National Institute of Technology Rourkela

Defence Seminar

Seminar Title	: AtMTHFR2 regulated one carbon metabolism contributes towards broad-spectrum disease resistance in Arabidopsis
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Venue	: Online, LS Seminar hall
Date and Time	: 23 Jun 2025 (09:00 AM)
Abstract	: Rice is the staple crop consumed worldwide and has a major impact on global food security. The fungal disease counts 2/3rd of the total crop loss, Magnaporthe oryzae is the 3rd most devastating pathogen that causes rice blast disease, leading to 30% crop loss annually. The non-host mediated resistance development is one of the alternatives to curtail blast disease and can pave wider and better opportunities toward resistance variety development. In the present study, we characterized the involvement of AtMTHFR in plant immunity against rice blast disease. Attenuating mthfr2 in the pen2 background leads to hypersusceptibility against the pathogen in comparison to wild-type Col-0. The breach of immunity leads to hypersensitive responses, hyper ROS accumulation, target cell death, dysregulation of ROS scavenging enzyme, and morphological changes in cellular chloroplast arrangement in the mutant. Histochemical staining and qRT-PCR analysis of defense-responsive genes illustrate the involvement of both SA and JA/ET synergistic cross-talk in pen2 mthfr2 mutant during susceptibility. We also evaluated the independent role of mthfr2, irrespective of PEN2 mutation that contributes to NHR. Active metabolites in Col-0 leaf extract completely arrested M. oryzae conidial germination, while pen2 mthfr2 fresh leaf extract facilitated the conidial germination, indicating an altered metabolic signature due to MTHFR2 function disruption. Thus, we performed untargeted global metabolomic profiling between Col-0 and pen2 mthfr2 leaf sample and found enrichment in folate-dependent 1C metabolic pathway in pen2 mthfr2. The disease phenotyping in methionine metabolism-related key intermediate genes like ms2, sahh1, hmt3, and mthfr1 (the noncanonical isoform of mthfr2) mutants leads to compromised plant immunity, indicating a significant role of methionine to homocysteine homeostasis in plant immunity. Methionine levels stayed constant, suggesting compensatory regulation, but untargeted metabolomics showed a hazardous buildup of homocysteine thiolactone, which worsened oxidative stress in mthfr2. Fungal growth was inhibited by exogenous methionine administration, demonstrating its dual function in immunological signaling and direct protection. The synergistic effects of MTHFR2's genetic connections with nonhost resistance genes (HSP90, SOBIR1, and AtRBOHF) showed impacts on ROS signaling and defense gene expression, including PR1, PR5, PDF1.2, and WRKY53. These disturbances impacted salicylic acid and jasmonic acid-mediated pathways, wakening immune responses. The elevated susceptibility indicates an independent role of MTHFR in plant immune response. We further checked the involvement of MTHFR2 in broad-spectrum disease resistance against Rhizoctonia solani, and Pseudomonas syringae. We found that disruption in 1C metabolism leads to susceptibility to R. solani, while it contributes negatively to resistance against the bacterial pathogen. Our findings establish MTHFR2 as a pivotal regulator linking 1C metabolism to plant immunity. This study advances understanding of primary metabolism's role in defense and provides a foundation for engineering crops with enhanced resistance to multiple pathogens. Key words: Arabidopsis, Non-host resistance, One carbon metabolism, Broad-spectrum immunity, methylenetetrahydrofolate reductase.