

Pearl millet is a staple food for more than 90 million farmers in arid and semi-arid regions of sub-Saharan Africa, India and South Asia. Downy mildew of pearl millet caused by the biotrophic oomycete *Sclerospora graminicola* is the most devastating disease which impairs pearl millet production causing huge yield and monetary losses. Chitosan nanoparticles (CNP) were synthesized from low molecular weight chitosan having higher degree of acetylation was evaluated for their efficacy against downy mildew disease of pearl millet caused by *Sclerospora graminicola*. Seed treatment with CNP induced systemic and durable resistance and showed significant downy mildew protection under greenhouse conditions in comparison to the untreated control. Seed treatment with CNP showed changes in gene expression profiles wherein expression of genes of phenylalanine ammonia lyase, peroxidase, polyphenoloxidase, catalase and superoxide dismutase were highly upregulated. Downy mildew protective effect offered by CNP was found to be modulated by nitric oxide and treatment with CNP along with NO inhibitors cPTIO completely abolished the gene expression of defense enzymes and PR proteins.

Transcriptome and GWAS-based approaches to understand the mechanisms of *Fusarium fujikuroi* resistance in rice

D. SPADARO (1), S. Matic (2), I. Siciliano (2), P. Bagnaresi (3), A. Volante (4), M. Aragona (5), A. Infantino (6), M. L. Gullino (7), G. Valè (4), (1) DISAFA and AGROINNOVA, University of Torino, Torino, ITALY; (2) Agroinnova - University of Torino, Grugliasco, ITALY; (3) CREA - Genomic Research Centre, Fiorenzuola (PC), ITALY; (4) CREA - Rice Research Unit, Vercelli, ITALY; (5) CREA - Plant Pathology Research Centre, Roma, ITALY; (6) CREA - Plant Pathology Research Centre, Rome, ITALY; (7) Agroinnova - University of Torino, Grugliasco, Torino, ITALY

Fusarium fujikuroi, causal agent of Bakanae disease, is the main seedborne pathogen on rice. Profiles of defense-related phytohormones and phytoalexins were investigated. In the resistant genotype Selenio, the pathogen induced high levels of sakuranetin and other phytoalexins. In the susceptible genotype Dorella, the pathogen induced gibberellins and abscisic acid, inhibited jasmonic acid, and Bakanae symptoms were observed. A RNA-seq transcriptome study was performed. The basic rice resistance machinery against *F. fujikuroi* involved PR genes, glucanases and peroxidases. The resistance mechanisms activated in the resistant cultivar included WRKY transcriptional factors, MAPK cascades, and cytochrome P450 genes. When the gibberellin production was controlled, Selenio plants activated the jasmonic acid metabolic pathway. A germplasm collection of japonica rice was screened for *F. fujikuroi* resistance, allowing the identification of accessions with high-to-moderate levels of resistance to bakanae. A genome-wide association study (GWAS) uncovered two genomic regions highly associated with the observed phenotypic variation for response to bakanae infection. A search for candidate genes with a putative role in bakanae resistance was conducted considering all the annotated genes and *F. fujikuroi*-related DEGs included in the two genomic regions highlighting several gene functions that could be involved in resistance, thus paving the way to functional characterization of the resistance loci.

Identifying susceptibility genes for citrus Huanglongbing in sweet orange

F. NOGALES C. VASCONCELOS, Z. Pang, N. Wang, University of Florida, Lake Alfred, FL, USA

The phloem limited bacteria, *Candidatus Liberibacter asiaticus*, *Ca. L. americanus* and *Ca. L. africanus*, are the putative causal agents of citrus Huanglongbing (HLB), which is devastating citrus industry worldwide. Identifying susceptibility genes is crucial to understand its interaction with citrus host and therefore engineer tolerant and resistant plants. In this study, we will present our current progress in identification of putative susceptibility genes against HLB. To identify putative susceptibility genes, we used yeast-two-hybrid system to screen for potential host target proteins of putative virulence factors of *Ca. L. asiaticus*. We also employed gene expression essays in order to identify differentially expressed genes in HLB diseased trees, giving us insights of microbe-host interactions at molecular level. The CRISPR technology is being used to mutate putative HLB susceptibility genes to test whether they affect citrus resistance or tolerance to HLB.

Intensification on PAMP-triggered immunity confers disease resistance against bacterial soft rot

Z. JING-LIN (1), Y. H. Lin (2), (1) National Pingtung University of Science and Technology, Pingtung, TAIWAN; (2) National Pingtung Univ of Science & Tech, Pingtung, TAIWAN

Bacterial soft rot disease is a devastating disease affecting a variety of plants worldwide. Possible strategy for controlling this disease consists of introducing the expression of the plant ferredoxin-like protein (pflp) gene in plants and usage of beneficial microorganisms. The PFLP-mediated resistance is associated with the intensification of PAMP-triggered immunity (PTI), have been demonstrated. To gain further insights regarding the enhanced intracellular PTI signaling contributed by PFLP, *Arabidopsis* mutants in MAPK pathway were used to assay the responses triggered by flg22_{Pst}. Firstly, we confirmed that the rapid generation of H₂O₂, callose deposition, and hypersensitive response (HR) triggered by flg22_{Pst} was intensified by PFLP. Then, we demonstrated that the flg22_{Pst}-induced MAPK pathway was intensified by PFLP based on the expression of the *FRK1* gene mapk mutants. In addition to the rapid H₂O₂ generation, callose deposition and expression of *FRK1* were still intensified by *Bacillus amyloliquefaciens* PMB05 upon the treatment of flg22_{Pst}. Moreover, *B. amyloliquefaciens* PMB05 confers resistance against soft rot disease. These suggested that the disease resistance enhanced by *B. amyloliquefaciens* PMB05 was associated with the intensification on PTI through signaling in MAPK pathway. Taken together, we showed the intensification on PAMP-triggered immunity by PFLP or *B. amyloliquefaciens* strain PMB05 could be as a dominant factor to increase resistance against bacterial soft rot.

Intensification on PAMP triggered immunity by *Bacillus* strains to control bacterial wilt of tomato

T. H. HO (1), Y. H. Lin (2), (1) Department of Plant Medicine, National Pingtung University of Science and Technology, Pingtung, TAIWAN; (2) National Pingtung Univ of Science & Tech, Pingtung, TAIWAN

Tomato bacterial wilt caused by *Ralstonia solanacearum* is a devastating disease in tomato production. Currently, there is still no effective chemical to control this disease. To control this disease, reports showed transgenic plants express extracellular PFLP protein are resistant against bacterial wilt. The mechanism of this resistance is associated with the intensification of harpin-mediated HR. Therefore, we sought to screen bacterial strains of *Bacillus* spp. that can enhance harpin-mediated HR and further evaluate their efficacy on controlling bacterial wilt of tomato. Before screening, the harpin from *R. solanacearum*, PopW, was cloned and expressed in pET system. Results revealed that the HR on tobacco leaves could be induced by PopW. Among 9 rhizosphere *Bacillus* strains we assayed, *B. amyloliquefaciens* PMB05 was one strain which could intensify PopW-induced HR. Bacterial suspension of PMB05 applied in the rhizosphere of 2-week old tomato seedlings exhibited strong resistant against bacterial wilt. Finally, we demonstrated that the flg22_{RS}-induced ROS generation in tomato roots was intensified by *B. amyloliquefaciens* strain PMB05. Taken together, we concluded that the PAMP triggered immunity intensified by *B. amyloliquefaciens* strain PMB05 confers resistance of tomato plants against bacterial wilt disease.

Lignin reduction in alfalfa (*Medicago sativa*) does not affect foliar disease resistance

D. A. SAMAC (1), S. Ao (2), M. Dornbusch (1), A. Grev (2), S. Wells (2), K. Martinson (2), C. Sheaffer (2), (1) USDA-ARS, St Paul, MN, USA; (2) University of Minnesota, St. Paul, MN, USA