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Godronia Canker (*Godronia cassandrae* f. sp. *vaccinii*) in Highbush Blueberry

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ABSTRACT

Godronia canker, caused by the fungus *Godronia cassandrae* f. sp. *vaccinii*, may cause severe stem dieback in highbush blueberry (*Vaccinium corymbosum*), and it is especially damaging in young plantings. This paper is an overview of presently available literature on taxonomy, biology and epidemiology of *G. cassandrae* f. sp. *vaccinii*, and management options of the disease. The pathogen has been reported from highbush blueberry growing areas in North America and in Northern and Central Europe. The pathogen overwinters in cankers or infection sites on young stems. Symptoms become visible in spring as reddish brown lesions that enlarge progressively, and eventually may girdle and kill the stems. The sexual stage has been found in North America, but not in Europe. However, the imperfect stage seems to dominate on both continents. Pycnidia are produced abundantly in the lesions, and conidia may be spread by rain splash from early spring to late autumn. The peak period of conidia formation has mostly been reported to occur in spring and early summer. Infections mainly take place in spring and autumn. Control measures include use of healthy planting material, use of cultivars with high resistance, and pruning and burning of infected stems. There are no fungicides available that sufficiently control the disease, and it is difficult to optimise the timing of fungicide applications.

Keywords: plant disease, stem lesion, *Topospora myrtillii*, *Vaccinium corymbosum*

CONTENTS

INTRODUCTION

Godronia canker (*Godronia cassandrae* (Peck) f. sp. *Vaccinii* Groves, anamorph: *Topospora myrtilli* Feltg. syn. *Fusicoccum putrefaciens* Shear) has caused severe losses in commercial production of highbush blueberry (*Vaccinium corymbosum* L.) in North America and Europe. The disease has been known since the 1930's (Conners 1932), but research on the pathogen and the disease it causes has been limited, and most reports on Godronia canker were published between 1965 and 1980. However, recent publications from Norway (Strømeng and Stensvand 2001) and Poland (Szmagara 2009) show that the disease continues to be problematic in highbush blueberry. In the early 1990's, farmers in Norway established highbush blueberry plantings, but many of the fields became seriously affected by Godronia canker (Strømeng and Stensvand 2001). In Poland, which has the largest highbush blueberry production in Europe, Godronia canker was the most serious disease of highbush blueberry stems in a study conducted in the southeastern region of the country (Szmagara 2009). Control of Godronia canker is difficult, primarily because there are no fungicides available to control the disease and because it affects establishment of new plantings. This paper provides an overview of the presently available knowledge on taxonomy, biology and epidemiology of *G. cassandrae* f. sp. *vaccinii*, areas where Godronia canker has been reported, cultivar resistance and other possibilities to manage the disease.

DISTRIBUTION AND DISEASE SEVERITY

Canada

In 1931, a fungus causing dieback of stems in highbush blueberry was found in Quebec, Canada, and identified as *F. putrefaciens* (Conners 1932). Within two years, 30% of the bushes in the plantation had died. This was the first report of *G. cassandrae* f. sp. *vaccinii* in highbush blueberry. In Canada, *G. cassandrae* f. sp. *vaccinii* was reported in 1943 from British Columbia (Conners and Savile 1944), and later it was reported from Nova Scotia (Conners and Savile 1948) and New Brunswick (Creelman 1958). In British Colombia, the disease caused severe damage to young plantings, and it was reported that 40% of the plants were killed in a two-year old plantation (McKeen 1958). Although older plants rarely died of the disease, up to 25% of the stems could be destroyed, causing severe yield loss. In

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Invited Mini-Review

Nova Scotia, Creelman (1958) considered Godronia canker to be the most important disease in highbush blueberry. The disease is still the most serious canker disease of highbush blueberry in British Columbia (Pscheidt 2010).

USA

Godronia canker in highbush blueberry was reported from USA for the first time in 1957 in Massachusetts (Zuckerman 1959), where it caused severe outbreaks and became an economic threat to the blueberry industry (Zuckerman 1960). One year later, the disease was reported from Maine (Creelman 1958). Barnes and Tweedie (1964) reported the disease in highbush blueberry in Michigan in 1962, where it was found in areas of commercial highbush blueberry production along the coast of Lake Michigan. Caruso and Ramsdell (1995) also mention the disease being present in Oregon, Washington and in the mountains of North Carolina. The disease may seriously affect establishment of new plantings in Washington (Pscheidt 2010). Godronia canker also occurs in highbush blueberry in New Jersey (Anonymous 1997) and Missouri (Fuqua *et al.* 2005), but is not considered important.

Europe

In Europe, Godronia canker was first reported on highbush blueberry in Finland, where it was the most serious fungal disease in this crop (Hårdh 1959), and two years later it was reported from Great Britain (Wilcox and Falconer 1961). Godronia canker was found in a planting in Bavaria, Germany in 1966 (Sprau 1969), and the disease started to cause severe dieback in Niedersachsen in 1974 (Menzinger and Krupp 1977). The disease was detected in a highbush blueberry planting in Poland in 1973 (Borecki and Pliszka 1978), and in Moscov, Russia in 1984 (Mukhina and Sizova 1984). In Russia, it was first found on bog bilberry (*V. uliginosum*), but it has also damaged cultivated highbush blueberry (Mukhina *et al.* 1993). In a European trial on highbush blueberry cultivars, Godronia canker severely affected the bushes in many of the participating countries, including Germany, Italy, the Netherlands, Poland, Switzerland, and UK (Liebster 1979). Whether the plant material used in the trials originated from the same nurseries is not mentioned, but since all fields were affected by the disease it seems likely that the disease was introduced with the highbush blueberry planting material. From Sweden, only severe winter damage was reported in the same European trial, but later it was confirmed that the disease was present in the country (Liebster 1980). Damage by *G. cassandrae* f. sp. *vaccinii* was also reported in a cultivar trial in Slovenia, but the disease was controlled by pruning and burning of infected canes (Oblak 1977). Plizka (1997) in Poland reported that highbush blueberry planted in the Baltic countries was severely damaged by Godronia canker.

Other countries

Godronia canker has, to our knowledge, never been reported from blueberry producing areas in Australia, New Zealand, Japan, and Chile.

GODRONIA CANKER IN NORWAY

First detection

G. cassandrae has been reported in Norway on non-cultivated *Vaccinium*-species; bilberry (*V. myrtillus*), lingonberry (*V. vitis-idaea*), and also on common heather (*Calluna vulgaris*) (Eriksson 1970). Highbush blueberry production started in Norway in the late 1980's and early 1990's. In 1990, a research project on highbush blueberry was initiated at the University of Life Sciences (UMB) to find cultivars suitable for Norwegian growing conditions (Vestrheim *et al.* 1997). Seven cultivars were planted in a research field in 1990, and an additional eight cultivars were planted in 1993. The disease was detected in highbush blueberry in Norway for the first time in 1995 in many commercial fields in southern Norway and in the cultivar research field at UMB (Stensvand and Langnes 1995).

Disease surveys

Yearly disease assessments were carried out in the research field during 1995-1999, and symptoms were found on all 13 cultivars planted (Strømeng and Stensvand 2001). Overall, Godronia canker was found on 29 to 81% of the one-year old stems, depending on cultivar. In 1998, a survey in 51 commercial highbush blueberry fields in southern Norway was carried out to determine how widespread Godronia canker was and to find possible differences in susceptibility between cultivars (Strømeng and Stensvand 2001). The survey revealed that the disease was present in 35 of the 51 fields examined, and it was found in all 11 counties included, but the damage was most severe in the south eastern parts and along the southern coast of the country. For the 15 most commonly grown cultivars symptoms were found on 7 to 47% of the one-year old stems included in the examination (Strømeng and Stensvand 2001). Many of the fields became so seriously affected by Godronia canker that further production ceased.

TAXONOMY AND HOST SPECIFICITY

Host plants for *Godronia* spp.

The genus *Godronia* includes a group of inoperculate fungi in the class Leotiomycetes (Groves 1965). The fungi in this genus are generally host specific, and they are found in plants of many different genera: *Alnus*, *Andromeda*, *Betula*, *Calluna*, *Chamadaphne*, *Clethra*, *Diervilla*, *Kalmia*, *Ledum*, *Linnaea*, *Menziesia*, *Phragmites*, *Prunus*, *Rhamnus*, *Rhus*, *Ribes*, *Salix*, *Spiraea*, *Sorbaria*, *Symphoricarpos*, *Vaccinium* and *Viburnum* (Groves 1965; Smerlis 1968).

First descriptions and reports of *Godronia cassandrae*

The species *Godronia cassandrae* Peck was described for the first time in 1885, when it was found on dead twigs of *Cassandra calyculata* L. (current name: *Chamadaphne calyculata*) in New York, USA, by Dr. C. H. Peck (Shear and Bain 1929). In 1917, an imperfect fungus causing end-rot of cranberry (*V. macrocarpo*n Ait., syn. *Oxycoccus macrocarpus* (Ait.) Pers.) in several northern states of USA; Massachusetts, New Jersey, Maine, Michigan, Wisconsin, Oregon, and Washington, was named *Fusicoccum putrefaciens* (Shear 1917). It was suggested that a Discomycete often found in association with *F. putrefaciens* on mummified cranberry fruits was the teleomorphic stage of the pathogen. The genetic relationship between the two forms was established by Shear and Bain (1929), and the teleomorph was identified as *G. cassandrae.*

Special forms of the species

In a study of the genus *Godronia*, Groves (1965) described 24 species of the genus, and five different forms (f. sp.; formae specialis) of the species *G. cassandrae*. The fungus infecting plant species of the genus *Vaccinium* was named *G. cassandrae* f. sp. *vaccinii*. Although host specificity was not investigated, it was desirable to keep it separated from the other forms due to the pathogenic significance on *Vaccinium* (Groves 1965). Smerlis (1968) indicated that different forms of *G. cassandrae* existed, and based on inoculation tests with isolates of the fungus collected from nine host plant species, it was concluded that three forms of the species occurred in Quebec. The form *G. cassandrae* f. sp. *vaccinii* was differentiated from *G. cassandrae* f.sp. *betulicola* based on pathogenicity, and from *G. cassandrae* f.sp.

Table 1 Morphological characteristics of *Godronia cassandrae* (after Groves 1965).

Fungal structure	Colour	Shape	Size
Apothecia	Tawny to reddish brown to dark olivaceous brown	Separate or in clusters, sessile, narrowed below, subglobose, but opening circularly and becoming urceolate	0.5-1.0 mm diam. 0.3-0.8 mm height
Asci	-	Cylindrical to cylindrical-clavate, containing 8 ascospores	95.0-115.0 × 7.5-9.0 µm
Ascospores	Hyaline	Filiform, three to seven septate, pointed at ends, straight or curved	50.0-70.0 × 2.0-3.0 µm
Pycnidia	Black	Immersed to erumpent, mostly separate, subglobose	0.15-0.3 mm diam. and height
Conidiophores	Hyaline	Cylindrical to cylindrical-subulate, simple or branched, continuous or septate	6.0-25.0 × 1.5-2.0 µm
Conidia	Hyaline	Narrow fusiform, mostly straight, mostly two-celled, sometimes one-celled	7.0-18.0 × 1.5-3.5 µm

cassandrae based on cultural differences (Smerlis 1968). In a later experiment, an isolate from lowbush blueberry (*V. angustifolium*) was inoculated in four *Vaccinium*-species and two species of other genera; *V. corymbosum*, *V. myrtilloides* (velvet-leaf blueberry), *V. nubigenum* (Newfoundland bilberry), *V. uliginosum* (bog bilberry), *Ribes glandulosum* (skunk currant), and *Spiraea latifolia* (meadowsweet) (Smerlis 1969). All the *Vaccinium* species were infected, but the other two plant species were not. Lockhart and Delbrigde (1972) carried out an inoculation experiment on potted plants in the greenhouse and found that an isolate from *V. angustifolium*, inoculated on *V. angustifolium*, *V. corymbosum*, and *V. macrocarpon*, resulted in infections in all three species. However, when McKeen (1958) inoculated stems of field-grown highbush blueberry with an isolate pathogenic to cranberry and two isolates from highbush blueberry, only the blueberry isolates resulted in infection. It was also noticed that the conidial morphology of the cranberry isolate was different in that conidia were straighter and slightly larger than those from the blueberry isolates (McKeen 1958). To our knowledge, no further research has been aimed at investigating possible host-specialisation of *G. cassandrae* within the *Vaccinium* genus.

The anamorph

The name *Fusicoccum putrefaciens* Shear has been commonly used on the anamorphic stage of *G. cassandrae.* However, the correct name should be *Topospora myrtilli* Feltg., since *Topospora* was used on anamorphic stages of *Godronia* earlier than *Fusicoccum* (Groves 1965). Furthermore, the genus *Fusicoccum* was established based on *F. aesculi* Corda which, according to descriptions and illustrations, was not the conidial stage of *Godronia.* The type species of *Topospora*; *T. uberiformis*, is however, the conidial stage of *G. uberiformis* (Groves 1965). In the literature, the disease has been referred to as Godronia canker, Fusicoccum canker, blueberry canker and stem canker. Because there are several other pathogens that cause stem canker in blueberry, the latter two names should be avoided, and the name *Fusicoccum* should be avoided for reasons mentioned above. We use the genus name of the teleomorph, because generally only fungi without known sexual stages are referred to by the name of the anamorph. Below, the disease is therefore referred to as Godronia canker.

MORPHOLOGY

The morphology of the fungus on highbush blueberry has not been thoroughly described in the literature. The morphology of *G. cassandrae* was described by Groves (1965) and is summarised in **Table 1**. McKeen (1958) measured the diameter of apothecia, and length and width of asci, ascospores and conidia to be in approximately the same range as that described by Groves (1965). The size of the pycnidia (**Table 1**) agrees with measurements of pycnidia from lesions on stems of highbush blueberry in Massachusetts (Zuckerman 1960). However, Melzer and Hoffmann (1980a) in Germany found that the pycnidia were larger; 0.8-1.2 mm in diameter in lesions on stems of highbush blueberry, and 1.5 to 2 mm in pure culture. Furthermore, the conidia were slightly curved (Melzer and Hoffmann 1980a), which agrees with later publications (Ramsdell 1995; Szmagara 2009). Melzer and Hoffmann (1980a) described conidia in mass to have a pale pinkish to yellowish colour. Isolates from highbush blueberry in Massachusetts yielded yellowish colonies on potato-dextrose agar (PDA) (Zuckerman 1960). In Germany, two distinct mycelial types occurred when colonies from highbush blueberry were grown on PDA. One type was yellowish to greenish with sparse aerial mycelium, while the other was greyish with abundant aerial mycelium (Melzer and Hoffmann 1980a).

SYMPTOMS AND SIGNS

Lesion and pycnidia formation

The initial lesions on young stems are red to reddish brown and appear on one- and two year old stems. McKeen (1958) concluded from observations and results of inoculation tests that symptoms did not appear on current-season growth, but became visible during winter on stems formed the previous year, often centred around leaf scars. In Michigan, symptoms were observed as early as October, as water-soaked lesions which turned red by December (Weingartner and Klos 1975a). The lesions generally develop on the lower part of the stems, sometimes partly hidden by ground mulch, and as they enlarge, the bark in the middle turns grey and later brown, while the margins remain reddish (Creelman 1958). In some cases the lesions are dark brown or black (McKeen 1958), and sometimes they show a pattern of bands of different colours encircling the infection site, caused by alternating periods of growth and inactivity of the pathogen (Zuckerman 1960). Brown or black pycnidia may develop in the lesions. They may be scattered in an irregular pattern, but are often formed in concentric bands from the centre of the lesions (Sprau 1969). The lesions may reach a length of more than 10 cm (Creelman 1958; Menzinger and Krupp 1977), and lesions developing adjacent to each other usually coalesce (McKeen 1958).

Wilting of young stems

Complete girdling of stems generally causes wilting of the stems above the infection site, although it was observed in Michigan that completely girdled stems did not necessarily wilt (Weingartner and Klos 1975a). Girdling is usually completed in one season, but partially girdled stems may survive into the next growing season (Creelman 1958). Wilting occurs rapidly during periods of dry, warm weather, often within a few hours (Creelman 1958). McKeen (1958) reported that the disease generally killed two to three stems in each bush, and therefore young bushes were more exposed to complete wilting than older plants which contained more stems. In British Columbia (McKeen 1958) and Massachusetts (Zuckerman 1960), wilting of stems started in May and continued throughout the summer. In Massachusetts, it was observed that stems with small lesions in February were girdled and killed within 18 weeks (Zuckerman 1960). It was assumed that the length of this period depended on climatic conditions, so that there would be variations from one year to the next. In Michigan, wilting of stems started in April, but most wilting occurred from mid-June (Weingartner and Klos 1975a). In Norway, small red lesions

Fig. 1 Stem pieces of highbush blueberry (*Vaccinium corymbosum*) with symptoms of Godronia canker caused by *Godronia cassandrae* f. sp. *vaccinii*. Photo: Rolf Langnes, Bioforsk.

started to appear on one-year old stems in March, and during April these lesions expanded and pycnidia generally appeared in the lesions (Strømeng and Stensvand, unpublished data) (**Fig. 1**). Infected stems started to wilt in June, but living stems with symptoms of Godronia canker were observed all through the growing season, and as observed in Nova Scotia (Creelman 1958), stems that were only partially girdled could survive another winter (Strømeng and Stensvand, unpublished data).

On older wood

In woody canes older than two years, the disease caused fissures and flaking of the bark (Menzinger and Krupp 1977), and pycnidia were observed in both lesions and bark fissures (Weingartner and Klos 1975a). In North America, apothecia have occasionally been observed at the base of branches three years or older (Creelman 1958; McKeen 1958; Zuckerman 1960; Weingartner and Klos 1975a).

Stem galls

Weingartner and Klos (1975a) observed stem galls in association with cankers in Michigan, but it was not determined if this was caused by *G. cassandrae* f. sp. *vaccinii*. Stem galls in association with Godronia canker were also reported from Germany (Menzinger and Krupp 1977; Melzer and Hoffmann1980a).

On leaves

In Nova Scotia, symptoms caused by *G. cassandrae* f.sp. *vaccinii* were observed on leaves of highbush blueberry (Lockhart 1970). Lesions were circular, light brown to dark brown, with darker margins, and pycnidia developed when detached leaves were incubated in saturated air. In Germany, artificial inoculations with *G. cassandrae* f.sp. *vaccinii* on leaves of highbush blueberry resulted in symptom development, and pycnidia of the fungus started to form two weeks after inoculation (Melzer and Hoffmann 1980b).

On fruits

The fungus has never been reported to cause symptoms on blueberry fruits.

BIOLOGY AND EPIDEMIOLOGY

Fungal growth in stem tissue

G. cassandrae f. sp. *vaccinii* infects young stems that are not yet lignified. Infections most often occur close to the ground level on stems coming from the base of the bushes (Creelman 1958; McKeen 1958). Time from infection to dieback generally takes one year (over two growing seasons). *G. cassandrae* f. sp. *vaccinii* overwinters as mycelium in the infection sites on stems, or in infected crowns (Creelman 1958). According to Creelman (1958), lesions on stems could result from spores infecting stems, or from mycelium in the crowns invading the stems, but the latter has not been suggested by other authors. Weingartner and Klos (1975b) examined alterations in the stem tissue after natural infections by *G. cassandrae* f. sp. *vaccinii*. They found intracellular growth by the fungal hyphae, but hyphae growing into living cells were never observed. The xylem started to turn brown by the time the lesions were about 1 cm long. In wilted stems, 30-40% of the xylem was occluded by hyphae and fungal deposits. It was concluded that the stems probably died due to occlusion of the vessels (Weingartner and Klos 1975b), which agrees with the sudden wilting that often occurs during warm and dry weather.

Time of pycnidia formation

Weingartner and Klos (1975a) observed pycnidia in Michigan from March to June, which was similar to findings in Massachusetts (Zuckerman 1959). In British Columbia (McKeen 1958), Russia (Mukhina and Sizova 1984), and Germany (Menzinger and Krupp 1977), pycnidia were also observed in spring. In Norway, pycnidia were observed from April and throughout the growing season (Strømeng and Stensvand, unpublished data). On the other hand, Creelman (1958) in Nova Scotia reported that pycnidia appeared in the lesions from late July and were visible for about five weeks. When the pycnidia mature, they tear open at the top (Groves 1965), and conidia are exuded during rainy periods (Weingartner and Klos 1975a).

Seasonal distribution of conidia

In Michigan, it was demonstrated that conidia were mainly water dispersed by trapping both airborne and waterborne spores in the field (Parker and Ramsdell 1977). Moreover, Parker and Ramsdell (1977) investigated the seasonal occurrence of conidia in Michigan during two consecutive growing seasons. They found that conidia were present in the field throughout the growing season, but the highest peak occurred in the last half of May, and the amount of conidia decreased during June. In August and September, conidia were more abundant, but in November very few conidia were found. In Norway, a similar pattern was found in a study carried out during three years, where stem lesions from highbush blueberry were sampled regularly during the season, and numbers of conidia were recorded (Strømeng and Stensvand, unpublished data). In Germany, conidia were present all year in 1974 and 1975 (Menzinger and Krupp 1977), but the numbers were not quantified. The authors suggested that the mild winters could explain presence of conidia at that time of the year.

Time of infection

An experiment aimed at determining the major periods for infection was carried out in Michigan by placing two-year-old potted highbush blueberry plants beneath heavily diseased bushes in the field for periods of one month during the growing season, after which the potted plants were placed in isolation from inoculum sources (Parker and Ramsdell 1977). Assessment of disease development was carried out the following year. The main periods of infection occurred from April to mid June and from mid August to the end of September (Parker and Ramsdell 1977). A similar pattern was reported by Weingartner and Klos (1969) in Germany.

Infection sites

The fungus infects the host tissue trough wounds, including leaf scars, but it is also capable of penetrating intact tissue (Weingartner and Klos 1975b; Parker and Ramsdell 1977).

Weingartner and Klos (1975b) observed hyphal penetration trough stomata of intact stem tissue. Nevertheless, Melzer and Hoffmann (1975b) concluded that infection through intact tissue required optimal conditions for the fungus and was less likely to occur under field conditions. In a field where the bushes were naturally infected, 94% of the lesions larger than 1.5 cm developed around leaf scars, and 44% of all the lesions examined developed around leaf scars (Weingartner and Klos 1975a). However, if the spring and early summer is an important period for infection, it seems less probable that these infections occurred through leaf scars. Weingartner and Klos (1969) carried out spray inoculations on bushes two to three weeks prior to leaf drop in the autumn, and this resulted in more than 80% of the lesions developing around leaf scars. Furthermore, *G. cassandrae* f. sp. *vaccinii* was isolated from 45% of attached leaf petioles that were collected in October. It was suggested that most infections of leaf scars occurred via attached leaf petioles prior to leaf drop (Weingartner and Klos 1969). Regarding that spring is an important period for conidia formation and infection, it may be speculated if the fungus is able to infect young leaves in the spring without causing symptoms. In a study of seasonal incidence of fungi in leaves and fruits of cranberry, *G. cassandrae* was detected in symptomless leaves from budbreak (early May) until harvest (late September) (Jeffers 1991). Melzer and Hoffmann (1980b) reported that *G. cassandrae* f. sp. *vaccinii* could infect leaves of highbush blueberry under optimal conditions, and Lockhart (1970) observed leaf spots caused by *G. cassandrae* f. sp. *vaccinii* on highbush blueberry in Nova Scotia in summer and autumn.

Temperature requirements

The fungus seems well adapted to grow at the low temperatures occurring in spring and autumn. Optimum temperature for mycelial growth on PDA has been reported to be 20°C (Melzer and Hoffmann 1980a), 22°C (Parker and Ramsdell 1977), and 18–22°C (Szmagara and Zalewska 2008). Growth was limited at 30°C (Melzer and Hoffmann 1980a). However, *G. cassandrae* f. sp. *vaccinii* was able to maintain growth at a slow rate at temperatures close to 0°C (Creelman 1958; Szmagara and Zalewska 2008). Although the optimum temperature range for germination of conidia was 20–25°C (100% germination after 72 hrs), conidia still germinated at 2°C (10% germination after 96 hrs), as long as high humidity was provided (Melzer and Hoffmann 1980a). Optimum temperature for production of pycnidia in culture was 15°C (Melzer and Hoffmann 1980a). Szmagara and Zalewska (2008) found that the colony growth rate of three different isolates on PDA were significantly higher at 12 than at 28°C, and the growth at 28°C were more equal to the growth rate at 2 and 6°C. Similar results were obtained for a *G. cassandrae* f. sp. *vaccinii* isolate from lowbush blueberry in Nova Scotia (Lockhart 1975). When lowbush blueberry plants were inoculated and placed at two different temperature regimes; 4.5°C night/15°C day or 9.5°C night/21°C day, the cankers on the stems developed faster at the lowest temperature regime, with an average lesion length of 7.9 cm after 6–8 months compared to 5.0 cm at the highest temperature (Lockhart 1975). The poor growth at temperatures above 22°C may explain why inoculations carried out in the field during July failed to cause infection (McKeen 1958; Lockhart and Craig 1967).

Enzyme production

An investigation of mycelial growth on agar containing different nutrient sources; glucose, saccarose, maltose, starch, pectin or cellulose, showed that the growth rate was similar regardless of nutrient source and that the fungus was able to make use of different polysaccharides (Melzer and Hoffmann 1980a). The enzymes pectinase, cellulase and oxidase were produced by the fungus at all temperatures tested, and the activity of these enzymes was sufficient at temperatures below 10°C, although the activity was highest at 10-15°C.

Fungal growth affected by pH

In culture, the fungus grew well at pH from 4 to 6, while the growth was markedly reduced at pH 3-3.5 which is the pH of ripening blueberry fruits, and this could explain why the fungus does not infect fruit (Melzer and Hoffmann 1980a).

Importance of the teleomorph

The role of the teleomorphic stage of the fungus is unclear, and to our knowledge, it has never been reported from Europe. In British Columbia, apothecia only appeared occasionally, and ascospores were assumed to be of little importance for disease development (McKeen 1958). In Nova Scotia, apothecia were observed on wood that was at least three years old, and it was suggested that both conidia and ascospores initiated stem cankers (Creelman 1958). Weingartner and Klos (1975a) found abundant apothecia in Michigan, which suggested that they were of importance in the life cycle of the pathogen. Immature apothecia were observed in April, and they generally matured by July. However, Parker and Ramsdell (1977) in Michigan only trapped trace numbers of ascospores in the field. They also observed heavy infections in highbush blueberry in areas where apothecia were not observed, which indicated that ascospores were relatively unimportant for the disease development. Inoculations with ascospores failed to cause infections in field experiments in Michigan (Parker and Ramsdell 1977).

CULTIVAR SUSCEPTIBILITY

Different opinions on susceptibility

Cultivars may differ greatly in susceptibility to Godronia canker. For some cultivars, there is agreement on the susceptibility between different geographical regions, but for other cultivars opinions may differ. Disagreements could be due to differences in climate between regions and also cultural factors, both decisive for how the plants thrive, and it is likely that stressed plants are more susceptible to disease than plants well adapted to their growing conditions. Disease severity would also likely be affected by how old the plants are at the time of infection, because older plants containing an older, less susceptible basis of stems may more easily resist dieback.

Differences in disease tolerance

There are indications that the resistance is based on restraining growth of the pathogen after infection by development of callus barriers (Melzer and Hoffmann 1980b), so that resistant cultivars may not necessarily be symptom-free. Melzer and Hoffmann (1980b) observed that in detached, inoculated stems of different cultivars, lesions developed twice as fast in ‘Jersey’ as in ‘Hardyblue’ (13-16-A) and ‘Rancocas’. In Michigan, it was observed that ‘Rancocas’ was often severely cankered, but was much more resistant to wilting than other cultivars (Weingartner and Klos 1975a). In an experimental field in Germany more than 30% of the plants of ‘Berkeley’, ‘Heerma I’ and Heerma II’ showed symptoms of Godronia canker, but only ‘Berkeley’ plants died from the attack (Menzinger and Krupp 1977). In Norway, the cultivars ‘Hardyblue’ and ‘Patriot’ showed severe symptoms of Godronia canker on the stems in a few fields with a high disease level, but no wilting was observed (Strømeng and Stensvand 2001).

Ranking of susceptibility

We have assessed the relative susceptibility of cultivars on a scale from 1 to 5, where 1 is highly resistant and 5 is highly

Table 2 Assessment of relative susceptibility of highbush blueberry cultivars to *Godronia cassandrae* f. sp. *vaccinii* based on reports from five countries (scale: 1 = highly resistant, 2 = resistant, 3 = intermediate, 4 = susceptible, 5 = highly susceptible).

Cultivar	Nova Scotia, Canada[1]	Oregon, USA[2]	Germany[3]	Norway[4]	Switzerland[5]
Ama				2	
Berkeley	3	5	5	3-4	4
Bluecrop	4	5	1	3-4	3
Blueray	3			5	4
Bluetta				2	
Burlington	2				
Collins				4	2
Concord	1				
Coville	3				4
Darrow			1		5
Duke				3-4	
Earliblue	4	5		5	
Goldtraube			1-2	1	3
Hardyblue				1	
Heerma			5	3	3-4
Herbert			2		4
Ivanhoe			3	2	2
Jersey	5	5	5	5	
Johnson	4				
Patriot				2	
Pemberton		5			
Pioneer	3				
Rancocas	1	1			
Rubel		1			
Stanley	2				

[1]Lockhart and Craig (1967), [2]Pscheidt (2010), [3]Menzinger and Krupp (1977), [4]Strømeng and Stensvand (2001), [5]Kobel (1977)

susceptible (**Table 2**). The assessments are based on five publications containing the most detailed information on cultivar susceptibility (Lockhart and Craig 1967; Kobel 1977; Menzinger and Krupp 1977; Strømeng and Stensvand 2001; Pscheidt 2010). 'Jersey' is ranked as very susceptible, and several other authors have also reported that 'Jersey' has been among the most susceptible cultivars (Conners and Savile 1952; Hårdh 1959; Weingartner and Klos 1975a). 'Earliblue' is also considered susceptible to highly susceptible, while 'Berkeley' and 'Blueray' have been considered from intermediately to highly susceptible (**Table 2**). The cultivar 'Bluecrop', which is a very important cultivar for commercial highbush blueberry production worldwide, has been considered both highly resistant and highly susceptible (**Table 2**). 'Goldtraube' has been considered intermediate to highly resistant (**Table 2**), which is in agreement with Liebster (1979). However, 'Heerma' is considered as intermediate to highly susceptible (**Table 2**), but was regarded as resistant by Liebster (1980). 'Rancocas' is considered highly resistant (**Table 2**), which agrees with Weingartner and Klos (1975a).

CONTROL

Disease resistance

Selecting cultivars with high degree of resistance to Godronia canker is likely the most important control measure.

Disease-free planting material

Since *G. cassandrae* f. sp. *vaccinii* is particularly severe in young bushes, it is important to use disease-free planting material. Plantlets propagated from diseased fields may carry the disease (Creelman 1958). Wilcox and Falconer (1961) found severe outbreaks of Godronia canker on newly established, imported highbush blueberry plants in Scotland, where the disease had not been found earlier. In Germany, Sprau (1969) discussed the need for quarantine regulations when importing young plants from USA due to the many diseases that occurred on highbush blueberry there. In Norway, it was observed that micro propagated, disease free planting material developed far less Godronia canker a few years after planting than planting material propagated by cuttings (Strømeng 1999).

Cultural practices

Pruning and removal of diseased stems is important, but in many cases not sufficient to eradicate the fungus from a field (Creelman 1958; Parker and Ramsdell 1977). In established fields it is possible to maintain a satisfactory control level by pruning, but heavy pruning in young fields can have adverse effects on the plants (Creelman 1958). Redundant and late fertilisation gives vigorous vegetative growth and weak shoots and should be avoided (Liebster and Sprau 1970). Creelman (1958) reported that a heavy layer of sawdust for weed control favoured disease development at ground level due to high humidity at stem basis.

Chemical control

At present, there are no fungicides available that control Godronia canker at a satisfactory level. Attempts to control the disease by applying fungicides often failed or only had effect when the treatments were repeated at short intervals (Creelman 1958; Zuckerman 1960; Sprau 1969; Menzinger and Krupp 1977). In Germany, a copper fungicide reduced the disease incidence if applied once in autumn after leaf fall and once in spring before bud burst (Melzer and Hoffmann 1980b). Studies on control of Godronia canker by two biopreparations of animal or plant origin or mancozeb were recently carried out on potted 'Jersey' plants under controlled conditions in Poland (Szmagara 2008). Stems were wound inoculated with conidia and mycelia of *G. cassandrae* f. sp. *vaccinii* and then immediately treated. Disease assessment after 27 days showed that only mancozeb significantly reduced the number of infections compared to the positive control. The result was the same for the size of the lesions and number of pycnidia produced in the lesions (Szmagara 2008).

CONCLUSION

G. cassandrae f.sp. *vaccinii* causes severe stem wilting in highbush blueberry, and the fungus seems to be adapted to relatively cool, humid regions such as in northern Europe, Canada and northern USA. The ascigerous stage of the fungus seems to have limited importance as inoculum and is not found in Europe. Conidia are spread throughout the growing season, but the highest releases seem to occur in spring and early summer. Stem infections can take place during the entire growing season, but high temperatures probably limit infections during summer. Leaves can become infected and probably serve as inoculum sources for stem infections. Lesions on young stems develop rapidly in spring the year following infection, but may easily be overlooked because they often appear at the lower part of stems, or even partly hidden under ground mulch. Thus, sudden wilting of stems during periods of warm weather may be the first symptoms observed. It is difficult to control the disease with fungicides, mainly because infections may occur over a wide time period, and there seem to be few or no fungicides with high efficacy. Consequently, disease free planting material, well aerated plantings, and most importantly use of less susceptible cultivars are the best options in controlling Godronia canker. Diseased stems should be pruned out as soon as they are detected in the field, and although resistant cultivars may not wilt from the attack, pruning is important to keep a low inoculum pressure in the field.

REFERENCES

Anonymous (1997) Crop profile for highbush blueberry in New Jersey. Rutgers New Jersey Agricultural Experiment Station. Available online: http://www.pestmanagement.rutgers.edu/NJinPAS/index.htm

Barnes EH, Tweedie HC (1964) *Fusicoccum* canker of highbush blueberry in Michigan. *Plant Disease Reporter* **48**, 687-689

Borecki Z, Pliszka K (1978) Canker of highbush blueberry caused by *Godronia cassandrae* f. *vaccinii* (Peck.) Groves. *Acta Agrobotanica* **31**, 159-171

Caruso FL, Ramsdell DC (1995) *Compendium of Blueberry and Cranberry Diseases*, APS Press, St. Paul, MN, USA, 87 pp

Conners IL (1932) *Canadian Plant Disease Survey*, 11th Annual Report, Canadian Department of Agriculture, p 67

Conners IL, Savile DBO (1944) *Canadian Plant Disease Survey*, 23rd Annual Report, Canadian Department of Agriculture, p 92

Conners IL, Savile DBO (1948) *Canadian Plant Disease Survey*, 27th Annual Report, Canadian Department of Agriculture, p 96

Conners IL, Savile DBO (1952) *Canadian Plant Disease Survey*, 31st Annual Report, Canadian Department of Agriculture, p 100

Creelman DW (1958) *Fusicoccum* canker of the highbush blueberry especially with reference to its occurrence in Nova Scotia. *Plant Disease Reporter* **42**, 843-845

Eriksson B (1970) On ascomycetes on Diapensiales and Ericales in Fennoscandia. *Symbolae Botanicae Upsaliensis XIX* **4**, 71 pp

Fuqua B, Byers P, Kaps M, Kovacs L, Waldstein D (2005) Growing blueberries in Missouri. Bulletin 44, 47 pp. Available online: http://mtngrv.missouristate.edu/Publications

Groves JW (1965) The genus *Godronia*. *Canadian Journal of Botany* **43**, 1195-1276

Hårdh JE (1959) Pensasmustikan viljelyä haittavista tekijoistä suomessa [Factors affecting blueberry culture in Finland]. *The Journal of the Scientific Agricultural Society of Finland (currently: Journal of Agricultural Science in Finland)* **31**, 131-140 (in Finnish)

Jeffers SN (1991) Seasonal incidence of fungi in symptomless leaves and fruit treated with fungicides during bloom. *Phytopathology* **81**, 636-644

Kobel F (1977) Experiments on highbush blueberries in Switzerland. *Acta Horticulturae* **61**, 117-120

Liebster G (1979) Kulturheidelbeer-Sortenversuch in sechs europäischen Ländern [Highbush blueberry cultivar trial in six European countries]. *Obstbau* **4**, 428-432 (in German)

Liebster G (1980) Das durch den Pilz *Godronia cassandrae* Peck. Hervorgerufene Triebsterben an Kulturheidelbeeren [Stem blight of highbush blueberry caused by the fungus *Godronia cassandrae* Peck]. *Obstbau* **5**, 384-387 (in German)

Liebster G, Sprau F (1970) Das Triebsterben bei Kulturheidelbeeren [Stem blight in highbush blueberry]. *Erwerbsobstbau* **12**, 220-223 (in German)

Lockhart CL (1970) Leaf spot of highbush blueberry caused by *Godronia cassandrae* f. *vaccinii*. *Canadian Plant Disease Survey* **50**, 93-94

Lockhart CL (1975) Effect of temperature on the development of *Godronia cassandrae* f. *vaccinii* cankers on lowbush blueberry. *Canadian Plant Disease Survey* **55**, 29-30

Lockhart CL, Craig DL (1967) *Fusicoccum* canker of highbush blueberry in Nova Scotia. *Canadian Plant Disease Survey* **47**, 17-20

Lockhart CL, Delbridge RW (1972) Occurrence and pathogenicity of *Godronia cassandrae* f. *vaccinii* on lowbush blueberry in Nova Scotia. *Canadian Plant Disease Survey* **52**, 119-121

McKeen WE (1958) Blueberry canker in British Columbia. *Phytopathology* **48**, 277-280

Melzer RR, Hoffmann GM (1980a) Triebsterben an der Kulturheidelbeere (Erreger: *Godronia cassandrae* Peck) [Stem blight in highbush blueberry (cause of disease: *Godronia cassandrae* Peck)]. *Gartenbauwissenschaft* **45**, 7-14 (in German)

Melzer RR, Hoffmann GM (1980b) Pathogenität und Bekämpfung von *Godronia cassandrae* Peck an *Vaccinium corymbosum* Lin. [Pathogenicity and control of *Godronia cassandrae* Peck on *Vaccinium corymbosum* Lin.]. *Gartenbauwissenschaft* **45**, 89-96 (in German)

Menzinger W, Krupp J (1977) Dieback disease on highbush blueberries caused by *Godronia cassandrae*. *Acta Horticulturae* **61**, 197-200

Mukhina LN, Sizova TP (1984) [Identification of *Godronia cassandrae* Pk., a new fungus for the USSR]. *Mikologiya i Fitopatologiya* **18**, 70-72 (in Russian) (Abstract in Review of Plant Pathology (1984) **63**, 326)

Mukhina LN, Danilova IA, Golovkina IN (1993) [Field resistance in *Vaccinium* to *Godronia*]. *Byulleten Glavnogo Botanicheskogo Sada*, No. 167, Glavnyĭ Botanicheskiĭ Sad, Moscow, Russia, pp 112-116 (in Russian) (Abstract in *Review of Plant Pathology* (1994) **73**, 551)

Oblak M (1977) Advanced testing of blueberry varieties in Slovenia. *Acta Horticulturae* **61**, 145-149

Parker PE, Ramsdell DC (1977) Epidemiology and chemical control of *Godronia* (*Fusicoccum*) canker of highbush blueberry. *Phytopathology* **67**, 1475-1480

Pscheidt JW (Ed) (2010) Blueberry – Godronia canker. 2010 PNW Plant Disease Management Handbook. Oregon State University Extension Service. Available online: http://ipmnet.org/plant-disease/disease.cfm?RecordID=180

Plizka K (1997) Overview on *Vaccinium* production in Europe. *Acta Horticulturae* **446**, 49-52

Shear CL (1917) Endrot of cranberries. *Journal of Agricultural Research* **11**, 35-42

Shear CL, Bain HF (1929) Life history and pathological aspects of *Godronia cassandrae* Peck (*Fusicoccum putrefaciens* Shear) on cranberry. *Phytopathology* **19**, 1017-1024

Smerlis E (1968) The occurrence and pathogenicity of forms of *Godronia cassandrae* in Quebec. *Canadian Journal of Botany* **46**, 597-599

Smerlis E (1969) Pathogenicity of some species of *Godronia* occurring in Quebec. *Plant Disease Reporter* **53**, 807-810

Sprau F (1969) Untersuchungen über das Rutensterben bei Kulturheidelbeeren [Investigations on twig blight in highbush blueberry]. *Mitteilungen aus der Biologischen Bundesanstalt für Land und Forstwirtschaft* **132**, 132-135 (in German)

Stensvand A, Langnes R (1995) Greindød – ny soppsjukdom i hageblåbær [Godronia canker – a new fungal disease in highbush blueberries]. *Gartneryrket* **86 (8)**, 25-26 (in Norwegian)

Strømeng GM (1999) Stem canker caused by *Godronia cassandrae* f. sp. *Vaccinii* in highbush blueberry. Diploma thesis, Agricultural University of Norway, 84 pp (in Norwegian)

Strømeng GM, Stensvand A (2001) Susceptibility of highbush blueberry (*Vaccinium corymbosum* L.) cultivars to Goderonia canker (*Godronia cassandrae* f. sp. *vaccinii*) in Norway. *Gartenbauwissenschaft* **66**, 78-84

Szmagara M (2008) Possibilities of growth and development suppression of *Topospora myrtilli* (Feltg.) Boerema on artificial media and stems of highbush blueberry (*Vaccinium corymbosum* L). *Acta Scientiarum Polonorum, Hortorum Cultus* **7 (3)**, 103-111

Szmagara M, Zalewska E (2008) The effect of cultivation conditions on growth, sporulation and formation of morphological structures of *Topospora myrtilli* (Feltg.) Boerema. *Acta Agrobotanica* **61**, 167-174

Szmagara M (2009) Biodiversity of fungi inhabiting the highbush blueberry stems. *Acta Scientiarum Polonorum, Hortorum Cultus* **8 (1)**, 37-50

Vestrheim S, Haffner K, Grønnerød K (1997) Highbush blueberry production and research in Norway. *Acta Horticulturae* **446**, 177-180

Weingartner DP, Klos EJ (1969) Seasonal infection and sites of infection by *Godronia cassandrae* f. *vaccinii*. *Phytopathology* **59**, 1056

Weingartner DP, Klos EJ (1975a) Etiology and symptomatology of canker and dieback diseases on highbush blueberries caused by *Godronia* (*Fusicoccum*) *cassandrae* and *Diaporthe* (*Phomopsis*) *vaccinii*. *Phytopathology* **65**, 105-110

Weingartner DP, Klos EJ (1975b) Histopathology of blueberry stems naturally infected with *Godronia cassandrae*. *Phytopathology* **65**, 1327

Wilcox HJ, Falconer MA (1961) New or uncommon plant diseases and pests. *Plant Pathology* **10**, 123-124

Zuckerman BM (1959) *Fusicoccum* canker of the highbush blueberry in Massachusetts. *Plant Disease Reporter* **43**, 803

Zuckerman BM (1960) Studies of two blueberry stem diseases recently found in Eastern Massachusetts. *Plant Disease Reporter* **44**, 409-415