Breeding plant broad-spectrum resistance without yield penalties

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A central goal of crop improvement is to breed varieties with broad-spectrum resistance (BSR) to pathogens, but most of the major resistance (R) genes identified to date confer race-specific resistance to their adapted pathogens. Although these R genes are effective for a specific pathogen, their durability in the field is typically short due to mutations in the pathogen population that overcome the resistance. An alternative strategy of incorporating multiple R genes against different pathogens into elite cultivars is time-consuming and technically challenging, and usually results in a yield penalty as the crop diverts energy to implementing disease resistance (1). Many pathogens infect rice (Oryza sativa), the staple food crop of over half of the world’s population. Among them, the fungal pathogen Magnaporthe oryzae and the bacterial pathogen Xanthomonas oryzae pv oryzae (Xoo) are two of the most destructive and can cause devastating yield losses in most rice-growing countries (2). Over the past two decades, many rice R genes have been identified, but none confers resistance to both pathogens. Although manipulating the expression of several defense-responsive genes, or genes in defense signaling pathways, has led to BSR (3, 4), few such genes have been successfully deployed in rice production for disease control. In PNAS, Zhou et al. (5) report the identification of the broad-spectrum resistance Kitaake-1 (Bsr-k1) gene, which negatively regulates BSR, and the bsr-k1 allele, which confers nonspecific BSR to both M. oryzae and Xoo without a yield penalty.

The resistant bsr-k1 mutant was identified from an ethylmethane sulfonate-treated mutant population of the japonica cultivar Kitaake that had been inoculated with seven Kitaake-compatible M. oryzae isolates in the field (5). In addition, the bsr-k1 mutant conferred enhanced resistance to 10 Kitaake-compatible Xoo isolates. Map-based cloning identified Bsr-k1, and further analysis showed that Bsr-k1 gene expression is not induced by M. oryzae infection and the BSR-K1 protein localizes in the cytoplasm.

The disease resistance phenotype of the bsr-k1 mutant results from a mutation that causes early termination of a tetratricopeptide repeat (TPR)-containing protein (5). Some TPR-containing proteins are known to be involved in mRNA metabolism (6); therefore, Zhou et al. (5) performed in vitro RNA immunoprecipitation assays and found that the BSR-K1 protein binds to the mRNAs of the phenylalanine ammonia-lyase (PAL) genes and promotes mRNA turnover in rice (Fig. 1A). In contrast, the truncated bsr-k1 protein (1–276 aa) in the bsr-k1 mutant does not bind to OsPAL mRNAs (Fig. 1B). Consistent with the RNA binding results, overexpression of OsPAL1 in transgenic rice enhances resistance to M. oryzae and causes an increase in lignin content. Together, these results demonstrate that BSR-K1 negatively regulates BSR by modulating turnover of OsPAL mRNAs.

PAL proteins are key enzymes in the phenylpropanoid pathway involved in the biosynthesis of lignin and flavonoids, and they contribute to disease resistance (7). In rice, there are nine members of the PAL gene family (8). Importantly, four of them (OsPAL1–4) are colocozed with the major resistance quantitative trait locus against Rhizoctonia solani and Xoo in the rice genome (8), and most OsPALS genes are induced by M. oryzae (9). The ospal4 mutant shows increased susceptibility to three rice pathogens (R. solani, Xoo, and M. oryzae) (8), and the OsPAL06 knockout mutant displays increased susceptibility to M. oryzae (9). These results demonstrate the importance of the OsPAL genes in BSR against diverse pathogens in rice. Similarly, Zhou et al. (5) found that the bsr-k1 mutant has higher transcript levels of OsPAL1-OsPAL7 than does Kitaake and that OsPAL1 overexpression lines have increased resistance to M. oryzae. However, unlike OsPAL4 and OsPAL06 (8, 9), suppression of OsPAL1 does not compromise resistance to M. oryzae in the bsr-k1 mutant or Kitaake background (5), probably due to function redundancy with other OsPAL genes.

Although dozens of BSR genes have been identified in crop plants (3), few of them have been used extensively in crop production due to the consequent yield penalty (1, 10). For example, the mlo gene of barley has an average yield penalty of 4.2% (11). Interestingly, the bsr-k1 mutation does not have clear adverse effects on major agronomic traits, compared with wild-type cultivars.
Kitaake, when growing in rice fields with or without high disease pressure. Furthermore, Zhou et al. (5) transferred the bsr-k1 allele into three elite rice cultivars that are popular restoring lines for hybrid rice in Southwest China. These bsr-k1-containing cultivars showed similar yields to those of their parental lines in three field tests at two locations.

Recently, the same team identified a natural allele of the transcription factor gene Bsr-d1 in rice that confers BSR to M. oryzae. A single-nucleotide mutation in the promoter of the bsr-d1 gene results in the binding of the repressive MYB transcription factor, leading to an inhibition of H$_2$O$_2$ degradation and enhanced resistance (12). The identification of the bsr-k1 allele provides another candidate gene for BSR breeding in rice.

TPR-containing proteins play central roles in the biogenesis of the photosynthetic apparatus and plant development (6). For example, the TPR-containing protein CGL71 participates in the assembly of photosystem I in Chlamydomonas reinhardtii (13). The chloroplast-localized TPR-containing proteins SG1 and WTG1 are required for chloroplast development in Arabidopsis (14, 15). TPR-containing proteins are also known to be involved in plant immunity. Suppressors of rps4-RLD (SRFR1), encoding a TPR-containing protein, modulates resistance to the bacterial pathogen Pseudomonas syringae pv. tomato strain DC3000 expressing avrRps4 (16). SRFR1 negatively regulates the accumulation of multiple R proteins, including SNC1, RPS2, and RPS4. Interestingly, SRFR1 directly interacts with another TPR-containing protein, SGT1, which is required for the function of a number of R proteins (17). These results suggest that SRFR1 associates with SGT1 to suppress R gene-mediated disease resistance. Although BSR-K1 does not interact with the rice defensome complex, including OsSGT1, as shown in this study (5), it remains to be determined whether BSR-K1 associates with any R protein that recognizes factors from M. oryzae or Xoo. Another possibility is that BSR-K1 protein may act as a susceptibility locus targeted by M. oryzae and/or Xoo.

This intriguing study establishes a link between a TPR-containing protein and PAL mRNA turnover in the context of plant BSR (Fig. 1); it also suggests a new strategy to breed durably resistant plants. However, the results also suggest several important areas of future investigation. First, the mechanism by which the BSR-K1 protein preferentially binds to different OsPAL mRNAs and promotes their turnover during pathogen infection is not known. Second, the relationship between BSR-K1 and R proteins, the OsRac1-mediated defense and other defense signaling components needs to be elucidated. Third, the location in the cytoplasm where BSR-K1 binds to OsPAL mRNAs warrants study. Fourth, it is unclear whether, and how, the higher level of lignin content directly induces the pathogenesis-related (PR) gene expression in the bsr-k1 mutant. Finally, an important question is whether moderate overexpression of OsPAL1, OsPAL2, OsPAL3, and OsPAL4 in rice leads to BSR without a yield penalty. Addressing these questions will result in deeper mechanistic understanding of BSR-K1–mediated BSR, and ultimately result in better strategies for breeding durable resistance in crops.

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