

Muscadine Grapes: Some Important Diseases and Their Control

Grape growing (viticulture) is truly an international vocation, and for many an avocation, throughout Europe, the United States, and countries of the Southern Hemisphere. Viticulture and wine making go back more than 6,000 years. Today grapes are grown in 39 countries and are the most economically important of all fruit crops.

The genus *Vitis*, which includes all grapes, is divided into two groups or subgenera known as *Euvitis* and *Muscadinia* (2,15). The larger number of species is included in the *Euvitis* group, commonly known as "bunch grapes." Cultivars in the *Euvitis* group have bark that peels off in loose fragments, forked tendrils, nodes with diaphragms across the pith, and berries that, when mature, adhere to the stem. The bunch or true grape is further divided into the *Vinifera*, which is grown in California and Europe, and the American *Euvitis*, which is represented by many species, including *V. labrusca* L. and *V. rupestris* Scheele. Cultivars in the *Muscadinia* group have simple tendrils, nodes that lack diaphragms across the pith, and mature berries that detach and fall to the ground singly (18). Of the many species of the bunch grape, only two *Muscadinia* species are found in the United States: *V. rotundifolia* Michx. and *V. munsoniana* Simpson. *V. munsoniana* is indigenous to southern Georgia and Florida and is of no importance commercially.

Muscadine grapes are native to the southeastern United States and are

grown on the east coast from Virginia to Florida, the Gulf states, Arkansas, Missouri, and into central Texas. Cultivars of *V. rotundifolia* are often called muscadines, a name derived from the musky flavor of the grapes. Other common names include southern fox grapes (as opposed to the northern fox or labrusca type), bullace or bullis, big white grape, rotundifolia grape, and scuppernong (18). The scuppernong is America's oldest native grape and is also the name of one of the better known cultivars. The name is derived from the Algonquin Indian word *ascuponong*, meaning "at the place of the sweetbay tree" (*Magnolia virginiana* L. = *M. glauca* L.).

History

The earliest written account of the muscadine grape appears in a Florentine navigator's logbook. While exploring the Cape Fear River valley of North Carolina for France in 1524, Giovanni da Verrazzano wrote that he saw "many vines growing naturally there [that] would yield excellent wines." Captains Phillip Amadas and Arthur Barlowe, Sir Walter Raleigh's explorers, wrote in 1584 that the coast of North Carolina was "so full of grapes as the very beating and urge of the sea overflowed them . . . in all the world, the like abundance is not to be found" (9).

Muscadines were used in wine by the country's first settlers. Sir John Hawkins, when relieving the French at Fort Caroline in 1565, found 20 hogsheads of muscadine wine. Even Thomas Jefferson was a devotee of the muscadine grape and the rich amber wine it produced. In 1810, when James Blount took a census of Washington County, North Carolina, he reported that 1,368 gallons of wine were made in the town of Scuppernong. A year later, an article in the Raleigh *Star* commented about Blount's census

and referred to the native white grape as the "Scuppernong grape." The 18th century maps of Washington County show a village, a lake, and a river named Scuppernong (9). In the late 1850s, most of the scuppernong wine in North Carolina was, as stated by John Le Conte, "spoiled by the infusion of whiskey, cider, spirits, or peach brandy, and after fermentation honey is often added," the result being "a mixture of wine and half-fermented mead." However, 2 years later, samples of wine from the most promising grapes then available were sent to the noted Charles T. Jackson of Boston (who introduced surgical anesthesia to the world) for chemical analysis. Despite, or perhaps because of, the fact that the sample of scuppernong wine had a whiff of whiskey, Jackson announced that the scuppernong wine was the best thus far produced in the United States. With proper attention and care, scuppernong wine may be so fine as to excel all other wines made on this continent (9).

One of North Carolina's most famous and prosperous winemakers was Paul Garrett. Garrett made scuppernong the most popular wine in the United States prior to prohibition under the name "Virginia Dare." Over 1 million cases of this wine were sold each year. To increase the limited quantity of the popular scuppernong wine, he blended in Concord and California wines, while maintaining the scuppernong taste.

The muscadine grape industry was brought to a conclusion in 1919 with the adoption of national prohibition and was not renewed until the 1960s (9). Since then, it has undergone dramatic changes. Production and area under cultivation expanded rapidly from 1960 to 1980 in response to favorable prices offered by wineries. The estimated area of muscadine grapes in eight southeastern states in 1966 was about 450 ha, with most plants confined to home gardens. By

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1978 the area had increased substantially, with more than 920 ha planted in North Carolina alone. Production and area under cultivation have declined in North Carolina over the past 10 years but have increased in other states. In Mississippi, for example, 40 ha were cultivated in 1985 and 400 ha in 1990.

Production of muscadine grapes has not reached its full economic potential. Problems include lack of concentrated areas of production, uneven ripening of grapes, large capital investments over a 3-year period, intensive labor for vineyard maintenance, and a general lack of consumer familiarity. However, there still is a strong interest among growers and processors in establishing a muscadine industry throughout the South. In addition to wine, the processing of jam, jelly, and juice represents another potential area of expansion for this unusual grape with the unique flavor. The development of new products and of solutions to handling and market problems will be essential.

Culture

Muscadine grapes are enjoyed primarily as fresh fruit in the areas where they are grown. Cultivars with white (bronze) fruit (Fig. 1) and black fruit are available (1,18). The predominant bronze cultivars are Carlos, Magnolia, Scuppernong, Fry, Higgins, Sterling, and Doreen. The main black grapes are Noble, Hunt, and

Tarheel. Some cultivars have self-fertile flowers, and others have self-sterile pistillate flowers. Self-fertile cultivars do not need a pollinizer, but cultivars with pistillate flowers require pollinizers and should be planted with a self-fertile pollen-producing cultivar.

Muscadine grapevines are not cold-hardy and are limited to areas where the temperature rarely falls below -8°C (1). Commercial vineyards are planted on loam or sandy loam soil; soils that have poor drainage should be avoided. Muscadine grapes are normally grown on a vertical one-wire trellis or a Geneva double-curtain trellis system. Young vines properly trained will bear a crop the third year after planting. Harvesting begins in September and continues through October, depending on the cultivar and the location. Fruit is either harvested mechanically for processing (Fig. 2) or handpicked for fresh market. Some growers allow the consumer to "pick-your-own."

Diseases

Muscadine grapes are inherently more vigorous and generally less affected by diseases than bunch grapes (3). Most pathogens are native and occur on wild as well as cultivated varieties. Loss from diseases varies from year to year with cultivar susceptibility and type of cultural practices. The warm, wet weather of the Southeast favors development of

several organisms that cause fruit rot. Nematodes are very common and can be damaging on bunch grapes but are of no importance on muscadine grapes. Likewise, viruses have not been observed or reported to be a problem for commercial muscadine vineyards. Diseases of muscadine grapes (Table 1) and their control are a major concern of commercial growers and home gardeners alike.

Ripe rot. This fruit rot is considered to be one of the most important disease of muscadine grapes, in some years reducing crops 50% or more. The warm, humid weather of the southeastern United States is ideal for development of this disease (3). The causal fungus, *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc. (teleomorph *Glomerella cingulata* (Stonem.) Spauld. & Schrenk), also causes rot of other fruit and vegetables. Affected berries develop reddish brown spots that continue to enlarge to cover the entire berry. Salmon-colored masses of spores develop as the grapes ripen and mature (Fig. 3). Rotting fruit may remain attached to the vine or drop to the ground. The fungus overwinters in mummified fruit and infected pedicels on the vine (Fig. 4), and spores are released and spread during rainy periods in spring (6). Fruit are susceptible to infection at all stages of development but do not show symptoms until ripening. Frequent rains during the ripening period often result in severe losses.

Macrophoma rot. Bunch and muscadine grapes are affected by the disease frequently described as "bot rot." *Botryosphaeria dothidea* (Moug. ex Fr.) Ces. & de Not (syn. *B. ribis* Grossenbacher & Duggar, anamorph *Fusicoccum aesculi* Corda = *Macrophoma* sp.), the cause of Macrophoma rot, has become important on muscadine grapes in North Carolina and other regions in the Southeast (3,15). Circular, flat, or slightly sunken lesions, 1-4 mm in diameter, develop on infected berries. Later, tan to brown soft rot spreads over the fruit of susceptible cultivars (Fig. 5). Infected berries drop from the vine, shrivel, and eventually become hollow shells with abundant fruiting structures (pycnidia) scattered over the surface. Spores are



Fig. 1. Muscadine grape cultivar Carlos.



Fig. 2. Mechanically harvested muscadine grapes to be sold for wine.

Table 1. Causes and relative severity of diseases on muscadine and bunch grapes

| Disease | Causal organism | Relative severity* on: | |
|-------------------|--|------------------------|-------|
| | | Muscadine | Bunch |
| Ripe rot | <i>Colletotrichum gloeosporioides</i> | +++++ | + |
| Macrophoma rot | <i>Botryosphaeria dothidea</i> | ++++ | + |
| Powdery mildew | <i>Uncinula necator</i> | ++++ | ++++ |
| Bitter rot | <i>Greeneria uvicola</i> | +++ | +++ |
| Black rot | <i>Guignardia bidwellii</i> f. <i>muscadinii</i> | +++ | - |
| | <i>G. b. f. euvitis</i> | - | ++++ |
| Angular leaf spot | <i>Mycosphaerella angulata</i> | ++ | - |
| Rust | <i>Physopella ampelopsidis</i> | + | + |
| Pierce's disease | <i>Xylella fastidiosa</i> | + | +++ |
| Anthraco-nose | <i>Elsinoë ampelina</i> | - | +++ |
| Downy mildew | <i>Plasmopara viticola</i> | - | ++++ |
| Eutypa dieback | <i>Eutypa lata</i> | - | +++ |

*+++++ = Most severe, + = least severe, - = unimportant.



Fig. 3. Ripe rot of muscadine grapes caused by *Colletotrichum gloeosporioides*, showing salmon-colored spore masses on rotting fruit.

released during wet weather and are disseminated during the growing season by wind and splashing rain.

Powdery mildew. The importance of this disease was recognized on bunch grapes in the mid-1800s, but it was not until the 1960s that powdery mildew was observed on muscadine grapes (3,15). The causal fungus, *Uncinula necator* (Schw.) Burr. (anamorph *Oidium tuckeri* Berk.), produces whitish mycelial growth on the surface of young grapes just after bloom (Fig. 6A); only young tissues are infected. Affected berries become russeted and often crack (Fig. 6B). Lesions can occur on leaves in late fall but cause very little damage (Fig. 6C). The pathogen is able to grow and multiply only on or in living tissues. Cleistothecia are produced on the leaves during the latter part of the growing season. The fungus overwinters as hyphae inside dormant vegetative buds or as cleistothecia. It grows in temperatures ranging from 5 to 30 C (40–86 F), but the optimum for infection and disease development is 20–27 C (68–80 F). Rainfall may harm the development of mildew by removing the spores and disrupting mycelium.

Bitter rot. The fungus that causes bitter rot imparts a bitter flavor to wine, making it unpleasant and unacceptable. Bitter rot occurs occasionally on bunch grapes but is most important on muscadines. *Greeneria uvicola* (Berk. & Curt.) Punithalingam (syn. *Melanconium fuligineum* (Scribner & Viala) Cav.), the cause of bitter rot, is widely distributed (3,13,16). The fungus infects all above-ground vegetative parts of the grape plant. Leaf infections occur as brown flecks with pale yellowish halos. Lesions that develop on petioles, tendrils, and stems are round to elliptic, black, slightly raised, and up to 1.5 mm long. Symptoms first develop on the young green berries as brown lesions, and the infected berry becomes covered with black fruiting bodies (Fig. 7). The infected fruit shrivels and becomes a dry, black mummy. The fungus overwinters on infected petioles and mummies, which remain on the vine or fall to the ground. Infection occurs in early spring and throughout the growing season during rainy periods when temperatures are between 12 and 30 C (55–85 F).

Black rot. Black rot is extremely important on bunch grapes in the eastern United States. It is very common on muscadine grapes but does not cause serious damage. A distinct form (race) of the fungus, *Guignardia bidwellii* (Ellis) Viala & Ravaz f. *muscadinii* Luttrell, is pathogenic to muscadines (3,12,15). It differs in pathogenicity from *G. bidwellii* on American bunch grapes and has different cultural and morphological features. Symptoms appear on new leaves, stems, and tendrils early in the season. Leaf spots appear as water-soaked areas that gradually turn from tan to reddish

brown (Fig. 8A). Fruiting structures (pycnidia) that develop in the necrotic areas produce the spores for later infections. Small, black, scabby lesions develop on petioles, young stems, and fruit (Fig. 8B). On susceptible cultivars, the scabs may coalesce and cover a large part of the surface of the berry, and eventually the skin splits. Infection of leaves and stems in the spring results from conidia produced in overwintered leaves and from cankers on stems infected the previous year. Spores are disseminated by wind or splashing rain to new growth, where they germinate and infect either through stomata or directly through the epidermis. Infection commences on immature berries from the time they are set until they reach full size. Lesions do not spread or cause decay of mature berries, as with bunch grapes.

Angular leaf spot. This important leaf disease causes premature defoliation, which results in reduced plant vigor and yield. The causal organism is *Mycosphaerella angulata* Jenkins (anamorph *Cercospora brachypus* Ell. & Ev.) (11). Symptoms first appear as small chlorotic spots on the upper leaf surface (Fig. 9). Small, dark brown to black areas in the center of developing lesions are surrounded by distinct halos. Lesions become angular to irregular in shape and vary in diameter from 1 cm to several centimeters. Invasion through either leaf surface is by direct penetration or by entry through stomata. Conidia and ascospores produced on the lower leaf surface are disseminated by wind to other leaves during the spring.

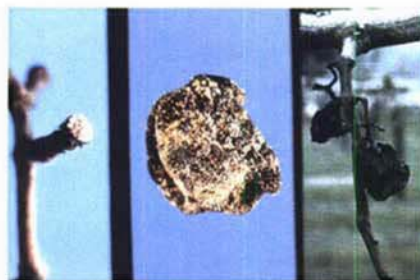


Fig. 4. In early spring, the ripe rot fungus produces spore masses on (left) pedicel and (center) fruit of (right) overwintered mummified fruit.

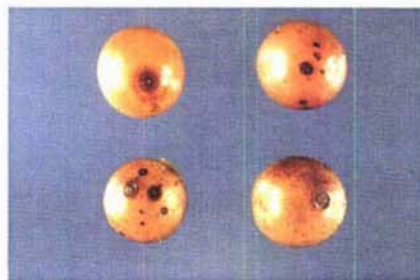


Fig. 5. Various stages of *Macrophoma* rot on muscadine grapes caused by *Botryosphaeria dothidea*.

Pierce's disease. Pierce's disease has been a serious threat to grapevines and a challenge to the scientific community since its discovery by Newton B. Pierce in 1892. It limits bunch grapes in the grape-producing areas of the Southeast and in California where it was first described, has been reported in Mississippi and Texas, and probably occurs in most areas of Central and South America. The pathogen was finally isolated and identified, and Koch's postulates were completed in 1978 (5). The pathogen, *Xylella fastidiosa* Wells et al, is a gram-negative, xylem-limited bacterium measuring 0.25–0.50 μm in diameter \times 1.0–4.0 μm in length (17). Bunch grapevines infected with the bacterium show necrosis at the leaf margins, decline in vigor and yield, and eventually die. In contrast, muscadine grapevines are naturally resistant or tolerant to Pierce's disease, although cultivars vary in degrees of tolerance or resistance. For example, infected grapevines of the cultivar Carlos may show marginal leaf burn (Fig. 10) but do not die and do not show symptoms each year (14).

Symptom expression in response to infection by *X. fastidiosa* is influenced by time of bacterial accumulation and

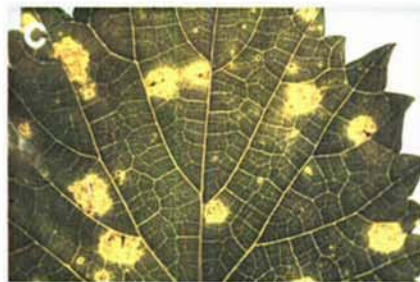


Fig. 6. Powdery mildew caused by *Uncinula necator* on the muscadine grape cultivar Carlos: (A) White mycelium on green berries, (B) russeting of fruit, and (C) leaf spots.

threshold population (7,10). The fastest increase in populations of the pathogen in naturally infected petioles, leaf veins, and stems occurs some 8-10 weeks later in muscadine vines than in bunch grapevines. The delay in symptom development in muscadine vines may be due to the delay in accumulation of the bacterium. Bacterial populations of 1×10^6 colony-forming units per centimeter of leaf vein have been correlated with symptom expression in infected grapevines. Structural barriers, including bacterial aggregates, gums, tyloses, and pectins, and subsequent water stress have been proposed as the primary factors involved in symptom expression.

X. fastidiosa has a wide host range in both monocots and dicots. Sharpshooter leafhoppers (Cicadellidae) and spittlebugs (Cercopidae) serve as vectors of *X. fastidiosa* (15). The insects acquire the bacteria from the water-conducting elements (xylem) when feeding on infected grapevines. Recent studies indicate that perhaps movement of *X. fastidiosa* is restricted in infected resis-

tant cultivars of muscadine grapevines. This localization response prevents systemic movement of the bacteria upward in the plant. Perhaps the localization response acts together with the rapid aging (morphological change of the bacterium from rippled to smooth cell walls) of *X. fastidiosa* in muscadine grapevines to provide resistance (8).

Rust. Grape rust is of minor importance in the United States but has been found on muscadine plants in North and South Carolina and Florida (4). It can be a very serious problem in Asia and Central America if not controlled. The disease is caused by the fungus *Physopella ampelopsidis* (Diet. & Syd.) Cumm. & Ramachar (syn. *Physopella vitis* Arth.). It is a macrocyclic rust that produces pycnidia and aecia on *Meliosma myriantha*, a deciduous tree in Japan (15). Only uredia were found on muscadine grapevines in North Carolina when the fungus was identified in 1967. Infections occur on both upper and lower surfaces of leaves. Small, orange-colored uredial pustules develop on the lower

surface (Fig. 11), and dark necrotic spots develop on the upper surface. Severely affected leaves become yellow and defoliate prematurely.

Control Strategies

The major diseases affecting muscadine grape are caused by fruit-rotting fungi. Control measures include the use of fungicides, sanitation, and cultural practices. Although the use of resistant cultivars would be highly beneficial, many vineyards in production today consist of older cultivars (Magnolia, Carlos, Scuppernong) that are highly susceptible to the major diseases. Many of the vineyards planted in the 1970s and 1980s might still be in production in the year 2000.

A successful grape grower needs a carefully planned strategy for applying fungicides. The effectiveness of disease control is influenced by the fungicides used and by the method and timing of fungicide application. Most fungicides and spray schedules recommended for controlling muscadine grape diseases for the past 20 years originated from the experiments conducted by C. N. Clayton from 1966 to 1974 (3). Angular leaf spot, black rot, bitter rot, and powdery mildew are easily controlled with the fungicides that are currently registered for use on muscadine grapes, but control of ripe rot is much more difficult. Losses from this disease can amount to 50% or more when rainy periods persist during ripening. At present, no registered fungicides are effective for controlling this disease. Since chemical formulations and labeling undergo frequent changes, no specific suggestions for fungicides are included here. Because recommended control practices vary from one region to another,



Fig. 7. Bitter rot on muscadine grapes caused by *Greenaria uvicola*, with numerous pycnidia.

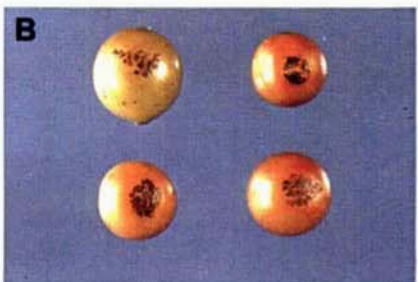
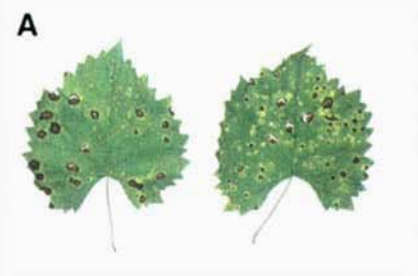


Fig. 8. Black rot of muscadine grapes caused by *Guignardia bidwellii* f. *muscadinii*: (A) Leaf spots and (B) fruit lesions.

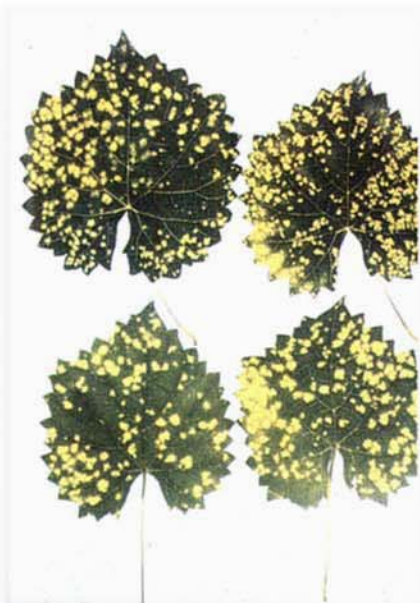


Fig. 9. Angular leaf spot on muscadine grape leaves caused by *Mycosphaerella angulata*.



Fig. 10. Marginal leaf necrosis on the muscadine grape cultivar Carlos caused by Pierce's disease bacterium, *Xylella fastidiosa*.



Fig. 11. Uredial pustules of *Physopella ampelopsidis* on lower surface of muscadine grape leaf.

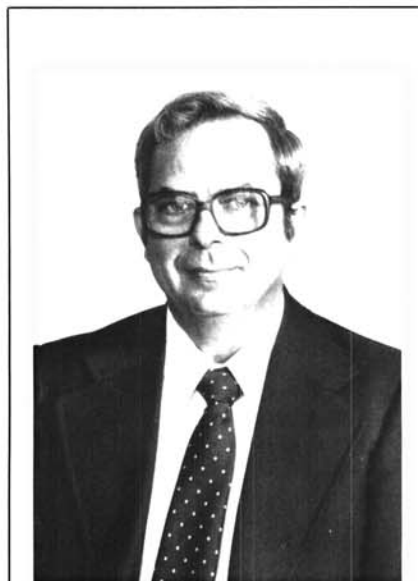
state extension service recommendations must be followed in applying chemical fungicides.

Maintaining clean, healthy plants through sanitation and cultural practices helps to ensure high yields and quality fruit. Mummies that remain attached to the vine, infected pedicels, and fruit spurs are primary sites of overwintering for *C. gloeosporioides*, and conidia are disseminated from these sites throughout the growing season (6). The sporadic development of ripe rot throughout a vineyard and lack of general spread indicate that mummies on the vine, and not those on the ground, are important sources of inoculum. The practice of severely pruning vines during the dormant season to remove these mummified fruit and dead, damaged, or otherwise undesirable wood is one of the best control measures for ripe rot and other fruit rot diseases of muscadine grapes. There are some pathogens, e.g., *M. angulata*, for which inoculum levels could be minimized by removing or destroying infected plant parts from the ground area beneath the vines.

In addition to reducing inoculum levels and disease severity, cultural practices can increase the effectiveness of chemical control. Planting in sites with good air circulation as well as removing excess vines and foliage during the growing season will provide better spray penetration and coverage. Maintaining a more open canopy also will help to lower the humidity, thereby reducing the duration of leaf wetness and disease development.

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