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Management of tomato diseases caused by *Fusarium oxysporum*

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ABSTRACT

Fusarium wilt (FW) and Fusarium crown and root rot (FCRR) of tomato (*Solanum lycopersicum*) caused by *Fusarium oxysporum* f. sp. *lycopersici* and *F. oxysporum* f. sp. *radicis-lycopersici*, respectively, continue to present major challenges for production of this important crop world-wide. Intensive research has led to an increased understanding of these diseases and their management. Recent research on the management of FW and FCRR has focused on diverse individual strategies and their integration including host resistance, and chemical, biological and physical control.

1. Background

Fusarium oxysporum represents a species complex that includes many important plant and human pathogens and toxigenic microorganisms (Nelson et al., 1981; Laurence et al., 2014). Diseases caused by *Fusarium* spp., especially Fusarium wilt (FW) and Fusarium crown and root rot (FCRR) in tomato (*Solanum lycopersicum* L., formerly, *Lycopersicon esculentum* Mill.), have been, and continue to be, among the most intensively studied plant diseases. FW, caused by *F. oxysporum* f. sp. *lycopersici* Snyder and Hansen (*Fol*), was first described over 100 years ago in the UK (Massee, 1895), and FCRR, caused by *F. oxysporum* f. sp. *radicis-lycopersici* Jarvis and Shoemaker (*Forl*), was first observed in 1969 in Japan (Sato and Araki, 1974). Tomato is considered the second most important vegetable crop after potato; worldwide tomato production was estimated at about 162 million MT in 2012 (Anonymous, 2014). At present, both pathogens cause extensive losses to this important vegetable crop in the field and greenhouse, and remain major limiting factors for tomato production. Losses from FW can be very high given susceptible host-virulent pathogen combinations (Walker, 1971); yield losses of up to 45% were recently reported in India (Ramyabharathi et al., 2012). Losses from FCRR in greenhouse tomato have been estimated at up to 90% and 95% in Tunisia and Canada, respectively, and the disease has been observed at an incidence of 100% in the field in the USA (Hibar et al., 2007; Jarvis et al., 1983; McGovern et al., 1998).

2. Biology and epidemiology

2.1. Survival and dissemination

2.1.1. Conidia

Both *Fol and Forl* produce three types of asexual infectious spores: macroconidia, microconidia and chlamydospores; a sexual or anamorphic stage for *F. oxysporum* has not been described. *Fol* and *Forl* are indistinguishable morphologically but can be differentiated by host range, the symptoms that they cause in tomato, optimal disease environment, and by molecular techniques (refer to *Host range*, *Symptoms* and *Molecular techniques* below).

Macroconidia have been implicated in aerial dissemination of *Fol*, and both microconidia and macroconidia have been linked to

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the spread of *Forl* (Katan et al., 1997; Rekah et al., 2000; Rowe et al., 1977). Such aerial spread suggests the possibility of a polycylical phase for FW and FCRR, which is unusual for soilborne diseases. Mycelia of the pathogens can survive in association with plant debris as saprophytes and alternate hosts, and, most importantly, as thick-walled chlamydospores which enable long-term survival. Chlamydospores arise from modification (wall-thickening) of hyphal or conidial cells. Induction of chlamydospore formation in *F. oxysporum* is related to stress factors such as absence of the host (nutrient depletion) and unfavorable environmental conditions (Smith, 2007). As would be expected chlamydospores germinate when favorable conditions return including the presence of host root exudates (nutrient abundance) (Kommedahl, 1966). Chlamydospores of *F. oxysporum* f. sp. *niveum* were more heat-resistant and survived longer in the soil than conidia (Freeman and Katan, 1988). De Cal et al. (1997) reported that inoculation with chlamydospores of *Fol* caused more severe symptoms in tomato than with microconidia. A higher disease-producing potential of microchlamydospores compared to microconidia of *F. oxysporum* f. sp. *lini* was also observed in flax (Couteaudier and Alabouvette, 1990).

2.1.2. Host range

Fol and *Forl* can exist as necrotrophs by killing and consuming the nutrients contained in cells of their primary host, tomato, and, in some cases, as biotrophs in association with the root systems of unrelated plants. In addition to *S. lycopersicum*, Fol can infect and cause symptoms in *S. melogena*, *S. pimpinellifolium* and other *Solanum* spp. (Katan, 1971; Subramanian, 1970). *Amaranthus, Chenopodium, Digitaria, Malva* and *Oryzopsis* spp. were found to be symptomless carriers of *Fol* (Katan, 1971; Fassihiani, 2000).

The host range of *Forl* is considerably larger and includes both symptomatic and symptomless hosts in the Anacardiaceae (*Schinus terebinthifolius*); Cruciferae [*Brassica juncea* L., *B. oleracea* L. (five vars.), *Capsella bursa-pastoris* (L.) Medik.]; Cucurbitaeae [*Citrullus lanatus* var. *lanatus* (Thunb.) Matsum. & Nakai]; Leguminosae (*Arachis hypogaea* L., *Glycine max* L., *Melilotus alba* Medik., *Phaseolus vulgaris* L., *Pisum sativum* L., Trifolium *pretense* L., *Trifolium repens* L., *Vicia faba* L.); Molluginaceae (*Mullugo verticillata* L.); Plantaginaceae (*Plantago lanceolata* L., *Scoparia* sp.); Solanaceae (*Capsicum frutescens* L., *S. lycopersicum* L., *S. melongena* L.); and Umbelliferae [*Apium graveolens* L. var. *dulce* (Mill.) pers., *Daucus carota* L.,] (Jones et al., 1991; McGovern and Datnoff, 1992; Menzies et al., 1990; Rowe, 1980). According to these references certain leguminous plants are very susceptible to *Forl*, and the host range and virulence of the fungus varied by isolate.

2.1.3. Tomato seeds and transplants

Contamination/infection of tomato seeds by *Fol* has been documented (Elliott and Crawford, 1922; Elwakil et al., 1998). Contaminated seed was a suspected source of the movement of *Fol* race 3 in Brazil (Reis and Boiteux, 2007). Al-Askar et al. (2014) recovered isolates of *F. oxysporum* from tomato seeds at a high frequency that caused seed and root rot. Contamination of tomato seeds by *Forl* was detected at a low incidence (0.1–0.01 %) in fruit on stem-infected plants, and also occurred through transmission by the *Forl*-infested hands of workers (Menzies and Jarvis, 1994). Tomato transplants infected by *Forl* have been implicated in the long distance spread of the fungus (Hartman and Fletcher, 1991; McGovern and Datnoff, 1992). McGovern et al. (1993) determined that outbreaks of FCRR were linked to the infection of tomato transplants grown in reused Styrofoam and plastic transplant trays contaminated by *Forl*.

2.1.4. Soil and other media

Estimations of the survival of *Fol* in field soil range from more than 10 years (Katan, 1971) to indefinitely (Agrios, 2005). Presumably the survivability of *Forl* in the field is very similar; in addition, this fungus possesses the added ability of surviving in association with many unrelated alternate hosts. Although FCRR outbreaks have occurred in rock wool-based hydroponic systems, because extensive plant to plant spread was not observed, it was concluded that the primary factor in such outbreaks was the use of infected transplants (Mihuta-Grimm et al., 1990). Hartman and Fletcher (1991) also observed only limited spread of the pathogen in rock wool-grown tomato. Once contaminated, a growing medium can also be a source of pathogen inoculum and dissemination via wind, water, shoes, tools and equipment.

2.1.5. Irrigation water

Although there have been a number of reports of dissemination of other *formae speciales* of *F. oxysporum* in either surface water or closed hydroponic systems (Anderson and Nehl, 2006; Davis, 1980; Jenkins and Averre, 1983), there have been few reported cases of the spread of either *Fol* or *Forl* in this manner (Rattink, 1992; Xu et al., 2006). Increasing use of recycled irrigation in plant production, mandated by water conservation and reduction of environmental impacts from agriculture, would seem to make the movement of these and other plant pathogens in irrigation water more likely.

2.1.6. Structures/supports

Both *Fol* and *Forl* can infest and survive on and inside of wooden stakes used to support field-grown tomato (Jones and Woltz, 1968; McGovern and Datnoff, 1992). *Forl* could be recovered from stakes for at least 5 years (McGovern, unpublished data). In addition, *Forl* isolated from plastic stakes used to secure drip tubes in rock wool cubes was implicated in greenhouse outbreaks of FCRR (Toro et al., 2012). Shlevin et al. (2003) indicated that contaminated greenhouse structures (walls, poles) were a likely source of *Forl* inoculum.

2.1.7. Insects

Transmission of both *Fol* and *Forl* to tomato by shore flies (*Scatella stagnalis* Fall. Diptera) has been reported (Corbaz and Fischer, 1994; Matsuda et al., 2009). In addition, transmission of *Forl* by fungus gnats (*Bradysia* spp. Diptera) from diseased plants to healthy tomato transplants has been demonstrated (Gillespie and Menzies, 1993). These vectors may be controlled through cultural, chemical and biological tactics (Jandricic et al., 2006; Price et al., 1991; Van Eppenhuijsen et al., 2001).

2.1.8. Root-knot nematodes

Although plant-parasitic nematodes including *Meloidogyne* spp. have been reported to predispose plants to a number of soilborne pathogens (Powell, 1979), there have been contrasting opinions on the ability of root-knot nematodes to cause loss of resistance to *Fol*. *Meloidogyne incognita* was reported to cause loss of resistance in tomato to race 1 of *Fol* (Jenkins and Courson, 1957; Sidhu and Webster, 1977). However, other researchers found that simultaneous, or prior infection of tomato plants by *M. incognita* did not affect resistance to either race 1 or 2 of the pathogen (Jones et al., 1976). Abawi and Barker (1984) also found that resistance to *Fol* race 1 was unaffected by root-knot nematode populations, but observed additive damage by the two pathogens in non-*Fol*-resistant cvs. Prior infestation of greenhouse soil with *M. incognita* did not appear to predispose tomato plants to *Forl* infection (Jarvis et al., 1977). Despite uncertainty as to the resistance-breaking ability of root-knot nematodes, the importance of their control in their own right is certain.