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Short communication

Expression of a repressor form of the *Arabidopsis thaliana* transcription factor TCP16 induces the formation of ectopic meristems

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ABSTRACT

Plants that express a fusion of the *Arabidopsis thaliana* class I TCP transcription factor TCP16 to the EAR repressor domain develop several phenotypic alterations, including rounder leaves, short petioles and pedicels, and delayed elongation of sepals, petals and anthers. In addition, these plants develop lobed cotyledons and ectopic meristems. Ectopic meristems are formed on the adaxial side of cotyledon petioles and arise from a cleft that is formed at this site. Analysis of the expression of reporter genes indicated that meristem genes are reactivated at the site of emergence of ectopic meristems, located near the bifurcation of cotyledon veins. The plants also show increased transcript levels of the boundary-specific *CUP-SHAPED COTYLEDON* (*CUC*) genes. The results suggest that TCP16 is able to modulate the induction of meristematic programs and the differentiation state of plant cells.

1. Introduction

TCP proteins are plant-specific transcription factors that regulate several aspects of plant development (Martín-Trillo and Cubas, 2010; Uberti-Manassero et al., 2013). They show a conserved domain, the TCP domain, which is involved in DNA binding and dimerization and resembles the basic-helix-loop-helix (bHLH) domain present in many eukaryotic transcription factors. There are 24 members of the TCP family in Arabidopsis, which can be divided into class I (also named PCF-like proteins) and class II according to sequence conservation within the TCP domain. Class II proteins can be further divided into two major clades: CYC/TB1 and CIN-like proteins (Martín-Trillo and Cubas, 2010; Uberti-Manassero et al., 2013). Class I and class II TCP transcription factors show different, though rather similar, DNA binding preferences: GTGGGNCC and GTGGNCCC, respectively (Kosugi and Ohashi, 2002; Viola et al., 2012). It has been hypothesized that proteins from both classes play antagonistic roles in cell differentiation and organ growth (Li et al., 2005). Class I proteins are characterized by a shorter basic region, since they lack a putative four-amino-acid loop that joins two basic stretches presumably involved in direct interactions with DNA (Aggarwal et al., 2010). Class I and class II basic regions also differ in certain amino acids otherwise conserved inside each class, which may explain the different DNA binding preferences of proteins from the two classes. Indeed, one of these residues, located at the equivalent positions 11 and 15 of the class I and II TCP domains, respectively, has been shown to determine the binding preference for a class I or a class II binding site (Viola et al., 2012). Class I proteins usually contain Gly at this position, while class II proteins have Asp. An exception is Arabidopsis class I TCP16, which contains Asp at position 11 and, according to the rule, shows a preference for a class II binding site. This raises questions about the role of TCP16.

Previous studies showed that *TCP16* is expressed in young leaf primordia and at early stages of pollen development (Takeda et al., 2006). RNA interference studies expressing a double stranded RNA from the native *TCP16* promoter suggested a role of this gene in pollen development. Considering the specific features of the TCP16 TCP domain, we were interested in performing more detailed functional studies of this protein. For this purpose, we expressed a dominant repressor form of TCP16 in Arabidopsis and found that this protein is able to promote the formation of

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ectopic meristems and shoots from cotyledon tissues and that this is related to the activation of meristem-specific genes. This suggests that TCP16 is able to modulate the differentiation state of plant cells, as it has been suggested for Arabidopsis class II proteins.

2. Materials and methods

2.1. Plant material and growth conditions

Arabidopsis thaliana ecotype Columbia (Col-0) was used as the wild type for this study. The *KNAT1* (Ori et al., 2000) and *CLV3* (Brand et al., 2002) reporter lines were obtained form the Arabidopsis Biological Resource Center (ABRC, Ohio State University). The *STM* reporter line (Spinelli et al., 2011) was a kind gift of Javier Palatnik (IBR, Rosario, Argentina). Plants were grown on soil at 22–24 °C under long-day photoperiod (16 h light/8 h darkness) at an intensity of about 100 μE m^{-2} s^{-1}.

2.2. Cloning and plant transformation

To express a C-terminal fusion of TCP16 (At3g45150) with the EAR repressor domain (Hiratsu et al., 2003), the *TCP16* coding sequence was amplified from Arabidopsis genomic DNA with specific primers (Supplemental Table 1), digested with *Xho*I and ligated with a double-stranded oligonucleotide encoding the EAR domain. The fusion was cloned in a modified pBI121 vector under the control of the *35SCaMV* promoter. Arabidopsis was transformed by floral dip (Clough and Bent, 1998). Seven independent homozygous T3 and T4 lines were used to analyze *TCP16* expression levels and plant phenotype.

2.3. Gene expression analysis

Total RNA was prepared using Trizol (Invitrogen). Northern blots were performed using standard protocols (Ausubel et al., 1987). RT-qPCR was performed on total RNA from rosettes of 25-day-old plants or the apical portion of the inflorescence of 45-day-old plants. cDNA synthesis was performed using an oligo dT primer and MMLV reverse transcriptase (Promega). qPCR was performed using SYBR Green detection and specific primers (Supplemental Table 1) in an MJ Research Chromo4 or a Stratagene MX3000 apparatus. *ACT2* and *ACT8* actin genes (Charrier et al., 2002) or *PP2AA3* (Czechowski et al., 2005) were used for normalization. Relative transcript levels were calculated based on the ΔΔCt method in three biological replicates. miR164 levels were determined by stem-loop RT-qPCR, as described (Chen et al., 2005). Histochemical β-Glucuronidase (GUS) staining was performed with 5-bromo-4-chloro-3-indolyl-ß-D-glucuronic acid (X-gluc) as described by Hull and Devic (1995).

2.4. Scanning electron microscopy (SEM) and histological analysis

Seedlings were fixed in formalin-acetyl alcohol-acetic anhydride-water (10:50:5:35 v/v) and dehydrated with ethanol. For SEM, samples were transferred to acetone, dried using CO_2 in a critical point drier and coated with gold-palladium. Images were taken in a Leitz AMR 1000 (Cambridge, England) scanning electron microscope located at the Centro Científico Tecnológico CONICET Rosario (Argentina). For histological analysis, samples were cleared, embedded in paraffin, sectioned with a microtome and stained with 0.1% toluidine blue. For Nomarski visualization of cell size, leaves were cleared with chloral hydrate (2.5 g/ml in 30% glycerol).

3. Results

3.1. Expression of a repressor form of TCP16 alters plant development

To gain insight into TCP16 action, we expressed in plants a dominant negative form of TCP16 fused to the EAR repressor domain (Hiratsu et al., 2003). This strategy has been successfully applied to study several transcription factors, including members of the TCP family (Aguilar-Martínez and Sinha, 2013; Hervé et al., 2009; Kieffer et al., 2011; Koyama et al., 2007; Uberti-Manassero et al., 2012; Viola et al., 2011). Several lines of plants that express TCP16-EAR from the *35SCaMV* promoter (TCP16-EAR plants) were obtained (Supplemental Fig. 1). Lines 3 and 7, with very low transcript levels, looked similar to wild type and were not further analyzed. TCP16-EAR rosette leaves from the other lines were rounder than wild type, which showed an elliptical shape (Fig. 1A,B). Estimation of circularity using the program LAMINA (Bylesjö et al., 2008) in lines with higher expression (lines 4 and 6) resulted in significantly different ($P < 0.001$) values of 89.7 ± 2.1% for TCP16-EAR plants and of 68.5 ± 2.4% for wild-type plants. Circularity values for the other lines under study were lower (81.8 ± 4.5%, $P < 0.001$), but still significantly different from wild type. Total leaf area and mesophyll cell size (Fig. 1C,D) were not significantly different from wild type. This suggests that TCP16-EAR differentially affects longitudinal and lateral blade expansion. TCP16-EAR plants also showed a compact inflorescence, with shorter internodes and pedicels (Fig. 1E,F). At earlier stages of development, flowers contained shorter and rounder sepals and, in extreme cases, organs from the three external whorls were shorter, giving rise to flowers with exposed carpels (Fig. 1F). Upon flower maturation, the outer whorls elongated and carpels were no longer exposed (Fig. 1G). The plants produced fertile pollen and microscopic analysis did not reveal any difference with pollen from wild-type plants. Plants that express native TCP16 from the *35SCaMV* promoter to similar levels than those of lines 1, 2 and 5 showed no phenotypic differences with wild type.

3.2. TCP16-EAR induces the formation of ectopic meristems in cotyledons

The cotyledons of TCP16-EAR plants, like the leaves, were rounder and had shorter petioles compared to those of wild-type plants (Fig. 2A,B). In lines with relatively high expression levels (lines 4 and 6), lobes were observed near the base (Fig. 2B, arrowheads). In addition, about 60–65% of plants from these lines showed leaf-like structures emerging from cotyledon petioles (Fig. 2C, arrowheads). Upon growth, a rosette and flowering stem developed from this site (Fig. 2D, arrowheads), suggesting that these ectopic growths have meristematic activity. Analysis using SEM showed that the emergence of ectopic meristems took place at symmetrical sites located in the distal part of cotyledon petioles (Fig. 2F). Meristems emerged from a cleft that was present at this site in cotyledons from TCP16-EAR plants but not in cotyledons from wild-type plants (Fig. 2E–H). This cleft extended longitudinally along the adaxial side of the petiole and ectopic meristems originated at the distal end of the cleft, near the cotyledon lamina. This probably indicates that the ectopic meristems originated from internal tissues. Indeed, longitudinal sections along the petiole showed the presence of small, probably proliferating cells, arising from or near the vascular tissue of the cotyledon (Fig. 2I,J). In addition, the vascular tissues of the meristem and the petiole were connected. The formation of ectopic meristems, although at lower frequency, was also observed in two lines with lower expression levels (8% and 16% in lines 1 and 2, respectively), but not in line 5.

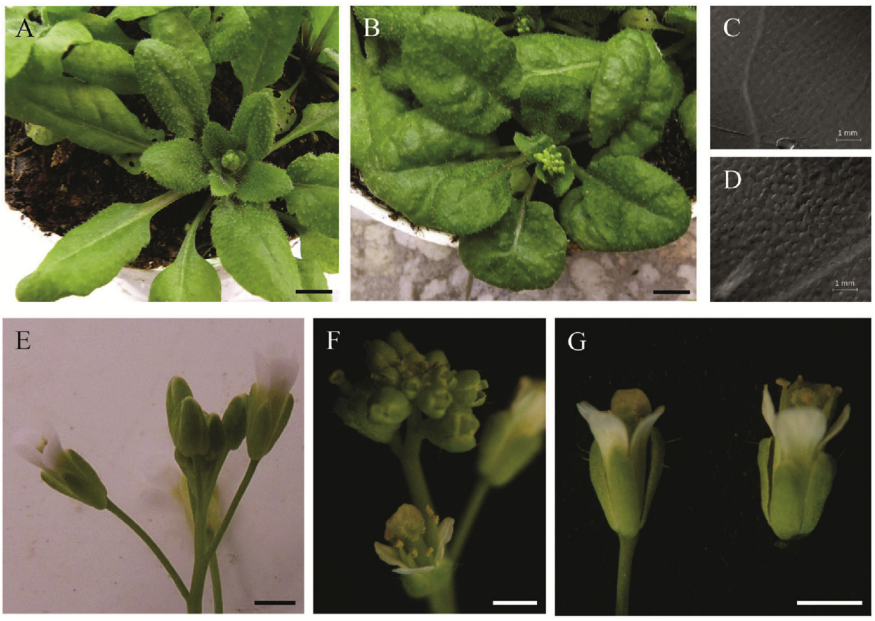

Fig. 1. Phenotype of TCP16-EAR plants. (A,B) Rosettes of 5-week-old wild-type (A) and TCP16-EAR (line 4) plants (B). (C,D) Nomarski images of fixed, fully expanded wild-type (C) and TCP16-EAR leaves (D) showing parenchymatic cell size on the adaxial face. Leaves were cleared with chloral hydrate. (E,F) Inflorescence of wild-type (E) and TCP16-EAR plants (F). (G) Open flowers of wild-type (left) and TCP16-EAR plants (right).

3.3. *TCP16-EAR induces the ectopic expression of meristem genes*

Arabidopsis apical meristems express a set of genes involved in the maintenance of the undifferentiated state of cells (Barton, 2010). These genes are not normally expressed in differentiated cotyledon or leaf tissues. To analyze if the formation of ectopic meristems involves the reactivation of meristem-specific genes, we crossed TCP16-EAR line 4 with GUS reporter constructs for *SHOOT*

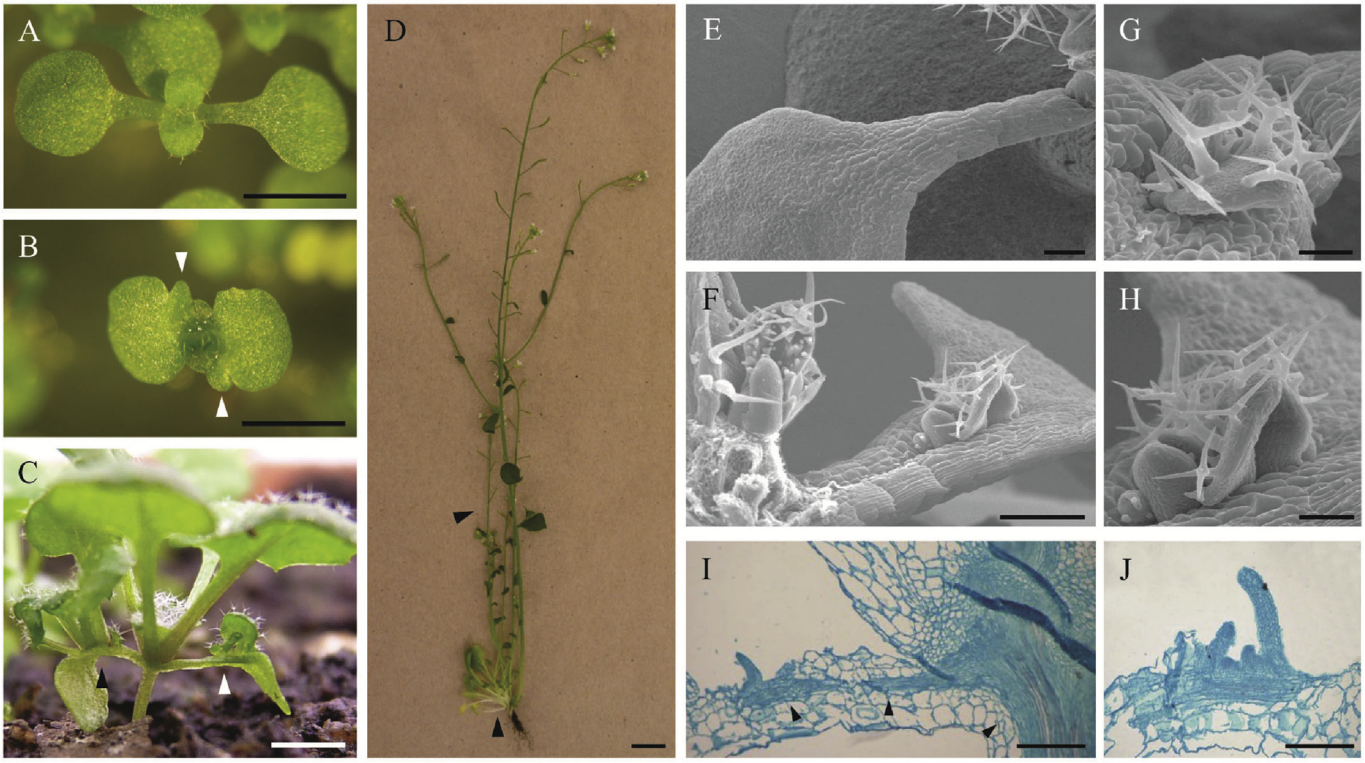

Fig. 2. TCP16-EAR plants develop ectopic meristems in cotyledons. (A,B) Cotyledons of wild-type (A) and TCP16-EAR (line 4) seedlings (B). Arrowheads in (B) point to lobes present in TCP16-EAR cotyledons. (C) TCP16-EAR plants showing the development of ectopic shoots from cotyledons (arrowhead). (D) TCP16-EAR plant at the reproductive stage, showing a flowering ectopic shoot (arrowheads). (E) SEM image of a wild-type cotyledon. (G–H) SEM images of TCP16-EAR cotyledons showing the development of ectopic leaves. (I,J) Longitudinal sections of TCP16-EAR cotyledons showing the development of ectopic meristems.

MERISTEMLESS (*STM*), *BREVIPEDICELLUS/HOMEOBOX PROTEIN KNOTTED-1-LIKE 1* (*KNAT1*) and *CLAVATA3* (*CLV3*). As shown in Fig. 3, these genes are specifically expressed in the region of the apical meristem in wild-type plants. In TCP16-EAR plants, however, expression was also observed in a group of cells located near the distal end of cotyledon petioles (Fig. 3, arrowheads), where ectopic meristems develop. Specifically, expression was associated with the point of bifurcation of cotyledon veins, suggesting that this is the site of emergence of ectopic meristematic tissue. The results indicate that TCP16-EAR is able to trigger the dedifferentiation of a specific group of cells in the cotyledon and the activation of meristem-specific genes at this point.

A survey of the literature indicated that plants that overexpress the boundary-specific gene *CUP-SHAPED COTYLEDON1* (*CUC1*) also develop ectopic meristems in cotyledon petioles at a similar location of those found in TCP16-EAR plants (Hibara et al., 2003; Takada et al., 2001). We then measured the expression of *CUC1* and the related genes *CUC2* and *CUC3* in TCP16-EAR plants. As shown in Fig. 4, increased transcript levels of the three genes were observed in rosettes of TCP16-EAR plants, while *CUC1* and *CUC3* also displayed higher transcript levels in flowers. This suggests that the effect of TCP16-EAR on ectopic meristem formation may be related to the activation of *CUC* genes in these plants. In agreement with this, plants that overexpress *CUC1* also display lobed cotyledons (Hibara et al., 2003; Takada et al., 2001), like TCP16-EAR plants. We also measured the levels of miR164, which regulates the expression of *CUC1* and *CUC2*, and found no differences between wild-type and TCP16-EAR plants (Fig. 4), suggesting that TCP16-EAR does not modulate the expression of *CUC* genes through this miRNA. Transcript levels of *STM* and *KNAT1* were also similar to wild-type in rosettes of TCP16-EAR plants, in agreement with the fact that the activation of these genes occurs locally at the site of ectopic meristem emergence, as indicated by reporter gene expression assays.

4. Discussion

Arabidopsis contains 24 genes that encode TCP transcription factors (Martín-Trillo and Cubas, 2010; Uberti-Manassero et al., 2013). Analysis of single mutants in many of these genes led to the conclusion that there is a high degree of redundancy among them, since the mutants frequently show either no or extremely mild phenotypic alterations (Aguilar-Martínez and Sinha, 2013; Kieffer et al., 2011; Schommer et al., 2008). This led to the use of dominant approaches, like the expression of fusions to repressor or activation domains. Particularly, fusions to the EAR repressor domain (Hiratsu et al., 2003) were used to study the function of several class I and class II TCP proteins (Aguilar-Martínez and Sinha, 2013; Hervé et al., 2009; Kieffer et al., 2011; Koyama et al., 2007; Uberti-Manassero et al., 2012; Viola et al., 2011). Expression of repressor forms of class I TCP proteins is usually associated with changes in leaf development that lead to upward curling of the lamina (Aguilar-Martínez and Sinha, 2013; Kieffer et al., 2011;

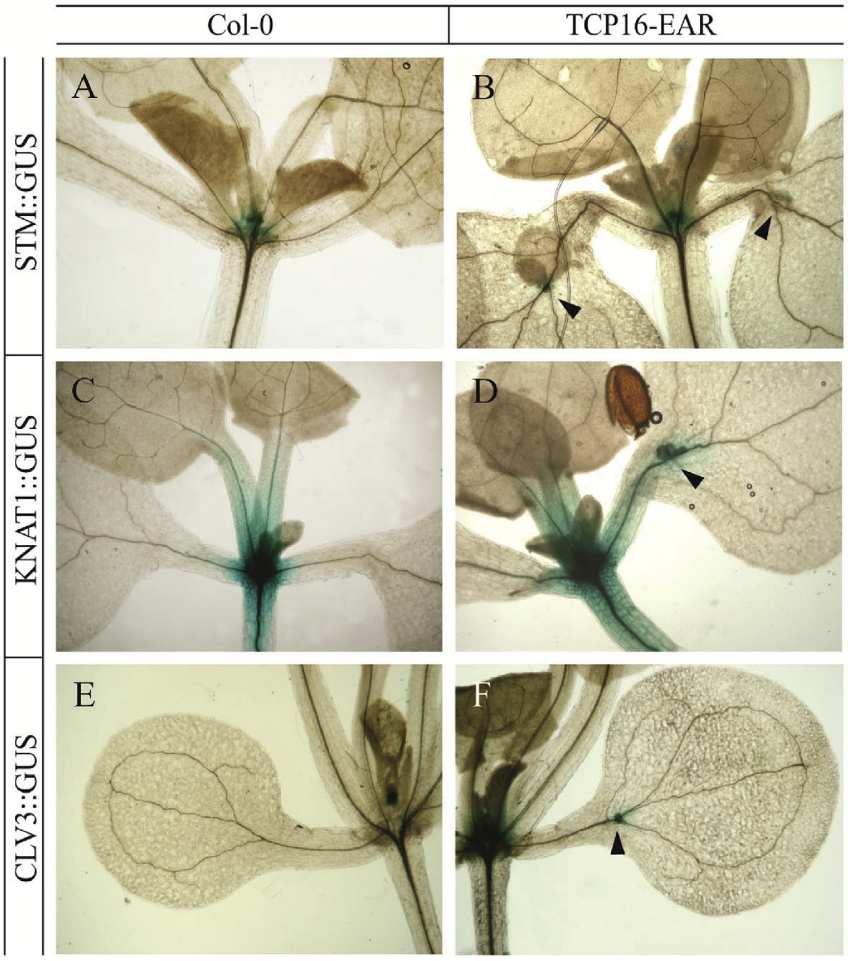

Fig. 3. Ectopic expression of meristem genes in TCP16-EAR plants. GUS reporter constructs were introduced by crossing into TCP16-EAR plants and the expression was analyzed by histochemical staining. (A,C,E) Col-0 control plants. (B,D,F) TCP16-EAR plants. Arrowheads show GUS expression in cotyledons.

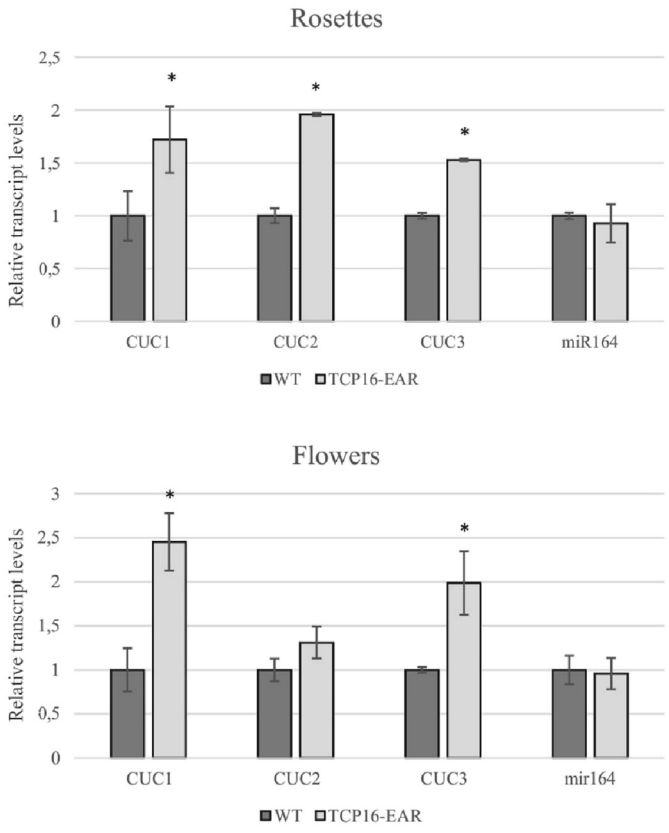

Fig. 4. Transcript levels of *CUC* genes in TCP16-EAR plants. Expression of *CUC* genes was analyzed by RT-qPCR in total RNA prepared from rosettes and flowers of wild-type and TCP16-EAR plants. Levels of *miR164*, which targets *CUC1* and *CUC2*, were also analyzed. Values are referred to levels in wild-type plants. Error bars indicate SD of three biological replicates. Asterisks indicate significant differences with wild type ($P < 0.05$).

Uberti-Manassero et al., 2012). In addition, more compact inflorescences with shorter pedicels and flowers with shorter sepals and petals were observed. The inflorescence of plants expressing TCP16-EAR, as reported here, is similar to the one produced after expression of repressor forms of other class I proteins. The phenotype observed in leaves, however, seems to be different, suggesting that TCP16 is acting on a different set of genes. This may be partly related to the different DNA binding specificity of TCP16. According to this, it has been shown that expressing TCP16-EAR forms with Asp11 or Gly11 affects leaf shape in different ways (Viola et al., 2012).

Another effect of expressing TCP16-EAR is the development of ectopic meristems in cotyledons. These meristems are fully functional since they produce an entire aerial part which occasionally produces flowers and siliques. The formation of ectopic meristems was reported for the ectopic overexpression of meristem genes like *STM* and *KNAT1*, the boundary-specific gene *CUC1*, or mutants in repressors of meristem genes (Brand et al., 2002; Byrne et al., 2000; Chuck et al., 1996; Hibara et al., 2003; Takada et al., 2001; Xu and Shen, 2008). In addition, double loss-of-function mutants in the *YABBY* genes *FILAMENTOUS FLOWER* and *YABBY3*, which specify abaxial leaf fate, and gain-of-function mutants in *PHABULOSA*, which specifies adaxial leaf fate, also develop ectopic meristems (Grigg et al., 2005; Kumaran et al., 2002; McConnell and Barton, 1998). The development of ectopic meristems in cotyledons was also observed in plants that express repressor fusions of the class II TCP protein TCP3 or related CINCINNATA-like class II proteins (Koyama et al., 2007), but was not reported for class I proteins. In the case of TCP3, the development of ectopic meristems was attributed to the increased expression of *CUC* genes. This also applies to TCP16, since *CUC* genes are induced in TCP16-EAR plants. A relevant difference is, however, that TCP3-EAR plants show aberrant cotyledons and leaves, while more moderate changes are observed with TCP16. Related to this, the expression domain of meristem genes is considerably expanded in TCP3-EAR plants but not in TCP16-EAR plants, where these genes are only expressed in the region where ectopic meristems arise. The lack of a generalized induction of meristem genes in the case of TCP16-EAR, as observed with TCP3-EAR, may indicate that, in addition to *CUC* genes, other genes which are differentially expressed in TCP16-EAR and TCP3-EAR plants are also responsible for the induction of meristem genes. In this sense, expression of a repressor form of the class I protein TCP15 was also shown to induce the expression of *CUC* genes, but formation of ectopic meristems was not observed (Uberti-Manassero et al., 2012). The fact that different TCP proteins produce partially overlapping but different phenotypic and gene expression changes when fused to a dominant repressor domain indicates the existence of specificity determinants in the structure of these proteins. Subtle changes in DNA binding preferences are most likely among the factors that contribute to different functional specificities. The results presented here suggest that TCP16 has the capacity to modulate the expression of meristematic gene programs and the differentiation state of plant cells.

Author contributions

NGUM designed, performed and analyzed experiments. ERC performed and analyzed experiments. DHG designed and analyzed experiments and prepared the manuscript.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.plaphy.2016.06.031.

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