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IDENTIFICATION OF A POSITIVE REGULATOR OF NITRIC OXIDE SIGNALLING DURING PATHOGEN INDUCED HYPERSENSITIVE CELL DEATH IN *ARABIDOPSIS THALIANA*

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Nitric oxide is a signal molecule largely diffused in different organism and, in particular, it plays a key role in the plants growth and stress tolerance. Previous studies have shown the relevance of this molecule in the activation and mediation of signalling during biotic stress. More in detail, the accumulation of nitric oxide typically occurring after invasion by avirulent pathogens modulates gene expression and contributes to the activation of an hypersensitive response including cell death at infection site aiming to restrict pathogen growth. Nevertheless, the molecular mechanisms at the base of nitric oxide network and activity during hypersensitive response and cell death triggering are still unclear. To further enquire these mechanisms, we have set up a genetic screening on 56.332 M_2 EMS-mutagenized *Arabidopsis thaliana* plants by using a fumigation system for plant treatment with nitric oxide. Treatment conditions leading to cell death were first established. Then mutant lines compromised in the nitric oxide induced activation of cell death were searched. This has allowed the identification of 14 mutant lines compromised in nitric oxide triggered cell death activation, as well as activation of cell death during avirulent pathogen infection. In order to identify the causal mutation, a mapping by sequencing approach was adopted, based on bulked pools in a backcrossed population for one of these mutants. NGS sequencing data highlighted a genomic region with allelic frequency enrichment for EMS-induced mutations in the initial portion of chromosome III in the phenotype selected pool. Nine candidate mutations in coding regions, one of which leading to a stop codon were found in this region. Putative candidate genes involvement was then further enquired by characterization of nitric oxide response and pathogen induced hypersensitive cell death in corresponding T-DNA knock-out Arabidopsis lines. Among these lines, the knock-out for the gene affected by the mutation leading to a stop codon showed a strong impairment in cell death activation, which was confirmed in both tested conditions. Further characterizations of the possible involvement of this gene as positive regulation in cell death activation during pathogen induced hypersensitive response are in progress, as well as complementation for phenotype rescue in the mutant line.