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A MUTATION IN THE FZL GENE OF *ARABIDOPSIS* CAUSES A LESION MIMIC PHENOTYPE

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Lesion Mimic Mutant (LMM), Hypersensitive Response (HR), chloroplast, Reactive Oxygen Species (ROS), signalling pathway

We report on the characterization of an Arabidopsis mutant showing reduction in size, compared with the wild type, and presence of chlorotic lesions on rosette leaves. These traits are characteristic of a group of mutants displaying spontaneous HR cell death, the lesion mimic mutants (LMM). They are useful tools to unravel the regulatory mechanisms of this form of PCD (Programmed Cell Death), in which reactive oxygen species (ROS), nitric oxide (NO) salicylic acid (SA), jasmonic acid (JA) and ethylene are known to play a central role as signalling molecules. Histochemical and expression analysis showed that lesions formation on mutant rosette leaves correlates with the presence of specific biochemical and molecular markers usually associated with the HR cell death program.

Analysis of double mutants, obtained by crossing our mutant with mutants affected in HR signalling pathways, showed that the mutant phenotype was dependent on SA but independent from JA and ethylene signalling.

Positional cloning and sequence analysis indicated that the mutation was in the FZL gene, encoding for a membrane-remodelling GTPase with a unique role in the determination of thylakoid and chloroplast morphology, and histological analysis confirmed the presence in mutant leaves of chloroplasts with altered shape, size and number in comparison with the wild type.

Chloroplasts are the major ROS source in plant cells and even if the signalling role played by ROS in HR progression has long been recognised many aspect of ROS function remain obscure, thus the characterisation of this LM mutant will help in understanding the role played by chloroplast-derived ROS in HR signalling pathway.