The Cultivated Highbush Blueberry

Austin C. Goheen

Our cultivated highbush blueberries are mostly hybrids and selected wild plants of *Vaccinium australe* and *V. corymbosum*, the native blueberries of eastern North America. A few are hybrids between the species *V. australe* and *V. lamarckii*, the lowbush blueberry that forms the bulk of the wild blueberry crop that is harvested in Maine.

When the commercial cultivation of blueberries was first started, the fields were small and isolated, and the plants remained relatively free from disease—blueberries, in fact, were hailed as one crop without serious disease problems. But that happy condition no longer prevails. Modern plantings, in large fields of genetically similar bushes, are subject to considerable damage from several disease-producing agencies. The most serious diseases are stunt, mummy berry, botrytis blight, powdery mildew, and stem canker.

**Stunt**, a virus disease of the yellows type, is prevalent in New Jersey and North Carolina. Entire fields may become so badly diseased that little or no crop is produced. The disease also occurs in Massachusetts, New York, Michigan, Maryland, and eastern Canada. It occurs in wild highbush blueberries in New Jersey.

In nature it is transmitted by the leafhopper, *Scaphytopius magdalenensis*. It can also be spread in diseased cuttings and nursery stock. In experiments it has been transmitted in diseased buds and grafting wood. We know from tests that dodder, a parasitic seed plant, can transmit the disease from blueberry plants to other blueberry plants and to *Vinca rosea*, but such transmission has not been observed under natural conditions. Inoculating healthy plants with the juices of diseased plants has not transmitted the disease. Stunt is not spread in the field by pruning knives or other mechanical methods.

Its symptoms are variable, differing with variety, time of year, stage of growth, and age of infection. Stunt dwarfs the bush, reduces the size of the leaves, and causes an abnormal coloring of the leaves. The fruit on bushes that have been infected for a number of years are inferior in size and quality. Diseased bushes eventually fail to set fruit.

The most marked symptom is found in the terminal leaves of young shoots, which in spring and early summer develop pale-green or yellowish margins. Often they are cupped. By holding the leaves perpendicular, one can see a pattern like a Christmas tree: The midrib and lateral veins remain dark green and the pale colors extend inward in the areas between the lateral veins. In late summer the interveinal areas redden brilliantly before healthy plants show any normal red fall coloring. Then the Christmas tree pattern is very striking. Leaf symptoms vary with variety. Rubel shows the spring symptoms throughout the season. Infected Rancocas bushes exhibit only late-summer symptoms. Bushes infected for some time frequently do not show the leaf symptoms.

Stunt was first observed in New Jersey in 1926. In 1942 R. B. Wilcox, of the Department of Agriculture, described the trouble as of virus origin. In 1945 C. A. Doehlert, of the New Jersey Agricultural Experiment Station, found that the disease could be transmitted by mixed colonies of leafhoppers. Subsequently P. E. Marucci and W. E. Tomlinson, Jr., of
the same station, proved that the vector was either *S. magdalensis* or *S. verecundus*, two similar insects that are widely distributed on ericaceous plants in swamps of eastern North America. M. T. Hutchinson, also of the New Jersey station, found that *S. verecundus* does not occur in appreciable numbers in the cultivated blueberry fields although it is common in nearby cranberry bogs. *S. magdalensis* therefore seems to be the principal vector for stunt virus.

No variety is immune to stunt. Rancocas appears to be tolerant to the virus. Plantings of Rancocas in badly infected fields have continued to produce crops of berries long after the disease has destroyed other varieties.

Three control practices are each partly successful. Prompt removal of plants that show stunt symptoms reduces the reservoir of diseased plants that the leafhopper may feed on. In districts where the disease occurs sporadically this practice is effective.

In areas where the disease is bad and the leafhopper is abundant, roguing alone is not successful. Here the second practice, control of the leafhopper, is necessary. Four applications of DDT or methoxychlor (3 pounds in 100 gallons of water) have greatly reduced the numbers of leafhoppers in tests in New Jersey but have never completely destroyed them. Roguing and insect control together are the best control procedures for use in New Jersey and North Carolina.

Use of disease-free plants in setting new fields is the third practice. Rigid specifications have been set up for the certification of healthy plants in New Jersey. But even with certified plants, new fields in New Jersey and North Carolina should be rogued and the leafhoppers controlled.

The ultimate control of stunt in areas where the vector is abundant seems to rest on the development of immune varieties or elimination of sources of the disease. No truly immune varieties were available in 1953. A number of wild species and varieties of blueberries have been put under test in New Jersey in the hope that among them an immune plant might be found to form the basis of a breeding program whose objective would be the development of resistance to stunt.

**Mummy Berry**, a fungus disease, is widespread in commercial blueberry fields in eastern North America. In the northern zones it sometimes destroys the crop of some varieties. In the Pacific Northwest and in North Carolina it is rare on the cultivated blueberry but more or less abundant on other blueberry species.

The causal fungus, *Monilinia urnula*, has a complicated life cycle. From harvest in early summer until the blueberry buds open the following spring, the fungus is found on the ground under the bushes in the form of mummies. The mummies are compact masses of fungus tissues that formed in infected berries the previous season. In early spring, when the blueberry buds start to open, the fungus renews its development, and mummy cups—the apothecia—are produced from the old mummies. Along the inner surface of the mummy cup sexual spores, or ascospores, of the fungus are produced in profusion. The ascospores, released during periods of rainy, cool weather, are carried by wind to the young flower and leaf buds. Ascospores landing on the moist surfaces of the young buds grow into the young leaf and flower bud tissues. After about a week the infections become apparent as necrotic areas on the petioles and along the midveins of the leaves in spur infections or at the base of the flowers in cluster infections. The infected spurs and clusters soon die, and conidia of the fungus are formed along the central axes of the spurs or clusters. The conidia are carried by wind or by insects to the stigmas of open flowers. The young berries
become infected and develop into new mummies, which carry the fungus through the summer and the following winter.

Weather conditions are important. In early spring the mummies must have moisture before apothecia and ascospores are produced. Primary infection of the spurs and clusters depends on wet weather at the time the buds are beginning to open. Secondary infection of the flowers depends on weather conditions that affect dispersal of the conidia. Differences in spring weather may explain the differences in the amount of damage done by mummy berry in different years. A cool, wet spring favors the outbreak of an epiphytotic.

Mummy berry was first identified in 1832 in Russia on the wild European lingberry, Vaccinium vitis-idaea. It was first discovered in North America on various wild blueberries in Michigan in 1898 by B. O. Longyear, of the Michigan Agricultural Experiment Station. He described the life cycle of the fungus essentially as we know it today.

Varieties differ considerably in susceptibility. June and Rancocas are blighted more severely by primary infections than are Weymouth, Cabot, and Stanley. There are also differences in the amount of secondary infection with different varieties.

It can be controlled at the time of primary infection in the spring. The young blueberry buds can be protected from infection through the use of ziram fungicide applied as a heavy spray over all the surfaces of the bushes, or the spores of the fungus can be destroyed before they blow to the bushes through the mechanical or chemical destruction of the mummy cups. Protection with ziram is more effective than destruction of the cups because the wind can blow spores to the bushes from distant sources. In order to obtain control, treatments must be applied just before the release of the spores from the cups. In New Jersey this usually occurs during the first or second week in April.

Botrytis blight is widespread in areas where blueberries are grown, but it often is overlooked because it is confused with mummy berry. It destroys the fruit when whole flower clusters are blighted at blossom time, when individual green berries become infected and rot, or when berries become infected at harvest time and rot during marketing or in cold storage.

The fungus can develop on stored berries at temperatures only slightly above freezing.

The disease attacks a variety of species. It is most severe in places where the weather is cool and wet when the blueberries are maturing and the bushes are producing new growth. The disease organism has been isolated from dead blueberry tips gathered in Washington, Michigan, Maryland, New Jersey, New York, Massachusetts, and Maine.

The cause is the common gray mold fungus, Botrytis cinerea. Besides producing blueberry fruit rots and tip blights, it occasionally produces leaf spots. It is abundant on such debris as fallen corollas, insect-damaged berries, and mummy lesions on or under the bush. Its production of spores reaches a peak during the period just after blossoming. After the weather becomes warm and dry in the summer, sporulation occurs only for short periods following rains. The fungus has been isolated from dead blueberry tips at all times during the year. In the spring the first sporulation of the fungus is observed on the dead tips.

The blight was one of the first diseases reported on commercial blueberries but no investigations of methods to control the disease were undertaken until 1949. No control method has been found for the disease.

Powdery mildew is widely distributed on cultivated and wild blueberries in eastern North America. It is not normally widespread on blue-
berry leaves until midsummer after the crop is harvested. For that reason it is not generally controlled in cultivated blueberry fields. Undoubtedly considerable damage results to the blueberry from the early defoliation that it causes in susceptible varieties, but the damage is hard to evaluate. On the lowbush blueberries, which are extensively harvested in Maine, it is one of the worst fungus disorders.

It is caused by the fungus *Microsphaera penicillata*, which occurs on a number of different plants. Probably blueberry strains of the fungus are confined in their host range to *Vaccinium* species and other closely related plants, but no cross-inoculation work with the organism has been done.

The fungus grows only on the surface of living blueberry leaves. In late summer the web of fungus growth on the surface of the blueberry leaf produces tiny, dark-brown, round bodies—cleistothecia—which fall to the ground with the leaves and carry the fungus over winter. Within them, in asci, the sexual spores of the fungus are produced. In spring, when the ascospores are mature, the cleistothecia rupture and release the ascospores into the air. Wind may carry them to young leaves of the blueberry. If conditions are favorable, the spores germinate and initiate small spots on the leaf surfaces. In late spring and early summer the spots remain so small that they are generally overlooked by the casual observer. The fungus continues to develop slowly, however, and when the infection is well established on susceptible varieties about the middle of the summer, a powdery layer of summer spores, or conidia, is produced on the leaf spots. These spores spread the fungus rapidly to uninfected areas. On susceptible varieties the fungus may cover the entire leaf surface. In late summer the infected areas produce a new mass of cleistothecia, which repeat the cycle for the following season.

The susceptibility of different varieties and selections to the disease varies considerably. Some are badly infected, others have scarcely a single infected leaf. No variety is immune to the disease although some resistant ones do not become infected until the middle of October. Most of the named varieties that have been introduced by the Department of Agriculture since 1940 are highly resistant. Jersey, which is planted most extensively in New Jersey is very susceptible. Cabot and Pioneer, which are most susceptible to the disease, are being replaced with newer varieties that have superior horticultural qualities and resistance to powdery mildew.

The disease can be controlled by dusting or spraying. On the lowbush blueberries in Maine, applications of copper-lime dust (25 pounds of monohydrated copper sulfate mixed with 75 pounds of hydrated lime) at the time of blossom drop and again 10 days later will check the disease. The dust should be applied at the rate of 40 pounds to the acre to be effective.

On cultivated blueberries, cover sprays with wettable sulfur (3 pounds in 50 gallons of water) or bordeaux mixture (4 pounds copper sulfate and 4 pounds hydrated lime made to 50 gallons of spray with water) at the end of blossoming give good control. Either material should be applied at the rate of 300 gallons an acre to be effective.

**STEM CANKER** is injurious on cultivated and wild highbush blueberries and the wild rabbiteye blueberry in the southeastern United States. It has also been found on cultivated bushes in a few places in New Jersey.

It is caused by *Physalospora corticis*, a fungus that lives on the living bark of the blueberry and produces cankers on the twigs and stems. Both sexual and asexual spores are produced on the surface of the cankers over a long period during the growing season. In wet weather, spores are liberated to uninfected parts of the bush or may be blown to nearby bushes.

If weather favors infection and the spores land on susceptible blueberry tissue, a new canker will form; it
appears as a reddish, conical swelling, which does not enlarge the first season. Thereafter it swells gradually. After a few years it may girdle and kill the stem. The use of infected cutting wood or rooted cuttings to establish new fields may carry the disease great distances.

Symptoms vary with the susceptibility of the infected bushes. Sometimes the fungus may penetrate the bark and make big cankers with deep cracks and fissures on the main stem of the plant. On less susceptible varieties the cankers are less extensive, they are not swollen, and they may form only on side branches.

Varieties differ in susceptibility. Only a few varieties—Angola, Wolcott, Murphy, Scammell, Atlantic, Ivanhoe, Jersey, Rubel, and Ranocas—are resistant enough to remain in commercial use in North Carolina.

Stem canker was first observed in North Carolina in 1938. Some fields then were already heavily infected. The disease was observed in New Jersey in 1951. It is hard to control in North Carolina. Fungicides have been ineffective because the fungus produces spores over a long period during the season. Roguing or removal of infected bushes is of little value because the disease may become reestablished from the wild. Resistant varieties, such as Murphy, Wolcott, and Angola, offer the only possible control. In New Jersey, where the disease does not occur in the wild, the eradication of individual cankers or entire infected bushes probably would eliminate the disease.

Several other diseases have been reported on blueberries in North America and Europe. Most are local or serious mainly on Vaccinium species other than the cultivated highbush blueberry.

Witches'-broom, or stem rust, is caused by the fungus Pucciniastrum goeppertianum, a heteroecious rust with fir, Abies species, as the alternate host. It occurs on cultivated and wild blueberries in New England and on wild blueberries in many places. It produces several short, swollen twigs on infected bushes, which are crowded on a small area near the point of infection. Heavily infected bushes produce no fruit. Possibly the disease can be controlled by removing all the true fir trees from the vicinity of the blueberry field. Pruning the infections from bushes might also be of some value.

Leaf rust is caused by the fungus Pucciniastrum myrtillii, another heteroecious rust with hemlock, Tsuga, as the alternate host. It normally occurs on various blueberry species only in areas where hemlock trees are present. In some seasons it occurs in severe outbreaks in cultivated fields far removed from the alternate host. The fungus can evidently be spread rapidly by uredospores after an infection is once established. The fungus produces small, irregular, dark-brown spots on the leaves. The spots may become numerous later in the season and cause early defoliation. No control is known for the disease on cultivated highbush blueberries, but copper-lime dusts applied at the time of blossom drop, followed by an additional application 10 days later, have been effective in controlling the disease on lowbush blueberries in Maine. This is the same treatment that is used against powdery mildew.

Several leaf-spot diseases have been reported on blueberries in North Carolina and other parts of the Southeast. The most important is double spot, caused by the fungus Dothichiza caroliniana. The disease occasionally causes severe defoliation in North Carolina and must be controlled by spraying with bordeaux mixture.

Crown gall of blueberries is caused by the bacterium Agrobacterium tumefaciens, which produces galls on many other plants. It is found on highbush blueberries in New York, New Jersey, Michigan, Washington, and British Columbia. It occurs on plants in the field, in nurseries, and in cutting beds.
It produces swollen galls along the stems and small twigs and occasionally at the base of the canes near the ground. The galls are irregular and of various sizes. Often the gall involves the entire circumference of the branch and affected branches and twigs die. In Washington certain varieties and selections are badly infected, while plants of a different selection in adjacent rows may be healthy. Although various other host plants become infected when inoculated by bacteria isolated from blueberry galls, the blueberry strain of the crown gall organism appears to be best adapted to the blueberry. The disease is probably spread in the field or nursery when plants are pruned or when softwood cuttings are made. No attempts have been made to control the disease in blueberry fields.

Tip blights of blueberries occasionally are common. They are probably caused by winter injury, excessive soil moisture, or other environmental factors that weaken the bush. Frequently stem tips of bushes, weakened by other causes, are infected by weakly parasitic fungi. *Diaporthe (Phomopsis) vaccinii* is reported to be a cause of tip blight in North Carolina, New Jersey, and New England. That fungus is not often encountered, however, and is only one of a number of fungi that may be found in blighted tips. In Washington *Diaporthe vaccinii* occurs as a saprophyte on dead blueberry tips.

AUSTIN C. GOHEEN, a native of Washington State, received his undergraduate training at the Western Washington College of Education and the University of Washington and his graduate training at the State College of Washington. He has been with the Bureau of Plant Industry, Soils, and Agricultural Engineering since 1950. He has worked on blueberry and cranberry diseases in New Jersey in cooperation with the New Jersey Agricultural Experiment Station. He was transferred to the Plant Industry Station at Beltsville in 1953.

Disorders of Cranberries

Herbert F. Bergman

Fruit rots cause an annual loss of 10 to 15 percent in our crop of cranberries, which are grown extensively in Massachusetts, Wisconsin, and New Jersey and less widely in Washington, Oregon, Maine, and Rhode Island. The value of the crop is 10 million to 20 million dollars a year.

Two or three decades ago the losses went as high as 25 percent, but they have been reduced by changes in cultural practices, improvements in fungicides, greater efficiency in handling and storing, and the development of the canning industry, which offers an immediate and profitable outlet for the berries that—although sound at harvest—probably would develop excessive decay if they were stored and shipped as fresh fruit.

Directly affecting the abundance of rot are the amount and frequency of rain and the temperature and humidity. Humidity and temperature (to a lesser extent) are affected by local conditions. A local condition that bears on humidity is the density of vine growth. Excessive growth prevents the evaporation of water among and under the vines after rain, fog, or dew. Thus the atmosphere may remain saturated or nearly saturated for hours after the air and soil surface under the thinner vines have dried. Poor drainage also keeps the soil wet and makes a high humidity under the vines.

The application of nitrogenous fertilizers, late holding of the winter flood