Brief Communication

Loss of OsHRC function confers blast resistance without yield penalty in rice

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Abstract

Loss of OsHRC function in rice increases resistance to blast disease caused by Magnaporthe oryzae without negatively impacting yield. OsHRC encodes a histidine-rich calcium-binding protein, which interacts with OsSPL13 to participate in the regulation of rice blast resistance. The role of OsHRC in rice blast disease resistance remains to be investigated. In the present work, we conducted CRISPR/Cas9-mediated gene editing to knock out OsHRC in rice. Using host resistance is the most effective method for blast control. However, most rice resistance genes are race-specific (Su et al., 2017). Discovering race-non-specific resistance genes will enhance blast resistance in rice cultivars.

Keywords: rice blast resistance, histidine-rich calcium-binding protein, loss-of-function mutation, susceptibility gene, wheat FHB.

Introduction

Rice blast, caused by Magnaporthe oryzae, is a serious disease in rice (Oryza sativa), worldwide. Because the pathogen mutates rapidly, rice blast has become one of the major threats for global rice production (Wang and Valent, 2017). Using host resistance is the most effective method for blast control. However, most rice resistance genes are race-specific (Su et al., 2017). Discovering race-non-specific resistance genes will enhance blast resistance in rice cultivars.

Results

Through host resistance in wheat Fusarium head blight (FHB) and shares a similar lifestyle with M. oryzae

Discussion

Plant nuclear proteins do not have transactivation activity and share the same biological functions. To verify the function of OsHRC on rice blast resistance, we designed a guide RNA to target a 20-nt sequence in the 59 bp downstream of the translation start codon of OsHRC and conducted CRISPR/Cas9-mediated gene editing to knock out OsHRC in ZH11. Among the five OsHRC mutants generated, three (KO-1, KO-2 and KO-3) showed premature translation termination due to frameshifts, and were predicted to generate truncated proteins of 39, 236, and 39 amino acids, respectively (Figure 1c). The three mutants and ZH11 were inoculated with RB22, a highly virulent M. oryzae strain from China, 60 days after planting. All the three mutants showed significantly smaller lesions and less fungal biomass than their non-edited controls at 14 days post inoculation (DPI) (Figure 1d). To examine whether the resistance is race non-specific, the three mutants were inoculated with another virulent strain S005 and showed the same level of resistance as to RB22 (Figure 5a, b), demonstrating that the loss-of-function of OsHRC confers race non-specific resistance to M. oryzae.

To explore the molecular basis of OsHRC resistance to rice blast, RNA-seq was conducted to identify differentially expressed genes (DEGs) between an OsHRC KO mutant and ZH11. At 14 DPI with RB22, a total of 1333 DEGs were detected at the fold-change of 2 and false discovery rate (FDR) < 0.05 using the DESeq2 package (Figure 1g). KEGG analysis indicated that the DEGs were mainly enriched in the pathways of photosynthesis, biosynthesis of secondary metabolites, and phenylpropanoid biosynthesis (Figure 1h). Phenylpropanoids play important roles in plant responses to biotic and abiotic stresses (Su et al., 2008). Quantitative RT-PCR results demonstrated that the genes in the phenylpropanoid biosynthesis pathway showed significant changes...