Transcriptome analysis reveals that *SlNPR1* mediates tomato fruit resistance against *Botrytis cinerea* by modulating phenylpropanoid metabolism and balancing ROS homeostasis

Rui Li, Yujing Li, Yuelin Zhang, Jiping Sheng, Hongliang Zhu and Lin Shen

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Abstract

Tomato is the fourth most popular fresh-market fruit, whereas most commercial tomato cultivars are particularly susceptible to *B. cinerea*. *Nonexpressor of pathogenesis-related gene 1* (*NPR1*) is a critical regulator in plant resistance against various pathogens. However, the underlying mechanism of how *SlNPR1* influences the defense against *B. cinerea* in tomato fruit remains unclear. In this study, two independent lines carrying homozygous mutation in *SlNPR1* were used for studying its role in the interaction between tomato fruit and *B. cinerea*. Our results showed that knockout of *SlNPR1* decreased the disease development of *B. cinerea* in tomato fruit. *slnpr1* fruit exhibited smaller lesion sizes, higher activities of defense enzymes, and upregulated expressions of defense genes compared to wild type (WT). In addition, reactive oxygen species (ROS) homeostasis in *slnpr1* fruit was balanced by increased activities of peroxidase (POD), superoxide dismutase (SOD) and glutathione S-transferase (GST), as well as decreased activity of catalase (CAT). Furthermore, *SlNPR1*-mediated differential expression genes (DEGs) were significantly enriched in the secondary metabolic pathways, represented by phenylpropanoid biosynthesis. Taken together, these findings revealed that knockout of *SlNPR1* resulted in increased activities of defense enzymes, changes in ROS homeostasis and activation of phenylpropanoid biosynthesis and some other signaling pathways, which contributes to resistance against *B. cinerea* in tomato fruit.