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Rpi-blb2 Gene-Mediated Late Blight Resistance in Plants

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Phytophthora infestans is the causal agent of potato and tomato late blight, one of the most devastating plant diseases. *P. infestans* secretes effector proteins that are both modulators and targets of host plant immunity. Among these are the so-called RXLR effectors that function inside plant cells and are characterized by a conserved motif following the N-terminal signal peptide. In contrast, the effector activity is encoded by the C terminal region that follows the RXLR domain. Recently, I performed *in planta* functional profiling of different RXLR effector alleles. These genes were amplified from a variety of *P. infestans* isolates and cloned into a Potato virus X (PVX) vector for transient *in planta* expression. I assayed for *R*-gene specific induction of hypersensitive cell death. The findings included the discovery of new effector with avirulence activity towards the *Solanum bulbocastanum Rpi-blb2* resistance gene.

The *Rpi-blb2* encodes a protein with a putative CC-NBS-LRR (a coiled-coil-nucleotide binding site and leucine-rich repeat) motif that confers *Phytophthora* late blight disease resistance. We examined the components required for *Rpi-blb2*-mediated resistance to *P. infestans* in *Nicotiana benthamiana*. Virus-induced gene silencing was used to repress candidate genes in *N. benthamiana* and to assay against *P. infestans* infections. *NbSGT*1 was required for disease resistance to *P. infestans* and hypersensitive responses (HRs) triggered by co-expression of *AVRblb2* and *Rpi-blb2* in *N. benthamiana*. *RAR1* and *HSP90* did not affect disease resistance or HRs in *Rpi-blb2*-transgenic plants. To elucidate the role of salicylic acid (SA) in *Rpi-blb2*-mediated resistance, we analyzed the response of *NahG*-transgenic plants following *P. infestans* infection. The increased susceptibility of *Rpi-blb2*-transgenic plants in the *NahG* background correlated with reduced SA and SA glucoside levels. Furthermore, *Rpi-blb2*-mediated HR cell death was associated with H_2O_2, but not SA, accumulation. SA affects basal defense and *Rpi-blb2*-mediated resistance against *P. infestans*. These findings provide evidence about the roles of SGT1 and SA signaling in *Rpi-blb2*-mediated resistance against *P. infestans*.