INTERACTIONS OF NEMATODES AND FUNGAL WILT PATHOGENS

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The interaction between plant-parasitic nematodes and fungal wilt pathogens is of both historical and practical significance. In 1892, Atkinson reported that a type of eel worm (*Meloidogyne incognita* (Kofoid & White) Chitwood) was associated with increased severity of a disease of cotton known at that time as frenching (later identified as *Fusarium* wilt caused by *F. oxysporum* Schlecht f. sp. *vasinfectum* Atk. Sny. & Hans.). This report not only was the first time a nematode and a fungal pathogen were associated in a disease complex, but it was also one of the first reports of a plant-parasitic nematode species parasitizing a crop in the United States.

Interactions between nematodes and various fungi have been reported in a number of crop species, but the association between *Meloidogyne* spp. and *Fusarium* spp. are by far the most common. Interactions between these organisms have been reported for numerous crop species including alfalfa, beans, cabbage, cowpea, muskmelon, tobacco, and tomato (Mai & Abawi, 1987). In cotton, limited studies indicate an association of *F. oxysporum* f. sp. *vasinfectum* with the sting nematode, *Belonolaimus longicaudatus* (Holdeman & Graham, 1954), the reniform nematode, *Rothylechulus reniformis* (Neal, 1954), and the lance nematode, *Hoplolaimus seinhorsti* (Rajaram, 1979). Both *M. incognita* and *R. reniformis* have also been associated with increased incidence of *Verticillium* wilt, caused by *Verticillium dahlae* (Bridge, J. 1992), although the significance of these associations in the field have not been determined.

Interactions between nematodes and fungal pathogens, or disease complexes, are generally defined as being either additive or synergistic (Powell, 1979). Additive interactions result in host damage that equals the sum of the damage caused by each organism alone. Synergistic interactions, on the other hand, result in damage to the host that is greater than would be expected from the sum of the damage due to each individual. Unfortunately, these definitions may not appropriately describe many pathogen-nematode associations, including the *Fusarium* wilt-root-knot nematode disease complex, where fungal and nematode population densities and environmental factors influence the degree and severity of the interaction (Starr, *et al.*, 1989).

Various hypotheses have been developed to explain the nature of the interaction between *M. incognita* and *F. oxysporum* f.sp. *vasinfectum* in cotton. One suggested mechanism is the possibility that the nematode vectors the fungus, introducing fungal propagules into the cotton root during penetration or feeding. However, vectoring by the nematode, at least in the sense of actual introduction of the fungus into the root system through the feeding process, does not appear to be plausible because the stylet of most plant-parasitic nematodes is much too small for fungal spores to enter. Transport into the root via conidia adhering to the cuticle of the nematode may, however, be possible although the phenomenon has not been documented (Mai & Abawi, 1987). Non-stylet-bearing nematodes may play a role in the epidemiology of the disease complex by transporting spores within the rhizosphere, although vectoring as defined in most vector-virus associations does not occur (Taylor, 1979).

A much more widely held view of the role of the nematode in the disease complex has been that nematodes provide entry points for the fungus through wounds produced in the root during nematode penetration. However, studies have demonstrated that wilt is much more severe when cotton plants are inoculated two to four weeks prior to inoculation by the fungus (Powell, 1971). Penetration wounds made by *M. incognita* juveniles were not attractive to *F. oxysporum* f. sp. *vasinfectum*, and there was no increase in fungal colonization at these sites (Perry, 1963). In addition, studies in tomato and cowpea have demonstrated that mechanical wounding of roots increased susceptibility to *F. oxysporum* f. sp. *vasinfectum* to a much lower degree than when plants were inoculated by root-knot nematodes (Jenkins & Coursen, 1957; Thomason *et al.*, 1959). It is possible root wounding that occurs when egg masses of mature females erupt through the root cortex rather than penetration wounds at infection may provide entry to the fungus (Shepherd & Huck, 1989). In cotton, root-knot females mature within three to four weeks after infection and egg masses are generally present on the root surface. This timing appears to correspond well with the period of greatest susceptibility to the fungus.

The enhanced susceptibility of cotton and other crops to *F. oxysporum* f. sp. *vasinfectum* due to nematode infection appears most likely to be the result of changes in the host physiology brought about by nematode parasitism. Nematode-induced susceptibility appears to be systemic, and plants infected by the nematode show increased susceptibility to stem inoculation (Hillocks, 1985). Cellular changes in the host in response to infection may be primarily responsible for the increased susceptibility to the wilt fungus. Giant cells and parenchyma cells adjacent to them are characterized by an increase in nutrient content (Owens & Specht, 1966; Sidhu & Webster, 1977).

In tobacco, hyphal size and growth rate are increased in galls produced by the nematode than in non-infected root tissue (Melendez & Powell, 1967). Giant cells and those cells adjacent to them are most metabolically active three to four weeks after infection, and these cells are unlikely to form tyloses or balloon-like expansions of xylem parenchyma cell

Reprinted from the *Proceedings of the Beltwide Cotton Conference*
Volume 1:123-125 (1998)
National Cotton Council, Memphis TN

walls that restrict the growth of the fungus in the vascular system (Webster, 1985).

In contrast to tomato, genetic resistance in cotton to *F. oxysporum* f. sp. *vasinfectum* is not broken by infection by root-knot. Infection by the nematode increases susceptibility in both resistant and susceptible cultivars, but wilt resistant cultivars still exhibit lower disease severity than susceptible cultivars in its presence (Hillocks, 1992). Root-knot appears to lower the threshold of *F. oxysporum* f. sp. *vasinfectum* required to elicit symptoms in cotton. In greenhouse pot studies, wilt symptoms were similar with 650 fungal propagules g^{-1} and 3,000 *M. incognita* juveniles as with 647,000 fungal propagules g^{-1} alone (Garber *et al.*, 1979). Significant interaction, measured by plant mortality, between the organisms occurred at intermediate fungal propagule densities and high nematode populations, but interactions did not occur at low nematode or high fungal population densities (Starr *et al.*, 1989). It appears that in cotton, the incidence and severity of the interaction between *F. oxysporum* f. sp. *vasinfectum* and *M. incognita* is dependent on the population densities of both pathogens, particularly the population density of the nematode.

Control of the *Fusarium* wilt-root-knot nematode disease complex has been accomplished through soil fumigation for nematode control and through genetic resistance. Fumigation has been shown to effectively control the complex in the field (Jorgenson, *et al.*, 1978). Unfortunately, use of fumigants for nematode control has been severely curtailed due to cost and difficulty of application, and to health and environmental impact concerns. Non-fumigant nematicides may be somewhat effective in lowering wilt severity, but efficacy is not as dependable as with fumigants (Colyer, *et al*, 1997). Genetic resistance to both organisms has been utilized. Effective resistance must include resistance to both organisms, but commercially available cultivars with high levels of root-knot resistance are extremely limited. Very effective levels of nematode resistance have been reported in cotton breeding lines (Shepherd, R.L., 1982a), and the utility of this germplasm in minimizing the effects of the disease complex have been demonstrated (Shepherd, R.L., 1982b). However, this material has not been effectively incorporated into commercially acceptable, high yielding cotton cultivars (A.F. Robinson, 1998). However, moderate levels of *M. incognita* resistance have been utilized in a few cultivars (Jones, *et al.*, 1991; Robinson, 1998). This degree of nematode resistance significantly lowers wilt severity and yield suppression due to the disease complex. Future improvements in agronomic characteristics of cultivars with both a high level of root-knot resistance and resistance to *F. oxysporum* f. sp. *vasinfectum* should further improve control of this disease complex.

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