ABSTRACT

RINIKER, STEVEN DOUGLAS. Variation Among Virginia Market-Type Peanut (*Arachis hypogaea*) Genotypes in Susceptibility to *Tomato spotted wilt virus* Vectored by Thrips (Thysanoptera: Thripidae). (Under the direction of Rick L. Brandenburg).

Tomato spotted wilt virus (TSWV), a thrips-vectored *tospovirus* is one of North Carolina's most important pathogens of peanut (*Arachis hypogaea* L.). Development of resistant cultivars remains one of the most promising methods to manage the disease. Thirty-two genotypes of virgina market-type peanut were monitored in field tests for TSWV incidence and severity during 2004 and 2005. Cultivar Gregory had a higher density of adult thrips than all other genotypes; breeding lines N01057 and N03054E had the least. Line N03036EJ had the greatest TSWV incidence but did not differ significantly from cultivars Gregory or Perry. Line N00033 had the least TSWV incidence and differed significantly from both Gregory and Perry. The occurrence of late-season chlorosis or peanut yellowing death (PYD) in North Carolina was confirmed to be highly associated with TSWV infection. Breeding line N02051ol (9) had the greatest incidence of PYD, but did not differ significantly from cultivars Gregory or Perry. Lines N03023EF and N01083 had the least PYD incidence and differed significantly from cultivars Gregory and Perry. Plants infected with TSWV but asymptomatic were found in greater abundance than TSWV-infected with symptoms for many of the genotypes. Line N03036EJ had the greatest proportion of infected but asymptomatic plants, having significantly more asymptomatic plants infected with TSWV than Gregory. Line

N03054E had the least number of infected but asymptomatic plants, having significantly less asymptomatic plants infected with TSWV than Perry. Cultivar NC-V 11 had significantly more thrips feeding injury and greater density of adult thrips than Perry. No correlation was found between thrips feeding damage or population densities and TSWV incidence, PYD incidence, or the number of TSWV-infected but asymptomatic plants. No significant differences were detected among genotypes for thrips damage ratings or virus severity ratings.

VARIATION AMONG VIRGINIA MARKET-TYPE PEANUT (*ARACHIS HYPOGAEA*) GENOTYPES IN SUSCEPTIBILITY TO *TOMATO SPOTTED WILT VIRUS* VECTORED BY THRIPS (*THYSANOPTERA: THRIPIDAE*)

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BIOGRAPHY

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INTRODUCTION

In 2005, Virginia, Texas, South Carolina, and North Carolina planted a total of 195,000 acres to virginia market-type-peanut (*Arachis hypogaea* L.), with North Carolina growing 48% of all virginia market-type peanuts (Brown 2006). Comparatively, in 2001, before the Federal Farm Bill peanut support program ended, 242,000 acres were planted to peanut in the United States. The average price per ton in 2005 was $425-475 for peanuts sold under contract and near $358 for peanuts sold without a contract, which was less than prices from 2003-2004, which were near $500 a ton (Brown 2006). Decreasing price motivates growers to more effectively manage insect and disease pests.

Tomato spotted wilt virus

Tomato spotted wilt virus (TSWV), the t*ospovirus* that causes spotted wilt of peanut (*Arachis hypogaea* L.) (Culbreath et al. 2003, Todd et al. 1990) and a contributing factor to the occurrence of peanut yellowing death (PYD) (Mitchell 1996), is a limiting factor in peanut production in North Carolina. *Tomato spotted wilt virus* was first reported to cause yield loss in peanut throughout Texas in 1984 (Black and Smith 1987). By the early 1990's TSWV was widespread in the southeastern United States (Pappu et al. 1999). In Georgia, TSWV infection was present in 100% of the sampled fields in a number of counties (Camann et al. 1995), with an average percent of infection of 2%, and by 1996, proportions of infected plants as high as 100% were detected in some areas (Brown et al. 1996).

Spotted wilt can severely limit yield of peanut (Baldwin and Williams 2002). Tomato spotted wilt epidemics on peanut in GA during 1997, the worst year on record, resulted in up to an estimated 86% of peanut plants infected in some areas (Culbreath et al. 1999b). This translated into an estimated $40 million of lost income for area growers (Baldwin and Williams 2002, Brown et al. 2005). *Tomato spotted wilt virus* was first reported on peanut in North Carolina in 1990, and by 1995 TSWV was present in all peanut production areas in the state (Garcia and Brandenburg 2000). The virus is known to infect peanut in all commercial production areas in North Carolina, and to be present in all counties (Lyerly et al. 2002). During 2005, TSWV infection was verified in nearly 50% of plants in experiment test plots (Brandenburg 2006).

Tomato spotted wilt virus also causes economically-limiting disease in a number of crops other than peanut. Tobacco (*Nicotiana tabacum* L.), tomato (*Lycopericon esculentum* L.), pepper (*Capsicum annum* L.), lettuce (*Lactuca sativa L.*) and chrysanthemum (*Chrysanthemum morifolium*), are crops that that can suffer economic yield loss under TSWV infection (Brown et al. 2001, Camann et al.1995, Chamberlin et al. 1992, Cho et al. 1987, Eckel et al. 1996, German et al. 1992, Jain et al. 1998, Magbanua et al. 2000, Pappu et al. 1999, Sakimura 1962b). Losses in susceptible crops can reach 50-90% (Cho et al. 1987).

TSWV has one of the largest host ranges of any plant virus and is able to infect over 500 species of plants (German et al. 1992, Whitfield et al. 2005). In North Carolina, TSWV was first detected in tomato and tobacco fields in 1988 (Cho et al. 1995), but incidence remained low for the next several years. However, by

1997 tobacco, tomato and pepper had suffered losses of about 25-50% in individual fields throughout the state (Groves 2001). An assessment of monetary losses attributed to TSWV has not yet been performed in North Carolina.

Detection of TSWV in plant tissue is based on observation of expressed symptoms, serology, molecular techniques, and the presence of inclusion bodies in peanut cells (Sherwood 1990). Commercially-produced DAS-ELISA (double antibody sandwich-enzyme-linked immunosorbent assay) assays are commonly used for routine diagnosis. However, the procedure has limitations. Assays of asymptomatic leaves from an infected plant may not test positive (Hoffmann et al. 1998). Also, dead plants or tissue may not test ELISA positive, as ELISA only detects activity-replicating virus (Magbanua et al. 2000). ELISA can also be used to test thrips for infection. However, adult thrips must be allowed time to clear their gut contents before testing, or resulting positives may be due to presence of virus in the ingested food rather than infection of the thrips (Chamberlin et al. 1993, Ullman et al. 1992).

Tomato spotted wilt virus is a member of the genus *Tospovirus* of the family *Bunyaviridae* (Ullman et al. 1996, 1995a, 1992, Wijkamp et al. 1995b). Virus particles are 80-110nm, spherical, and membrane bound (German et al. 1992). The RNA of TSWV is comprised of 3 segments; the S RNA encodes for viral nucleocapsid protein, and nonstructural protein (NSs); the M RNA encodes for the precursor of two glycoproteins (G1 and G2) and a nonstructural protein (NSm); and the L RNA encodes for viral polymerase (Ullman et al. 1995b). The virus is sap transmissible and transmitted in a persistent-propagative manner (Sakimura 1962b,

Ullman 1996, Ullman et al. 1993, Wetering et al. 1996, Wijkamp and Peters 1993), solely by thrips in the family Thripidae (Thysanoptera) (German et al. 1992, Peters et al. 1991).

Thrips

Members of Thripidae are haplo-dipoid. Males develop from unfertilized eggs; females are derived from fertilized eggs, but in the absence of males, females can be produced in some spp. by thelytoky (chromosome doubling). The life cycle is begun after an adult female inserts eggs into plant tissue; in the case of peanut females oviposit into terminal buds (Kresta et al. 1995, Todd et al. 1996). Eggs typically hatch in 3-5 days and larvae emerge and begin feeding. Two larval instars take about 3.6-12 days to complete before the insect enters a non-feeding pupal stage. The prepupal and pupal stages typically occur in the soil from which an adult emerges 2.5-13 days later under favorable conditions (Sparks and Riley). A generation takes about 20 days to complete, taking longer under cooler temperatures (Mound 1996).

Members of the Thripidae are highly polyphagous (Mound 1996), and feed on the leaves and flowers of a number of plant species (Whitfield et al. 2005). Thrips have high fecundity and short reproductive cycles (Whitfield et al. 2005) and population size can increase rapidly (Puche and Funderburk 1992), allowing thrips the potential to become economic pests. In peanut, heavy feeding damage can cause terminals to blacken and die, while mild feeding can cause scarring and leaf deformities (Mitchell et al. 1995).

Thrips cause economically-damaging levels of leaf damage and plant stunting to peanut in North Carolina (Barbour and Brandenburg 1994). Peak damage corresponds to the peak number of larvae rather than adults (Todd et al. 1995). Though an individual adult is more damaging than an individual larva (Mitchell et al. 1995), larvae are present in much higher densities than adults in peanut (Brecke et al. 1996). For this reason the majority of thrips feeding damage is attributed to larval feeding.

In areas outside of North Carolina, where virginia market-type peanuts are less predominant, peanut can be damaged by thrips feeding, but little if any yield loss is reported (Mitchell et al. 1995). In North Carolina, thrips achieve their peak density mid-May to early-June (Groves et al. 2003). The economic threshold for thrips feeding injury in North Carolina is 25% leaf damage (Brandenburg 2006). Systemic insecticides are typically applied in the furrow at planting to control thrips in North Carolina. This practice has reduced, if not eliminated losses, attributed to thrips feeding damage. The current concern of grower is the role of thrips in the transmission and spread of TSWV.

Symptom expression

Spotted wilt symptoms vary largely across species and even among genotypes within species. Typical symptom expression begins with chlorotic spots, which may develop into concentric rings or ringspots. General chlorosis and bronzing of foliage may also occur. As the disease progresses stunting and distortion of terminal foliage and reduction of growth commonly occurs (Hoffmann et al. 1998). The earlier the infection occurs, the more severe the symptoms typically

become (Culbreath et al. 1992a, Marois and Wright 2003). In peanut, early infection tends to be expressed in one of two ways: severe stunting or wilting with seedling death (Lyerly et al. 2002), or with some combination or progression of the occurrence of chlorotic spots, ringspots, mottling, silvering, chlorotic streaks, vein streaking, chlorosis, distortion of foliage, defoliation, stunting, wilting, bud necrosis, local lesions, and death is typical (Culbreath et al. 1992a, Lyerly et al. 2002, Peters et al. 1991, Shew 2006). Spotted wilt can also cause plants to set fewer pods with those that are set to be undersized (Culbreath et al. 1992a) and seeds to be discolored (Shew 2006).

In peanuts inoculated in the greenhouse, symptoms first become visually apparent approximately 13 days after infection occurs (Lyerly et al. 2002). Latency until symptom expression has been reported in TSWV-resistant cultivars of chrysanthemum (Broadbent and Allen 1995). This is important because it suggests visual inspection alone may not identify all infected plants. Some plants may also become infected with both TSWV and a *potyvirus,* such as *Peanut mottle virus*. In such cases, TSWV symptoms are usually the dominant symptoms expressed (Hoffmann et al. 1998).

In addition to spotted wilt of peanut, TSWV has been linked as a causal agent in another disease phenomenon. Peanut yellowing death (PYD) occurs in older plants under moist conditions about 80-120 days after planting (DAP) in Texas (Mitchell 1996). Symptoms are expressed in the form of foliar chlorosis and root necrosis (Culbreath et al. 1991) that leads to water stress, severe wilting, and rapid death of the plant (Mitchell 1996). Culbreath et al. (1991) showed the association

between TSWV infection and the disease. *Tomato spotted wilt virus* was detected in the roots of 90%, 70%, 92% and 88% of late-season chlorotic plants from experimental plots, while 0%, 0%, 32%, and 8% of asymptomatic plants from the same plots tested positive. A later study detected TSWV in the roots of 90% and 23% of chlorotic plants while 23% and 0% of asymptomatic plants tested positive (Culbreath et al. 1991).

Even though a strong association has been shown between PYD and TSWV infection (Culbreath et al. 1991), Mitchell (1996) determined that TSWV infection is not required in order for a plant to express PYD symptoms, but that TSWV infection can increase the severity of the condition. As the growing season progressed, increasing frequency of fungi were found in the vascular system of the roots of peanut plants. *Penicillum* spp, *Aspergillus niger* and *Fusarium* spp. were the predominant invading fungi found. The invasion of fungi suggests that TSWV may act as a stressor and that peanut becomes more susceptible to late season infection by fungal pathogens under moist conditions (Mitchell 1996). However, Culbreath et al. (1991), found *Fusarium* spp. equally frequent in both symptomatic and asymptomatic plants. Furthermore, fumigation has not been found to reduce the incidence of PYD (Mitchell 1996). There is difficulty in visually identifying PYD. Peanut yellowing death symptoms can be confused with Cylindrocladium black rot (CBR), which is a fungal disease caused by *C. parasiticum* that causes similar symptoms to PYD (Shew 2006) and is also present in peanut growing areas of North Carolina (Shew 2006).

Tomato spotted wilt virus is commonly found in the roots of asymptomatic peanuts plants (Mandal et al. 2001b). Culbreath et al. (1992b), reported that some samples that test ELISA positive for TSWV never show TSWV symptoms, and that the incidence of asymptomatic TSWV infected plants can be 2-3 times greater than the incidence of visually symptomatic plants. This is in agreement with Rowland et al. (2005), who reported that assays of below-ground parts have a higher incidence of infection based on ELISA positives than above ground parts. Curiously, the inocula from the roots of TSWV-infected but asymptomatic plants is not mechanically transmissible, but inocula from the roots of TSWV-infected and symptomatic plants is always mechanically transmissible (Mandal et al. 2001b).

Why some TSWV-infected plants express symptoms and others do not in unclear. A likely cause is an interaction between resistance mechanisms within the plant and environmental conditions. Temperature affects symptom expression and disease progression of TSWV in plants. In chrysanthemum cultivars, infection at cool temperatures (16-21C) resulted in greater levels of symptom expression than warm temperatures (18-42C) (Broadbent and Allen 1995). The difference of symptom expression across temperatures was 40-95% higher under cooler temperatures (Broadbent and Allen 1995). In tobacco, temperature appears to have the opposite effect. Tobacco plants express more severe symptoms at higher temperatures. Virus translocation is encouraged by temperatures around 29C, as compared to temperatures near 23C. This results in reduced movement, and instead an accumulation of TSWV in the inoculated leaves (Llamas-Llamas et al.

1998). Lower temperatures may favor replication rather than translocation (Hull 1989).

In TSWV-infected peanut, incubation at a temperature range of 30-37C resulted in more severe symptom expression than at 25-30C (Mandal et al. 2002). Plants grown at the higher temperature had localized symptom expression, reduced systemic infection, and restricted viral movement. ELISA assay results suggested that peanut grown at higher temperatures had reduced levels of TSWV antigen. Severity was reduced across temperature; lower temperatures produced more plants with full mosaics, while at higher temperatures ringspots were more commonly expressed. When considering breeding line C11-2-39 at 30-37C, 50-80% of symptomatic plant's roots tested negative for TSWV. This proportion is greater than the number of these plants that tested positive for TSWV under leaf assay. These data suggest that infection was localized and did not become systemic (Mandal et al. 2002).

Asymptomatic plants can be debilitated by TSWV infection. Rowland et al. (2005) reported infected but asymptomatic peanut plants have 30-51% reduced photosynthesis, and that transpiration and water efficiency is also significantly decreased. It has not been determined if yield is lost due to TSWV infected plants that remain asymptomatic.

Virus distribution does not appear to be uniform in the plant, but becomes more concentrated in young and developing terminal tissue (Kresta et al. 1995). Mandal et al. (2001b) reported that after peanut becomes infected with TSWV, the virus first moves to the roots and then the terminals. Using ELISA to assay peanut

plant tissue, Mandal found that antigen levels were initially highest in the roots but, as the plant was continued to grow, antigen levels in terminals became greater than roots. This is in agreement with data reported by Rowland et al. (2005).

Varying virus concentrations throughout the plant can play a role in symptom expression. Symptom expression and severity appear to be positively correlated to virus concentration (Kresta et al. 1995). An oddity is that some older symptomatic plants, plants generally over 90 days post inoculation (DPI), can have high antigen but not yield transmissible virus (Mandal et. al. 2001b).

TSWV is not known to be seed-borne (Garcia and Brandenburg 2000). The virus can be detected in parts of the pod, but it is not found in the embryonic cotyledons; thus peanuts grown from infected seed will not inherently be infected (Pappu et al. 1999).

Vector thrips

Thysanoptera is comprised of nearly 5000 species, of which only eight are confirmed vectors of *tospoviruses* (Ullman et al. 1995a). Of these only seven, all of which are members of the Thripidae are known to be competent vectors of TSWV (Camann et al. 1995**,** Mound 1996, Sin et al. 2005, Ullman et al. 1995a). Mound (1996) reviewed and assembled adult keys for the seven confirmed TSWV vectors: *Frankliniella fusca* (Hinds); *F. intonsa* (Trybom); *F. occidentalis* (Pergande); *F. schultzei* (Trybom); *Thrips palmi* Karny; *T. setosus* Moulton; and *T. tabaci* Lindeman (Mound 1996). *F. fusca*; tobacco thrips (TT), and *T. tabaci*; onion thrips (OT), are considered native to the Southeast and are found in peanut-producing areas (Sakimura 1963, 1962b, Todd et al. 1990). *F. occidentalis*, western flower thrips

(WFT), has more recently become endemic in areas where peanut is grown, for a total of three TSWV vectors extant in North Carolina (Barbour and Brandenburg 1994).

Vector competency

Numerous studies have reported no significant differences in vector competency. Eckel et al. (1996) reported no difference between WFT and TT in the ability to acquire or transmit TSWV. Assis et al. (2002) reported no detectable differences in replication or acquisition of TSWV between WFT and TT. Sakimura (1963, 1962a) reported no difference between TT and OT. However, OT's ability to act as a competent vector has been disputed. Wijkamp et al. (1995c) suggested that most OT populations are not competent vectors. Chatzivassiliou et al. (1999) confirmed that OT from a leek (*Allium porrum* L.) population could transmit TSWV. Numerous studies have reported that certain OT populations can no longer transmit contemporary isolates of TSWV (Nagata et al. 2004, 2002, Ullman 1996, Wijkamp et al. 1995a, 1995c).

Wing forms, color morphs, and gender have also been examined for association with vector competency. A number of thrips species develop different wing morphs: brachypterous (small winged), or macropterous (large winged) (Groves et al. 2001). No difference in vector ability was found between the morphs of TT (Sakimura, 1993, Wells et al. 2002b). An earlier study reported no difference in transmissibility for color morphs in WFT (Sakimura 1962a). Sakimura (1963, 1962a) also reported that there were no differences across gender. However, Wetering et al. (1999, 1998) reported significant difference across gender in regards

to efficiency of transmission, with males being more efficient vectors of TSWV than females.

Thrips ingest the contents of multiple cells per probe (Kindt et al. 2002). It has been suggested that males are more efficient transmitters. Individual female thrips are larger and feed in a more damaging manner than individual males. Damage to growing tissue results in a wrinkling effect as the cells fed upon die, while the cells around them continue to grow (Mitchell et al. 1995). Wetering (1998) states that males cause less scarring and suggests less scarring results in higher transmission efficiency, since TSWV needs living cells in which to replicate in to induce infection. Shallow feeding may result in higher levels of transmission (Sakimura 1962b).

Vector virus interaction

Knowledge about TSWV-thrips interaction has increased dramatically over the last decade. TSWV infects and replicates within its thrips vector (Ullman et al. 1993). The route of infection has been hypothesized to start when virons are ingested by juvenile thrips and come in contact with the epithelial tissue of midgut1 (Assis et al. 2002, Ullman et al. 1995a, Whitefield et al. 2005). From the epithelial cells the virus rapidly moves organ to organ and cell to cell as a viral-protein-RNA complex, rather than mature virons (Ullman et al. 1995b). The virus is hypothesized to move from midgut1 into midugut2 and the foregut (Assis et al. 2002), infecting the epithelial and visceral muscle cells of the gut lumen (Ullman et al. 1992, Whitefield et al. 2005). Finally, the virus infects the salivary glands (Whitefield et al. 2005), by way of a ligament tissue pathway (Assis et al. 2002), where it replicates (Wijkamp et

al. 1995b) and forms complete virons that can be transmitted to plants upon thrips feeding (Ullman et al. 1995b).

A haemocoel pathway into the salivary glands has also been proposed, but the failure for thrips to become viruliferous if injected with active virus suggests the ligament pathway theory is more probable (Nagata et al. 2002). The ligament pathway is further supported by Nagata et al. (2002), who found that TSWV could be found sequentially in the midgut epithelium, midgut muscles, ligament, and finally the salivary glands in WFT. In non-transmitting OT the infection process does not proceed into the ligament tissue. This helps explain both the manner in which TSWV infects thrips and the reason some OT populations have lost their ability to transmit. TSWV can replicate in non-transmitting species, but the virus is lost during pupation (Nagata et al. 2004).

An oddity in the vector-virus relationship of TSWV is that only adult thrips (Sakimura 1963 1962a, Ullman 1996, 1995b, Wetering et al. 1996) and late instar larvae (Peters et al. 1991, Sakimura 1963, 1962b, Wetering et al. 1996, Wijkamp and Peters 1993, Wijkamp et al. 1995a) that acquired the virus as first instars can transmit TSWV to plants. Second instar WFT larvae failed to acquire and retain the virus and could not transmit (Wetering et al. 1996). Adults were reported to be unable to acquire TSWV even when given prolonged access to infected material (Chamberlin 1993, Sakimura 1963, 1962b). However, Assis et al. (2004, 2002) showed that adults TT and WFT can be infected, but do not transmit the virus. This apparent inability of later instar thrips and adults to acquire and transmit TSWV has been attributed to development of the midgut barrier (Ullman et al. 1992). Ullman et

al. (1992) observed virons clumping in the cytoplasm of midgut cells and not disseminating beyond the epithelia in WFT. This suggests midgut cells can become infected but the virus is unable to spread to other areas of the host.

Virus has been detected in pupae (Chamberlin et al. 1993) and infectivity is not known to be impaired by pupation (Sakimura 1962b). TSWV is not transmitted transovarially; mother to offspring, and consequently each generation must acquire the virus (Barbour and Brandenburg 1994). Acquisition and inoculation have been reported to occur in periods as short as 5 minutes, though efficiency is known to increase with the length of the access period (German et al. 1992, Wijkamp and Peters 1993). Longer acquisition periods will increase the percentage of thrips that become infected (Sakimura 1962b). Transmission efficiency in adults is known to decrease with the age that the larvae acquire the virus, until the ability is lost as larvae progress into their second instar (Wetering et al. 1996).

Tomato spotted wilt virus may cause disease in plants but the presence of the virus also appears to have an impact on the thrips it infects, as well as thrips that feed on infected plant tissue. Ullman (1996) reported that *tospoviruses* have a negative impact on the fecundity and longevity of thrips hosts and suggested infection or altered host nutrition as the cause. Garcia et al. (2000) reported that thrips survival and reproduction is reduced on infected peanut when compared to non-infected plants. However, Groves (2001) and Groves et al. (2001) reported that thrips population did not differ on infected verses non-infected plants, though populations did vary across species of plants. In 2005, Stumpf and Kennedy (2005) reported that infected thrips fed on infected plant tissue on average take longer to

mature and become smaller adults than non-infected thrips. In contrast, non-infected thrips fed on healthy plant tissue on average take longer to develop than non-infected thrips reared on infected tissue (Stumpf and Kennedy, 2005). This study shows direct effects of TSWV on the thrips host as well as indirect effects of plant suitability on the thrips, suggesting that viral infection can have a negative impact on a thrips, but that plant host infection can make a plant become a more suited thrips host.

Vector acquisition

Once acquisition occurs, there is a latent period that varies across species. The latent period in TT is 9 days, while in WFT and OT latent periods last approximately 10 and 11 days, respectively (Sakimura 1962b). The mean latent period is 9.3 days and the range is 4-12 days. Sakimura (1963, 1962b) suggested that allowing a longer acquisition period would result in longer retention of vector virulence. Though the virus is often retained and transmitted continuously until insect death (Peters et al. 1991) sporadic transmission can occur (Sakimura1963, 1962b).

Todd et al. (1990) suggested a longer average latent period for TT, reporting that 10 days are needed for virus transmission to occur. Such discrepancies could be the result of varying temperature regimes used in experiments. The latent period in WFT becomes shorter at higher temperatures. A temperature of 20C results in a 171 hour (h) latent period, while a temperature of 27C necessitates only an 84 h latent period before transmission can occur (Wijkamp et al. 1995a, Wijkamp and Peters 1993). However, in the same study it was noted that the percentage of thrips

able to transmit decreased with increasing temperatures. It is hypothesized that this is because thrips mature faster than the virus infection can spread within the thrips (Wijkamp et al. 1995a, Wijkamp and Peters 1993).

Mechanical transmission

It is difficult to achieve a high rate of TSWV transmission using mechanical methods to inoculate peanut (Mandal et al. 2001a). *Tomato spotted wilt virus* has an *in vitro* longevity of about 1-4.5 h at 18C and 10 minutes at 42C (Sakimura 1962b). There is also, reportedly, a lack of correlation between results produced by mechanical and thrips transmission (Hoffman et al. 1998), possibly due to differences in virus titers between the two types of inoculation (Mandal et al. 2002). A lack of correlation between the two inoculation methods suggests resistance to mechanically-inoculated TSWV cannot be used as means to judge a plant's resistance to thrips transmission.

Repeated mechanical inoculation can result in an isolate losing the ability to be transmitted by thrips (Nagata et al. 2000). Glycoproteins (Ullman et al. 2005) have been determined to be necessary for thrips transmissibility (Sin et al. 2005). Free nucleocapsids will not infect thrips, but will continue to infect plants (Nagata et al. 2000). During successive mechanical inoculations in plants, M-RNA mutants that are unable to construct glycoproteins accumulate (Sin et al. 2005). These isolates lack the receptors needed for infection of thrips, so subsequent thrips to plant transmission cannot occur (Nagata et al. 2000).

Vector prominence

Three TSWV vectors occur in North Carolina. Thrips distribution and host selection varies among species. Groves et al. (2001) suggests WFT and TT are the most efficient vectors of TSWV in the Southeast. Eckel et al. (1996) suggests that transmission efficiency and occurrence makes WFT the prominent vector of concern on tomato and pepper, whereas TT is more important on tobacco. Similarly, though, TT, OT, and WFT occur in peanut producing areas of North Carolina; WFT and OT are collected only rarely from peanut (Garcia and Brandenburg 2000). Eastern flower thrips (*F. tritici*) also occasionally occur on peanut but are not known to transmit TSWV (Barbour and Brandenburg 1994). Emergence studies based in peanut agrosystems have found all thrips emerging from the soil to be TT (Barbour and Brandenburg 1994). Aerial trap collections suggest TT is the dominant vector in central and eastern North Carolina. Groves et al. (2003) reported 98% of TSWV vectors captured were TT, with WTF comprising the remaining 2%. An earlier report stated 96% of vectors were TT, while 2.5% and 1.5% were WFT and OT, respectively (Groves 2001). This supports Eckel et al. (1996), who reported WFT occur infrequently in eastern North Carolina. WFT, though non-native, is able to overwinter in North Carolina (Cho et al. 1995).

Tobacco thrips are by far the most abundant thrips found on peanut in the southeastern United States (Barbour and Brandenburg 1994, Brown et al. 1996, Cho et al. 1995). This also makes TT the most common and most important TSWV vector found on peanut in North Carolina (Barbour and Brandenburg 1994, Cho et al. 1995, Eckel et al. 1996). In Texas, TT adults were the most abundant vector

found on terminals and blooms, comprising 75% of the vector species; WFT was the only other vector of consequence (Lowry et al. 1995). Interestingly, only 2% of the collected thrips (101 of 4930) were actually infected with TSWV (Lowry et al. 1995). In Oklahoma, Mulder et al. (1991) reported 75% of thrips found in blooms were TT. In Florida, majority of the thrips found in terminals were immatures, but of the adults found >80% were TT, with the only other adult species of number being WFT (Brecke et al. 1996). Due to lack of sufficient larval keys, identifying larvae is difficult. However, it is known WFT reproduces poorly if at all on peanut, while TT can reproduce successfully (Chamberlin et al. 1992, 1993, Todd et al. 1995). Thus, we can assume the immatures found on peanut are TT. This is supported by Todd et al. (1995) who reported 484 of 486 immature thrips collected from peanut reared out to TT adults, with the remaining 2 rearing to WFT.

Tobacco Thrips

Tobacco thrips are known to overwinter mostly as brachypterous, adult females (Barbour and Brandenburg 1994, Cho et al. 1995), though smaller numbers of macropterous adults and larvae can also be found (Chamberlin et al. 1992, 1993). Since brachypterous thrips are unable to fly this suggests some thrips remain in the field during winter (Chamberlin et al. 1993). A number of these overwintering thrips (0-10% depending on location and sample date) have tested positive for TSWV infection (Chamberlin et al. 1993), suggesting thrips populations do not need to re-acquire TSWV infection for spring, as the thrips themselves act as transitional virus hosts. In North Carolina, TT produce a generation before peanut is planted by utilizing weed hosts (Cho et al. 1995, Groves 2001,) or cereal crops, reducing the

importance of overwintering thrips populations in the role or TSWV infection. The focus instead falls on the monitoring of subsequent generations (Barbour and Brandenburg 1994).

Peak TT flights occur in spring in mid to late May in North Carolina (Groves et al. 2003). This results in thrips populations peaking, while peanuts are still seedlings and in their most susceptible stage (Brown et al. 2001). Temperature can play a role in thrips population dynamics. The interval between generations is shortened under warmer temperatures (Broadbent and Allen 1995). Stumpf and Kennedy (2005) reported a positive correlation between larval survival and increasing temperatures over a range of 18.3-29.4C with TT. Though both wing morphs can exist at all times, it is more common to see more brachypterous forms in the winter and early spring and greater numbers of macropterous forms in late spring (Groves et al. 2001).

Distribution of thrips varies across types of peanut vegetation. Generally immatures are more common in terminals (Brecke et al. 1996, Tappan 1986) and adults are more common in flowers (Lowry et al. 1995, Mulder et al. 1991, Tappan 1986, Todd et al. 1995). Sampling time had no effect on the number of thrips found per terminal, and approximately 90% of thrips collected from terminals were immatures (Tappan 1986). Adults TT are the predominant stage found in flowers but the number of thrips collected from flowers is dependent on the time of day samples are taken (Tappan 1986). Peanut flowers are ephemeral (Kresta et al. 1995) and start to wilt around noon (Tappan 1986), which could explain why significantly more thrips were collected in flowers from 9-11am than later in the day. This is in agreement with Todd et al. (1995), who suggest adults are predominant in

flowers. Tappan (1986) suggested that thrips population in flowers and terminals are independent of each other, that thrips aren't lost from terminals to flowers or flowers to terminals. Adult female TT are more common than adult males (Mulder et al. 1991, Puche and Funderburk 1992).

Managing TSWV in the field

Since the 1990's efforts have been made to help growers reduce their potential losses due to TSWV infection in peanut. Some evidence of success can be found in the reduction of reported losses in Georgia, for which annual losses in peanut are reported as being below $5 million (Brown et al. 2005). A variety of mostly cultural techniques has been shown to have a beneficial impact.

Planting date has been shown to have a significant effect on TSWV incidence. Early and late plantings have higher incidences of infection than peanut planted in the middle of the typical planting period (Brown et al. 2005, 2001, 1996, Culbreath et al. 1999b, Hurt 2005). In North Carolina, peanuts are planted from about May 5 through June 5 (Jordan 2006), with the recommended planting date being after May 15th if TSWV is a concern (Brandenburg 2006).

Spotted wilt incidence is inversely related to seeding rate in peanut (Hurt et al. 2005, Wehtje et al. 1994). This does not necessarily mean fields with higher stands will have fewer infected plants, but that the proportion of infected to uninfected plants is higher in fields with lower plant densities. This relationship is evident when observing clusters of spotted wilt symptomatic plants (Culbreath et al. 1990) within certain areas of the field where "skips" occurred (Baldwin and Williams 2002). It should be noted that lower plant densities could result in more thrips per plant

(Brown et al. 2005, 2001, 1996, Culbreath et al. 1999b). Row spacing also has an impact on incidence. Twin row plantings with an interion spacing of 18-25cm have been reported to have 25-30% lower incidence than traditional single row plantings (91.4cm between rows) (Culbreath et al. 1999b). Twin row planting has also been reported to result in higher yields and better grades (Brown et al. 2005, 2001, 1996, Lanier et al. 2004), while also reducing weeds (Baldwin and Williams 2002). When attempting to encourage TSWV disease pressure for field experiments it is common to plant early season and at low plant populations (Culbreath et al. 1999a).

Targeting the vector with insecticides fails to control TSWV in the Southeast (Brown et al. 1996). Insecticides give poor control of immigrating adult thrips (Brown et al. 1996). Immigrating adults are the source of primary infection (Brown et al. 2006, Todd et al. 1990) and are believed to be the source of most TSWV infections (Brown et al. 2006, Camann et al. 1995). Several insecticides are recommended to control thrips and reduced feeding damage. Acephate, aldicarb, carbaryl, disulfoton, lambda-cyhalothrin, malathion and phorate have activity on thrips but do not kill rapidly enough to prevent virus transmission (Brown et al. 2005, Sakimura 1962b). However, phorate (Thimet 20G or Phorate 20G), an insecticide used to control thrips, also appears to stimulate a defense response in peanut that results in a lower incidence of infection (Gallo-Meagher et al. 2001). The exact mechanism is still unknown, but the reduction of incidence is not attributed to thrips suppression (Brandenburg 2006, Brown et al. 2005, 2001, Culbreath et al. 1999b, Hurt et al. 2003).

Peanuts planted with reduced or minimum tillage have less thrips feeding damage, compared to conventional tillage (Brandenburg et al. 1998). Reduced tillage has also been shown to reduce TSWV incidence (Brown et al. 2005, 2001, Hurt et al. 2003, Jordan et al. 2003). Johnson et al. (2001) reported 42% lower incidence of TSWV in reduced tillage systems compared to conventional tillage systems. Though conventional tillage has been reported as producing better yields when not under disease pressure (Jordan 2006), reduced tillage systems also offer the grower the advantage of reduced wind and water erosion (Johnson et al. 2001).

Cultivar selection has repeatedly been shown to have the greatest and most consistent impact on reducing incidence of and losses to TSWV (Brown et al. 1996, Marois and Wright 2003). Varietal resistance was originally attributed to non-preference by thrips (Black and Smith 1987). Thrips are believed to use a number of long and short range cues when selecting a host (Terry 1997). Since larvae cannot move significant distances, host selection is dependent on the adult female. Thus, the number of thrips larvae present on a particular host would act as a relative measure of that plant's suitability as a reproductive host (Terry 1997). Varying levels of thrips preference (antixenosis) or reproductive ability (antibiosis) (Terry 1997) would indicate resistance to TSWV could be gauged by counting thrips alone. However, field resistant cultivars, as identified by reduced incidence or severity of TSWV infection do not exhibit differences in thrips feeding preference or suitability as reproductive hosts (Brown et al. 2005, Culbreath et al. 2000, 1999a, 1997a, 1997b, 1996). Culbreath et al. (2000) detected no differences in the number of adult WTF or TT across peanut cultivars, and reported that differences in larval number

were inconsistent. Broadbent and Allen (1995) reported similar findings across chrysanthemum cultivars. This suggests that differences among cultivars are due to a differential response to TSWV infection, and not to thrips resistance (Wells et al. 2002a). However, a correlation between disease incidence and mean weekly thrips per month has been reported (Cho et al. 1987). This suggests that although counting thrips per plant at a specific location may not be an indicator of TSWV resistance, monitoring thrips populations can allow a location's general risk of TSWV infection to be estimated.

No peanut cultivar totally resistant to TSWV; resistance implies reduced incidence or severity (Brown et al. 2001). The emphasis placed on the development of TSWV resistant cultivars has increased over time. In a 2003 peanut cultivar selection guide, of eight virginia market-type peanuts mentioned, NC-V 11 was the only cultivar listed with any TSWV resistance (Huber 2002). In 2006, the same variety guide had a listing of ten virginia market-type peanuts, with Brantley, NC 7, and Perry being listed as TSWV-susceptible varieties, while Georgia Hl-O/L, and NC-V 11 were listed as resistant varieties (Huber 2006). This agrees with data presented by Lanier et al. (2004), who reported Perry with higher virus incidence than NC-V 11, and Hurt (2003) who reported that Perry which is resistant to CBR was more susceptible than Gregory. In North Carolina, VA 98R, NC-V 11, and Gregory tend to have lower TSWV incidence (Brandenburg 2006). Many factors need to be considered when selecting a variety; for example, Perry often has greater TSWV incidence than NC-V 11, but can produce greater yields than NC-V 11 under most conditions (Coker 2005, Lanier et al. 2004).

All currently available cultivars can suffer severe loses to TSWV. In 1996 a Spotted Wilt Risk Index for Peanut was created in Georgia (Brown et al. 2005). The index uses a point-based system and takes variety, planting date, density, insecticide use, row pattern, and tillage into consideration (Baldwin and Williams 2002). The index has gone through multiple revisions and is updated as new information becomes available (Brown et al. 2003). The index has been adapted to North Carolina and takes variety, planting date, plant density, insecticide use, and tillage into account (Brandenburg 2006).

Engineering resistance to TSWV

There is considerable research underway using molecular techniques to attempt to better understand the complexities of TSWV infection and to help confer resistance to TSWV in peanut. One of the biggest limiting factors in the progress of our molecular knowledge was that the polyphenols present in peanut tissue inhibit normal Reverse transcriptase-polymerase chain reaction (RT-PCR) (Jain et al. 1998). This difficulty has been overcome by using Immunocapture-RT-PCR (Jain et al. 1998). This allows gene locations to be identified in peanut, which enables gene transformations to be tracked with much less difficulty (Jain et al. 1998).

Peanut transformed to express the TSWV nucleocapsid gene, derived from the S-RNA (Nascimento et al. 2003b), were reported to not suffer systemic infection (Zhijian et al. 1997). Peanut transgenic for the nucleocapsid gene has been reported to express field resistance to TSWV (Yang et al. 2004). Magbanua et al. (2000) reported reduced symptom expression, with 76% of transgenic plants appearing asymptomatic, while only 42% of normal plants were asymptomatic

(Magbanua et al. 2000). Gene markers, as expressed sequence tags have been generated and placed into GenBank (Luo et al. 2005) and should help expedite future research.

North Carolina State University Peanut Breeding Program's TSWV Resistance Breeding Project examines a large number of peanut breeding lines each growing season. Thorough exanimations of the incidences of TSWV infection and severity across wide selections of genotypes has done much to assist future breeding accessions. However, not all aspects of TSWV infection have been examined in North Carolina.

There are a number of gaps in our understanding of the interactions between TSWV, peanut and vector thrips. The role thrips density plays in TSWV incidence and severity is unclear. Peanut yellowing death has only briefly been examined. The occurrence of PYD in North Carolina has yet to be determined across a range of genotypes. It is unknown whether any correlation exists between TSWV and PYD incidence, or if resistance to TSWV also confers resistance to PYD. The amount of underlying TSWV incidence has yet to be estimated across genotypes. The proportion of TSWV infected plants that show symptoms compared to those that remain asymptomatic has yet to be determine in North Carolina. In order to better manage TSWV efforts need to continue to be made to increase our understanding of these uncertainties.

Experiments were designed to evaluate the influence and interaction of peanut genotype selection, early season thrips populations, and feeding injury on the incidence and severity of TSWV infection in virginia market-type peanut grown in

eastern North Carolina. The objectives of the project were to 1) identify peanut lines with low incidence of TSWV or reduced severity of infection; 2) to examine the portion of TSWV-infected plants that remain asymptomatic across a large range of peanut genotypes; 3) to examine the occurrence of late season foliar chlorosis across peanut genotypes and determine the level of association it shares with TSWV infection; 4) to examine the interactions among thrips density, damage, and TSWV incidence and severity.

MATERIALS AND METHODS

Influence of Genotype and Thrips Density on TSWV Incidence and Severity (ALT)

Investigations were conducted within plots of the North Carolina State University Peanut Breeding Program's ongoing ALT trials. Breeding lines within the ALT trials were selected based on attributes for a purpose other than disease resistance; such as, high content of bright jumbo or fancy pods, or if they are high oleic.

Experiments were conducted in 2004 in field C7 at the Peanut Belt Research Station (PBRS) near Lewiston-Woodville, NC. In 2005 experiments were conducted at PBRS field A1. Field C7 contained soil type Rains sandy loam (fine-loamy, siliceous, thermic, typic paleudults) and Goldsboro sandy loam (fine-loamy, siliceous, thermic, aquic paleudults). In A1 soil types included Bonneau loamy sand (loamy, siliceous, thermic, arenic paleudults,), Norfolk sandy loam (fine-loamy, siliceous, thermic, typic paleudults), and Goldsboro sandy loam. Peanut was planted into conventional raised seed beds. Plots were two rows, 7.3m long and spaced 91cm apart, with a within-row seed spacing of 51cm. It is a common practice to increase plant spacing to encourage TSWV infection for experimental purposes (Culbreath et al. 1999a, Hurt et al. 2005). The experiments were planted on 15 May 2004 and 11 May 2005 using a custom planter equipped with John Deere (Moline, IL) Flexiplanter 20 planting unit equipped with Swanson "gatling gun" type seed-metering hoppers.

In 2004, 49 peanut lines were planted (Table 1), including two commonly used cultivars Gregory and Perry. Gregory and Perry are two lines that have differing levels of resistance to TSWV. Perry is considered highly susceptible to TSWV while Gregory is moderately resistant (Brandenburg 2006, Hurt et al. 2005). In 2005, 24 lines were planted and retested (Table 1). No insecticides were applied either year. The experiment was designed as a randomized complete block with three replicates. Aside from insecticide applications all plots were managed based on Cooperative Extension recommendations for the region (Jordan et al. 2006).

Thrips injury and damage rating

Thrips damage was recorded on 2 June and 17 June 2004 (18 and 33 DAP) and 5 June, 14 June, and 27 June 2005 (25, 34, and 47 DAP). Feeding injury was assessed by randomly examining 10 plants within each plot for evidence of thrips feeding on a recently-emerged quadrifoliolate. A plant was considered thrips-injured if the leaf that was examined had any scarring. Data were recorded as the number of damaged plants per plot. A thrips damage rating was also conducted in conjunction with the injury sampling. The damage rating system was designed to assess the general effect of thrips feeding on a specific peanut line. Plots were accessed using a simple visual rating system. While standing in front of the plot plants were visually scanned and the plot was subjectively scored in regards to the degree thrips feeding pressure has had negative impact. Three rating levels were defined: Three (3) was defined as a plot with a high degree of stunting; two (2) was defined as a plot with a moderate degree of stunting; and one (1) was defined as a plot with a minimal degree or absence of stunting that could be attributed to thrips

feeding. The number of injured plants per plot and the rating of each plot were recorded.

Thrips density

To evaluate thrips density, samples were collected on 2 June and 17 June 2004 (18 and 33 DAP), and on 5 June and 27 June (25 and 47 DAP) in 2005. Samples consisted of ten random, non-opened quadrifoliolates that were collected from each plot and placed immediately into 20 ml vials of 70% ETOH and refrigerated until processing. Samples were examined under a stereoscope and a complete count of tobacco thrips (TT) (Mound 1996) was performed. All thrips were removed from the leaf tissue and sorted according to life stage. The total numbers of adults and larvae per plot was recorded.

TSWV incidence and severity

In 2004, visual assessment of foliar TSWV symptoms occurred 29 June, 5 July, 13 July, 21 July, 27 July, 4 August, 10 August, 19 August, 31 August, and 9 September (45, 51, 59, 67, 73, 81, 87, 96, 108, and 117 DAP). In 2005, assessments occurred 7 July, 20 July, 2 August, 16 August, 27 August, 11 September, and 23 September (56, 69, 82, 96, 107, 122, and 134 DAP). On these dates, plants were examined for symptoms including chlorotic spots, ringspots, mottling, silvering, chlorotic streaks, vein streaking, chlorosis, distortion of foliage, defoliation, stunting, wilting, bud necrosis, and local lesions (Culbreath et al. 1992a, Lyerly et al. 2002, Peters et al. 1991, Shew 2006). A plant was considered symptomatic for TSWV if one or more leaflets exhibited any combination of the described symptoms. Observations were conducted by walking between the rows of

each plot and inspecting every plant. Any plant that was symptomatic was marked with a survey flag and immediately labeled, scored, and then data recorded. Labeling consisted of the date of flagging, and an alphanumeric identifier, in the format of: date (mm/dd), plot number (xxx), and serial, where the first plant in the plot is identified as “plant a” the second as “plant b” and the twenty-seventh as “plant aa.” Flags were placed adjacent to the main stems of plants.

Scoring consisted of rating the severity of symptoms from 3 to 1; where three (3) was defined as plants that were severely stunted or malformed; two (2) was defined as plants with widespread symptoms but no, or limited stunting; and one (1) was defined as plants with only mild and localized symptoms. Once a plant was flagged its symptom expression was reevaluated on each subsequent date to examine any changes in symptom severity over time. This system allowed the symptom progression of individual plants to be followed from the date symptoms were first observed until digging or death. For clarity, plant death was defined as a plant that was >80 necrotic or <20% “green.” When a flagged plant was recorded as dead, its flag was bent to a 45^{o} angle, and its rating was set at “3” for all subsequent dates. “Mourning” flags were left in the field to ensure that if a dead plant completely disintegrated its serial would still be held. The labeling system also ensured that if a flag were lost, the plant would be able to be re-flagged accurately without a lapse of data collection. The number of symptomatic plants per plot and the rating of each of those plants were recorded for each date.

For each date, after all plots were surveyed, foliar samples were collected from each newly-flagged plant. Samples consisted of at least three symptomatic

leaflets hand pulled from a symptomatic plant and placed into a plastic bag labeled identically as the flag marking the plant. Samples collected in this manner were processed the day of collection using the ImmunoStrip® assay from Agdia Inc. (STX 39300, ACC 00996, ACC 00925, Elkhart, IN). The assay is simple, efficient, and comparable to the DAS-ELISA produced by Agdia Inc. (Elkhart, IN). The kit consists of a plastic extraction pouch containing buffer lined with a plastic mesh. Samples were placed between the mesh and homogenized using a marking pen and the then the testing strip was inserted. The assay provides a very clear positive response in the form of two solid lines on the test strip if TSWV is present in the sample, opposed to only a single line if TSWV is absent. The assay was used to verify infection in all flagged plants. Flagged plants that did not test positive for TSWV infection during their initial assay were retested at 2 weeks intervals until infection was confirmed, the plant died, or the plant was dug. A positive or negative response was recorded for each flagged plant.

In September, visual scouting was expanded to include plants exhibiting severe foliar chlorosis or peanut yellowing death (PYD) (Mitchell 1996). In 2004, on 14 September and 23 September (122 and 131 DAP), and in 2005 on 2 October (143 DAP) the chlorotic plants in each plot were dug by hand. Taproot samples were collected by cutting the root off at the crown and again at least 2.5cm below the crown and placing the tissue into a plastic bag labeled with the plot number. The remainder of the plant was removed from the field. The number of samples per plot was counted, and processed the same day as collection using ImmunoStrip® assay

from Agdia Inc. (STX 39300, ACC 00996, ACC 00925, Elkhart, IN). The number of tested plants and the number of infected plants per plot was recorded.

In order to prepare the field for asymptomatic sampling all flagged plants that tested TSWV positive were manually removed from the field on 23 September 2004 and 2 October 2005 (131 and 143 DAP, respectively). All flags were also removed from the field at this time. Plants were dug and inverted mechanically using a commercial peanut digger and stand counts were taken on 5 October and 6 October (141 and 147 DAP) for 2004 and 2005, respectively. Stand was determined by counting the number of taproots per plot and adding in the number of plants that were previously removed. Once the plants were inverted there were no distinguishing markers in the field other than plot numbers. To assess the number of asymptomatic plants that were infected with TSWV, root samples, were collected as previously described from 10 randomly selected plants per plot on 7 October and 13 October (143 and 154 DAP) for 2004 and 2005, respectively. The samples were refrigerated no more than 72 h until processed using ImmunoStrip® assay from Agdia (STX 39300, ACC 00996, ACC 00925, Elkhart, IN). The number of infected plants per plot was recorded.

In September 2004, several previously asymptomatic plants began to express foliar chlorosis and severe wilting that progressed to necrosis within a period of approximately 2 weeks (personal observation). This is atypical of *Tomato spotted wilt* disease on peanut in North Carolina. Leaf tissue from chlorotic plants tested negative for *Tomato spotted wilt virus* (TSWV) using the ImmunoStrip® assay (Agdia Inc. STX 39300, ACC 00996, ACC 00925, Elkhart, IN). However, a large proportion

of samples collected from the root tissue of the same chlorotic plants tested positive for TSWV. In 2004, 92% and in 2005, 97% of chlorotic plants tested positive for TSWV infection. The observed symptoms and assay results suggested that the plants were suffering from peanut yellowing death disease (PYD) as defined by Mitchell (1996), which is attributed, in part, to TSWV infection. One focus of this experiment was to examine differences in the number of TSWV symptomatic-infected plants among genotypes. To determine if PYD can be considered a symptom of TSWV infection in North Carolina, the proportion of TSWV-infected plants exhibiting PYD symptoms was compared to the proportion of TSWV-infected asymptomatic plants. Proportions were compared using the general models procedure (SAS 9.1.3, SAS Institute Inc., Cary, NC) with logit link and PSCALE options enabled. By comparing the proportion of PYD plants infected with TSWV to the underlying TSWV infection level throughout the field, it was possible to confirm that PYD symptoms could be considered a late-season symptom of TSWV infection (Table 2) in both years. Therefore, "TSWV incidence" was comprised of plants that exhibited symptoms of either spotted wilt or PYD and when assayed were verified to be infected with TSWV.

Influence of Genotype and Thrips Density on TSWV Incidence and Severity (DAT)

Investigations were conducted within plots designated for North Carolina State University Breeding Program's ongoing DAT trials conducted at the Peanut

Belt Research Station (PBRS) near Lewiston-Woodville, NC. Breeding lines within the DAT trials were selected based on their potential for disease resistance.

No insecticide treatments were to be applied to any of the experiments conducted in fields C7 and A1. However, in 2005 seven lines were mistakenly treated with an insecticide. Lines N98003, N99103ol (9), N00090ol (7), N02020J, N02060ol (Per), N03004F, and N03005J were treated with 1.18 kg ai/ha of Temik 15G insecticide (aldicarb, Bayer CropScience LP, Research Triangle Park, NC) in-furrow at planting and 5.33 kg ai/ha of Ridomil Gold EC fungicide (mefenoxam, Syngenta Crop Protection, Greensboro, NC). All other management and experimental practices were constant across tests. Due to the non-consistency of this test as compared to the ALT experiment, data have been segregated and analyzed separately.

Statistical analysis

Data were pooled across years for all variables except thrips damage rating for which data were drawn only from the 2004 ALT experiment and the 2005 DAT test. Thrips data were separated and analyzed based on sampling date (2 June 2004 and 5 June 2005; 17 June 2004 and 27 June 2005). TSWV incidence data were drawn from the final sampling dates (9 September 2005 and 25 September 2006). Statistical analyses were performed at the 5% Type I error rate (α) based on the appropriate transformations using the SAS 9.1.3 (SAS Institute Inc., Cary, NC) general linear model (GLM) procedure for ANOVA. Thrips densities were transformed to square-roots. Virus incidences were transformed into proportion

infected and then subjected to angular transformation. Transformed means are reported. Thrips injured plants, and thrips damage ratings did not require transformation. Fisher's protected least significant difference (LSD) values were calculated for comparison of genotypes. Pearson's correlation coefficients were calculated to examine the correlations among virus incidences, and thrips densities, and damage across lines.

Influence of Cultivars Perry and NC-V 11 on Thrips Density and TSWV Incidence and Severity

In 2005, experiments were conducted at North Carolina on research farms located at the Peanut Belt Research Station (PBRS) near Lewiston-Woodville and the Upper Coastal Plain Research Station (UCPRS) located near Rocky Mount, and on private farms located on US route 76 near Cerro Gordo in Columbus County, Taylor Town Road near Faison in Duplin County, and on Big Oak Road near Bethel in Pitt County. The field at PBRS contained soil type Rains sandy loam (fine-loamy, siliceous, thermic, typic paleudults) and Goldsboro sandy loam Goldsboro sandy loam (fine-loamy, siliceous, thermic, aquic paleudults). Soil at UCPRS and Duplin County was a Goldsboro sandy loam. The Columbus County site contained soil series Wakulla (sandy, siliceous, thermic, psammentic hapludults) and Pitt County was soil series Exum (fine-silty, siliceous, thermic, aquic paleudults). Peanut was planted on raised, conventional seed beds. At PBRS and UCPRS plots were two rows, 9.1m long and spaced 91cm apart, with a within-row spacing of 7.62cm to achieve a seeding rate of 120 seed per row. At Columbus, Duplin, and Pitt Counties

plots were 12.2m long with a seeding rate of 160 seed per row. Peanut was planted on 2 May 2005 at PBRS and 24 May 2005 at UCPRS. Peanut was planted at Columbus County on 17 May 2005, Duplin County 10 May 2005, and at Pitt County 13 May 2005. All locations received 1.32 kg ai/ha of aldicarb (Bayer CropScience LP, Research Triangle Park, NC) in furrow at planting. All other production and management decisions were based on Cooperative Extension recommendations for the region (Jordan et al. 2006).

Plots consisted of two cultivars, Perry and NC-V 11. NC-V 11 has some field resistance to TSWV, whereas Perry is highly susceptible (Brandenburg 2006). The experiment was designed as a randomized complete block with four replicates.

Thrips injury and damage rating

Thrips feeding injury was recorded on 5 June, 14 June, and 27 June (34, 43, and 56 DAP) for PBRS. Thrips feeding injury was surveyed at UCPRS and Pitt County on 14 June and 1 July (21 and 38 DAP, 32 and 49 DAP, respectively) and Columbus and Duplin Counties 8 June, 15 June, and 22 June (22, 29, and 36 DAP and 29, 36, and 43 DAP, respectively). Feeding injury was assessed by randomly examining 20 plants within each plot for evidence of thrips feeding on a recently-emerged quadrifoliolate. A plant was considered thrips-injured if the leaf that was examined had any scarring. Data were recorded as the number of damaged plants per plot. A thrips damage rating was also conducted in conjunction with the injury sampling. The damage rating system was designed to assess the general effect of thrips feeding on a specific peanut line. Plots were accessed using a simple visual rating system. While standing in front of the plot plants were visually scanned and

the plot was subjectively scored in regards to the degree thrips feeding pressure has had negative impact. The rating levels defined as previously described were used to assess overall plot health. The number of injured plants per plot and the rating of each plot were recorded.

Thrips density

To evaluate thrips density, samples were collected on 5 June and 27 June (34 and 56 DAP), and 14 June and 1 July (21 and 38 DAP) for PBRS and UCPRS, respectively. Samples were collected at Columbus County on 15 June and 22 June (29 and 36 DAP), Duplin County on 8 June and 22 June (29 and 36 DAP), and at Pitt County on 14 June and 1 July (32 and 49 DAP). Ten random, non-opened quadrifoliolates were collected from each plot and processed as previously described. The total numbers of adults and larvae per plot was recorded.

TSWV incidence and severity

Visual assessment of foliar TSWV symptoms was conducted at PBRS on 7 July, 20 July, 2 August, 16 August, 27 August, and 11 September (66, 79, 92, 106, 117, and 132 DAP). Virus severity ratings were recorded in conjunction with assessments on 2 August and 11 September (92 and 132 DAP). UCPRS was evaluated on 1 July, 12 July, 20 July, 2 August, 16 August, 27 August, 11 September (49, 57, 70, 84, 95, 110 DAP), and rated on 12 July and 11 September (70 and 110 DAP). Columbus and Duplin Counties were assessed on 13 July, 27 July, 12 August, 20 August, and 3 September (57, 71, 87, 95, and 109 DAP and 64, 78, 94, 102, and 116 DAP, respectively), and rated on 27 July and 3 September (at 71 and 109 DAP and 78 and 116 DAP, respectively); Pitt County was assessed on

12 July, 20 July, 2 August, 16 August, 27 August, 11 September (60, 68, 81, 95, 106, and 121 DAP), and rated on 2 August and 11 September (81 and 121 DAP). On these dates plants were examined for expressed symptoms as previously described. The number of symptomatic plants was tallied and recorded for each plot. Virus severity ratings were performed twice at each location. In addition to counting the number of symptomatic plants per plot, symptomatic plants were divided into ranked categories as previously described on these same dates. The number of symptomatic plants in each category was also recorded for each plot.

Plants were dug and inverted mechanically using a commercial peanut digger. Peanuts were inverted at PBRS on 28 September (149 DAP), UCPRS on 12 October (141 DAP), Columbus County on 27 September (133 DAP), and Duplin and Pitt Counties on 30 September (143 DAP and 140 DAP, respectively). Stand counts were taken on 1 October (152 DAP) and 13 October (142 DAP) for PBRS and UCPRS, respectively. Stand counts were taken at Columbus County on 27 September (133 DAP); at Duplin County on 5 October (148 DAP); and Pitt County on 1 October (141 DAP). Stand was determined at Columbus County by counting the number of taproots in a 3.04m row section and using the results to estimate stand for the entire plot by multiplying. Stand was determined at Duplin County by counting the number of taproots for one row and using that count to estimate the stand for both rows by multiplying by two. Stand was determined at PBRS, UPCRS, and Pitt County by counting every taproot in the plot. Once inverted there were no distinguishing markers in the field other than plot number.

To access the number of asymptomatic plants taproot samples were collected on 1 October (152 DAP) from PBRS, 13 October (142 DAP) from UPCRS, 27 September (133 DAP) from Columbus County, 5 October (148 DAP) from Duplin County, and 1 October (141 DAP) from Pitt County. Samples were collected from 10 randomly selected plants per plot by cutting the root off at the crown and again at least 2.5cm below the crown and placing the tissue into a plastic bag labeled with the plot number. The samples were refrigerated for no more than 72 h until processed using ImmunoStrip® assay from Agdia (STX 39300, ACC 00996, ACC 00925, Elkhart, IN). The number of infected plants per plot was recorded.

Statistical analyses were performed at the 5% Type I error rate (α) based on the appropriate transformations using the SAS 9.1.3 (SAS Institute Inc., Cary, NC) general linear model (GLM) procedure for ANOVA. Thrips densities were transformed to square-roots. Virus incidences were transformed into proportion infected and then subjected to angular transformation. Transformed means are reported. Thrips injured plants, and thrips damage ratings did not require transformation. Fisher's protected least significant difference (LSD) values were calculated for comparison of genotypes

RESULTS AND DISCUSSION

Influence of Genotype and Thrips Density on TSWV Incidence and Severity (ALT)

The main effect of year was significant for densities of adult thrips and larval thrips densities (Table 3) and asymptomatic TSWV incidence (Table 4). Plants in 2005 had 2.1x greater adult densities than in 2004; plants in 2004 had 1.5x greater larval densities than in 2005, and plants in 2005 had 3.7x higher asymptomatic TSWV incidence than 2004 (data not shown). There were no significant year-by-genotype interactions (Table 3; 4). Therefore, data from 2004 and 2005 were pooled prior to analysis, and pooled means were used for genotype comparisons. The main effect of genotype was significant for adult density (sample1) (Table 3), the percentage of symptomatic plants infected with *Tomato spotted wilt virus* (TSWV incidence), the percentage PYD symptomatic plants infected with TSWV (PYD incidence), and the percentage of asymptomatic plants infected with TSWV (asymptomatic incidence) (Table 4). No differences were detected among genotypes for thrips damage ratings (Table 5), thrips injury, larval density (Table 3), or the percentage of TSWV infected plants at particular severity ratings (sev1, sev2, or sev3) (Table 4).

Thrips injury, damage, and density

No differences across genotype were detected for thrips injury or thrips damage ratings. However, the main effect of year had significant impact on thrips densities (Table 3). A total of 22,284 *F. fusca* were collected and sorted based on

their maturity (data not shown). On 2 June 2004, 941 adult thrips (8%) and 10,570 larvae thrips (92%) were collected, and on 17 June 2004 30 adult thrips and 786 larvae thrips were collected. On 5 June 2005, 2068 adult thrips (23%) and 7018 larvae thrips (77%) were collected, and on 27 June 2005 171 adult thrips and 600 larvae thrips were collected. This is in agreement with Groves et al. (2003), who found tobacco thrips reach their peak density in North Carolina in late May and early June. Peanut is considered most susceptible while in the seedling stage (10-20 DAP) (Brown et al. 2001). The earlier sample dates (2 June 2004 and 5 June 2005) fall within this interval more so than the later dates (17 June 2004 and 27 June 2005). The high thrips densities observed during the earlier sample dates and the overlapping of those dates with the seedling stage of peanuts make the earlier sample dates more relevant to TSWV incidence than the later dates. If combined, the data from the early counts would depreciate the data from the late counts. Therefore, thrips density was divided into two separate groups before analysis. Data from 2 June 2004 and 5 June 2005 were pooled together as sample1 and data from 17 June 2004 and 27 June 2005 were pooled together as sample2. When analyzed separately the sample2 counts are not significant for either adult thrips density or larvae thrips density, while the sample1 counts have significant difference among genotypes for adult density (Table 3).

Across years significant variation was detected among genotypes for adult density (sample1). Gregory, a commonly grown cultivar had the highest density of adult thrips (33.2), while genotypes N01057 (9.4) and N03054E (10.4) had the lowest densities (Table 6; Figure 1). The overall mean across all genotypes was

19.3. The strong genotype effect suggests that adult *F. fusca* may exhibit host preference in North Carolina during the stage in which peanut is most susceptible to TSWV infection (Brown et al. 2001). Since TSWV is transmitted almost entirely by adult thrips, genotypes that have higher densities of adult thrips could be at higher risk for TSWV infection than genotypes with lower densities. However, previous research suggests that this assumption may not be true. Hurt (2003) reported that Gregory had more feeding damage and lower TSWV incidence than Perry. Perry is considered highly susceptible to TSWV (Huber 2006), yet Perry has lower thrips densities and feeding damage than cultivars that are considered resistant (Hurt 2003). Conversely, Gregory is considered a resistant cultivar (Brandenburg 2006, Huber 2006), yet had the highest adult thrips density of any of the genotypes tested in these experiments and significantly more adult thrips than Perry (Table 6). Therefore, thrips densities did not necessarily result in high TSWV incidence.

Using Pearson's correlation coefficients (N=144), the relationships between the following pairs of data were examined: TSWV incidence and thrips injury (r=-.1067, *p*=0.2030); TSWV incidence and thrips damage rating (r=0.0059, *p*=0.9444); and TSWV and adult density sample1 (r=0.1072, *p*=0.2008). None of the pairs had a significant correlation. These results suggest that TSWV incidence cannot be accurately predicted across a wide variety of genotypes using thrips density (Figure 2) or feeding injury data.

All thrips damage ratings on 5 June and 27 June 2005 were scored as "1" (data not shown). Thrips damage ratings were designed to measure damage intensity. In 2005, ratings were taken later in the growing season than during 2004.

Peanut had already recovered from injury at this date. For this reason thrips damage rating analysis is drawn from 2004 data alone, (Table 5) as all ratings were identical during 2005.

TSWV incidence and severity

Across years and genotypes a total of 525 TSWV infected plants were confirmed of 559 symptomatic plants tested. Of those plants symptomatic and infected with TSWV, 369 (70%) were diagnosed as suffering from spotted wilt of peanut; the remaining 156 (30%) plants were diagnosed as suffering from PYD (data not shown). The main effect of genotype was significant for TSWV incidence (Table 4). Genotype N03036EJ had the highest TSWV incidence (32.2%). Perry (30.2%), a commonly grown cultivar had the second highest TSWV incidence, while genotype N00033 (1.5%) had the lowest TSWV incidence (Table 6). The mean incidence of TSWV across all genotypes was 13.8%. Perry had significantly greater TSWV incidence than Gregory. This is in agreement with past research conducted in North Carolina (Hurt 2003). Even though Gregory had the highest adult thrips density and a significantly higher adult density than Perry, Perry had the second highest TSWV incidence overall (Table 6). These data suggest that higher adult thrips densities do not account for higher incidence of TSWV (Figure 1; 2). Gregory is considered to be one of the more resistant cultivars grown in North Carolina (Brandenburg 2006, Huber 2006). Across all genotypes only line N00033 had significantly less TSWV incidence than Gregory (Table 6). These data serve to further reinforce Gregory remaining a robust cultivar choice in areas throughout the Carolina region under high TSWV pressure.

In 2004, 79 (92%) of the PYD symptomatic plants tested positive for TSWV infection, while in 2005, 77 (97%) of the PYD symptomatic plants tested positive for TSWV (data not shown). Analysis confirmed that PYD is associated with TSWV infection (Table 2), which is in agreement with past research (Culbreath et al. 1991). The main effect of genotype was significant for PYD incidence (Table 4). Genotype N02051ol (9) (12.7%) had greater PYD incidence than other genotypes, while genotypes N01083, N03023EF, and N01054 had the least (0.2%) closely followed by line N00033 (Table 6; Figure 3). The overall mean across genotypes was 3.2%. Although, genotype N03036EJ and the cultivar Perry had the greatest TSWV incidences, they did not have the greatest PYD incidence (Figure 3). However, genotype N00033 which had significantly less TSWV incidence than all other genotypes also had one of the lower PYD incidences. Perry was not significantly different than Gregory in PYD incidence (Table 6).

The etiology of PYD has not been clearly elucidated. Only a few research studies have been published (Culbreath et al. 1991, Mitchell 1996), and none that have investigated the occurrence of PYD in virginia market-type peanut grown in the Carolina region. Research conducted in Texas suggests that PYD is caused in part by fungi that invade the vascular system of the root of peanut late in the season, often after a rain event (Mitchell 1996). However, other research has detected no significant difference in the amount of fungi among PYD symptomatic and asymptomatic peanuts (Culbreath et al. 1991). Fungi disrupting the vascular system could help to explain the rapid wilting that is observed. PYD symptoms first appear in North Carolina in mid September, which corresponds to decreasing temperatures

(personal observations). It is possible TSWV acts as a stressor that causes infected peanut plants to become more susceptible to fungal organisms. Regardless of whether PYD is caused solely by TSWV infection or some combination of stressors, these data (Table 2) strongly suggest that PYD can be considered as a symptom of TSWV infection within virgina market-type peanut and that PYD can make up a large proportion of the total TSWV incidence found in a field. These data (Table 6) indicate that genotypes have varying resistance to PYD, and that genotypes resistant to TSWV will have lower PYD incidence than genotypes that are not resistant. Further research is required to fully understand the factors that cause PYD in peanut.

In 2004, 10% of asymptomatic plants were determined to be infected with TSWV; in 2005, 40% of asymptomatic plants were infected (data not shown). The overall mean across genotypes was 20.3%. The main effect of genotype was significant for asymptomatic incidence (Table 4). Genotype N03036EJ had the greatest asymptomatic incidence (48.9%), closely followed by line N01057 (47.4%). Genotypes N03054E (5.1%), N03023EF (5.6%), and N00033 (5.9%) had less asymptomatic incidence than all other genotypes (Table 6; Figure 4). These data suggest it is common for a high proportion of infected plants to never show symptoms within a growing season; the number may be higher than the number of infected plants that actually express symptoms (Figure 4). This is in agreement with past research in other states (Culbreath et al. 1992b, Mandel et al. 2001b).

Interestingly, a significant correlation was detected between TSWV incidence and asymptomatic incidence ($r=0.4328$, $p=0.0001$). This moderate correlation

suggests that genotypes that have more symptomatic infected plants also have higher incidences of asymptomatic infected plants (Figure 5). This suggests lines susceptible to TSWV are actually more likely to become infected, rather than just more likely to express symptoms if infected.

These data indicate that high proportions of infected but asymptomatic plants occur within virginia market-type peanuts grown in North Carolina in some years. These data suggest that genotypes with lower TSWV incidence, such as N00033 and Gregory (Figure 4), also have lower asymptomatic incidence, suggesting that those genotypes are actually less likely to become infected with TSWV rather than simply better able to tolerate infection. The reason some TSWV-infected plants never express visual symptoms has yet to be elucidated. The strong difference detected among years (p=<.0001) suggests that environmental conditions could play a role in development of symptoms. Further research needs to be conducted in order to better identify the factors affecting symptom expression and tolerance to TSWV infection.

The main effect of genotype did not have significant impact on severity of infection (Table 4). The number of plants that were severely stunted or malformed or became necrotic (sev3) did not differ among genotypes. Less severe infections (sev2 and sev3), also did not differ among genotypes. Past research has shown that any plant infected with TSWV is to some degree debilitated (Rowland et al. 2005). Thus it remains important to continue to identify such plants as infected.

TSWV incidence was assessed over time to show disease progression in the field. During 2004 (Figure 6) and 2005 (Figure 7) TSWV incidence increased

gradually until leveling off in the middle of August. Another increase in incidence seen in September was attributed to the onset PYD symptoms. A similar trend can be seen in the DAT trial (Figure 8).

Influence of Genotype and Thrips Density on TSWV Incidence and Severity (DAT)

In 2005, all thrips damage ratings were scored as "1" (data not shown). Plants were mistakenly treated with 1.18 kg ai/ha of Temik 15G insecticide (aldicarb, Bayer CropScience LP, Research Triangle Park, NC) in-furrow at planting. Aldicarb is recommended to control thrips feeding damage (Brandenburg 2006). The effect of insecticide treatment explains why little thrips damage was detected. As all ratings were identical analysis was not performed. All other parameters were examined. The main effect of genotype was significant for larval density (Table 7). No differences were detected among for the remaining parameters (Table 7; 8). Genotype N02020J (35.8) had greater larval density than the other genotypes. Genotype N02060ol (Per) (10.6) had the lowest larval density but differed only from N02020J (Table 9). The overall mean among genotypes was 17.8.

Influence of Cultivars Perry and NC-V 11 on Thrips Density and TSWV Incidence and Severity

The main effect of location was significant for thrips injury, adult and larval thrips densities (Table 10), TSWV incidence (Table 11) and asymptomatic incidence (Table 13). Pitt (75.9%) and Columbus (64.1%) County plots had greater thrips injury than other locations (data not shown). Columbus County (5.9) plots had greater adult density than other locations (data not shown). Columbus County (19.1) trials had greater larval density than other locations. Duplin County (9.2) and Pitt County (6.8) plots had greater larval density than plots at PBRS (2.7) and UCPRS (2.5). Trials at Duplin County (1.8%), Pitt County (1.2%) and Columbus County (0.9%) had greater TSWV incidence than plots at UCPRS (0.3%) and PBRS (0.2%). No location-by-cultivar interaction was detected for thrips injury, larval or adult densities (Table 10), or TSWV incidence (Table 11). Therefore, data were pooled across locations for these parameters. However, a location by cultivar interaction was detected for asymptomatic incidence (Table 13), requiring data for asymptomatic incidence to be analyzed separately among locations (Table 14).

No difference among cultivars was detected for larval density (Table 10), or virus severity ratings (Table 16). No difference in TSWV incidence was detected for the main effect of cultivar when data were pooled across weeks (Table 11), nor if data are separated and analyzed over weeks (Table 12). The overall mean for TSWV across locations was 0.9%; the overall virus pressure across all locations was low. The main effect of cultivar was significant for thrips injury (Table 10). Cultivar NC-V 11 (55.6%) had greater thrips damage than cultivar Perry (43.3%). The

overall mean across cultivars was 49.4%. The main effect of cultivar was significant for adult density (Table 10). Cultivar NC-V 11 (4.2) had greater adult density than cultivar Perry (2.0). The overall mean across cultivars was 3.1. When analyzed among locations the main effect of cultivar was significant for asymptomatic incidence at Columbus and Pitt County and UPCRS plots (Table 14). Within Columbus County and UCPRS, cultivar Perry had greater asymptomatic incidence than cultivar NC-V 11 (Table 12). However, at Pitt county cultivar NC-V 11 had higher asymptomatic incidence (Table 15). No significant difference in asymptomatic incidence was detected for other locations (Table 12; 15). The main effect of cultivar was not significant for virus severity (Table 16).

When comparing TSWV incidence to asymptomatic incidence across locations (Figure 9) no correlation is apparent, thus one must assume that asymptomatic incidence varied among locations due to environmental influences. Duplin County does not have a history of peanut production as the other experimental locations do and was not brought into production until after 2001. It should be noted that if Duplin County is removed as an outlier, the R^2 value increases from 0.0358 (Figure 9) to 0.8914 (data not shown). Further research will be necessary in order to better understand the relationship between infected plants that become symptomatic and plants that do not.

TSWV was assessed over time to show disease progress throughout the season. At Columbus (Figure 10), Duplin (Figure 11) and Pitt Counties (Figure 13) TSWV incidence was highest around 70 DAP and then became less apparent. At PBRS (Figure 12) TSWV incidence increased throughout the entire season except

for the last sampling date for which no virus was detected in cultivar NC-V 11. UCPRS (Figure 14) was different in the fact that instead of gradual changes over time sudden spikes in TSWV incidence were found that quickly dissipated.

These data suggest that in areas with low virus pressure little difference exists between cultivars Perry and NC-V 11. Past research has shown that in areas with low virus pressure Perry will outperform NC-V 11 (Lanier et al. 2004). These data suggest that virus pressure can vary significantly throughout the region (Table 11), but overall that incidence in 2005 was low in production fields.

Summary

Results from these experiments suggest that *Tomato spotted wilt virus* (TSWV) incidence is heavily influenced by cultivar selection. Breeding line N03036EJ had more than 20x the amount of TSWV incidence as line N00033. The cultivar Perry had the second highest incidence but did not differ from Gregory. N00033 was the only line that was found to have less TSWV incidence than Gregory, suggesting Gregory remains a robust selection in areas with high TSWV pressure.

Late season foliar chlorosis was identified as peanut yellowing death (PYD) and found to be very highly associated with TSWV infection. Breeding line N02051ol (9) had the greatest PYD incidence but did not differ from Perry or Gregory. A large proportion of asymptomatic peanut plants were found to be infected with TSWV. In specific lines, the amount of asymptomatic infection was more than double that of symptomatic TSWV infection. These findings suggest that overall incidence of TSWV infection in peanut throughout North Carolina may be greater than visual estimates alone would imply. Breeding line N03036EJ had nearly 10x greater asymptomatic incidence than line N00033. A moderate degree of correlation was detected between symptomatic TSWV incidence and asymptomatic incidence. This suggests that lines that are more susceptible to TSWV are more likely to become infected, rather than just more likely to express symptoms if infected.

Tomato spotted wilt virus incidence varied throughout the state. Columbus, Duplin and Pitt county plots had higher incidence of symptomatic TSWV incidence than plots at Peanut Belt Research Station (PBRS) or Upper Costal Plains Research Station. Duplin County and the PBRS had less asymptomatic infection while the other locations had greater incidences. Differences between cultivars Perry and NC-V 11 were inconstant across locations. In some areas Perry was found to have greater asymptomatic incidence, while in others NC-V 11 had a larger proportion of infected plants.

Greater numbers of adult thrips were collected from Gregory than any other line. Though Gregory had a greater adult density at the time of sampling than Perry, neither cultivar differed in any other of the other treatment parameters. NC-V 11 had greater adult density than Perry. No correlation was detected between thrips density or damage or any occurrence of TSWV incidence. This suggests that thrips density per breeding line does not serve as a good indicator to resistance to TSWV.

Appendix

Influence of Host Genotype on Transmissibility of TSWV by Thrips

Preliminary experiments were conducted from 2004 to 2006 in an effort to develop understanding of mediated TSWV inoculations in a laboratory and greenhouse setting. Peanut is known to be most susceptible to TSWV while a seedling (Brown et al. 2001) and becomes increasingly difficult to infect as the plant ages (Mandal et al. 2001a). The pathway by which resistance effectively reduces TSWV incidence in peanut is unknown. One possibility is that the duration of seedling susceptibility is prolonged in susceptible versus resistant peanut lines. By exposing resistant and susceptible peanuts lines to thrips-vectored TSWV for varying time periods after germination, one could detect varying durations of susceptibility.

Preliminary experiments

In late 2004 through early 2005, using the inoculation method described by Hoffmann et al. (1998), efforts were made to infect the cultivar “Perry” with TSWV. Due to availability, and previous consistency in the lab, the TSWV isolate “Parker” was used. The Parker isolate was collected from potato (*Solanum tuberosum* L.) during 2002 in Pasquotank County, NC. Two mechanical inoculation attempts were made, but no inoculated peanut tested positive for TSWV infection by ImmunoStrip® assay (Agdia Inc., STX 39300, ACC 00996, ACC 00925, Elkhart, IN). Another inoculation method was attempted using a mixture of celite and carborundum as described by Mandal et al. (2001a). One attempt was made with this method and

one inoculated plant tested positive under ImmunoStrip® assay. In early 2006, in an effort to avoid the difficulties of mechanical inculcation and to conserve seed, which was limited, a thrips transmission from *Emilia sonchifolia* into Perry was attempted using Parker. No transmission was detected. However, because Parker was not a peanut-derived isolate of TSWV, its ability to infect peanut was unknown. Those results prompted the decision to initiate the experiment using a peanut derived isolate, hypothesizing that the derivation would make a difference in transmissibility.

Laboratory trial

To acquire a suitable and relevant TSWV isolate that could be used in a prolonged series of experiments, TSWV infected leaves were collected from peanut (*Arachis hypogaea* L.) at PBRS in 2005. The peanut isolate was passed into *Emilia sonchifolia* (Hoffmann et al. 1998). Infection was confirmed using ImmunoStrip® assay (Agdia Inc. STX 39300, ACC 00996, ACC 00925, Elkhart, IN). Infected peanut tissue was triturated in 0.01 M Tris, pH 7.8, containing 0.01 M Na_2SO_3 and 1% cysteine hydrochloride. Inoculations were made by rubbing carborundum (320 grit) dusted leaves with an inoculum-soaked cotton swab. The isolate was maintained in *E. sonchifolia* in cylindrical isolation cages (25ht x 40cm) made from plastic (5mm clear poly, AIN Plastics Inc., Philadelphia, PA). The top opening of the cylinder was covered with BedBug 110 screening (Greenthumb Group, Downer's Grove, IL). The resultant TSWV infected plants were stored under greenhouse conditions of 28:20C and 14:10 (L:D) h photoperiod and used as the source material for all experiments.

Peanut cultivar Perry and genotype N00033 were selected for use in the experiment based on their observed field incidence of TSWV during 2004 and 2005 and seed availability. Observations indicated that Perry was highly susceptible to TSWV, while N00033 expressed a high degree of resistance. The experiment was designed to expose both lines to viruliferous thrips at cracking, the peanut growth stage in which the cotyledons first begin to open, and the first growth stage in which thrips would have feeding access to peanut in the field. Subsequent experiments were designed to expose the lines to viruliferous thrips at progressing time intervals.

The experiment was conducted April 2006 through June 2006. On 20 April, peanut seed was treated with Vitavax PC fungicide (Bayer CropScience LP, Research Triangle Park, NC) and allowed to begin germination in a moist paper towel roll in a plastic bag placed in an incubation chamber at 24C, 65% RH, with a photoperiod of 14:10 (L:D) h for 10 days. After 10 days, seedlings of Perry and N00033 were planted into individual 296 ml clear plastic cups containing soil filled (Metro-Mix 200 series, Sun Gro Horticulture Distribution Inc. Bellevue, WA) 6cm “jiffy pot” peat pots (Jiffy Products Ltd., Shippaman, Canada) and set at ambient room temperature (20 to 25C) for 48 h. The cups were covered by placing another cup on top and securing with Parafilm®. The top cup had a 1.5cm circular cut made into the top, covered with BedBug 110 screening.

In conjunction with the seedling production previously described, a vector acquisition cycle was completed. *Frankliniella fusca* were obtained from a colony maintained on pole bean pods (*Phaseolus vulgaris* L.). Viruliferous adults were obtained by placing first instar thrips (0-12h old) into 100 x 15mm Petri dishes

containing moistened filter paper and *E. sonchifolia* infected with the presumptive peanut-derived TSWV isolate from PBRS and sealing the dishes with Parafilm®. After an acquisition period of 48, h the thrips were transferred to uninfected pole bean in 473 ml clear plastic cups (Sweetheart Cup Co., Owings Mills, MD) with BedBug 110 screen lids. Thrips were reared in a incubation chamber at 24C, 65% RH with a photoperiod of 14:10 (L:D) h. Fresh bean pods were added to the containers every 3 days until adult eclosion. On 2 May 2006, 520 potentially viruliferous thrips were transferred, in sets of 10, into 1.5 ml microcentrifuge tubes.

On 2 May, 26 seedlings from each line were selected at cracking stage. Microcentrifuge tubes containing 10 potentially viruliferous thrips were placed into the cups containing the peanut and opened to expose the plant to infection. Thrips were removed on 5 May, and plants were allowed to continue to grow at ambient room temperature (20 to 25C) for 72 h at which time plants were transplanted into 15.24cm plastic pots filled with Metro-Mix, (200 potting medium, Sun Gro Horticulture Distribution Inc. Bellevue, WA) covered with a plastic cylinder as previously described, and held under greenhouse conditions of 28:20C with a photoperiod of 14:10 (L:D). On 12 June, taproots were collected from all peanut plants and excised as previously described. TSWV infection was examined using ELISA testing kits (PSA 39330, Agdia Inc., Elkhart, IN). The assay is based on a double antibody sandwich protocol with a monoclonal antibody used for both capture and detection. Roots were individually triturated using general extract buffer (ACC 00955, Agdia Inc., Elkhart, IN) and then 1 ml of each triturate was pipetted into a well in a pre-coated ELISA plate sensitive to TSWV. Plates were read on a

THERMOmax microtiter plate reader (Molecular, Devices Corp., Menlo Park, CA) at a wavelength of 405nm. A plant was considered infected if the optical density (OD at 405nm) was greater than the mean plus 3 standard deviations of the non-infected controls.

In conjunction with the above experiment, an effort was made to verify the ability of the *F. fusca* colony to transmit the presumptive peanut-derived isolate of TSWV using *E. sonchifolia*. On 2 May 2006, 100 potentially viruliferous thrips were placed onto individual *E. sonchifolia* seedlings held in 60x15mm Petri dished with moist filter paper. A 48hr inoculation period was allowed after which all thrips were removed from the plants. Plants were subjugated to ELISA (PSA 39330, Agdia Inc., Elkhart, IN) 10 days after the inoculation period. The number of plants infected plants was recorded. This experiment was not carried to a second repetition.

Table 1. List of genotypes[a] used in the 2004 and 2005 ALT and DAT experiments.

Genotype (Accession)	Identity or Parentage	Pedigree
N90014E	NC 7 / NC9	F2-33-B-B-A01:F08
N91026E	NC 7 / NC9	F2-18-B-B-L01:F08
N98002	N90014E / N91024	F2-S-S-01-02:F07
N98003	Phillips	
N99103ol (9)	NC 9 // X90047 (F2-S-S-18: F05), NC 9 / F435	BC1F2-02-02-04:F07
N00033	N90010E / N92020	F2-01-08-02-01:F07
N00035J	N90010E / N92020	F2-03-07-01-01:F07
N00090ol (7)	Brantley	
N00098ol	Gregory // Random ol1ol2, NC 9*2 / F435, X95253 (F1-02-04: F02) /3/ Gregory , X96201 (BC1F-01: F01) /4/ Gregory	BC2F1-01-03:F04
N01012T	PI 371853 / 2*N90010E	BCF1-05-02-01-01:F07
N01051	Gregory / VA 93B	F2-01-2-02-S-01:F08
N01054	N90010E / VA 9210162	F2-02-S-02-S-02:F08
N01057	N90014E / VA 93B	F2-01-S-03-S-01:F08
N01060	N91003E / VA 9210162	F2-02-S-02-S-01:F08
N01083	N91054E / VA 9210162	F2-04-S-03-S-02:F08
N02012	N90010E / N91047E	F2-05-01-01-01-01:F08
N02020J	N91003E / Gregory	F2-09-01-01-01-01:F08
N02051ol (9)	NC 9 // X90047 (F2-S-S-25: F05), NC 9 / F435 (Knauft high O/L), X94068 (BC1F2-05: F02) /3/ NC 9, X95020 (BC2F1-01-08: F02) /4/ NC9	BC3F1-01-B-01:F05
N02053ol (11)	NC-V 11 // X90048 (F2-S-S-11: F05), NC-V 11 / F435 (Knauft high O/L), X94071 (BC1F2-02 : F02) /3/ NC-V 11, X95035 (BC2F1-01-03: F02) /4/ NC-V 11	BC3F1-01-B-01:F05
N02060ol	Perry // Random ol1ol2, NC 9*2 / F435, X95249 (F1-01-04: F02) /3/ Perry, X96224 (BC1F1-01-04: F02) /4/ Perry	BCF2-06-01:F05
N03004F	NC 12C*2 / N96076L	BC1F1-06-02-S-01-S-02:F08
N03005J	NC 12C*2 / N96076L	BC1F1-06-05-S-03-S-01:F08
N03006J	Perry*2 / N96076L	BC1F1-07-01-S-05-01:F08

[a] Shaded blocks are lines planted in both 2004 and 2005, non shaded blocks were only planted in 2004

Table 1. (Cont.)[a]

Genotype	Identity or Parentage	Pedigree
N03020E	VA 98R // X98011 (F1), Perry / N96076L	F1-04-01-S-03-S-01:F08
N03023EF	VA 98R // X98011 (F1), Perry / N96076L	F1-04-02-S-02-S-03:F08
N03025J	Perry // X98007 (F1), NC 12C / N95003C	F1-01-01-S-02-S-01:F08
N03026EJ	Wilson*2 / Tamrun 98	BC1F1-05-01-S-02-S-01:F08
N03027EF	Wilson*2 / Tamrun 98	BC1F1-05-01-S-03-S-01:F08
N03028E	Gregory / Andru 93	F2-01-03-03-01-01:F08
N03031EJ	Gregory / MARC I	F2-04-02-01-02-01:F08
N03032EJ	Gregory / MARC I	F2-04-04-01-01-01:F08
N03033EJ	Gregory / MARC I	F2-04-05-01-01-02:F08
N03034EJ	Gregory / MARC I	F2-04-05-01-01-03:F08
N03035EJ	Gregory / MARC I	F2-04-05-01-01-04:F08
N03036EJ	Gregory / MARC I	F2-04-05-01-02-01:F08
N03037E	N90010E / Andru 93	F2-02-01-01-01-02:F08
N03038E	N90010E / Andru 93	F2-03-01-01-02-01:F08
N03040E	N90010E / MARC I	F2-03-03-01-02-03:F08
N03043EF	NC 12C / Andru 93	F2-01-01-01-01-01:F08
N03052EF	N91019E / Andru 93	F2-02-01-01-02-02:F08
N03053EF	N91019E / Andru 93	F2-02-05-01-01-02:F08
N03054E	N92025 / Andru 93	F2-06-03-01-01-01:F08
N03057EF	N93017E / Andru 93	F2-02-04-01-01-02:F08
N03061E	N93017E / Andru 93	F2-02-04-01-02-02:F08
N03066EF	PI 315631 (F4 Sel # 209 made in Israel) / PI 221068 (nambyquarae from Brazil), X90064 (F2-S-S-S-01: F08) // Andru 93	F2-01-02-01-01-02:F08
17404	NC 9	
N90009	Gregory	
N93112C	Perry	
N96076L	N90004 / GP-NC WS 13	F2-S-S-08:F06

[a] Shaded blocks are lines planted in both 2004 and 2005, non shaded blocks were only planted in 2004

Table 2. Comparison of late season chlorotic plants to underlying asymptomatic infected with TSWV for ALT experiments.

Year	Proportion of chlorotic plants infected (%)[b]	Proportion of asymptomatic plants infected (%)[c]	Chi Square	Pr>|Z|
2004	92	10	65.48	<.0001
2005	97	40	26.20	<.0001

[a]Shaded cells indicate |Z|≤0.05.
[b]Infected over stand.
[c]Infected per 10 randomly selected plants.

Table 3. Analyses of variance (*p* values) for percentage of plants with thrips injury, and the densities of adult and larvae for ALT experiments.

Treatment Factor	Df	Thrips Injury (%)[b]	Adult Density Sample 1[c]	Larval density Sample 1[c]	Adults Density Sample 2[d]	Larval density Sample 2[d]
Year	1	0.0962	<.0001	0.0002	0.0037	0.0072
Block(year)	4	0.2336	0.0510	<.0001	0.0075	0.0641
Genotype	23	0.8532	<.0001	0.7061	0.0973	0.1072
Year X Genotype	23	0.1529	0.1877	0.2900	0.1928	0.6469
Error	92	-	-	-	-	-

[a]Shaded cells indicate p≤0.05.
[b]Proportion of thrips injured plants of 10 randomly selected plants.
[c]Collected 2 June 2004 and 5 June 2005.
[d]Collected 17 June 2004 and 27 June 2005.

Table 4. Analyses of variance (*p* values) for percentages of symptomatic plants infected with *Tomato spotted wilt virus* (TSWV incidence), peanut yellowing death symptomatic plants infected with TSWV (PYD incidence), asymptomatic plants infected with TSWV (asymptomatic incidence), and percentages of plants with final TSWV severity ratings of rank 1(sev1), rank 2(sev2), and rank 3(sev3) for ALT experiments.

Treatment Factor	Df	TSWV incidence (%)[b]	PYD incidence (%)[b]	Asymptomatic incidence (%)[c]	Sev1 (%)[b]	Sev2 (%)[b]	Sev3 (%)[b]
Year	1	0.3623	0.2103	<.0001	0.6879	0.2649	0.0399
Block(year)	4	0.3920	0.7076	0.8478	0.0256	0.5530	0.6366
Genotype	23	0.0333	0.0082	0.0047	0.5171	0.9517	0.2395
Year X Genotype	23	0.2554	0.5588	0.2660	0.1888	0.4767	0.5960
Error	92	-	-	-	-	-	-

[a]Shaded cells indicate p≤0.05.
[b]Proportion of TSWV infected plants over stand.
[c]Proportion of TSWV infected per 10 randomly selected plants.

Table 5. Analyses of variance (*p* values) for thrips damage rating[a] for ALT experiments.

Treatment Factor	Df	Thrips Damage Rating[c]
Block	2	0.0046
Genotype	23	0.1998
Sample	1	0.8487
Sample X Genotype	23	0.0695
Error	94	-

[a]Shaded cells indicate p≤0.05.
[b]Data is drawn from 2004 only.
[c]Based on a rating scale of 1-3, where 3 is severely damaged.

Table 6. Influence of genotype on adult thrips density (adult density), percentages of symptomatic plants infected with *Tomato spotted wilt virus* (TSWV incidence), peanut yellowing death symptomatic plants infected with TSWV (PYD incidence), and asymptomatic plants infected with TSWV (asymptomatic incidence) for ALT experiments.

Genotype	Adult density[b]	LSD[a]	TSWV incidence (%)[c]	LSD[a]	PYD incidence (%)[c]	LSD[a]	Asymptomatic incidence (%)[c]	LSD[a]
N03036EJ	20.0	BCDEF	32.2	A	7.6	AB	48.9	A
Perry	21.9	BCD	30.2	AB	5.0	ABCD	23.5	ABCDEF
N02051ol (9)	18.6	CDEF	23.3	ABC	12.7	A	15.9	CDEF
N01057	9.4	G	22.0	ABC	6.8	ABC	47.4	AB
N90014E	12.0	FG	18.5	ABCD	3.9	ABCDEF	23.6	ABCDEF
N98002	20.8	BCDE	15.3	ABCD	2.2	BCDEF	20.7	BCDEF
N00035J	24.8	ABC	15.1	ABCD	0.5	DEF	13.7	CDEF
Gregory	33.2	A	14.8	ABCD	4.0	ABCDEF	7.6	EF
N03025J	19.8	BCDEF	14.4	ABCD	6.7	ABC	15.9	CDEF
N03040E	18.8	CDEF	14.2	ABCD	4.2	ABCDEF	32.6	ABCD
N03032EJ	18.9	CDEF	13.5	ABCD	2.2	BCDEF	12.1	DEF
N03020E	12.9	EFG	13.2	BCD	4.4	ABCDE	26.7	ABCDE
N00098ol (Gre)	22.5	ABCD	12.8	BCD	4.9	ABCD	14.6	CDEF
N01054	30.3	AB	12.7	BCD	0.2	F	30.6	ABCD
N03006J	20.1	BCDE	11.5	CDE	1.6	CDEF	21.4	BCDEF
N91026E	15.5	CDEF	11.3	CDE	2.1	BCDEF	37.2	ABC
N0205ol (11)	22.5	ABCD	11.0	CDE	2.1	BCDEF	23.6	ABCDEF
N0102T	22.6	ABCD	8.4	CDE	1.1	CDEF	6.8	EF
N03054E	10.4	G	8.1	CDE	1.0	CDEF	5.1	F
N96076L	15.1	DEFG	7.8	CDE	2.5	BCDEF	9.6	DEF
N03026EJ	14.1	DEFG	7.5	CDE	0.5	DEF	18.6	CDEF
N03023EF	15.1	DEFG	5.9	DE	0.2	F	5.6	F
N01083	23.0	ABCD	5.4	DE	0.2	F	18.6	CDEF
N00033	20.8	BCDE	1.5	E	0.2	EF	5.9	F

[a]Means[b] followed by the same letter are not significantly different according to Fisher's Protected LSD test p≤0.05.
[b]Means shown have been sqrt transformed.
[c]Means shown have been subjected to angular transformation.

Table 7. Analyses of variance (*p* values) for percentage of plants with thrips injury, and the densities of adult and larvae for DAT experiment.

Treatment Factor	Df	Thrips Injury (%)[b]	Adult Density	Larval density
Block	2	0.0475	0.1412	0.4705
Genotype	6	0.3919	0.4679	0.0092
Error	12	-	-	-

[a]Shaded cells indicate p≤0.05.
[b]Proportion of thrips injured plants of 10 randomly selected plants.

Table 8. Analyses of variance (*p* values) for percentages of symptomatic plants infected with *Tomato spotted wilt virus* (TSWV incidence), peanut yellowing death symptomatic plants infected with TSWV (PYD incidence), asymptomatic plants infected with TSWV (asymptomatic incidence), and percentages of plants with final TSWV severity ratings of rank 1(sev1), rank 2(sev2), and rank 3(sev3) for DAT experiment.

Treatment Factor	Df	TSWV incidence (%)[b]	PYD incidence[b] (%)[b]	Asymptomatic incidence (%)[c]	Sev1 (%)[b]	Sev2 (%)[b]	Sev3 (%)[b]
Block	2	0.7938	0.5904	0.1513	0.0833	0.1865	0.2513
Genotype	6	0.3012	0.9497	0.1245	0.2440	0.7484	0.1530
Error	12	-	-	-	-	-	-

[a]Shaded cells indicate p≤0.05.
[b]Proportion of TSWV infected plants over stand.
[c]Proportion of TSWV infected per 10 randomly selected plants.

Table 9. Influence of genotype on larvae thrips density (larval density) for DAT experiment.

Genotype	Larvae Desnity[a]
N02020J	35.8a
N03004F	19.7b
N99103ol (9)	16.6b
Brantley	14.7b
N03005J	13.9b
Phillips	13.3b
N02060ol (Per)	10.6b

[a]Means[b] shown have been sqrt transformed.
[b]Means with the same letter are not significantly different according to Fisher's Protected LSD test p≤0.05.

Table 10. Analyses of variance (*p* values) for percentage of plants with thrips injury, and the densities of adult and larvae for Perry and NC-V 11.

Treatment Factor	Df	Thrips Injury (%)[b]	Adult Density	Larval density
Location	4	<.0001	0.0005	<.0001
Sample(loc)	5	<.0001	<.0001	<.0001
Block(loc)	15	0.1973	0.8385	0.0183
Block*Sample(loc)	15	.04355	0.2052	0.3382
Cultivar	1	0.0409	0.0178	0.5150
Loc X Cultivar	4	0.4328	0.3806	0.1493
Cultivar*Sample(loc)	5	0.1055	0.4445	0.0732
Error	49	-	-	-

[a]Shaded cells indicate p≤0.05.
[b]Proportion of thrips injured plants of 10 randomly selected plants.

Table 11. Analyses of variance (*p* values) for percentage of symptomatic plants infected with *Tomato spotted wilt virus* (TSWV incidence) for Perry and NC-V 11.

Treatment Factor	DF	TSWV Incidence (%)[b]
Location	4	<.0001
Block(loc)	15	0.0018
Cultivar	1	0.9745
Loc X Cultivar	4	0.0890
Loc X Block X Cultivar	15	0.0065
Week	4	<.0001
Loc X Week	16	0.0140
Week X Cultivar	4	0.4920
Loc X Week X Cultivar	16	0.4202
Error	120	-

[a]Shaded cells indicate p≤0.05.
[b]Proportion of TSWV infected plants over stand.

Table 12. Analyses of variance (*p* values) for percentage of symptomatic plants infected with *Tomato spotted wilt virus* (TSWV incidence)[b] by weeks for Perry and NC-V 11.

Treatment Factor	DF	TSWV Incidence Week 1	TSWV Incidence Week 2	TSWV Incidence Week 3	TSWV Incidence Week 4	TSWV Incidence Week 5
Location	4	<.0008	<.0001	0.0788	0.0322	0.0163
Block(loc)	15	0.9931	0.2150	0.4463	0.2224	0.1260
Cultivar	1	0.9556	0.3120	0.5960	0.1609	0.8981
Loc X Cultivar	4	0.8097	0.0261	0.7438	0.4051	0.6975
Error	15	-	-	-	-	-

[a]Shaded cells indicate p≤0.05.
[b]Proportion of TSWV infected plants over stand.

Table 13. Analyses of variance (*p* values) for percentage of asymptomatic plants infected with TSWV (asymptomatic incidence) for Perry and NC-V 11.

Treatment Factor	Df	Asymptomatic incidence (%)[b]
Location	4	0.0002
Block(loc)	15	0.0073
Cultivar	1	0.3188
Loc X Cultivar	4	0.0316
Error	15	-

[a]Shaded cells indicate p≤0.05.
[b]Proportion of TSWV infected per 10 randomly selected plants.

Table 14. Analyses of variance (*p* values) for percentage of asymptomatic plants[b] infected with TSWV (asymptomatic incidence) by locations for Perry and NC-V 11.

Treatment Factor	Df	Columbus	Duplin	PBRS	Pitt	UCPRS
Block	3	0.1790	0.0891	0.5000	0.5000	0.0795
Cultivar	1	0.0250	0.2262	0.3910	0.0414	0.0460
Error	3	-	-	-	-	-

[a]Shaded cells indicate p≤0.05.
[b]Proportion of TSWV infected per 10 randomly selected plants.

Table 15. Influence of genotype on the percentage of asymptomatic plants infected with TSWV (asymptomatic incidence) for Perry and NC-V 11.

Location	Columbus		Duplin		Pitt		PBRS		UCPRS	
Cultivar	Perry	NC-V 11	Perry	NC-V 11	Perry	NC-V 11	Perry	NC-V 11	Perry	NC-V 11
	34.7a	16.8b	7.5a	2.5a	31.8b	50.0a	2.5a	0.0a	21.9a	9.1b

[a]Means[b] followed by the same letter are not significantly different according to Fisher's Protected LSD test p≤0.05.
[b]Means shown have been subjected to angular transformation.

Table 16. Analyses of variance (*p* values) for percentage of plants with final TSWV severity ratings of rank 1(sev1), rank 2(sev2), and rank 3(sev3) for Perry and NC-V 11.

Treatment Factor	DF	Sev1 (%)[b]	Sev2 (%)[b]	Sev3 (%)[b]
Location	4	<.0001	<.0001	<.0001
Block(loc)	15	0.1537	0.6644	0.0066
Cultivar	1	0.5437	0.4451	0.4245
Loc X Cultivar	4	0.7329	0.2680	0.3114
Loc X Block X Cultivar	15	0.4134	0.2167	0.1531
Sample	4	<.0001	0.3495	0.1539
Loc x sample	16	0.0641	0.1747	0.0440
Vrating X Cultivar	4	0.2240	0.6975	0.6415
Loc X sample X Cultivar	16	0.7869	0.4189	0.6641
Error	30	-	-	-

[a]Shaded cells indicate p≤0.05.
[b]Proportion of TSWV infected plants over stand.

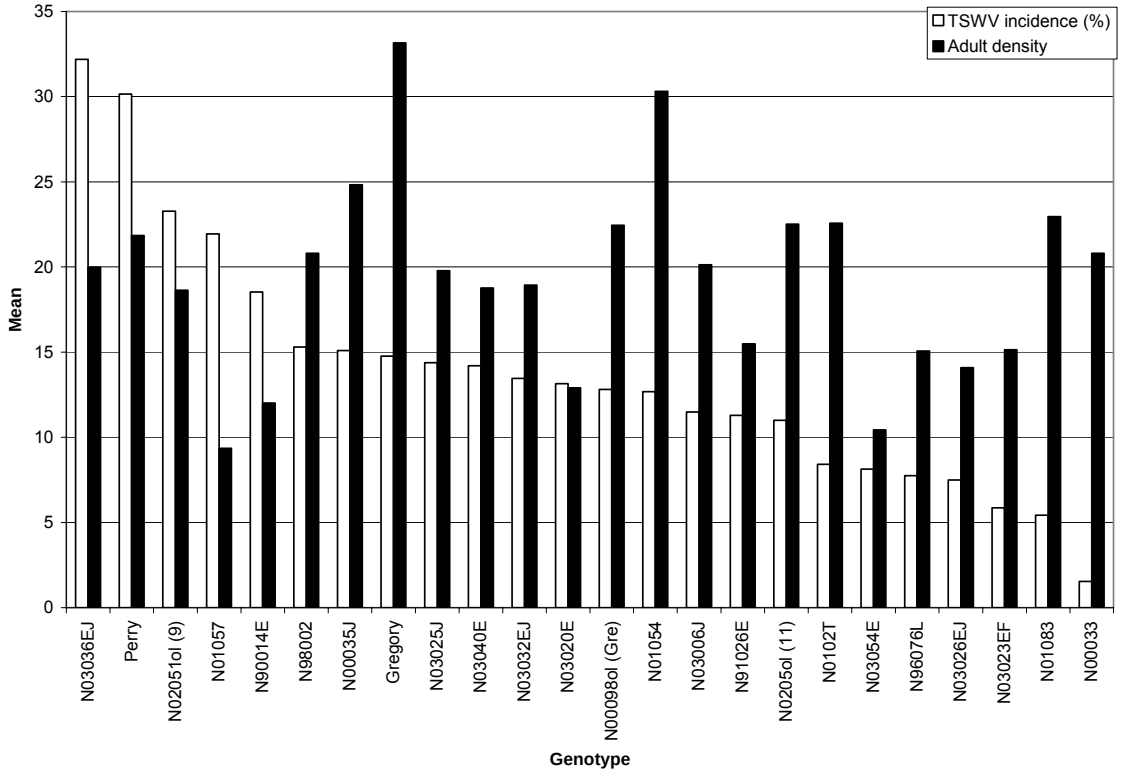

Figure 1. Comparison of adult thrips density[a] and incidence of TSWV[b] among genotypes

[a]Adult thrips density per 10 leaves pooled across 2 June 2004 and 5 June 2005.

[b]TSWV incidence from June through October as the percentage of total plants at digging exhibiting TSWV symptoms at some point throughout the growing season that also confirmed to be infected by ImmunoStrip® assay.

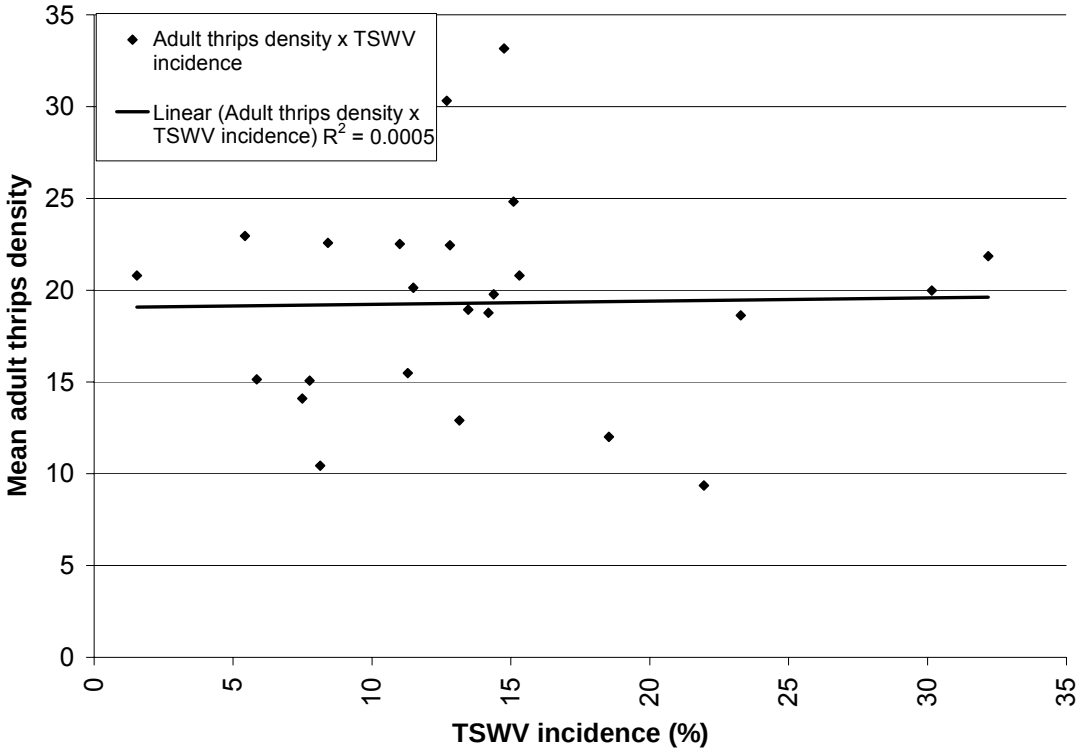

Figure 2. Comparison of adult thrips density[a] and incidence of TSWV[b]

[a]Adult thrips density per 10 leaves pooled across 2 June 2004 and 5 June 2005.

[b]TSWV incidence from June through October as the percentage of total plants at digging exhibiting TSWV symptoms at some point throughout the growing season that also confirmed to be infected by ImmunoStrip® assay.

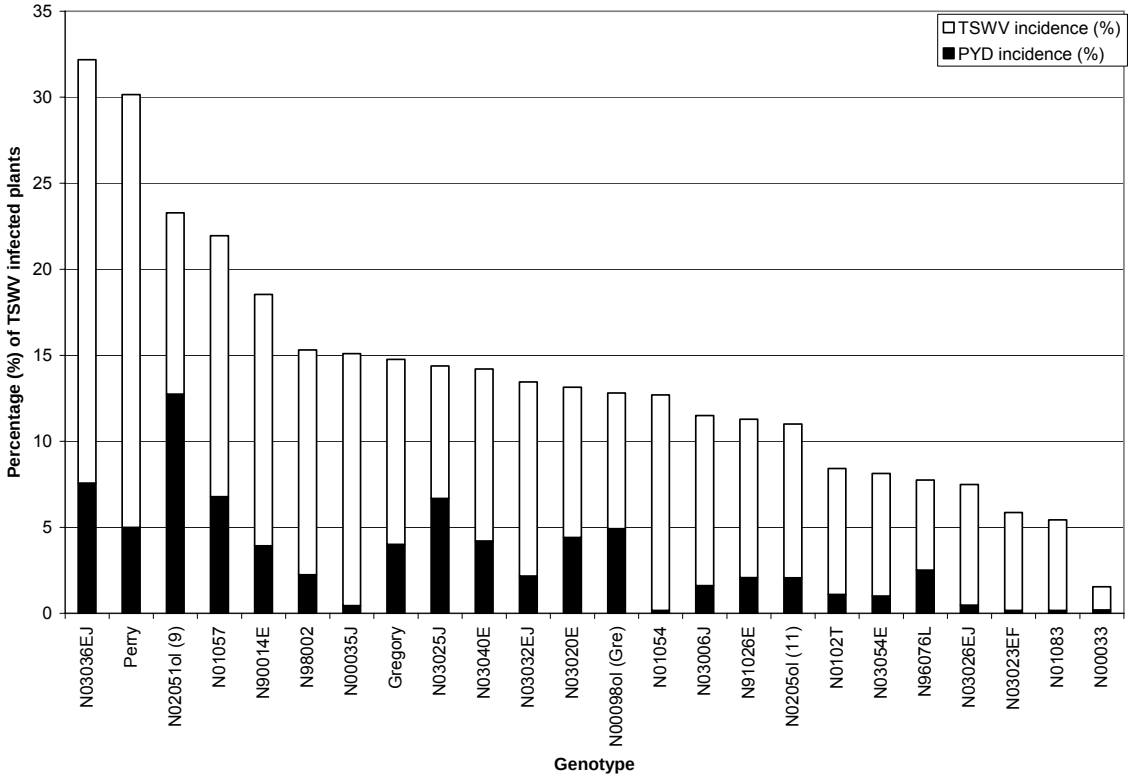

Figure 3. Comparison of PYD[a] and TSWV[b] incidence among genotypes.

[a]Peanut yellowing death (PYD) incidence as the percentage of total plants pooled over 23 September 2004 and 2 Oct. 2005 exhibiting PYD symptoms that also confirmed to be infected by ImmunoStrip® assay .

[b]TSWV incidence from June through October as the percentage of total plants at digging exhibiting TSWV symptoms at some point throughout the growing season that also confirmed to be infected by ImmunoStrip® assay.

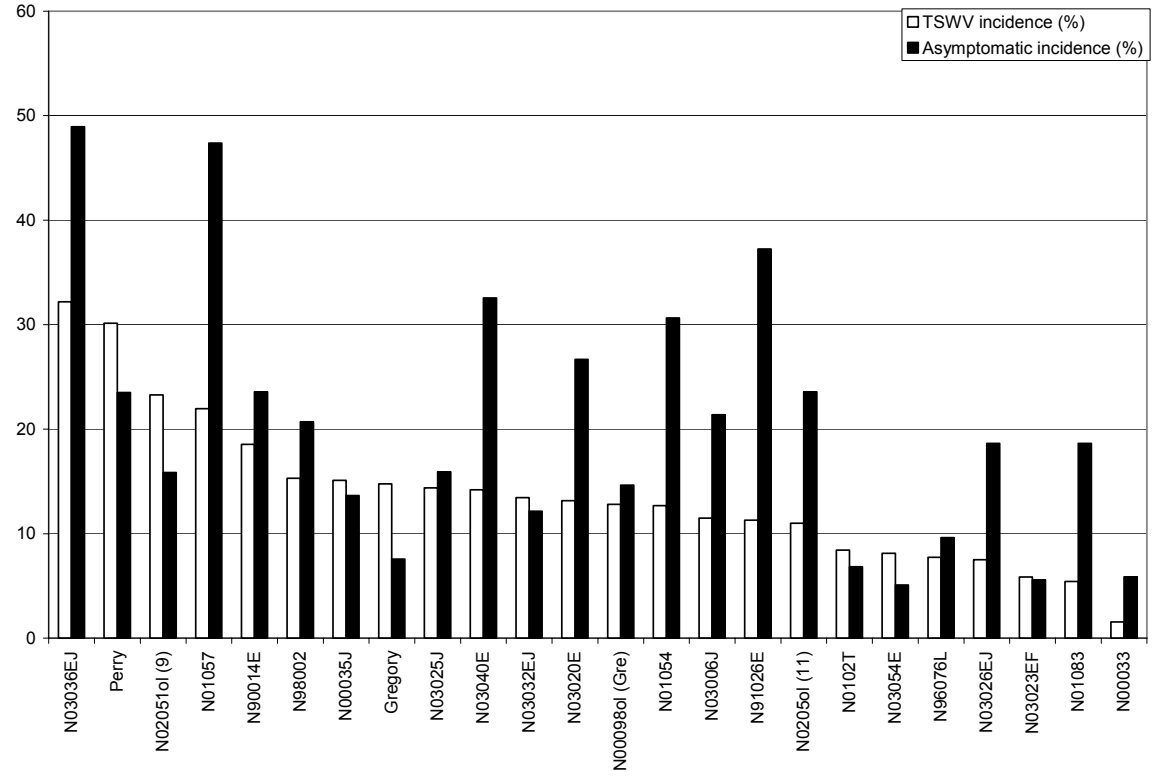

Figure 4. Comparison of asymptomatic incidence[a] and symptomatic incidence[b] of TSWV among genotypes.

[a] Asymptomatic incidence from Oct. 7, 2004, and Oct. 13 2005 as the percentage of roots from 10 randomly selected plants that confirmed to be infected with TSWV by ImmunoStrip® assay.

[b]TSWV incidence from June through October as the percentage of total plants at digging exhibiting TSWV symptoms at some point throughout the growing season that also confirmed to be infected by ImmunoStrip® assay.

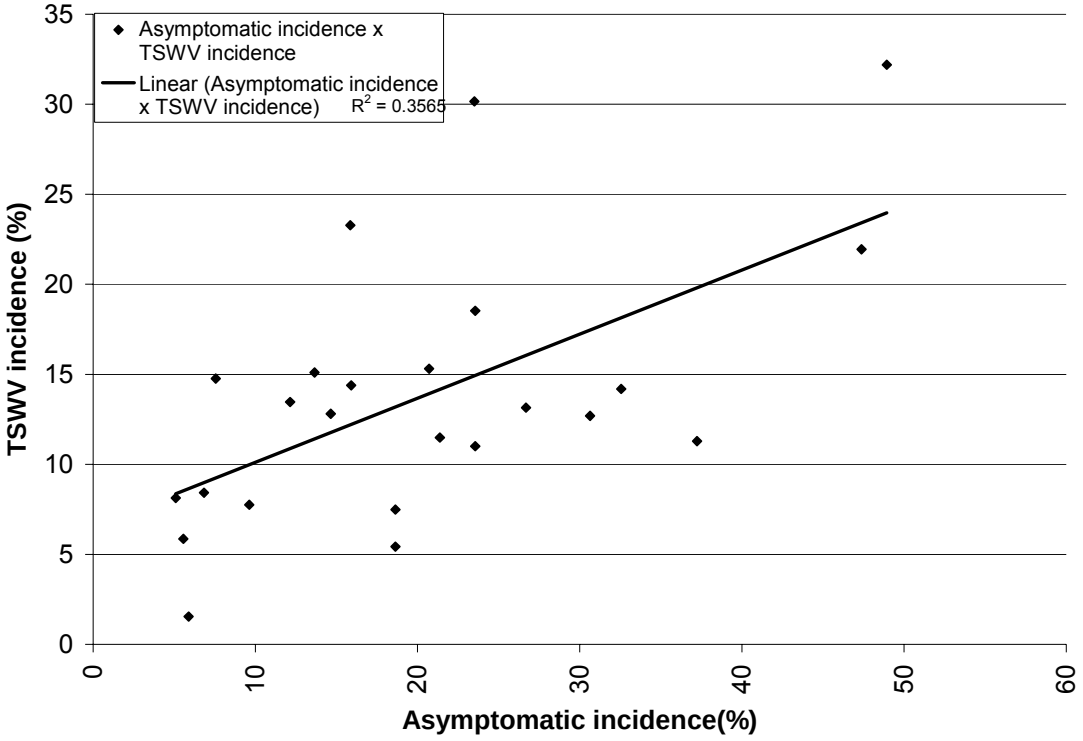

Figure 5. Comparison of asymptomatic incidence[a] and symptomatic incidence[b] of TSWV.

[a] Asymptomatic incidence from 7 Oct. 2004, and 13 Oct. 2005 as the percentage of 10 randomly selected plants that confirmed to be infected with TSWV by ImmunoStrip® assay.

[b]TSWV incidence from June through October as the percentage of total plants at digging exhibiting TSWV symptoms at some point throughout the growing season that also confirmed to be infected by ImmunoStrip® assay.

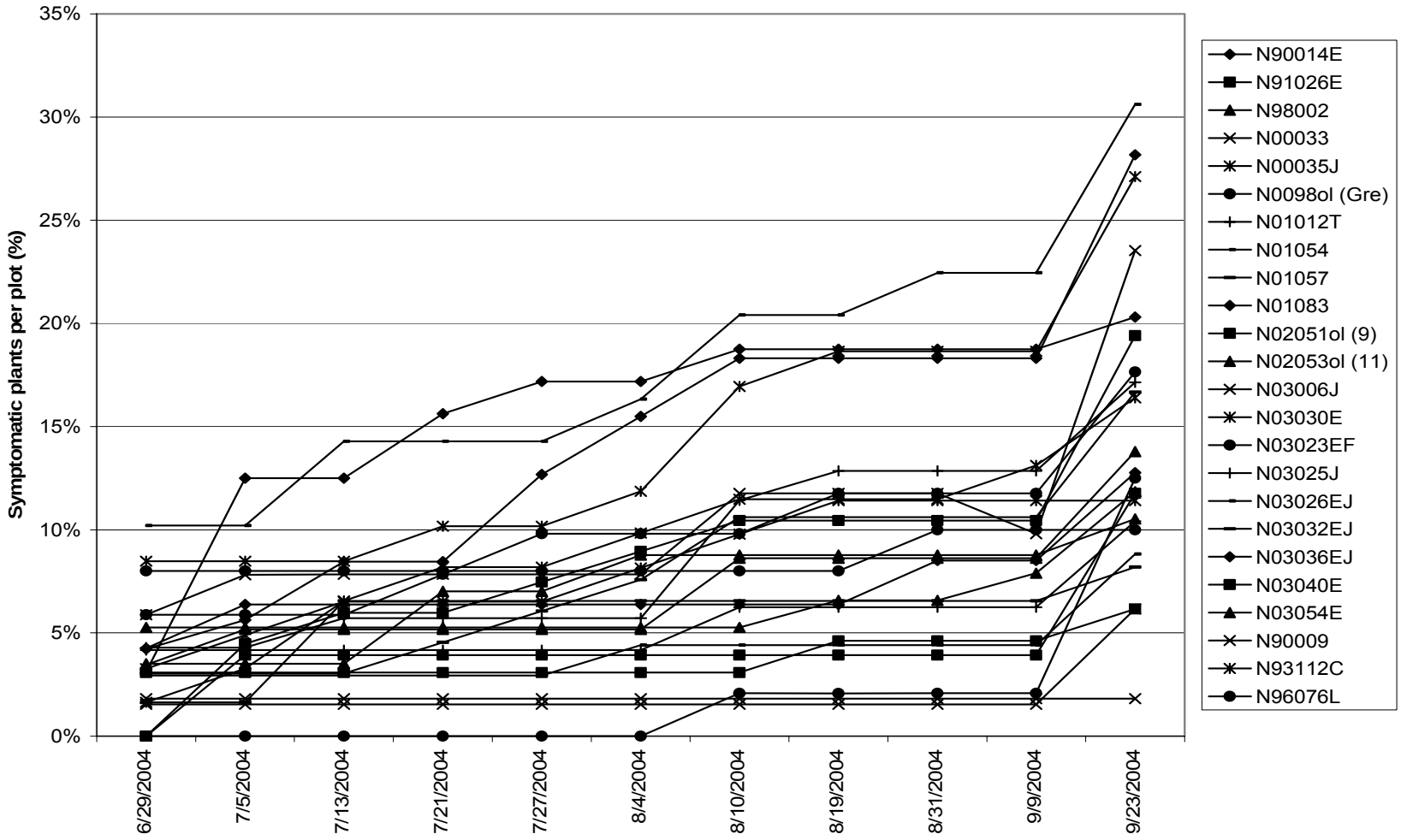

Figure 6. Cumulative incidence of *Tomato spotted wilt virus* symptomatic and infected plants at PBRS 2004 (ALT)

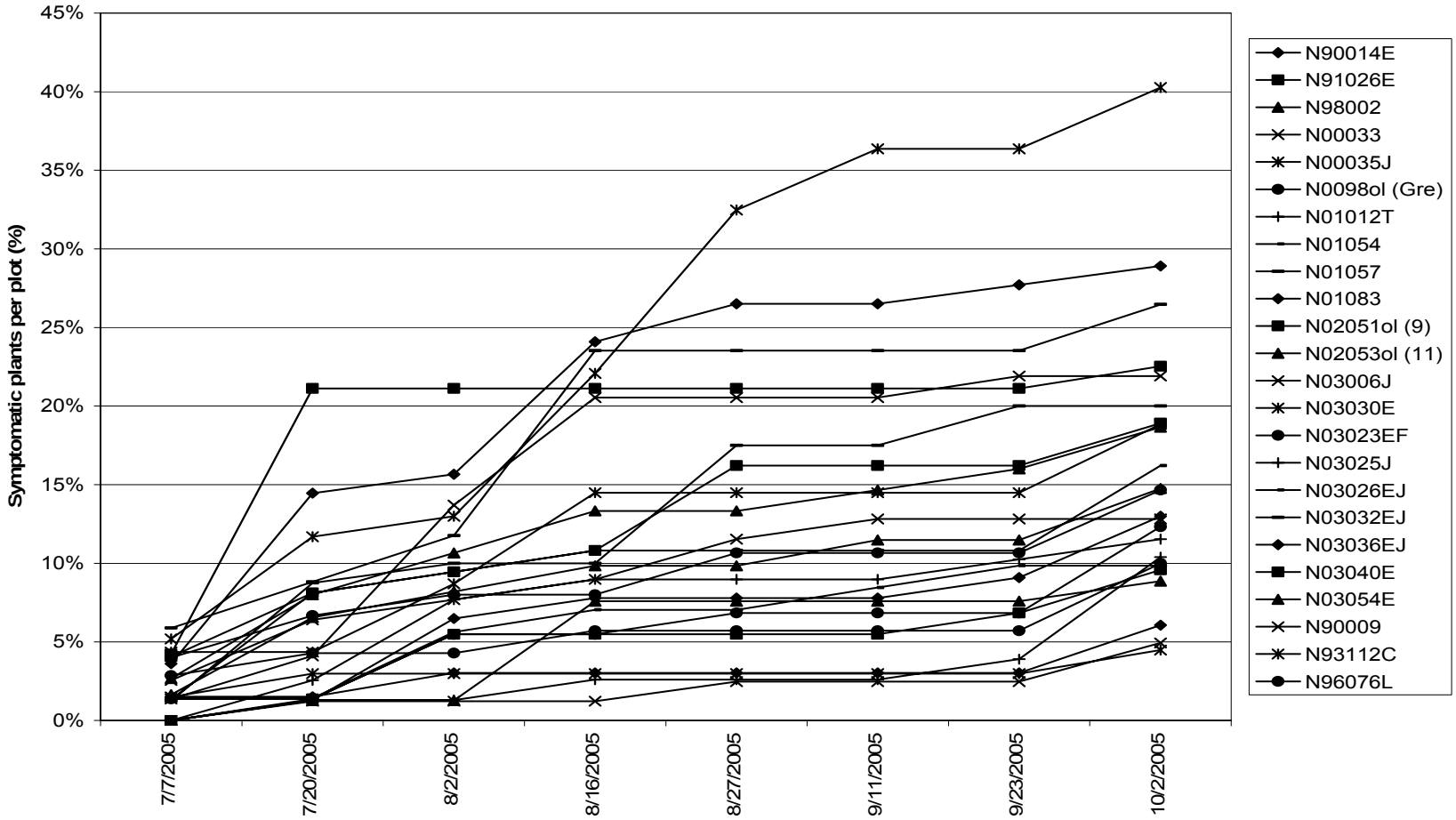

Figure 7. Cumulative incidence of *Tomato spotted wilt virus* symptomatic and infected plants at PBRS 2005 (ALT)

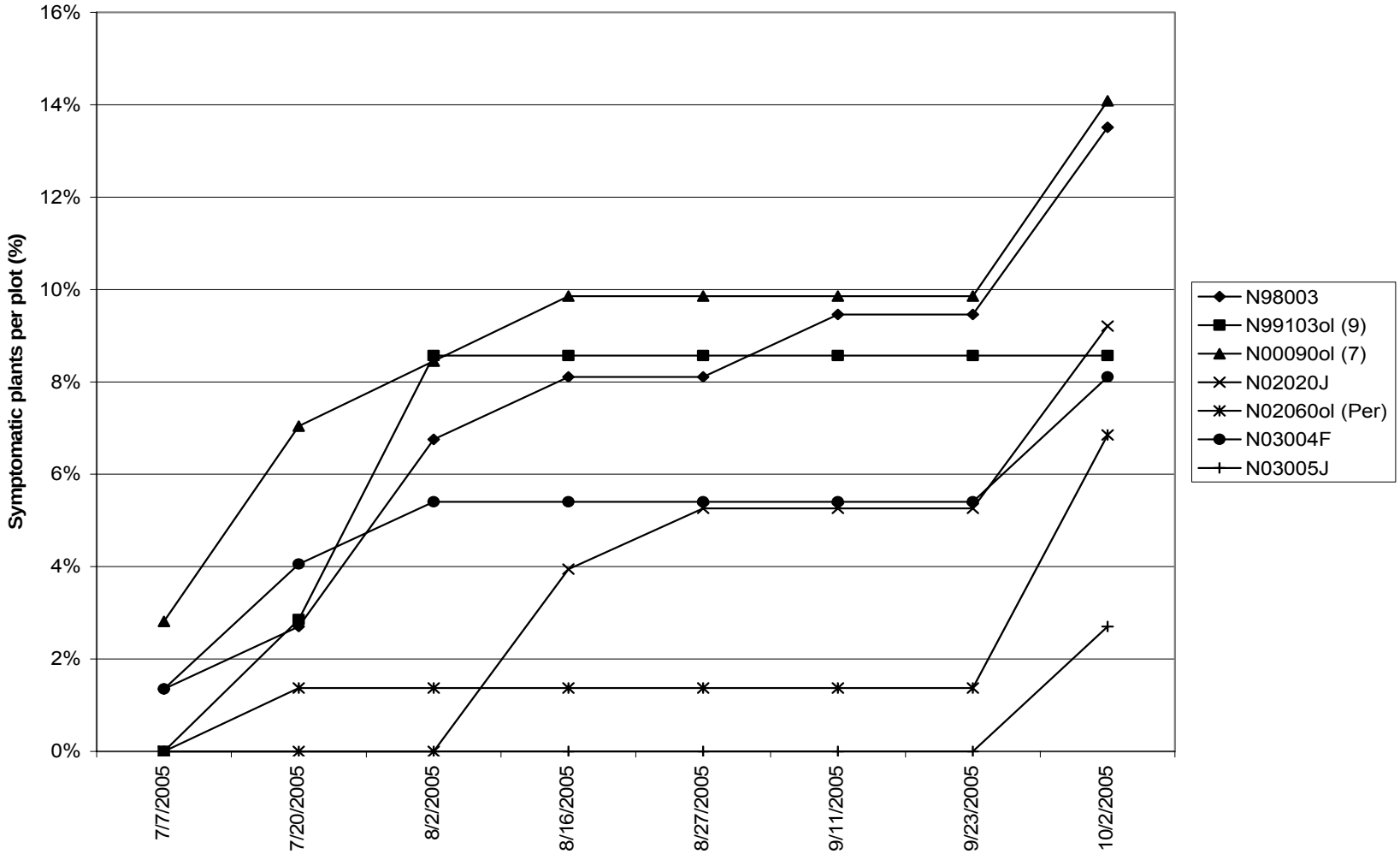

Figure 8. Cumulative incidence of *Tomato spotted wilt virus* symptomatic and infected plants at PBRS 2005 (DAT)

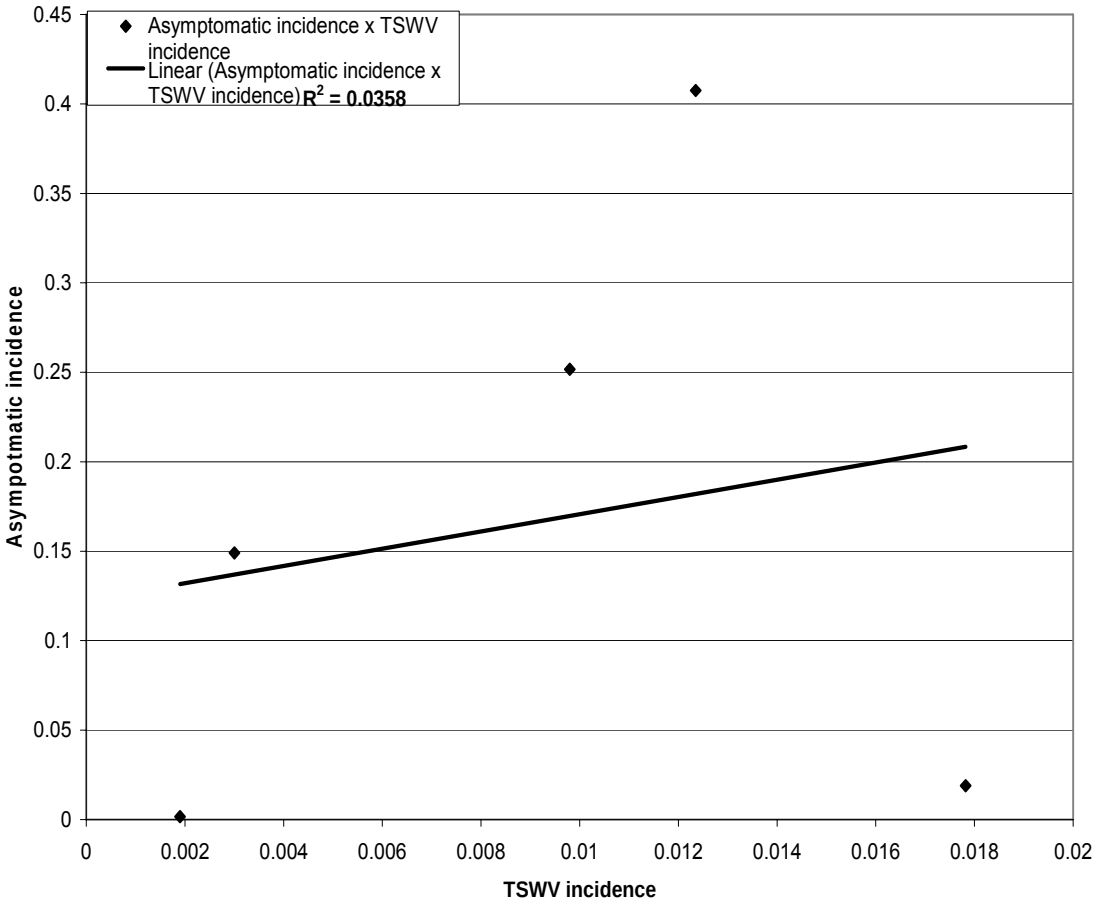

Figure 9. *Tomato spotted wilt virus* incidence[a] by asymptomatic incidence[b].

[a]TSWV incidence from June through October as the percentage of total plants at digging exhibiting TSWV symptoms at some point throughout the growing season that also confirmed to be infected by ImmunoStrip® assay.

[b]Asymptomatic incidence from 2005 as the percentage of 10 randomly selected plants that confirmed to be infected with TSWV by ImmunoStrip® assay

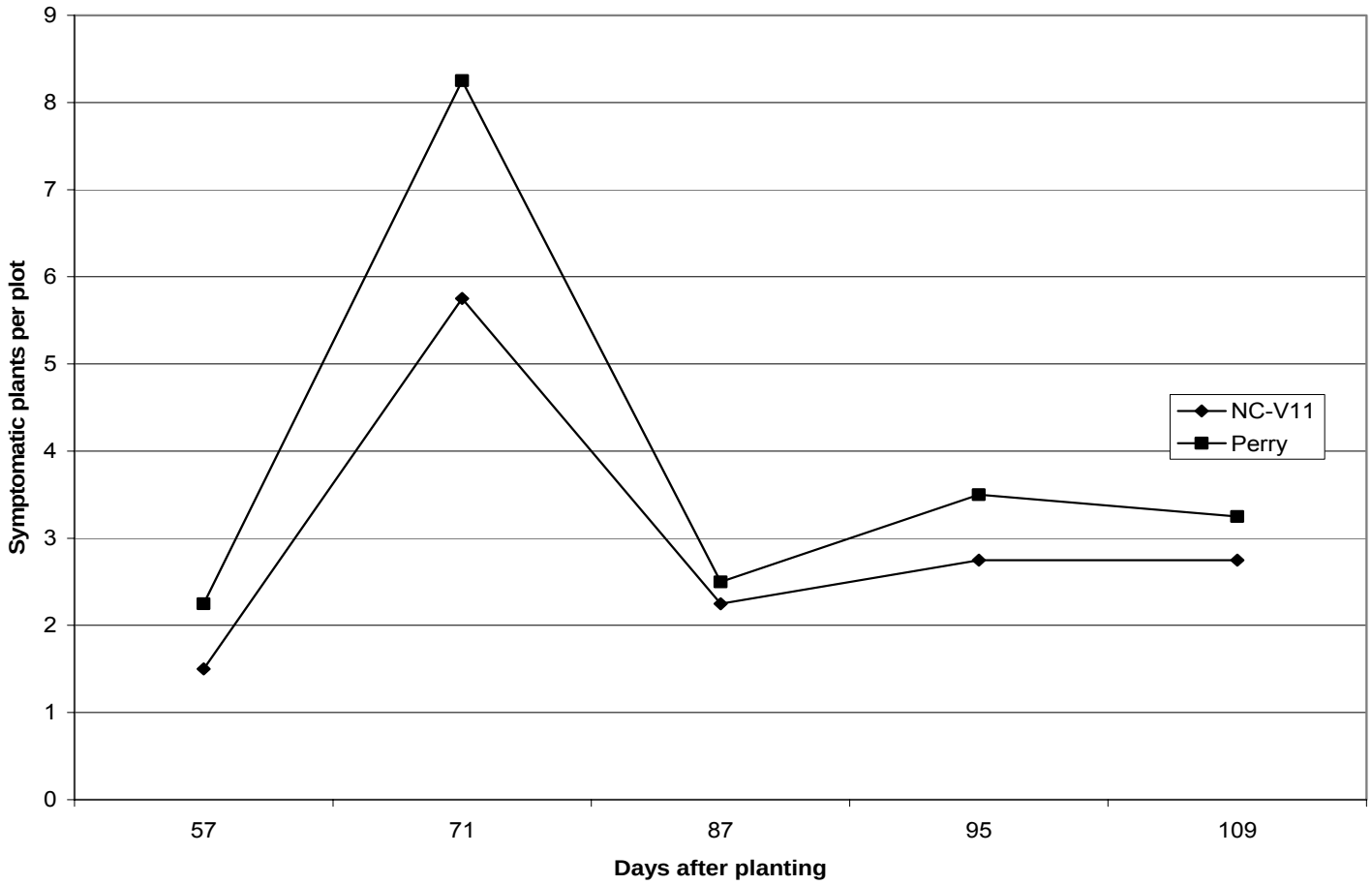

Figure 10. *Tomato spotted wilt virus* symptomatic plants at Columbus County 2005
[a]Peanut was planted 17 May.

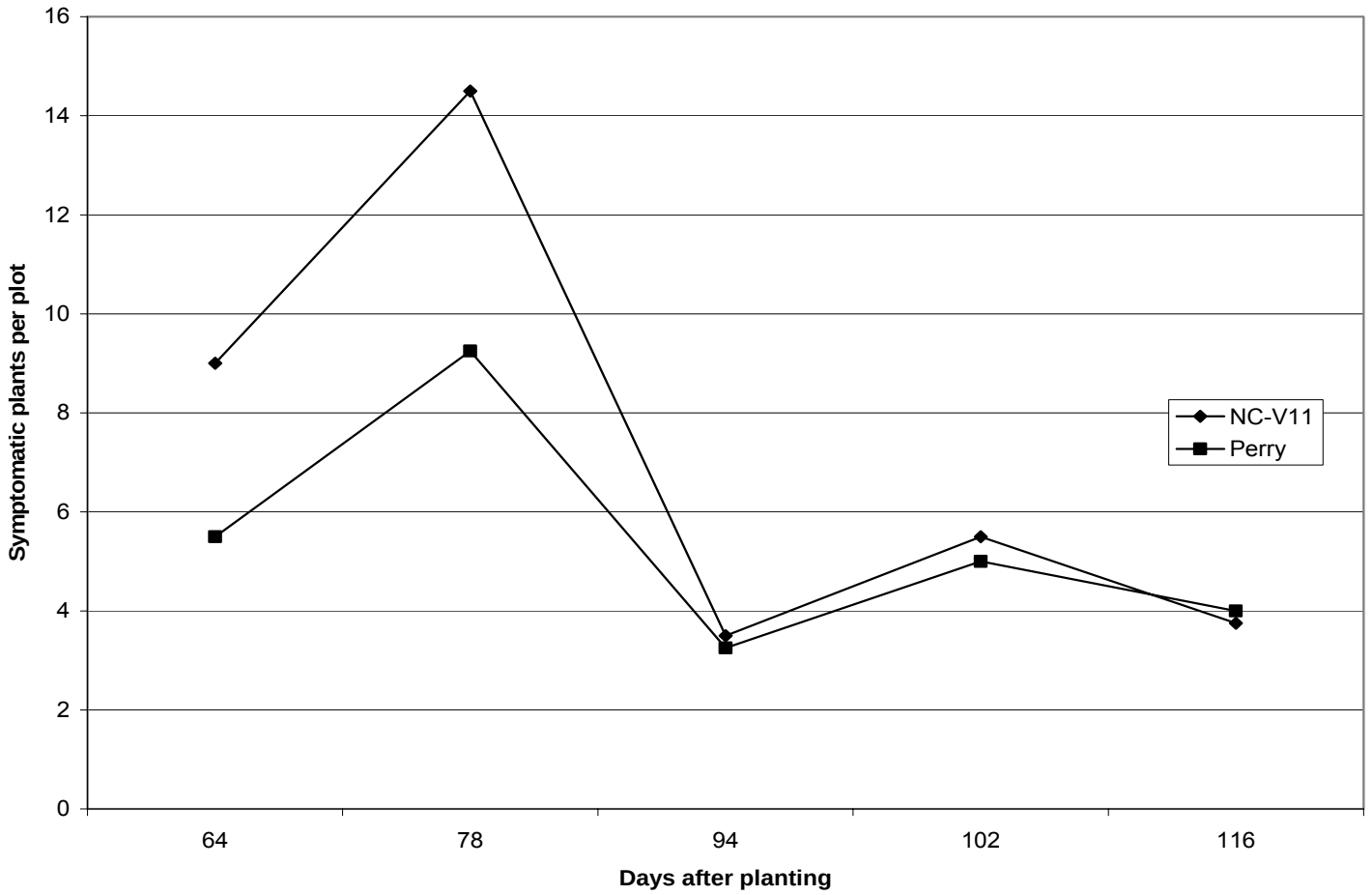

Figure 11. *Tomato spotted wilt virus* symptomatic plants at Duplin County 2005
[a]Peanut was planted 10 May.

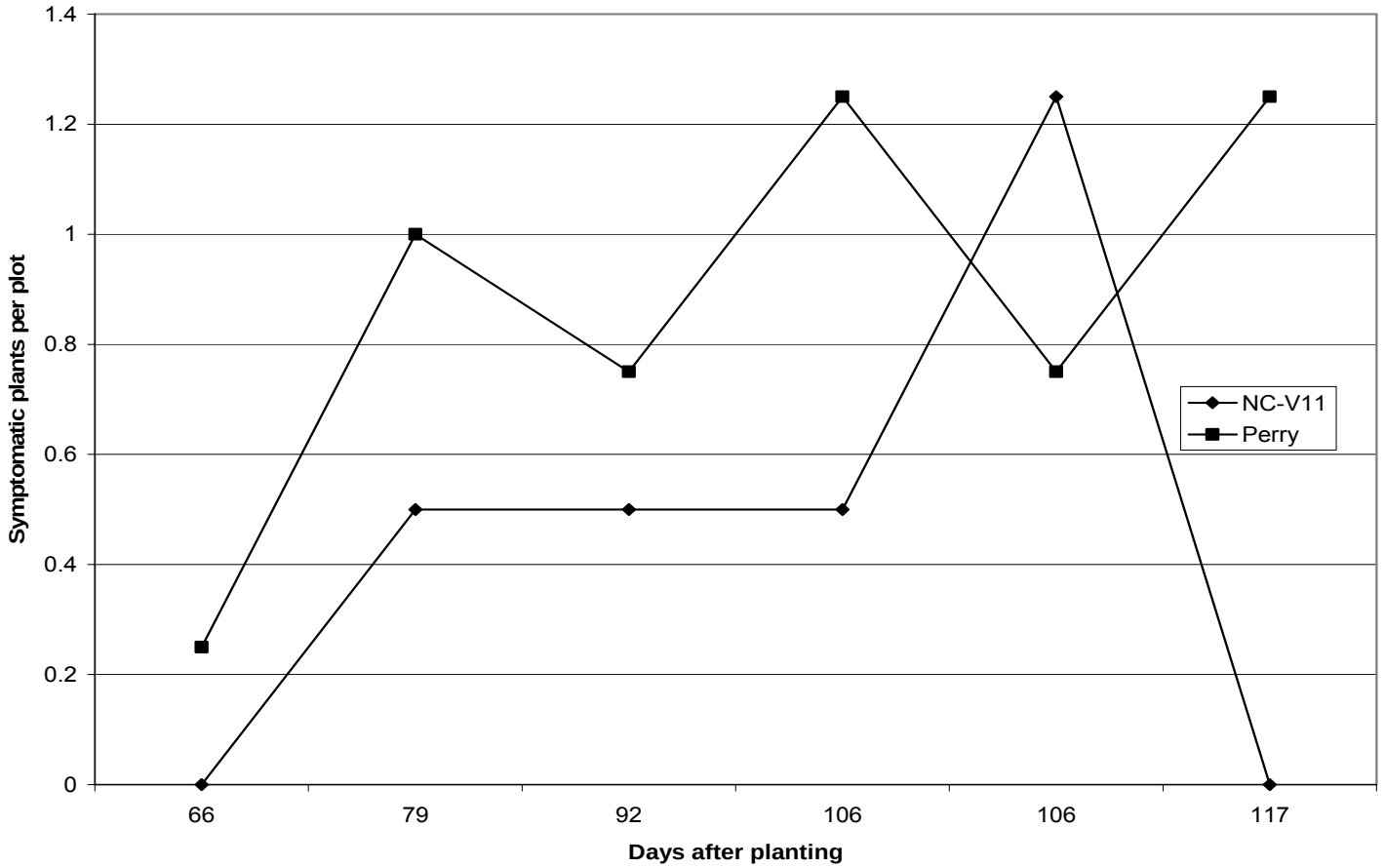

Figure 12. *Tomato spotted wilt virus* symptomatic plants at PBRS 2005
[a]Peanut was planted 2 May.

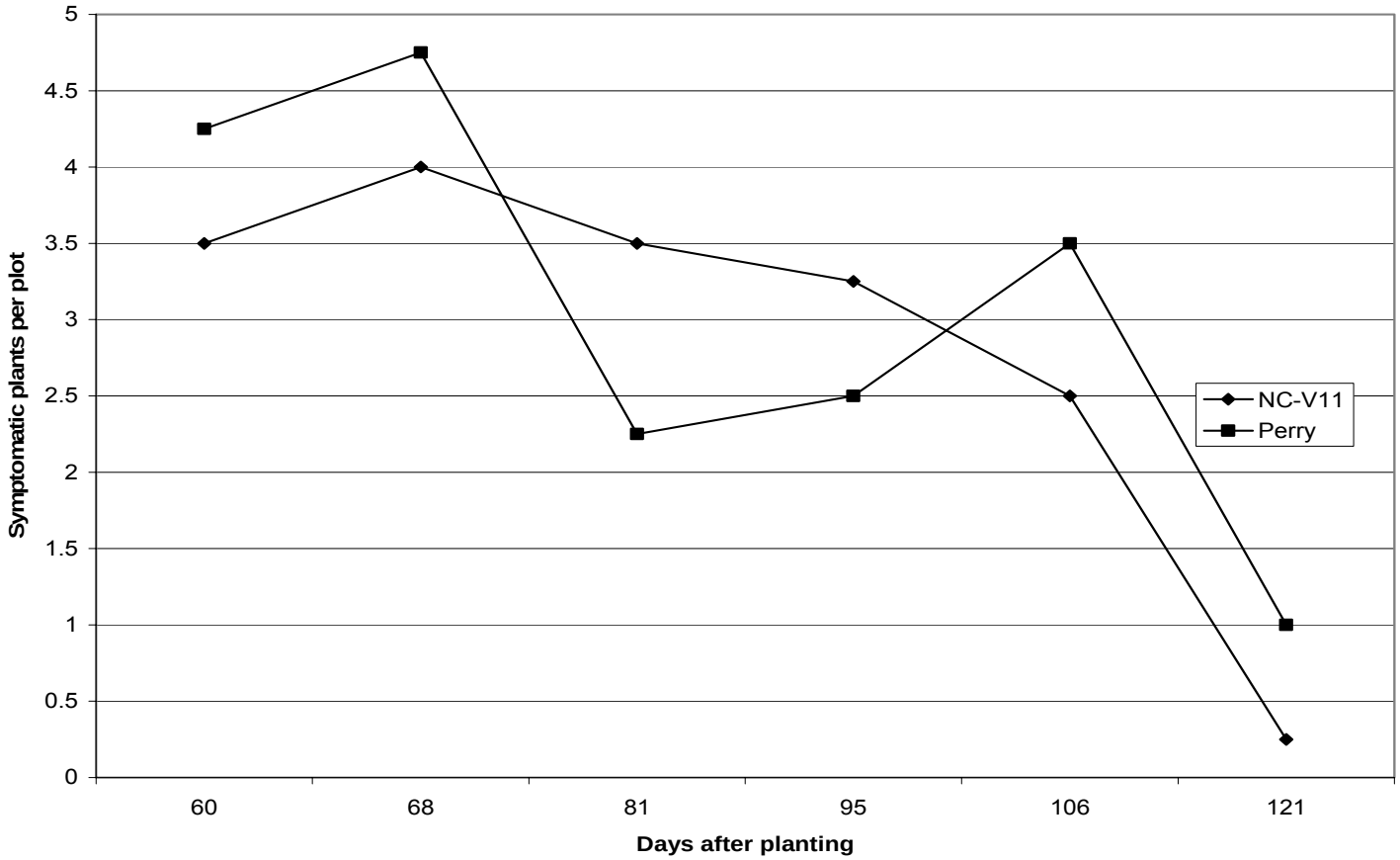

Figure 13. *Tomato spotted wilt virus* symptomatic plants at Pitt County 2005
[a]Peanut was planted 13 May.

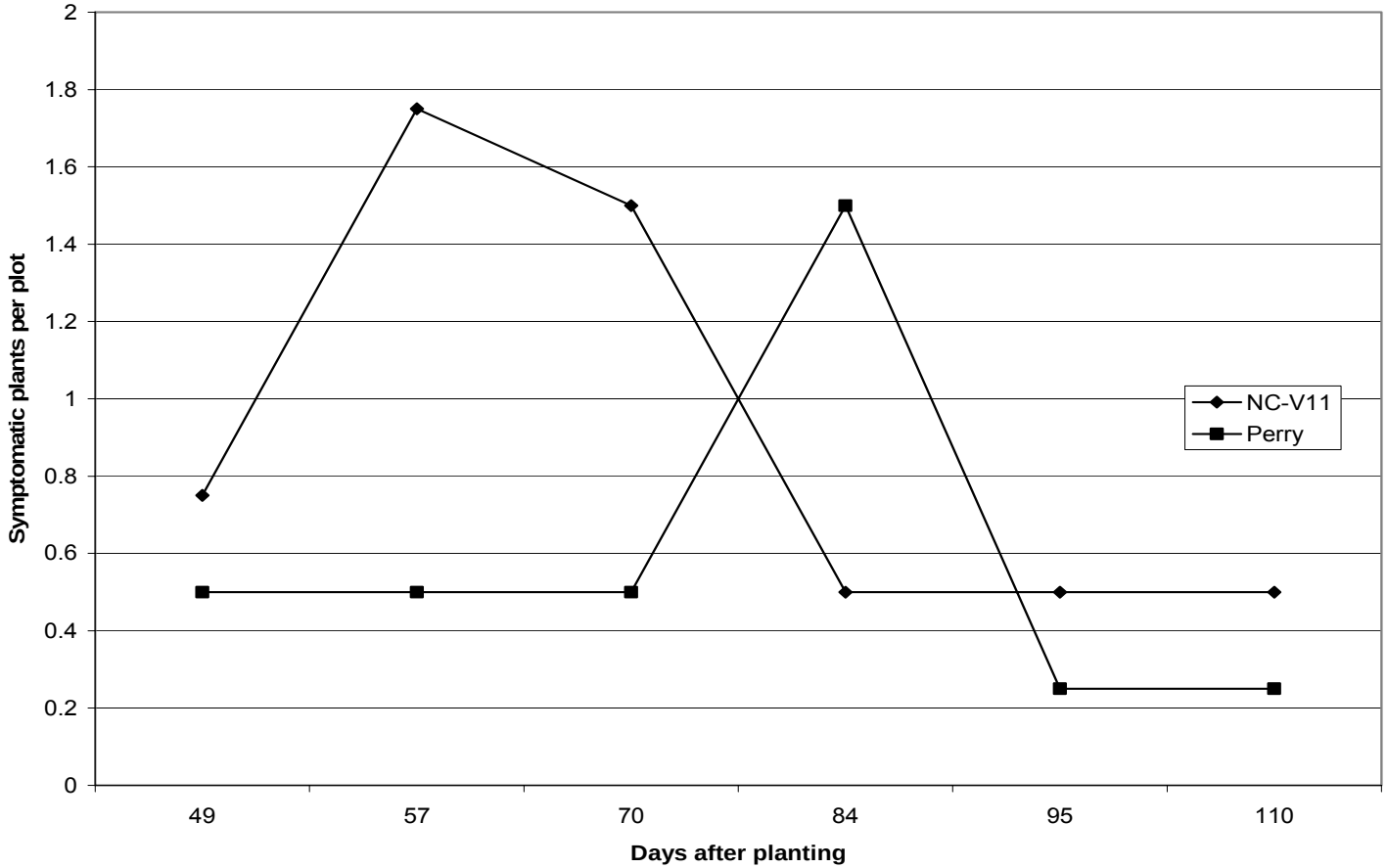

Figure 14. *Tomato spotted wilt virus* symptomatic plants at UCPRS 2005
[a]Peanut was planted 24 May.

REFERENCES CITED

Assis F.M., Deom C.M., Sherwood J.L. 2004. Acquisition of Tomato spotted wilt virus by adults of two thrips species. Phytopathology 94:333-336.

Assis, F. M., R. A. Naidu, C. M. Deom, and J. L. Sherwood. 2002. Dynamics of *Tomato spotted wilt virus* replication in the alimentary canal of two thrips species. Phytopathology. 92:729-733.

Baldwin, J. and J. Williams. 2002. Effect of twin rows on yield and grade. The Peanut Grower. November 2002:28-29.

Barbour, J. D. and R. L. Brandenburg. 1994. Vernal infusion of thrips into North Carolina peanut fields. J. Econ. Entomol. 87:446-451.

Black, M. C., and D. H. Smith. 1987. Spotted wilt and rust reactions in south Texas among selected peanut genotypes. Proc. Am. Peanut Res. Ed. Soc. 19: 31 (abstr.).

Brandenburg, R. L. 2006. Peanut Insect and Mite Management, pp. 61-77. In 2006 Peanut Information, North Carolina Coop. Ext. Ser. Series AG-331.

Brandenburg, R. L., D. A. Herbert, Jr., G. A. Sullivan, G. C. Naderman and S. F. Wright. 1998. The impact of tillage practices on thrips injury of peanut in North Carolina and Virginia. Peanut Sci. 25:27-31.

Brecke, B. J., J. E. Funderburk, I. D. Teare, and D. W. Gorbet. 1996. Interaction of early-season herbicide injury, tobacco thrips injury, and cultivar on peanut. Agronomy Journal. 88:14-18.

Broadbent, A. B. and W. R. Allen. 1995. Interaction within the western flower thrips/*Tomato spotted wilt virus*/host plant complex on virus epidemiology, pp. 185-196. In B. L. Parker et al. [eds.], Thrips Biology and Management. Plenum Press, NY.

Brown, A. B. 2006. Virginia-Type Peanuts: Situation and Outlook, pp. 1-9. In 2006 Peanut Information, North Carolina Coop. Ext. Ser. Series AG-331.

Brown, S. L., A. K. Culbreath, J. W. Todd, D. W. Gorbet, J. A. Baldwin, J. P. Beasley, Jr. 2005. Development of a method of risk assessment to facilitate integrated management of spotted wilt of peanut. Plant Disease. 89:348-356.

Brown, S. L., Todd, J., Culbreath, A., Baldwin, J., Beasley, J., Kemerait, B., and Prostko, E. 2003. Minimizing spotted wilt of peanut. Univ.Georgia Coop. Ext. Serv. Bull. 1165.

Brown, S., J. Todd, A. Culbreath, J. Baldwin, J. Beasley, B. Kemerait and H. Pappu. 2001. Tomato spotted wilt of peanuts: Identifying and avoiding high-risk situations. The Peanut Grower: April 2001:12-15.

Brown S. L., J. W. Todd, and A. K. Culbreath. 1996. Effect of selected cultural practices on incidence of *Tomato spotted wilt virus* and populations of thrips vectors in peanuts. Acta Horticulturae 431:491-498.

Camann M. A., A. K. Culbreath, J. Pickering, J. W. Todd, and J. W. Demski. 1995. Spatial and temporal patterns of spotted wilt epidemics in peanut. Phytopathology. 85:879-985.

Chamberlin J. R., A. K. Culbreath, J. W. Todd and J. W. Demski. 1993. Detection of *Tomato spotted wilt virus* in tobacco thrips (Thysanoptera: Thripidae) overwintering in harvested peanut fields. J. Econ. Entomol. 86:40-45.

Chamberlin J. R., J. W. Todd, R. J. Beshear, A. K. Culbreath, and J. W. Demski. 1992. Overwintering hosts and wingform of thrips, frankliniella spp., in Georgia (Thysanoptera: Thripidae) implications for management of spotted wilt disease. Eviron. Entomol. 21:121-128.

Chatzivassiliou, E. K., T. Nagata, N. I. Katis, and D. Peters. 1999. Transmission of tomato spotted wilt tospovirus by Thrips tabaci populations originating from leek. Plant Pathology. 48:700-706.

Cho, J. J., W. C. Mitchell, and R. F. L. Mau. 1987. Epidemiology of tomato spotted wilt virus disease on crisphead lettuce in Hawaii. Plant Disease. 71:505-508.

Cho, K., C. S. Eckel, J. F. Walgenbach, and G. G. Kennedy. 1995. Overwintering of thrips (Thysanoptera: Thripidae) in North Carolina. Eviron. Entomol. 24:58-67.

Coker, D. 2005. Peanut Variety and Quality Evaluation Results: Agronomic and Grade Data. June 2006. Information Services No. 432-301.

Culbreath, A. K., J. W. Todd, and S. L. Brown. 2003. Epidemiology and management of tomato spotted wilt in peanut. Annu. Rev. Pytopathol. 41:53-75.

Culbreath, A. K., J. W. Todd, D. W. Gorbet, S. L. Brown, J. Baldwin, H. R. Pappu, and F. M. Shokes. 2000. Reaction of peanut cultivars to spotted wilt. Peanut Sci. 27:35-39.

Culbreath, A. K., J. W. Todd, D. W. Gorbet, S. L. Brown, J. A. Baldwin, H. R. Pappu, C. C. Holbrook, and F. M. Shokes. 1999a. Response of early, medium and late maturing peanut breeding lines to field epidemics of tomato spotted wilt. Peanut Sci. 26:100-106.

Culbreath, A. K., J. W. Todd, S. L. Brown, J. A. Baldwin, and H. Pappu. 1999b. A genetic and cultural "package" for management of *Tomato spotted wilt virus* in peanut. Biological and Cultural Tests. 14:1-8.

Culbreath, A. K. J. W. Todd, D. W. Gorbet, F. M. Shokes, and H. R. Pappu. 1997a. Field response of new peanut cultivar UF 991108 to *Tomato spotted wilt virus*. Plant Disease. 81:1410-1415.

Culbreath, A. K. J. W. Todd, D. W. Gorbet, F. M. Shokes, and H. R. Pappu. 1997b. Field performance of advanced runner and Virginia-type peanut breeding lines during epidemics of TSWV. Peanut Sci. 24:123-128.

Culbreath, A.K., J. W. Todd, and D. W. Gorbet. 1996. Disease progress of *Tomato spotted wilt virus* in selected peanut cultivars and advanced breeding lines. Plant Disease. 80:70-73.

Culbreath, A. K., J. W. Todd, J. W. Demski. 1992a. Productivity of Florunner peanut infected with *Tomato spotted wilt virus*. Peanut Sci. 19:11-14.

Culbreath, A. K., J. W. Todd, J. W. Demski. 1992b. Comparison of hidden and apparent spotted wilt epidemics in peanut. Proc. Am. Peanut Res. Ed. Soc. 24:39 (abstr.).

Culbreath, A. K., A. S. Csinos, and T. B. Brenneman. 1991. Association of *Tomato spotted wilt virus* with foliar chlorosis of peanut in Georgia. Plant Disease. 75:863 (abstr.).

Culbreath, A. K., J. W. Todd, and J. W. Demski. 1990. Epidemiology if TSWV On Peanut. Proc. Am. Peanut Res. Ed. Soc. 22:81 (abstr.).

Eckel, C. S., K. Cho, J. F. Walgenbach, G. G. Kennedy, and J. W. Moyer. 1996. Variation in thrips species composition in field crops and implications for tomato spotted wilt epidemiology in North Carolina. Entomol. Exp. App. 78:19-29.

Gallo-Meagher, M. K. Chengalraya, J. M. Davis, and G. E. MacDonald. 2001. Phorate-induced peanut genes that may condition acquired resistance to Tomato spotted wilt. Proc. Am. Peanut Res. Ed. Soc. 33:29 (abstr.)

Garcia, L. E., and R. L. Brandenburg. 2000. Incidence of Tomato spotted wilt virus (Bunyaviridae) and tobacco thrips in Virginia-type peanuts in North Carolina. Plant Diseaee. 84:459-464.

Garica. L. E., G. G. Kennedy, and R. L Brandenburg. 2000. Survival and reproductive success of tobacco thrips on three *Tomato spotted wilt virus* infected and noninfected peanut cultivars. Peanut Sci. 27:49-52.

German, T. L., D. E. Ullman, and J. W. Moyer. 1992. Tospoviruses: diagnosis, molecular biology, phylogeny, and vector relationships. Annu. Rev. Phytopathology. 30:315-348.

Groves, R. L., J. F. Walgenbach, J. W. Moyer, and G. G. Kennedy. 2003. Seasonal dispersal patterns of Fankliniella fusca (Thysanoptera: Thripidae) and *Tomato spotted wilt virus* occurrence in central and eastern North Carolina. J. Econ. Entomol. 96:1-11.

Groves, R. L., J. F. Walgenbach, J. W. Moyer, and G. G. Kennedy. 2001a. Overwintering of Frankliniella fusca (Thysanoptera: Thripidae) on winter annual weeds infected with *Tomato spotted wilt virus* and patterns of virus movement between susceptible weed hosts. Phytopathology. 91:891-899.

Groves, R. L. 2001b. The role of weed hosts and tobacco thrips, Frankliniella fusca Hinds (Thysanoptera: Thripidae), in the epidemiology of tomato spotted wilt tospovirus. Ph.D. diss., North Carolina State University, Raleigh.

Hoffman, K. S., M. Geske, and J. W. Moyer. 1998. Pathogenesis of tomato spotted wilt virus in peanut plants dually infected with peanut mottle virus. Plant Disease. 82:610-614.

Huber, A. 2002. 2003 variety guide. The Peanut Grower. November 2002:21-27.

Huber, A 2006. 2006 variety guide. The Peanut Grower. February 2006: 1-6. Retrieved April 11, 2006, from http://www.peanutgrower.com/home/2006_FebVarietyGuide.html.

Hull, R. 1989. The movement of viruses in plants. Annu. Rev. Phytopathology. 27:213-240.

Hurt, C. A., R. L. Brandenburg, D. L. Jordan, G. G. Kennedy, and J. E. Bailey. 2005. Management of spotted wilt vectored by Frankliniella fusca (Thysanoptera: Thripidae) in Virginia market-type peanut. J. Econ. Entomol. 98:1435-1440.

Hurt, C. A. 2003. Evaluation of cultural practices to reduce the incidence of *Tomato spotted wilt virus* in North Carolina peanut (Arachis hypogaea). M.S. Thesis, North Carolina State University, Raleigh.

Jain, R. K., S. S. Pappu, H. R. Pappu and A. K. Culbreath. 1998. Molecular diagnosis of tomato spotted wilt tospovirus infection of peanut and other field and greenhouse crops. Plant Disease. 82:900-904.

Johnson, W. C. III, T. B. Brenneman, S. H. Baker, A. W. Johnson, D. R. Summer, and B. G. Mullinix, Jr. 2001. Tillage and pest management considerations in a peanut-cotton rotation in the southeastern coastal plain. Agronomy Journal. 93:570-576.

Jordan, D. L. 2006. Peanut Production Practices, pp. 15-34. In 2006 Peanut Information, North Carolina Coop. Ext. Ser. Series AG-331.

Jordan, D. L., J. Spears, R. L. Brandenburg, A. B. Brown, B. Shew, G. T. Roberson, and S. G. Bullen. 2006 Peanut Information, North Carolina Coop. Ext. Ser. Series AG-331.

Jordan, D. L., J. S. Barnes, C. R. Bogle, R. L. Brandenburg, J. E. Bailey, P. D. Johnson, and A. S. Culpepper. 2003. Peanut response to cultivar selection, digging date, and tillage intensity. Agronomy Journal. 95:380-385.

Jordan, D. L., R. L. Brandenburg, J. E. Bailey, P. D. Johnson, B. M. Royals and V. L. Curtis. 1999. Cost effectiveness of pest management strategies in peanut (Arachis hypogaea L.) grown in North Carolina. Peanut Sci. 26:85-94.

Kresta, K. K., F. L. Mitchell and J. W. Smith, Jr. 1995. Survey by ELISA of thrips (Thysanoptera: Thripidae) vectored *Tomato spotted wilt virus* distribution in foliage and flowers and field-infected peanut. Peanut Sci. 22:141-149.

Kindt, F., N. N. Joosten, D. Peters, and W. F. Tjallingii. 2003. Characterisation of the feeding behavior of western flower thrips in terms of electrical penetration graph (EPG) waveforms. Journal of Insect Physiology. 49:183-191.

Lanier, J. E., D. L. Jordan, J. F. Spears, R. Wells, P. D. Johnson, J. S. Barnes, C. A. Hurt, R. L. Brandenburg, and J. E. Bailey. 2004. Peanut response to planting pattern, row spacing, and irrigation. Agronomy Journal. 96:1066-1072.

Llamas-Llamas, M. E., E. Zavaleta-Mejia, V. A. Gonzalez-Hernandez, L. Cervantes-Diaz, J. A. Santizo-Rincon, and D. L Ochoa-Martinez. 1998. Effect of temperature on symptom expression and accumulation of *Tomato spotted wilt virus* in different host species. Plant Pathology. 47:371-347.

Lowry, V. K., J. W. Smith, Jr. F. L. Mitchell, and C. R. Crumley. 1995. Thrips vectors responsible for the secondary spread of *Tomato spotted wilt virus* in Texas peanut, pp. 167-170. In B. L. Parker et al. [eds.], Thrips Biology and Management. Plenum Press, NY.

Luo, M., P. Dang, B. Z. Guo, G. He, C. C. Holbrook, M. G. Bausher, and R. D. Lee. 2005. Generation of expressed sequence tags (ESTs) for gene discovery and marker development in cultivated peanut. Crop Science. 45:346-353.

Lyerly, J. H., H. T. Stalker, J. W. Moyer and K. Hoffman. 2002. Evaluation of Arachis species for resistance to *Tomato spotted wilt virus*. Peanut Sci. 29:79-84.

Magbanua, Z. V., H. D. Wilde, J. K. Roberts, K. Chowdhury, J. Abad, W. Moyer, H. Y. Wetzstein and W. A. Parrott. 2000. Field resistance to *Tomato spotted wilt virus* in transgenic peanut (Arachis hypogaea L.) expressing an antisense nucleocapsid gene sequence. Molecular Breeding. 6:227-236.

Mandal, B., H .R. Pappu, and A. K. Culbreath. 2002. Differential response to selected peanut (Arachis hypogaea) genotypes to mechanical inoculations by *Tomato spotted wilt virus*. Plant Disease. 86:939-943.

Mandal, B., H. R. Pappu, and A. K. Culbreath. 2001a. Factors affecting Mechanical transmission of *Tomato spotted wilt virus* to peanut (Arachis hypogaea). Plant Disease. 85:1259-1263.

Mandal, B., H. R. Pappu, A. K. Culbreath, and J. W. Todd. 2001b. Movement and accumulation of *Tomato spotted wilt virus* in peanut (peanut). Phytopathology. 91:S57. (abstr.).

Marois, J. J., and D. L. Wright. 2003. Effect of tillage system, phorate, and cultivar on tomato spotted wilt of peanut. Agronomy Journal. 95:386-389.

Mitchell, F. L. 1996. Implementation of the IPM planting window for management of *Tomato spotted wilt virus* and avoidance of peanut yellowing death. Final Compliance Report, Texas Pest Management Association, Biologically Intensive Integrated Pest Management Grant Program. http://stephenville.tamu.edu/~fmitchel/ento/tswv3.pdf.

Mitchell, F. L., V. K. Lowry, K. K. Kresta, and J. W. Smith, Jr. 1995. Histological study of tobacco thrips feeding on peanut foliage, pp 175-178. In B. L. Parker et al. [eds.], Thrips Biology and Management. Plenum Press, NY.

Mound, L. A. 1996. The Thysanoptera vector species of tospoviruses. Acta Horticulturae. 431: 298-309.

Mulder, P. G., C. L. Cole, M. A. Karner, and J. R. Bolte. 1991. Seasonal prevalence of the Thysanoptera in an Oklahoma peanut ecosystem and potential for *Tomato spotted wilt virus*. Southwestern Entomologist. 16:108-116.

Nascimento, L. C., V. Pensuk, F. M. Assis Filho, C. M. Deom, and J. L. Sherwood. 2003a. Evaluation of peanut genotypes for resistance to *Tomato spotted wilt virus* by mechanical and thrips inoculation. Phytopathology. 93:S64 (abstr.).

Nascimento, L. C., F. M. Assis Filho, O. R. Paguio, C. M. Deom, and J. L. Sherwood. 2003b. Transformation of peanut with the *Tomato spotted wilt virus* non-structural gene from the S RNA. Phytopathology. 93:S64 (abstr.).

Nagata, T., A. C. L. Almeida, R. O. Resende, and A. C. de Avila. 2004. The competence of four thrips species to transmit and replicate four tospoviruses. Plant Pathology. 53:136-140.

Nagata, T., A. K. Inoue-Nagata, J. van Lent, R. Goldbach, and D. Peters. 2002. Factors determining vector competence and specificity for transmission of *Tomato spotted wilt virus*. Journal of General Virology. 83: 663-671.

Nagata, T., A. K. Inoue-Nagata, M. Prins, R. Goldbach and D. Peters. 2000. Impeded thrips transmission of defective *Tomato spotted wilt virus* isolates. Phytopathology. 90:454-459.

Pappu, S. S., H. R. Pappu, A. K. Culbreath, and J. W. Todd. 1999. Localization of *Tomato spotted wilt virus* (Genus Tospovirus, Family Bunyaviriae) in peanut pods. Peanut Sci. 26:98-100.

Peters, D., A. C. de Avila, E. W. Kitajima, R. de Resende, P. de Haan and R. W. Goldbach. 1991. An overview of *Tomato spotted wilt virus*, pp. 1-14. In H. T. Hsu and R. H. Lawson [eds.] Virus-Thrips-Plant Interactions of *Tomato spotted wilt virus*. Proceedings USDA Workshop, United States Department of Agriculture Research and Service ARS-87; Beltsville, MD.

Puche, H. and J. Funderburk. 1992. Intrinsic rate of increase Frankliniella fusca (Thysanoptera: Thripidae) on peanuts. Florida Entomologist. 75:185-189.

Rowland, D., J. Dorner, R. Sorensen, J. P. Beasley, Jr., and J. Todd. 2005. *Tomato spotted wilt virus* in peanut tissue types and physiological effects related to disease incidence and severity. Plant Pathology. 54:431-440.

Sakimura, K. 1963. Frankliniella fusca, an additional vector for the tomato spotted wilt virus, with notes on Thrips tabaci, another vector. Phytopathology. 53:412-415.

Sakimura, K. 1962a. The present status of thrips-borne viruses, pp 33-40. In K. Maramorosch [ed.], Bilog. Transm. Of Dis. Agents. Academic, NY.

Sakimura, K. 1962b. Frankliniella occidentalis (Thysanoptera: Thripidae), a vector of the *Tomato spotted wilt virus*, with special reference to the color forums. Ann. Entomol. Soc. Of Am. 55:387-389.

Sherwood, J. L. 1990. Methods for detection of *Tomato spotted wilt virus*. Proc. Am. Peanut Res. Ed. Soc. 22:82 (abstr.).

Sin, S.-H., B. C. McNulty, G. G. Kennedy, and J. W. Moyer. 2005. Viral genetic determinant for thrips transmission of *Tomato spotted wilt virus*. Proc. Natl. Acad. Sci. USA. 102:5168-5173.

Shew, B. Peanut Disease Management, pp. 79-104. In 2006 Peanut Information, North Carolina Coop. Ext. Ser. Series AG-331.

Stumpf, C. and G. G. Kennedy. 2005. Effects of *Tomato spotted wilt virus* (TSWV) isolates, host plants, and temperature on survival, size, and development time of Frankliniella fusca. Entomologia Experimentalis et Applicata. 114:215-225.

Sparks, A. and D. G. Riley. Insect identification and management: Thrips (Order: Thysanoptera, Family: Thripidae). Retrieved Sep. 4, 2006, from University of Georgia, Dept of Entomology. http://www.ent.uga.edu/veg/solanaceous/thrips.htm.

Tappan, W. B. 1986. Relationship of sampling time to Tobacco Thrips (Thysanoptera: Thripidae) numbers in peanut foliage buds and flowers. J. Econ. Entomol. 79:1359-1363.

Terry, L. I. 1997. Host selection, communication and reproductive behavior, pp. 65-118. In T. Lewis [ed.], Thrips as crop pests. CAB International.

Todd, J. W., A. K. Culbreath, J. R. Chamberlin, R. J. Beshear, and B. G. Mullinix. 1995. Colonization and population dynamics of thrips in peanuts in the southern United States, pp. 453-460. In B. L. Parker et al. [eds.], Thrips Biology and Management. Plenum Press, NY.

Todd, J. W., A. K. Culbreath, J. W. Demski, and R. Beshear. 1990. Thrips as vectors of TSWV. Proc. Am. Peanut Res. Ed. Soc. 22:81 (abstr.).

Ullman, D. E., A. E. Whitfield, and T. L. German. 2005. Thrips and tospoviruses come of age: mapping determinants of insect transmission. Proc. Natl. Acad. Sci. USA 102:4931-4932.

Ullman, D. E. 1996. Thrips and tospoviruses: Advances and future directions. Acta. Horticulturae. 431:310-324.

Ullman, D. E., T. L. German, J. L. Sherwood, and D. M. Westcot. 1995a. Thrips transmission of Tospoviruses: future possibilities for management, pp. 135-151. In B. L. Parker et al. [eds.], Thrips Biology and Management. Plenum Press, NY.

Ullman, D. E., D. M. Westcot, K. D. Chenault, J. L Sherwood, T. L. German, M. D. Bandla, F. A. Cantone, and H. L. Duer. 1995b. Compartmentalization, intracellular transport, and autophagy of Tomato spotted wilt tospovirus proteins in infected thrips cells. Phytopathology. 85:644-654.

Ullman, D. E., T. L. German, J. L. Sherwood, D. M. Westcot, and F. A. Cantone. 1993. Tospovirus replication in insect vector cells: immunocytochemical evidence that the nonstructural protein encoded by the S RNA of tomato spotted wilt tospovirus is present in thrips vector cells. Phytopathology. 83:456-463.

Ullman, D. E., J. J. Cho, R. F. L. Mau, D. M. Westcot, and D. M. German. 1992. A midgut barrier to *Tomato spotted wilt virus* acquisition by adult Western Flower thrips. Phytopathology. 82:1333-1342.

Wehtje, G., R. Weeks, M. West, L. Wells, and P. Pace. 1994. Influence of planter type and seeding rate on yield and disease incidence in peanut. Peanut Sci. 21:16-19.

Wells, M. L., A. K. Culbreath, J. W. Todd, S. L. Brown, and D. W. Gorbet. 2002a. A regression approach for comparing field resistance of peanut cultivars to tomato spotted wilt tospovirus. Crop Protection. 21:467-474.

Wells, M. L., A. K. Culbreath, J. W. Todd, A. S. Csinos, B. Mandal, and R. M. McPherson. 2002b. Dynamics of spring tobacco thrips (Thysanoptera: Thripidae) populations: Implications for *Tomato spotted wilt virus* management. Environmental Entomology. 31:1282-1290.

Wetering, F. van de, M. van der Hoek, R. Goldbach, and D. Peters. 1999. Differences in *Tomato spotted wilt virus* vector competency between males and females of Frankliniella occidentalis. Entomologia Experimentalis et Applicata. 93:105-112.

Wetering, F. van de, J. Hulshof, K. Posthuma, P. Harrewijn, R. Goldbach, and D. Peters. 1998. Distinct feeding behavior between sexes of Frankliniella occidentalis results in higher scar production and lower tospovirus transmission by females. Entomologia Experimentalis et Applicata. 88:9-15.

Wetering, F. van de, R. Goldbach, and D. Peters. 1996. Tomato spotted wilt tospovirus ingestion by first instar larvae of Frankliniella occidentalis is a prerequisite for transmission. Phytopathology. 86:900-905.

Whitfield, A. E., D. E. Ullman, and T. L. German. 2005. Tospovirus-thrips interactions. Annu. Rev. Phytopathol. 43:459-89.

Wijkamp, I., N. Almarza, and D. Peters. 1995a. Median latent period and transmission of tospoviruses vectored by thrips, pp. 153-156. In B. L. Parker et al. [eds.], Thrips Biology and Management. Plenum Press, NY.

Wijkamp, I., J. van Lent, R. Kormelink, R. Goldbach, and D. Peters. 1995b. Multiplication of *Tomato spotted wilt virus* in western flower thrips, pp. 157-161. In B. L. Parker et al. [eds.], Thrips Biology and Management. Plenum Press, NY.

Wijkamp, I., N. Almarza, R. Goldbach, and D. Peters. 1995c. Distinct levels of specificity in thrips transmission of tospoviruses. Phytopathology. 85:1069-1074.

Wijkamp, I. and D. Peters. 1993. Determination of the median latent period of two tospoviruses in Frankliniella occidentalis, using a novel leaf disk assay. Phytopathology. 83:986-991.

Yang, H., P. Ozias-Akins, A. K. Culbreath, D. W. Gorbet, J. R. Weeks, B. Mandal, and H. R. Pappu. 2004. Field evaluation of *Tomato spotted wilt virus* resistance in transgenic peanut (Arachis hypogaea). Plant Disease. 88:259-264.

Zhijian, L., R. L. Jarret, and J. W. Demski. 1997. Engineered resistance to *Tomato spotted wilt virus* in transgenic peanut expressing the viral necleocapsid gene. Transgenic Research. 6:297-305.