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Current Opinion in Plant Biology

# Hormone signaling in plant development

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Hormone signaling plays diverse and critical roles during plant development. In particular, hormone interactions regulate meristem function and therefore control formation of all organs in the plant. Recent advances have dissected commonalities and differences in the interaction of auxin and cytokinin in the regulation of shoot and root apical meristem function. In addition, brassinosteroid hormones have recently been discovered to regulate root apical meristem size. Further insights have also been made into our understanding of the mechanism of crosstalk among auxin, cytokinin, and strigolactone in axillary meristems.

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**Current Opinion in Plant Biology** 2012, **15**:92–96

This review comes from a themed issue on
Growth and Development
Edited by Xuemei Chen and Thomas Laux

Available online 11th January 2012

1369-5266/$ – see front matter
Published by Elsevier Ltd.

**DOI** 10.1016/j.pbi.2011.12.004

## Introduction

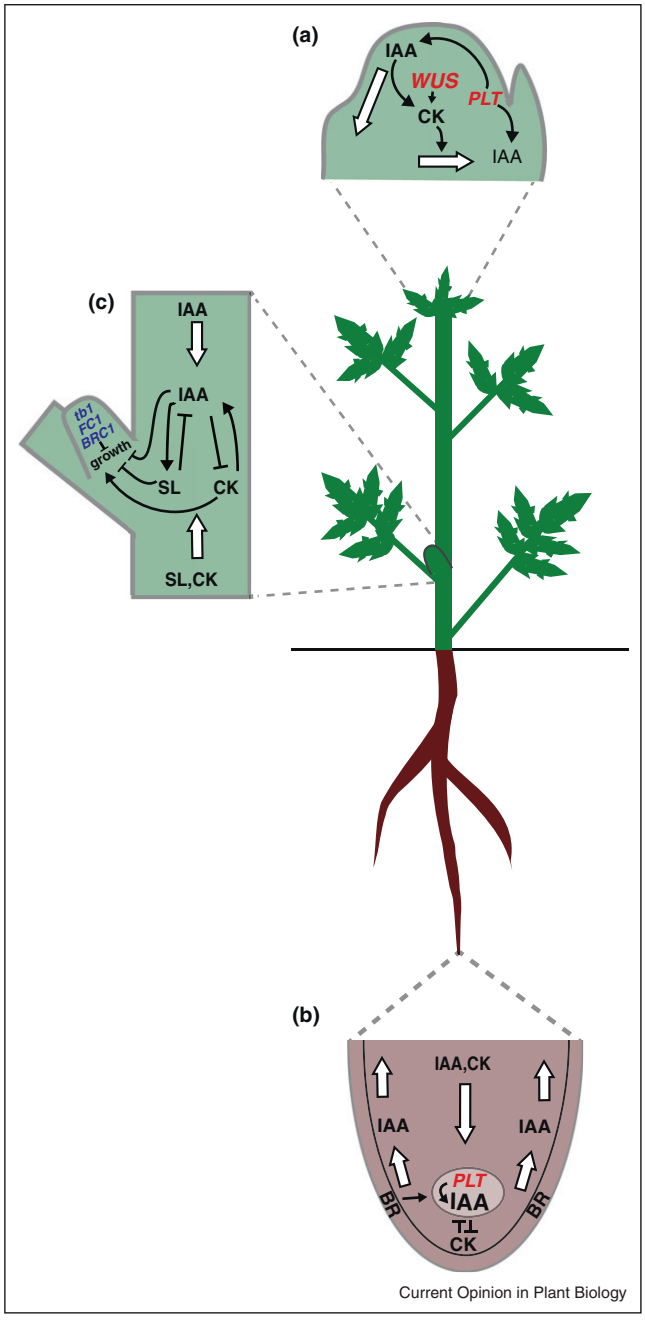
Plant growth hormones, primarily auxin, cytokinin (CK), brassinosteroids (BRs), gibberellins (GA), and strigolactones (SLs), play an amazing array of roles during plant development, from embryogenesis to senescence. In this review, we focus on the role of hormones in meristems, as continuous growth of new organs throughout the plant's life cycle is achieved through the activity of these stem cell populations. The primary meristems in a plant are the shoot apical meristem (SAM), responsible for generating all aboveground organs, and the root apical meristem (RAM), responsible for producing all underground organs. Initiation and outgrowth of axillary meristems (AMs) are responsible for producing all secondary axes of growth including flowers. The last 20 years have seen remarkable advances in our understanding of hormonal regulation of meristem function and plant development. This review focuses on advances in the last two years, in particular on the molecular mechanism of interaction between hormones. Two themes emerge in the mechanisms of hormone crosstalk: first, hormones regulate biosynthesis and transport of other hormones, and second, hormone interactions converge on regulation of particular transcription factors which integrate and coordinate the developmental response.

## Hormone signaling in the shoot apical meristem

In the SAM, several hormones, including auxin, CK, and GA, act both independently and in combination to regulate meristem function [1–3]. In *Arabidopsis*, low levels of GA and high levels of CK are known to maintain the stem cell population in the SAM center. This stem cell population is defined by the activity of the homeodomain transcription factor *WUSCHEL* (*WUS*) [4]. Previously, it was shown that high CK levels in the meristem are maintained by *WUS*-mediated repression of two negative regulators of CK signaling, *ARABIDOPSIS RESPONSE REGULATORS*, *ARR7* and *ARR15* [5]. For further information on the mechanisms of hormone signaling see the review by Shan *et al.* in this issue [6]. Recently, auxin was also discovered to play a role in maintenance of high CK levels in the meristem. The auxin-activated *AUXIN RESPONSE FACTOR/MONOPTEROUS* (*ARF5/MP*) transcriptional regulator was found to directly repress *ARR7* and *ARR15* expression [7••]. Therefore, high levels of CK in the SAM are maintained not only by *WUS*, but also by auxin signaling (Figure 1a).

Along with maintenance of SAM stem cells, auxin and CK regulate organogenesis, with auxin being required for organ initiation in the peripheral zone [8], while CK acts to modulate auxin distribution [9,10]. The site of organ initiation and outgrowth occurs in areas of high auxin, and the localization of auxin is determined by transport of auxin via the PINFORMED (PIN) family of proteins [8]. Many factors that regulate organ position or phyllotaxy in the SAM, including auxin and CK, converge on regulation of the *PIN* genes [9–11]. Studies on the maize *aberrant phyllotaxy1* (*abph1*) gene, which encodes an A-type *ARR* CK signaling regulator, show that *abph1* is required for proper *Zea mays PIN1* (*ZmPIN1*) expression and auxin localization in incipient leaf primordia in the SAM [10]. In addition, CK treatment was found to induce *ZmPIN1* expression in the SAM. In *Arabidopsis*, a recent study using a hypocotyl explant *in vitro* system as a model system for organogenesis found that high CK levels strongly induced expression of *PIN3* and *PIN6* but reduced the expression of *PIN2* [9]. CK also has differential effects on *PIN* expression in the root [9,12]. These results suggest that CK regulates auxin transport through

**Figure 1**




Hormonal regulation of plant development. **(a)** In the SAM, high cytokinin (CK) levels are maintained in the center of the meristem through the combined effects of auxin (IAA) and *WUS* signaling. Meanwhile, high levels of IAA are transported to the periphery of the meristem to sites of organ initiation, facilitated by upregulation of *PIN1* by *PLT*. Upon primordia initiation, IAA is immediately transported to the next site of organ initiation. CK also impacts organogenesis through modulation of auxin transport. **(b)** In the root, IAA and CK are transported basipetally through the vasculature into the meristem. High CK levels in the root tip restricts IAA signaling and allows cell differentiation, while high IAA levels above the tip leads to establishment of the QC (tan oval) and the root stem cell population through activation of *WOX5* and *PLT*. *PLT* in turn upregulates *PIN* expression, helping maintain high IAA levels in the QC. Meanwhile, brassinosteroid (BR) signaling initiated from the epidermal layer acts in maintenance of the root stem cell population. **(c)** IAA travels basipetally in the stem and indirectly inhibits the outgrowth of axillary buds, while CK and SL travel acropetally in the stem and directly regulate (promote and suppress, respectively) axillary bud outgrowth. Proper endogenous levels of IAA, CK, and SL may be maintained by interactions between these hormones through feedback loops. The TCP family of transcription factors, including the maize *tb1*, rice *FC1*, and *Arabidopsis BRC1* genes suppress bud outgrowth in response to IAA, CK, and SL. White arrows indicate direction of hormone transport. Black arrows indicate regulation. Genes in blue function in downregulation. Genes in red function in upregulation. IAA: indole-acetic acid or auxin; CK: cytokinin; BR: brassinosteroids; SL: strigolactone.

transcriptional regulation of the *PIN* genes in both the root and the shoot.

Recent work has shown that *PIN1* expression is also controlled by several members of the *PLETHORA* (*PLT*) family of transcription factors (Figure 1a) [13•]. Loss of *PLT* activity in the SAM periphery was found to reduce *PIN1* expression and alter auxin localization patterns, resulting in predictable changes in phyllotaxy [13•]. Interestingly, *PLT* gene family members were previously shown to induce *PIN1* expression in the RAM [14] but functions in meristem maintenance in the RAM rather than organogenesis [15].

## Hormone signaling in the root apical meristem

In contrast to the SAM, where auxin and CK act in conjunction to establish the stem cell population, auxin and CK act antagonistically to establish and maintain the RAM stem cell population (Figure 1b). The RAM consists of a small group of rarely dividing cells known as the quiescent center (QC), surrounded by more rapidly dividing stem cells that give rise to the various root tissue types. Similar to *WUS* in the SAM, expression of *WUSCHEL-RELATED HOMEOBOX* 5 (*WOX5*) is required for maintenance of the root stem cell population [16]. *WOX5* facilitates proper expression of the *PLT* genes and is restricted to the QC by auxin signaling at the root tip [15,17•]. Crosstalk between auxin and CK in the RAM converges on the regulation of auxin transport and signaling. Under high auxin conditions, the *SHORT HYPOCOTYL2* (*SHY2*)/*IAA3* repressor is degraded, allowing ARF proteins to activate *PIN* expression, leading to high accumulation of auxin in the root tip [18]. In contrast, high CK levels induce expression of *ARR1* and *ARR12* genes, activating *SHY2*, which represses auxin signaling at the root tip beneath the QC [18,19].

Previously, the majority of auxin–CK interactions were found to occur at the transcriptional level. A new study has found that CKs can also directly regulate auxin transport through control of PIN1 localization [20]. CK reduces PIN1 levels by targeting PIN1 proteins for lytic degradation in the vacuole [20]. In another recent study, multiple *arr* mutants, which have a reduced RAM phenotype, were found to have reduced PIN protein levels, but not decreased *PIN* transcript levels [21], providing

further evidence that CK-mediated regulation of PIN1 occurs at the post-transcriptional level. It would be interesting to determine if post-transcriptional regulatory mechanisms are utilized in other auxin/CK controlled developmental processes in the plant.

Although much of the hormonal regulation of the RAM occurs through auxin and CK, other hormones are known to have an effect on the RAM, especially BRs. Two recent studies have shown that mutations in the BR receptor gene *BRASSINOSTEROID INSENSITIVE 1* (*BRI1*) result in aberrant cell cycle progression in the RAM [22•,23••] and that these mutants produce smaller RAMs in a BR dosage-dependent manner [22•]. Although auxin is known to stimulate BR biosynthesis [24], BR activity does not affect the expression of *PIN* genes [23••] and BR mutants do not show the same root tip phenotypes as auxin mutants [22•]. These data suggest that BRs act on the meristem independently of auxin. Interestingly, the analysis of BRs on RAM function revealed that *BRI1* expression in the root epidermis was sufficient to promote root meristem expansion and cell proliferation, while expression in the inner endodermis, QC, or stele could not rescue the *bri1* mutant phenotype [23••]. As the epidermal layer of the SAM has also been shown to play a critical role in the regulation of SAM size [8,25,26], this exciting result suggests that similar modes of regulation exist in the SAM and the RAM epidermis.

## Hormone signaling in axillary meristems

Shoot branching is a two-step process that begins with initiation of AMs in the axils of leaves to form lateral buds followed by bud outgrowth to form branches. Auxin is critical for this process, since genes involved in auxin biosynthesis, transport, and signaling are required for AM initiation [27]. The maize *barrenstalk1* (*ba1*) gene and its rice ortholog *LAX PANICLE1* (*LAX1*), encode a basic helix–loop–helix transcription factor that functions in auxin-mediated regulation of AM initiation [28,29]. Recent papers have identified several other genes that act with *ba1/LAX1* to regulate AM function. The maize *barren stalk fastigiate1* (*baf1*) gene, which encodes an AT-hook transcription regulator, was found to be required for a threshold level of *ba1* expression [30]. The rice *LAX2* gene, which encodes a novel nuclear protein that physically interacts with LAX1, was also shown to promote AM formation [31]. As *ba1/LAX1* related genes are expressed in AMs of many plant species, these genes are likely to be also relevant to branching in dicots [32].

Hormonal regulation of lateral bud outgrowth involves antagonistic action of auxin, CK, and SL (Figure 1c) [27,33–35]. Auxin synthesized in the shoot apex moves basipetally in the polar auxin transport stream and indirectly inhibits outgrowth of axillary buds, a phenomenon known as apical dominance. On the other hand, CK travels acropetally through the xylem into the axillary bud, directly promoting its outgrowth. SLs, a new class of hormones, are synthesized mostly in roots and transported acropetally to directly suppress axillary bud outgrowth. Recently it was shown that SL biosynthesis in *Medicago truncatula* and rice is positively regulated by the GRAS-type transcription factors *NODULATION SIGNALING PATHWAY, NSP1* and *NSP2* [36]. However, further research is required to fully elucidate the SL biosynthesis pathway and the perception of SL in axillary buds.

Auxin and CK interact antagonistically to control branching in the shoot [35,37]. Auxin inhibits the expression of *IPTs* (*ISOPENETENYL TRANSFERASES*) to downregulate CK biosynthesis [38]. Additionally, auxin upregulates expression of *CKX2* (*CYTOKININ OXIDASE2*), involved in the degradation of CK [37]. On the other hand, it was recently shown that increased CK levels induce auxin biosynthesis in young, developing root and shoot tissues in *Arabidopsis*, while decreased CK levels have the opposite effect [39]. These data suggest that proper endogenous auxin and CK levels are maintained by a homeostatic feedback loop.

A feedback loop may also exist between auxin and SL [33,34]. Auxin upregulates expression of the SL biosynthetic genes [40,41]. Moreover, decreased SL increases auxin levels to promote SL biosynthesis. A recent study in *Arabidopsis* showed that SLs also dampen basipetal auxin transport in the stem and reduce PIN1 accumulation in a SL-dependent manner [42••]. Recently in pea (*Pisum sativum*), decapitation was shown to cause polar localization of PIN1 in the buds followed by induction of *PIN1* expression in the stem to establish directional auxin export from bud to stem [43•]. These results support the hypothesis that export of auxin from the bud is important for bud outgrowth.

Environmental and hormonal regulation of shoot branching is integrated by members of the TCP family of transcription factors including the maize *teosinte branched1* (*tb1*), rice *FINECULM1* (*FC1*), and the *Arabidopsis BRANCHED* (*BRC1*) genes, which suppress bud outgrowth [27]. *FC1* and *BRC1* have been shown to interact with auxin and SL in rice and *Arabidopsis*. Recently, negative regulation of *FC1* expression by CK was reported in rice [44]. A new gene in this pathway was recently identified in maize, the homeodomain leucine zipper gene *grassy tillers1* (*gt1*), which suppresses bud outgrowth [45]. Expression of *gt1* is upregulated by shading and is *tb1*-dependent [45]. Further study of the interactions between hormones, *tb1*, *gt1* and the environment will lead to an understanding of the characteristic plant architectures of different species.

## Conclusions

Although crosstalk between hormones is incredibly complex, recent years have seen a dissection of the molecular

mechanisms of hormone interaction. This will certainly continue in the future, aided by modeling studies to dissect the complex feedback and feed forward regulatory mechanisms. It is striking that hormone interaction modules are redeployed over and over again during development. The interaction between auxin, CK and GA plays a fundamental role in SAM function. Interestingly, the auxin–CK–GA module is redeployed in the regulation of compound leaf development in tomato [46–48]. Auxin and CK have long been known to have opposite functions in the root and shoot, and the auxin–CK interaction module is deployed to different effect in the SAM and RAM. Auxin and CK also have opposing roles in the axillary bud. Is this a coincidence or accident of evolution? More likely this interaction reflects a common regulatory mechanism in meristems intersecting with different downstream transcription factors to achieve the appropriate developmental responses.

## Acknowledgements

Research in the McSteen lab on the role of auxin in plant development is funded by the National Science Foundation (IOS-1114484). We apologize to the authors of the deluge of papers in the area of hormonal regulation of development that we could not cite due to space constraints.

## References and recommended reading

Papers of particular interest, published within the period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

1. Depuydt S, Hardtke CS: **Hormone signalling crosstalk in plant growth regulation**. *Curr Biol* 2011, **21**:R365-R373.

2. Stahl Y, Simon R: **Plant primary meristems: shared functions and regulatory mechanisms**. *Curr Opin Plant Biol* 2010, **13**:53-58.

3. Sablowski R: **Plant stem cell niches: from signalling to execution**. *Curr Opin Plant Biol* 2011, **14**:4-9.

4. Mayer KF, Schoof H, Haecker A, Lenhard M, Jurgens G, Laux T: **Role of WUSCHEL in regulating stem cell fate in the *Arabidopsis* shoot meristem**. *Cell* 1998, **95**:805-815.

5. Leibfried A, To JP, Busch W, Stehling S, Kehle A, Demar M, Kieber JJ, Lohmann JU: **WUSCHEL controls meristem function by direct regulation of cytokinin-inducible response regulators**. *Nature* 2005, **438**:1172-1175.

6. Shan X, Yan J, Xie D: **Comparison of phytohormone signaling mechanisms**. *Curr Opin Plant Biol* 2012, **15**:84-91.

7. •• Zhao Z, Andersen SU, Ljung K, Dolezal K, Miotk A, Schultheiss SJ, Lohmann JU: **Hormonal control of the shoot stem-cell niche**. *Nature* 2010, **465**:1089-1092.
This paper demonstrates how auxin and cytokinin inputs are coordinated in the SAM. Inhibition of *ARR7/15*, two negative cytokinin response regulators, by *WUS* allows for high cytokinin signaling in the SAM, while the auxin response factor *ARF5/MP* also represses *ARR7/15*, further establishing high cytokinin in the center of the SAM. Therefore, auxin-mediated regulation of cytokinin signaling explains the interaction between cytokinin and auxin in the SAM.

8. Reinhardt D, Pesce ER, Stieger P, Mandel T, Baltensperger K, Bennett M, Traas J, Friml J, Kuhlemeier C: **Regulation of phyllotaxis by polar auxin transport**. *Nature* 2003, **426**:255-260.

9. Pernisova M, Klima P, Horak J, Valkova M, Malbeck J, Soucek P, Reichman P, Hoyerova K, Dubova J, Friml J *et al.*: **Cytokinins modulate auxin-induced organogenesis in plants via regulation of the auxin efflux**. *Proc Natl Acad Sci U S A* 2009, **106**:3609-3614.

10. Lee BH, Johnston R, Yang Y, Gallavotti A, Kojima M, Travençolo BAN, Costa LDF, Sakakibara H, Jackson D: **Studies of aberrant phyllotaxy1 mutants of maize indicate complex interactions between auxin and cytokinin signaling in the shoot apical meristem1[W][OA]**. *Plant Physiol* 2009, **150**:205-216.

11. Heisler MG, Ohno C, Das P, Sieber P, Reddy GV, Long JA, Meyerowitz EM: **Patterns of auxin transport and gene expression during primordium development revealed by live imaging of the *Arabidopsis* inflorescence meristem**. *Curr Biol* 2005, **15**:1899-1911.

12. Laplaze L, Benkova E, Casimiro I, Maes L, Vanneste S, Swarup R, Weijers D, Calvo V, Parizot B, Herrera-Rodriguez MB *et al.*: **Cytokinins act directly on lateral root founder cells to inhibit root initiation**. *Plant Cell* 2007, **19**:3889-3900.

13. • Prasad K, Grigg SP, Barkoulas M, Yadav RK, Sanchez-Perez GF, Pinon V, Blilou I, Hofhuis H, Dhonukshe P, Galinha C *et al.*: ***Arabidopsis* PLETHORA transcription factors control phyllotaxis**. *Curr Biol* 2011, **21**:1123-1128.
This work reveals three members of the *PLT* transcription factor family to be major regulators of phyllotaxis in the shoot apical meristem. The nonrandom switch from spiral patterning at ~137° to either 90° or 180° in *plt3/5/7* triple mutants was found to be due to changes in PIN1 localization and subsequent loss of auxin maxima in the shoot meristem. Although *PLT* genes have been previously shown to be involved in stem cell maintenance and in growth and division, this work proposes a new role for *PLT* in regulation of organ patterning.

14. Blilou I, Xu J, Wildwater M, Willemsen V, Paponov I, Friml J, Heidstra R, Aida M, Palme K, Scheres B: **The PIN auxin efflux facilitator network controls growth and patterning in *Arabidopsis* roots**. *Nature* 2005, **433**:39-44.

15. Aida M, Beis D, Heidstra R, Willemsen V, Blilou I, Galinha C, Nussaume L, Noh YS, Amasino R, Scheres B: **The PLETHORA genes mediate patterning of the *Arabidopsis* root stem cell niche**. *Cell* 2004, **119**:109-120.

16. Sarkar AK, Luijten M, Miyashima S, Lenhard M, Hashimoto T, Nakajima K, Scheres B, Heidstra R, Laux T: **Conserved factors regulate signalling in *Arabidopsis thaliana* shoot and root stem cell organizers**. *Nature* 2007, **446**:811-814.

17. • Ding Z, Friml J: **Auxin regulates distal stem cell differentiation in *Arabidopsis* roots**. *Proc Natl Acad Sci U S A* 2010, **107**:12046-12051.
Through studies of the distal stem cells (DSCs) in *Arabidopsis* roots, Ding, and Friml show that long distance auxin signals act upstream of the *WOX5* and *PLT* transcription factor network to regulate the RAM stem cell population. Auxin mediated ARF10/16 proteins repress *WOX5* expression in the DSCs, thus restricting its expression to the QC. This work connects long distance hormone signaling with local cell–cell signaling in the RAM.

18. Dello Ioio R, Nakamura K, Moubayidin L, Perilli S, Taniguchi M, Morita MT, Aoyama T, Costantino P, Sabatini S: **A genetic framework for the control of cell division and differentiation in the root meristem**. *Science* 2008, **322**:1380-1384.

19. Moubayidin L, Perilli S, Dello Ioio R, Di Mambro R, Costantino P, Sabatini S: **The rate of cell differentiation controls the *Arabidopsis* root meristem growth phase**. *Curr Biol* 2010, **20**:1138-1143.

20. Marhavy P, Bielach A, Abas L, Abuzeineh A, Duclercq J, Tanaka H, Parezova M, Petrasek J, Friml J, Kleine-Vehn J, Benkova E: **Cytokinin modulates endocytic trafficking of PIN1 auxin efflux carrier to control plant organogenesis**. *Dev Cell* 2011, **21**:796-804.

21. Zhang W, To JP, Cheng CY, Eric Schaller G, Kieber JJ: **Type-A response regulators are required for proper root apical meristem function through post-transcriptional regulation of PIN auxin efflux carriers**. *Plant J* 2011, **68**:1-10.

22. • Gonzalez-Garcia MP, Vilarrasa-Blasi J, Zhiponova M, Divol F, Mora-Garcia S, Russinova E, Cano-Delgado AI: **Brassinosteroids control meristem size by promoting cell cycle progression in *Arabidopsis* roots**. *Development* 2011, **138**:849-859.

This study sheds light on the role of brassinosteroids (BRs) in root development. Through use of cell cycle markers, the authors show that BR signaling mutants have decreased cell cycle activity in the RAM, suggesting a direct cause for the short root phenotype observed in BR mutants. This work provides the first evidence of a role for BRs in meristems.

23. •• Hacham Y, Holland N, Butterfield C, Ubeda-Tomas S, Bennett MJ, Chory J, Savaldi-Goldstein S: **Brassinosteroid perception in the epidermis controls root meristem size**. *Development* 2011, **138**:839-848.

In this paper, the authors show that the small meristems observed in *bri1* mutant roots are due to both impaired mitotic activity as well as impaired cell expansion. In addition, the authors find that expression of wild type *BRI1* solely in the root epidermis is competent to rescue the *bri1* mutant phenotype, and that signal transduction from the epidermis to the meristem does not occur through BES1 and BZR1 signaling. This work sheds light on a distinct mode of meristem-size control.

24. Chung Y, Maharjan PM, Lee O, Fujioka S, Jang S, Kim B, Takatsuto S, Tsujimoto M, Kim H, Cho S *et al.*: **Auxin stimulates DWARF4 expression and brassinosteroid biosynthesis in *Arabidopsis***. *Plant J* 2011, **66**:564-578.

25. Savaldi-Goldstein S, Peto C, Chory J: **The epidermis both drives and restricts plant shoot growth**. *Nature* 2007, **446**:199-202.

26. Abe M, Katsumata H, Komeda Y, Takahashi T: **Regulation of shoot epidermal cell differentiation by a pair of homeodomain proteins in *Arabidopsis***. *Development* 2003, **130**:635-643.

27. McSteen P: **Hormonal regulation of branching in grasses**. *Plant Physiol* 2009, **149**:46-55.

28. Gallavotti A, Zhao Q, Kyozuka J, Meeley RB, Ritter MK, Doebley JF, Pe ME, Schmidt RJ: **The role of barren stalk1 in the architecture of maize**. *Nature* 2004, **432**:630-635.

29. Komatsu K, Maekawa M, Ujiie S, Satake Y, Furutani I, Okamoto H, Shimamoto K, Kyozuka J: **LAX and SPA: major regulators of shoot branching in rice**. *Proc Natl Acad Sci U S A* 2003, **100**:11765-11770.

30. Gallavotti A, Malcomber S, Gaines C, Stanfield S, Whipple C, Kellogg E, Schmidt RJ: **BARREN STALK FASTIGIATE1 is an AT-hook protein required for the formation of maize ears**. *Plant Cell* 2011, **23**:1756-1771.

31. Tabuchi H, Zhang Y, Hattori S, Omae M, Shimizu-Sato S, Oikawa T, Qian Q, Nishimura M, Kitano H, Xie H *et al.*: **LAX PANICLE2 of rice encodes a novel nuclear protein and regulates the formation of axillary meristems**. *Plant Cell* 2011, **23**:3276-3287.

32. Woods DP, Hope CL, Malcomber ST: **Phylogenomic analyses of the BARREN STALK1/LAX PANICLE1 (BA1/LAX1) genes and evidence for their roles during axillary meristem development**. *Mol Biol Evol* 2011, **28**:2147-2159.

33. Domagalska MA, Leyser O: **Signal integration in the control of shoot branching**. *Nat Rev Mol Cell Biol* 2011, **12**:211-221.

34. Beveridge CA, Kyozuka J: **New genes in the strigolactone-related shoot branching pathway**. *Curr Opin Plant Biol* 2010, **13**:34-39.

35. Muller D, Leyser O: **Auxin, cytokinin and the control of shoot branching**. *Ann Bot* 2011, **107**:1203-1212.

36. Liu W, Kohlen W, Lillo A, Op den Camp R, Ivanov S, Hartog M, Limpens E, Jamil M, Smaczniak C, Kaufmann K *et al.*: **Strigolactone biosynthesis in *Medicago truncatula* and rice requires the symbiotic GRAS-type transcription factors NSP1 and NSP2**. *Plant Cell* 2011, **23**:3853-3865.

37. Shimizu-Sato S, Tanaka M, Mori H: **Auxin–cytokinin interactions in the control of shoot branching**. *Plant Mol Biol* 2009, **69**:429-435.

38. Nordstrom A, Tarkowski P, Tarkowska D, Norbaek R, Astot C, Dolezal K, Sandberg G: **Auxin regulation of cytokinin biosynthesis in *Arabidopsis thaliana*: a factor pf potential importance for auxin cytokinin regulated development**. *Proc Natl Acad Sci U S A* 2004, **101**:8039-8044.

39. Jones B, Gunneras SA, Petersson SV, Tarkowski P, Graham N, May S, Dolezal K, Sandberg G, Ljung K: **Cytokinin regulation of auxin synthesis in *Arabidopsis* involves a homeostatic feedback loop regulated via auxin and cytokinin signal transduction**. *Plant Cell* 2010, **22**:2956-2969.

40. Hayward A, Stirnberg P, Beveridge C, Leyser O: **Interactions between auxin and strigolactone in shoot branching control**. *Plant Physiol* 2009, **151**:400-412.

41. Brewer PB, Dun EA, Ferguson BJ, Rameau C, Beveridge CA: **Strigolactone acts downstream of auxin to regulate bud outgrowth in pea and *Arabidopsis***. *Plant Physiol* 2009, **150**:482-493.

42. •• Crawford S, Shinohara N, Sieberer T, Williamson L, George G, Hepworth J, Muller D, Domagalska MA, Leyser O: **Strigolactones enhance competition between shoot branches by dampening auxin transport**. *Development* 2010, **137**:2905-2913.

The mechanism of strigolactone action in branching inhibition is still not clear. This study showed that in *Arabidopsis*, strigolactones may act on auxin canalization by dampening basipetal auxin transport in the stem and reducing PIN1 accumulation on the basal membranes of xylem parenchyma cells to suppress branching.

43. • Balla J, Kalousek P, Reinohl V, Friml J, Prochazka S: **Competitive canalization of PIN-dependent auxin flow from axillary buds controls pea bud outgrowth**. *Plant J* 2011, **65**:571-577.

The mode of action of auxin in inhibiting branching is a matter of debate. This study provided direct evidence to support the auxin canalization model. It was found that decapitation in pea caused a fast polarization of PIN1 in the buds followed by induction of PIN1 expression in the stem to establish directional auxin export from bud to stem, while apically applied auxin prevented these changes.

44. Minakuchi K, Kameoka H, Yasuno N, Umehara M, Luo L, Kobayashi K, Hanada A, Ueno K, Asami T, Yamaguchi S, Kyozuka J: **FINECULM1 (FC1) works downstream of strigolactones to inhibit the outgrowth of axillary buds in rice**. *Plant Cell Physiol* 2010, **51**:1127-1135.

45. Whipple CJ, Kebrom TH, Weber AL, Yang F, Hall D, Meeley R, Schmidt R, Doebley J, Brutnell TP, Jackson DP: **grassy tillers1 promotes apical dominance in maize and responds to shade signals in the grasses**. *Proc Natl Acad Sci U S A* 2011, **108**:E506-E512.

46. Jasinski S, Tattersall A, Piazza P, Hay A, Martinez-Garcia JF, Schmitz G, Theres K, McCormick S, Tsiantis M: **PROCERA encodes a DELLA protein that mediates control of dissected leaf form in tomato**. *Plant J* 2008, **56**:603-612.

47. Shani E, Ben-Gera H, Shleizer-Burko S, Burko Y, Weiss D, Ori N: **Cytokinin regulates compound leaf development in tomato c w**. *Plant Cell* 2010, **22**:3206-3217.

48. Koenig D, Bayer E, Kang J, Kuhlemeier C, Sinha N: **Auxin patterns *Solanum lycopersicum* leaf morphogenesis**. *Development* 2009, **136**:2997-3006.