Muscadine Grape Berry Rot Diseases in Mississippi: Disease Epidemiology and Crop Reduction

N. Kummuang, Former Graduate Student, **S. V. Diehl**, Assistant Professor, and **B. J. Smith**, Research Plant Pathologist, USDA-ARS, Small Fruit Research Station, Poplarville, MS 39470, and **C. H. Graves, Jr.**, Emeritus Plant Pathologist, Department of Entomology and Plant Pathology, Mississippi State University, Mississippi State 39762

ABSTRACT

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Four berry rot diseases that can cause severe losses of muscadine grapes in Mississippi were monitored throughout the 1991 and 1992 growing seasons on four cultivars (Doreen, Sterling, Carlos, and Cowart) at three locations in south Mississippi. Bitter rot (*Greeneria uvicola*) caused significant drop of young, half-size, full-size, and mature berries and was responsible for most of the fruit drop during the two growing seasons. Cultivars Sterling and Cowart had significantly more berry drop associated with this disease than did Doreen. Black rot (*Guignardia bidwellii* f. *muscadinii*) did not cause significant berry drop. Macrophoma rot (*Botryosphaeria dothidea*), ripe rot (*Colletotrichum* sp.), and russet (unknown etiology) were not generally associated with berry drop of the cultivars in this study. At harvest in 1991, black rot was most severe on Cowart; whereas bitter rot was more severe on Carlos and Sterling cultivars. Incidences of Macrophoma rot and ripe rot were very low both years at all locations. The incidence of berry diseases at harvest was lower in 1992 than in 1991, probably because of the higher amount of rainfall during the early summer of 1991 compared to 1992. Only conidia of *G. uvicola* were abundant in rainwater runoff from the vines. Spore traps showed that the highest conidial peak of *G. uvicola* at Beaumont coincided with the highest peak of bitter rot disease on berries (young stage berries). Only the bitter rot pathogen overwintered in abundance on pedicels, fruit spurs, and mummified berries.

Additional keywords: *Vitis rotundifolia*

Five muscadine berry rot diseases (black rot, bitter rot, Macrophoma rot, ripe rot, and russet [6]) have been identified as important in Mississippi because they can significantly reduce muscadine yields. However, their occurrence and severity in muscadine (*Vitis rotundifolia* Michx.) vineyards has been sporadic due to environmental conditions, location, and cultivar type. Reduction in muscadine yields from berry rot diseases across the southeast has varied from 10 to more than 50% (1,9,10).

Present address of first author: Rachamachala Technology Institute, Bangpra Siracha, Chenburi 20210, Thailand.

Present address of second author: Mississippi Forest Products Laboratory, Mississippi State University, Box 9820, Mississippi State 39762; E-mail: svdiehl@fpl.msstate.edu

Send reprint requests to the third author: USDA-ARS, Small Fruit Research Station, P. O. Box 287, Poplarville, MS 39470; E-mail: bjsmith@ag.gov

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Black rot of muscadine is caused by *Guignardia bidwellii* (Ellis) Viala & Ravaz f. *muscadinii* Luttrell (anamorph *Phyllosticta ampelicida* (Engelm.) van der Aa). Black rot can cause a small amount of defoliation and usually does not result in berry drop. This disease is considered of little importance in reducing yield; however, it may affect fruit quality (1,7,9,13). The pycnidial stage of the black rot fungus overwinters in infected canes; whereas the perithecial stage overwinters on leaves and mummified berries on the vine or ground. Both ascospores and conidia are primary sources of infection of leaves and stems in the spring (1,9,10,13).

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 in Mississippi (6). In North Carolina, this disease alone may account for a 10 to 30% loss of ripening berries (1,12). The name "bitter rot" is derived from the bitter flavor the fungus imparts to infected fruit, producing an unpleasant and unacceptable taste (10,11). The bitter rot fungus overwinters as a saprophyte on leaves, pedicels, and mummified berries on the vine or on the ground (1,9,10). No perfect stage of the fungus has been found (9,13).

Macrophoma rot is caused by *Botryosphaeria dothidea* (Moug.:Fr.) Ces. & De Not. (syn. *B. ribis* Gross. & Duggar, anamorph *Fusicoccum aesculi* Corda = *Macrophoma* sp.). In Mississippi, the disease incidence is low (6), but it has been severe on muscadine grapes in other areas of the southeastern United States (9,10). Twenty to 30% losses of the ripening berries of susceptible cultivars have been reported (10). On susceptible cultivars, a tan to brown soft rot can spread to cover the entire berry. Infected berries drop from the vine, shrivel, and become hollow shells with abundant pycnidia produced over the surface (1,9,10). The fungus overwinters as pycnidia on infected berries and pedicels, and as ascocarps on stems (8,9,10,13).

Ripe rot, caused by *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc. in Penz. and *Colletotrichum acutatum* Simmonds ex Simmonds, is not prevalent in Mississippi (6); however, the disease has been reported to be a major problem on muscadines in North Carolina (3). The characteristic symptoms of ripe rot occur on ripe berries at harvest. Salmon-colored spore masses are produced on the rotting skin of berries (2,3). Daykin and Milholland (3) found that large numbers of conidia were released in the early spring and in the fall, and infections occur at all stages of fruit development. The ripe rot fungus overwinters on infected pedicels, fruit spurs, and mummified fruit on the vine (1,3,10).

Russet symptoms (unknown etiology) were first identified in Mississippi, mainly on susceptible cultivars such as Sterling and Carlos (6). Symptoms include a severe brown scarring over some areas of the berries. *G. uvicola* was always isolated from this type of scarring. It has been suggested that the russet symptom is an expression of a type of resistance by some cultivars to early infection or colonization by *G. uvicola* (6).

Control of muscadine berry rots is currently hindered by limited epidemiological information. Unless these diseases are appropriately controlled, the quality and quantity of the crop are markedly reduced. A full understanding of the etiology and epidemiology of berry rot diseases as they occur in Mississippi is an essential prerequisite for successful control programs. These studies were undertaken to (i) de-

termine the mode of overwintering and period of spore release, (ii) estimate crop reduction due to berry rot diseases, and (iii) examine the relationship of temperature and rainfall to occurrence of berry rot diseases.

MATERIALS AND METHODS

This research was conducted 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, 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. Collections of samples were made at 2-week intervals from May through September 1991, and from April through October 1992. Overwintering samples were also collected at an additional vineyard planted in Noble and Magnolia muscadine cultivars at the Plant Science Research Farm, Mississippi State University, Starkville, MS.

Mode of overwintering. One hundred fruit spurs, pedicels, attached overwintering mummies, and mummified berries on the ground were collected from two cultivars (Noble, Magnolia) at Starkville in March 1992. Ninety-two Carlos mummified berries were also collected from the ground at the same time at Poplarville, MS. The fruit spurs, pedicels, and attached mummies were surface-sterilized and plated on potato-dextrose agar (PDA) acidified with 25% lactic acid. The fungi were subsequently transferred to PDA and identified after 15 days of growth at 25°C.

Pedicels and attached mummified berries on vines, and mummified berries from the ground were collected from Carlos, Doreen, Sterling, and Cowart at Beaumont and McNeill in February 1993. The samples were processed as described above.

Period of spore release. Conidia were collected in rainwater runoff from muscadine vines from June through October 1992. Five 4-liter plastic milk jugs were connected by tubing to plastic funnels to collect rainwater runoff (4,5). The spore traps were positioned beneath the arms of unsprayed Sterling muscadine vines at Beaumont and unsprayed Carlos muscadine vines at Poplarville. Rainwater was collected weekly by exchanging the jugs with new empty ones, and the total water collected from each jug was recorded. Thirty ml of a 5% $CuSO_4$ solution was added to each jug to prevent spore germination (3), and the water was examined for the presence of conidia.

Conidia determinations were done by removing 30 ml of rainwater from each jug, centrifuging the suspension for 20 min at 4 × 10^3 revs/min (5), resuspending the pellet in 1 ml of distilled water using a mixer, and counting the conidia using a hemacytometer. Conidia were identified on the basis of shape and size.

Crop reduction from berry rot diseases. At harvest, a 1.8-kg sample of ripe berries was taken from each vine of each cultivar at each location in 1991, and 3.7-kg samples were taken in 1992. The percentage of each berry rot disease was recorded by counting the number of diseased berries. The data were analyzed by using a completely randomized design (CRD) at each location with repeated measures and three replications. Replications consisted of three different vines per cultivar per location. Fisher's protected LSD was used to compare treatment means. There was an interaction between cultivars and locations; thus each year was analyzed separately.

Berries that dropped prior to harvest in the 1992 growing season were collected in a 0.6 × 0.6 m basket hung from two cordons of a single vine per cultivar at each location for evaluation of crop reduction. The percentage of berry drop for each disease was recorded by counting diseased berries. The data were analyzed as above.

Relationship of temperature and rainfall to occurrence of berry rot diseases. Temperature and rainfall were recorded during the 1991 and 1992 growing season at Beaumont, McNeill, and Crystal Springs. Multiple regression analyses were used to evaluate the relationships of temperature and rainfall to disease occurrence. The disease incidence data used were reported on in a previous publication (6). The incidence data for each disease were transformed for regression analyses to obtain a best model. Prediction models of disease occurrence were developed and analyzed using the SAS Max-R^2 variable selection procedure of the SAS Statistics (SAS Institute, Cary, NC).

RESULTS

Mode of overwintering. *Macrophoma* sp. was found in only two of 60 pedicel samples from Carlos collected at Beaumont and two of 60 pedicels from Sterling collected at McNeill. *P. ampelicida* and *Colletotrichum* sp. were not found in any of the samples. Only *G. uvicola* occurred in abundance. *G. uvicola* infection occurred on 62% of the fruit spurs, 56% of the pedicels, and 48% of mummified berries from the vines collected at Mississippi State University at Starkville in 1992. *G. uvicola* was isolated from 90 and 65% of Carlos pedicels, 68 and 53% of Doreen pedicels, 72 and 78% of Sterling pedicels, and 75 and 63% of Cowart pedicels collected from Beaumont and McNeill, respectively. Ninety-five percent of the berries collected from the ground were contaminated, probably by soil saprophytes.

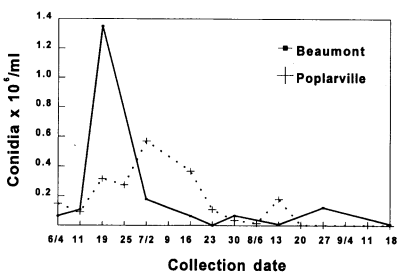

Fig. 1. Spore dispersal patterns of *Greeneria uvicola* in traps placed under muscadine grape vines at Beaumont (cv. Sterling) and at Poplarville (cv. Carlos) during the 1992 growing season.

Period of spore release. Both *G. uvicola* and *P. ampelicida* conidia were found in the water collected from the spore traps. The number of *P. ampelicida* conidia was always small and of infrequent occurrence. At Beaumont, the number of *G. uvicola* spores trapped under Sterling was highest on 19 June (Fig. 1), which is 1 week prior to the period in which bitter rot was most prevalent on leaves. At Poplarville, the highest number of spores trapped under Carlos was half of what was trapped at Beaumont (Fig. 1). Other fungal contaminants, such as *Helminthosporium* and *Fusarium* spp., were also present in samples of both locations.

Crop reduction from berry rot diseases. During 1991, black rot at harvest was most prevalent at McNeill, and Cowart was the most susceptible of the four cultivars (Table 1). The disease incidences for Macrophoma rot were very low (Table 1). When it did occur, Macrophoma rot was most prevalent at Crystal Springs and on Doreen and Sterling. Ripe rot incidences were also very low (<2.8%) at all locations, and there were no significant differences among locations or cultivars (data not shown). There was an interaction between locations and cultivars for bitter rot. Bitter rot was more severe on Carlos, Sterling, and Cowart than on Doreen at Beaumont (Table 2). The disease was most prevalent on Carlos at McNeill. Bitter rot was more prevalent on Sterling and Carlos than on Doreen at Crystal Springs.

Table 1. Incidence of black rot and Macrophoma rot on muscadine grape berries among locations and cultivars at harvest in 1991

	Black rot (%)[z]	Macrophoma rot (%)[z]
Location		
Beaumont	2.9 b	0.5 b
McNeill	7.5 a	1.0 b
Crystal Springs	0.6 c	2.6 a
LSD (0.05)	1.7	0.9
Cultivar		
Carlos	2.6 b	0.8 b
Sterling	2.1 b	1.6 a
Cowart	11.5 a	0.2 b
Doreen	1.3 b	2.1 a
LSD (0.05)	4.4	0.8

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

During 1992, there was an interaction between locations and cultivars for black rot. At Beaumont, black rot was least prevalent on Sterling, but there were no significant differences among cultivars at McNeill or Crystal Springs (Table 3). There was an interaction between locations and cultivars for both Macrophoma rot and ripe rot, but there were no significant differences among locations or cultivars. The incidence of Macrophoma rot was less than 2%. The incidence of ripe rot was less than 1% (data not shown). There was no interaction between locations and cultivars for bitter rot. The percentage of bitter rot was very low (1 to 3%), and there were no significant differences among cultivars (data not shown). There was an interaction between locations and cultivars for russet. Sterling was the most susceptible to russet at Beaumont and Crystal Springs; whereas Carlos was the most susceptible to the disease at McNeill (Table 3).

Early berry drop due to black rot was very low, with no interaction between locations and cultivars. There were no significant differences among cultivars; however, there were significant differences among all three locations, with the highest incidence occurring at Beaumont (0.63%), followed by McNeill (0.19%), and the lowest incidence at Crystal Springs (0.03%). For bitter rot, there were significant differences among locations, with the highest incidence of berry drop due to bitter rot occurring at Beaumont and the lowest incidence at McNeill (Table 4). Among cultivars, Sterling and Cowart had significantly more berry drop associated with this disease than did Doreen (Table 4). There were no interactions between locations or cultivars in the incidence of berry drop associated with russet, Macrophoma rot, or ripe rot. Berry drop associated with these three diseases was very low, <1.9% for russet and <0.1% for Macrophoma rot and ripe rot.

Relationship of temperature and rainfall to occurrence of berry rot diseases. Total rainfall for the sample collection periods in 1991 in all three vineyards were 51.3, 43.6, and 25.9 cm for June, July, and August, respectively, and in 1992 were 42.8, 41.8, and 64.9 cm (including 18.1 cm collected in a single day at Crystal Springs). Temperature and rainfall had an effect on incidence of black rot and bitter rot; however, the relationships were not linear.

The best general model of black rot on leaves during the 1991 growing season was $\text{Dis}(y) = b_0 + b_1(\text{logtemp}) + b_2(\text{lograin})$, at all locations except Beaumont. The best model of black rot on leaves during the 1992 growing season was $\text{Dis}(y) = b_0 + b_1(\text{logtemp})$, except for the Cowart cultivar at Beaumont and all cultivars at Crystal Springs, where the best model was $\text{Dis}(y) = b_0 + b_1(\text{logtemp}) + b_2(\text{lograin})$. The best general model of black rot on berries varied with locations and cultivars; however, the R^2 were low (0.07 to 0.41) and not significant ($P \leq 0.05$) for most situations.

The best models of bitter rot on leaves varied with locations and cultivars, and the R^2 values were low (0.04 to 0.65) and not significant for most situations. The best model for bitter rot on berries was $\text{Dis}(y) = b_0 + b_1(\text{logtemp})$, with the exception of the disease on Sterling at Crystal Springs, where the best model was $\text{Dis}(y) = b_0 + b_1(\text{logtemp}) + b_2(\text{lograin})$.

DISCUSSION

Isolations from mummified muscadine berries on vines, pedicels, and fruit spurs during the 1991 and 1992 overwintering periods indicate that these are possible primary overwintering sites for *G. uvicola*. Savage (13) also found that *G. uvicola* overwinters on mummified berries and pedicels of berry clusters left hanging on vines. *G. uvicola* was found in most samples, but *P. ampelicida* was not. There may be some competition in growth rate or nutrient consumption between these fungi, with *G. uvicola* being a better competitor. These results coincided with results of the isolations from black rot symptoms, where *G. uvicola* was often found instead of *P. ampelicida* (6). During the 1991 and 1992 overwintering periods, *Macrophoma* and *Colletotrichum* spp. were rarely found in the samples, perhaps because of the low disease incidences observed during the growing season (6).

Abundant conidia of *G. uvicola* were trapped in rainwater runoff from the vines. From the pattern of spore dispersion of *G. uvicola* at Beaumont, the highest conidial peak was at the same time as the highest peak of bitter rot disease on berries (young stage berries) (6). It seems that the use of spore traps was begun at least 2 months too late in the growing season in this study to provide an adequate understanding of spore dispersion phenomena throughout the season. Conidia of *P. ampelicida* were trapped infrequently and in small quantities. This may be related to the apparent greater resistance exhibited by the cultivar employed, namely Sterling at the Beaumont station.

Bitter rot caused significant drop of young, half-size, full-size, and mature berries and was responsible for most of the berry drop during the two growing seasons. We were not able to count the drop of very small berries (1 to 2 mm) due to bitter rot because they dried out rapidly. As a result of the early berry drop, the level of bitter rot recorded decreased drastically after the young berry stage. Con-

Table 2. Incidence of bitter rot on muscadine grape berries at three locations in Mississippi at harvest in 1991

	Bitter rot (%)[y]		
Cultivar	**Beaumont**	**McNeill**	**Crystal Springs**
Carlos	13.1 a	5.2 a	4.0 ab
Sterling	14.9 a	1.2 c	5.7 a
Cowart	11.0 a	3.1 b	–[z]
Doreen	0.8 b	1.0 c	2.4 b
LSD (0.05)	7.5	1.4	3.1

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

Table 3. Incidence of black rot and russet on muscadine grape berries at three locations in Mississippi (Beaumont, McNeill, and Crystal Springs) at harvest in 1992

	Black rot (%)[y]			Russet (%)[y]		
Cultivar	**Beaumont**	**McNeill**	**Crystal Springs**	**Beaumont**	**McNeill**	**Crystal Springs**
Carlos	27.5 a	11.9 a	1.6 a	3.7 b	15.2 a	2.6 b
Sterling	6.3 b	12.1 a	1.3 a	29.2 a	7.4 b	19.9 a
Cowart	21.7 a	17.3 a	–[z]	5.6 b	2.3 b	–[z]
Doreen	19.5 a	10.2 a	0.4 a	5.0 b	6.5 b	2.5 b
LSD (0.05)	8.1	7.8	1.4	6.5	7.1	9.9

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

Table 4. Incidence of early berry drop due to bitter rot on muscadine grape berries among locations and cultivars in 1992

	Bitter rot (%)[z]
Location	
Beaumont	9.5 a
McNeill	3.0 b
Crystal Springs	7.0 a
LSD (0.05)	3.5
Cultivar	
Carlos	5.0 ab
Sterling	9.7 a
Cowart	8.4 a
Doreen	3.4 b
LSD (0.05)	4.8

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

trary to previous reports (1,9), black rot did not cause significant berry drop in our study. Neither Macrophoma rot nor ripe rot was generally associated with berry drop of the cultivars in this study. However, berry drop as a result of Macrophoma rot was observed in abundance just prior to harvest on vines of an undetermined cultivar located in the Poplarville vineyard.

The incidence of bitter rot disease at harvest was much lower in 1992 than in 1991. This was probably related to the higher amount of rainfall during the early summer of 1991 compared to 1992. When environmental conditions were not favorable for disease development, the incidence of bitter rot was low on all cultivars. The poor growth of the bitter rot pathogen when conditions are unfavorable may be related to the capacity of the muscadine plant to react to the bitter rot pathogen and cause russet instead of typical bitter rot symptoms.

Black rot on leaves responded to the greater rainfall in 1991 than in 1992 in much the same manner as bitter rot on leaves. This is accounted for in the best general model of black rot on leaves in both years ($\text{Dis}(y) = b_0 + b_1(\text{logtemp}) + b_2(\text{lograin})$). However, rainfall was not important in the model for bitter rot on berries where most often the best model was $\text{Dis}(y) = b_0 + b_1(\text{logtemp})$. One reason why rainfall may have less effect than temperature is that muscadine berries tend to grow under the leaf canopy, where small amounts of rainfall do not contact the berries. At Crystal Springs, there was a large amount of rainfall before harvest in 1992 (over 18 cm in a single day), and these rains had an obvious effect on bitter rot on berries, especially on the most susceptible cultivar, Sterling. The best model for Sterling at Crystal Springs was $\text{Dis}(y) = b_0 + b_1(\text{logtemp}) + b_2(\text{lograin})$. Overall, the best general model of bitter rot on berries was $\text{Dis}(y) = b_0 + b_1(\text{logtemp}) + b_2(\text{lograin})$, which would cover most environmental situations, especially heavy rainfall in some years.

The relationship of temperature and rainfall to the incidences of berry rot diseases gives a low R^2 value for most models, especially for black rot on berries and bitter rot on leaves. This means that temperature and rainfall do not manipulate disease incidence independently. Several other factors influence disease incidence, such as timing of weather events, plant age, plant cultivar, size of canopies, amount of inoculum, differences in pathogen race, and pathogen virulence. Other factors that influence disease incidence should be included in a predictive model. Information from this study on the best general models for rainfall and temperature may be useful for developing a model incorporating weather pattern changes in each year. These models can be used to better time fungicide applications and probably reduce the number of applications in most years.

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