

ADVANCES IN THE CHARACTERIZATION OF TOMATO MUTANTS PUTATIVELY AFFECTED IN CLASS B MADS-BOX TRANSCRIPTION FACTORS

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The interest towards male sterile mutants in tomato dates back long time ago due to the perspective of using male sterility (MS) in hybrid seed production. In order to solve problems related to the use of genic MS, the selection of conditional MS mutants, where sterile anthers are restored to fertility by permissive growth conditions, has been regarded as a useful strategy. Therefore, mutants with conditional expression, such as *stamenless* (*sl*), *stamenless-2* (*sl-2*), *7B-1* and *variable male sterile* (*vms*), have been deeply studied in the past. Literature data and examination of the phenotype indicated as candidates for these mutations members of the class B MADS-box transcription factors family, that specify petal and stamen organ identity. In *Arabidopsis thaliana* and *Antirrhinum majus*, there are two class B genes that, referring to the *A. majus* nomenclature, are known as *DEFICIENS* (*DEF*) and *GLOBOSA* (*GLO*). In tomato, as in most solanaceae, there are four class B MADS-box genes, two *DEF*-like members (*SIDEF* and *SITM6*) and two *GLO*-like members (*SIGLO1* and *SIGLO2*). In this research, we describe our recent advances towards the identification of the gene underlying the *7B-1* mutation. The tomato *7B-1* mutant, is male sterile in long days, while in short days it produces several male fertile flowers. *7B-1* mutant has a strong potential for production of tomato hybrid seeds. To identify the gene underlying this mutation would be very important in order to pursue marker-assisted selection strategies and the discovery of new alleles by the screening of large mutagenized populations. Through genetic analysis, we demonstrated that *7B-1* is allelic to *sl-2*, but not to *sl* (involved in *SIDEF*) as it was previously hypothesized. Accordingly, analysis of two independent mapping populations, one segregating *sl-2* and one segregating *7B-1*, excluded the involvement of these mutants in the *SIDEF* locus (Chromosome 4). To identify the candidate gene of *7B-1* mutation, we developed a backcross mapping population after crossing the mutant and *S. pennellii*. We have characterized the mapping population at molecular level using markers representing the class B MADS box genes or associated to them. The molecular screening showed that the best candidate gene to represent the *7B-1* mutation is the *SIGLO2* gene (Chromosome 6). Complementation of the mutant to confirm the candidate gene is under way.