A foot and root rot disease of tomato caused by Fusarium oxysporum

W. R. Jarvis, H. J. Thorpe', and B. H. MacNeill[2]

A widespread and serious foot rot disease of tomato which occurred in the greenhouse crops of the Leamington area of Ontario in the spring of 1974 was caused by ***Fusarium oxysporum*** of undetermined forma specialis. Isolates were pathogenic to cultivars resistant to races 1 and 2 of ***F. oxysporum*** f. sp. ***lycopersici.*** The fungus caused cortical rotting in the hypocotyl where adventitious roots arose, as well as a root rot. Vascular discoloration was present in the stem, but only for short distances from the rotted zone, though wilting reminiscent of that caused by *F. **oxysporum*** f. sp. lycopersicioccurred.

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Un genre de pourridie grave et repandu de la tomate qui s'est manifeste dans les cultures de serre de la region de Leamington (Ont.) au printemps de 1974, a ete cause par ***Fusarium oxysporum*** de la forme indeterminee specialis. Les isolats de ce champignon se sont révélés pathogenes pour les cultivars resistants aux races 1 et 2 de ***F. oxysporum*** f. sp. ***lycopersici.*** Ce champignon a cause la pourriture du cortex de l'hypocotyle ou poussent les racines adventives, ainsi qu'un genre de pourridie. On a constaté une decoloration vasculaire de la tige, mais seulement sur de courtes distances depuis les zones de pourriture. même s'il s'est produit un fletrissement comparable a celui cause par *F. **oxysporum*** f. sp. ***lycopersici.***

Symptoms

In the spring of 1974 and again in the fall our attention was drawn to a widespread wilt of tomatoes in greenhouses of the Leamington area, southwestern Ontario, the cultivars affected being WR25, Vendor, and MR13, with some crops affected *to* the extent of about 30%. The same syndrome occurred simultaneously at Cleveland, Ohio (Dr. **J.** M. Farley, personal communication). The disease was characterized by a sudden wilt, particularly in sunny weather, the upper leaves wilting first, the lower leaves then turning golden-yellow from the tip and dying. These symptoms, however, followed a chocolate-brown cortical rot at soil level and reddish-brown vascular discoloration extending upwards for 4 or 5 cm. Primary, secondary, and tertiary roots, as well as the initial tap root, had dark reddish-brown lesions, many confluent with hypocotyl lesions.

Causal organism

Isolations from naturally infected plants consistently yielded ***Fusarium oxysporum,*** usually alone. The fungus was identified mycologically by Dr. R.A. Shoemaker, Biosystematics Research Institute, Ottawa, and chemotaxonomically in the Guelph laboratory by means of the benomyl-plate test (3). The fungus, for the most part, was limited to the discolored sites on hypocotyl and stem; in this respect it differed from the wilt pathogen ***F. oxysporum*** f.sp. ***lycopersici*** which is systemic in its host (2). The disease resembles the root rot described in California (1), except for the limited leaf necrosis noted under Ontario conditions.

Inoculations

Small numbers of seedlings of the cultivars WR25, MR13, and Vendor raised in infested soil damped off about 1 week after emergence. Those that survived had brown lesions in the transition zone, ranging from only slight discoloration to complete rotting of the tap root. When these survivors, 16 days old, were repotted into steamed compost,. most quickly developed adventitious roots, but 4 weeks after potting, the lesions had enlarged, often resulting in the total destruction of the true root system, the breakdown of the lower hypocotyl cortex, and reddish-brown discoloration extending 2-4 cm up the vascular system. Some of the adventitious roots also developed brown lesions at the tip and these often extended back to the hypocotyl where annular cortical lesions then formed. In some cases several cortical lesions surrounding infected roots had coalesced into large brown lesions 2-3 cm above the basal lesion. In young roots infected from the tip, the stele became discolored early and the lesion progressed faster there than in the cortex. The metaxylem often rotted out completely leaving a hollow stele that extended into the hypocotyl vascular system.

When seedlings, 25, 32, or 39 days old, were inoculated by pouring a spore suspension into the soil around them, small discrete (<0.5 mm) superficial brown lesions were formed at soil level, but they have not been seen to develop further. Also, annular cortical lesions sometimes appeared in the hypocotyl around symptomless adventitious roots; infection appeared to have occurred in the ruptured hypocotyl epidermis and cortex as the root broke through. Here, the lesion progressed along the endogenous root, rotting its cortex as well as the neighboring hypocotyl cortex until it reached the hypocotyl vascular system; then the vascular system of both stem and root became discolored. Root tips and

[1] *Research Station. Agriculture Canada, Harrow, Ontario, NOR 1GO*

[2] *Department of Environmental Biology, University of Guelph, Guelph, Ontario N1G 2W1*

secondary root initials were also sites of aggressive infection.

Seedlings, 27 days old, inoculated by the bare-root-dip method and planted into sterilized potting compost were usually quickly killed by large lesions on the tap root and hypocotyl, though some survived by means of adventitious roots above the lesions. The highly aggressive nature of the isolate could be modified by lowering the inoculum level. When the fungus was grown in Czapek's-vermiculite medium and then added at high dilutions to natural soils, the onset of symptoms could be delayed until the plants began to produce flowers, a situation comparable to that encountered in growers' greenhouses. In such plants the fungus was found to be somewhat more systemic than when the inoculum level was high, and could be reisolated from the fifth node of the host.

Epidemiology

A limited analysis of the distribution and spread of the disease made in the fall in commercial ground beds suggests that infection was at random from the soil and that the disease then spread along the rows. Wilted plants often bore lesions with profuse sporulation from soil level to about 5 cm upwards but occasionally to as much as 30 cm. It seems likely that spores could be readily dispersed along and across rows by water from mobile irrigation systems.

Control

There is no control measure yet known; commercial steaming, either by buried tile ducts or by surface application beneath plastic sheets, and Vorlex fumigation have failed to eradicate the disease between spring and fall crops, though the incidence in some cases was considerably lower in the fall crop. The locally important cultivars so far tested, WR25, MR12, MR13, Vendor, and Walter, together with Bonny Best, were all susceptible when inoculated by the root-dip method. The susceptibility of other cultivars is unknown.

Conclusions

This disease appears to be distinct from the epinasty caused by *F. oxysporurn* f. sp. *lycopersici* (4) in that here cortical necrosis and limited vascular discoloration are different symptoms. Further, the fungus seems to be limited in its invasion of the plant. If the fungus is *F. oxysporurn* f. Sp. *lycopersici,* then it is a new race, both on the grounds of cultivar host range and of symptoms.

Acknowledgments

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