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# Epidemiology of spotted wilt disease of peanut caused by *Tomato spotted wilt virus* in the southeastern U.S.

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ABSTRACT

Spotted wilt disease of peanut (*Arachis hypogaea*) (SWP), caused by *Tomato spotted wilt virus* (TSWV) (genus *Tospovirus*, family *Bunyaviridae*), was first observed in Alabama, Florida, and Georgia in the late 1980s and rapidly became a major limiting factor for peanut production in the region. Tobacco thrips (*Frankliniella fusca*) and western flower thrips (*Frankliniella occidentalis*) both occur on peanut throughout the southeastern U.S., but *F. fusca* is the predominant species that reproduces on peanut, and is considered to be the more important vector. Several non-crop sources of potential primary vectors and TSWV inoculum have been identified, but their relative importance has not been determined. The peanut growing season in Alabama, Florida, and Georgia is from April through November, and 'volunteer' peanut plants can be present for much of the remainder of the year. Therefore peanut itself has huge potential for perpetuating both vector and virus. Symptoms are often evident within a few days of seedling emergence, and disease progress is often rapid within the first 50–60 days after planting. Based on destructive sampling and assays for TSWV, there is often a high incidence of asymptomatic infections even in peanut genotypes that produce few and mild symptoms of infection in the field. Severity of SWP epidemics fluctuates significantly from year to year. The variability has not been fully explained, but lower incidences have been associated with years categorized as "La Niña" in the El Niño-Southern Oscillation. Planting date can have a large effect on disease incidence within a location. This may be linked to the thrips reproductive cycle and environmental effects on the plant and plant–thrips–virus interactions. Row pattern, plant population, and in-furrow applications of phorate insecticide can also affect epidemics of SWP. Considerable progress has been made in developing cultivars with natural field resistance to TSWV. Use of cultivars with moderate field resistance combined with other suppressive measures has been very successful for managing spotted wilt disease. Several new cultivars with higher levels of field resistance can improve control and allow more flexibility in the integrated management programme. Although effects of these factors on epidemics of SWP have been documented, mechanisms responsible for disease suppression by most factors have not been fully elucidated.



## 1. Introduction

Production of peanut (*Arachis hypogaea*) in the U.S. is heavily concentrated in the coastal plain region of Alabama, Florida, and Georgia. These three states account for over 70% of the U.S. production, and almost half comes from Georgia (U.S.D.A. National Agricultural Statistics Service, 2010). Historically, diseases caused by fungal pathogens have been major limiting factors for peanut production in the southeastern U.S. However, since the late 1980s, spotted wilt disease of peanut (SWP), caused by thrips-transmitted *Tomato spotted wilt virus* (TSWV) (genus *Tospovirus*, family *Bunyaviridae*) has had a major deleterious impact on peanut production in the region, both in terms of losses to the disease and in major shifts in cropping practices.

## 2. History

A disease of peanut caused by TSWV was first reported by Costa (1941) in Brazil. Subsequent occurrences of SWP or diseases caused by other tospoviruses in peanut have since been reported from other parts of the world (Halliwell and Philley, 1974; Helms et al., 1961; Klesser, 1966; Reddy et al., 1968).

Although SWP occurs on peanut in production areas of South America (Costa, 1941), the impact of the disease in larger peanut producing areas there has not been as great as in North America. In the U.S., SWP was first reported in Texas in 1971 (Halliwell and

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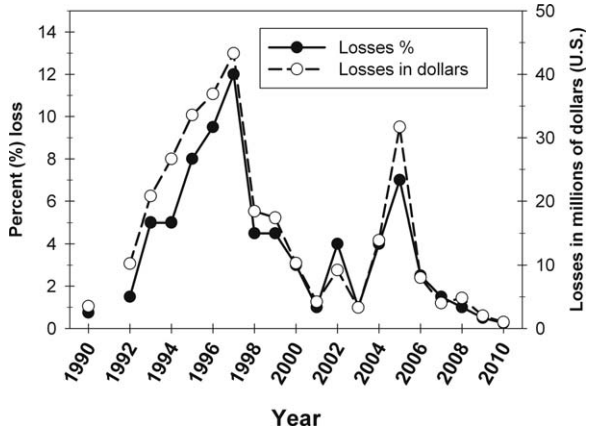


**Fig. 1.** Losses caused by spotted wilt disease of peanut in Georgia from 1990 to 2010. Closed circles represent percentage of the peanut crop lost to the disease. Open circles represent estimated losses in U.S. dollar value.

Philley, 1974). It has since become one of the most serious diseases of peanut in the U.S., especially in Alabama, Florida, and Georgia (Culbreath et al., 2003). In 1985, losses of approximately 50% of the peanut crop were incurred in production areas of southern Texas, with almost total losses in some individual fields (Black et al., 1986). Severe losses were also reported in the small production area in Mississippi in 1986 (Reed and Sukamto, 1995). In that year, SWP was identified in Alabama (Hagan et al., 1990) and Georgia (Todd et al., 1995). Losses due to SWP in Georgia were first noted in 1990 (Hadden, 1991) (Fig. 1).

Losses to SWP in peanut increased dramatically from the late 1980s through 1997, when losses were estimated to be 12% of the entire crop for Georgia alone, representing an approximate value of $40 million (Bertrand, 1998) (Fig. 1). More recently, significant reductions in severity and losses have been noted (Williams-Woodward, 2010) (Fig. 1). Since the emergence of SWP as a serious threat to peanut production, interdisciplinary, inter-state and inter-agency cooperative efforts have resulted in the development of an integrated programme that has been very successful for managing this disease. The objective of this paper is to discuss the epidemiological aspects of the key factors that are used or have potential for use in that programme.

## 3. Symptoms

Symptoms of SWP vary greatly. They include concentric ringspots (Fig. 2A), and various patterns of chlorosis on leaflets (Fig. 2B), stunting of all above-ground plant parts (Fig. 3), small and/or misshapen geocarpophores (usually referred to as “pegs”), pods and kernels, and reddish discoloration and cracking of seed coats (Costa, 1941; Culbreath et al., 1992a; Halliwell and Philley, 1974). Severity of symptoms ranges from minor spotting on one or a few leaflets with little apparent damage to yield, to severe stunting and death of entire plants. Number of pods produced, kernel size and yield per plant can be greatly reduced, and plants showing symptoms early in the season typically yield less than those in which symptoms develop later (Culbreath et al., 1992a). TSWV is also associated with a general chlorosis and wilting of peanut plants that may not be accompanied by typical above-ground symptoms (Culbreath et al., 1991).

Asymptomatic infections are also common. Based on immunoassays of root tissue from field-grown plants, Culbreath et al. (1991) and Murakami et al. (2006) reported an incidence of asymptomatic infections as high as that of disease incidence based on visible foliar symptoms. Even genotypes with high levels of field resistance to TSWV, based on incidence and severity of symptoms can have high incidence of inapparent infection. In the 2004 field trial at Tifton, Georgia, average final incidence of SWP ranged from 68.1% in cv SunOleic 97R to 2.3% in breeding line F NC 94022 (LSD = 10.2, $P = 0.05$) (Culbreath et al., 2005). In addition to evaluations reported in that paper, tap root samples were collected from nine plants of each genotype in each of the six replications after the plants were uprooted. Roots were assayed by ELISA for the presence of TSWV. Incidences of detection of TSWV for cvs SunOleic 97R, Georgia Green, C11-186, C11-2-39 and F NC 94022 were 100, 100, 85, 85, and 69%, respectively, (LSD = 12, $P = 0.05$).

## 4. Initial inoculum and thrips vectors

In natural epidemics in peanut, transmission of TSWV by viruliferous thrips is the only known significant means of dissemination (Sakimura, 1962, 1963; Todd et al., 1995; Ullman et al., 2002). TSWV can be mechanically inoculated into peanut, but often not easily (Mandal et al., 2001). Although TSWV is detectable in pods and testae of peanut kernels (Pappu et al., 1999), there is no indication that seed transmission occurs in peanut (Costa, 1941; Pappu et al., 1999; A.K. Culbreath, unpublished data).

Tobacco thrips, *Frankliniella fusca* and western flower thrips, *Frankliniella occidentalis*, are competent vectors of TSWV (Sakimura, 1962, 1963), and both species occur in peanut producing areas of the southeastern U.S. (Todd et al., 1995). Emergence of SWP as a problem in peanut in the southeastern U.S. followed soon after initial detection of western flower thrips in the region (Beshear, 1983; Hagan et al., 1990; Todd et al., 1995). The timing of those occurrences prompted speculation that the two events were related. Although the western flower thrips occurs on peanut in much of the southeastern U.S., typically it is not as numerous, and does not reproduce as well on peanut as the tobacco thrips (Todd et al., 1995, 1996). Once TSWV became established, the tobacco thrips that was already endemic to the southeastern U.S. acted as a competent vector of the virus. In most of the southern U.S., it is considered the predominant vector species of TSWV in peanut (Todd et al., 1995, 1996; Weeks and Hagan, 1991).

Determining the sources of primary vectors and TSWV inoculum has been a major goal since the initial occurrence of SWP. Several potential sources have been identified, but the relative importance of these sources for epidemics in peanut remains largely unknown. TSWV, western flower thrips and tobacco thrips all have a wide host range, and infest many crop and noncrop plant species that occur in peanut growing areas (Chamberlin et al., 1992a, 1993a,b; Todd et al., 1995, 1996). Several species of winter and spring weeds have been found to have significant incidences of infection with TSWV (Mullis and Martinez-Ochoa, 2009). Some of those species were also among those reported from surveys in North Carolina (Groves et al., 2001, 2002), and some are hosts for one or both of the key thrips vectors. For a plant to be a significant source of inoculum, it must be a host of TSWV, and also must support reproduction of at least one of the vector species. Moreover, thrips must able to acquire the virus from the plant, and the plant must occur at a time that would complement disease cycles. The role of weeds in the disease cycle in the southeastern U.S is not clearly elucidated. There are stronger indications of weeds as a key source of inoculum for epidemics in peanut and other crops to the north in North Carolina (Groves et al., 2002). However, in Georgia, with over 200,000 hectares of peanuts grown in most years, and a climate that often allows ‘volunteer’ peanut plants to survive much of the year, a weed ‘bridge’ may be unnecessary. The peanut growing season typically lasts from April through early November. ‘Volunteer’ peanuts often emerge shortly after harvest in many fields and may survive much of the winter. In the spring, emergence of additional ‘volunteer’ peanut plants may