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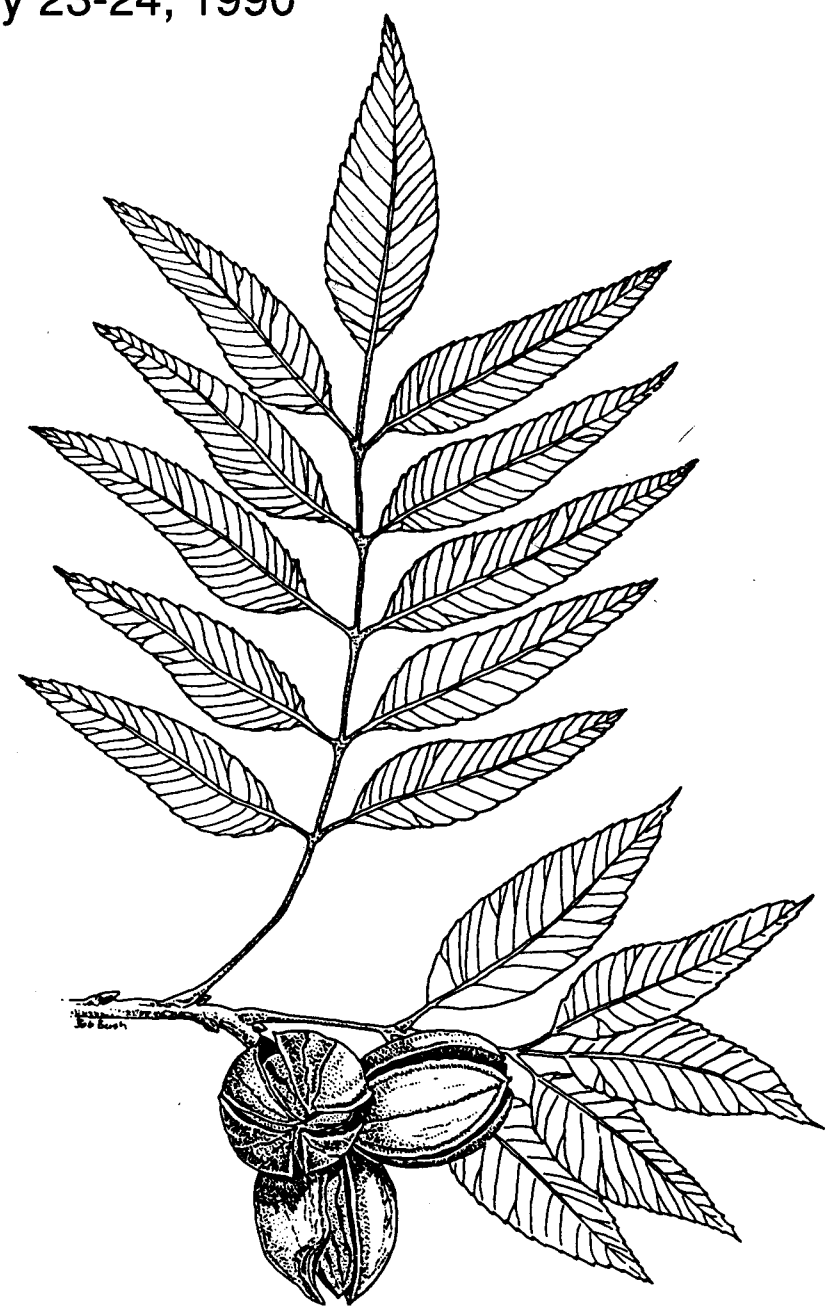

PECAN ANTHRACNOSE: REOCCURRENCE OF AN OLD PROBLEM

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ABSTRACT

Anthracnose of pecan *[Carya illinoinensis* (Wangenh.) C. Koch] caused by *Glomerella cingulata* (Ston.) Spauld. and Schrenk was first reported in the United States in 1914 (Rand 1914). Although widespread, it was not reported to cause substantial damage, and the disease received little attention during the next 75 years. Recent outbreaks in Georgia have demonstrated that the pathogen can cause significant late season yield loss on some varieties. Although originally reported on both shucks and leaves, recent observations have indicated only shuck symptoms which consist of sunken, black lesions usually initiated near the proximal end of the shuck. The fungus can penetrate the shell and kernel resulting in decreased kernel size, abortion of the nut, or shucks clinging to the shell at maturity. Symptoms were readily reproduced by inoculating detached nut clusters in the lab, but results of field inoculations were more erratic. *G. cingulata* and its anamorph, *Colletotrichum gloeosporioides* (Penz.) Sacc., are known pathogens of ripening and senescing tissues and are often associated with long latent periods. The frequency of isolation from shuck tissues increases steadily throughout the summer on susceptible cultivars and sometimes reaches 100% by late season. However, growth can also be saprophytic and successful isolations can be made from shucks with a variety of symptoms late in the season. More work is needed to determine the role of *G. cingulata* on different varieties and clarify the etiology of this and other shuck necroses.

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In 1911, Rand (Rand 1911) described a pecan leaf-blotch which he attributed to *Mycosphaerella convexula* (Schwein. Thüm, Sacc.). He described specimens obtained from Alabama, Georgia, Florida, South Carolina and Ohio. In 1914, Rand (Rand 1914) published a more extensive work in which the causal organism was positively identified as *Glomerella cingulata*. He described the anamorph, now known to be *Colletotrichum gloeosporioides* Penz., and indicated that it was found more frequently than the teleomorph, although many strains were holomorphic. Rand (Rand 1914) described in detail the symptoms observed on leaves and nuts of both natural infections and those resulting from inoculations in the laboratory. He reported that symptoms did not occur until mid to late season. Pecan isolates of *G. cingulata* were found to also cause bitter-rot symptoms on apples (Rand 1914).

After this extensive initial work by Rand, little attention was focused on this problem. It was mentioned in some subsequent pecan production summaries such as that of Stuckey and Kyle in 1925. They reported that "Anthracnose is well distributed throughout most localities..., attacks have been light and the industry has not suffered any serious effects from the disease" (Stuckey and Kyle 1925).

The disease faded even further into obscurity and was pretty much forgotten until recent years when pecan growers in Georgia started experiencing problems with shuck necrosis late in the growing season. Symptoms unrelated to scab were linked to a high incidence of *G. cingulata* on diseases tissues. This was documented by Brenneman and Reilly (1989) who successfully reproduced the symptoms by inoculating detached nut clusters in the laboratory. They also reported that the fungus could penetrate the shell and rot the kernel as well as infect the shuck to cause "stick-tights." Cultivars reported to be most susceptible based on field observations were Wichita, Grabohl, Van Deman and Schley (Brenneman and Reilly 1989).

Unfortunately the etiology of this disease is confounded by several factors. First, there are a number of shuck disorders reported on pecan. Stein and McEachern (1983) discussed several of these as well as possible causes including pathogens, irrigation, nutrition and physiological factors. For example, shuck dieback is a condition commonly affecting the variety Success and Success hybrids. This "shuck disease" as originally described by Schaller et al. (1968) begins with a black spot on the shuck adjacent to

the stem, similar to the symptoms later reported with *G. cingulata* (Brenneman and Reilly 1989). Shuck disease later withers the apex end and may envelop the entire shuck. It is thought to be caused by abiotic factors (Herrera 1982, Schaller et al. 1968) and the potential damage caused by this condition was documented by Schaller (1971).

Pecan stem end blight has been described by Halliwell and Johnson (Halliwell and Johnson 1978 and is reported to be of biotic origin and thus controlled by the use of fungicides. The suspected pathogen is *Botryosphaeria* sp. (Halliwell and Johnson 1978) and the nut-rotting potential of *B. ribis* has been demonstrated in India (Saharan 1974). A variety of symptoms were reported including severely rotted nuts, shucks fusing with shells, and simple colonization of shucks only. To further complicate the issue, a disease known simply as "dieback" affecting only twigs and limbs has been attributed to *Botryosphaeria berengeriana* DeNot (Marz 1918).

The second factor confounding the etiology of shuck diseases is the occurrence of both obvious and inconspicuous nut splitting problems. Worley and Taylor (15) reported this in 1972 and subsequent work by Daniell and Prussia (2) demonstrated the relationship between these symptoms and high osmotic pressures reached within the developing nuts of some varieties. Of interest to this discussion is the fact that affected nuts may exhibit obvious longitudinal splits or only minor cracks that result in a discolored spot on the shuck. Also, the variety found to be most susceptible to this split, Wichita, is also one of the most likely to exhibit symptoms of infection by *G. cingulata*.

Research conducted during 1989 demonstrated that *G. cingulata* can cause a significant reduction in yield and quality of pecan cv. Wichita, even when symptoms develop very late in the season (T.B. Brenneman, C.C. Reilly and M.W. Hotchkiss, unpublished data). Approximately 30% of individual nuts showing some symptoms (<25% of shuck discolored) had progressed to more than 75% discoloration at harvest two weeks later. There were 98.5 nuts/lb in this category versus 87.0 nuts/lb where there was still <25% necrosis at harvest. Those with no disease had 75.8 nuts/lb.

This study also demonstrated that higher severities of anthracnose are correlated with higher rates of late season nut drop. Unseasonal fruit drop occurs in most dicotyledonous tree crops and has been associated with *Colletotrichum* spp. in some of those (Wellman 1972). It is also of interest to note that Rand (Rand 1914) reported an association between pecan anthracnose and nut drop in his initial studies on the disease.

Isolation studies have demonstrated that the incidence of *G. cingulata* isolations from pecan shucks rises steadily through the season and may reach 100% in some orchards (C.C. Reilly, T.B. Brenneman and M.W. Hotchkiss, unpublished). This occurs even before visual symptoms are apparent on the shucks. This symptomless colonization indicates that latent infections are involved, something that occurs frequently with *Colletotrichum* spp. (Wellman 1972). Usually these infections are not evident until triggered by a change in the physiological state of the maturing host tissue. This appears to be true for pecan as well.

This process of latent infection along with the possibility of systemic infections via the branches adds to the confusion of determining etiology. Isolation studies from specimens collected throughout Georgia have often found *G. cingulata* associated with a variety of shuck necrosis symptoms, but it also appears to be an excellent saprophyte. Excellent growth occurs on shuck tissues damaged by other causes such as physiological split described previously.

Our biggest challenge currently is to determine the etiology of the various shuck necrosis problems found on a number of pecan varieties throughout the state. Disease ratings taken near harvest in 1989 indicated definite differences among varieties with regards to symptoms (T.B. Brenneman, unpublished). Results are given in Table 1, and those with basal symptoms known to be associated with *G. cingulata* infection are listed separately from those with shuck dieback as typified by "Success." It is apparent that there are different shuck necrosis symptoms and that varieties differ significantly in susceptibility to them. For example, most varieties exhibited no symptoms of *G. cingulata* infection whereas Wichita had more than 50% of the clusters affected.

The role of other factors such as nutrition, irrigation, other pathogens, etc. needs to be elucidated. However, we also need to learn more about the biology of *G. cingulata* in pecan orchards. Then, where the fungus is involved, we will have a basis to formulate control strategies, and where it is not involved, alternate programs can be developed.

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Table 1. Occurrence of shuck necrosis on various pecan varieties, Tifton, GA (Oct. 3, 1989).

Variety	# Clusters w/*Glomerella*[1]/ # Clusters evaluated	% w/*Glomerella* symptoms	% w/tip dieback
Stuart	0/20	0	0
Money maker	1/20	5.0	20
Schley	2/20	10.0	0
Van Deman	8/46	17.4	0
Kiowa	0/20	0	0
Frotcher	0/20	0	0
Alley	0/20	0	0
Curtis	0/30	0	0
Summers	2/21	9.5	0
Moore	0/22	0	4.5
Pabst	0/22	0	4.5
Mobile	0/27	0	0
Ivey	0/30	0	0
Woodard	0/32	0	0
Desirable	0/31	0	0
Farley	0/22	0	0
Tejas	0/30	0	6.7
Big Z	0/20	0	0
Bradley	0/20	0	0
Tesche	0/28	0	0
Melrose	0/20	0	0
Mahan	0/30	0	10
	Note - 11 additional Mahan clusters died at about 1/2 of full size development from unknown causes.		
Burkett	0/32	0	0
Williamson	0/30	0	6.7
W. Schley	2/31	6.5	25.8
Brooks	1/33	3.0	6.1
Oklahoma	0/36	0	0
Wichita	26/47	55.3	0
Penn Cluster	0/30	0	0
French TW	0/30	0	3.3
Harris	0/35	0	0
Hasting	0/33	0	0
Barton	0/30	0	56.7
Cape Fear	0/33	0	0

[1]Clusters with ≥1 nut exhibiting lesions similar to those known to be associated with *G. cingulata*.