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A mutation, *tl2*, in pea (*Pisum sativum* L.) affects leaf development only in the heterozygous state

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Abstract After gamma irradiation of pea seeds, a mutation designated as *tendril-less2* (*tl2*) was induced. In the heterozygous state, it transforms tendrils into very narrow leaflets that resemble the heterozygote phenotype of the classic *tl* mutation. The tendrils of the double heterozygote *tl2*/+, *tl*/+ are converted into oval leaflets. Unlike *tl*, the novel mutation in the homozygous state does not affect tendrils. The leaf phenotype of homozygotes *tl2*/*tl2* and *Tl2*/*Tl2* do not differ in the *tl*/+ background. However, the anthocyanin pigmentation is strongly suppressed in petals of *tl2*/*tl2* plants. Some hypotheses to explain the unusual phenotypic manifestation of *tl2* are suggested.

Introduction

Flowering plants dominate the vegetation of most terrestrial ecosystems and comprise about 300,000 species. This evolutionary success obviously results from an ability for rapid adaptation to diverse environments. A substantial role in these processes belongs to alterations in morphology of the leaves, which are the main photosynthetic organs of a plant. There are two types of leaves: the simple leaf that consists of a single lamina and the compound leaf with the blade divided into a set of leaflets. While simple leaves vary in shape and size, the variation of compound leaves involves various leaflet characteristics such as the shape, number, spatial arrangement, and even functional differentiation (Esau 1977). Genetic mechanisms responsible for this variability are yet to be understood. Only two plant species with compound leaves, pea (family Fabaceae) and tomato (family Solanaceae), are well studied genetically. Fabaceae leaves are mostly pinnate, that is, flat, bilateral, feather-like structures with the central stem-like rachis and lateral organs, called pinnae, arranged along the rachis. The pinnae of most tribes of Papilionoideae subfamily are represented by oval leaflets, but in two closely related tribes, Vicieae and Cicereae, the distal leaflets are substituted by tendrils—filamentous organs able to wind around the objects of appropriate size (Yakovlev 1991). For example, in pea (belonging to the tribe Vicieae), the leaf rachis supports pairs of proximal leaflets, distal tendrils, and a terminal tendril (Fig. 1). Climbing up grasses, bush branches, or rocks with the aid of the tendrils, a herbaceous plant can rapidly expose its leaves to the sunlight. Such a tactic minimizes the expense of strengthening the plant's stem and may represent a competitive advantage.

For about a century, a semidominant mutation, *tl*, has been known in pea that affects the leaflet–tendril transition (Vilmorin and Bateson 1912). In the homozygous state, this mutation replaces filamentous tendrils with oval leaflets indistinguishable from the proximal pinnae of a wild-type leaf. In the heterozygote *tl*/+, the tendrils are transformed into leaflets with very narrow (sometimes hardly visible) laminae (Fig. 1b). Since, in legumes, tendrils of this type are found only in two closely related tribes, it is probable that the gene *Tl* arose rather recently in evolution (Makasheva 1962), but the genetic basis of this innovation remains unknown.

In this paper, we describe a new mutation, *tl2*, not linked to *tl*, which (like *tl*) in the heterozygous state causes formation of leaflets with narrow laminae in place of tendrils. Paradoxically, however, plants homozygous for the mutant allele *tl2* possess normal filamentous tendrils. At the same time, anthocyanin pigmentation in the flowers of *tl2*/*tl2* plants is strongly suppressed.

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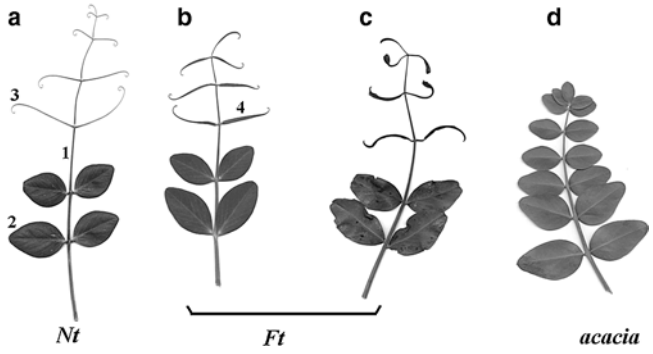

Fig. 1 Three phenotypes, normal tendrils (*Nt*), Flat tendrils (*Ft*), and tendrils replaced with leaflets (*acacia*), associated with lamina development of distal pinnae. **a** Wild-type leaf, **b** leaf of the heterozygote *tl*/+, **c** leaf of the heterozygote *tl2*/+, **d** leaf of the homozygote *tl*/*tl*. *1* Rachis, *2* leaflet, *3* tendril, *4* flat tendril (narrow leaflet)

Materials and methods

Plant growing

Seeds were planted in a greenhouse in hydroponic beds filled with drainage gravel (claydite) and fed by standard Knop nutrient solution (0.8 g/l calcium nitrate 0.2 g/l magnesium sulfate, 0.2 g/l acid potassium phosphate, 0.2 g/l potassium nitrate, and traces of ferric phosphate). Plants were illuminated by 8 h daylight/16 h incandescent light of 10,000–12,000 cd.

Genetic markers used and their chromosome location

- *tl* (chromosome 3, linkage group V): *tl*/*tl*, tendrils replaced with leaflets (phenotype acacia), *tl*/+, tendrils acquire a very narrow lamina. tl^x, a recessive embryonic lethal very tightly linked to locus *r* (see Berdnikov et al. 1999).
- *r* (chromosome 3, linkage group V), wrinkled seeds.
- *His(2-6)* (pericentromeric region of chromosome 6, linkage group II), a block of five tightly linked genes coding for subtypes of histone H1. $His6^S$ and $His6^F$ are slow and fast electromorphs of subtype 6 of histone H1.
- *crd* (the distal part of the short arm of chromosome 6, linkage group II), a long rachis with reduced pinnae.
- *af* (linkage group I), proximal pinnae are transformed into rachillae.

Lines

- WL1238 (*r tl*) and WL2715 (*af, R Tl*), standard testerlines from the Weilbullsholm (Landscrona) collection.
- SPARKLE ($His6^S$, *r Tl*), a kind gift by Dr. N. Weeden (Bozeman, Mont., USA).
- SG (*R Tl*), an original line derived from the accessions VIR6135 (Greece) and VIR320 (*Pisum sativum syriacum*, Palestine).
- DELTA ($R\ tl^x/r\ Tl$), a line with a maintained heterozygosity in the *r-tl* segment that carries the tl^x mutation (for details, see Berdnikov et al. 1999).
- WHAF (*af,* crd^{wh}), originated from a mutant crd^{wh} induced by EMS in the line SG (for details, see Berdnikov et al. 2000).
- MONO ($His6^F\ crd^{wh}$), derived from the accession VIR320 and line WHAF.

Linkage estimates

Significance of genetic linkage was evaluated by chi-square criterion; recombination fraction was estimated by the maximum likelihood method with the aid of the CROS program developed in our laboratory.

Histone H1 isolation and electrophoresis

Histone H1 was isolated with an express method (Kosterin et al. 1994). Two hundred to 400 mg of pea leaves were rubbed with a rubber-headed pestle through a stainless steel grid into a vessel containing 12 ml 0.15 M NaCl, and the resulting homogenate centrifuged (1,500 *g* for 10 min). Histone H1 was extracted by resuspending the pellet in 1 ml 5% perchloric acid. After centrifugation (3,000 *g* for 30 min), the protein was recovered from the supernatant by adding sulfuric acid to a final concentration of 0.5 M and 6 vol cold acetone. The precipitated protein was centrifuged (1,500 *g* for 10 min) and then dissolved in 0.2 ml of a medium containing 0.9 M acetic acid, 8 M urea, and 15% (w/v) sucrose.

The preparations were subjected to electrophoresis in slabs of 15% polyacrylamide gel containing 6.25 M urea and 0.9 M acetic acid (Kosterin et al. 1994). After electrophoresis, the gels were stained in 0.01% Coomassie R-250 in 0.9 M acetic acid and destained by diffusion in 0.9 M acetic acid.

Results

Phenotype of heterozygote *tl2*/*Tl2*

After treatment of seeds of the SG line with gamma rays, a plant with aberrant foliage was found in the M_2 population. This exceptional plant had leaves with tendrils converted in leaflets with very narrow laminae (Fig. 1c); this phenotype designated as Ft (Flat tendrils). Since a very similar phenotype has been described for heterozygotes for the classical allele *tl*, we supposed that this mutation represented a new allele of the *tl* locus. To test this, the exceptional plant was crossed with the multimarker line WL1238 homozygous for *tl*. In the homozygote *tl*/*tl*, tendrils are con-

verted into normal oval leaflets (acacia phenotype, Fig. 1d); therefore, if the exceptional plant was heterozygous for *tl*, the hybrids from this cross should be of two phenotypes—acacia and Ft. In accordance with this expectation, seven F_1 plants obtained were of these two phenotypes—acacia (two plants), and Ft (five plants). If the F_1 plants with the acacia phenotype were homozygous for *tl*, their self-pollination should give rise to the progenies of the same phenotype. In fact, however, 77 offspring of selfed F_1 acacia plants segregated into three phenotypic groups: 42 plants (54%) had the acacia phenotype, 26 plants (34%)—phenotype Ft and 9 plants (12%) had normal wild-type tendrils; the latter phenotype will be designated Nt (Normal tendrils). This segregation suggested that the Ft mutation was not allelic to *tl*. The new mutation was symbolized as *tl2*.

The original mutant plant with flat tendrils was also crossed with the cultivar SPARKLE (*Tl2*, *Tl*). Of five F_1 individuals, three had flat tendrils, and two had wild-type tendrils. One of the Ft plants was chosen to establish the line FLAT-1. For eight generations, one vigorous plant with flat tendrils was chosen and self-pollinated. After this period of isogenization, the line was maintained by selfing of Ft plants. F_1 hybrids from the crosses of FLAT-1 Ft plants with unrelated lines, carrying wild-type tendrils (genotype *Tl2*/*Tl2*), segregated into two phenotypic classes, Nt and Ft, in a ratio close to 1:1 (Table 1). This mode of inheritance suggested that Ft plants of the line FLAT-1 had the genotype *tl2*/*Tl2*.

The progeny of self-pollinated Ft plants of the line FLAT-1 also contained plants of only two phenotypic classes—Ft and Nt in a ratio close to 1:1 (Table 1). It remained unclear why we did not find plants with the acacia phenotype among the offspring of selfed Ft plants. If homozygotes *tl2*/*tl2* died, the ratio of phenotypes Ft:Nt would be close to 2:1, but we observed a 1:1 ratio.

We noticed that the first cross of the original mutant plant with the line WL1238, homozygous for *tl*, produced hybrids some of which had the acacia phenotype. This result was reproduced in crosses of the homozygote *tl*/*tl* with Ft plants from the line FLAT-1 (Table 1). The progenies from these crosses segregated into two phenotypes, acacia and Ft, in a ratio close to 1:1. Since the supposed genotype of Ft plants is *tl2*/+, the F_1 individuals with the acacia phenotype should be double heterozygotes, *tl2*/*Tl2*, *tl*/*Tl*.

Phenotype of homozygote *tl2*/*tl2*

Among F_1 hybrids from the cross FLAT-1 (*His6*^{S}/*His6*^{S}, *tl2*/+,*Crd*/*Crd*) × MONO (*His6*^{F}/*His6*^{F}, *Tl2*/*Tl2*, *crd*/*crd*), we chose four plants with flat tendrils and analyzed their progenies from self-pollination (Table 2). Eighty of 85 plants heterozygous for *tl2* (Ft phenotype) were also heterozygous for the gene *His6*, a member of the cluster *His(2-6)* of genes encoding subtypes of histone H1. This implied a strong linkage between the loci *tl2* and *His6*. Since the mutant allele *tl2* in the FLAT-1 line was coupled with the allele *His6*^{S}, coding for the slow electromorph of H1 subtype 6, the majority of *His6*^{S} /*His6*^{S} segregants must be also homozygous for *tl2*. Table 2 shows that 39 of 43 *His6*^{S}/*His6*^{S} plants had wild-type tendrils (phenotype Nt). Therefore, we concluded that homozygotes *tl2*/*tl2* should have phenotype Nt. To our surprise, all 39 homozygotes *His6*^{S}/*His6*^{S} with normal tendrils had "pale" flowers, practically lacking anthocyanin pigmentation in the petals except for rose rims of wings in some flowers (Fig. 2a). Other parts of the plants (leaf axils, stems, pedicles, pods, and seed coats) retained normal anthocyanin coloration. Similar phenotype referred to as "albicans" is recorded for some pea mutations, e.g. *am1* (Blixt 1972).

Forty-nine of 50 homozygotes for the "fast" allele *His6*^{F} (encoding the fast electromorph of H1 subtype 6) had wild-type tendrils and flowers with the normal

Table 2 Phenotypes of F_2 progeny from the cross FLAT-1 (*His6*^{S}, *tl2*/+, *Crd*) × MONO (*His6*^{F}, *Tl2*, *crd*)

Leaf and flower traits[a]	Allelic composition of *His6*		
	S	S/F	F
Nt, albicans, Crd	39	4	0
Nt, Bright, Crd	0	0	5
Nt, Bright, crd	0	7	44
Ft, Bright, Crd	4	70	1
Ft, Bright, crd	0	10	0

[a] *crd* Number of pinnae reduced, *Crd* normal number of pinnae, *Albicans* pale flowers, *Bright* bright-colored flowers

Table 1 Phenotypes of the offspring from crosses of *tl2* carriers with different testers used as a male (*m*) or female (*f*). *Nt* Normal tendrils, *Ft* Flat tendrils, *Acacia* leaflets instead of tendrils

Genotype of tester	Phenotype of *tl2* carrier	Nt	Ft	Acacia	Presumptive genotype of *tl2* carrier	χ^2 [1:1]
Tl/*Tl*,*Tl2*/*Tl2*; m	Ft	49	48	0	*Tl*/*Tl*, *tl2*/+	0.01^{NS}
Tl/*Tl*,*Tl2*/*Tl2*; f	Ft	12	10	0	*Tl*/*Tl*, *tl2*/+	0.18^{NS}
tl/*tl*, *Tl2*/*Tl2*; m	Ft	0	33	29	*Tl*/*Tl*, *tl2*/+	0.26^{NS}
Self-pollination	Ft	90	86	0	?	0.09^{NS}

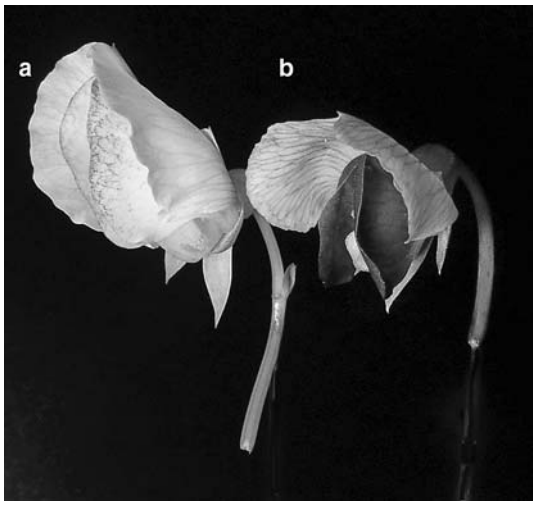

Fig. 2 Flower of the homozygote (**a**) and heterozygote (**b**) for *tl2*

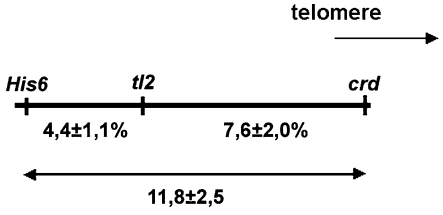

Fig. 3 Recombinational relationship among three loci of chromosome 6

bright anthocyanin pigmentation. So we concluded that both types of homozygotes, *tl2*/*tl2* and *Tl2*/*Tl2*, do not differ in the leaf morphology but strongly differ in anthocyanin pigmentation of the flower.

Further, we made crosses of *tl2* carriers with unrelated plants homozygous for the wild-type allele *Tl2* and obtained several hundreds of F_2 progenies. However, we failed to find individuals combining pale flowers with flat tendrils: all plants with flat tendrils had brightly colored flowers, and all plants with pale flowers had normal filamentous tendrils. In the course of FLAT-1 breeding, we left for propagation only Ft plants, while Nt plants were removed before flowering. However, when allowed to grow up to flowering, about a half of Nt plants had pale flowers. One such plant with pale flowers was chosen to establish the line FLAT-2. All plants of this line had wild-type tendrils and pale flowers. The progenies produced by crosses of FLAT-2 with unrelated lines, homozygous for the wild-type alleles, *Tl2* and *Tl*, had brightly colored flowers and flat tendrils. Thus, we can conclude that the drastic decrease of anthocyanin pigmentation in flowers is caused by the *tl2* mutation in the homozygous state. In other crosses, the level of flower pigmentation of *tl2*/*tl2* plants was found to be the same in both *A*/*A* and *A*/*a* backgrounds. The *a*/*a, tl2*/*tl2* plants were fully acyanic.

Table 2 allows an estimate of the linkage relations among *His6, tl2*, and *crd* to be made (Fig. 3). We assumed that the plants with wild-type filamentous tendrils and pale flowers were homozygous for *tl2*, the plants with wild-type tendrils and normally colored flowers were homozygous for *Tl2*, and the plants with flat tendrils and normal flowers were heterozygotes *tl2*/*Tl2*. According to consensus linkage map of pea (Weeden et al. 1998), the gene cluster *His(2-6)* resides in the pericentromeric region of chromosome 6, and *crd* is mapped to the distal part of its short arm; therefore, *tl2* should be situated within the short arm of chromosome 6, linkage group II. Noteworthy, the locus *am1* resides on chromosome 1, linkage group VI (Weeden et al. 1998).

Phenotype of *tl2*/*tl2* in the background of heterozygote *tl*/+

Earlier we induced by gamma irradiation a mutation tl^x (most probably a short deletion), which is embryonic lethal in the homozygous state, while heterozygotes with the wild-type allele, *Tl*, have flat tendrils (Gorel et al. 1994). In the consensus linkage map (Weeden et al. 1998), the *tl* locus is separated from the *r* locus (wrinkled seeds) by about 4.5 cM, whereas the tl^x mutation reduced this distance to 0.1 cM (Berdnikov et al. 1999). The lethality of tl^x and its strong linkage to the dominant allele *R* (round seeds) allow us to maintain easily the chromosome segment *R*-tl^x in the heterozygous state. In the line DELTA (*r Tl*/*R* tl^x), embryos homozygous for *R* tl^x die during early development, so that actually, all round seeds produced by the selfed *r Tl*/*R* tl^x plants are of the parental genotype. It should be mentioned that the lamina width of narrow leaflets of heterozygote tl^x/*Tl* is very susceptible to action of modifiers (Berdnikov et al. 1999; Bogdanova et al. 2000). Therefore, it is quite easy to test the phenotypic effects of the allele composition in *Tl2* locus in the background of tl^x/*Tl*.

Plants heterozygous for both *tl2* and *tl* were grown from the round seeds resulting from the cross FLAT-2 (*tl2*/*tl2; r Tl*/*r Tl*) × DELTA (*Tl2*/*Tl2, r Tl*/*R* tl^x). They had the genotype *tl2*/*Tl2, r Tl*/*R* tl^x and the phenotype acacia, that is, oval leaflets instead of tendrils. Among their progeny from selfing, the plants with the genotype *tl2*/*tl2, r Tl*/*R* tl^x can be easily recognized. These are the plants with pale flowers grown from the round seeds. All the plants selected in such a way had flat tendrils, and produced after selfing both round and wrinkled seeds. The wrinkled seeds gave rise to plants with wild-type tendrils and pale flowers (the genotype *tl2*/*tl2, r Tl*/*r Tl*), while the plants grown from the round seeds reproduced the parental phenotype, that is, flat tendrils and pale flowers (the genotype *tl2*/*tl2, R* tl^x /*r Tl*). Leaf phenotypes at different combinations of alleles in the loci *Tl* and *Tl2* are given in Table 3. Thus, even in the background of the tl^x /+, very susceptible to modifier

Table 3 Leaf and flower phenotype depending on allelic composition of *Tl* and *Tl2* loci

Allelic composition of *Tl*	Allelic composition of *Tl2*	Leaf phenotype	Flower phenotype
+/+	+/+	Nt	Bright
+/+	*tl2*/*tl2*	Nt	albicans
+/+	*tl2*/+	Ft	Bright
tl/+	+/+	Ft	Bright
tl/+	*tl2*/*tl2*	Ft	albicans
tl/+	*tl2*/+	Acacia	Bright
tl/*tl*	+/+	Acacia	Bright

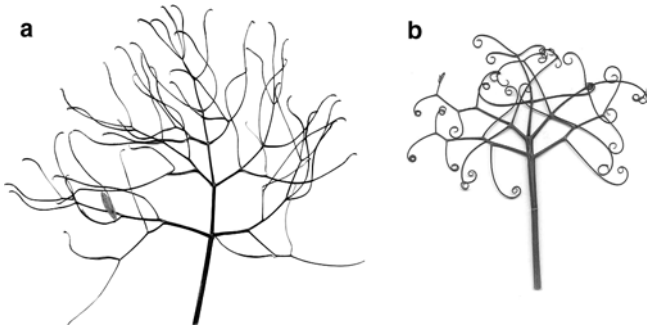

Fig. 4 Leaf of the heterozygote *tl2*/+ in the *afila* background. **a** genotype *tl2*/+, *af*/*af*; **b** genotype *Tl2*/*Tl2*, *af*/*af*

action, the leaf phenotype of *tl2*/*tl2* plants, does not differ from that of the homozygotes for wild-type allele *Tl2* (compare rows 4 and 5 in Table 3).

Expression of *tl2* in the background of mutation *afila*

There is a specific effect of the *tl* mutants that is expressed in a background homozygous for the *afila* (*af*) mutation. In *af*/*af* plants, the leaflets are converted in the second-order raches (rachillae) bearing unbranched tendrils (Kujala 1953). In the *af*/*af* background, rachillae of *tl*/*tl* plants undergo up to four rounds of branching, and the terminal branches bear miniature leaflets (Goldenberg 1965). In *af*/*af*, *tl*/*Tl* leaves, the proximal rachillae often exhibit additional (in comparison with *af*/*af*, *Tl*/*Tl*) round of branching (Villani and DeMason 1999).

In F_2 progeny from the cross FLAT-2 (*Af, tl2, Tl*) × L2715 (*af, Tl2, Tl*), we obtained all combinations of alleles in the *Tl2* locus in the *af*/*af* background. The *tl2*/*Tl2*, *af*/*af* plants (recognized by flat tendrils and brightly colored flowers) exhibited increased complexity of proximal rachillae (Fig. 4a). That is, the leaf phenotype of *af*/*af*, *tl2*/+ plants closely resembled that of the *af*/*af*, *tl*/+ plants. At the same time, the leaf architecture of the double homozygotes *tl2*/*tl2*, *af*/*af* (recognized by pale flowers) did not differ from that of *af*/*af* plants. So we may conclude that, even in combination with *af*/*af*, the mutant allele *tl2* in the homozygous state does not affect the pattern of rachilla branching.

Discussion

We have shown that the new mutation *tl2*, in the homozygous state, suppresses antocyanin pigmentation in the flowers and, in the heterozygous state, causes formation of tendrils with very narrow laminae. The heterozygous effect of *tl2* does not differ from that of the classical mutation *tl* (Lamm 1957; Makasheva 1962) or the null mutation tl^x (Berdnikov et al. 1999). Moreover, the double heterozygote *tl*/+, *tl2*/+ has oval leaflets instead of tendrils and does not differ in this repect from the homozygote *tl*/*tl*. The heterozygotes *tl2*/+ and *tl*/+ resemble one another in the *af*/*af* background as well, suggesting that both genes affect in a similar way not only lamina formation but also the branching of rachillae. However, homozygotes for these two mutations are strikingly unalike. In homozygotes *tl*/*tl*, the tendrils are replaced with oval leaflets, while homozygotes *tl2*/*tl2* have wild-type filamentous tendrils. This equality of *tl2*/*tl2* and *Tl2*/*Tl2* effects on leaf architecture is retained in the background of *af*/*af* and even in a very provocative background of *tl*/+.

Unlike *tl*, *tl2* affects also flower coloration. Since *tl2* was induced by gamma rays, it is feasible to assume that this mutation is a deletion covering two different genes. Noteworthy, we always observed good correspondence to Mendelian segregation ratios 1:2:1 and 1:1 for the *tl2* alleles, implying that the putative deletion was not too large to affect gametophyte viability. If the entire *Tl2* gene were deleted, its product would be missing; however, the tendrils of *tl2*/*tl2* plants are indistinguishable from those of *Tl2*/*Tl2*, suggesting that the protein product of the mutant allele *tl2*, TL2*, is quite effective as a factor required for development of wild-type tendrils. Hence, we have to suppose that the putative deletion could only modify the product function.

However, the most intriguing feature of *Tl2* is that leaf development is disturbed only in the presence of the products of both mutant and normal alleles, *tl2* and *Tl2*. The normal phenotype of homozygotes for the mutant gene can hardly be explained if the corresponding protein product Tl2 functions as monomer. In this case, the mutant phenotype of the homozygote *tl2*/*tl2* would most probably be more severe than that of the heterozygote. More likely, Tl2 could function as a subunit of multimeric complex. We may hypothesize that the protein product of *Tl2* acts as a dimer, and only dimers with identical subunits (produced by mutant or wild-type allele) are functional, e.g., if *tl2* mutation changes the length of the TL2 molecule, creating steric difficulties for proper dimerization.

We also cannot rule out a possibility that both the leaf morphology and flower coloration effects result from the same mutation affecting one gene. Since in various plants, the genes regulating antocyanin pigmentation encode transcription factors (Dooner et al. 1991), it is plausible that TL2 protein is a transcriptional activator of some structural genes encoding enzymes of

the anthocyanin pathway. One can hypothesize that the region of TL2 affected by mutation *tl2* participates directly in regulation of the anthocyanin gene expression, and so the effect of *tl2* mutation is manifested in the homozygous state. We may suppose that *Tl2* participated originally in the regulation of flower pigmentation, but then it was involved into the program of leaf development retaining the ability to regulate anthocyanin expression in petals.

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