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USE OF WILD *LYCOPERSICON* ACCESSIONS AND NEAR ISOGENIC LINES OF *LYCOPERSICON HIRSUTUM* FOR THE IDENTIFICATION HOST FACTORS INVOLVED IN THE SUSCEPTIBILITY TO *CUCUMBER MOSAIC VIRUS* AND ITS SATELLITE RNA

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Several accessions of wild and cultivated species of the genus *Lycopersicon*, including *L. esculentum* (tomato), *L. hirsutum*, *L. chilense*, *L. pimpinellifolium*, *L. pennellii* and *L. peruvianum*, were tested for their susceptibility to combinations of *Cucumber mosaic virus* (CMV) with variants of satellite RNA (satRNA) co-inducing diverse disease phenotypes in tomato. The following CMV preparations have been used for the characterisation of *Lycopersicon* spp./CMV/satRNA interactions: a) CMV-Fny (no satRNA); b) CMV-FB (Fny + a Benign satRNA variant, symptomless on tomato); c) CMV-FS (Fny + a tomato Stunting-inducing satRNA variant); d) CMV-FN (Fny + a tomato Necrosis-inducing satRNA). CMV-Fny induced on all hosts the typical leaf malformation ("shoestring", reduced leaflet blade), and no resistance was observed on any of the tested genotypes. CMV-FB induced on all hosts a phenotype characterised by latent infection accompanied by the down-regulation of viral RNA replication, referred to as LIDR (Latent Infection, Down-Regulation). Interestingly, LIDR was observed also on some wild *Lycopersicon* accessions inoculated with CMV-FS and CMV-FN that are aggressive on tomato. In particular, accessions of *L. chilense* showed LIDR with both the aggressive CMV/satRNA combinations tested, whereas accessions of *L. hirsutum* and *L. pennellii* showed LIDR upon inoculation with CMV-FS, but susceptibility to systemic necrosis induced by CMV-FN.

A screening of 99 near isogenic lines, containing single introgressions from *L. hirsutum* accession LA1777 in the *L. esculentum* cv. E6206 genetic background (Monforte and Tanksley, Genome/Génome 43: 803, 2000), was undertaken to map host factors determining differential susceptibility to CMV/satRNA in *Lycopersicon* spp. Observing a temporary resistance to CMV-FS on one of these lines, a possible host factor interfering with stunting, but not with systemic necrosis, was mapped genetically to *L. hirsutum* chromosome 6. Other loci were identified, correlating with changes in symptoms expression rather than with absence of symptoms.

From these preliminary studies the following conclusions are drawn: a) CMV/satRNAs diverse symptoms (leaf malformation, stunting, necrosis) on host *Lycopersicon* spp. are due to alterations of apparently individual and uncoupled pathways; b) *L. chilense* can be a source of gene(s) responsible for protection from CMV/satRNA-induced lethal necrosis; c) the LIDR phenotype appears to be regulated by a multigenic character not directly transferable to cultivated tomato genotypes.