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DISEASES OF LOWBUSH BLUEBERRY AND THEIR IDENTIFICATION

Canada

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The authors of this text:

P.D. Hildebrand, Agriculture and Agri-Food Canada, Kentville, NS.
W.E. Renderos, Agriculture and Agri-Food Canada, Kentville, NS.
R.W. Delbridge (1949-2015), Delbridge Disease Management, Kentville, NS.

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Introduction

The lowbush blueberry plant (*Vaccinium angustifolium, V. myrtilloides*) is susceptible to many diseases, some of which are more serious than others. Several diseases require intense management while others, despite being present annually, do not significantly impact the crop and do not require any management efforts. An accurate diagnosis of a disease and its cause is therefore essential in making wise decisions on implementing control measures.

The purpose of this guide is to aid in the identification of diseases commonly found in lowbush blueberry fields in Eastern Canada and to assist in disease management decisions. Time of symptom development is based on observations in Nova Scotia, and so anticipated times of symptom development in other provinces may need to be adjusted to account for seasonal differences. Discussions on diseases in this guide are arranged in categories of importance with those of high importance presented first.

In general, plant pathogens that cause disease exist in the form of microscopic fungi, bacteria and viruses. Lowbush blueberries are commonly affected by fungal pathogens and typically not by bacteria or viruses which can be more difficult to manage. Most fungal pathogens are highly influenced by weather conditions and so disease may be serious in one year and less so in another year. Thus, it is important to keep track of diseases that are developing or have been a problem in the past.

Fungal pathogens that attack blueberry reproduce by the formation of spores. Spores are microscopic bodies that exist in a variety of sizes, shapes and colour and are spread by a variety of means such as wind, rain or even by human activity. Some are designed for reproduction purposes only (e.g. conidia, urediniospores) while others are involved in fungal genetic exchange (e.g. ascospores, aeciospores). Spores of each pathogen have the ability to germinate on leaf surfaces within defined ranges of temperature and wetness duration, much like seeds of plants. Once germination and growth into the host tissue has occurred, there is a latent period of a few days, several weeks or even months before disease symptoms appear. Each fungal pathogen and the resulting symptom (wilting and dying of leaves and stems, spots on leaves and stems, shrivelling berries etc.) are unique to the disease in question and thus aid in its accurate diagnosis.

From a disease perspective, there is perhaps no other production practice in the lowbush blueberry industry that has influenced diseases as much as pruning. Blueberries are grown primarily on a two-year production cycle where fruit are harvested in one year and then stems are pruned to ground level to encourage regrowth of straight, strong stems with flower buds. Years ago, burn pruning was the principal means of pruning blueberry fields. Straw was spread onto fields and then ignited during dry periods in early spring or fall. The advent of oil-fired tractor-drawn burners replaced straw and more recently the advent of flail mowing largely replaced the use of fire for economic and environmental reasons. Most of the fungal pathogens affecting blueberry overwinter (survive) in leaf and stem debris and the practice of repeated burn pruning consumed much of this infected debris

and was therefore an unintentional benefit in controlling diseases. Flail mowing, on the other hand, has no effect on pathogen survival and so severity of some existing diseases has increased markedly and new ones have appeared. Unfortunately, a return to burn pruning in only a single year may not effectively destroy all infected debris thereby allowing disease to build-up rapidly again. Also, spores of some pathogens can travel long distances (e.g. leaf rust) on air currents so all fields in a local area or region would need to be burn pruned repeatedly for effective control. Recently, some growers have experimented with sickle-bar mowing in the fall and then igniting the dried stems in the spring to achieve a relatively hot burn. Where feasible, this practice appears to reduce many diseases that overwinter within the field.

The shift away from burn pruning has resulted in an increased reliance on fungicides. While fungicides can be effective for managing many diseases, they need to be applied at the right time in relation to the life cycle of a pathogen. If they are applied too early or late, their effectiveness will not be realized. For almost all diseases, fungicides should be applied early in the development of the pathogen's life cycle and as protectant sprays that protect the plant from infection by spores. There is only one disease, Monilinia blight, for which some fungicides can be effectively applied after infection. Good spray coverage is essential for good disease control. While some fungicides are known to have systemic activity (movement within the plant) which helps to improve effectiveness, this does not mean that one can be more relaxed about spray coverage and rely on movement of the chemical throughout the plant. The degree of systemic movement of almost all modern fungicides is limited to the chemical moving only from the top leaf surface to the lower leaf surface and/or a few millimeters laterally from a spray droplet and not, for example, from one leaf to another. Provincial production guides should be consulted for recommended and timing of fungicides.

The increased use of fertilizer has also, in some cases, resulted in increased disease pressure. Increased levels of nitrogen may cause the plant to become intrinsically more susceptible to infection. Fertilizers may also increase the density and height of stems within fields. Dense canopies can remain wet for long periods further favouring many diseases by promoting spore infections and subsequent spore production. Provincial production guides should be consulted for appropriate use of fertilizers.

MONILINIA BLIGHT (MUMMY BERRY)

(Monilinia vaccinii-corymbosi)

Importance: high

Symptoms

Monilinia blight is the earliest occurring disease in the growing season and can lead to substantial losses if not controlled (Fig. 1). The disease affects leaves, flowers and fruit of the blueberry plant. The earliest symptom of Monilinia blight is a drooping of developing leaves and shoots beginning in late May, followed shortly (24 h) by a dark browning of the midrib and veins of leaves (Fig. 2). Infected flower clusters become dark purple-brown in colour and shrivel (Fig. 3). A whitish-grey growth is produced on the axis of leaf shoots and midveins of infected leaves (Fig. 2) and at the base of flowers (Fig. 3). All infected tissue eventually falls off the plant. After the spring symptoms, affected plants appear disease free until the berries begin to ripen. Infected berries become cream to salmon pink and eventually turn tan or silvery gray in early August (Fig. 4). Initially soft, they eventually shrivel, harden and drop to the ground, usually before commercial harvest. The skin is eventually sloughed off, exposing a hard, black fungal mass called a mummy berry (pseudosclerotium).

Disease Cycle

Mummy berries are the overwintering structure of the fungus and can remain viable in the blueberry leaf litter for several years. During bud break in late April and early May, the mummy berries germinate to produce small cup-like structures called apothecia (Fig. 5). The inside of the cup is lined with microscopic sacs that contain primary spores (ascospores; Fig. 6). The spores are released by a puffing action in response to changes in humidity and wind (Fig. 7). Moist soils and temperatures between 10 and 16 C favor emergence of apothecia. At higher temperatures (20-25 C) apothecia mature more rapidly but also senesce rapidly, whereas at lower temperatures (10-16 C) they last longer and therefore produce more spores. Exposure to freezing temperatures, as low as -6 C, reduces spore production and release, but only temporarily. Viability of ascospores exposed to freezing temperatures down to -8 C is reduced but not eliminated. Apothecia typically release ascospores over a 3-week period in Atlantic Canada after which they expire and the mummy berry disintegrates. Unseasonably low temperatures during May can extend the period of ascospore release, while unusually warm, dry conditions may cause the apothecia to dry up earlier than usual thereby reducing the period of ascospore release. In some years, conditions may be too hot and dry for apothecia to develop at all and the threat of disease is minimal. In these cases, the mummy berries will go dormant for the remainder of the growing season until the following spring.

Flower buds are resistant to ascospore infections when they are still tight, but they become susceptible to infection when they reach the F2 stage (bud scale leaves beginning to separate giving the appearance of a crown) (Fig. 8) of development and continue to be susceptible at later stages. Similarly, leaf buds become susceptible to infection at the V2 stage (primary leaves extending 2-5 mm, but not yet unfolding) (Fig. 9) and thereafter. Development of apothecia generally coincides with these early stages of bud development, but differences in timing of bud development among lowbush blueberry clones often leads to patchiness of symptoms in a field.

Infection of susceptible tissue by ascospores requires free water from rain, fog or wet snow and the degree of infection is dependent on temperature. For example, infection occurs within as little as 4 hours at the optimal temperature of 18 C, but requires 10 hours at 2 C. Field frost during bud development can increase susceptibility to infection, and this heightened effect can last 4 days. Once infection has occurred, disease symptoms become evident in 10-20 days, typically in late May. Secondary spores (conidia) are produced on diseased tissues and appear as a whitish-gray growth (Fig. 1, 2). The conidia are carried by wind and pollinating insects to open flowers where infection occurs. The fungus grows down the stigma of the flower into the ovary (eventual berry) and remains latent until shortly before harvest when it begins to aggressively colonize the fruit to produce a mummy berry. Newly opened flowers are more susceptible to infection than are older flowers and incidence of infected berries is reduced if pollination precedes the arrival of conidia.

Disease is usually more severe in low-lying, moist areas of fields where conditions favor the production of apothecia and where plants are subject to spring frosts that may affect their susceptibility. Mummy berries floating on water may also drain into these areas creating a more concentrated source of ascospores available for infection.

Disease symptoms may occasionally be observed on new shoots in sprout fields if favourable weather conditions and early development of sprouts occur simultaneously (Fig. 10), but the effect on sprout stem density is usually not of concern.

Disease Management

Properly timed fungicides can be highly effective in managing this disease. The decision to spray for this disease depends almost entirely on past disease severity of blight in a particular field. The first fungicide spray should be applied when 40-50% of the flower buds have reached the F2 stage. This usually coincides with similar development of the vegetative buds (V2). A second application is usually needed in 7-10 days to protect all developing plant tissue when ascospores are present. A third application is rarely required. Subsequent applications in an effort to control the secondary conidia are usually not cost effective. An alternative spray strategy that may reduce the number of fungicide applications in some years relies on the systemic activity of some fungicides and knowledge of when an infection period has occurred. By monitoring weather conditions and estimating when infection has occurred, applications of fungicides with back action can be applied up to 72 hours after the beginning of an infection period. Monilinia blight is the only disease of lowbush blueberry where the back action of fungicides can be effectively employed. If frost occurs during the early morning hours and a fungicide application is planned, the application should be delayed until the temperature rises a few degrees above freezing. If fungicides are applied under freezing conditions, severe burning of the developing buds may occur. The fine mist from the sprayer becomes super cooled at low temperatures causing tissue injury on contact.

Burn pruning can substantially reduce this disease by destroying the mummy berries, but this practice has largely been replaced by flail mowing which has no effect on mummy berries. A return to burn pruning may take several cycles to effectively reduce disease pressure in severely infested fields since the mummy berries become buried and insulated from the heat by the leaf litter thereby remaining viable for several years.

Fig. 1
Severe disease resulting from improper timing of fungicide applications

Fig. 2
Blighted leaf shoots resulting from ascospore infections. Conidia are produced on diseased tissue.

Fig. 3
Blighted flower clusters resulting from ascospore infections. Conidia are produced on diseased tissue.

Fig. 4
Mummified fruit form as berries ripen

Fig. 5
Fruiting bodies or spore cups (apothecia) emerging from overwintered mummy berries

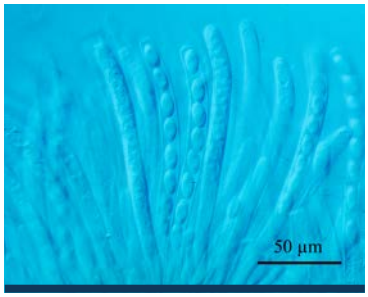

Fig. 6
Inside surfaces of apothecia are lined with sacs that contain spores (ascospores)

Fig. 7
Apothecia puffing out spores (ascospores)

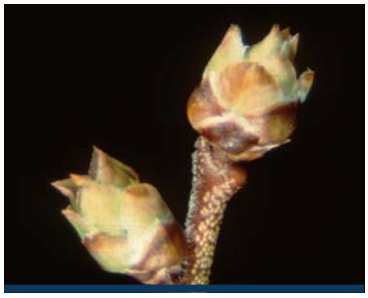

Fig. 8
F2 stage of flower bud development

Fig. 9
V2 stage of vegetative (leaf) bud development

Fig. 10
Blighted sprout resulting from ascospore infection

BOTRYTIS BLIGHT (GREY MOLD)

(Botrytis cinerea)

Importance: high

Symptoms

Botrytis blight occurs on blueberry flowers, leaves and stems in fruiting fields, but it has no impact on stems in sprout fields. The first symptom of disease is a premature browning or blighting of the corollas (petals) of open flowers in early-mid June (Fig. 11). The flower calyces and ovaries may also turn a dark purple-brown colour. Under periods of prolonged wet weather, diseased flowers develop a fuzzy or whiskery fungal growth (Fig. 12) characteristic of grey mold which is common to many other crop plants. Brown lesions may also develop on leaves that have come into contact with diseased flowers. Depending on the duration of wet periods, leaf shoots and stem tips may also become severely diseased (Fig. 13). Because clones vary in their susceptibility to infection and rate of development during the growing season, the disease is often limited to distinct clones and so appears in patches throughout a field (Fig. 14). Berries may become infected, but the disease incidence is usually low and not of concern in the processed frozen product. However, if the crop is fresh marketed, a few infected berries may decay making the product less marketable.

Botrytis blight is often confused with Monilinia blight and frost which also produce patches of blighted flowers and leaves. However, Botrytis blight is easily differentiated by the whiskery appearance of the fungus on the dead flowers (Fig. 12) in contrast to the whitish-gray growth resulting from Monilinia blight. Symptoms of Monilinia blight begin earlier in the season and flower clusters are usually killed before they fully open. Frosted flowers also turn brown, but do not show evidence of fungal growth initially. However, the Botrytis fungus can quickly colonize frosted flowers and may become evident several days after the frost event.

Disease Cycle

The fungus overwinters in diseased blueberry flowers, leaves, stems and fruit, but numerous weed hosts such as sheep sorrel, wild strawberry and wild raspberry commonly found within fields and along border areas are also susceptible to the fungus and they often provide the initial inoculum in fruiting fields during the spring. Abundant spore production can often be seen on dead tissues of these weed hosts before disease appears on blueberry flowers. Early flowering clones are the first to become infected because they come into bloom first. The fungus can become well established on these clones and act as a source of infective spores for later flowering clones throughout the field.

Spores (conidia; Fig. 15) that are produced on weed hosts or blueberry debris are wind-blown to developing flowers. Flowers are resistant to infection at the early stages of development, slightly susceptible at the pink (prebloom) stage and highly susceptible when fully open. Infection and spore production also occur readily on corollas that have fallen after pollination. Green berries are relatively resistant to infection unless contacted by infected tissue. Flower infection is favoured by wet periods with warm temperatures. At 20 C, high levels of infection require only 10 hours of wetness, but at lower temperatures, longer wet periods are required. If wet periods are relatively short but still adequate for infection, infected corollas turn brown without further noticeable symptoms and drop prematurely frequently contributing to unexplained yield losses. However, if wet periods extend for more than two days and temperatures are favourable, the fungus rapidly colonizes the flower and entire flower cluster and may extend into the main stem causing it to turn brown. Under these conditions, affected flowers tend to cling together and remain attached to the stem (Fig. 13) while others that have fallen may land on foliage or stem tissues where the fungus can infect healthy tissues causing further damage to the crop. Spore production occurs on infected tissue within only a few days after infection and so the fungus may cycle many times during prolonged wet periods during bloom. Severe disease outbreaks typically occur in coastal areas that experience prolonged periods of rain or fog, but outbreaks may occur further inland when conditions favour repeated cycles of spore production and infection. Frost during the bloom period increases the susceptibility of tissues to infection.

Disease Management

Botrytis blight may be controlled with applications of fungicides. The decision to use fungicides is usually based on the history of the field and location; fields in coastal areas typically require treatment. Fungicides should be applied before forecasted wet weather beginning at midbloom and thereafter on a 7-10 day schedule according to need until the end of bloom. Two applications are typically required, but sometimes a third is needed if disease is severe or flowering is prolonged due to cool weather.

Over fertilization leads to tall, dense canopies that do not dry quickly and promote disease buildup. In such canopies, the disease often goes unnoticed continuing to attack tissues long after the bloom period resulting in weakened stems and reduced yields. Unfortunately, if the disease becomes well established in the canopy, post bloom fungicide applications are not very effective.

Burn pruning in an attempt to destroy infected blueberry debris does not affect this disease since there are numerous other plant hosts in and around blueberry fields that act as sources of spores to initiate an infection.

Fig. 11
Blighted flowers

Fig. 12
Botrytis cinerea producing spores on blighted flowers

Fig. 13
Blighted stem; the disease has progressed from flowers into the stem

Fig. 14
Severely blighted blueberry clone with most of the flowers killed

Fig. 15
Spores (conidia) of *Botrytis cinerea* produced on tree-like fungal growth

LEAF RUST

(Thekopsora minima formerly *Pucciniastrum vaccinii)*

Importance: high

Symptoms

The first symptoms of leaf rust occur in mid to late July on the foliage of both fruiting and sprout fields, although the disease is usually more severe in sprout fields. Water soaked lesions appear on the undersurface of leaves that soon turn bright red and penetrate to the upper surface. One or more small yellow-orange blisters (pustules) form in the centre of lesions on the leaf undersurface (Fig. 16). With time, the number of lesions with pustules increases causing the upper leaf surface to become covered in red spots that eventually turn brown, and the undersurface to become covered with the yellow-orange pustules called uredinia that contain spores (Fig. 17, 18). Leaves become severely discoloured and drop from the stems prematurely. There is wide variation in susceptibility among clones with some showing only flecking while others show the more typical well-defined lesions and severe discolouration. Depending on the susceptibility of clones, defoliation becomes noticeable beginning in early September and by mid to late September, clones may become completely defoliated. Clones in sprout fields that become severely defoliated prematurely in September tend to have fewer and smaller fruit buds and reduced yields the following season.

Disease Cycle

The fungus that causes leaf rust has a complicated life cycle. It overwinters in the diseased leaves of blueberry and other hosts related to blueberry. In the spring, specialized spores called teliospores are produced on overwintered leaves that soon give rise to another flush of spores called basidiospores that are blown by wind and infect newly expanding needles of the alternate host Eastern hemlock during early to mid-June. In late June to early July, small, yellow, cylindrical fruiting bodies called aecia are formed on the undersides of hemlock needles that contain spores called aeciospores (Fig. 19). The effect on hemlock is negligible since infection occurs only sparsely. Aeciospores become windblown and infect leaves of blueberry and other hosts related to blueberry. By mid to late July, spores called urediniospores are pushed out of pustules called uredinia that form on the undersides of infected leaves (Fig. 20) and are blown about and infect more blueberry leaves. The fungus continues to cycle on blueberry until late October. As it cycles, numbers of lesions increase dramatically during August and early September. Depending on temperature, the fungus completes its cycle from infection to new spores in about 10-14 days. The optimum temperature for infection is about 21 C and the fungus appears to be favoured more by dew than rain.

Disease Management

Leaf rust affects both fruiting and sprout stems, but the consequence of premature defoliation is significantly more pronounced in sprout fields. The disease does not reach high levels in the fruiting year until September by which time most of the crop is harvested. Also, leaves of fruiting stems appear to be more resistant to infection than those of sprout stems.

The disease can be effectively managed by a fungicide application in sprout fields during the early stages of disease development in mid to late July, at a time when the sprouts cease growing (often referred to as the tip dieback stage of growth). Fungicide application delays disease development and subsequent defoliation allowing adequate time for flower buds to form in the autumn. If the fungicide is applied too early, disease control is less effective because the tip dieback stage has not yet occurred leaving the newest leaves unprotected. It is important to protect the upper most leaves of sprout stems as they contribute most to flower bud formation and maturation. The decision to apply a fungicide is based on the past history of disease severity and premature defoliation in sprout fields.

Burn pruning of blueberry fields and cutting of nearby hemlock trees does not reduce this disease because regionally there are many field sources of spores and they can travel long distances on wind currents. Interestingly, prior to the mid 1990's, leaf rust was not a major disease, but since then, it has become increasingly more severe. It may be that the disease was kept in check on a regional basis since most blueberry fields were burn pruned instead of mowed.

Over fertilization appears to increase the susceptibility of leaves on sprout stems.

Fig. 16
Early leaf rust symptoms resulting from aeciospore infections

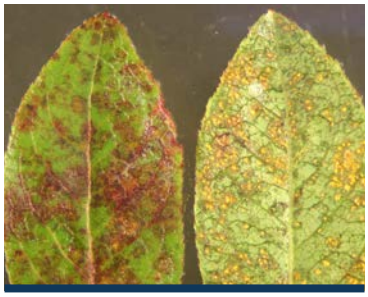

Fig. 17
Red to brown spots occur on the upper leaf surface (left) and pustules (uredinia) that produce masses of yellow coloured spores (urediniospores) occur on the lower leaf surface (right)

Fig. 18
Leaf rust symptoms on many leaves of a sprout stem

Fig. 19
Fruiting bodies (aecia) containing masses of yellow coloured spores (aeciospores) on Eastern hemlock needles

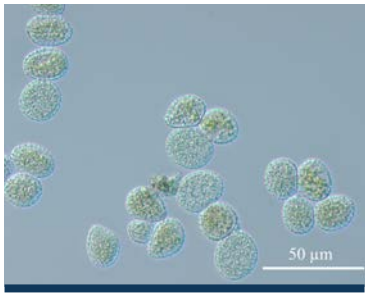

Fig. 20
Light microscope image of urediniospores

SEPTORIA LEAF SPOT AND STEM CANKER

(Septoria spp.)

Importance: high

Symptoms

In fruiting fields, leaf lesions occur throughout the canopy whereas in sprout fields, lesions occur mostly on the lower leaves of stems. The lesions first appear in mid-late June as minute water soaked spots on the leaf undersurface and are visible only with a hand lens (Fig. 21). The spots are translucent with transmitted light. With time, the spots increase in number and size and become slightly raised and visible to the naked eye. The centre of a spot on the leaf undersurface may become dark brown and slightly sunken while the outer edge remains water soaked and slightly raised. Tissues between lesions may become brown and lesions may be more pronounced along leaf veins. The leaf undersurface may have many lesions of different ages, and later in July, they begin to merge producing larger, irregular shaped spots that are variable in size up to about 5 mm in diameter (Fig. 22). The lesions penetrate to the upper leaf surface and appear as red/purple spots with irregular borders. Symptom intensity and colour of spots varies among clones. Leaves deep in the canopy may turn yellow instead of red/purple and appear peppered with the dark green water soaked spots. In sprout fields, the diseased lower leaves (Fig. 23) may drop during July and August, but this often goes unnoticed because they are deep in the canopy. In the upper canopy of fruiting fields, the reddish/purple discolouration continues to intensify into late July and early August when affected clones take on an unhealthy reddish/brown appearance. Clones may become severely defoliated with many dropped berries by harvest time (Fig. 24). By mid-August, it is difficult to differentiate leaf spots caused by this disease from leaf rust, powdery mildew or other leaf spotting pathogens.

Infections on stems in the sprout year remain latent until they become visible in late April-early May of the following fruiting year. Lesions initially appear as red/purple spots (Fig. 25) variable in size (0.5-10 mm long) and are confined to the lower half of the stem. They expand with time and by late May-early June the centres turn brown and become slightly sunken at which time they are referred to as cankers. By late June, several cankers may merge causing much of the lower half of stems to be affected, but stems are usually not killed. This is in contrast to Godronia canker which causes lesions only at leaf nodes and kills stem tissues above the point of infection.

Infections may also occur on immature green fruit which appears as water soaked raised pimples with blackened centres, but the effect on yield is considered minimal.

Disease Cycle

Small black fruiting bodies of the fungus called pycnidia mature on diseased fallen leaves (Fig. 26) and in stem cankers (Fig. 27) during April and May. When mature, spores (conidia; Fig. 28) are released by rain and splashed about. Spore release occurs most intensively over a 3-4 week period beginning in late May to early June. In fruiting fields, this occurs when 1-10% of flowers are open. By the end of the bloom period, the majority of spores have been released. Spore production closely follows the bloom period regardless if the season is early or late. In general, prolonged rainy weather during June favours infection, but high humidity (>95%) alone can also cause spores to germinate. The optimum temperature for infection is about 25 C. Following infection, the characteristic minute water soaked spots begin to appear on leaves about 10-14 days later, but do not become visible to the naked eye until about 4 weeks later. Hot, dry weather causing moisture stress during July and August promotes the drop of infected leaves and fruit. Although a few secondary spores from pycnidia produced on fallen, current season leaves may occur in late July and August, they are considered far less important than the large numbers of primary pycnidia and spores that occur on overwintered leaves during late May and June. The fungus also produces sexual spores (ascospores) during June, but too few are produced to contribute significantly to an epidemic.

In sprout fields, only those leaves and stem portions that have developed during June are exposed to spores explaining why the lower leaves of sprout stems become more severely diseased and why cankers on fruiting stems appear only on the lower portions.

Disease Management

Burn pruning can reduce this disease, but if conditions are highly favourable for infection, disease levels may remain high. In order for burning to reliably reduce this disease, it must be done intensively and uniformly throughout a field to destroy infected leaf and stem litter. Fungicide applications during the spore release period (beginning at 1-10% bloom) can protect foliage from infection, but the decision to apply a fungicide may be difficult to make. In general, if premature leaf drop has been severe before harvest in previous years, fungicide applications may be beneficial in the current fruiting year. The percentage of stems with spots/cankers observed in early May of the fruiting year may also help to guide the decision whether to apply a fungicide. High numbers of infected fruiting stems indicate that infection was severe in the previous sprout year and that many cankers and overwintered leaves may produce high spore populations posing a threat to the foliage of the current fruiting year. Attempting to control the disease with fungicides in the sprout phase may not be cost effective since repeated applications would be required to protect the continually expanding stems during June.

Fig. 21
Early disease symptoms appear as flecks on the leaf undersurface

Fig. 22
Advanced disease symptoms appear as irregular shaped brown spots on the leaf undersurface

Fig. 23
Lower leaves of sprout stems are more diseased than upper leaves

Fig. 24
Severe leaf and fruit drop resulting from disease

Fig. 25
Septoria leaf spot and stem canker symptoms on fruiting stem

Fig. 26
Small black fruiting bodies (pycnidia) on overwintered leaves

Fig. 27
Small black fruiting bodies (pycnidia) in cankered areas on fruiting stem

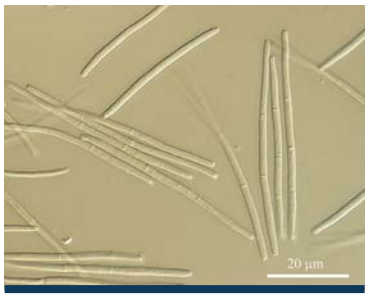

Fig. 28
Light microscope image of spores (conidia)

VALDENSINIA LEAF SPOT

(Valdensinia heterodoxa)

Importance: high

Symptoms

The disease appears on leaves of sprout and fruiting stems as circular, brown lesions sometimes with a dark brown or purple-red border and variable in size up to 1 cm in diameter (Fig. 29, 30). A single spore in the centre of a lesion may be visible on either side of the leaf with a hand lens. Infected leaves fall rapidly from the plant while still green. When the disease is serious, many fallen green leaves with lesions can be seen on the soil surface. Flowers and developing fruit may also become infected and drop from the plant (Fig. 31, 32). Infections in early June result in spots on lower leaves deep in the canopy, but with time, symptoms appear progressively higher eventually reaching the upper leaves. This leads to defoliated patches which become visible from a distance. Affected localized areas continue to expand and merge resulting in large defoliated areas in fields (Fig. 33). Severely affected fruiting fields have poor yields. Severely affected sprout fields may appear to recover a few weeks later, but this is due to vegetative re-growth from the leaf axils where fruit buds would normally form and so reduced yields may result in the following year.

First symptoms frequently appear in the shade of wooded areas, but symptoms eventually appear anywhere in the field. All blueberry clones are susceptible. The fungus can often be seen causing spots on many other plants in blueberry fields, but this is not important since the fungus does not easily produce spores or overwinter on these infected hosts. Such plants include sheep sorrel, bunchberry, birch, wild strawberry and raspberry

Disease Cycle

The fungus overwinters in the mid vein of an infected leaf as a hardened, black material called a sclerotium (Fig. 34). Sclerotia are hard to find since most of a diseased leaf decomposes during the winter, unlike healthy leaves which remain intact and decompose much more slowly. Large, star-shaped spores (conidia) are first produced in early-mid June on the sclerotia (Fig. 35, 36). About 2-3 days of continuous wetness are required for the first crop of spores to be produced and released from sclerotia. Once released, spore infection occurs rapidly with continued leaf wetness. Relatively few spores are produced, but each spore is highly aggressive and will cause a large lesion. Lesions reach about 5-10 mm in diameter in 24-48 hours after which leaves begin to drop. At this point, a further 48 hours of wetness are required for new spores to be produced on the infected leaves (Fig. 37). As a result, multiple spore production/infection cycles can occur during a week of wet weather. As the season progresses, the leaves tend to become more resistant. While infection still occurs, lesions tend to be smaller and leaves tend not to drop as quickly. However, any new re-growth on stems still remains highly susceptible.

Spore production occurs at 10-25 C with an optimum of 15-20 C. No spores are produced at 5 C and 30 C. Spores infect rapidly within 6-8 hours of wetness at temperatures of 15-25 C with an optimum at 20C. Infection is moderate at 10 C (12 hours of wetness), slow at 5 C (24 hours of wetness), and does not occur at 30 C. After the leaves drop, the fungus will colonize the mid vein of leaves where it overwinters. The fungus can survive as sclerotia in infected leaves for at least two years.

The spores of the fungus are forcibly discharged from sclerotia or the surface of infected leaves. They can be propelled upward about 20-30 cm and then fall downward due to their very large size if they have not impacted a leaf on the upward trajectory. This type of discharge results in localized areas of disease which then expand as the spores continue to land on healthy leaves. The spores are not spread by wind or rain, and so the disease often remains confined to affected fields. However, under wet conditions, the infected leaves become very soft and stick to machinery, tires and footwear and so the fungus is easily spread from field to field or within fields by mechanical transmission.

Disease Management

Growers should thoroughly inspect their fields for signs of disease when the canopy is dry. If a diseased patch is discovered, it is important to flag the area and then leave the area ensuring that no leaves are sticking to footwear or pant legs. It is important not to spread the disease by human activity. Work activities in affected areas should be planned so that they are completed last or affected fields should not be entered at all until other healthy fields have been worked. After exiting affected fields, equipment should be power or steam washed to remove all leaves before entering healthy fields.

If disease is found on a small scale, an immediate, thorough burning with a hand held weed burner under dry conditions may be adequate to destroy infected foliage thereby eradicating the disease. If disease is found on a larger scale, a thorough field burn may reduce disease the following year. However, the burn must be intense and uniform enough to destroy the leaf litter and so must be done under ideal burning conditions. Despite these measures, the disease may return if infected blueberry plants in nearby wooded areas are harbouring the disease.

If the disease is widespread in a field, fungicide applications will reduce the effect of the disease, but will not eradicate it from fields. For optimum disease control, the first application must be made when disease symptoms first occur and wet weather is forecasted. A second application may be necessary 10-14 days later if wet weather persists or before the next forecasted period of extended wet weather. Good spray penetration into the canopy is essential.

The fungus is highly responsive to nitrogen levels in leaves. Over-fertilization should be avoided.

Fig. 29
Large brown spots sometimes with dark brown or purple-red borders develop quickly on leaves after infection

Fig. 30
Several large spots may appear on a leaf

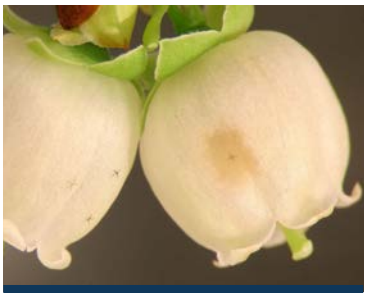

Fig. 31
The unusually large star-shaped spores (conidia) infecting flowers

Fig. 32
Spots on leaves and sunken spots on fruit

Fig. 33
Severe defoliation caused by Valdensinia leaf spot

Fig. 34
Overwintering fungal bodies (sclerotia) of *V. heterodoxa* on leaves

Fig. 35
Spores (conidia) of *V. heterodoxa* produced on sclerotia of overwintered leaves

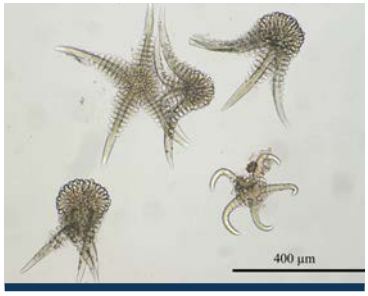

Fig. 36
V. heterodoxa spores (conidia) of varying maturity

Fig. 37
Spores (conidia) of *V. heterodoxa* forming on a diseased leaf

POWDERY MILDEW

(Microsphaera vaccinii)

Importance: moderate

Symptoms

Symptoms of powdery mildew vary substantially among clones due to their levels of disease resistance. In susceptible clones, the first symptoms of disease appear as small blotches of reddish discolouration on the upper leaf surface in mid to late July. This symptom can be confused with Septoria leaf spot but in the case of the latter disease, the spots on the upper leaf surface are always associated with water soaked lesions on the leaf undersurface. The blotches caused by powdery mildew expand and may be covered with very fine, white fungal threads visible with a hand lens or microscope (Fig. 38). With time (early to mid-August) the red discolouration may become severe (Fig. 39) and the leaf undersides may also turn red. The blotches begin to turn brown by mid-August and severely affected leaves begin to curl and drop making it difficult to distinguish powdery mildew from Septoria leaf spot. On some clones, the fungal growth on the upper and lower leaf surfaces becomes evident as a white deposit (Fig. 40) whereas other clones may show only the reddish discolouration. Small spherical, yellow bodies that eventually turn black may be observed on the leaves of clones showing the powdery deposit (Fig. 41, 42). These are the overwintering structures of the fungus called cleistothecia. Powdery mildew can cause severe defoliation in both fruiting and sprout fields. Under severe disease in sprout fields, flower and leaf buds may be affected. Although the exterior tissues may appear normal, when cut open in October, the internal bud tissues may appear darkened due to fungal colonization resulting in reduced viability.

In addition to affecting foliage, powdery mildew can also affect the fruit when it is still immature. Early signs of disease appear as a reddish discolouration on green berries (Fig. 43) and with time affected areas turn brown and berries may shrivel and drop. Very fine fungal threads may be visible on the green berries (Fig. 44) with a hand lens or a microscope. Clones that are severely diseased usually have reduced yields.

Leaves of resistant clones may be completely free of the disease or they may show numerous small red rings which is considered to be a defense response of the plant.

Disease Cycle

Research on the powdery mildew disease cycle has not been extensively conducted and so it remains poorly understood. The small fruiting bodies (cleistothecia) on diseased leaves are possibly an important source of inoculum in spring. In other powdery mildews that have been more extensively studied, the cleistothecia rupture during wet weather and forcibly eject spores (ascospores) that are blown by wind to young leaves. Once established on a leaf, the fungus produces secondary spores (conidia) that are carried by wind to establish new infections. Many cycles may occur throughout the summer months. Although not proven in research, it is possible that the fungus can also overwinter

within infected leaf and flower buds as do many other species of powdery mildew on other perennial crops. From here, the fungus produces conidia.

Initial infections occur on leaves during June, but symptoms usually are not visible until later in July. The disease is present annually in most lowbush blueberry fields to some degree, but the severity is highly dependent on weather conditions. Unlike most other pathogens of blueberry, the powdery mildew fungus is inhibited by rainy weather. Instead, the fungus is favoured by long periods of warm, dry, humid weather, particularly in July. Under these conditions, powdery mildew severity can increase at a surprisingly rapid rate. However, if rainy weather occurs following these conditions, disease development may be substantially reduced.

Disease Management

In most years, powdery mildew is not severe enough to be of concern, but occasionally high disease levels can result in yield reductions. Little research has been conducted on powdery mildew and so there are few well-defined management strategies available. Burn pruning does not appear to reduce this disease suggesting that sufficient inoculum is blown in from sources outside of the pruned field or the disease overwinters in infected buds. Foliage that is deficient in phosphorous tends to be more susceptible, so appropriate tissue sampling is advisable. Fungicides have recently become available for this disease and may be applied when symptoms first become visible (usually July), but the cost benefit of this has not been explored.

Fig. 38
Early symptoms appear as red blotches on leaves

Fig. 39
Severe powdery mildew symptoms on leaves

Fig. 40
Leaves covered with a white powdery fungal growth; leaves of some clones do not turn red

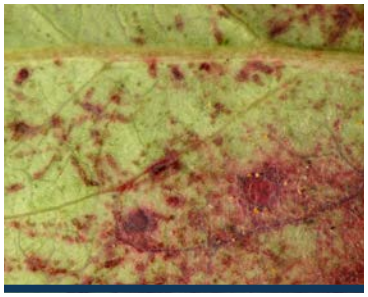

Fig. 41
Immature yellow fruiting bodies (cleistothecia) forming on the leaf undersurface

Fig. 42
Yellow coloured cleistothecia maturing to become black in colour

Fig. 43
Infected berries initially show red speckling followed by brown blotches

Fig. 44
Early symptoms on berry with inconspicuous wispy threads of the fungus

RED LEAF

(Exobasidium vaccinii)

Importance: moderate

Symptoms

As the name suggests, red leaf disease appears as a bright red discolouration of the foliage (Fig. 45). The disease is most conspicuous in fruiting fields and occurs on single stems or often in patches of many stems. The disease can be observed in mid-late May as the foliage begins to expand, but it is most conspicuous during June and early July when affected foliage stands out in contrast to lush green healthy foliage. Affected leaves may be uniformly discoloured red or they may have sections of green mixed with red tissue. Diseased leaves growing on stems in shaded areas may be pale pink, yellowish or even pale green. During late June to early July, the underside of affected leaves develop a white, felt-like fungal deposit (Fig. 46). By late July to early August, the leaves turn brown and fall from stems giving the false impression that a field has recovered from the disease. Affected stems typically have fewer and weaker flower buds and if pollination is successful, the berries usually shrivel, turn brown and drop from the stem before harvest. Infected rhizomes are weakened by the disease and eventually die, but this process goes unnoticed as new healthy rhizomes fill in the diseased patches.

Diseased foliage in sprout fields is usually not as conspicuously discoloured as in fruiting fields. Leaves on young sprout stems may show some signs of reddish discolouration in June, but this can be easily confused with the natural reddish hue on foliage of some clones. As the season progresses, diseased foliage may turn brighter red, but still less than in fruiting fields.

Disease Cycle

Research on this disease has been hampered by the fact that infection under laboratory conditions is difficult to achieve. Under field conditions, infection of healthy stem tissue occurs during wet weather in late June to early July when spore release from affected foliage occurs. It is not known if infection occurs on leaves. However, the infection efficiency is very low in relation to the large number of spores produced on diseased foliage. Following infection, the fungus colonizes the stem and then grows downward into the rhizome. However, symptoms do not appear in the year of infection and it may be several years before diseased stems become noticeable. Once infection becomes well established, symptoms will occur annually in stems because the fungus grows systemically from the rhizomes.

Fields that have been in production for many years tend to have a higher incidence of disease, but this increases only 0.1% per year due to the low infection efficiency of the fungus. The incidence of diseased stems seldom exceeds 5% in most fields and so the effect on yield is negligible. However, incidence levels as high as 30% may occur in some fields and in these instances lower yields consistently occur. The reason that some fields have a high disease incidence is not fully understood, but a high level of disease may have already been present at the time of field development.

Disease Management

There are no disease management strategies available for red leaf disease. Some fungicides may inhibit the pathogen from causing new infections, but none have been shown to move downward into the rhizomes where the fungus overwinters. Similarly, burn pruning may destroy new infections that are not yet in the rhizomes, but in general it does not reduce disease levels because the fungus remains protected below soil level. Research has shown that fields consistently pruned by burning or mowing have similar disease levels.

Eradication of infected stems with herbicides may be effective, but damage to surrounding healthy stems usually results in yield loss greater than the disease and so this practice is not advised. Moreover, there are no registered herbicides for this purpose.

The bright red foliage of affected stems during June can be so striking causing one to believe that the disease is more severe than it actually is. Accurate estimates of disease can be determined by collecting about 200 stems along a W pattern encompassing an entire fruiting field and then determining the incidence (%) of diseased stems. If an estimate of disease over years is desired, it is recommended that stem collection occur roughly in the same area of a field and at the same time each year.

Fig. 45
Bright red leaf symptoms in a fruiting field

Fig. 46
Undersurface of leaves covered with masses of white spores

FALSE VALDENSINIA

(Unnamed species)

Importance: low-moderate

Symptoms

The name of this disease has not been formally determined and should be considered temporary. It is derived from the fact that its symptoms are almost identical to those of Valdensinia leaf spot, but the two diseases are caused by different fungal pathogens. Like Valdensinia leaf spot, False Valdensinia appears as large (up to 1 cm in diameter) circular brown lesions on leaves (Fig. 47; left 2 leaves are False Valdensinia; right 2 leaves are Valdensinia leaf spot). However, the amount and speed of defoliation is much reduced with False Valdensinia resulting in diseased foliage which often is more noticeable (Fig. 48) in the field compared with Valdensinia leaf spot which causes leaves to drop quickly. Lesions caused by False Valdensinia tend to remain brown whereas those caused by Valdensinia leaf spot tend to develop a lighter centre with time, if the leaves happen to remain on the plant. One way to be certain that the disease is False Valdensinia is to examine both surfaces of several leaves with lesions with a hand lens. If there are no signs of the large spore in the centre of a lesion typical of Valdensinia leaf spot, then the disease is likely caused by False Valdensinia. Like Valdensinia leaf spot, False Valdensinia also initially appears in small patches (about 1 m diameter) within fields, but these affected areas do not usually expand and spread within the field as does Valdensinia leaf spot. The disease also attacks fruit. Lesions on green fruit are dark brown and affected areas become sunken (Fig. 49) and the fruit eventually drop.

Disease Cycle

The life cycle of the fungus has not been studied in detail. However, it is known that the fungus overwinters in diseased leaves and produces spores directly on the leaves in early June, at the same time as the Valdensinia leaf spot fungus. Spores are very small (Fig. 50) and not visible with a hand lens compared with those of the Valdensinia leaf spot fungus. Spores are thought to be dispersed mostly by rain splash, but conditions for infection have not been determined. Initial disease symptoms appear during June at the same time as Valdensinia leaf spot adding to the confusion between the two diseases. The period of spore production is thought to be restricted because the disease does not appear to spread extensively within a field over time.

Disease Management

The disease is of moderate concern since it mimics the symptoms of Valdensinia leaf spot which is a disease of great concern. False Valdensinia can give the impression that Valdensinia leaf spot is present and should be treated with a fungicide, when in fact, disease control measures are not warranted. For this reason, an accurate identification is important. No disease control strategies have been studied, but it is believed that fungicides used for the control of Valdensinia leaf spot will also control False Valdensinia. Burn pruning is presumed to help reduce disease.

Fig. 47
Symptoms of False Valdensinia leaf spot (left) are very similar to Valdensinia leaf spot (right); spots of False Valdensinia leaf spot tend to remain brown whereas those of Valdensinia leaf spot develop a lighter centre

Fig. 48
Many leaves may be affected with several spots on a leaf

Fig. 49
Spots on fruit are dark brown and sunken

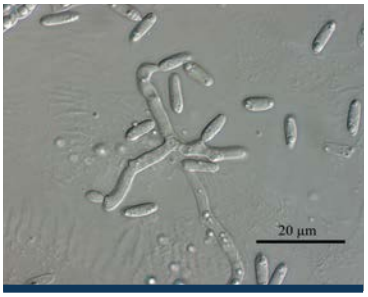

Fig. 50
Spores (conidia) of the fungus that cause False Valdensinia leaf spot

ANTHRACNOSE FRUIT ROT

(Colletotrichum acutatum)

Importance: moderate; emerging

Symptoms

The first noticeable symptom of this disease occurs as softening, shrivelling and slight sinking of the blossom end of ripe or nearly ripe fruit. Under wet conditions, small droplets of salmon coloured ooze (Fig. 51) may develop on berries. If shrivelled berries are noticed on the plant, but no ooze is present, affected berries can be incubated in a moist plastic bag overnight and if the fungus is present, droplets of ooze will appear. If no ooze develops after a further 24 hours, the shrivelling is likely due to a number of other possible causes such as Botrytis blight, Septoria leaf spot and stem canker, powdery mildew, mummy berry, insect maggot or simply drought. Berries affected by anthracnose eventually drop from the plant.

Disease symptoms also occur during late June on the foliage as small, sunken, circular to angled spots with dark brown or blackened centres and red borders (Fig. 52), but this symptom is usually difficult to detect and goes unnoticed.

Disease Cycle

The life cycle has not been studied in detail in lowbush blueberry, but it is well understood in highbush blueberry and presumably occurs similarly in lowbush blueberry. The fungus overwinters in blueberry debris. Spores (conidia) are released throughout the growing season and infect fruit at all stages, but in leaves only when they are young. Blueberry flowers also may become blighted as a result of infection. Infection occurs during periods of rain when continuous wetness exceeds 12 hours and temperatures range from 15-27 C. Infected fruit remain symptomless until shortly before ripening when masses of secondary spores are released in the salmon-coloured ooze. These spores may infect healthy berries and new symptoms may develop rapidly on ripe or nearly ripe fruit. In highbush blueberry, substantial losses can occur with prolonged periods of warm, wet weather during bloom and just before harvest. Losses also occur in whole packs of fresh fruit when infected berries with ooze contact healthy berries causing new infections.

Disease Management

Anthracnose in lowbush blueberry was first observed in Nova Scotia and New Brunswick in 2013 with estimated yield losses of 5-10% in some fields. Disease management strategies have not been developed for this disease in lowbush blueberry due to its recent appearance. Burn pruning likely will help reduce this disease.

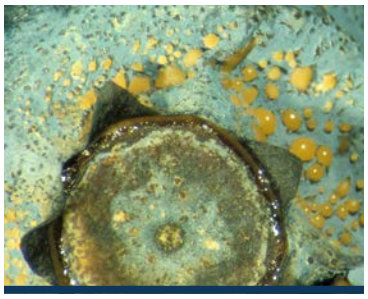

Fig. 51
Masses of spores (conidia) in salmon-coloured ooze on infected berries

Fig. 52
Infections on young leaves appear as small dark sunken spots with red borders

PHOMOPSIS CANKER

(Phomopsis vaccinii)

Importance: low

Symptoms

Phomopsis canker affects both fruiting and sprout stems. Diseased stems have foliage that is orange-brown in colour and are relatively easy to identify against the green background of healthy stems and leaves (Fig. 53). Initial disease symptoms are more difficult to see as they begin at the soil level where stems begin to turn brown after infection has occurred (Fig. 54). The brown discolouration continues up the stem and soon leaves above the basal canker begin to turn reddish in colour and eventually orange-brown. The basal canker and the orange-brown discolouration of leaves are diagnostic for this disease (Fig. 55). The disease usually affects single stems scattered throughout a field, but sometimes the disease can be more intense and affects many stems in small patches.

Disease Cycle

The disease cycle has not been extensively studied in lowbush blueberry and is not fully understood. The fungus overwinters on dead stem tissue and produces fruiting bodies called pycnidia. Two types of spores (alpha and beta conidia; Fig. 56) are released during rainfall and are splashed about. A second type of fruiting body (perithecium) is produced on overwintered dead stems that release airborne spores (ascospores), but their role in the disease cycle is not understood. Infection by conidia occurs at the base of both fruiting and sprout stems during June and symptoms begin to develop in late June and early July.

Disease Management

Disease severity in lowbush blueberry is usually low and of little consequence so specific control measures have not been necessary. Burn pruning is likely beneficial for disease control.

Fig. 53
Foliage on stems affected by Phomopsis canker turns reddish brown

Fig. 54
Initial cankers occur at the base of stems

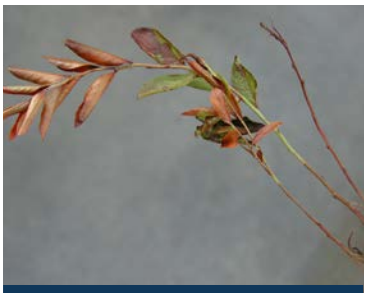

Fig. 55
Basal cankers eventually cause entire stem to collapse

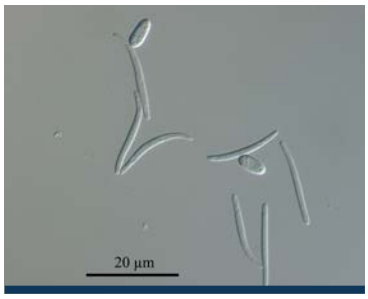

Fig. 56
Light microscope image of two spore types of *Phomopsis vaccinii*

GODRONIA CANKER (FUSICOCCUM CANKER)

(Godronia cassandrae)

Importance: low

Symptoms

Godronia canker appears as lesions on fruiting stems that result in a dieback symptom. Stem cankers first appear on fruiting stems in late May or early June about midway up the stem and are almost always centered around leaf buds. Cankers are orange-brown in colour, surrounded by a purple border and may be up to 2 cm in length. The centres of the cankers become tan coloured and sunken as the growing season progresses. Small black fruiting bodies called pycnidia are formed in the centre of the canker and tend to encircle the leaf bud (Fig. 57). Leaf and flower buds and green stem tissues above the canker are killed resulting in the tip dieback symptom. Leaf buds below the canker are not affected and they begin to grow vigorously (Fig. 58), so by early July a severely infected field will appear to have recovered due to an abundance of green foliage, but it will have a reduced yield. Lesions on some clones may develop more slowly and so tip dieback is not as severe, but stems remain unthrifty, foliage turns reddish and berries are small. The disease may appear on isolated stems or more densely in patches throughout a field.

Disease Cycle

The disease cycle has not been extensively researched and so it is not fully understood. Initial infection by the fungus is believed to occur during the sprout year, but stems remain symptomless until the following spring when they develop on fruiting stems. The sprout year infections are believed to occur during early July when the sprouts are about half-grown accounting for the fact that cankers are usually found midway up the stem in the following fruiting year. The fungus produces fruiting bodies called pycnidia (Fig. 57) in the cankers that release spores (conidia; Fig. 59). The spores are released during rain and are splashed about. Although the optimum for spore germination is known to be 20-25 C, specific wetness requirements for infection have not been determined. The fungus also produces cup-like fruiting bodies (apothecia) on old, dead stems along ditch banks during the growing season that release windblown spores (ascospores), but their role in the disease cycle has not been determined.

Disease Management

Godronia canker was severe and widespread in Nova Scotia and to a lesser extent in New Brunswick and Prince Edward Island in 2002. In previous and in subsequent years, the disease has occurred only at very low levels and it is not known why the disease was severe in only one year. However, fields that had been mowed in the previous production cycle had substantially higher levels of disease than those fields that had been burn pruned indicating that burn pruning has a beneficial effect in disease management.

Fig. 57
Fruiting bodies (pycnidia) in canker surrounding a leaf bud

Fig. 58
Stem death occurs above a canker

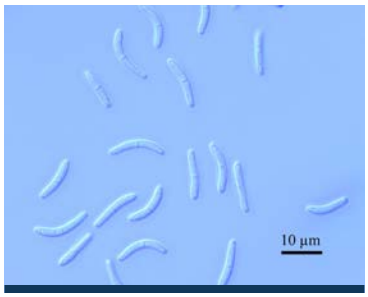

Fig. 59
Spores (conidia) of the fungus that cause Godronia canker

RED WART

(Phomopsis spp.)

Importance: low

Symptoms

Initial disease symptoms appear as small, raised, red coloured spots or warts on sprout stems during late June to mid-July (Fig. 60). Leaves may also show small red coloured lesions that stand out in contrast to the lush green foliage of new growth. The warts on stems do not increase in number as the season progresses. By September and October, the warts turn a red-brown colour (Fig. 61) and some may split open longitudinally. During May and June of the following year on the current season fruiting stems, the centres of warts turn gray and splitting of the bark expands (Fig. 62). By late July, cracking of stems can be severe if they are covered with many warts. Lesions also appear on the foliage and increase in intensity from June through the growing season. The lesions initially appear as numerous raised water-soaked spots on the undersides of leaves that eventually turn a reddish brown (Fig. 63) and are associated with a red discolouration on the upper leaf surface. Substantial defoliation can occur before harvest if the disease is severe (Fig. 64). Advanced foliar symptoms can be difficult to discern from those of Septoria leaf spot, leaf rust or powdery mildew.

Disease Cycle

This disease has not been studied in detail. The old scientific literature indicates that the disease has been attributed to the piercing action of unknown insects, but more recent laboratory studies have shown the disease is most likely caused by a fungus closely related to *Phomopsis vaccinii*, the cause of Phomopsis canker. The red wart fungus overwinters in diseased stem and leaf tissue and produces fruiting bodies called pycnidia that release spores (conidia) during rain. Infection of stems and leaves most likely occurs only during June based on the fact that warts on sprout and subsequently fruiting stems occur only half way up the stem. Hot, dry weather causing moisture stress during July and August promotes drop of infected leaves and fruit.

Disease Management

The disease can be found in most blueberry fields and is occasionally moderately severe in some. Disease management studies have not been conducted, but it is believed that burn pruning will help to destroy infected leaf and stem debris in which the fungus overwinters. It is not known if fungicide application during June will reduce disease severity.

Fig. 60
Brightly coloured and raised Red wart lesions on sprout stems during early summer

Fig. 61
Red wart lesions on sprout stems turning red-brown in early autumn

Fig. 62
Bark of gray coloured wart lesions splitting open on fruiting stem in spring

Fig. 63
Raised lesions on leaf undersurface

Fig. 64
Severe defoliation caused by Red wart

EXOBASIDIUM FRUIT AND LEAF SPOT

(Exobasidium spp.)

Importance: low

Symptoms

This disease appears initially as small scattered white to pale yellow circular spots on leaves in fruiting fields during late June (Fig. 65). As the season progresses, the lower surface of the spots eventually become covered with a thin powdery deposit which consists of masses of spores. With more time, the lesions thicken slightly and the upper surface becomes reddish-brown and the centres of spots sometimes show faint target-like concentric rings. Heavily diseased leaves drop prematurely, but damage to plants is negligible. The fungus also attacks berries causing small, circular, firm spots that fail to ripen and remain green or pink (Fig. 66). The firm tissue of the infected area extends about 2-3 mm into the berry tissue. The disease is usually not observed in sprout fields.

Disease Cycle

Laboratory studies have shown that the fungus associated with this disease is similar to, but different from the species that causes red leaf disease, *Exobasidium vaccinii*. Little is known about the disease cycle. The appearance of disease during late June indicates that infection occurs when leaves are young. The disease appears to be more prominent following prolonged wet periods in June. The fungus does not appear to become systemic in the rhizome as occurs with red leaf disease.

Disease Management

The disease is usually not severe and so control measures have not been studied. Affected berries are easy to see in whole fresh packs and can be removed. Burn pruning likely reduces this disease.

Fig. 65
White to pale yellow spots on leaves of fruiting stems

Fig. 66
Spots on fruit fail to ripen

WITCHES' BROOM

(Pucciniastrum goeppertianum)

Importance: low

Symptoms

Symptoms of this disease can be observed in fruiting fields. The fungus stimulates the production of multiple swollen shoots from a rhizome resulting in isolated broomlike masses of shoots scattered throughout the field (Fig. 67). Infected shoots are swollen, turn reddish or yellow and have smaller leaves (Fig. 68). As the season progresses, the stems turn brown and shiny, and eventually dry and crack. Affected stems usually produce few or no fruit.

Disease Cycle

The disease cycle of this pathogen is somewhat similar to that of the leaf rust fungus, except in the case of Witches' broom the alternate host is balsam fir instead of hemlock. In the spring, spores (basidiospores) are produced on overwintered, diseased fruiting stems and are carried by wind and infect newly expanding balsam fir needles during June. The effect, however, is negligible since infection occurs only sparsely. In late June to early July, the fungus produces small, white, cylindrical fruiting bodies called aecia on the undersides of the needles (Fig. 69) that contain spores called aeciospores. Aeciospores are windblown and infect the stems and leaves of blueberry. It is not known if sprout stems are more susceptible to infection than fruiting stems. After initial infection, the stem remains more or less symptomless and the fungus eventually grows into the rhizome. Once in the rhizome, the fungus becomes perennial, much like the fungus that causes red leaf disease, and will stimulate the growth of masses of swollen stems annually until the rhizome eventually dies. Unlike leaf rust, the Witches' broom fungus does not continue to produce spores throughout the growing season accounting for the low disease levels in most fields.

Disease Management

Witches' broom is not a serious disease and so development of control strategies has not been required. Burn pruning does not reduce the incidence of diseased plants because the fungus is protected within the rhizome.

Fig. 67
Symptoms of Witches' broom appear as thickened brown stems in a broomlike mass

Fig. 68
Early symptoms show thickened, but still green stems and small reddish discoloured leaves

Fig. 69
Fruiting bodies (aecia) containing white coloured spores (aeciospores) on the undersides of needles of Balsam fir

FALSE SEPTORIA

(Mycocentrodochium spp.)

Importance: low

Symptoms

The name of this disease has not been formally described and should be considered as temporary. Its name is derived from the fact that symptoms and fungal spore shape appear somewhat similar to those caused by Septoria species occurring on other crops. Symptoms of False Septoria begin to appear in late June in fruiting fields as small red-brown spots (Fig. 70) that soon develop a tan coloured centre. The spots may expand slightly, but the outer borders remain a red-brown to purple colour. The centres of the spots become distinctly sunken (Fig. 71). The leaves usually become peppered with many spots. By mid-July, small, brown fruiting bodies form in the centres of the spots (Fig. 72) on the upper leaf surface, but are visible only with a strong hand lens. In late July to early August, affected leaves begin to shrivel and drop. The disease occurs in small patches and remains localized, but because of the distinct spotting, it attracts attention.

Disease Cycle

The disease cycle has not been studied in detail. Based on symptom development, initial infection is presumed to occur in mid-June. The causal fungus likely overwinters in infected leaves, but the amount of inoculum produced appears to be limited since the disease tends to develop only in small patches with little consequence. It may also be that leaves become resistant to infection as they age. The fungus produces small fruiting bodies called sporodochia (Fig. 73) that produce spores (conidia; Fig. 74) on their outer surface. These spores tend to resemble those of Septoria species on other crops, but they are much larger. Despite large numbers of spores produced, the disease does not appear to spread from the initial infected patches.

Disease Management

To date, the disease has been of little consequence and so management strategies have not been studied.

Fig. 70
Spots on leaves of sprout stems caused by False Septoria

Fig. 71
Lesions on leaves are initially brown becoming tan coloured, distinctly sunken and surrounded by a red-brown to purple border

Fig. 72
Small brown fruiting bodies of the fungus in a lesion

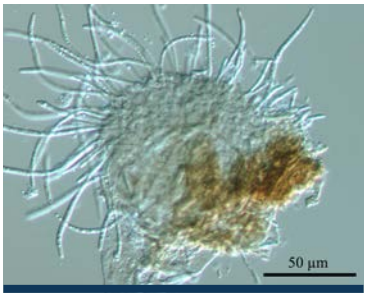

Fig. 73
Fruiting body (sporodochium) producing long slender spores

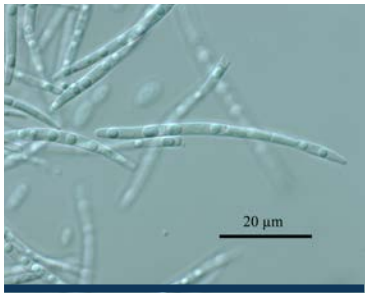

Fig. 74
Spores are similar to those of true species of *Septoria*

LOW TEMPERATURE INJURY

Low temperature injury occurs during the winter months, during early spring before bud break or during the bloom period. Some of the resulting injuries may be incorrectly attributed to diseases.

Winter Kill

Winter kill occurs when the tips of overwintering sprout stems are not adequately protected by snow during periods of unusually low temperatures and high winds. These conditions desiccate stem tissues causing the bark to shrivel. Symptoms may be visible soon after snow melt. Leaf and flower buds fail to develop and the bark eventually turns dark brown (Fig. 75). Depending on the depth of snow cover, symptoms may occur only at the very tips of stems or more than half of the stem may be affected. Symptoms occur at the same height on stems within affected areas of fields reflecting the level of snow cover at the time of injury. Symptoms often occur more commonly on knolls in open areas of fields. Some blueberry clones appear to be more tolerant than others.

Winter kill can be reduced by planting coniferous trees around fields to trap snow and reduce damaging wind speeds.

Early Spring

Unusually low temperatures during early spring when the buds are beginning to swell, but before they crack open, may lead to poor leaf and flower development. Contrary to expectations, it is the innermost tissues of the bud that are affected. Symptoms are most easily seen on leaf buds. The first outermost leaves of the bud appear to unfold normally, but as the growing point of the shoot becomes visible, it appears blackened and further development of leaves does not occur (Fig. 76). Overall, leaf development within a field soon appears to be stunted. Similarly, individual flowers within buds fail to develop or are distorted (Fig. 77) resulting in reduced flowers available for pollination. A useful method to determine if flower buds have been damaged by frost is to cut them longitudinally and observe for internal brown tissues.

Frost during Bloom

In general, as blueberries advance in their development through spring, their tolerance to low temperatures decreases. Fully open flowers can tolerate light frosts of temperatures down to about -2 C, but significant damage occurs at lower temperatures. Depending on the severity of an early morning frost episode, symptoms of frost injury on flowers may appear only in low lying areas within a field, or throughout an entire field if the injurious temperatures were widespread. Affected corollas of flowers begin to shrivel and turn tan coloured and calyces turn purple (Fig. 78). After several hours, a smell of crushed tissue may be noticed. By the next day, flowers turn fully brown and may appear as though they have been affected by Botrytis blight. In severe frost episodes, leaves may also appear

blistered (Fig. 79) and eventually turn brown. Tissues affected by frost have a heightened susceptibility to Botrytis blight, so conditions for this disease should be monitored carefully. Cutting frosted flowers through the ovary and observing for darkened tissues is a useful method to determine if frost has been severe enough to potentially affect berry development.

In rare instances when frost is severe, sprout stems may also become affected. Symptoms appear as slightly sunken brown coloured spots on the developing stems and leaves may shrivel and turn brown (Fig. 80).

Fig.75
Winter kill appears as dark brown dead tissue at the tips of stems

Fig. 76
Vegetative buds affected by early spring low temperatures show blackened internal leaves that fail to develop

Fig. 77
Developing flowers within flower buds affected by early spring low temperatures are brown and fail to develop or become distorted

Fig. 78
Corollas of open flowers affected by frost during bloom turn brown and calyces turn purple

Fig. 79
Leaves affected by a severe frost during bloom show blistering of the leaf surface

Fig. 80
Sprout stem and leaves affected by a severe frost