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New insights into molecular basis of red rot resistance in sugarcane

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Red rot of sugarcane caused by Colletotrichum falcatum Went is one of the devastating diseases of sugarcane causing significant loss to sugarcane production in the country. Complex polyploidy and lack of information on inheritance to red rot in sugarcane make breeding for red rot resistance more difficult. Hence researchers studied the mechanism of red rot resistance in sugarcane in detail. Initial studies based on biochemical tools identified oxidative enzymes and red rot pigments in disease resistance. Further studies revealed the role of pathogenesis-related (PR) proteins and 3-deoxyanthocyanidin phytoalexins especially luteolinidin and apigeninidin in red rot resistance. Recent studies using semiquantitative RT-PCR after pathogen inoculation from sugarcane cultivars varying in red rot resistance, revealed differential accumulation of transcripts of the flavanoid biosynthetic pathway like coumarate-4-hydroxylase, chalcone synthase, chalcone reductase, flavanoid 3'-5' hydroxylase and flavanoid glycosyl transferase and this transcript analysis, further confirmed the role of sugarcane phytoalexins in red rot resistance. Similarly the role of PR- proteins like chitinase and β -1,3-glucanase was established at the transcript level. Detailed molecular studies using differential display (DD)-RT-PCR identified expression of more number of differentially expressed transcripts during the host-pathogen interaction. Full length sequences of many potential transcripts were identified and are being characterized. Also to identify specific proteins involved in host resistance, proteomic approach has been attempted by optimizing sample preparation from stalk tissues, 2-D electrophoresis (2-DE), downstream processing of identified spots and bioinformatics. Several resistance associated proteins spots were identified and they are being analyzed critically.

O1 Identification of differentially expressed proteins in chickpea upon Fusarium oxysporium infection

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One of the most important diseases affecting chickpea is Fusarium wilt, caused by the soil borne fungus Fusarium oxysporum f sp ciceri (FOC), a root pathogen, which colonizes the xylem vessels and blocks them completely leading to wilt. Several studies have demonstrated that infection with F. oxysporum preceeds various chemical and biochemical changes in chickpea. Active plant defense against invading pathogens is achieved by recognition of pathogen followed by changes in structural and biochemical components that are differentially regulated depending on the incoming stress. Perception of both general and specific pathogen-associated molecules triggers defense responses via signal transduction cascades and transcriptional activation of numerous genes. However, information about differentially expressed protein that determines the outcome of interactions between F. oxysporum and chickpea roots needs a