Fruiting Stress Induces Shuck Decline and Premature Germination in Pecan

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Abstract. The influence of fruiting stress on shuck decline, nut quality, and premature germination was evaluated on trees of pecan [*Carya illinoensis* (Wangenh.) C. Koch]. Fruit at the liquid endosperm stage were removed from trees with a mechanical shaker to reduce crop load by 0%, 25%, 41%, 56%, or 77%. Shuck decline and premature germination decreased and kernel quality increased with a reduction in crop load. An excessive fruit load or fruit stress elevated the incidence of shuck decline, previously referred to as shuck disease, tulip disease, shuck die-back, or late season shuck disorder; decreased kernel development; and increased premature germination. Shucks were dissected from fruit ranging from healthy to those with premature shuck opening and examined by scanning electron, transmission electron, and light microscopy. Fungal growth was detectable, but only after tissue degeneration had occurred. Thus, results indicate the onset of shuck decline is caused by stress associated with an excessive crop load and not a pathological disorder. Fungal growth is a secondary, not a primary, factor in deterioration of shucks with decline.

Fruit development in pecan is an exhaustive process that imposes stress on the tree (Davis and Sparks, 1974; Sitton, 1931; Smith et al., 1993; Sparks and Brack, 1972). Stress from fruit development is manifested in poor nut quality (incomplete kernel development) often produced by prolific pecan cultivars, especially by mature trees (Sparks, 1990). Often poor quality is concomitantly associated with shuck (involucre) deterioration that may result in premature shuck opening in severe cases. This condition has been designated by a variety of terms including shuck dieback, shuck disease, and tulip disease (Halliwell, 1968; KenKnight, 1968; Payne et al., 1979; Schaller and KenKnight, 1972; Schaller et al., 1968), although data were not presented to support a disease (Halliwell, 1968; Schaller et al., 1968). Shuck deterioration associated with tree stress was not considered by Schaller et al. (1968) to be an infectious disease or by Halliwell and Johnson (1972) to be caused by a pathogen. Halliwell and Johnson (1972) induced shuck deterioration by ethylene treatment, a result that supports the involvement of tree stress in development of this malady. Contrary to the association of tree stress with shuck deterioration, Reilly and co-workers (Brenneman and Reilly, 1989; Hotchkiss et al., 1993; Reilly, 1989, 1990, 1992; Reilly and Hotchkiss, 1991) recently concluded the causative agent to be the fungus *Glomerella cingulata* (Stoneman) Spauld. and H. Schrenk [anamorph = *Colletotrichum gloeosporioides* (Penz.) Penz. and Sacc. in Penz.].

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Shuck deterioration associated with fruit stress is often, but should not be, confused with another shuck disorder that has been termed stem-end blight (Schaller and KenKnight, 1972). Stem-end blight is characterized by a gray-brown or black spot that first appears at or near the proximal end of the immature shuck. The spot enlarges and can engulf the entire shuck. The dead shuck may stick to the nut producing a sticktight. Stem-end blight occurs earlier in the growth cycle of the fruit (early August in the southeastern United States) than shuck deterioration from fruiting stress (early to late September). Fungicide sprays reduce the incidence of stem-end blight (Schaller and KenKnight, 1972) but have not been shown to reduce the incidence of shucks deteriorating from fruiting stress (Hotchkiss et al., 1993; Latham and Campbell, 1991).

At least two reasons account for the confusion in the literature on the nature of shucks failing to develop normally. First, symptom descriptions are ambiguous. Second, several abnormal conditions are often grouped into one category, such as shuck disorder. The terminology shuck decline will be used henceforth in this study to designate shuck deterioration associated with tree stress. Shuck decline begins as a thin, dark, necrotic line on the inner surface of the shuck at the junction with the shell, then spreads rapidly towards the exterior surface of the shuck. The interior of the shuck turns dark green and slimy and the surface of the shuck appears to be water-soaked exhibiting a green sheen. Later, the entire shuck turns black. If decline begins during early fruit development, the black fruit falls from the tree or opens prematurely and remains in the cluster (Sparks, 1992a, 1992b).

Circumstantial evidence for fruiting stress as the prerequisite for shuck decline has accumulated from extensive observations of crop load by year, tree, cultivar, and shoot vigor. Years of severe shuck decline are often concurrently accompanied by high fruit

loads; whereas, years with little or no shuck decline are usually associated with light to moderate fruit loads (Sparks, 1992b, 1993a). Observations in a mature orchard with variable fruit set among trees, likewise, show shuck decline occurs in direct proportion to the degree of fruiting. Also, prolific cultivars, such as 'Success', 'Wichita', 'Cherokee', 'Cape Fear', 'Choctaw', 'GraBohls', and 'Chickasaw', usually produce high quality nuts without shuck decline as young trees; but quality decreases and shuck decline increases in these cultivars as the leaf to fruit ratio decreases with tree maturity. Mature trees of prolific cultivars have good nut quality and a low incidence of shuck decline during the "off" year of production with the reverse occurring during the "on" cycle of the alternate bearing sequence. This alternation of shuck decline has long been observed with 'Success', a prolific cultivar that is classically known for poor quality and a high incidence of shuck decline during the "on" cycle of fruit production (Sparks, 1992a). Furthermore, shuck decline on 'Success' trees with similar crop loads decreases as shoot vigor increases (Schaller et al., 1968; Sparks, 1992a). An inadequate leaf to fruit ratio (Sitton, 1931) from less leaf area on short shoots (Sparks, 1966) probably accounts for the increase in shuck decline with decreasing vigor.

Premature germination (vivipary) can occur concurrently with shuck decline or may occur without shuck decline (Sparks, 1993a). Premature germination is associated with delayed shuck opening and high ambient temperature (Finch, 1937; Finch and Van Horn, 1936) and is a major problem for pecans growing at low elevations in hot climates (Sparks, 1993a). A combination of maintaining adequate soil moisture to enhance shuck dehiscence and early nut harvesting can reduce premature germination (Sparks, 1993a). However, in years of excessive fruit load, premature germination occurs before the fruit mature, resulting in a substantial loss of marketable nuts. Also in such seasons, shuck decline is frequently a major problem. Preliminary data indicate that reducing crop load will reduce premature germination to an acceptable level (Sparks, 1993b).

The two problems, shuck decline and premature germination, are apparently associated with excessive fruiting. The objective of the current study was to determine the effect of fruiting stress on shuck decline and premature germination.

Materials and Methods

The experimental site was near Crystal City, Texas, in a 13-year-old flood-irrigated orchard. The site was selected because 'Wichita', the major cultivar in the orchard, is very susceptible to premature germination and shuck decline (Sparks, 1993a). Thirty trees, with crop loads judged to be excessive, were selected for study on 22 July 1993. Previous experience in this orchard had shown that trees with excessive crop loads developed premature germination, shuck decline, and poor kernel quality. Heavy crop load was verified by determining that 99% of the shoots were fruiting and that cluster size of fruiting shoots averaged 4.5 fruit.

Fruit were thinned mechanically on 22 and 23 July to remove a projected 0%, 20%, 40%, 60%, or 80% of existing fruit from the selected trees. Actual percentages of fruit removed were calculated at harvest to be 0%, 25%, 41%, 56%, and 77%, respective to the projected percentages. Fruit were thinned during the liquid endosperm stage of development. Most fruit were near maximum liquid endosperm stage with a few fruit ($\approx 10\%$) slightly past this stage. Initial shell hardening was completed on some ($\approx 10\%$), but not all, fruit. Fruit development stage was within the range recommended for mechanical fruit thinning of pecan (Reid et al., 1993; Smith et al., 1993) which is before the major stress of kernel development (Davis and Sparks, 1974). The fruit removed from

each tree were weighed. A sample of ≈ 225 fruit was taken per tree to determine fruit weight which, in turn, was used to calculate the total number of fruit removed from each tree.

Nuts were harvested by tree on 24 Sept. Before harvest, the number of fruit on each of 100 shoots was recorded by tree. On the same day and also before harvest, a random sample of ≈ 100 fruit were collected from each tree to rate shuck decline. Fruit from this sample were then dehulled, weighed, and separated into germinated and nongerminated nuts. Nuts were air-dried at $\approx 25^\circ\text{C}$. Nut, shell, and kernel weight, percent kernel, and percent edible kernel (U.S. No. 1 grade) were determined individually for nongerminated nuts. The remainder of nuts were harvested by tree; dehulled; weighed; dried in a pecan wagon by ambient, forced air for 2 days; and weighed again. Before drying, a sample of ≈ 125 freshly dehulled nuts were collected by tree and weighed. Premature germination (Fig. 1) was estimated from pre- and postharvest nut samples. Fresh weight of dehulled nuts of both samples was used to calculate the number of fruit per tree at the time of harvest. Percentage of fruit removed by mechanical thinning was calculated from the number of fruit removed on 22 July and the number on the tree at harvest time.

Decline was categorized relative to no decline on the basis of increasing severity on the shuck exterior into three stages as water-soaked (Fig. 2A), black shuck (Fig. 2B), and black shuck that had opened prematurely (Fig. 2C). Salmon-colored fruiting bodies described as characteristic of *G. cingulata* (Reilly, 1989, 1990; Reilly and Hotchkiss, 1991) were evident on the shucks of some fruit in the last two stages of decline. The first symptom of decline, the thin, dark, necrotic line on the inner shuck (Fig. 2D) was not categorized.

Shucks were dissected from fruit ranging from healthy to premature shuck opening for examination by light microscopy (LM), scanning electron microscopy (SEM), and transmission electron microscopy (TEM). Sections were taken at the midpoint of the fruit perpendicular to the long axis of the fruit and, thus, cross sections of the vascular system. Samples prepared for hand sectioning were fixed in FAA [70% ethyl alcohol : glacial acetic acid : formalin (90:5:5, v/v)] for 1 month or longer at room temperature. Hand sections were examined after fixation either without any further treatment, with clearing, or with histological stains. Sections were cleared for 3 weeks in BB-41/2 [2 lactic acid : 2 chloral hydrate : 2 phenol : 2 clove oil : 1 xylene : 1 benzyl benzoate (by

Fig. 1. Premature germination in 'Wichita' pecan.

Fig. 2. Stages of shuck decline: Water-soaked (A); black shuck on left fruit and no decline on right fruit (B); premature shuck split on left fruit and no decline on right fruit (C); dark, necrotic line on inner shuck on left fruit and no decline on right fruit (D).

weight)] (Herr, 1982) and examined with a Leitz Dialux 22 EB microscope equipped with differential interference contrast (DIC) (LEICA, Wetzlar, Germany). Histological stains used were potassium iodide for starch (Johansen, 1940), ferric chloride for polyphenols, Sudan IV for lipids (Pearse, 1961), and Coomassie brilliant blue for proteins (Fisher, 1968).

At least 24 samples for microscopy were fixed overnight at 4°C with 4% glutaraldehyde in 0.1 M sodium cacodylate buffer, pH 7.2. Following glutaraldehyde fixation for SEM, samples were dehydrated (30 min in each of 50%, 70%, 100% ethanol), critical-point dried with CO_2 , mounted on stubs, coated with gold-palladium, and examined at 15 kV with a Philips 505 SEM (North American Philips Corp., Mahwah, N.J.). Following glutaraldehyde fixation for LM and TEM, samples were then treated 1 h in an ice bath with 1% OsO_4 buffered with 0.1 M sodium cacodylate, stained with 1% uranyl acetate for 1 h in an ice bath, dehydrated in an ethanol series and embedded in resin (Spurr, 1969). Sections were cut using an ultramicrotome (model Ultracut E; Reichert-Jung, Vienna, Austria) at 90 nm for TEM and 0.5 μm for LM. Ultrathin sections were studied in a Jem-100 CX II TEM (JEOL, Peabody, Maine). For LM, sections were stained with a mixture of 1% azure II and 1% sodium borate (1:1, v/v) by applying gentle heat to the glass before examination with bright field illumination using a Leitz Dialux 22 EB microscope.

The experimental design was a randomized complete block replicated six times with one tree per experimental unit. Results were delineated by regression analysis (Gomez and Gomez, 1984).

Results and Discussion

Healthy pecan shucks had outer, central, and inner tissue zones that were distinguished on the bases of cell size and shape (Fig. 3A). The outer zone consisted of about four layers of small, rectangular cells whose long axis was parallel to the surface of the fruit (Fig. 3A and B). The central zone composed the major portion of the shuck and began about 0.1 mm from the surface (Fig. 3A–D). Cell size increased and shape became more elongated perpendicular to the shuck surface with each progressive layer toward the inner zone. The third and inner tissue zone of the shuck was made up of ≈ 10 layers of small, rounded cells (Fig. 3A, C, and D). Globules of $\approx 50 \mu\text{m}$ in diameter (Fig. 3D) filled some, but not all, inner zone cells and were not observed at all in cells of the other two tissue zones.

An obvious difference, microscopically and macroscopically, between healthy and black shucks (Figs. 3A and 4A, respectively) was the reduced thickness of black shucks. Another difference was in the shuck consistency. Healthy shucks were firm, so cuts made with a razor blade were smooth. Shucks with decline, however, were flaccid and therefore not adaptable to sectioning with a razor blade, as evidenced by tearing. The outer, central, and inner tissue zones could be identified in shucks blackened from decline (Fig. 4A) as in healthy shucks. However, differences in cell shape among the zones were not as accentuated as in a normal shuck, especially elongation in the central zone (Figs. 4A and B vs. 3A and B). Globules occurred in cells of the inner and central zones of

black shucks (Fig. 4 C–E); whereas, they were restricted to the inner zone of healthy shucks (Fig. 3 C and D). Thus, basic tissue zonation in the pecan shuck was not altered by decline, but cell shape and content were altered in the central tissue zone. Furthermore, differences in tissue consistency would indicate that the rigidity of the cells was affected by shuck decline.

Shucks, dried and black from the normal process of senescence, were examined for similarities and/or differences to shucks in the black stage of decline. Many cells in shucks black from normal senescence had convoluted walls (Fig. 5A) in contrast to the regular shape of cell walls in shucks with decline (Fig. 4C). Some cells in the central zone were filled with globules with a smooth

surface (Fig. 5A) characteristic of those observed in shucks with decline. Other cells had structures with a crystalline appearance (Fig. 5B), which were presumed to be calcium oxalate (Gallaher and Jones, 1976). However, we propose that the smoother appearing globules are the products of normal shuck senescence. Because globules occur earlier and are more numerous in declining shucks, we further propose that shuck decline is an acceleration and accentuation of the condition occurring with normal senescence.

The progression of decline through the tissue zones was characterized from free hand sections with increasing severity of decline. Light microscopy of these sections (Fig. 6) confirmed SEM observations that rounded cells of the inner tissue zone

Fig. 3. SEM of normal shuck. Cross section of entire shuck (A) demonstrates relative proportion of the outer, central, and inner tissue zones. Enlarged outer (B) and inner (C) zones of shuck. Further enlargement of inner zone (D) to show globules. The top and bottom sides of boxes define limits of indicated tissue zones. Abbreviations: o = outer, c = central, I = inner. Bars = 0.5 mm (A and C), 0.25 mm (B) and 0.2 mm (D).

adjacent to the shell had two different cytoplasmic contents. Healthy shucks (Fig. 6A) without a macroscopically visible blackness had many cells with a dense, reddish brown cytoplasm. Interspersed among the dense cells were layers of cells with a translucent cytoplasm. Once decline became obvious as a thin

black line, most inner tissue zone cells appeared occluded with a black deposit (Fig. 6B). As decline progressed to the water-soaked stage, black globules appeared in an occasional elongated cell in the central tissue zone (Fig. 6C). Most of the inner tissue zone appeared as a black, amorphous mass. In black shucks, dark

Fig. 4. SEM of shuck in the black shuck stage of decline. Cross section of entire shuck (A) demonstrates relative proportion of the outer, central, and inner tissue zones. Enlarged outer (B) and inner (C) zones of shuck. The top and bottom sides of boxes define limits of indicated tissue zones. Globules present in cells of inner (D) and central (E) zone. Abbreviations: o = outer, c = central, I = inner. Bars = 0.5 mm (A and C), 0.25 mm (B), 10 μ m (D), and 50 μ m (E).

Fig. 5. SEM of central zone cells with globules (A) and crystalline deposits (B) in a normal shuck following natural senescence. Abbreviations: w = wall, g = globule, c = crystalline deposit. Bars = 10 μ m (A and B).

globules were common throughout the central zone with most cells containing one or more of these structures (Fig. 6D). In summary, free hand sections demonstrated the macroscopic black appearance of shucks was due to intracellular dark globules. However, these sections were not satisfactory for defining the stage of shuck decline that might be associated with fungal growth as proposed previously (Brenneman and Reilly, 1989; Hotchkiss, et al., 1993; Reilly, 1989, 1990, 1992; Reilly and Hotchkiss, 1991).

An apparent secondary role of microorganisms in the progression of shuck decline was documented in tissue sections made with a microtome and examined with LM and TEM (Fig. 7). Analyses were confined to shuck tissue near the shell in which decline was first manifested, the area at the junction of the inner and central zones. Cells appeared to have a translucent cytoplasm in healthy shucks from free hand sections which was confirmed by microtome sections at the level of LM and TEM to have a sparse cytoplasm (Fig. 7A and B, respectively). Cytoplasm was restricted primarily to the cell periphery and composed of small, dense globules of various dimensions. The middle lamella of adjoining cells and lacunae present at some sites were evident from TEM (Fig. 7B). No evidence of fungi was present at the level of either LM or TEM for healthy shucks (Fig. 7A and B, respectively). Shucks with macroscopic evidence of early stages of shuck decline, i.e., a thin black line next to the shell (Fig. 2D), had cells similar to the normal shucks except for an increase in the size and

abundance of globules (Fig. 7C and D, respectively). No evidence of fungal growth was present either inter- or intracellularly (Fig. 7D).

Intercellular fungal growth became obvious after deterioration advanced to the water-soaked stage of shuck decline (Fig. 2A) in microtome sections by LM (Fig. 8A) and was confirmed by TEM (Fig. 8B and C). Fungi were observed growing between cells in the region of the middle lamella (Fig. 8B) as well as the lacunae (Fig. 8C).

Free hand shuck sections were treated with histological stains for starch, protein, fat, tannin, and polyphenol in an attempt to determine the chemical nature of the globules. However, no definitive identification was possible based on histological procedures because of the interference of the natural coloration of the globules with evaluation of the uptake of stains.

In summary, anatomical evidence indicated fungi became established only after the plant cells were deteriorating. The major change in the cytoplasmic content of plant cells in the early stages of decline was an abundance of electron dense globules that occurred before fungal growth. No penetration sites of *G. cingulata* on the shuck exterior were found in a concentrated study involving biweekly field inoculations of the fungus onto shucks from mid-June until August (Kerrigan, 1993). The same study reported that acervuli of *G. cingulata* emerged through shuck surfaces, sometimes on previously asymptomatic tissues. The current investigation along with that of Kerrigan (1993) indicates that fungal growth that followed shuck decline was initiated in the inner tissue zone of the shuck and progressed to the exterior.

Midseason fruit removal had a dramatic effect on the incidence of shuck decline. For each stage of decline, decreasing fruit load decreased the percentage of fruit exhibiting shuck decline symptoms (Fig. 9). Kernel quality, measured as percent kernel or as edible kernel, increased with the percentage of fruit thinned (Fig. 10). The data clearly document that shuck decline is due to fruit stress. Our data also show that poor kernel development, concomitantly associated with shuck decline, is likewise due to a fruiting stress. Although a fungus or fungi are present in shucks affected with decline, microbial growth occurs after the onset of decline. Thus, as emphasized by Halliwell and Johnson (1972) and Schaller et al. (1968), shuck decline is not caused by a pathogen. Lack of a pathogen is also supported by the fact that application of conventional pecan fungicides has not been shown to prevent shuck decline (Hotchkiss et al., 1993; Latham and Campbell, 1991) and by the inability to induce decline after field inoculations of *G. cingulata* (Kerrigan, 1993).

Premature germination decreased in direct proportion to fruiting stress (Fig. 10), confirming observations and preliminary data (Sparks, 1993b). Thus, in addition to delayed shuck opening (Finch, 1937; Finch and Van Horn, 1936), fruiting stress accentuates premature germination.

Fruit that was thinned up to 25% maximized total weight of nuts; thinning 25% to 41% maximized nongerminated nuts; 41% fruit thinning maximized nongerminated kernels; maximum edible kernel occurred at 56% to 77% thinning (Fig. 11). The key parameter is edible kernel because it represents marketable yield. Maximum marketable yield occurred with 56% fruit thinning. This thinning level corresponded to 72% of the shoots retaining fruit with an average cluster size of 2.9 fruit on fruiting shoots. Although shuck decline (Fig. 9) and premature germination (Fig. 10) were minimal at the highest level of fruit thinning, both were still present, suggesting a stress factor in addition to fruiting. Insufficient soil moisture was indicated by occasional dead and nonabscised leaves, a symptom of moisture stress.

Although fruiting stress often is a prerequisite for shuck decline, high fruit load does not result necessarily in shuck decline. Instead, data and many observations indicate that shuck decline most commonly occurs when fruit stress is accentuated by other factors. Inadequate sunlight and soil moisture stress are apparently two major accentuating factors for shuck decline. Shuck decline on 'Success' and other cultivars can be more pronounced on the shady than the sunny side of the tree, as on border rows. Similarly, decline has been observed to be worse in crowded than open areas of an orchard (Schaller et al., 1968; Sparks, 1992a). Excessive shade, as suggested by repeated observations, appears to be a major factor in the induction of shuck decline even on cultivars that are not prolific, e.g., 'Desirable'.

Soil moisture stress was associated with acute shuck decline in the southeastern United States in 1991, a year of excessive fruit load (Sparks, 1992b). Recordbreaking rainfall occurred during most of the growing season until about mid-August. Waterlogging, which Schaller et al. (1968) also observed to be associated with shuck decline, occurred frequently. Stress was further accentuated by a record-breaking drought from mid-August to mid-September. During the drought, massive premature defoliation occurred in nonirrigated orchards. Premature defoliation under conditions of a high fruit set, as in this case, is especially stressful (Sparks and Brack, 1972). Additionally, poor nut quality could be expected under soil moisture stress as kernel development is critically dependent on adequate soil moisture (Sparks, 1992a,b).

Fig. 6. LM of normal shucks (A) and shucks with progressive stages of decline including fine black line (B), water-soaked (C), and black shuck (D). The top and bottom sides of boxes define limits of inner tissue zone. Point indicates black globules. Bars = 100 μ m (A-D).

Fig. 7. Normal shucks (A and B) and shucks with thin black line (C and D) examined by LM (A and C) and TEM (B and D). Abbreviations: l = lacunae, m = middle lamella, c = cytoplasm. Bars = 100 μm (A and C), 10 μm (B and D).

Pecans had massive shuck decline in southern Brazil in 1993, a year with weather patterns similar to the southeastern United States in 1991 when rains were excessive during most of the growing season followed by a drought during kernel development (Geraldo T. Linck, personal communication). Also, as in the southeastern United States, shuck decline was severe on the prolific cultivars, 'Wichita', 'Barton', 'Shoshoni', and 'Cape Fear', but was only minor on the nonprolific 'Desirable'.

In 1991, shuck decline in Georgia was less severe in orchards with adequate irrigation during the drought than in orchards with no or inadequate irrigation. The role of soil moisture was strikingly

Fig. 8. Water stage of shuck decline examined by LM (A) and TEM (B and C). Abbreviations: p = plant cell, f = fungal cell, l = lacunae. Bars = 100 μ m (A), 10 μ m (B and C).

Fig. 9. Shuck decline on 'Wichita' pecan fruit as a function of fruit thinned during the water stage of fruit development. The relationship of cumulative shuck decline, water-soaked stage, black shuck stage, and premature shuck split stage to fruit thinned is described by $Y = 80.8 - 8.86\sqrt{X}$, $r^2 = 0.796$; $Y = 48.8 - 5.23\sqrt{X}$, $r^2 = 0.698$; $Y = 14.7 - 1.57\sqrt{X}$, $r^2 = 0.633$; and $Y = 18.0 - 2.15\sqrt{X}$, $r^2 = 0.600$, respectively. All regression coefficients are statistically significant from zero, $P = 0.05$.

Fig. 10. Percent kernel, edible kernel, and germination of 'Wichita' nuts as influenced by fruit thinned during the water stage of fruit development. Data are for nongerminated nuts only. The relationship of percent kernel, edible kernel, and germination to fruit thinned is described by $Y = 71.23[1 - 0.1e^{-0.018277(x-80.59)}]$, $r^2 = 0.755$; $Y = -5.66 + 1.22x$, $r^2 = 0.669$; and $Y = 41.31 - 0.47x$, $r^2 = 0.620$, respectively. All regression coefficients are statistically significant from zero, $P = 0.05$.

Fig. 11. Total nuts, germinated nuts, nongerminated nuts, nongerminated kernels, and edible kernel of 'Wichita' pecan as a function of fruit thinned during the water stage of fruit development. The relationship of total nuts, nongerminated nuts, nongerminated kernels, and germinated nuts to fruit thinned is described by $\sqrt{Y} = 5.87 + 0.0385X - 0.0008X^2$, $r^2 = 0.550$; $\sqrt{Y} = 4.16 + 0.0664X - 0.0009X^2$, $r^2 = 0.334$; $\sqrt{Y} = 2.90 + 0.0549X - 0.0007X^2$, $r^2 = 0.356$; and $Y = 15.33 - 0.184X$, $r^2 = 0.584$, respectively. All regression coefficients are statistically significant from zero, $P = 0.05$.

illustrated in orchard blocks where inadequate irrigation resulted in massive shuck decline caused by water stress. In the same orchard, shuck decline was not a problem in blocks with adequate irrigation (Sparks, 1992b). Yields of pecans in Georgia, in 1992, were below normal with favorable soil moisture resulting in few problems of shuck decline or nut quality. In 1993, a drought year, pecan yields were above normal. Good nut quality with minimal, if any, shuck decline occurred in orchards that had adequate irrigation or timely rains during kernel development. However, orchards with insufficient soil moisture from lack of either rain or adequate irrigation during kernel development sustained damage from shuck decline and poor quality nuts.

An extreme case of water-logging occurred in an orchard with heavy fruit set in southwest Texas during 1990 (Sparks, 1993a). Water-logged conditions, which are detrimental to pecan (Smith and Ager, 1988; Smith and Bourne, 1989), were created by excessive irrigation during most of the growing season followed by excessive rains (229 mm) during kernel development. Shuck decline was obvious long before fruit maturity occurred, resulting in reduced kernel quality. Pecan yields for 1991 were light, with good nut quality and little, if any, shuck decline. Yields were high in 1992, but in contrast to 1990, soil moisture conditions were more favorable for healthy fruit development. Shuck decline was not observed until near shuck dehiscence and with no detectable effects on kernel quality. Other workers (Schaller et al., 1968) also observed variable effects of shuck decline on kernel quality depending on the developmental stage of the kernel at the time of stress. Stress imposed at the middle of kernel development was detrimental to kernel quality, but late season stresses imposed near fruit maturity had little, if any, effect on quality.

The accentuation of fruiting stress by other accompanying

stress factors, i.e., drought, premature defoliation, water-logged soils, shady conditions, etc., will make it difficult to establish an absolute threshold of fruiting intensity at which shuck decline will not occur. Observations indicate that shuck decline is normally not a problem if stress factors other than fruiting are not in effect in nonprolific 'Desirable', 'Schley', and 'Stuart', the major cultivars in the southeastern United States. However, with prolific cultivars, such as 'Wichita', selective fruit thinning is apparently essential to prevent shuck decline induced by excessive fruiting as such.

In summary, decreasing tree stress by mechanical fruit thinning had a direct impact on fruit maturation by decreasing shuck decline and premature germination and increasing kernel quality. Thus, shuck decline, previously referred to as shuck disease, tulip disease, shuck die-back, or late season shuck disorders is due to excessive tree stress, as is poor kernel development associated with shuck decline. Onset of shuck decline should be treated as a problem of tree physiology and not a pathological problem, as fungal growth was not detectable microscopically until cellular breakdown was evident.

Literature Cited

- Brenneman, T.B. and C.C. Reilly. 1989. Recent occurrence of pecan anthracnose caused by *Glomerella cingulata*. Plant Dis. 73:775.
- Davis, J.T. and D. Sparks. 1974. Assimilation and translocation pattern of carbon-14 in the shoot of fruiting pecan trees, *Carya illinoensis* Koch. J. Amer. Soc. Hort. Sci. 99:468-480.
- Finch, A.H. 1937. The use of ethylene to improve pecan harvesting. Proc. Amer. Soc. Hort. Sci. 34:74-77.
- Finch, A.H. and C.W. Van Horn. 1936. The physiology and control of pecan nut filling and maturity. Arizona Agr. Expt. Sta. Tech. Bul. 62.
- Fisher, D.B. 1968. Protein staining of ribboned epon sections for light microscopy. Histochemie 16:92-96.
- Gallaher, R.N. and J.B. Jones. 1976. Total, extractable, and oxalate calcium and other elements in normal and mouse ear pecan tree tissues. J. Amer. Soc. Hort. Sci. 101:692-696.
- Gomez, K.A. and A.A. Gomez. 1984. Statistical procedures for agricultural research. Wiley, New York.
- Halliwell, R.S. 1968. Research on shuck disease. Proc. Southeastern Pecan Growers Assn. 61:149-150.
- Halliwell, R.S. and J. Johnson. 1972. Etiology of the pecan shuck disease. Phytopathology 62:762.
- Herr, Jr., J.M. 1982. An analysis of methods for permanently mounting ovules cleared in four-and-a-half type clearing fluids. Stain Technol. 57:161-169.
- Hotchkiss, M.W., C.C. Reilly, and B.W. Wood. 1993. Orchard management—Decisions affecting pecan diseases and nut quality. Proc. Southeastern Pecan Growers Assn. 86:87-91.
- Johansen, D.A. 1940. Plant microtechnique. McGraw-Hill, New York.
- KenKnight, G. 1968. Resistance of pecan to disease. Proc. Southeastern Pecan Growers Assn. 61:168-170.
- Kerrigan, J.L. 1993. Light and electron microscopic observations of *Colletotrichum gloeosporioides*, causal agent of pecan anthracnose. MS thesis. Univ. Georgia.
- Latham, A.J. and H.L. Campbell. 1991. Evaluation of selected fungicides for control of scab and anthracnose of pecans. Proc. Southeastern Pecan Growers Assn. 84:78-83.
- Payne, J.A., H.L. Malstrom, and G.E. KenKnight. 1979. Insect pests and diseases of the pecan. U.S. Dept. of Agr. ARM-S-5.
- Pearse, A.G.E. 1961. Histochemistry—Theoretical and applied. Little, Brown and Co., Boston.
- Reid, W., S.M. Huslig, M.W. Smith, N. Maness, and J. Whitworth. 1993. Fruit-removal time influences return bloom in pecan. HortScience 28:800-802.
- Reilly, C.C. 1989. Late season diseases of pecan. Proc. Southeastern Pecan Growers Assn. 82:67-70.
- Reilly, C.C. 1990. Major nut diseases of pecan. Proc. Southeastern Pecan

- Growers Assn. 83:123-128.
- Reilly, C.C. 1992. Pecan diseases and the 1991 season. Proc. Southeastern Pecan Growers Assn. 85:117-122.
- Reilly, C.C. and M.W. Hotchkiss. 1991. Pecan anthracnose and Phytophthora shuck and kernel rot are production limiting diseases. Proc. Southeastern Pecan Growers Assn. 84:98-102.
- Schaller, C.C. and G.E. KenKnight. 1972. Fungicides reduce the incidence of stem-end blight of *Carya illinoensis*. Plant Dis. Rpt. 56:276-281.
- Schaller, C.C., F.N. Dodge, and G.E. KenKnight. 1968. Increased occurrence of shuck disease of pecan (*Carya pecan*). Plant Dis. Rpt. 52:189-190.
- Sitton, B.G. 1931. A preliminary report on the relation of foliage to filling of pecan. Proc. Natl. Pecan Assn. Bul. 30:120-125.
- Smith, M.W. and P.I. Ager. 1988. Effects of soil flooding on leaf gas exchange of seedling pecan trees. HortScience 23:370-372.
- Smith, M.W. and R.D. Bourne. 1989. Seasonal effects of flooding on greenhouse-grown seedling pecan trees. HortScience 24:81-83.
- Smith, M.W., W. Reid, B. Carroll, and B. Cheary. 1993. Mechanical fruit thinning influences fruit quality, yield, return fruit set, and cold injury of pecan. HortScience 28:1081-1086.
- Sparks, D. 1966. A rapid method for estimating the leaf area of 'Stuart' pecan, *Carya illinoensis* Koch. HortScience 1:93-94.
- Sparks, D. 1990. Inter-relationship of precocity, prolificacy, and percentage kernel in pecan. HortScience 25:297-299.
- Sparks, D. 1992a. Pecan cultivars: The orchard's foundation. Pecan Production Innovations. Watkinsville, Ga.
- Sparks, D. 1992b. Stress factors affecting the Georgia pecan crop in 1991 and fruit set in 1992. Annu. Rpt. Northern Nut Growers Assn. 83:57-62.
- Sparks, D. 1993a. Growing pecans at low elevation in a hot climate. Annu. Rpt. Northern Nut Growers Assn. (In press.)
- Sparks, D. 1993b. Manejo de huertas de nuez pecanera en climas calidos enfasis en la germinacion prematura y apertura del ruzno. Conferencias Internacionales sobre el Cultivo del Nogal. 12:1-14.
- Sparks, D. and C.E. Brack. 1972. Return bloom and fruit set of pecan from leaf and fruit removal. HortScience 7:131-132.
- Spurr, A.R. 1969. A low-viscosity epoxy resin embedding medium for electron microscopy. J. Ultrastructure Res. 26:31-43.