GRMZM2G145104, a RING E3 domain containing ubiquitin ligase partially suppresses Rp1D21 induced cell death in maize

Shailesh Karre, Saetbyul Kim, Peter Balint-Kurti, Gurmukh Johal, Brian Dilkes and Bong Kim

In order to defend against diverse microbial pathogens, plants rely on an innate immunity strategy which comprises recognition of pathogen elicitors by membrane bound pattern recognition receptors and intracellular nucleotide binding leucine-rich repeat receptors (NLRs) that recognize specific proteins produced by the pathogen to facilitate pathogenesis. This recognition initiates a defense response often including the so-called hypersensitive response (HR), a rapid localized cell death at the point of pathogen penetration. In maize, the intragenic recombination of two NLR's, Rp1D and Rp1dp2 produced an auto-active NLR, Rp1D21 which confers a spontaneous HR phenotype. A genome wide association study identified several SNP loci associated with variation in the Rp1D21-induced HR response. One was located within a gene *GRMZM2G145104*, encoding a predicted RING E3 ubiquitin ligase. Transient co-expression studies in *Nicotiana benthamiana* showed suppression of Rp1D21-induced HR by *GRMZM2G145104* coupled with a decrease in the levels of Rp1D21 protein suggesting that Rp1D21 may be a plausible target for *GRMZM2G145104*-mediated ubiquitination. Similarly, *GRMZM2G145104* co-expression reduced the levels of another auto-active NLR, RPM1D505V. Interestingly, additional co-expression studies indicated that co-expression with *GRMZM2G145104* did not decrease protein levels of Rp1D and Rp1dp2. These results suggest that *GRMZM2G145104* may specifically target auto-active but not 'wild-type' NLRs for degradation.

In related work using chimeric mutants we demonstrated that while the HR induced by Rp1D21 is cell autonomous, the defense response signal that induced expression of pathogenesis-related genes is not.