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Auxin–cytokinin interactions in the control of shoot branching

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Abstract In many plant species, the intact main shoot apex grows predominantly and axillary bud outgrowth is inhibited. This phenomenon is called apical dominance, and has been analyzed for over 70 years. Decapitation of the shoot apex releases the axillary buds from their dormancy and they begin to grow out. Auxin derived from an intact shoot apex suppresses axillary bud outgrowth, whereas cytokinin induced by decapitation of the shoot apex stimulates axillary bud outgrowth. Here we describe the molecular mechanisms of the interactions between auxin and cytokinin in the control of shoot branching.

Keywords Adenosine phosphate-isopentenyltransferase · Apical dominance · Basipetal auxin flow · Cytokinin oxidase · Pea · *PIN1* · Shoot branching

Abbreviations

CK	Cytokinin
CKX	Cytokinin oxidase
GUS	β-glucuronidase
IAA	Indole-3-acetic acid
IPT	Adenosine phosphate-isopentenyltransferase
SAM	Shoot apical meristem
TIBA	2, 3, 5-triiodobenzoic acid

Introduction

Apical dominance is a phenomenon in which a main shoot in an intact plant grows predominantly while suppressing the outgrowth of axillary buds. After loss of the main shoot, the dormant axillary buds immediately begin to grow out as a main shoot to replace the lost apex, thereby allowing the plant to survive (Cline 1991; Shimizu-Sato and Mori 2001). A main shoot derives from the activity of the primary shoot apical meristem (SAM), which arises during embryogenesis (Kerstetter and Hake 1997). Axillary buds also derive from the primary SAM in a developmental process that generally involves two phases. (1) The axillary meristem is formed from groups of meristematic cells, which originate directly from detached parts of the primary SAM of the main shoot. The axillary meristem produces axillary buds located on the axil of the leaf primordia. (2) After axillary buds are fully developed and have reached a certain size depending on the plant species, growth ceases and the axillary bud becomes dormant. In Arabidopsis, pea, petunia, and tomato, some mutants show a phenotype that is characteristic of a specific developmental phase of the axillary buds. For example, the *lateral suppressor* (*ls*) mutant in tomato shows decreased branching, because the *ls* mutation prevents the initiation of axillary meristems during the vegetative phase (Malayer and Guard 1964). The *more axillary growth* (*max*) mutant of Arabidopsis shows enhanced shoot branching (Stirnberg et al. 2002), caused by the release of dormant axillary buds.

Shoot branching has an important role in generating a large variety of diverse plant forms, because the degree of apical dominance varies depending on the plant species. Branching pattern complexity also depends on plant age. Generally, older plants show weaker apical dominance resulting in a bushier form in which the total number of leaves or flowers are increased to be more fruitful. Shoot branching is controlled by environmental cues (such as light strength and nutrition) as well as by temporal and spatial developmental programs (Cline 1996). In some

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species, axillary buds of plants damaged by excessive bending continue to grow in the intact shoot apex, suggesting that gravity is also one of the environmental cues that controls axillary bud outgrowth. Interestingly, gravity-regulated release from apical dominance seems to be distinct and unique from decapitation-regulated release from apical dominance (Kitazawa et al. 2008). When the upper part of the main shoot of the Japanese morning glory (*Pharbitis nil*) is bent down, the axillary buds located on the uppermost node of the bent region begin to grow. Despite this phenomenon, shoot bending does not induce significant changes in either the cytokinin (CK) level or the auxin response, which are typical responses to the release from apical dominance caused by decapitation. The response to gravity, therefore, might be a separate molecular mechanism for survival.

Apical dominance is regulated by two plant hormones, auxin and CK. In this review, we describe the molecular mechanisms underlying the interactions between auxin and CK in the control of shoot branching. Recently, studies of a series of recessive mutants with enhanced shoot branching revealed that a third novel hormone is also involved in inhibiting the outgrowth of axillary buds. These mutants include the pea *ramosus* (*rms*) mutant, the petunia *decreased apical dominance* (*dad*) mutant, the Arabidopsis *more axillary growth* (*max*) mutant, and the rice *dwarf* (*d*) and *high-tillering dwarf* (*htd*) mutants (Beveridge et al. 1994; Napoli 1996; Stirnberg et al. 2002; Ishikawa et al. 2005). Dun et al. and Ongaro and Leyser have provided excellent reviews of other important signals involved in shoot branching (Dun et al. 2006; Ongaro and Leyser 2008).

The basipetal auxin flow in the stem suppresses axillary bud outgrowth

More than 70 years ago, Thimann et al. reported that decapitation of *Vicia* spp. plants induces axillary bud outgrowth, whereas the application of indole-3-acetic acid (IAA) to the stump prevents axillary bud outgrowth (Thimann and Skoog 1934). Axillary bud growth cannot be prevented by the direct application of auxin to the axillary buds after decapitation. Further, radiolabeled auxin applied to the stump is not transported into the axillary buds (Hall and Hillman 1975). These findings indicate that auxin does not directly prevent axillary bud outgrowth. Stump application of 2, 4-dichlorophenoxyacetic acid, which cannot be transported basipetally in plants, also does not prevent the outgrowth of axillary buds (Brown et al. 1979). The auxin-transport inhibitor 2, 3, 5-triiodobenzoic acid (TIBA) in lanolin applied to the stem of an intact plant can, however, reduce or abolish apical dominance (Snyder 1949). These observations indicate that basipetal auxin flow derived from a shoot apex inhibits axillary bud outgrowth.

Auxin, produced mainly in young leaves and in the shoot apex (Ljung et al. 2001), is transported basipetally down the stem in a polar manner by active transport in the vascular parenchyma (Lomax et al. 1995). Several proteins regulate auxin transport, such as the auxin efflux carriers PIN-FORMED (PIN) and p-glycoprotein, and the auxin influx facilitators AUXIN RESISTANT 1 (AUX1)/LIKE-AUX1 (LAX) (Okada et al. 1991; Marchant et al. 1999; Murphy et al. 2002). Although the molecular mechanisms of active auxin transport are becoming clear, it remains unknown how auxin flow derived from the shoot apex induces axillary buds to enter dormancy.

CK promotes axillary bud outgrowth

The effects of CK in apical dominance are antagonistic to those of auxin. Direct application of CK to axillary buds promotes axillary bud outgrowth, even in intact plants (Wickson and Thimann 1958). To date, CK is the only chemical known to release axillary buds from dormancy. After decapitation in chickpea, CK levels in the axillary buds increase 7-fold within 6 h and 25-fold within 24 h, and axillary bud outgrowth is well correlated with the CK level in the axillary buds (Turnbull et al. 1997). Roots are the main CK-producing organ in plants, and CK biosynthesized in the root system is transported into the shoot system via the xylem (Hopkins and Huner 2004). CK levels in bean xylem exudates increase within 16 h after decapitation and gradually return to basal levels (Bangerth 1994). Application of 1-naphthylacetic acid to the stump prevents the increase in CK in the bean xylem exudates (Li et al. 1995). These observations led to the suggestion that CK derived from roots promotes axillary bud outgrowth after decapitation, and auxin derived from a shoot apex regulates CK transport in plants (Letham 1994). It is now known, however, that the CK that promotes axillary bud outgrowth after decapitation is locally biosynthesized in the nodal stem rather than in the roots (Tanaka et al. 2006), as described below.

An adenosine phosphate-isopentenyltransferase (*IPT*) gene is expressed specifically in nodal stems after decapitation

Several molecular approaches have been used to characterize the biochemical events associated with axillary bud dormancy and outgrowth. In pea (*Pisum sativum* L. cv. Alaska) axillary buds, the molecular mechanisms of cell cycle control during the dormancy-to-growth transition

were characterized based on the expression patterns of cell cycle regulators such as *histone H4* (S phase marker), *cyclinB* (G2/M phase marker), *cyclinD* (G1 phase marker), and *proliferating cell nuclear antigen* (G1/S phase marker) (Shimizu and Mori 1998). These analyses demonstrated that for every 2-cm increase in the distance from the site of decapitation to the second node, there is a 2-h delay in the induction of *proliferating cell nuclear antigen* mRNA in the axillary buds (Shimizu-Sato et al. unpublished data). This finding suggests that sustained outgrowth of the axillary bud is regulated by a decrease in the auxin level in the stem around the second node, although the rapid but transient initial growth of the axillary bud is dependent on hydrostatic pressure (McIntyre and Damson 1988) and independent of auxin (Morris et al. 2005). The genes, including *IPT*, expressed in nodal stems after decapitation were identified by a subtraction method using mRNA prepared from nodal stems before or after decapitation (Tanaka et al. 2006). *IPT* encodes a key enzyme in CK biosynthesis (Kakimoto 2001; Takei et al. 2001). Transgenic *Agrobacterium tumefaciens* plants overexpressing *IPT* exhibit elevated levels of CK, and reduced apical dominance (Medford et al. 1989). The petunia mutant *sho*, which was identified by activation tagging, also has increased levels of CK, and reduced apical dominance (Zubko et al. 2002). *sho* encodes an IPT protein. These observations indicate that IPT has an important role in the control of CK levels in plants. *IPT* cDNA has been identified and characterized in various plants such as Arabidopsis, rice, and maize (Kakimoto 2001; Takei et al. 2001). In pea, two full-length *IPT* cDNAs (*PsIPT1* and *PsIPT2*) were isolated from the stem. PsIPT2 in the stem seems to have a main role in controlling axillary bud outgrowth after decapitation, because *PsIPT2* was the only isolated clone identified from a subtracted library prepared from nodal stems after decapitation.

Auxin derived from a shoot apex suppresses local biosynthesis of CK in the nodal stem through the regulation of *PsIPT* expression

Members of the *IPT* family regulate CK levels in Arabidopsis in response to plant developmental processes (Miyawaki et al. 2004; Takei et al. 2004). In pea plants, *PsIPT2* mRNA expressed transiently in the nodal stems increases at 1 h, reaches a maximum level at 3 h, decreases by 9 h, and is undetectable 24 h after decapitation. This expression pattern suggests that the induction of *PsIPT2* mRNA in the nodal stem correlates with the release of dormant axillary buds, and that PsIPT2 is involved in CK biosynthesis to stimulate axillary bud outgrowth. Together, these results and the report that basipetal auxin transport in the pea stem is 1 cm h^{-1} (Johnson and Morris 1989) suggest that auxin derived from the shoot apex represses *PsIPT2* expression in the nodal stem in intact plants, and that the depletion of auxin in the nodal stem immediately induces the expression of *PsIPT2* mRNA after decapitation (Tanaka et al. 2006).

Some members of the Arabidopsis *IPT* family are downregulated by CK, and others are upregulated by auxin (Miyawaki et al. 2004). *PsIPT2*, however, is downregulated by auxin, as demonstrated by the following two experiments. (1) Decrease of *PsIPT2* expression by exogenous IAA in nodal excised stem segments, in which *PsIPT2* mRNA had accumulated. Three hours after decapitation 1 cm above the second node, nodal stem segments in which *PsIPT2* mRNA was increased were excised. The excised nodal segments were incubated in 2-(*N*-morpholine)-ethanesulfonic acid (MES) buffer with or without IAA. Northern blot analyses showed that *PsIPT2* mRNA was maintained at the same level in the IAA-free buffer, whereas it decreased immediately after incubation with IAA buffer. (2) Increase of *PsIPT2* expression by IAA-depletion in nodal excised stem segments, in which *PsIPT2* mRNA had been undetectable. The excised nodal segments from intact seedlings were incubated in MES buffer with or without IAA. Northern blot analyses demonstrated that *PsIPT2* mRNA was induced in the IAA-free buffer, whereas it was not detected in the IAA buffer. In addition, *PsIPT2* responds to the auxin level as well as the basipetal auxin flow in the stem, as indicated by the following two observations. (1) Induction of *PsIPT2* mRNA is inhibited by the application of IAA in lanolin to the stump after decapitation. (2) *PsIPT2* mRNA is induced by the application of TIBA in lanolin to the internode above the second node as well as by removal of the shoot apex. Auxin negatively regulates CK biosynthesis to control some plant developmental stages (Eklöf et al. 1997; Nordström et al. 2004), but there is little information on the molecular mechanisms occurring at the gene level. These above observations in pea plants indicate that auxin directly affects CK biosynthesis by regulating *IPT* expression, which is involved in the first step in CK biosynthesis.

CK is mainly synthesized in the root, which contains high levels of CK. In Arabidopsis, *AtIPT1*, *AtIPT3*, *AtIPT5*, and *AtIPT7* transcripts are detected in the root (Miyawaki et al. 2004; Takei et al. 2004). In intact pea seedlings, the *PsIPT2* expression level is very low in various organs, including root. Therefore, *PsIPT2* may not contribute to CK biosynthesis in the root. *PsIPT*, however, may induce CK biosynthesis in the roots and other tissues to control various aspects of plant development. Members of the *IPT* family each have individual roles in controlling the complicated interactions between auxin and CK, depending on plant development.

Transgenic Arabidopsis seedlings containing the β-glucuronidase (GUS) reporter gene under control of the *PsIPT2* promoter region (2,025 bp) were generated to examine whether the depletion effect of auxin on *PsIPT2* expression in pea is a common molecular mechanism in plants. The *GUS* gene was expressed before treatment with IAA, but *GUS* mRNA was completely depleted by IAA treatment for 5 h. This response of *GUS* expression to auxin in transgenic Arabidopsis seedlings is similar to that of *PsIPT2* expression in the pea stem, suggesting that plants, at least, pea and Arabidopsis, have similar molecular mechanisms in which auxin suppresses *PsIPT2* expression (Tanaka et al. 2006).

CK is locally biosynthesized in the nodal stem

CK is mainly biosynthesized in roots, and xylem exudates contain high levels of CK. Based on the changes in the levels of CK in xylem exudates, it was thought that CK is transported from the root to promote axillary bud outgrowth (Letham 1994). In pea, CK levels in the stem and axillary buds increase 3 h after decapitation despite the fact that *PsIPT2* is not expressed in growing axillary buds; therefore, CK is biosynthesized in the stem and transported into axillary buds to promote their outgrowth after decapitation.

Axillary bud outgrowth occurs in excised nodal stem segments without the root (Tamas et al. 1989). In pea, the amount of CK synthesized in the excised stem segments is the same as that in the stem with an intact root. Application of IAA in lanolin to the stump inhibits the increase in CK in the excised nodal stem segments. Together, these observations indicate that the CK in the stem is biosynthesized in the nodal stem and not transported from the root. The amount of CK locally biosynthesized *de novo* in the nodal stems is sufficient for promoting axillary bud outgrowth.

PsPIN1 expression pattern follows the auxin flow in plants

PIN1 has a very important role in polar auxin transport, because the Arabidopsis loss of *PIN1* mutant has a very severe phenotype. The distribution of PIN-family proteins in Arabidopsis root follows the auxin flow (Blilou et al. 2005). *PsPIN1* was isolated as a gene expressed specifically in the nodal stem of intact pea seedlings in a subtraction experiment. Although Gocal et al. had previously reported IAA levels in axillary buds of *Phaseolus vulgaris* (Gocal et al. 1991), more detailed measurements of IAA levels in axillary buds and nodal stems were made before and after decapitation in pea plants (Tanaka et al. unpublished data). The decrease in the IAA level in the stem after decapitation corresponded to the decrease in *PsPIN1* expression in the stem. When auxin flow is recovered by *de novo* biosynthesis in outgrowing axillary buds, *PsPIN1* expression in the stem also recovers. In axillary buds, the IAA level is low during dormancy, and increases in outgrowing axillary buds. In a similar manner, the *PsPIN1* expression level in dormant axillary buds is low, whereas it increases in outgrowing axillary buds. These findings suggest that the *PsPIN1* expression pattern follows the auxin flow in the stem, both before and after decapitation.

The decrease in CK in the stem is also regulated by auxin through the regulation of cytokinin oxidase (CKX)

Endogenous plant hormone levels are controlled by biosynthesis and metabolism. CKX is the only enzyme that inactivates CK by irreversibly degrading active CK (Jones and Schreiber 1997), and is a key factor in controlling the endogenous levels of active CK (Werner et al. 2001). *CKX* cDNA has been identified and characterized in various plants, such as maize, Arabidopsis, rice, *Hordeum vulgare*, *Dendrobium hybrid*, and *Dendrobium huoshanense* (Houba-Hérin et al. 1999; Morris et al. 1999). Two *CKX* cDNAs (*PsCKX1* and *PsCKX2*) from pea stem were isolated (Tanaka et al. unpublished data). *PsCKX2* may contribute mainly to regulate CK levels in the stem, because *PsCKX2* transcript levels are very high compared with those of *PsCKX1* in the stem. *PsCKX2* transcripts are expressed at a low level in the stem in intact plants. After decapitation, *PsCKX2* mRNA is undetectable from 3 to 9 h after decapitation, and then rapidly increases after 9 h. The application of TIBA in lanolin to the stem causes a similar *PsCKX2* expression pattern. These *PsCKX2* expression patterns in the stem are opposite to the CK level patterns before and after decapitation, suggesting that *PsCKX2* induces a decrease in CK levels in the stem.

In many cases, CKX activity and *CKX* mRNA are promoted by CK, indicating that CK levels are regulated by negative feedback through the regulation of CKX (Terrine and Laloue 1980; Brugière et al. 2003). On the other hand, auxin might induce some CKXs to regulate CK levels in various developmental processes in plants, because auxin induces the CKX activity during leaf development under canopy shade (Carabelli et al. 2007). In pea stem, *PsCKX2* mRNA is strongly induced by auxin, indicating that auxin positively regulates *PsCKX2* expression (Tanaka et al. unpublished data). The decrease in CK in the stem 12 h after decapitation might be caused by both the suppression

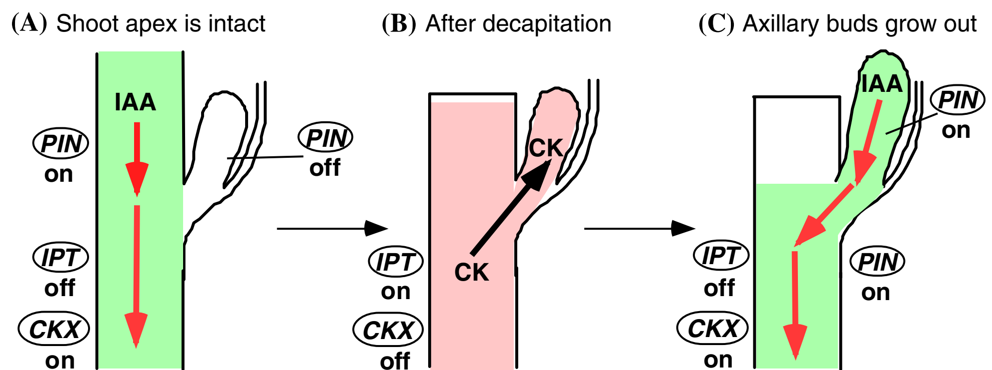

Fig. 1 A model of the interactions between auxin and CK in shoot branching through functions of *PsIPT*, *PsCKX*, and *PsPIN1*

of *PsIPT2* expression and the induction of *PsCKX2* in the stem. Auxin derived from the shoot apex might regulate CK levels in the stem by inducing CK degradation through the regulation of *CKX* expression, as well as by the suppression of CK biosynthesis through the regulation of *IPT* expression to control shoot branching. Plants seem to regulate plant hormone interactions to control developmental programs through complex crosstalk in which the hormones affect the expression of genes related to the biosynthesis as well as hormone metabolism.

A model of interactions between auxin and CK in shoot branching through functions of *PsIPT*, *PsCKX*, and *PsPIN1*

We propose the model of molecular mechanisms in shoot branching shown in Fig. 1. In an intact plant, basipetal auxin flow derived from the shoot apex represses *PsIPT* expression and maintains *PsPIN1* expression in the stem. Consequently, axillary bud outgrowth does not occur. Once the shoot apex is decapitated, the auxin level in the stem decreases and repression of *IPT* expression is released. CK is then biosynthesized *de novo* in the stem and transported into dormant axillary buds to initiate their sustained outgrowth. After axillary bud outgrowth, *de novo* synthesized IAA derived from a new shoot apex flows to the stem, where it represses *IPT* expression and induces CKX to reduce the steady-state CK level in the stem.

A third plant hormone involved in the control of shoot branching

Recently, strigolactone, a group of terpenoid lactones, was identified as a third plant hormone involved in the control of shoot branching (Gomez-Roldan et al. 2008; Umehara et al. 2008). Gomez-Roldan et al. and Umehara et al. demonstrated that the levels of strigolactones are significantly reduced in the roots of enhanced shoot branching mutants (*max*/*rms*/*dwarf*). Moreover, the bushy phenotype of these mutants can be restored toward wild type when exogenous strigolactones are supplied. Strigolactones were previously reported to be communication chemicals in symbiotic arbuscular mycorrhizal fungi that facilitate soil nutrient uptake. Parasitic weeds, *Striga* and *Orobanche*, utilize strigolactone as a plant-derived signal to promote seed germination. To survive under nutrient-deficient conditions, plants might use strigolactones as signal molecules to suppress shoot branching and to communicate with symbiotic arbuscular mycorrhizal fungi. At present, the strigolactone biosynthesis and signal transduction pathways are not clear, and there is still much to be learned about the relationships between strigolactones, auxin, and CK with regard to the control of shoot branching. More information about the interactions between these three plant hormones will provide further insight into the molecular mechanisms of shoot branching.

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