ABSTRACT

Fusarium oxysporum f. sp. *vasinfectum* (Fov) was first identified in Australia in 1993, and has since become one of the most significant threats to the country's thriving cotton industry. The interaction between a unique Australian biotype of Fov and cotton hosts with varying susceptibilities to Fusarium wilt was studied. This research described the infection process and associated host defence mechanisms of two commercial cotton varieties after inoculation with Fov, and quantified their subsequent accumulation of antimicrobial terpenoids.

A rapid, reliable glasshouse bioassay that correlated with field resistance was developed for the study of Fusarium wilt of cotton. Detailed observations of the infection process obtained through light microscopy were used to formulate the disease cycle of Australian Fusarium wilt of cotton. Using pathogen growth assays, varietal differences in root exudates and vascular tissues in the cotton hosts were documented. Root diffusate from the most susceptible cotton variety to Fusarium wilt, Siokra 1-4, contained a lipophilic compound that promoted the germination of Fov microconidia. On the other hand, a lipophilic compound present in diffusate from the least susceptible variety, Sicot 189, inhibited the growth of Fov germ tubes.

A bioassay using inoculated whole plants showed that Fov colonisation of the vascular tissues of Sicot 189 was restricted after 3 days. The basis for this inhibition was investigated further using light and transmission electron microscopy. Infection induced the reorganisation of contact cells in host vascular tissue, including an increase in cytoplasmic content and the partitioning of vacuoles, which was concurrent with the accumulation of materials in adjacent vessel lumens, via pits. Histochemical analysis indicated these globular materials secreted into the vessels were terpenoids. These structural and terpenoid responses in Siokra 1-4 and Sicot 189 were similar, however, they were more intense and rapid in the latter, less susceptible variety. The responses in Sicot 189 also corresponded to the time period that pathogen inhibition was observed. Thus, a correlation was demonstrated between the rapid and intense induction of both structural and biochemical responses with decreased susceptibility to Fusarium wilt. Detailed HPLC analysis of vascular tissues confirmed that terpenoids accumulated more rapidly and at higher concentrations in the less susceptible cotton variety. These findings provided strong evidence for the involvement of antimicrobial terpenoids in the determination of Fusarium wilt susceptibility of Australian cotton varieties.

This work represents the most complete survey to date of the interaction of Australian biotypes of Fov with cotton. These insights can contribute to future cotton breeding efforts and cultural management of Fusarium wilt in the field. Thus, each part of this study has advanced complementary facets of our understanding of Fov, and has provided a framework from which future studies on phytoalexins and other putative cotton defences can be studied.