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Anthracnose of Strawberry Caused by the *Colletotrichum* Complex in Florida

Strawberries, perhaps the most delectable of fruits, are grown for their attractiveness and exquisite flavor in most temperate and many tropical areas of the world. Some feel the world would indeed be a dull place without the strawberry to perk taste buds and enliven desserts and pastries.

Strawberry growing can be traced back thousands of years. The origins of strawberry cultivation in Europe is well documented in literature and art (5,10, 27). *Fragaria vesca* L. (diploid, $2n=14$ chromosomes) and *F. moschata* (hexaploid, $2n=42$ chromosomes) were cultured in palace and church gardens. Strawberry culture in the New World of North and South America is less well documented. North American natives appreciated the appearance of *F. virginiana* Duchesne (octoploid, $2n=56$ chromosomes) fruit in spring, but this species apparently was not cultivated until Europeans began exploring and settling North America. However, native inhabitants of Chile and Peru had selected and cultured superior variants of *F. chiloensis* (L.) Duchesne (octoploid, $2n=56$ chromosomes) well before the arrival of the conquistadores in the 16th century.

The modern strawberry, *Fragaria* × *ananassa* Duchesne, is of relatively recent origin. Not until the North American *F. virginiana* and the South American *F. chiloensis* were brought together and hybridized in Europe around 1750 did the progenitor of the strawberry we know today come into being. The high degree of genetic heterozygosity in *Fragaria* spp. has enabled the development of strawberry cultivars adapted to widely varying environmental conditions and resistant to several diseases and pests. Available today are cultivars that can survive the bitterly cold winters of Alaska and others that can perform well in the long, hot season of Pretoria, South Africa.

Although a minor crop, strawberries provide a major source of income for many fruit growers and nursery growers. Strawberries were grown on nearly 20,000 ha in the United States in 1989. Total production for fresh market and processing use totaled 555,000 t and was valued at $520 million. Florida is second only to California in the production of fresh-market strawberries. Strawberries for U.S. winter production (December through March) are grown on approximately 2,400 ha.

The major cultural problems for the Florida strawberry industry are 1) anthracnose diseases, 2) yearly infestations of the two-spotted spider mite (*Tetranychus urticae* Koch), and 3) variability in the quality of nursery transplants. Commercial fields are established on an annual basis, as in California. Freshly dug transplants are obtained from nurseries in Florida or in the northern states and Canada. Use of transplants from northern nurseries is necessary to avoid anthracnose disease problems and to obtain plants that have received some natural chilling to induce floral bud initiation.

The warm, humid climate of Florida that permits year-round growth of strawberry plants in nursery and commercial fields is ideal for the development of a large number of diseases, especially anthracnose. Anthracnose diseases of strawberry have become significant in other areas of North America and the world. Because of the importance of strawberry anthracnose in Florida and increasing concern for the disease in other strawberry-producing areas of the world, we are presenting the history of its occurrence, symptoms caused by the *Colletotrichum* complex in Florida, and control measures developed for Florida strawberry growers.

History of Anthracnose in the United States

The term "anthracnose" first was used to describe a new disease of strawberry caused by *Colletotrichum fragariae* Brooks (1). "Strawberry anthracnose" has continued to be used as a general term to identify all diseases of strawberry caused by species of *Colletotrichum*. Strawberry anthracnose diseases have been described as caused by *C. acutatum* J.H. Simmonds, *C. gloeosporioides* (Penz.) Penz. & Sacc. in Penz. (teleomorph of *Glomerella cingulata* (Stoneman) Spauld. & H. Schrenk.), and *C. dematium* (Pers.) Grove. *C. dematium* only occasionally causes strawberry fruit rots and is not discussed here.

Anthracnose fungi may attack crowns, petioles, leaves, fruit trusses, flowers and buds, and fruit. The ability of the fungi to attack more than one plant part, and the fact that several pathogen species are involved, adds to the complex nature of strawberry anthracnose.

Brooks reported in 1931 (1) that *C. fragariae* caused spotting and girdling of runners and later reported (2) that it also caused spotting of petioles and a crown rot in a summer nursery that resulted in wilting and death of plants. Researchers in Louisiana found all of these symptoms and reported that crown rot also occurred in the fruit production field (3,13,14). In 1972, Howard (16) described a fruit rot in Florida that was caused by *C. fragariae*. Fruit rots caused by anthracnose fungi have also occurred in several other areas of the United States, e.g., *Gloeosporium* sp. in Maryland (21), *C. acutatum* in Ohio (9), and *Colletotrichum* spp. in

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several eastern and western states (8; C. M. Howard and J. L. Maas, *unpublished*). In 1983, Howard and Albregts (18) described black leaf spot caused by *C. fragariae*.

Anthracnose became a problem in North Carolina during the mid-1970s after growers there had been producing plants of cvs. Tioga and Fresno for a few years for shipment to Florida. Anthracnose became serious in Tennessee and Arkansas in the late 1970s. However, plants from North Carolina, Tennessee, and Arkansas usually were infected by *G. cingulata* rather than by *C. fragariae*. *G. cingulata* was first found in Florida nurseries in 1978. Since the early 1980s, *G. cingulata* has been isolated more frequently than *C. fragariae* from Florida nurseries. In 1984, Howard and Albregts (19) reported anthracnose fruit rot caused by *G. cingulata* in Florida.

For many years, strawberry anthracnose was thought to be exclusively a disease of the southeastern United States. Since 1972, however, *C. fragariae* has been found occasionally in plants that were grown in California and then planted in Florida fields. Since 1982, *G. cingulata* also has been found in a few of these plants. Since the early to mid-1980s, *C. fragariae* or *G. cingulata* or both have been found in plants arriving in Florida from Michigan, Maine, New York, Nova Scotia, and Ontario and *C. acutatum* has been found in plants arriving from Michigan and Ontario. To date, the incidence of anthracnose crown rot has not been serious in northern-grown plants, but the disease has obviously occurred farther north as plant production of highly susceptible cultivars has moved in that direction.

C. acutatum first was identified as causing strawberry anthracnose in the United States in 1983 in Mississippi (25). However, a severe outbreak of fruit rot caused by a *Colletotrichum* sp., later identified as *C. acutatum*, occurred in California in 1982. This species was found causing fruit rot in Florida in 1984 (C. M. Howard, *unpublished*). Since then, *C. acutatum* has been found on runners, petioles, and fruit in most areas of the United States and on runner stubs, petioles, and leaves of plants from Ontario and Nova Scotia soon after arrival of the plants in Florida. This species is becoming increasingly important, primarily in causing fruit rot, in many areas of the United States.

Disease Phases and Symptoms

Anthracnose of strawberry may be manifested in several ways. The term applies to all phases of the disease but is often reserved for the lesions on stolons and petioloes. Other phases are usually described by the identifying symptoms, i.e., crown rot, flower blight, black leaf spot, etc.

Anthracnose. Lesions on stolons and petioles are classic: sunken, firm, dark, dry, with a sharp line of demarcation between diseased and symptomless tissue. When a lesion girdles a runner, unrooted daughter plants beyond the lesion wilt and die. Lesions may continue to elongate until the entire runner or petiole is infected. When a petiole is girdled, the leaf dies and turns brown. Lesions often form on the underside of a petiole. The petiole then bends sharply at this point, and the leaf hangs down. Leaves may continue to live in this position and remain green for an extended period of time.

Crown rot. Crowns of plants in the nursery become infected when the fungus grows into the crowns from runner or petiole lesions or when a sufficient number of spores germinate in the central buds (6), as when spores are splashed or washed into the central bud from runner and petiole lesions. Plants with crown rot may die in the nursery or after being transplanted into fields. Infected plants, after being planted in fruit production fields, may grow normally for some time, then wilt suddenly and die. A reddish brown, firm rot or reddish brown streaking in portions of the interiors of crowns of wilted plants (Fig. 1A) can be seen most readily by cutting through the crown lengthwise. The reddish tint is the diagnostic feature. The crown of a plant turns brown regardless of the cause of death, but a darker brown without the reddish hue usually means that agents other than the anthracnose pathogen killed the plant.

Bud rot. Bud rot may develop in plants within a few days after transplantation or a few weeks later after the plants have become established and formed multiple crowns. In either case, a damp, firm rot develops and the infected bud turns dark brown to black. Plants have a single bud (apical meristem) when they are planted, and when this single bud decays, the entire plant dies. Rot usually develops in only one bud of multicrowned plants. Then the entire plant may wilt and die as bud rot progresses, or only the infected crown may die while the remaining crowns continue to grow. Pink masses of *Colletotrichum* conidia are found on the surfaces of the decaying tissues when the infected buds are dissected. A longitudinal cut through the infected bud and the crown exposes a sharp line of demarcation between the dark infected bud tissue and the crown tissue below the bud (Fig. 1B). The interior portions of the crowns of these plants appear white and healthy. Even in multiple-crowned plants in which the entire plant wilts and dies, there is no reddish brown discoloration.

Flower blight. Flowers and flower buds of some cultivars, such as Pajaro and Irvine, are highly susceptible to anthracnose and may become infected at any time after the bud first begins to emerge from the crown until the flower is fully open. When newly emerging buds are infected, the sepals dry and turn dark brown to nearly black. At this stage, affected buds resemble those affected by tip burn resulting from excessive fertility. As the stem elongates, the entire bud becomes infected, dries, and turns light brown. When a flower bud becomes infected after the stem has begun to elongate, a dark lesion forms on the calyx or on the stem immediately below the calyx. Infection spreads throughout the bud, which dries and becomes light brown either before or after the flower opens. In the early stages, lesions on these buds are similar to those caused by *Rhizoctonia* spp., except that lesions caused by *Rhizoctonia* usually are purple. When an open flower becomes infected, the pistils and ovules turn black and the calyx and a portion of the stem becomes tan or light brown and dry.

Sticky, gelatinous masses often form on infected stems of flowers and flower buds. These masses apparently are sap exuded from the plants and sometimes contain spores of *Colletotrichum*. Stems usually become light red below the point of infection, sometimes even before lesions are visible. Lesions may extend 5–7.5 cm below the flowers. Red is not diagnostic of anthracnose, however; *Botrytis cinerea* Pers.:Fr. (the cause of gray mold), other organisms, and apparently some environmental conditions also can cause the stems to become red.

Fruit rot. Round, firm, sunken spots that develop on ripening fruit (Fig. 1C) usually become black but sometimes remain light tan for a few days, especially during wet weather. The spots may enlarge until the entire fruit is infected. The fruit then dries and mummifies. In most susceptible cultivars, this rot most often affects ripe fruit, but green fruit in all stages of development can be affected when rot becomes severe in a field. The green fruit of some cultivars, such as Pajaro and Irvine, are very susceptible and nearly all green fruit may become infected. Lesions on green fruit are dark brown to black and hard (Fig. 1D). One cannot distinguish between anthracnose and *Rhizoctonia* lesions on green fruit without magnification. Some seeds on anthracnose-infected fruit have very small, crusty masses of spores. These masses can be seen, with difficulty, with a 10× hand lens. Seeds on *Rhizoctonia*-infected berries are smooth and free of these crusty masses.

Black leaf spot. Spots are round and range in diameter from 0.5 to 1.5 mm and occasionally up to 3 mm. Lesions are usually black but may remain light gray (Fig. 1E). Leaflets can have numerous spots without dying. Black leaf spot usually is present in areas of nursery fields where symptoms of anthracnose are severe on runners and petioles but

often is found before anthracnose is detected on runners or petioles, usually on the expanding leaves of the youngest runner plants. Black leaf spot thus can serve as an early warning that the anthracnose pathogen is present in the nursery. At the first sign of black leaf spot, all possible efforts to control anthracnose should be initiated.

Irregular leaf spot. These dry lesions are dark brown to nearly black and occur on the margins and tips of leaflets (Fig. 1F). Lesions vary in size from those extending only about 3 mm along the margin and 1.5 mm toward the center of the leaflet to those extending 18 mm along the margin and inward. Lesions are variable but tend to be elongated with irregular margins.

In Florida, *C. fragariae* and *G. cingulata* can induce all of the symptoms described except irregular leaf spot and bud rot, and *C. acutatum* can induce all of the symptoms except crown rot and black leaf spot. *C. acutatum* occasionally causes crown rot and death of plants in Mississippi (26) and California (8) but has not been associated with crown rot in Florida even when spores are injected into the plants (C. M. Howard, *unpublished*). During the fall of 1990, however, *C. acutatum* caused bud rot that led to the wilting and death of some plants; these plants did not show the reddish brown discoloration in their crowns that is characteristic of crown rot infections.

Disease Development

The origin of the inoculum that initiates the first infection in a strawberry nursery is unknown. *C. fragariae* does attack coffeeweed (*Cassia obtusifolia* L.) (17) and might survive from one summer to the next on this weed, as well as on other nonstrawberry hosts. *C. fragariae* has been reported to infect *Duchesnea indica* (Andr.) Focke (1) and *Potentilla canadensis* L. (7) after controlled inoculation. *G. cingulata* and *C. acutatum* may survive also on some nonstrawberry hosts. Many of the fungi that cause anthracnose of tomato, eggplant, cucumber, watermelon, and other crops are closely related to the strawberry pathogens and can infect strawberry fruit under experimental conditions (23), but we do not know if they can infect strawberries in the field.

The strawberry anthracnose pathogens do not survive from year to year in the soil in Florida and Louisiana. In Louisiana, strawberry plants potted into soil obtained from a planting severely affected by *C. fragariae* did not become infected (15). Also, the pathogen was grown on oat medium, mixed into sterilized soil, and then placed in a large crock and buried in the field. Every 3 months for 1 year, portions of the soil were placed in pots and healthy strawberry plants were planted into it. None of the plants became diseased.

Numerous observations in Florida indicate that in commercial plantings in which up to 80% of the plants died from crown rot caused by *C. fragariae* or *G. cingulata*, no anthracnose or crown rot developed the following year if plants from nurseries were free of anthracnose. Also, isolation of either pathogen from crowns of wilted plants is uncommon 2 or 3 weeks after plants have died, whereas they are often the only organisms isolated from interior parts of crowns during the first few days after the plants have wilted.

In California, *C. acutatum* survived in soil and in infected, buried runners for up to 9 months but could not be detected in soil after fumigation with a methyl bromide–chloropicrin mixture (8). In the warmer, moister Florida climate, *C. acutatum* probably does not survive for extended periods in soil or buried plant debris. In Florida, when anthracnose caused by *C. acutatum* is severe in nurseries or on fruit on farms, it commonly is absent the following year. Thus, it appears that none of the three pathogens survives for extended periods in soil or infested plant debris in Florida. Regardless of the source of inoculum, after aerial parts of one or a few plants are infected, many spores are produced on the lesions that develop. These spores are blown or splashed by water to other plants, and the pathogens spread quickly under conditions of high temperature and moisture that favor disease development.

Crown rot occurs on farms when plants that become infected by *C. fragariae* or *G. cingulata* in the nursery are transplanted in the fall. *C. acutatum* has not caused typical crown rot in Florida even after spores have been injected into crowns. Crown rot is difficult to detect in shipments of plants, even by cutting through large numbers of crowns before planting, because nursery growers generally avoid digging plants from obviously diseased areas. Infections in plants from nurseries generally are in the initial stages, before red streaks have developed in crowns. The pathogens continue to invade crown tissue, however, and the plants die sometime during winter or spring. The time of death depends on the stage of infection at transplanting, on the temperature during fruit

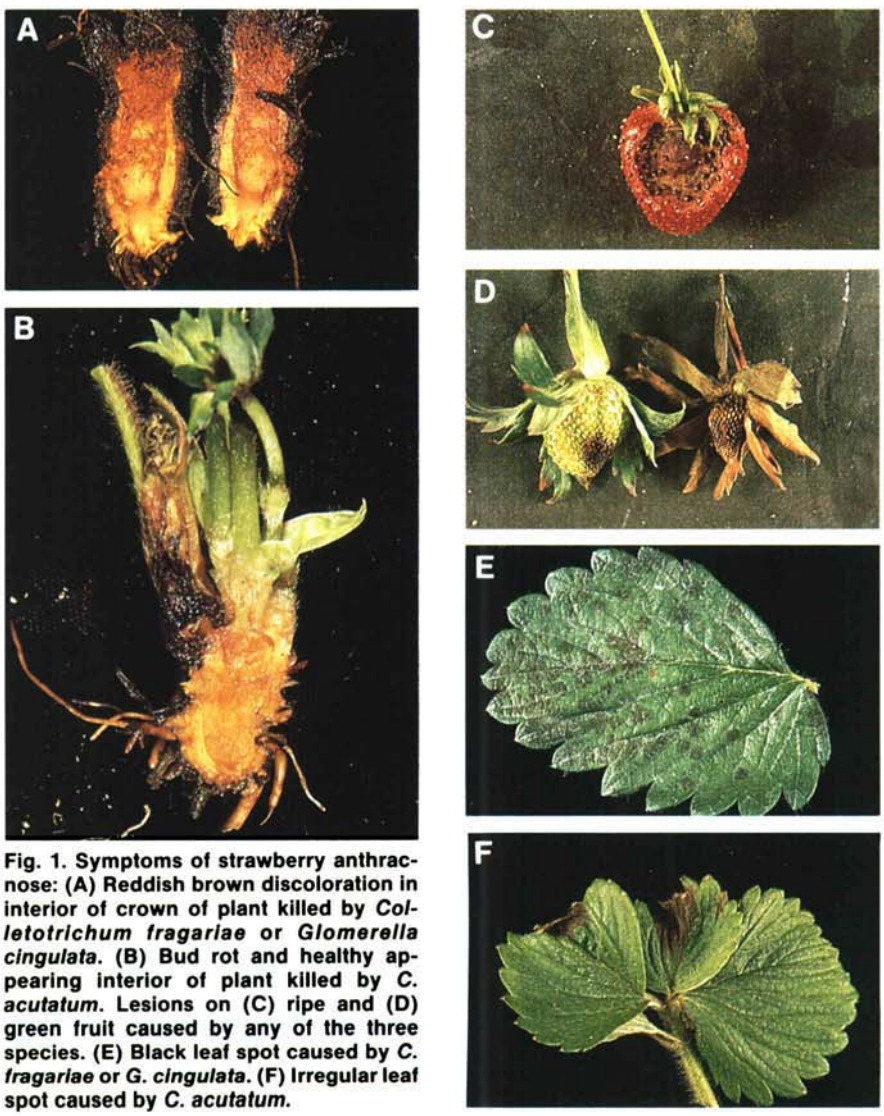

Fig. 1. Symptoms of strawberry anthracnose: (A) Reddish brown discoloration in interior of crown of plant killed by *Colletotrichum fragariae* or *Glomerella cingulata*. (B) Bud rot and healthy appearing interior of plant killed by *C. acutatum*. Lesions on (C) ripe and (D) green fruit caused by any of the three species. (E) Black leaf spot caused by *C. fragariae* or *G. cingulata*. (F) Irregular leaf spot caused by *C. acutatum*.

production, and perhaps on other factors. Under low temperatures, the fungi can survive in infected crowns for long periods without killing the plants. Horn and Carver (15) injected spores into crowns, allowed 4 days at 28–32 C for infection to occur, then placed the plants at 4.4 C with 10 hours of light daily. Every 6 weeks for 30 weeks, they removed some of the plants from 4.4 C and placed them at 28–32 C. None of the plants wilted or died at 4.4 C, but even after 30 weeks, some of the plants died from crown rot after they were placed at 28–32 C.

There apparently is little or no spread of crown rot in Florida's commercial fields. When plants from an anthracnose-free nursery are planted adjacent to those from an infested nursery (sometimes even on the same bed), plants from the disease-free nursery do not die even though high percentages of those from the infested nursery wilt and die. Because spores are abundant on infected fruit, it follows that petioles and crowns should become infected after fruit rot, but petiole infection has not been observed on farms in Florida. Very little infection of petioles occurs when fruiting plants are inoculated in the greenhouse, even though temperatures are favorable for anthracnose development. Thus, it appears that petioles at fruiting stages are less susceptible to infection than petioles of plants in the runner production stage, even though the fruit are highly susceptible.

Crown rot sometimes does occur after severe outbreaks of fruit rot caused by *C. fragariae* or *G. cingulata*. However, plants do not begin to wilt and die until April, near the end of the fruit production period, even when fruit rot occurs in December or early January. Thus, crown rot that is initiated by inoculum from the fruit usually is not economically important under Florida cultural conditions.

In past years, when all Florida strawberries were transplanted after the first of October, it appeared that crown rot was never initiated in commercial fields. In autumn 1990, however, crown rot in one field, planted in mid-September, appeared to originate there. Nearly all plants in an area approximately 60 × 60 m at one end of the field had died.

Black leaf spot occurs when spores of *C. fragariae* or *G. cingulata* are deposited on leaves and cause infections in the summer nursery or in May in the winter nursery. Black leaf spot has not been found in commercial strawberry production fields. When plants with black leaf spot are transplanted into commercial fields, the infected leaves die and dry up and new leaves do not become infected. Therefore, black leaf spot apparently is not the initial source of inoculum for flower blight or fruit rot.

Irregular leaf spot occurs when young expanding leaves become infected by *C. acutatum* in the summer nursery. The leaves with these lesions persist after the plants are transplanted, and the first one to three leaves that emerge after transplantation often become infected. Lesions cease to develop as the leaves continue to expand, thereby limiting lesion size. The infected leaves persist on the plants for at least 2 or 3 months.

Sporulation occurs on the lesions, thus supplying inoculum for infection of the first flowers and fruit that emerge from the crowns in the fall. Irregular leaf spot probably is not initiated on disease-free nursery plants after they are transplanted.

Fruit rot and flower blight may develop at any time during the fruit production season. The flower buds, flowers, and green fruit of some cultivars are extremely susceptible to all three *Colletotrichum* species. When irregular leaf spot, caused by *C. acutatum*, is present at the time of transplantation, spores from the lesions often infect the first few flower buds as they emerge from the crowns. Spores produced on infected buds and on leaf lesions are splashed or blown to other flower buds, flowers, and fruit on these and surrounding plants and initiate new infections. The disease spreads very quickly throughout a field of these cultivars, and effective control is impossible the rest of the season. Infected flowers and flower buds dry and remain on the plants for 2 or 3 months. Sporulation continues as long as they remain on the plants. Thus, secondary inoculum for infection of more fruit and flowers is produced continuously. In these fields, 50% or more of the potential fruit yield may be lost, even when the most stringent fungicide schedules are maintained and removal of all flower buds, flowers, and fruit with lesions is attempted immediately after each harvest.

Flower blight and fruit rot usually are not problems on anthracnose-susceptible cultivars in which the flowers and green fruit are not extremely susceptible, even if irregular leaf spot is present on the plants when transplanted. Generally, fruit rot becomes a problem only on ripe fruit of these cultivars, and then only if overripe fruit accumulates in the field. Fruit rot usually can be brought under control if it is not late in the season. In some instances, blight of the first flowers that emerge in the fall does become a problem on these cultivars but usually can be controlled with a stringent fungicide program.

Fruit rot and flower blight often do not occur until midseason or later. The initial inoculum for these infections probably consists of spores that enter fields from local nonstrawberry hosts. After a few flowers or fruit are infected, the disease spreads within the field. Fruit rot on cultivars with less susceptible flowers and green fruit can usually be controlled if overripe fruit is not allowed to accumulate in the field. Rainy periods of 3 or 4 days are especially favorable for very rapid buildup of disease on less susceptible cultivars.

A high incidence of crown rot caused by *C. fragariae* or *G. cingulata* does not necessarily indicate that fruit rot or flower blight will occur. Fields in which 30–40% of the plants have died from crown rot often have little or no fruit rot, presumably because the pathogens usually do not grow from the crowns into aerial parts of the plant. With the absence of spore production on aerial parts, inoculum resulting from crown infection is not available for dissemination and infection of flowers and fruit. When fruit rot occurs in fields in which crown rot is present, the inoculum probably was produced nearby on nonstrawberry hosts or on lesions of irregular leaf spot (caused by *C. acutatum*) that were present at transplantation. *C. acutatum* may be present with either of the other two species in the same lot of plants.

Bud rot was observed for the first time in Florida during fall 1990 in plants from three nurseries. Anthracnose lesions on many petioles and expanding bud leaves on the plants from two nurseries were present at time of transplantation. Buds in these plants began to rot within 2 or 3 days, and nearly all plants from these two nurseries were dead within 2 weeks. Only small irregular leaf spots were present on some plants from the third nursery at time of transplantation. Most plants became established and formed one to three new crowns. Rot developed in the buds of some plants, however. Only the infected bud died, and the remaining crowns continued to grow. Sometimes, entire plants wilted and died after rot progressed sufficiently to kill the infected bud. Approximately 10–15% of the plants from this nursery died during the October–April fruiting season. Only *C. acutatum* was isolated from plants with bud rot.

Control Measures

Stringent methods are needed to prevent or manage the various symptoms of disease in the summer nursery (20). Summer nurseries should be established only on land with very low residual fertility because high fertility favors development of the disease (20,24). One should use only enough fertilizer to establish plants and little or no fertilizer during July and August; applications may be resumed in September.

Fungicides should be applied at least every other day from mid-June through mid-September. The nursery should be carefully inspected frequently. If any symptoms of anthracnose, black leaf spot, or irregular leaf spot are found, all applications of nitrogen and potash fertilizer should be stopped and fungicides should be applied after every rain or period of overhead irrigation. Two fungicide applications per week should be continued from mid-September until

the plants are dug. Resistant cultivars such as Dover may be grown successfully under much higher fertility regimes and with two fungicide applications per week throughout the summer.

Crown rot and irregular leaf spot on farms can be prevented by controlling anthracnose in the summer nursery.

Fruit rot and flower blight are two of the most difficult diseases to control. They have caused loss of all fruit in some instances and early abandonment of many fields. All possible efforts must be made to prevent the occurrence of these diseases. Plants with irregular leaf spot must be avoided, especially for cultivars in which the flowers and green fruit are highly susceptible. Controlling fruit rot and flower blight is usually impossible on these cultivars if they have irregular leaf spot when planted. Without close inspections of the nursery, however, irregular leaf spot is very difficult to detect before planting. Even then, suspicious lesions often can be definitely diagnosed only with the aid of a microscope, and sometimes only by isolation from the lesions. Lesions caused by fertilizer burn and physiological tip burn sometimes appear nearly identical to those of irregular leaf spot.

For fruit production fields in Florida, approved fungicides should be applied twice each week on all susceptible cultivars beginning no later than the early blossom stage in the fall. After harvest begins, fungicides should be applied after each harvest if possible, especially on cultivars such as Pajaro and Irvine that have extremely susceptible flowers and fruit. Because fruit rot occurs primarily on ripe fruit on most of the susceptible cultivars, 3- or 4-day picking schedules should be employed to prevent accumulation of overripe fruit in the field. Frequent harvest is especially important during warm weather because the anthracnose pathogens are favored by temperatures above 24 C.

All infected fruit should be removed from the plants at each harvest. If fruit rot, in the absence of flower blight, becomes moderate to severe on ripe fruit, the most economical method may be to strip all ripe and pink fruit from the plants, then apply registered fungicides every other day until control has been achieved or until harvesting is nearly ready to resume. If many green fruit also are infected, it may be necessary to strip all ripe and green fruit from the plants before beginning the intensive fungicide program. These methods sometimes have controlled anthracnose fruit rot even when flowers and green fruit were extremely susceptible, but only when the rot problem began in early to mid season and only if flower blight had not yet occurred and irregular leaf spot was absent.

Sanitary procedures must be practiced during harvesting. Pickers should never move from an infested field to a noninfested field. Spores are produced abundantly in sticky masses on fruit and flower lesions and almost certainly would be moved easily from field to field on pickers' hands and clothing. If pickers must move from an infested to a noninfested field, they should be required to wash their hands and arms thoroughly before entering the noninfested field.

Resistance to Anthracnose

Strawberry cultivars vary in susceptibility to anthracnose diseases of petioles, stolons, crowns, leaves, flowers, and fruit (Table 1). Resistance to these diseases is being incorporated into new cultivars in breeding programs of state agricultural experiment stations at Dover, Florida, Baton Rouge, Louisiana, and Raleigh, North Carolina, and in USDA programs at Poplarville, Mississippi, and Beltsville, Maryland.

The genetics of anthracnose resistance is complicated (9), but observations in Florida indicate that strawberry clones resistant to any one of the three anthracnose pathogens also are resistant to the other two. Clones that are resistant to infection of stolons and crowns are resistant to all other phases of the disease (C. M. Howard, *unpublished*). Smith and Black (26) indicate that in Mississippi, clones that are resistant to *C. fragariae* tend to be resistant to *C. acutatum* as well. No cultivars are known to be resistant to all forms or races of *C. fragariae, C. acutatum*, and *G. cingulata*. Thus, a cultivar that is resistant to the predominant pathogen in one area may be susceptible to those in another area.

The interaction between environmental conditions and expression of cultivar resistance is a significant consideration in selection of planting stocks. For example, Sequoia and Cardinal are resistant at temperatures up to 25 C but become susceptible at temperatures at or above 32 C (5,25). The duration of high humidity conditions also affects resistance in cultivars. Delp and Milholland (6) reported that Sequoia remained resistant after 48 hours under high humidity conditions following inoculation but became susceptible after 72 hours of high humidity. Breeding programs are developing cultivars having high levels of resistance under conditions favorable for disease development while recognizing that pathogenic components of the disease may vary from one location to another (4,6,11,12,22,26).

Table 1. Susceptibility of several strawberry cultivars to various phases of anthracnose in Florida[a]

Cultivar	Ratings[b] of anthracnose according to phase						
	Stolon and petiole spot	Flower blight	Green fruit rot	Ripe fruit rot	Crown rot	Black leaf spot	Irregular leaf spot
Pajaro	VS	VS	VS	VS	VS	VS	VS
Irvine	VS	VS	VS	VS	VS	VS	VS
Selva	VS	MS	MS	VS	VS	VS	VS
Tufts	VS	S	S	VS	VS	VS	VS
Chandler	VS	S	S	VS	VS	VS	VS
Tioga	VS	S	S	VS	VS	VS	VS
Fresno	VS	S	S	VS	VS	VS	VS
Sequoia	S	R	R	MS	MS	...[c]	...
Florida 90	MR	VR	VR	MR	MR	...	...
Florida Belle	R	VR	VR	MR	MR	...	...
Dover	VR	VR	VR	MR	VR	VR	VR

[a] In Florida, *Colletotrichum fragariae* and *Glomerella cingulata* cause all phases of anthracnose except irregular leaf spot and bud rot, and *C. acutatum* causes all phases except crown rot and black leaf spot.
[b] VS = very susceptible, MS = moderately susceptible, S = susceptible, R = resistant, MR = moderately resistant, VR = very resistant.
[c] No record available.

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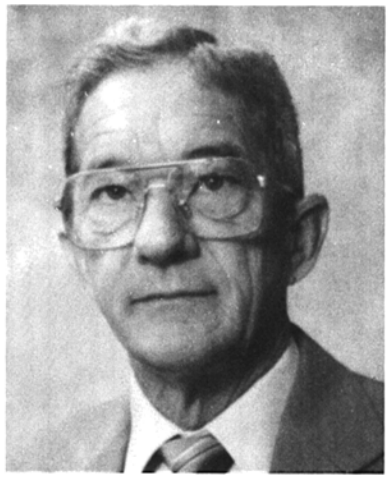
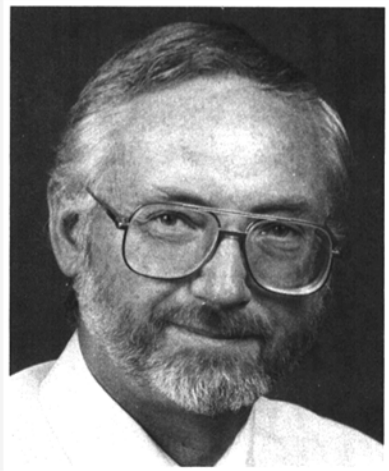


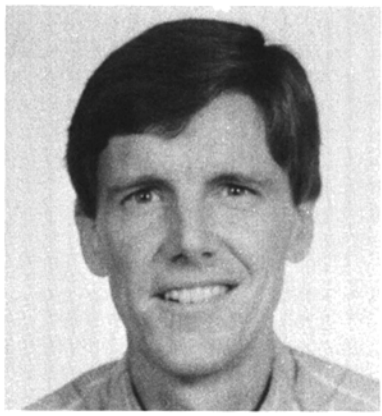
Charles M. Howard

Dr. Howard was professor of plant pathology at the University of Florida, Institute of Food and Agricultural Sciences, Agricultural Research and Education Center, Dover. He received a B.S. degree in agricultural science in 1960 and an M.S. degree in plant pathology in 1962 from West Virginia University and his doctorate from Cornell University in 1969. Dr. Howard began research on etiology and control of strawberry and vegetable diseases at the center in 1967. He initiated a strawberry anthracnose resistance breeding program in 1970 and conducted the program until 1987, when a strawberry breeder was added to the faculty. His major interest was in strawberry diseases, especially strawberry anthracnose.



John L. Maas

Dr. Maas is a research plant pathologist with the USDA-ARS Fruit Laboratory in Beltsville, Maryland. He earned his B.S. degree in botany from Michigan State University in 1962, his M.S. degree in mycology from the University of Washington in 1964, and his Ph.D. degree from Oregon State University in 1968 with epidemiological studies on Botrytis rhizome rot of iris. Since 1968, Dr. Maas has worked on diseases of small fruit crops, especially strawberry and brambles. He has conducted research on pathogen physiology, environmental and host factors that affect disease development, physiological responses of the host to infection, and identification and manipulation of genetic sources of disease resistance for crop improvement. Currently, Dr. Maas is investigating the polyphenol metabolism of strawberry as part of studies on host-pathogen interactions.



Craig K. Chandler

Dr. Chandler is an assistant professor at the University of Florida's Agricultural Research and Education Center in Dover. His duties include development of strawberry cultivars for Florida and study of commercially important traits of strawberry, including resistance to *Colletotrichum*. He received his B.S. degree in 1976 and M.S. degree in 1980 from the University of Florida and his Ph.D. degree in 1983 from the University of Maryland. Before coming to AREC-Dover, he served as a research scientist in the Department of Horticulture at The Ohio State University, Ohio Agricultural Research and Development Center, Wooster.

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