TRANSCRIPTOME ANALYSIS OF GOSSYPIUM HIRSUTUM RESPONSES TO MELOIDOGYNE INCOGNITA DURING COMPATIBLE AND INCOMPATIBLE INTERACTIONS

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Abstract

Root-knot nematodes (RKN; *M. incognita*) are one of the major limiting factors in crop production around the world. Case in point, US cotton yield losses associated with RKN increased from 1.0% in 1987 to 3.02% in 2017, resulting in losses of more than 136.8 million kilograms of cotton valued over $228.4 million (Cotton Disease Loss Estimate Committee Report 2017; Cotton Price Statistics Annual Report 2019). To address the issue, host resistance has gained traction as the most effective approach, wherein marker-assisted selection (MAS) is deployed to incorporate quantitative trait loci (QTLs) conditioning resistance in the breeding pipeline for developing commercial cultivars.

Building on our ongoing efforts towards identifying candidate genes conditioning Southern root-knot nematode (RKN) resistance in upland cotton, we are working to characterize the interactions between *G. hirsutum* and *M. incognita* using comparative transcriptomics and differential gene expression of a nematode resistant (incompatible interactions) and susceptible (compatible interactions) lines at five different stages of nematode development inside RKN infested roots. Our results show that basal defense responses are activated both during compatible and incompatible interactions; albeit the responses are more pronounced in RKN resistant genotype compared to the susceptible line. In summary, nematode responsive genes related to defense pathways are often repressed during compatible interactions, while earlier induction, greater diversity, and higher degree of up-regulation of those genes are archetypal of incompatible interactions. A wide spectrum of disease resistance and putative resistance genes, pathogenesis-related genes, and genes corresponding to ligands and receptors are differentially expressed in response to nematode parasitism in G. hirsutum. These genes are mapped across the cotton genome and include candidate genes Gh_A11G3090 (*PUB21*) and Gh_A11G2836 (*RPPL1*), and Gh_D02G0259 (*RLP12*) in the QTL regions of chromosomes 11 and 14, respectively.