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CHARACTERIZATION OF AN ARABIDOPSIS MUTANT IMPAIRED IN DEFENSE PATHWAYS

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Programmed cell death (PCD) is a metabolically active and genetically controlled process leading to cell death.

In plants this process occurs during normal development and senescence, and during interaction with the environment, in biotic and abiotic stress response.

One of the most studied form of PCD in plants is the cell death associated to an avirulent pathogen attack, known as hypersensitive response (HR). In this process, the death of cells challenged by the pathogen can be seen as a first barrier to limit pathogen growth in plant tissues.

We report on the characterization of the Arabidopsis mutant *chlorotic lesions1* (*cll1*).

The two main characteristics of this monogenic recessive mutant are a reduction in size respect to wild type plant and the presence of chlorotic lesions on rosette leaves. This latter trait reminds of the typical phenotype shown by the so called “lesion mimic mutants”, a group of mutants characterized by alterations in the hypersensitive response pathway.

To better understand the nature of the lesions present in *cll1* mutants we have checked for the presence, in mutant rosette leaves, of specific markers associated to HR by histochemical analyses. We have also analyzed the expression level of two senescence-associated genes (SAG) and a group of pathogen-related /stress-related genes, constitutively expressed in the lesion mimic mutants. Phenotypic, histochemical and molecular analyses have been performed also on *cll1* mutants aseptically grown on MS medium.

The data thus far obtained indicate that the lesions formation in *cll1* mutants correlates with the expression of histochemical and molecular markers of plant disease resistance responses.

The *cll1* mutant has been isolated in the Exotic collection, based on the *Ac/Ds* transposon system of maize. Cosegregation analysis showed that the mutant phenotype did not cosegregate with the *Ds* element, the positional cloning of the *cll1* mutation is in progress.