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Lesion Mimic Mutant 1 confers basal resistance to *Sclerotinia sclerotiorum* in rapeseed via a salicylic acid-dependent pathway

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Background:

Rapeseed *(Brassica napus)* is a major edible oilseed crop consumed worldwide. However, its yield is seriously affected by infection from the broad-spectrum non-obligate pathogen *Sclerotinia sclerotiorum* due to a lack of highly resistant germplasm. Here, we identified a *Sclerotinia*-resistant and light-dependent *lesion mimic mutant (lmm1)* from an EMS-mutagenised population of the rapeseed inbred Zhongshuang 11 (ZS11).

Objective:

The findings tried to shed light on the genetic mechanism underlying the role of salicylic acid metabolism in *lmm1* resistance against *S. sclerotiorum*. In addition, these results laid the foundation for the breeding of disease-resistant rapeseed cultivars as well as fresh insights into disease resistance breeding in *B. napus*.

Methods:

The mutant used in this study was obtained from the EMS (ethylmethane sulphonate, EMS) mutagenic treatment to *B. napus* cultivar ZS11. After three generation of self-crossings, the *lmm1* with stable lesion on leaves was chosen for further investigation. This study compared the phenotypic traits (including histochemical assay, agronomic trait evaluation, activities of antioxidant enzymes, photosynthesis characteristics, scanning electron microscopy observation to leaf epidermis, and transmission electron microscopy observation to the ultrastructure of chloroplasts, etc.) between *lmm1* and ZS11. A combination analysis of genetic mapping and transcriptome analysis was used to identify candidate *LMM1* gene.

Results:

Lesion mimic spots (LMS) appeared in the mutant at the early bolting stage. Histochemical analysis indicated that H2O2 strongly accumulated, and cell death occurred around the lesions in *lmm1*. Inheritance analysis suggested the phenotype of lmm1 is controlled by a single recessive gene, which we mapped onto chromosome C04 by bulked segregant analysis, corresponding to three continuous segments within a 2.71-Mb interval. Comparative transcriptome analysis identified 188 differentially expressed genes (DEGs) were enriched in defence response, including 95 DEGs involved in systemic acquired resistance (SAR), which is consistent with the measured higher salicylic acid levels in *lmm1*. We identified the DEG *BnaC4.PR2*, encoding a β-1,3-glucanase 2 protein involved in SAR, as the candidate gene for *LMM1*. We propose that *BnaC4.PR2* induces a reactive oxygen species burst by influencing the expression of *Bna.SnRK2.9* to trigger partial cell death and SAR, which enhances *Sclerotinia*-resistance in lmm1.

Conclusions:

Based on transcriptome analysis and QTL resequencing, and analysis of physiological indicators, we determined that the improved resistance to *S. sclerotiorum* of *lmm1* is likely due to a mutation in *BnaC4.PR2*, encoding BGL2. The overexpression of BGL2 in *lmm1* might break the integrity of the plant cell wall, allowing them to release cell wall-related immune stimulators under normal growth conditions. This, in turn, leads to the production of high concentrations of ROS, resulting in localized cell death and SA accumulation in the mutant. Additionally, SA-mediated SAR and *PR* genes might be induced in *lmm1* to further improve resistance to *S. sclerotiorum* under normal growth conditions, but further experimental evidence is needed to support this notion. These findings provide valuable insights into the molecular mechanisms that enhance resistance to *S. sclerotiorum* in rapeseed via the SA pathway.