

Data Sheets on Quarantine Pests

*Diaporthe vaccinii***IDENTITY**

Name: *Diaporthe vaccinii* Shear

Anamorph: *Phomopsis vaccinii* Shear

Taxonomic position: Fungi: Ascomycetes: Diaporthales

Common names: Phomopsis canker and dieback, twig blight, fruit rot, storage rot, viscid rot (English)

Chancre phomopsien, brûlure phomopsienne (French)

Notes on taxonomy and nomenclature: On the basis of superficial morphology Wehmeyer (1933) suggested that *D. vaccinii* was morphologically similar to the previously described *D. phaseolorum* (Cooke & Ellis) Saccardo var. *batatis* (Harter & Field) Wehmeyer and *D. phaseolorum* var. *sojae* (Lehman) Wehmeyer. Chao & Glawe (1985) concluded from host specificity tests and behaviour of isolates on agar media that *D. vaccinii* is best regarded as a species distinct from *D. phaseolorum*.

Since stem blight symptoms near the tip of twigs on blueberry caused by *D. vaccinii* are similar to those caused by *Botryosphaeria dothidea* it has been suggested that, in disease surveys based on symptoms, blight of blueberry caused by *D. vaccinii* may have been confused with twig blight symptoms caused by *B. dothidea* (Witcher, 1961; Witcher & Clayton, 1963).

Bayer computer code: DIAPVA

EPPQ A1 list: No. 211

EU Annex designation: II/A1

HOSTS

The principal hosts are American and European cranberries (*Vaccinium macrocarpon*, *V. oxycoccus*, *V. oxycoccus* var. *intermedium*), highbush blueberry (*V. corymbosum*) and rabbiteye blueberry (*V. ashei*). *D. vaccinii* is restricted to cultivated *Vaccinium* species. The wild European species *V. oxycoccus* which usually occurs in mountain bogs could be a potential reservoir for the pest.

GEOGRAPHICAL DISTRIBUTION

EPPQ region: Romania (found in experimental plots of introduced American cultivars, but did not establish; Teodorescu *et al.*, 1985), UK - found in plants originally imported from the Netherlands and USA (New Jersey) in 1956 and planted in Scotland, but did not establish (Wilcox & Falconer, 1961; Baker, 1972).

North America: Indigenous, mainly temperate areas: Canada (Nova Scotia), USA (Arkansas, Illinois, Indiana, Maryland, Massachusetts, Michigan, New Jersey, North Carolina, Oregon, Washington, Wisconsin) (Shear *et al.*, 1931; Wilcox, 1939; Wilcox, 1940; Carlson, 1963; Conners, 1967; Weingartner & Klos, 1975; Chao & Glawe, 1985; Ames *et al.*, 1988).

South America: Chile (introduced from USA; Guerrero & Godoy, 1989).

EU: Absent.

BIOLOGY

The fungus grows well in a variety of carbon sources, at optimum pH 5-6, and over a temperature range of 4-32°C (Carlson, 1963; Weingartner & Klos, 1975); the most favourable temperature for conidium germination and growth is 21-24°C (Wilcox, 1939). Conidia germinated 95% and entered through wounds or directly at the tips of young, succulent blueberry shoots held in a damp chamber at 21-24.5°C. Four days after inoculation 71% of the shoots became blighted. In artificially inoculated plants, *D. vaccinii* is reported to cause cankers and dieback at temperatures over 30°C (Weingartner & Klos, 1975).

The epidemiology of the fungus has been studied in the USA. *D. vaccinii* overwinters on dead vines (Shear *et al.*, 1931; Wilcox, 1939) and ascospores and conidia are disseminated in the crop under wet or humid conditions. In North Carolina, rain-dispersed conidia of *P. vaccinii* have been trapped throughout the growing season, the largest number being caught between blossom budbreak through to bloom (Milholland, 1982).

The fungus is believed to enter stems through the vascular tissue (Milholland, 1982) and progress downwards towards the base, girdling the old branches at their junction, and killing part of the plant above the girdle (Wilcox, 1939).

On the basis of inoculations with *D. vaccinii* and field trials, it is believed that blueberry blight develops primarily from infections of flower buds at budbreak through bloom in North Carolina (Milholland, 1982). The open flower buds have been suggested as a path for entering into the vascular tissue. The conidia on germination enter berries throughout the growing season at all stages of development and they remain dormant until maturation causing a soft rot and leakage of juice at harvest (Milholland & Daykin, 1983). Since 90% of isolations from stems of apparently healthy vines of *V. macrocarpon* from a bed with a history of high disease incidence in Wisconsin yielded cultures of *D. vaccinii*, it seems likely that the fungus is an endophytic colonizer of blueberry and cranberry (Friend & Boone, 1968).

Conidial germ tubes enter leaves producing spots (Wilcox, 1939). Pycnidia with conidia appear on stems and leaf spots 2-3 weeks after inoculation (Wilcox, 1939; Weingartner & Klos, 1975). *D. vaccinii* has been isolated from fruit bodies found on overwintered cranberry leaves in New Jersey (Wilcox, 1939) but was not recovered in Wisconsin from vines collected in the spring from beds in which dieback had been very severe in the late summer of the preceding year (Friend & Boone, 1968). Cultures of *D. vaccinii* on maize meal agar, naturally and artificially infected blueberry and decayed cranberry fruit left to overwinter out of doors, or in an unheated building in winter, produced perithecia and pycnidia in Maryland and Oregon (Shear *et al.*, 1931; Wilcox, 1940). These observations suggest that overwintering is necessary for ascocarp development, completing the life cycle, perpetuating the species and providing a source of inoculum for infection in the next season.

A correlation between vine dieback and dry conditions has been suggested (Friend & Boone, 1968) because serious dieback occurred when rainfall was below average, and temperatures were above the seasonal average in Wisconsin. Dry conditions have been reported to place vines under stress and make them susceptible to dieback. Poor drainage is considered to favour outbreak of stem blight of *Vaccinium* species (Witcher, 1961).

Other fungi often associated with twig blight of cranberry are *Godronia cassandrae* and *Botryosphaeria dothidea* (Witcher, 1961; Weingartner & Klos, 1975).

DETECTION AND IDENTIFICATION

Symptoms

In susceptible blueberry cultivars, blighting of 1-year-old woody stems with flower buds is the predominant symptom in North Carolina. Systemic invasion has also been reported (Milholland, 1982).

Infected succulent, current-year shoots wilt in 4 days and become covered with minute lesions. The fungus continues to travel downward through the stem at a rate averaging 5.5 cm in 2 months, killing major branches and often entire plants (Wilcox, 1939; Daykin & Milholland, 1990). Regardless of age of stems, cankers are long and narrow, and are covered by the bark or epidermis (Weingartner & Klos, 1975). On blueberry stems over 2 years old, *D. vaccinii* causes a brown discoloration of the stem xylem below wilt symptoms (Weingartner & Klos, 1975). Direct inoculation of woody stems only produces localized lesions. Infected leaves show spots enlarging to 1 cm with pycnidia appearing in 2 weeks. The fungus may also remain dormant until favourable conditions permit it to resume growth (Wilcox, 1939). Infection of crowns usually results in death of stems originating from the crown. Wilt of (symptomless) stems of blueberries occurs in July-August and continues until October in Michigan. Pycnidia appear from August to October mostly on dead stems 3-5 years old (Weingartner & Klos, 1975). In Arkansas, twig blight symptoms at early fruit set include blighting of 1-year-old woody stems with flower buds and occasional blighting of new, young, succulent growth without flower buds (Ames *et al.*, 1988). Infected fruits turn reddish-brown, soft, mushy, often splitting and causing leakage of juice (Milholland & Daykin, 1983).

Morphology

Pycnidia occur on stems, leaves and berries, and vary in size from 300 to 500 μm in diameter on cranberry fruit. Pycnidial stromata 1-2 mm wide form on cornmeal and potato agar cultures; they are black, leathery, thick-walled, locular (floor of cavity convoluted), rupturing irregularly, exuding masses of conidia which are white or with a slight pink tinge. Conidiophores spindle-shaped, 15-25 μm in young pycnidial conidiomata but longer in older conidiomata. A conidia (alpha conidia) hyaline, ellipsoid, aseptate, with two prominent guttules, 6-11 x 2-5 μm . B conidia (beta conidia) hamate, 14-20 x 0.35 μm . Perithecia occur on cranberries and vines, on stems between bark and wood with an eccentric neck protruding through the bark, nearly hemispherical, 300-500 x 200-400 μm ; perithecial wall black, carbonaceous. Asci oblong, fusoid, sessile, 37-51 x 6.8-11.7 μm . Ascospores ellipsoid, 2-celled, each cell biguttulate, 8.8-11.8 x 2.4-3.4 μm . Ascospores ellipsoid, 2-celled, each cell biguttulate, 8.8-11.8 x 2.4-3.4 μm .

A full description is given by Shear (in Shear *et al.*, 1931) and a range of measurements is given by Ames *et al.* (1988), Chao & Glawe (1985) and Wilcox (1940).

MEANS OF MOVEMENT AND DISPERSAL

Natural spore dispersal of *D. vaccinii* only occurs over short distances. Export of infected vines from North America to other countries has been the main source of vine infection at new sites (Wilcox & Falconer, 1961; Baker, 1972; Guerrero & Godoy, 1989).

PEST SIGNIFICANCE

Economic impact

The disease is commonly established in the USA on cranberries and blueberries (Friend & Boone, 1968; Farr *et al.*, 1989) and was considered in the late 1940s to be of minor importance (Wilcox, 1939) though it was responsible for a reduction of 18-35% of the cranberry crop in several plots in 1933 in Massachusetts (Bergman & Wilcox, 1936). The disease became serious in a few marshes in Wisconsin in 1966 and in isolated instances caused serious losses (Friend & Boone, 1968). In 1975, phomopsis dieback was reported epidemic in the centre of the blueberry-producing area in Indiana and southern Michigan and *D. vaccinii* was then considered to be a serious pathogen under favourable conditions (Weingartner & Klos, 1975). In Wisconsin, loss of up to 65% of cranberries in storage due to *Godronia cassandrae* (end rot), *D. vaccinii* (viscid rot) and *Ceuthospora lunata* (=Apostrasseria lunata) (black rot) have been reported (Carlson, 1963). Twig blight of susceptible blueberry cultivars has been estimated to cause fruit loss of 2-3 pints per bush in North Carolina (Milholland, 1982). In New York supermarkets in 1978 and 1979, phomopsis fruit rot accounted for 0.5% loss of the 15.2% defective fruits (Milholland & Daykin, 1983).

Control

All highbush blueberry cultivars and standard cranberry cultivars are susceptible to phomopsis canker (Wilcox, 1939; Weingartner & Klos, 1975; Milholland & Daykin, 1983; Ames *et al.*, 1988), though some variation in susceptibility has been reported (Teodorescu *et al.*, 1988). Bordeaux mixture with a suitable spreader has been tested as a means of protection against *D. vaccinii* (Bergman & Wilcox, 1936; Wilcox & Bergman, 1945). Early spraying, when nearly all the flowers are in the late-bud stage, has been reported to give good control (Wilcox & Bergman, 1945). Ferbam has also been tested and reported to give as good control as Bordeaux mixture applied before the flower buds opened. More recently, captafol and benomyl have given good reduction of canker in field trials (Parker & Ramsdell, 1977).

Phytosanitary risk

D. vaccinii was recently added to the EPPO A1 list, but has not been listed as a quarantine pest by any other regional plant protection organization. Since it was first reported in the UK over 30 years ago and disappeared without causing any problems, there could be some doubt about its potential importance. In Romania, it was apparently introduced with American *Vaccinium* cultivars being tested with a view to the development of *Vaccinium* cultivation. In the experimental plots, it was noted that cultivars differed in susceptibility and that infection was increasingly severe from year to year (Teodorescu *et al.*, 1985). The fungus has not apparently persisted or spread onto commercial crops. Since commercial production of *Vaccinium* is now becoming much more important in Europe, it is important to exclude North American *Vaccinium* pathogens such as *D. vaccinii*.

PHYTOSANITARY MEASURES

Since *D. vaccinii* occurs as an endophyte in apparently healthy vines, only vines known to be disease-free should be imported for cultivation. In countries where the disease is established, vines for export should be screened and certified as disease-free before distribution.

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