REDUCED EXPRESSION OF AN EARLY ENZYME IN THE GOSSYPOL PATHWAY REVEALS A LINK BETWEEN THE GOSSYPOL AND LACINILENE PATHWAYS DURING FUNGAL INFECTION
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Abstract
We have generated plants that contain an RNAi construct that severely knocks-down expression of a p450 gene involved in an early step in the production of gossypol and heliocides. The leaves of these plants have less than 10% of the normal level of these compounds. Since these secondary compounds protect the cotton plant from pathogens, we predicted that these plants would be more susceptible to *Fusarium* infection. To test this hypothesis, we grew WT and RNAi plants in growth chambers and treated the plants with either FOV11 or water via a root-dip assay. Three weeks after inoculation, we measured several disease indicators including: leaf symptoms, root and shoot weight, plant height, stem recovery of FOV11, and terpenoid aldehyde composition of leaves and roots. Surprisingly, we did not see any strong difference in disease severity between WT and RNAi plants. Non-treated WT plants produced, probably either as preemptive strike or induced by soil microbial contaminants, high amounts of gossypol pathway metabolites, which are toxic to soil microbes. Fungal infection did not induce more of these metabolites. The lacinilene pathway metabolites were produced in negligible amounts in both non-treated and treated WT plants. In RNAi plants, the gossypol pathway was knocked down and negligible amounts were present in both non-treated and treated. However, lacinilene pathway metabolites were induced more than 22 fold when attacked by fungus. Blocking the gossypol pathway resulted in diverting the shared intermediate into the lacinilene pathway during fungal infection. This establishes that the gossypol and lacinilene pathway are linked in the RNAi plants, and that the lacinilene metabolites may contribute to the seedling resistance toward Fov.