***Pieris brassicae* egg-induced cell death**

**in *Brassica nigra* is mediated**

**by a single locus containing**

**a cluster of TIR-NBS-LRR receptors**

## **Abstract**

The hypersensitive response (HR) characterizes monogenic qualitative resistance traits in several pathosystems. Its role in resistance to insects is relatively understudied and limited to a few resistance (*R*) gene-based defense responses against piercing-sucking insects.   
The hypersensitive response (HR)-like cell death induced by egg deposition ofcabbage white butterflies (*Pieris* spp.) in *Brassica* spp. reduces egg survival and represents an effective resistance trait before that feeding larvae emerges. However, its implementation as defence trait is conditional on the understanding of its genetic basis. In this study, we found that *P. brassicae* egg-induced HR segregates as a Mendelian trait in wild accessions of black mustard *B. nigra* L. Through bulk-segregant analysis coupled with whole-genome sequencing (BSA-seq), we identified a single dominant locus on chromosome B3 which we named *PEK (Pieris* egg-killing). Fine-mapping through recombinant analysis restricted the *PEK* locus to a ~50 kb region that contains several tandemly duplicated genes, including a cluster of potential candidate resistance TIR-NBS-LRR (TNL) receptor proteins. We found that *PEK* is polymorphic between the parental accessions of our crossing scheme and shows copy number variants (CNVs) of TNL genes among *B. nigra* reference genomes. These results highlight the need for a complete *de novo* assembly of the *PEK* locus from our parental accessions to precisely fine map the causal locus and/or polymorphism. Further fine-mapping of the *PEK* locus will reveal whether the TNLs are responsible for the HR phenotype, while studying the diversity of the locus among Brassicaceae will shed light on the evolutionary basis of HR.

**Keywords**

Crop wild relatives (CWR), *Brassica* crops, cabbage white butterfly, plant-insect interaction, Bulk Segregant Analysis, *k*-mers, nucleotide-binding leucin rich-repeat (NLR)