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ABSTRACTS

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level upon MYMIV infection suggests involvement of the *CYR1* gene in conferring resistance against the virus. *In silico* constructed 3D models of LRR region of the host protein and MYMIV-coat protein/PAMP revealed that the unique CYR1-LRR forms an active pocket and successively interacts with PAMP during docking. This phenomenon of host pathogen interaction is like that of receptor and ligand that has been elucidated by *in silico* studies. Such recognition induces signaling pathways that ultimately results in changes of the host gene expression pattern and synthesis of pathogenesis related proteins. To understand the incompatible interaction of *V. mungo* during MYMIV infection at the molecular level comprehensively, genomic and proteomic approaches have been undertaken in our laboratory. About 200 differentially expressed ESTs and 66 differential proteins present in high abundance have been identified during incompatible interactions. Seven functional categories of differential genes/proteins have been identified, which are energy/metabolism, photosynthesis, stress/defense, signal transduction, transcription, transport and secondary metabolism related. The involvement of these proteins in different metabolic pathways in an orchestrated manner will briefly be discussed.

Molecular Profiling 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 pattern of red rot resistance in sugarcane make breeding for the disease resistance more difficult. Hence, we have undertaken detailed studies on the mechanism of red rot resistance in sugarcane. Our initial studies based on biochemical tools, identified the possible involvement of 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. A set of red rot resistant and susceptible varieties were inoculated with *C. falcatum*, tissues were harvested after different intervals and RNA/protein were isolated for genomic/proteomic studies. Our recent genomic studies involving semiquantitative RT-PCR, differential display (DD)-RT-PCR and subtractive libraries identified many transcripts involved in red rot resistance. Differential accumulation of transcripts potentially involved in the flavanoid biosynthetic pathway like coumarate-4-hydroxylase, chalcone synthase, chalcone reductase, flavanoid 3`-5` hydroxylase and flavanoid glycosyl transferase was established and further confirmed the role of sugarcane phytoalexins in red rot resistance. Similarly the role of PR- proteins like chitinase, β-1,3-glucanase and TLPs in red rot resistance was established at the transcript level. Differential accumulation of chitinase, β-1,3-glucanase was further validated by qPCR and northern blot analysis. In subtractive libraries, ~400 transcripts associated with signal transduction, defense, transcription factors, energy metabolism, carbohydrate metabolism and cell structure maintenance were obtained. Our molecular studies using DD-RT PCR identified expression of more number of differentially expressed transcripts during the host-pathogen interaction. Using RACE technique, we have isolated full length sequences of the following four genes putatively involved in red rot resistance *viz.* 14-3-3 like protein, chitinase, xylanase inhibitor and basal antifungal peptide. Studies on regulation of five major families/classes of defense related transcription factors (TFs) revealed earlier induction of 14 red rot pathogen responsive TFs *viz.* TLP B, K, N, L, MYB 78, WRKY 40, NAC L, O, J, D, BZIP 18, 27, 23, and 11 in resistant variety. 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), down stream processing of identified spots by MS analysis and bioinformatics. About 125 up/down regulated proteins were characterized by peptide mass finger printing, some of the identified important proteins were putative callose synthase, R2R3-MYB transcription factor MYB6, p-coumarate 3-hydroxylase, PrLTP1 and PISTILLATA-like protein. Further validation of the differential expression of identified proteins/transcripts by qPCR and RNA blots are in progress. Further work is under progress on characterization of the cDNA encoding the elicitor and identification of the cognate membrane binding pathogen defense signalling in sugarcane.

Interactions between Fungi and Bacteria to Fulfil the Oxalate Carbonate Pathway, A Potential Sink for Atmospheric CO_2 in Tropical Soils under Oxalogenic Trees

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The oxalate-carbonate pathway combines oxalate biosynthesisby plants from atmospheric CO_2 through photosynthesis, calcium oxalate precipitation and accumulation in specialized cells (idioblasts), oxalate oxidation to CO_2 and OH^- by oxalotrophic bacteria, and precipitation of $CaCO_3$ (calcite) through the resulting alkalinisation. As calcite normally lasts for very long periods once accumulated in soils (1000 to 1'000'000 years), this phenomenon represents a long-term sink for atmospheric CO_2.Such accumulations of $CaCO_3$ were observed in acidic soils devoid of carbonates in the intertropical belt, in Africa, Asia and Latin America, under a number of oxalogenic trees without systematic relationships. However, in microcosms under laboratory conditions, a distinct alkalinisation was only observed in the simultaneous presence of oxalotrophic bacteria and filamentous fungi. Fungi may act at different levels in this cycle. First, many of them, particularly the wood decaying ones, produce calcium oxalate in their medium or on their hyphae. Second, they participate to the decay of plant litter, freeing plant Ca-oxalatecrystals and exposing them in contact with the oxalotrophic bacteria. Third: fungi may intervene in calcium leaching and translocation from the Ca-containing, non carbonate minerals. Finally, ligninolytic fungi may oxidize oxalate extracellularly, through the secretion of oxalate-oxidase, ahydrogen peroxide-producing enzyme. Hydrogen peroxide is a substrate required for fungal lignin oxidative degradation (e.g. in white rot), directly through ligninases (lignin peroxidases), or indirectly by manganese peroxidases producing Mn^{+3} "shuttles" which, in turn, penetrate the

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