Muscadine Grape Berry Rot Diseases in Mississippi: Disease Identification and Incidence

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ABSTRACT

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Berry rot diseases of muscadine grapes were monitored throughout the 1991 and 1992 growing seasons on four cultivars (Doreen, Sterling, Carlos, and Cowart) at three locations in south Mississippi. The etiology and symptom development of each berry rot disease were studied. Disease incidence data were collected at 2-wk intervals during both growing seasons. Fruit diseases observed on berries included black rot (*Guignardia bidwellii* f. *muscadinii*), bitter rot (*Greeneria uvicola*), russet (unknown etiology), Macrophoma rot (*Botryosphaeria dothidea*), and ripe rot (*Colletotrichum* sp.). Bitter rot was the most important disease in Mississippi, followed closely by black rot. The incidence of Macrophoma rot and ripe rot was low. On leaves, the incidence of black rot was greatest during the middle and late growing seasons. On berries, black rot was most severe as berries approached full size. Cowart and Carlos cultivars were most susceptible to black rot. The incidence of bitter rot on leaves was most severe on young leaves following bud break through the young berry stage. The incidence of bitter rot on berries was severe on small berries, especially those 1 to 3 mm in diameter. The cultivar, Sterling, was most susceptible to bitter rot and russet, but was resistant to black rot. Russet was most severe on full-size berries. *G. uvicola* was always associated with the russet symptom, and it may be a type of resistance expression by some cultivars to early infection or colonization by this pathogen. Both *Colletotrichum acutatum* and *C. gloeosporioides* were identified as causal agents of ripe rot on muscadine grapes in Mississippi.

Additional keywords: *Vitis rotundifolia*

Muscadine grape (*Vitis rotundifolia* Michx.) is native to the southeastern United States and has been cultivated in this area for more than 400 years (12). Muscadine and bunch grapes are of the same genus, *Vitis*, but muscadine is in the subgenus *Muscadinia* and bunch grape is in the subgenus *Euvitis*. Muscadines are generally less susceptible to diseases than are bunch grapes (1). However, the warm, humid weather of the southeastern United States favors development of certain fungi that cause fruit rots, specifically black rot, bitter rot, Macrophoma rot, and ripe rot.

Black rot of muscadine is caused by *Guignardia bidwellii* (Ellis) Viala & Ravaz f. *muscadinii* Luttrell (anamorph *Phyllosticta ampelicida* (Engelm.) van der Aa). This disease is known to cause spots on both leaves and fruit, as well as canker lesions on petioles and young shoots (1,5,8,13,17). On leaves, the spots can spread and collapse, resulting in leaf blight. Small, black, superficial, scabby lesions appear on berries prior to maturity. These lesions do not continue to increase in size or cause mature berries to decay or mummify as they do on bunch grapes. Black rot symptoms also include flower cluster blight (8).

Bitter rot, caused by *Greeneria uvicola* (Berk. & M.A. Curtis) Punithalingam (syn. *Melanconium fuligineum* (Lams.-Scrib. & Viala) Cav.), is the most important berry rot disease of muscadine grape and is also important on bunch grapes (1,10,15,16). Flecks develop on young leaves, shoots, and tendrils, and on individual flower buds and berries. Severe bitter rot infection can cause a blight of young berries and pedicels, causing berries to shrivel and drop. Typical symptoms of bitter rot on mature berries are olive brown lesions covered with black spore masses. These lesions increase in size rapidly and cause soft rot (1,9,10,16). The perfect stage of the fungus has not been found (10,17).

Macrophoma rot, caused by *Botryosphaeria dothidea* (Moug.:Fr.) Ces. & De Not. (syn. *B. ribis* Gross. & Duggar, anamorph *Fusicoccum aesculi* Corda = *Macrophoma* sp.), occurs on both bunch and muscadine grapes but is most severe on muscadines (10,13). Macrophoma rot generally occurs on full-size berries and is most prevalent on ripe berries (1,17). Spots on berries are circular, flat, or slightly sunken, and on susceptible cultivars they can result in a soft rot that covers the entire berry. Abundant pycnidia are produced in the lesions (1,10,13).

Ripe rot has been reported to be caused by the fungus *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc. in Penz. Infection may occur at all stages of fruit development, but the disease remains latent through most of the season (2). Symptoms appear only as berries ripen. Circular brown spots that initially develop on berries can enlarge and cover most of the berry. Rotting fruits are covered with salmon-colored masses of conidia (1,3,13). *C. gloeosporioides* has been reported to rarely cause leaf spots and sunken shoot and stem cankers on grapes in the Philippines (14).

Mississippi now has about 300 ha of muscadines, mostly bronze cultivars. As muscadine production is expanded, berry rot diseases are likely to become a greater problem. Control of berry rots is hindered by limited epidemiological information. The objectives of this portion of the study were (i) to establish or confirm the etiology of each symptom type observed on fruit and vegetative parts from Mississippi vineyards and (ii) to determine the incidence and severity of each disease as the season progressed.

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MATERIALS AND METHODS

Fruit and leaf diseases were monitored on unsprayed vines of four muscadine cultivars (Carlos, Sterling, Doreen, and Cowart) at the Mississippi Agricultural and Forestry Experiment Station (MAFES), South Mississippi Branch, Beaumont, MS, and the USDA Small Fruit Research Station, McNeill, MS, and on three cultivars (Carlos, Sterling, and Doreen) at the MAFES Truck Crops Branch Experiment Station, Crystal Springs, MS. Disease observations and collections were made at 2-week intervals from May through September 1991, and from April through October 1992.

Disease identification. Disease identification was achieved by pathogen isolation and culture followed by confirmatory pathogenicity studies. Disease progress through the season was followed by visual identification and confirmed by frequent pathogen isolation. Diseased tissues were surface-sterilized, placed on potato-dextrose agar (PDA) acidified with 25% lactic acid, and incubated for 1 to 2 weeks at room temperature (~25°C). Fungi were identified by conidial shape and size and by their culture characteristics on PDA. Symptomatic tissue was also incubated in a moist chamber (100% relative humidity) and cross-sectioned for closer examination of the causal agent's characteristics.

Permanent, stained mounts of thin sections were prepared of the typical bitter rot symptoms (flecks) from Sterling and Doreen berries, and of russet symptoms collected from Beaumont. Symptomatic tissues were fixed in FAA, dehydrated in tertiary butyl alcohol, embedded in paraffin followed by paraplast, and stained with Modified Johansen's Quadruple Stain (6).

Inoculum was prepared using conidia of *P. ampelicida* and *G. uvicola* collected from 30-day-old PDA cultures and conidia of *Colletotrichum* sp. collected from 20-day-old PDA cultures. Inoculum consisted of suspensions adjusted to 10^6 conidia per ml or 5-mm mycelium disks of each fungus cut from the edge of 7-day-old cultures.

Half-size and full-size berries (nearly ripe) of Carlos, Sterling, Doreen, and Cowart cultivars were collected from the vineyard at Crystal Springs for artificial inoculation studies. Berries were surface-sterilized and placed on screens in jars with the pedicels of the berries immersed in sterile 2% sucrose. Wounded (needle pricks) and nonwounded berries were inoculated by the respective spore suspensions and mycelium disks and were incubated at room temperature for 7 days.

Five plants of each of the muscadine cultivars Carlos, Sterling and Doreen, and five plants of the bunch grape cultivar Orlando Seedless, propagated by meristem culture procedures (19), were atomized with spore suspensions of each fungal species until runoff. Plants were held in a greenhouse mister for 20 h, moved to the greenhouse, and examined every day after

Fig. 1. Black rot on berries of muscadine grapes: **(A)** rough, black, star-shaped lesions and **(B)** close-up of scabby lesions.

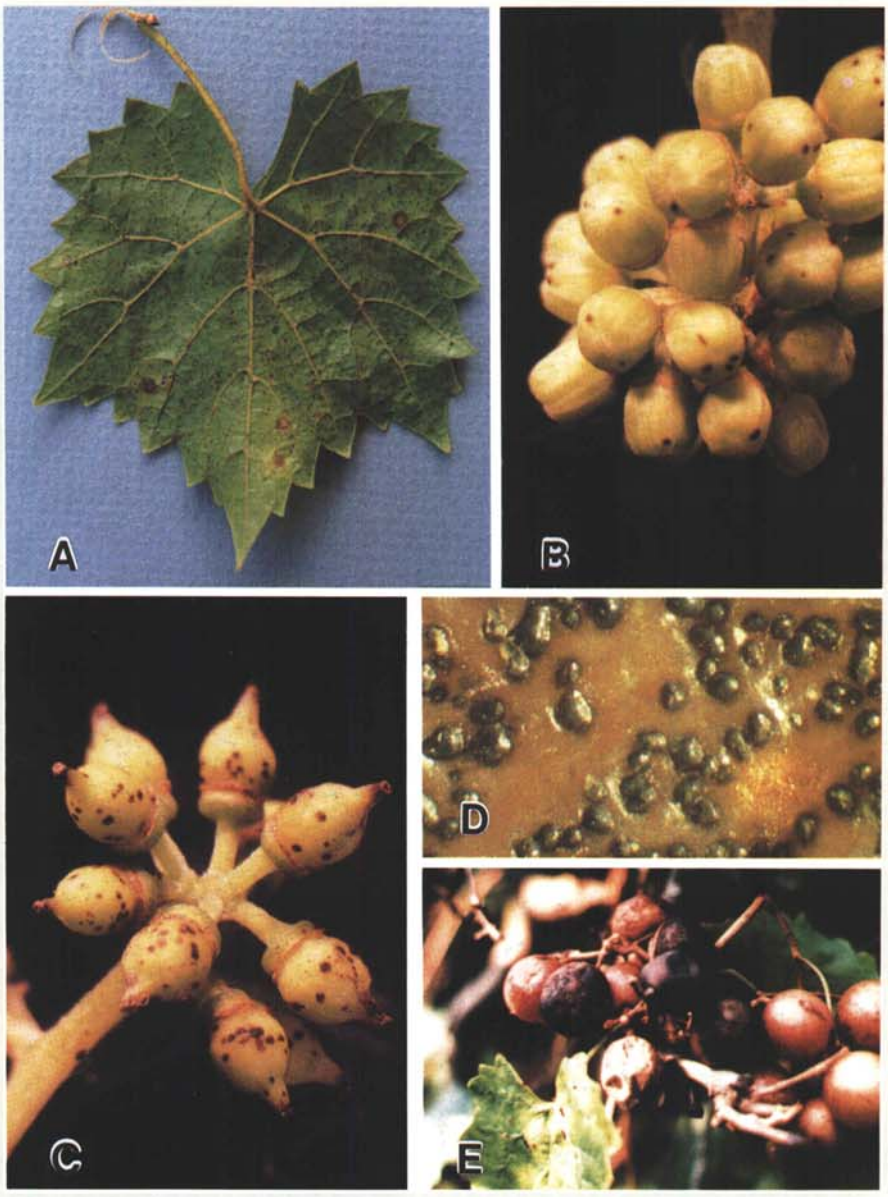

Fig. 2. Bitter rot symptoms on muscadine grapes: **(A)** flecks on leaf and petiole, **(B)** flecks on flower buds, **(C)** flecks on young berries, **(D)** black acervuli on berry, and **(E)** soft rot with black spore masses.

inoculation for symptom appearance. Once symptoms were observed, infected tissues were removed and the pathogen was reisolated. The isolated fungi were then reinoculated on other muscadine and bunch grape plants in the greenhouse to confirm the identity of the causal agent and to complete Koch's postulates.

Disease incidence and severity on leaves and berries. Development of symptoms on naturally inoculated, field-grown vines for each associated disease on vegetative parts and berries was recorded from the beginning of the season through harvest. Disease incidence on leaves was quantitated by collecting three 0.6-m-long shoot terminals from each of two cordons per vine, three vines per cultivar for each location on each observation date. All leaves from each vine sampled were removed from the shoot and intermingled, and 20-leaf random samples were taken. These were graded into four categories for black rot on leaves using a rating scale of 1 to 4 as follows: 1 (light) = 1 to 10 spots; 2 (medium) = 11 to 25 spots or symptom area covering 10 to 25% of the leaf; 3 (severe) = more than 25 spots, or symptom area covering more than 25 to 50% of the leaf; and 4 (very severe) = symptom area covering more than 50% of the leaf. Disease incidence was determined by weighing the counts in each category, adding, and calculating as a percent of the weighted total. Bitter rot on leaves was rated in 1992 by using a rating scale of 1 to 4 as follows: 1 (light) = symptom area 1 to 25% of leaf area; 2 (medium) = symptom area more than 25 to 50%; 3 (severe) = symptom area more than 50 to 75%; 4 (very severe) = symptom area more than 75% or some leaf area turned brown. The sampling method was intended to reflect incidence of new infections on new growth with each successive sampling date.

Disease incidence on berries was quantitated only in the 1992 growing season by collecting six clusters from each of two cordons per vine. Berries were sampled as was done for leaves; the number of diseased berries in a 20-berry sample was recorded for each disease and changed to percentage. The experimental design for disease incidence on leaves and berries was complete randomized design (CRD) with repeated measures with three replications. Replications consisted of three different vines per cultivar per location. Fisher's protected LSD was used to compare treatment means. This is a repeated measure design with cultivars arranged in a CRD at each of three locations. Data were combined over locations for analyses.

RESULTS

Disease identification. Four berry rot diseases, namely black rot, bitter rot, Macrophoma rot, and ripe rot, were observed and identified on muscadines during the 1991 and 1992 growing seasons. In addition, a russet disease (of unknown etiology) of young and developing berries was observed. This disease was associated in this study with early colonization by the bitter rot pathogen, *G. uvicola*, and the fungus was isolated from the russet symptom in all such cases.

Black rot symptoms appeared initially as light brown spots on muscadine leaves. These spots then turned dark, and numerous pycnidia developed on both upper and lower leaf surfaces when they were incubated in a moist chamber at 100% relative humidity. Black rot symptoms also appeared as black necrotic lesions on leaf veins, tendrils, petioles, and succulent branches. Circular or star-shaped scabby lesions were the major symptoms on berries (Fig. 1A). The star-shaped scabby lesions developed from a raised light brown area (Fig. 1B) and were severe on Carlos. Pycnidia were rarely observed on the scabby lesions, and no berry degradation (rot) occurred. Both *P. ampelicida* and *G. uvicola* were isolated from most black rot lesions.

Early bitter rot symptoms appeared as tiny spots (flecks) on young leaves as they emerged from the dormant bud (Fig. 2A). These symptoms also appeared on tendrils, branches, flower buds (Fig. 2B), and small berries (Fig. 2C). On very small berries (1 to 5 mm), tiny spots occurred, which did not enlarge as berries developed. These spots often sloughed off or disappeared as the berries enlarged. Occasionally, flecking was very severe (Fig. 2D), and the skin of entire small berries would turn brown (blight). Most small berries with severe flecking and blight dropped from the vine. When berries were about half-size, typical bitter rot symptoms (soft rot with black spore masses) usually appeared (Fig. 2E). Once berries were covered with black spore masses, they usually dropped to the ground and mummified; however, some mummified berries remained on the vines. Throughout the season, *G. uvicola* could also be isolated from symptomless berries.

As determined by the permanent-stained cross sections, bitter rot fleck symptoms from berries collected in the field produced cell necrosis in the lesion areas. Some of the flecks protruded above the epidermal level. Hyphae were observed in infected cells near the lesion areas. Acervuli were not observed on flecks of berries collected from the field. In the brown lesions covered with black spore masses, hyphae colonized intracellularly in the epidermis and in cells under the epidermis. Acervuli were found in the area under the epidermis. Hyphae grew in the cells under acervuli and were also found in cells deep within the berries.

When berries were half-size, a russet symptom was often observed (Fig. 3). The symptoms are similar to the netlike pattern of scar tissue caused by the powdery mildew pathogen or typical starfish pattern of berry scarring caused by thrips. Brown lines developed initially around the pedicel and spread down the berries. Severe brown scarring occurred over some areas of the berries. This symptom was most prevalent

Fig. 3. Russet, netlike pattern of scar tissue on muscadine grape cv. Sterling berries.

on Sterling, followed closely by Carlos. Only a slight occurrence was observed on Doreen and Cowart. Pathogen isolation procedures revealed that *G. uvicola* was always associated with this symptom. Based on permanent-stained mounts of berries with the russet symptoms, the epidermal cells and three to five layers of cells beneath the epidermis were necrotic. The tissues in areas with the russet symptom broke off easily from the normal tissues. On naturally infected berries, hyphae were not observed in necrotic cells of the russet areas.

Macrophoma rot symptoms were observed on berries beginning about 1 month prior to harvest. Berry symptoms associated with *Macrophoma* sp. on berries included both soft rot and 1- to 2-mm brown spots with dark brown halos. Pycnidia were observed on both lesion types during high moisture periods.

Both *Colletotrichum acutatum* Simmonds ex Simmonds (18) and *C. gloeosporioides* were isolated from berries with typical ripe rot symptoms (rot with salmon-colored spore masses). Symptoms caused by both species were identical. Ripe rot symptoms occurred only on berries during the same period as Macrophoma rot or later. Once the entire berry was rotted, it dropped to the ground. Symptoms were not found on leaves or vegetative parts of muscadine; however, both *Colletotrichum* species were isolated from dark, sunken lesions on pedicels and tendrils of the cultivar Orlando Seedless (B. J. Smith, *unpublished*).

Inoculations of half- or full-size wounded and nonwounded berries with *P. ampelicida* produced no symptoms on any of the cultivars tested. Following inoculation of wounded berries with conidia of *C. acutatum*, typical ripe rot symptoms occurred on only four of 15 half-sized Carlos berries. When inoculated with mycelium, one of 15 full-size nonwounded Carlos berries and four of 15 full-sized wounded Doreen berries developed symptoms. Symptoms were produced on both half-size and full-size berries of all cultivars inoculated by *G. uvicola*. Percent infection was highest when wounded berries were inoculated using mycelium of *G. uvicola*. This resulted in 100% infection of half-size berries of all cultivars, and from 46% infection of full-size berries on Cowart to 100% infection on Sterling. The skin of full-size berries turned brown within 3 or 4 days, and black spore masses covered the browned areas within another 2 days. In some cases, berries detached from pedicels. A low level of infection also occurred on noninoculated controls, indicating some colonization prior to collection and sterilization.

None of the greenhouse plants inoculated with *P. ampelicida* or *C. acutatum* developed symptoms on old leaves or 3-week-old shoots. Disease symptoms were not observed on muscadine plants with mature, fully expanded leaves following inoculation with *G. uvicola*. However, plants with 3-week-old shoots inoculated with *G. uvicola* exhibited symptoms. The first symptoms appeared within 3 days as flecks on leaves. Flecks on petioles and stems, and flecks on tendrils, appeared on days 4 and 5 after inoculation. The symptoms were generally severe on the young plant parts of all cultivars. The flecking was more severe on Sterling and Carlos than on Doreen. Disease development was much more severe on the Orlando Seedless bunch grapes than on muscadine cultivars. Symptoms were also observed on the older leaves of Orlando Seedless, in contrast to the muscadine cultivars, where symptoms occurred only on young leaves.

Disease incidence and severity on leaves and berries. During the 1991 growing season, black rot on muscadine leaves was most prevalent on Carlos and least prevalent on Sterling during the middle and late growing season at Beaumont and Crystal Springs. At McNeill, black rot on leaves was equally prevalent on Doreen, Sterling, and Carlos, and least prevalent on Cowart. The incidences of black rot on berries, bitter rot on leaves, and bitter rot on berries were not recorded in 1991.

The incidence of black rot on leaves during 1992 showed the same pattern as the incidence during the 1991 growing season. There was an interaction among locations, cultivars, and times for black rot on leaves; thus data could not be pooled (Fig. 4). Black rot on leaves was less prevalent on Sterling during the middle and late growing season at Beaumont. At McNeill, there were no differences among cultivars ($P \leq 0.05$) until late in the growing season, when black rot was least prevalent on Cowart. At Crystal Springs, disease incidence was very low on all cultivars.

There was also an interaction among locations, cultivars, and times for black rot on berries for 1992 (Fig. 5). At Beaumont, Sterling was the most resistant cultivar to black rot on berries. At Beaumont and McNeill, the percentage of black rot was highest when berries reached full size. The incidence of black rot on berries was very low throughout the growing season at Crystal Springs.

There was no interaction among locations, cultivars, and times for bitter rot on leaves in 1992. Bitter rot was most prevalent overall at Crystal Springs (Table 1). Sterling was the most susceptible cultivar to bitter rot on leaves. The incidence of disease was highest in early May (18.1%) during the young shoot stage, dropped in late May (9.4%), and increased in late June (18.9%) following the young berry stage.

There was an interaction among locations, cultivars, and times for bitter rot on berries during 1992 (Fig. 6). Bitter rot was most severe on Sterling at Beaumont and Crystal Springs. The disease was most prevalent on all cultivars in June (young berry stage).

There was an interaction among locations, cultivars, and times for russet on

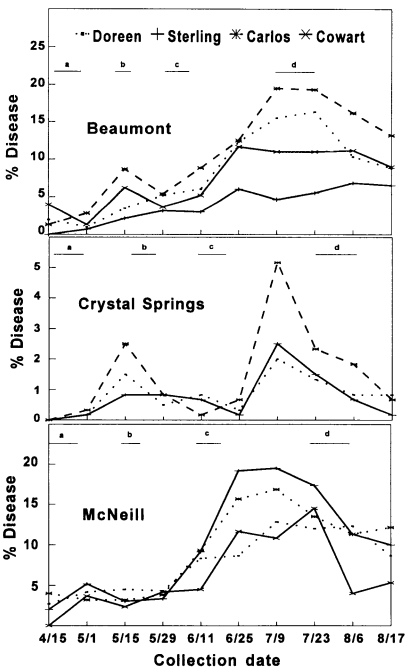

Fig. 4. Incidence of black rot on leaves of four muscadine grape cultivars at three locations in Mississippi during the 1992 growing season. Berry stages are given across the top of the graph: a = shoot, b = bloom, c = berry 1 to 3 mm, and d = berry full size.

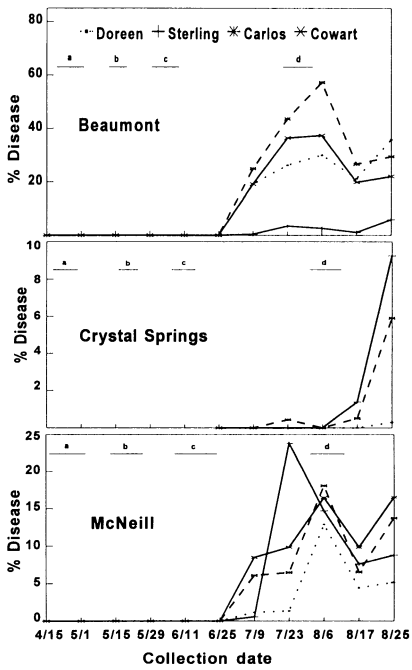

Fig. 5. Incidence of black rot on berries of four muscadine grape cultivars at three locations in Mississippi during the 1992 growing season. Berry stages are given across the top of the graph: a = shoot, b = bloom, c = berry 1 to 3 mm, and d = berry full size.

berries during 1992 at the three locations. Russet was most prevalent on full-size berries. At Beaumont and Crystal Springs, russet symptoms first appeared on Sterling in late July, with 30.0 and 27.6% disease incidence, respectively. The percent russet incidence was highest on Sterling in mid- to late August, with 48.0% at Beaumont and 41.2% at Crystal Springs. No other cultivars showed russet incidence above 10% at either location. At McNeill, russet was more prevalent on Sterling and Carlos, although disease incidence was less than 10% in all samplings.

DISCUSSION

The berry rot diseases observed on muscadines in Mississippi included bitter rot, black rot, russet (unknown etiology), Macrophoma rot, and ripe rot. Bitter rot was the most widespread berry rot disease in Mississippi, followed closely by black rot. Macrophoma rot and ripe rot were minor diseases during both growing seasons.

Table 1. Incidence of bitter rot on leaves of four muscadine grape cultivars at three locations in south Mississippi during the 1992 growing season

	Bitter rot (%)[z]
Location	
Beaumont	10.8 b
McNeill	9.4 b
Crystal Springs	14.5 a
LSD (0.05)	2.4
Cultivar	
Carlos	9.5 b
Sterling	14.5 a
Cowart	9.7 b
Doreen	11.0 b
LSD (0.05)	2.2

[z] Numbers followed by the same letter in a column are not significantly different by LSD at $P \leq 0.05$.

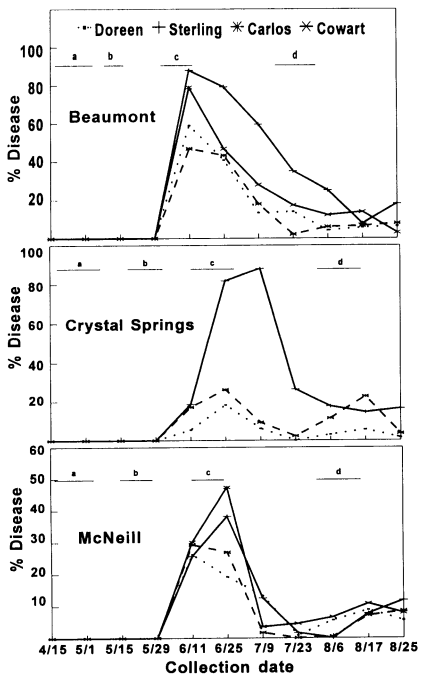

Fig. 6. Incidence of bitter rot on berries of four muscadine grape cultivars at three locations in Mississippi during the 1992 growing season. Berry stages are given across the top of the graph: a = shoot, b = bloom, c = berry 1 to 3 mm, and d = berry full size.

Flecking, associated with *G. uvicola* colonization, was evident on leaves immediately after young shoots emerged from the lateral buds. Bitter rot was most severe on leaves when the shoots were 7 to 15 cm long, indicating that the bitter rot fungus infects only young leaves. Bitter rot infection on berries was most severe at the 1- to 4-mm stage, although bitter rot symptoms had already been observed on some flower buds. The level of bitter rot recorded decreased drastically after the young berry stage as a result of the early berry drop experienced (7) and the sloughing off or disappearance of the flecking symptoms from some berries as they enlarged. The symptoms observed on berries were the same as indicated in previous studies (1,9,16,17). *G. uvicola* was consistently isolated from symptomless leaves and berries, and especially from berries late in the growing season. Our results indicate that colonization by *G. uvicola* without symptom expression was prevalent on leaves and berries during the late part of the growing season.

Infection by *P. ampelicida* (*G. b.* f. *muscadinii*) was first observed on leaves almost at the same time as the earliest bitter rot infection, but black rot infection was slight compared to bitter rot. The incidence of black rot on leaves increased drastically right after midseason. At about the same time, the incidence of black rot on berries began to increase as the berries reached full size. Black rot on leaves seems to serve as the source of inoculum for black rot infection on berries. Isolations from surface-sterilized pycnidia taken from these lesions produced only *P. ampelicida*; however, isolations from the lesions consistently demonstrated the presence of both *P. ampelicida* and *G. uvicola*. Savage (17) mentioned that *G. uvicola* usually gains entrance through black rot lesions.

Macrophoma rot and ripe rot were of minor consequences during the two seasons of our observations in Mississippi. This is contrary to previous observations in North Carolina (1), where both diseases were more severe than bitter rot. The differences in severity of these diseases between North Carolina and Mississippi may be due to environment and cultivar differences. *C. acutatum* was identified as a causal agent of ripe rot on muscadine grapes in Mississippi, along with the previously reported causal agent, *C. gloeosporioides*.

Russet was observed on berries during both growing seasons on some grape cultivars. However, the etiology of russet could not be proven, since this symptom could not be reproduced through inoculation procedures. The symptom we are calling "russet" was prevalent on all cultivars in our studies and was most severe on Sterling, especially at Beaumont. Excessive russet as observed at Beaumont may also be associated with plants of poor vigor. On other fruit, russet is often a symptom of high humidity or rainfall immediately at or around full bloom, frost, damage from harsh chemicals, or improper nutrition (4,11). Some chemicals may cause injury to grape berries similar to russet (13); however, vines in this study were not sprayed with fungicides or insecticides. Berries with russet symptom that were cross-sectioned showed no evidence of the powdery mildew pathogen. Mature berries with the russet symptom were collected from Beaumont in the 1992 growing season, surface-sterilized, and placed in a moist chamber. All of these berries produced typical bitter rot symptoms within 1 month. This did not occur when immature russeted berries were subjected to the same test. In 1993, clusters of berries with flecks were tagged for observation, and later berries within these clusters developed typical russet symptoms. In contrast, berries of tagged clusters with no symptoms did not develop the russet symptom. It is conceivable that this russet symptom is an expression of a type of resistance by some cultivars to early infection or colonization by *G. uvicola*. Some berries with severe fleck symptoms dropped from pedicels, but sometimes small, mummified berries were found on these clusters. Tagged clusters of symptomless berries exhibited no berry drop and no mummification. However, the role of *G. uvicola* in the development of the russet symptom disease is subject to question.

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