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Authors
Wang, Honggui
Zhang, Zenglin
Li, Hongyu
et al.

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CONSTANS-LIKE 7 regulates branching and shade avoidance response in *Arabidopsis*

Honggui Wang1,3,*, Zenglin Zhang2,* Hongyu Li2, Xiaoying Zhao1, Xuanming Liu1,†, Michael Ortiz3, Chentao Lin3 and Bin Liu2,†

1 College of Life Sciences, Hunan University, Changsha 410082, China
2 Institute of Crop Science, Chinese Academy of Agricultural Sciences, Beijing 100081, China
3 Department of Molecular, Cell and Developmental Biology, University of California, Los Angeles, CA 90095, USA

* These authors contributed equally to this work.
† To whom correspondence should be addressed. E-mail: liu-bin2011@caas.net.cn or xml05@126.com

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Abstract

Branching is an important trait of plant development regulated by environmental signals. Phytochromes in *Arabidopsis* mediate branching in response to the changes in the red light:far-red light ratio (R:FR), the mechanisms of which are still elusive. Here it is shown that overexpression of CONSTANS-LIKE 7 (*COL7*) results in an abundant branching phenotype which could be efficiently suppressed by shade or a simulated shade environment (low R:FR). Moreover, *col7* mutants develop shorter hypocotyls and *COL7* overexpression lines develop longer hypocotyls in comparison with the wild type in low R:FR, indicating that *COL7* acts as an enhancer of the shade avoidance response. In shade or transient low R:FR, transcriptional and post-transcriptional expression levels of *COL7* are up-regulated and positively associated with rapid mRNA accumulation of PHYTOchrome INTERACTING FACTOR 3-LIKE 1 (*PIL1*), a marker gene of shade avoidance syndrome (SAS). Taken together, the results suggest a dual role for *COL7* which promotes branching in high R:FR conditions but enhances SAS in low R:FR conditions.

Key words: Branching, COL7, light signal transduction, phytochrome B, PIL1, shade avoidance response.

Introduction

Branching is a significant developmental trait in agricultural and horticultural crops that determines the above-ground architecture of plants (Evers et al., 2011). As a lateral organ, a branch is developed from an axillary meristem. Several genes involved in axillary meristem initiation in *Arabidopsis* have been identified, such as LATERAL SUPPRESSOR, REVOLUTA, and BLIND (Schumacher et al., 1999; Otsuga et al., 2001; Schmitz et al., 2002). While their loss of functions dramatically impedes the formation of axillary meristems, little evidence exists on how environmental signals regulate branching via the initiation of axillary buds (Finlayson et al., 2010). Axillary branching is mainly regulated by breaking of bud dormancy and subsequent branch stem elongation, the processes of which are regulated by internal factors in responding to environmental cues such as light quality and intensity, nutrition, pruning, etc. (Leyser, 2009; Domagalska and Leyser, 2011).

As intrinsic factors, phytohormones play important roles in systemic control of branching. Auxin produced at the shoot apex is transported basipetally to inhibit shoot branching and establish apical dominance (Leyser, 2005). Strigolactone synthesized in the roots is transported acropetally, and also suppresses bud activity (Domagalska and Leyser, 2011). Cytokinins are mostly synthesized in the roots, and act within the bud to promote branch outgrowth (Chen et al., 1985; Nordstrom et al., 2004; Tanaka et al., 2006). Dozens of...
genes, including MORE AXILLARY GROWTH1 (MAX1), MAX2, MAX3, MAX4, AUXIN RESISTANT 1 (AXR1), BRANCHED1 (BRC1), and BRC2, are involved in the biogenesis, transport, or signal transduction of these phytohormones, and mutations of those genes lead to various abnormal branching phenotypes (Stirnberg et al., 2002; Sorefan et al., 2003; Booker et al., 2004, 2005; Aguilar-Martinez et al., 2007; Brewer et al., 2009; Domagalska and Leyser, 2011).

The shade avoidance syndrome (SAS) is characterized by adjustments in plant development in response to a low red light:far-red light ratio (R:FR) perceived by the plant. SAS in Arabidopsis triggers elongation of hypocotyls, stems, and petioles, elevation of leaf angles, suppression of branching, and promotion of flowering (Devin et al., 2003; Casal, 2012). In dense growing conditions, the R:FR decreases as red light is absorbed by photoactive pigments of neighbouring plants, and far-red light is mainly reflected by and transmitted through the neighbouring plants (Ballare, 1999; Franklin, 2008; Hornitschek et al., 2009). In Arabidopsis, red light and far-red light signals are detected by phytochromes (phyA–phyE), which act as major sensors of light quality (Franklin and Quail, 2010).

Among the five phytochromes, phyB plays a predominant role in SAS, and the phyB mutant displays constitutive SAS-like phenotypes including early flowering, hypocotyl elongation, and reduced branching (Reed et al., 1993). Furthermore, phyB exists in two photo-interconvertible forms: an inactive Pr form and an active Pfr form. Red light triggers conversion of phyB from the Pr to the Pfr form, while far-red light photoconverts phyB from Pr to Pr. The Pfr form of phyB is able to interact physically with a subset of basic helix–loop–helix (bHLH) transcriptional factors, named PHYTOCHROME INTERACTING FACTORS (PIFs), which act as positive regulators to promote hypocotyl elongation (Hornitschek et al., 2009; Franklin and Quail, 2010; Li et al., 2012). In high R:FR, most phyB is in the Pfr form which interacts with PIFs and promotes their degradation through the 26S proteasome (Leivar and Quail, 2011). During low R:FR, phyB is converted into the Pr form and is disassociated from PIFs, leading to the accumulation of PIF proteins. Among the seven PIFs characterized in Arabidopsis, PIF4, PIF5, and PIF7 have been implicated in the regulation of SAS (Lorrain et al., 2008; Leivar and Quail, 2011; Li et al., 2012). In contrast to other unstable, light-sensitive PIF proteins, PIF7 shows no rapid light-induced degradation (Leivar et al., 2008; Li et al., 2012). Furthermore, PIF7 is a major positive regulator of SAS that undergoes dephosphorylation and directly binds to G-boxes of auxinsynthesis genes to promote auxin biosynthesis and consequently enhances hypocotyl elongation in response to shade or a low R:FR (Li et al., 2012).

Additionally, phytochromes have been proposed to mediate branching by altering strigolactone signalling and polar auxin transport based on the observation that the inhibition of branching by phyB in response to low R:FR requires functional BRC1, BRC2, AXR1, MAX2, and MAX4 (Finlayson et al., 2010). These findings suggest a primary SAS signal transduction cascade from the perception of light quality to phytohormone biosynthesis, and finally to adjustments in growth and development. However, the network between those plant hormones and phytochrome-mediated control of branching remains elusive. In this study, it is shown that overexpression of COL7 results in an abundant branching phenotype which can be efficiently suppressed by shade. It is demonstrated that mutation of COL7 has suppressed, while overexpression of COL7 has enhanced shade avoidance responses. This study suggests that COL7 plays a positive role in branching and SAS signal transduction, and thus provides additional information about the SAS regulatory network.

Materials and methods

Plant material and growth conditions

The ecotype Col-4 of Arabidopsis thaliana was used as the wild type (WT) in this study. The col7 (GABI-639C04) mutant was ordered from NASC. The 35S::COL7 and 35S::MYC-COL7 transgenic lines are in the Col-4 background. Quantification of branch phenotype analysis was performed using a modified method (Finlayson et al., 2010). Seeds were sown on soil, stratified at 4 °C in darkness for 3 d, and then transferred to long days (LDs) (16 h light/8 h dark, 100–150 µE m⁻² s⁻¹ of white light, R:FR ratio of 1.2) for 2 weeks. The plants were then left in white light or transferred to simulated shade (R:FR ratio of 0.1–0.3) provided by a combination of white light and far-red light (LED panel, 730±30 nm). The number of primary rosette branches produced from the rosette buds was recorded when the first siliques of Arabidopsis turned yellow. Hypocotyl lengths in continuous red light or far-red light were measured as described previously (Yu et al., 2007). Briefly, the seeds were surface sterilized with 10% bleach for 10 min, stratified at 4 °C in darkness for 3 d, treated with white light for 12 h, and grown in red light or far-red light (red light, 19 µE m⁻² s⁻¹; far-red light 0.47 µE m⁻² s⁻¹) for 7 d. The hypocotyl length of at least 20 seedlings was measured. Hypocotyl elongation assays in response to shade were performed as previously described (Li et al., 2012). The seeds were incubated in continuous white light (30 µE m⁻² s⁻¹; R:FR ratio of 1.2) for 3 d, then either kept in white light or moved to simulated shade (LED: red light, 12 µE m⁻² s⁻¹; blue light, 0.5 µE m⁻² s⁻¹; and far-red light, 20 µE m⁻² s⁻¹; R:FR ratio of 0.6) for 5 d. The hypocotyl length of 220 seedlings was measured.

Vector construction and plant transformation

The open reading frame (ORF) of COL7 (AT1G73870) was amplified by reverse transcription–PCR (RT–PCR) using primers COL7-CDS-F and COL7-CD-S-R (primer sequences are provided in Supplementary Table S1 available at JXB online) and cloned into pDONR201 by BP reaction to generate pDONR201-COL7 (Gateway, Invitrogen). Then the COL7 coding sequence (CDS) was cloned into the binary vector pLeela (Liu et al., 2007) and 35S::MYC-GW by LR reaction (Gateway, Invitrogen) to generate 35S::COL7 and 35S::MYC-COL7, respectively. Agrobacterium tumefaciens strains GV3101(pMP90RK) and GV3101(pMP90) were used for Arabidopsis transformation with 35S::COL7 and 35S::MYC-COL7, respectively, following the floral dip method (Clough and Bent, 1998).

PCR genotyping, RNA isolation, and mRNA expression analysis

Genomic DNA of the WT or col7 mutant was used as template for PCR genotyping using prim-er P1, P2, and P3. Total RNA extraction and cDNA synthesis were performed as previously described (Yu et al., 2008). COL7 mRNA abundance in the WT and col7 mutant was evaluated by semi-quantitative RT–PCR using primer pairs P1 and P2 for COL7 and P4 and P5 for ACTIN2. mRNA
levels of the indicated genes were measured by quantitative PCR (qPCR) using P6 and P7 for COL7, P8 and P9 for PIL1, and P10 and P11 for ACTIN2. Sequences of the above primers are provided in Supplementary Table S1 at JXB online.

Immunoblot

Seeds were sterilized, sown on Murashige and Skog (MS) medium, grown, and moved into different treatment conditions. Samples were harvested, frozen in liquid nitrogen, and ground in 4× SDS protein extraction buffer for subsequent SDS–PAGE and immunoblot analysis probed with anti-MYC antibody (Millipore, Cat. #05-724). The same membrane was stripped and probed with anti-CRY1 antibody as internal control.

Results

Overexpression of COL7 in Col-4 results in an abundant branching phenotype

There are a total of 17 members of the CO-LIKE gene family which can be grouped into three phylogenetic clades (Khanna et al., 2009). The functions of some CO-LIKE genes belonging to clade I and II have been characterized, but the biological roles of the clade III CO-LIKE genes are still largely unknown (Hassidim et al., 2009). Here it was observed that overexpression of COL7, one of the clade III CO-LIKE genes, driven by the 35S promoter in WT Col-4 Arabidopsis, resulted in an obviously abnormal branching phenotype (Fig. 1). Multiple transgenic lines were obtained, and two representative lines, 35S::COL7 #10 and 35S::COL7 #11, in which the overexpression of COL7 was verified by qPCR (Supplementary Fig. S1 at JXB online), were used for the subsequent studies. The phenotype of 35S::COL7 lines was compared with that of WT plants and the col7 mutant in which the full-length mRNA expression of COL7 was impaired by a T-DNA insertion at the second exon (Supplementary Fig. S1A–C). As shown in Fig. 1A, when the indicated plants were grown at a low density (one plant per pot) in LDs, 35S::COL7 lines generated more rosette branches than the WT and the col7 mutant. To test if higher planting density can inhibit the abundant branching phenotype, the numbers of plants was increased and treatments of one, four, nine, or 20 seedling per pot in LDs were used for branch quantification. At low planting density of one plant per pot, the branch number of 35S::COL7 lines is ~5-fold greater than that of the WT and the col7 mutant (Fig. 1B). However, the branch number of the overexpression lines declines dramatically with increased planting density and is almost as low as that of the WT and the col7 mutant when the plants were grown at the highest density of 20 plants per pot. Since branching could be influenced by other factors such as nutrition, beside SAS, the branch numbers of each line grown at a low density of one plant per pot in low R:FR were compared. As show in Fig. 1C and D, sustained low R:FR efficiently suppressed the abundant branching phenotype of 35S::COL7 lines, indicating that the decline of R:FR at high planting density could result in the reduction of branch numbers of 35S::COL7 lines. In addition, when comparing the plants grown in high R:FR with those grown in low R:FR, the branch number of the 35S::COL7 lines decreases >4-fold, while that of the WT and col7 decreased <2-fold, suggesting that COL7 enhances the shade-induced suppression of branching.

COL7 promotes hypocotyl elongation in response to shade

During SAS, the hypocotyls of Arabidopsis tend to elongate to reach a higher position in response to low R:FR (Casal, 2012). To test the role of COL7 in the regulation of hypocotyl elongations, the hypocotyls of the 35S::COL7 line, the col7 mutant, and the WT were analysed in red light, far-red light, high R:FR, and low R:FR, respectively. All the lines showed no obvious difference when grown in continuous red light for 3–6 d (Supplementary Fig. 2A, C at JXB online). When grown in continuous far-red light, 35S::COL7 lines developed longer hypocotyls and col7 mutants developed shorter hypocotyls in comparison with the WT (Supplementary Fig. S2B, D), indicating that COL7 may suppress the far-red-light-dependent inhibition of hypocotyl elongation. Furthermore, quantification of hypocotyl elongation in response to shade treatment shows that overexpression of COL7 significantly promotes hypocotyl elongation, while loss of function of

Fig. 1. Branching phenotypes of WT, col7 mutant, and 35S::COL7 lines. (A) Branching phenotype of the WT, col7 mutant, and COL7 overexpression lines growing at the density of one plant per pot in LDs (16 h light/8 h dark). (B) Rosette branch number of each line as indicated grown at the density of one, four, nine, or 20 plants per pot, respectively. (C) Rosette branch phenotype of each line treated by low R:FR. Plants were grown in LDs for 3 weeks and then transferred to low R:FR. (D) Rosette branch number of each line grown in high R:FR or low R:FR. The number of the primary branches generated from the rosette axillary buds was measured. Means and standard deviations are representative of at least 20 plants. (This figure is available in colour at JXB online.)
**COL7** in the *col7* mutant suppresses hypocotyl elongation when grown in low R:FR but not in high R:FR (Fig. 2A, B). These observations demonstrate that **COL7** enhances hypocotyl elongation in response to sustained shade, a typical phenotype of SAS.

**Expression of COL7 is dynamically regulated at both the transcriptional and post-transcriptional level by shade**

To test if the transcription of **COL7** is regulated by low F:FR, an mRNA expression analysis was performed via qPCR using the seedlings transferred from simulated shade to white light (Fig. 3A) or the seedlings transferred from white light to simulated shade (Fig. 3B). The results indicate that high R:FR results in a rapid decline of **COL7** mRNA within 15 min (Fig. 3A). In contrast, mRNA expression of **COL7** was rapidly up-regulated in 15 min and then fell back to its original level within 2 h after the seedlings were transferred from high R:FR to low R:FR (Fig. 3B). To test further the stability of **COL7** proteins in different light conditions, the **35S::MYC-COL7** binary vector was constructed to transform *Arabidopsis*, and the **35S::MYC-COL7** transgenic line which showed a similar phenotype to **35S::COL7** transgenic lines was obtained (Supplementary Figs S1, S3 at JXB online). Seedlings of the **35S::MYC-COL7** line were grown on MS plates for 5 d in white light, transferred to dark for 3 d, and then treated by far-red light or red light. Immunoblots probed with anti-MYC antibody showed that **COL7** protein accumulated to a high level within 1–2 h but then declined gradually when the seedlings were transferred from dark to red light or far-red light (Fig. 3C, D, G, H), indicating that both red light and far-red light could dynamically increase the stability of the **COL7** protein. To explore the role of **COL7** in SAS further, the fluctuation of **COL7** protein in response to low R:FR was investigated. Seedlings grown in low R:FR were transferred into high R:FR (Fig. 3E, I) or vice versa (Fig. 3F, J). The results demonstrate that high R:FR destroys **COL7** protein but low R:FR increases its stability. These data taken together reveal that the expression of **COL7** is dynamically up-regulated by shade at both the transcription and post-transcriptional level, suggesting that the regulation of **COL7** expression is a part of the SAS.

**COL7 promotes the expression of PIL1 mRNA in response to shade**

**PIL1** is a bHLH protein associated with the SAS (Salter et al., 2003). Because its mRNA level is rapidly up-regulated in response to low R:FR treatment, **PIL1** is widely used as a marker gene of SAS. To investigate if **COL7** is involved in the rapid regulation of **PIL1** expression by low R:FR, seeds of WT, *col7* mutant, and **35S::COL7** lines were sown on MS plates, grown in 24 h diurnal cycles (12 h light/12 h dark) for 5 d, and treated by low R:FR (Fig. 4A) from Zeitgeber time 1 (1 h after dawn) for the period indicated (0, 5, 10, 30, and 60 min). The results indicate that **PIL1** mRNA abundance increases ~40-fold in **35S::MYC-COL7** lines but increases only 25-fold in the WT and 17-fold in the *col7* mutant in response to low R:FR treatment for 1 h, indicating that the abundance of **COL7** is positively correlated with the rapid increase in **PIL1** mRNA induced by low R:FR. To evaluate if **COL7** sustainably affects the expression of **PIL1**, **PIL1** mRNA abundance was further analysed in the WT, *col7* mutant, and **35S::COL7** lines grown in sustained high R:FR, low R:FR, or continuous far-red light. **PIL1** mRNA showed a minor increase in the *col7* mutant and a decrease in the **35S::COL7** lines in comparison with the WT grown in high R:FR (Fig. 4B). In contrast, **PIL1** mRNA showed a slight decrease in the *col7* mutant and an increase in the **35S::COL7** lines in comparison with the WT in low R:FR and continuous far-red light (Fig. 4C, D). These results suggest that **COL7** increases the **PIL1** mRNA abundance in continuous low R:FR or far-red light but decreases its abundance in continuous high R:FR.

**Discussion**

**COL7** is a CO-LIKE protein, which belongs to a putative transcriptional factor family containing 17 gene members sharing two conserved domains, the B-box domain and CCT (CO, COL, and TOC1) domain. Among them, CO is the first B-box protein identified in *Arabidopsis* which plays a pivotal role in regulation of photoperiod flowering in *Arabidopsis*, and its expression is regulated by light at both the transcriptional and post-transcriptional level (Suarez-Lopez et al., 2001; Laubinger et al., 2006; Jang et al., 2008; Turk et al., 2008; Zuo et al., 2011). The presence of multiple **COL** genes in the genome of *Arabidopsis* suggests that they may share redundant functions in regulation of photoperiod flowering. Until now only **COL5** was shown to induce flowering as CO does in SDs. Additionally, **col5** mutants were shown to flower...
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normally, in contrast to co mutants that flower extremely late in LDs (Hassidim et al., 2009). COL9 was shown to have the completely opposite effects to CO, where COL9 overexpression lines were late flowering while co-suppression lines and col9-t mutant lines were early flowering in LDs (Cheng and Wang, 2005). Also col3 mutants were also shown to flower early in both LDs and SDs (Datta et al., 2006). These unexpected observations imply that the COL genes have evolved wider roles in addition to regulation of flowering. It has been established that overexpression of COL1 affects circadian rhythms (Ledger et al., 2001) and COL3 regulates the formation of lateral roots, daylength, and light-dependent elongation and branching of shoots (Datta et al., 2006). However, the functions of other COL genes remain poorly understood.

In this study, the role of COL7 in branching, hypocotyl elongation, and marker gene expression in SAS was investigated. The results implicate a dual role for COL7 which acts oppositely in SAS signal transduction depending on the changes of R:FR. In high R:FR, COL7 promotes branching and suppresses hypocotyl elongation. In low R:FR, COL7 suppresses branching and enhances hypocotyl elongation and SAS marker gene expression. Unlike some of the other SAS...
regulators, COL7 positively expands the capacity for shade avoidance. For instance, an elevated level of phyB or PIFs in engineered overexpression lines results in a shorter or longer hypocotyl phenotype, respectively, but both overexpressing phyB and PIFs attenuate the extent of shade-induced hypocotyl elongation (Roig-Villanova et al., 2006; Lorrain et al., 2008; Leivar and Quail, 2011). In contrast, hypocotyl growth of 35S::COL7 lines was suppressed by high R:FR but promoted by low R:FR (Fig. 2), demonstrating an increased capacity for shade-induced hypocotyl elongation. Overexpression of COL7 also promotes branching in high R:FR and increases the sensitivity of SAS to suppress branching in low R:FR. Such an elastic branching trait of COL7 overexpression lines in response to varying light qualities may be utilized to optimize the agricultural architecture and planting density of crops to improve production in the field.

How COL7 dynamically regulates branching in response to competing neighbours and/or light quality is still not known. Plant branching is regulated by genes associated with the biogenesis, transport, and signal transduction of phytohormones such as auxin, strigolactone, and cytokinin. Phytohormone-associated mutants, such as max1, max2, max3, max4, axr1, brc1, and brc2, all showed an abundant branching phenotype similar to that displayed by 35S::COL7 lines (Stirnberg et al., 2002; Sorefan et al., 2003; Booker et al., 2004, 2005; Aguilar-Martinez et al., 2007; Brewer et al., 2009; Domagalska and Leyser, 2011). Low R:FR or disruption of phyB function can efficiently suppress the branch production in WT Arabidopsis plants but has little effect on the abundant branching phenotype of the phytohormone-associated mutants, indicating that SAS requires normal function of those phytohormone-associated genes to inhibit branching (Finlayson et al., 2010; Casal, 2012). Whether the abundant branching phenotype of 35S::COL7 lines could be suppressed by those phytohormone-associated genes in perception of low R:FR has yet to be investigated. It is summarized here that COL7 is involved in a complicated network of branching regulation and shade avoidance signal transduction, the mechanism of which need to be illustrated by further genetic and molecular investigations.

**Supplementary data**

Supplementary data are available at JXB online.

Figure S1. Abundance of COL7 mRNA in the wild type, col7 mutant, and overexpression lines.

Figure S2. COL7 affects the hypocotyl elongation in far-red light.

Figure S3. Hypocotyl phenotype of the 35S::MYC-COL7 line.

Table S1. Sequences of primers used in this study.
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