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Fusarium wilt of cotton: Management strategies

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ABSTRACT

Cotton is a globally important crop used for both its natural fiber and seed. Fusarium wilt, caused by the fungus *Fusarium oxysporum* f. sp. *vasinfectum*, is a major disease of cotton capable of causing significant economic loss. The fungus persists in soil as chlamydospores and in association with the roots of susceptible, resistant and non-cotton hosts as well as in seed. Management of Fusarium wilt is difficult and most successfully achieved through the use of resistant cultivars and pathogen-free cotton seed. Once inoculum has been introduced into the field, strategies such as soil solarization and fumigation are applied to manage inoculum levels. While experimentally successful, these techniques are of limited use in a commercial setting. Management of Fusarium wilt will continue to rely upon the development of new resistant cultivars. Additionally, understanding the mechanism of seed colonization and developing high throughput seed treatment and testing protocols will allow the industry to effectively avoid the introduction of inoculum.

1. Introduction

Cotton is a globally important crop used for both its natural fiber and seed. It is one of more than 4000 species in the family Malvaceae, which also includes okra and cacao. Global production of cotton is approximately 26.9 million metric tons per year. China and India produce nearly half the world's cotton followed by the United States with an annual production of approximately 3.7 million metric tons (Anonymous, USDA-FAS). The majority of cotton lint production is through the cultivation of two cotton species: *Gossypium hirsutum* (Upland) and *Gossypium barbadense* (Pima). These two species were independently domesticated in the new world approximately 4000 to 6000 years ago in the Yucatan peninsula and Peruvian Andes, respectively, and are the subjects of both public and private breeding programs. Of lesser commercial importance are *Gossypium herbaceum* and *Gossypium arboretum*, species native to India and Eastern Asia. Each of these four species is affected by Fusarium wilt.

2. Fusarium wilt

Several fungal diseases are problematic for cotton growers, including Verticillium wilt (*Verticillium dahliae*), damping-off (*Rhizoctonia* spp. and *Pythium* spp.), Ascochyta blight (*Ascochyta gossypii*), black root rot (*Thielaviopsis basicola*) and various leaf spots. Fusarium wilt is of particular concern to growers as the pathogen frequently causes severe economic losses and persists in fields for many years.

2.1. The causal organism

Fusarium oxysporum f. sp. *vasinfectum* W.C. Synder & H.N. Hans (FOV), first identified in 1892 in cotton fields in Alabama (Atkinson, 1892), produces asexual macro- and micro-conidia as well as chlamydospores, stable overwintering structures. The fungus also survives in association with non-hosts, a particularly challenging trait since the ability to colonize roots of weeds and other crops allows FOV to persist in soil for years or decades (Smith and Snyder, 1974). FOV also persists in seed, which can occur by contamination of cotton bolls in the field (Bennett et al., 2008; Elliot, 1923).

2.2. Races and geographical distribution

Worldwide, there are six recognized races of FOV, numbered in order of their discovery. The term “race” used to distinguish these genotypes does not imply a correlation in host gene resistance in

Abbreviations: FOV, *Fusarium oxysporum* f. sp. *vasinfectum*.

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the classical use of the term, but the designation of certain genotypes by race has been maintained by the cotton industry. Although eight races were initially identified, molecular classification has eliminated redundancy between races 4 and 7 and races 3 and 5 (Chen et al., 1985; Ibrahim, 1966; Nirenberg et al., 1994).

Fusarium wilt of cotton was first described in 1892 in the southern United States (Atkinson, 1892). The earliest described genotype, race 1, was characterized in the United States in 1958 (Armstrong and Armstrong, 1958) and has worldwide distribution. Race 1 is distinguished from race 2 by DNA sequence analysis (O'Donnell et al., 2009) as well as by pathogenicity on plants other than cotton, including okra (Armstrong and Armstrong, 1958; Elliot, 1923). These two genotypes are particularly devastating on cotton when the root knot nematode, *Meloidogyne incognita*, is also present (Garber et al., 1979). In the absence of nematodes, these races cause mild disease (Jorgenson et al., 1978). Genotypes in the race 3 lineage were initially reported on Pima cultivars in Egypt (Fahmy, 1927). Current distribution of these genotypes includes Egypt, Sudan, Israel, Uzbekistan, China and the United States (A. Cianchetta, unpublished data; Dishon and Nevo, 1970; Fahmy, 1927; Hillocks, 1992; Kim et al., 2005). Race 4, which is particularly virulent on certain cotton cultivars, was first reported in India in 1960 (Armstrong and Armstrong, 1960), and was later detected in California in 2001 (Kim et al., 2005). Unlike other races of FOV, this genotype causes economic losses independent of nematode pressure on both Pima and Upland cultivars (Ulloa et al., 2006). Race 5 was described in Sudan in 1966 (Ibrahim, 1966) but was later found to be the same as the previously described race 3 (Nirenberg et al., 1994). Race 6, which was first reported in Brazil in 1978, is genetically similar to races 1 and 2 but is distinct in its pathogenicity on certain cotton cultivars as well as okra (Armstrong and Armstrong, 1978). Isolates of this genotype were also reported in Paraguay in 1980 (Armstrong and Armstrong, 1980). Races 7 and 8 were initially described in China in 1985 (Chen et al., 1985) but race 7 was later found to be the same as race 4. The appearance of Fusarium wilt of cotton in Australia in 1993 (Kochman, 1995) led to the identification of several genotypes distinct from other known genotypes of FOV (Davis et al., 1996; Kim et al., 2005; Wang et al., 2010). Several isolates recovered from symptomatic cotton plants in Darling Downs of Queensland were similar to FOV race 6 and pathogenic on both Pima and Upland cultivars. However, esculin hydrolysis tests did not match these isolates with any known race. A wider survey of 856 isolates of FOV indicated distinct morphological and genetic differences from FOV races 1, 2, 3, 4, 6 and 8 (Wang et al., 2010). Further analysis indicated two closely related vegetative compatibility groups, suggesting a local origin of these isolates in response to wide-scale planting of highly susceptible cotton cultivars (Davis et al., 1996).

In addition to these six nominal races of FOV and the Australian isolates, four genotypes from the southeastern United States are highly virulent on some commercial cotton cultivars (Holmes et al., 2009). Designated LA108, LA110, LA112 and LA127/140, these genotypes have unique partial translation elongation factor (EF-1α) sequences (Holmes et al., 2009). LA108 and LA110 are particularly aggressive on Upland varieties and the occurrence of these genotypes is not rare (Holmes et al., 2009). In general, the nominal races and genotypes of FOV can be distinguished from each other by sequence analysis, restriction fragment length polymorphisms of the intergenic spacer region, mating type, vegetative compatibility (Abo et al., 2005; Skovgaard et al., 2001) and pathogenicity tests on cotton.

2.3. Disease symptoms

FOV colonizes the vascular system of susceptible cotton hosts, causing vascular discoloration, wilting and sometimes death of the plant. Discrete symptoms vary with inoculum density, susceptibility of the cotton cultivar and plant age (Hao et al., 2009). In greenhouse pathogenicity tests, diagnostic symptoms of the disease were not induced at inoculum levels below 10^3 conidia/gram of soil (Hao et al., 2009). At lower inoculum densities, the fungus did not compromise plant health and could not be recovered from stem tissue. Although FOV colonizes the vasculature of resistant cultivars, the infection is limited and may or may not result in obvious vascular discoloration (Hao et al., 2009).

The infection of young seedlings may result in the wilting and necrosis of cotyledons and occasionally, plant death. This creates an uneven stand in the field, a pattern that may be confused with seedling diseases such as damping-off caused by *Pythium* spp. and *Rhizoctonia* spp. Internal symptoms of FOV infection include brown discoloration in the vasculature. Plants infected at a later stage of growth may be wilted and stunted, and leaves may display chlorosis and necrosis. The characteristic brown vascular discoloration is most evident in the roots and lower stem, but may be apparent in upper branches as well.

Disease foci increase in size through the redistribution of infested soil and plant material by field equipment and water. The rate at which these foci expand and coalesce is dependent upon a combination of environmental factors such as the susceptibility of the cultivar, inoculum density, soil texture and pH (Hao et al., 2009; Mundkur, 1936; Wang et al., 1999b). Estimated average crop losses due to seedling diseases including Fusarium wilt in the United States are 2.85% per year; losses due to *Fusarium* spp. specifically are about 0.35% (National Cotton Council of America).

3. Disease management

Strategies for the management of Fusarium wilt include clean cotton seed, resistant cultivars, sanitation and the reduction of inoculum density. In some cases race-specific strategies are effective. Fusarium wilt caused by race 1 and race 2, for example, can be managed by the reduction of nematode populations and the use of nematode resistant cultivars.

3.1. General management strategies

Cotton seed treatments can decrease and eliminate seedborne inoculum, thereby limiting the risk of introducing FOV into a field. The seed transmissibility of FOV was established by Elliot (1923), who demonstrated transmission of FOV in surface sterilized seed, prompting the recommendation that infested fields be avoided for seed production. The identification of FOV race 4 in California spurred new research on seed transmission and protocols for eliminating seedborne inoculum while preserving germination and vigor. In a survey of seed collected from four Pima cultivars (Phytogen 800, DeltaPine 340, DeltaPine 744 and Bayer 4126) and one Upland cultivar (Phytogen 72) grown in California fields naturally infested with FOV race 4, Bennett et al. (2008) detected FOV in one of 25,742 seeds and in 16 of 26 bulk samples of 50 seeds each. The variability in the rate of transmissibility is similar to reports of seed infested with other races of FOV, which ranged from zero to 47% (Hillocks, 1992; Perry, 1962; Smith et al., 1981).

The efficacy of thermotherapy to eliminate FOV from seed was evaluated due to the success of the technique in eliminating *Glomerella gossypii*, the cause of cotton anthracnose, from seed without compromising seed germination (Barre and Aull, 1914). In a 2010 study, the effect of dry heat treatments of 60, 70 and 80 °C at two day intervals for 14 days on disinfesting seed infested with FOV race 4 was assessed (Bennett and Colyer, 2010). Although none of the dry heat treatments eliminated FOV from seed, a significant