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LATE SEASON SHUCK DISORDERS OF PECAN

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ABSTRACT

Late season shuck disorders have perplexed pecan specialists and growers for decades as to their description and cause. Confusion about these problems has been exacerbated because of several factors. A variety of different names apparently have been applied to the same symptoms; conversely, different problems may have been assigned the same names. There has been a lack of complete symptom description, which is compounded by variation in symptoms from year to year as well as differences between cultivars. In addition it has been difficult to obtain a definitive association of a specific symptomatology with a single specific cause. In this paper the literature concerning the late season shuck disorders is reviewed, and two distinct symptomtologies are identified. One named anthracnose is typified by necrosis beginning near the proximal end of the nut as a discrete sunken lesion that can spread over the shuck. Nuts with anthracnose often drop shortly after the onset of symptoms. Anthracnose had been reproduced in the lab by inoculations with *Glomerella cingulata*. Shuck dieback generally begins as a necrosis near the distal end of the nut. The entire shuck dies prematurely, and the nuts often remain attached to the terminals. Shuck dieback is associated with various environmental and cultural stress situations such as large crop loads.

All of the literature that comes under the umbrella of late season shuck disorders has at least one thing in common. It always describes a necrosis of the shuck that begins anywhere from around early August to nut maturity. Unfortunately, beyond the recognition of shucks becoming necrotic in late summer and early fall there was not always a detailed description of the symptoms given. The first published account of such a problem on the nuts was by Frederick Rand in 1914 (Rand 1914). The problem occurred in several southeastern states, and was reported as sunken necrotic spots on the shucks. Nuts with this necrosis often dropped from the trees. No particular location of the spots on the shucks was mentioned. The fungus *Glomerella cingulata* was isolated from these shucks and the necrosis was reproduced by inoculations in the lab. Rand named the problem anthracnose disease. In addition to the necrosis of the shucks, *Glomerella* also was reported to cause reddish brown lesions on the foliage (Rand 1914).

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During the late '60s and early '70s, Pete Schaller and Glen KenKnight, working at the USDA Pecan Station in Louisiana, published several papers in which they described late season shuck problems on pecan (Schaller et al.1968, Schaller 1971, Schaller and KenKnight 1972), they worked primarily with the 'Success' cultivar. In the last of these papers published in 1972 they recognized two distinct patterns of symptoms of shuck disorders (Schaller and KenKnight 1972). One of the patterns was a necrosis of the shuck beginning at the proximal end of the nut. The necrosis could spread to cover the entire shuck. The shuck thus affected would often stick to the shell of the nuts. They termed this problem 'stem-end blight'. Because the results of their tests made during two years indicated that applications of benomyl beginning in early August reduced the incidence of this malady they suggested that a fungus might be involved in causing the symptoms. They did not mention any specific fungus. They also described another pattern of shuck necrosis in which the shuck begins to turn black near the distal end of the nut. This symptom was associated with an excessively large crop. They termed this problem 'shuck dieback'. Both of these problems could occur at the same time and even on the same nut. In addition to the end of the nut on which the necrosis began, another common difference was that nuts with stem-end blight often had the shuck stick to the shell, while nuts with shuck dieback typically had the shuck flare open (Schaller and KenKnight 1972). It has been my observation that these are general tendencies but not absolute distinguishing characteristics; sometimes the shucks of nuts with either malady can stick to the shell or flare open. Schaller and KenKnight indicated in this paper that a variety of terms had been applied to late season shuck disorders and suggested that the term 'shuck disease' had been used in a general sense for any and all late season shuck necrosis and should not be used any longer. Unfortunately, this suggestion has not been followed.

In the late '70s and early '80s, it was reported from Texas that stem-end blight was caused by *Botryosphaeria* (Halliwell and Johnson 1978, Johnson 1980). However, I could not find any report on the confirmation of infection by inoculation studies. These same reports also indicated that shuck dieback appeared to be physiological and not involve a pathogen. The association of *Botryosphaeria* with stem-end blight was similar to a report from India in 1974 in which a nut rot was reported to be caused by this fungus (Saharan, 1974). The symptoms described for the nut rot of pecan in India caused by *Botryosphaeria* were different from the symptoms reported for late season shuck diseases in the U. S. in that numerous lesions on the shuck coalesced to completely cover the nut, and a watery exudate was often present with the rot.

Brenneman and Reilly described a late season problem of pecan that started with necrosis at the proximal end of the nut and in which the shucks often stuck to the shell (Brenneman and Reilly, 1989). *Glomerella cingulata* was isolated from these nuts and the disease reproduced by inoculation with *Glomerella* in lab tests. This was the fungus described as the cause of anthracnose by Rand in 1914. The symptoms described by Brenneman and Reilly

for anthracnose were very similar to the symptoms described as stem-end blight by Schaller and KenKnight (Schaller and KenKnight, 1972). Anthracnose and stem-end blight are probably the same phenomenon, and because the name anthracnose predates the term stem-end blight, anthracnose should be the proper name applied to this problem.

Reilly most recently indicated that not only could *Glomerella* inoculations produce the symptoms of necrosis beginning at the proximal end of the nut but that inoculations with another fungus isolated from pecan, a species of *Phomopsis,* also could reproduce the symptoms of anthracnose (Reilly 1994) . Additionally lab inoculations with either fungus could produce necrosis that began at the distal end of the nut (Reilly 1994). Prior to this it had been suggested by others that the necrosis beginning at the distal end of the nut was a physiological response to stress (Schaller and KenKnight 1972, Halliwell and Johnson 1972, Sanderlin, 1989).

A study of the incidence and damage caused by anthracnose and shuck dieback was conducted over a five-year period at the LSU Pecan Station from 1980 through 1984. The test was on cv. 'Success' trees that were about 50 years old. Comparisons were made on the occurrence of shuck dieback and anthracnose between trees that were not treated with fungicide and trees that received benomyl applications full season. Fungicide applications were made on a 3-4 week schedule from April through August. Insecticide and fertilizer applications were the same for all trees in the test each year. The same treatment was applied to the same individual test trees throughout each year of the test. There were twelve trees that received benomyl applications each year and six that got no fungicide. Each year twenty nut-bearing terminals were tagged on each tree and monitored regularly for shuck problems. Nut clusters on both the perimeter of the tree limbs and on the interior of the tree were tagged and the clusters varied in location from 8 feet to 50 above ground.

In four of the five years the trees treated with benomyl had a significantly lower number of nuts with anthracnose than the untreated trees (Table 1). The year without a significant difference (1984) apparently occurred because the incidence of anthracnose was low on the untreated trees. At the same time, the incidence of shuck dieback was significantly lower in only one year (Table 2). In two of the five years, the incidence of shuck dieback was higher on the benomyl-treated trees. This seems to support the observation that shuck dieback is a separate problem from anthracnose (Sanderlin 1989). The effect of both anthracnose and shuck dieback on nut weight in this test was variable and depended on when symptoms began to occur. If symptoms began before the end of September, the reduction in nut weight was significant. The closer the onset of shuck necrosis to the time of normal shuck split the lesser the effect on weight. Anthracnose caused a significant reduction in nut weight in three of the five test years, and shuck dieback reduced weight significantly in two of the years (Table 3). Over the five-year period of the test these two problems each reduced the average nut weight by 12 percent (Table 4). In addition to this circumstantial evidence that anthracnose and shuck dieback are separate entities is the observation that some orchards have both problems commonly occurring in them while others were observed with primarily the shuck dieback syndrome with anthracnose appearing rarely. Overall shuck dieback is observed more frequently than anthracnose in orchards in LA (Sanderlin 1989). It is important to realize that in the advanced stages of development shuck dieback and anthracnose cannot be distinguished from one another by their appearance.

There is some additional empirical evidence that correlates the shuck dieback problem with a physiological response to various stresses on crop development. The primary stress factor associated with shuck dieback is an excessive crop; this was originally reported by Schaller and KenKnight when they separated the two symptomtologies (Schaller and KenKnight 1972). This also was noted in work from Texas (Halliwell and Johnson 1978). Shuck dieback symptoms were induced by girdling the peduncle or treatment with ethylene (Halliwell and Johnson 1972). The most recent circumstantial evidence for stress induced by a large crop causing shuck dieback was presented by Sparks and co-workers (Sparks et al. 1995). They demonstrated that the incidence of shuck dieback on cv. 'Wichita' could be significantly reduced on a large crop by mechanically thinning the crop during the liquid endosperm stage of nut development. In their work they referred to late season shuck necrosis as shuck decline. They described shuck decline as initially beginning as a thin necrotic line in the shuck tissue at the junction of the shuck to the shell. Soon the entire shuck becomes necrotic. They did not indicate that necrosis began at either end of the nut, and apparently the entire shuck develops necrosis at the same time. However, the final stage of shuck decline as demonstrated in photos appears very much like the final stages of shuck dieback (Sparks et al. 1995). It is probable that shuck decline and shuck dieback are the same problem with differences in appearance the result of cultivar and other variations. Photos of shuck decline from Arizona on cv. 'Western' look identical to shuck dieback on cv. 'Success' described from Louisiana. Because shuck dieback was the original term used to describe this pattern of necrosis and has been commonly used in the literature since 1972, it should be retained in place of shuck decline to avoid addition confusion.

In summary, the situation with the late season shuck disorders is still somewhat murky; thus additional research is needed. But it appears that at least two distinct symptomtologies exist. One of these, anthracnose disease, is associated with one or more fungal pathogens. Evidence from several publications indicates that *Glomerella cingulata* is a pathogen involved in anthracnose development (Rand 1914, Brenneman and Reilly 1989, Reilly 1992, Reilly 1994). Two other fungi also have been implicated (*Botryosphaeria* and *Phomopsis)* (Halliwell and Johnson 1978, Reilly 1994) in anthracnose. For the other shuck necrosis pattern, referred to as shuck dieback, circumstantial evidence has been collected by several workers that suggest it is a separate problem from

anthracnose and may be a physiological response to cultural and environmental stresses (Schaller and KenKnight 1972, Halliwell and Johnson 1972, Sparks et al 1995, Sanderlin Table 1 & 2). The possibility that fungi also are involved in development of shuck dieback has been demonstrated (Reilly 1994).

In addition to the late season shuck disorders anthracnose and shuck dieback, there are other problems that can cause the shuck to become dark during late summer and fall. These include Phytophthora nut and kernel rot (Reilly and Hendrix 1989), water split, mechanical damage, and shuckworm feeding. Fortunately these problems are usually easily distinguished from anthracnose and shuck dieback.

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Table 1. Percent of nuts with anthracnose disease. Benlate treatment applied on a 3-4 week schedule from April through August (1.0 lb/acre). Check trees received no fungicide. Statistical significance based on LSD (P=0.05).

	PERCENT OF NUTS WITH ANTHRACNOSE				
TREATMENT	1980	1981	1982	1983	1984
BENLATE	3.8	3.2	5.6	11.2	1.1
CHECK	30.2	26.1	21.1	45.5	4.3
STATISTICAL SIGNIFICANCE	YES	YES	YES	YES	NO

Table 2. Percent of nuts with shuck dieback. Benlate treatment applied on a 3-4 week schedule from April through August (1.0 lb/acre). Check trees received no fungicide. Statistical significance based on LSD (P=0.05).

	PERCENT OF NUTS WITH SHUCK DIEBACK				
TREATMENT	1980	1981	1982	1983	1984
BENLATE	10.5	38.0	8.2	33.4	15.3
CHECK	19.5	25.2	25.4	25.4	19.5
STATISTICAL SIGNIFICANCE	NO	NO	YES	NO	NO

Table 3. Weight of nuts that had either shuck dieback or anthracnose disease compared to the weight of nuts with normal (healthy) shuck split. LSD (P=0.05).

	IN-SHELL WEIGHT (grams)				
VARIABLE	1980	1981	1982	1983	1984
SHUCK DIEBACK	8.11	8.31[A]	7.79[A]	5.51[A]	8.55
ANTHRACNOSE	8.28	7.33[B]	8.52[B]	6.35[A]	7.64
NORMAL	9.23	8.61[A]	9.13[C]	7.35[B]	9.25
LSD	NS	0.45	0.52	0.93	NS

Table 4. Effect of anthracnose and shuck dieback on nut weight (five-year average, 1980-1984).

	MEAN IN-SHELL WEIGHT (grams) FOR 5 YEARS	PERCENT DEVIATION FROM NORMAL
SHUCK DIEBACK	7.65	-12.2
ANTHRACNOSE	7.62	-12.5
NORMAL	8.71	-