower buds at budbreak through bloom in North Carolina. Phomopsis twig blight primarily resulted in reduced yield in North Carolina. This contrasts with Phomopsis stem canker in Michigan, in which stems or entire bushes may be killed (3).

Both isolates of *P. vaccinii* used were capable of causing twig blight lesions. Greenhouse inoculations of unwounded succulent stems with *P. vaccinii* resulted in small, raised lesions that failed to develop further. Previous inoculation tests with wounded succulent stems did result in dieback of stems (Milholland, *unpublished*). These tests indicate that a wound or another mode of entry besides penetration through unbroken epidermal tissue was required for systemic invasion by the fungus. The open flower buds not only acted as a catching-frame for rain-dispersed conidia but also provided the fungus with an avenue of entrance into the vascular tissue of the stem. Studies in Michigan on stem canker development by *P. vaccinii* (1) indicate that an abrasion wound or freeze damage is necessary for infection to occur. Although this occurred in North Carolina, it was not the primary means by which blueberry twig blight developed. Based on greenhouse inoculations, systemic infection also occurred when the fungus infected the leaf margins and progressed down the petiole into the stem.

Conidia of *P. vaccinii* were collected in spore traps from rainwater runoff from late February to early August. The greatest number of conidia were trapped during the early part of the growing season from blossom budbreak through bloom from overwintered twig blight lesions. These results are similar to those in Michigan, where maximum numbers of conidia from Phomopsis cankers were trapped during rains from bloom through petal fall in late May and June (1). Conidia from current-season infections did not extend the period of spore dispersal but did increase the total number of conidia available during the summer months, especially at harvest.

About four berries are normally produced from each developing bud. An average of five to six buds per stem were killed in 1980 and 1981 field inoculations, which would result in a loss of 20 to 24 berries per stem. Results of field testing of fungicides for twig blight control over the past 3 yr have shown a mean of 20 infected twigs per plant for unsprayed controls (Milholland, *unpublished*). Based on a reduction of 20–24 berries per stem, this could result in a loss of 2–3 pt per bush.

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