**Metabolite profiling of chickpea (*Cicer arietinum*) in response to necrotrophic fungus *Ascochyta rabiei***

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Ascochyta blight (AB) caused by a necrotrophic fungus, *A. rabiei* is one of the most damaging diseases of chickpeas worldwide. Resistance to AB is a highly complex and quantitative trait. The limited number of resistance sources and the erosion of resistance in commercial cultivars have posed a challenge for breeders to develop cultivars with durable resistance to aggressive pathogen populations. Despite this little or no research has been directed toward physiological and biochemical mechanisms to develop complementary crop protection strategies for the sustainable and profitable chickpea industry. Our study aims to identify the metabolites associated with resistance/susceptibility in chickpea in response to *A. rabiei* infection. Here, we present metabolite profiling of two chickpea accessions comprising a moderately resistant genotype (CICA1841) and a highly susceptible cultivar (Kyabra) in response to one of the most aggressive Australian *A. rabiei* isolates TR9571. Non-targeted metabolomics analysis using liquid chromatography-mass spectrometry (LC-MS) revealed constitutive or differentially altered metabolites in aerial tissue (leaf and stem) of CICA1841 and Kyabra. The host-pathogen interaction resulted in the accumulation and suppression of various metabolites, revealing a possible reason for susceptibility against *A. rabiei* in the highly susceptible chickpea cultivar. Several differential metabolites are the precursors for secondary metabolic pathways, including flavonoid biosynthesis, phenylalanine pathway, Aminoacyl-tRNA biosynthesis, pentose and glucuronate interconversions, arginine biosynthesis, valine, leucine, and isoleucine biosynthesis, and alanine, aspartate, and glutamate metabolism. This study has provided insight into how a necrotrophic fungus manipulates the host during infection to cause disease.