学位論文要旨	
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題目	Metabolomic study on the resistance mechanism of rice sheath blight disease (メタボローム解析を用いたイネ紋枯病抵抗性メカニズムに関する研究)

Rice is staple food for most of the world's population. In rice cultivation, sheath blight disease caused by *Rhizoctonia solani* is economical important. Cultural control with resistant varieties is one of techniques to control the disease. However, the engineering on resistant varieties of rice have problem due to its resistance to R. solani is determined by many genes (polygene). In this regards, two rice lines have developed in our laboratory, 32R is resistant to R. solani and 29S is susceptible, respectively. The objective of this study is to observe the resistance mechanism as response of two rice lines due to R. solani infection by using metabolomic. This study is important to explain the rice lines response to R. solani, metabolites involved and metabolism regulation as resistance mechanism, as well as the role of metabolomic in resistance mechanism observation.

Study on plant resistance by metabolomic approach is based on the ion mode, positive and negative ion mode. Metabolomic study using positive ion mode has successfully revealed an increase of chlorogenic acid in 32R due to *R. solani* infection that leads to the formation of lignifications and secondary cell walls. While in the 29S, *R. solani* infection causes the accumulation of specific amino acids, such as GABA, glutamate, histidine, phenylalanine, serine, tryptophan, and tyrosine. It suggests the relationship between specific amino acid and susceptibility of rice plants to *R. solani*. In addition, the amino acid accumulation in 29S suggests providing a good condition for inner cell as increasing a nutrient source for fungal pathogen growth. The pipecolic acid showed higher in 29S than 32R due to *R. solani* infection. It may support the hypersensitive response (HR) which leads to the susceptibility in plant to the infection of necrotrophic fungal pathogen. The metabolomic study using positive ion mode also revealed the existence of canavanine as potential antimetabolite and antimicrobial in 32R due to *R. solani* infection.

In the metabolomics study using negative ion mode, the high of glyceric acid in 32R indicates that photorespiration occurs to maintain the ROS threshold. The *R. solani* infection to 32R increases the carbohydrate metabolites which alter the sugar accumulation. Additionally, the infection of *R. solani* in 32R increases the ADP that suggested the increment of respiration. In 32R infected by *R. solani*, the mucic acid increased that leads to the activation of pectin synthesis in primary cell wall formation. Jasmonic acid activation in 32R after infected by *R. solani* suggested to the plant signaling activation that leads to lignifications and secondary cell wall formation. In the 29S, the infection of *R. solani* causes to the increase of inosine monophosphate (IMP) relating to nitrogen assimilation and mobilization.

The study concludes that each rice line has different response as resistance mechanism against R. solani infection. The resistance mechanism can be derived originated from rice line or induced by R. solani infection. Metabolomic study is able to complements the previous studies on plant resistance to R. solani. Finally, metabolomic study can provide a knowledge and information in order to explain the mechanism of resistance in resistant and susceptible rice lines by using simple and fast methods. Furthermore, this information is expected to be used as a reference in the engineering of rice resistant variety to R. solani.