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# **A transposable element from the hAT-superfamily is responsible for the absence or globose-shape phenotype of leaf extrafloral nectaries in peach (*Prunus persica* L. Batsch)**

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## **Abstract**

Most peach (*Prunus persica* L. Batsch) cultivars have extrafloral nectaries (EFNs), or leaf-glands, on the leaf petioles, stipules, or margins (Gregory 1915, Okie 1998). From an extensive study, Gregory (1915) observed that the great majority of varieties was found to have well-defined gland shapes. Moreover gland-shapes were generally homogenous on typical shoots, although some cultivars could exhibit mixed glands. This author identified three main types of leaves, those with reniform glands, those with globose glands and glandless leaves. Fruit breeding programs have inadvertently produced peach cultivars with glandless leaves (Okie 1998), yet without determining the effects on either natural enemies or herbivorous pests (Scorza and Sherman 1996). Empirical observations, however, showed that the absence of EFNs resulted in a higher susceptibility to peach powdery mildew (PPM), a major disease of the peach caused by *Podosphaera pannosa* var. *persicae* (Weinhold 1961; Monet 1983). As a result, peach seedlings without leaf-glands were systematically discarded in the breeding programs. The Mendelian inheritance of the leaf-gland phenotype (*E/e*) was first described by Connors (1921) and the *E* locus was latter mapped on chromosome 7 of the peach (Dettori et al., 2001), but without identifying the genetic factor involved. In order to address the latter point and implement early diagnosis tools, we developed a mapping population of 833 progenies derived from the selfing of ‘Malo Konare’, a canning peach cultivar with globose leaf-glands, from Bulgarian origin. This population was used to map and investigate the genetic factor underlying the *E* locus, using additional resources including NGS peach resources and 148 cultivars from various origins. Our findings demonstrated that a MITE-like *Moshan* transposable element inserted in the gene controlling the character was responsible for the absence or globose-shape phenotype of the EFNs.