RIBOFLAVIN-INDUCED RESISTANCE AGAINST SHEATH BLIGHT OF RICE FUNCTIONS THROUGH THE POTENTIATION OF LIGNIN FORMATION AND JASMONIC ACID SIGNALLING PATHWAY

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There are multiple defense strategies in plants to protect them against infection by pathogens. Formation of structural barriers including cell wall reinforcement and deposition of cell wall appositions by polymerization of lignin or suberin, and the dimerization of antimicrobial phenolic compounds can be activated by biologically or chemically induced resistance or after challenge inoculation of plants with various pathogens. Also, several defense signalling hormones such as salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) play important roles in activation of plant defense responses. In the present study, we investigated the mechanisms involved in activation of rice defense responses against the economically important sheath blight disease, caused by the necrotrophic fungus *Rhizoctonia solani*. Riboflavin-treatment of rice plants led to induction of resistance and suppression of disease progress. By investigating lignin formation using the phloroglucinol/HCL test and the thioglycolic acid assay it was shown that the observed resistance is linked with lignin formation at the infection sites. Expression of a cationic rice peroxidase, POC1, that is involved in lignin production, was examined using reverse transcription-polymerase chain reaction (RT-PCR). POC1 was upregulated at 18 h after inoculation in riboflavin treated rice plants. The expression in control plants was lower than that observed in riboflavin-treated plants. A correlation was found between induction of resistance by riboflavin and up-regulation of the POC1 gene. Also, marker genes of phenylpropanoid and octadecanoid pathways were upregulated in riboflavin-treated plants following infection. Application of the lipoxygenase (LOX) inhibitor 5, 8, 11, 14-eicosatetraynoic acid (ETYA) led to a strong reduction of riboflavin-induced resistance. In addition, riboflavin and JA protect rice plants against sheath blight pathogen in a similar way. The potentiated expression pattern of the LOX gene (which is a key marker of the JA pathway) by riboflavin, and the suppression of riboflavin-induced resistance by application of ETYA suggest an involvement of the JA signalling pathway as well as lignin deposition in riboflavin-induced resistance against *R. solani* in the monocot model plant rice.