

# Axillary bud outgrowth in herbaceous shoots: how do strigolactones fit into the picture?

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Received: 9 August 2009 / Accepted: 7 January 2010 / Published online: 29 January 2010  
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**Abstract** Strigolactones have recently been identified as the long sought-after signal required to inhibit shoot branching (Gomez-Roldan et al. 2008; Umehara et al. 2008; reviewed in Dun et al. 2009). Here we briefly describe the evidence for strigolactone inhibition of shoot branching and, more extensively, the broader context of this action. We address the central question of why strigolactone mutants exhibit a varied branching phenotype across a wide range of experimental conditions. Where knowledge is available, we highlight the role of other hormones in dictating these phenotypes and describe those instances where our knowledge of known plant hormones and their interactions falls considerably short of explaining the phenotypes. This review will focus on bud outgrowth in herbaceous species because knowledge on the role of strigolactones in shoot branching to date barely extends beyond this group of plants.

**Keywords** Shoot branching · Apical dominance · Strigolactone · Auxin · Decapitation · Light · Nutrient · Flowering

## Regulation of bud outgrowth

In different species, and also different positions along the plant stem, axillary buds exhibit differing degrees of

development and outgrowth during the vegetative phase. Along the main shoot axis in pea (*Pisum sativum*), axillary meristems develop into buds with immature leaves at most nodes, and the outgrowth of these buds typically remains arrested (Stafstrom and Sussex 1988, 1992; Stafstrom 1993). In *Arabidopsis* (*Arabidopsis thaliana*), axillary buds form late during vegetative growth and are generally suppressed until the switch to flowering (Grbić and Bleecker 2000). In monocots such as rice (*Oryza sativa*), the characteristic growth habit comprises the release of many axillary buds to form tillers, while in other monocot species such as corn (*Zea mays*) the outgrowth of axillary buds remains relatively restricted (McSteen 2009).

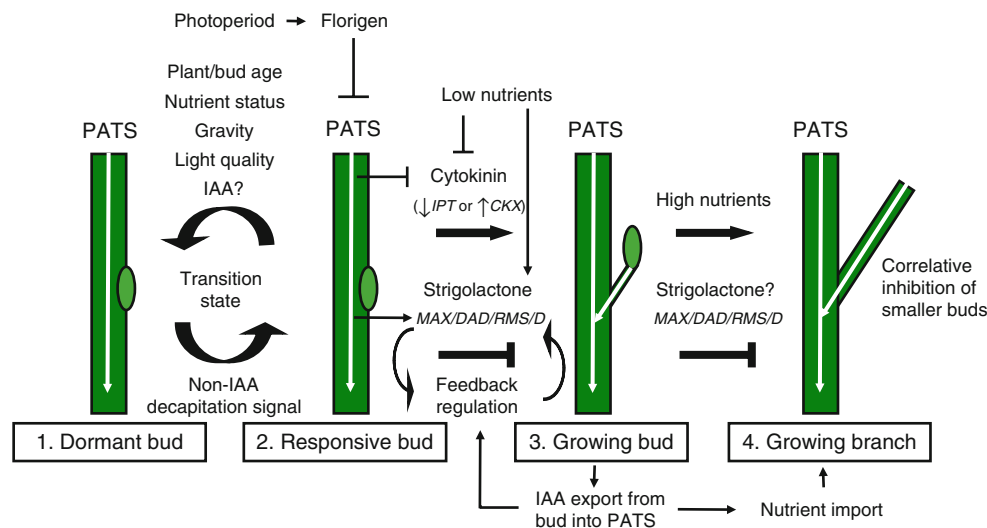
Despite the diversity in growth habits, bud outgrowth among herbaceous species is a multifactorial process determined largely by the same factors. In addition to the classical hormones auxin and cytokinin, and the recently identified strigolactone compound (Gomez-Roldan et al. 2008; Umehara et al. 2008), other external and internal cues also contribute to spatial and temporal regulation of bud outgrowth. Together, these are thought to impose three distinct states of dormancy: (1) endodormancy, by factors acting within the bud, (2) paradormancy, by signals within the plant but external to the bud, and (3) ecodormancy, by environmental factors external to the plant (Lang et al. 1987). Interpretation of multiple signals within buds may be accomplished, at least in part, by the existence of distinct stages at which different signals might act (Fig. 1; Dun et al. 2006). For example, numerous studies demonstrate that the transition of axillary buds from dormancy to growth is likely to comprise an intermediate phase of altered metabolic activity. This transition phase is characterised by increased cellular activity compared to cells within dormant buds, as evidenced by gene and protein expression levels and even small increases in bud size

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**Fig. 1** Stages of bud outgrowth. Buds may progress (*bold arrows*) through different growth states depending on different regulatory factors (*standard arrows*). Various cues including positional and developmental information can cause dormant buds (1) to enter a stage of transition, characterised by altered metabolic activity. (Stafstrom and Sussex 1988; Devitt and Stafstrom 1995; Shimizu and Mori 1998; Napoli et al. 1999; Shimizu-Sato and Mori 2001). Buds may then become responsive (2) to regulatory factors such as stem auxin level, which may be influenced by florigen signalling

(Stafstrom and Sussex 1988, 1992; Madoka and Mori 2000; Shimizu-Sato and Mori 2001; Morris et al. 2005; Shimizu-Sato et al. 2008). Importantly, buds undergoing this transition are capable of cycling back and forth between dormancy and growth (Stafstrom and Sussex 1988; Devitt and Stafstrom 1995; Shimizu and Mori 1998; Napoli et al. 1999; Shimizu-Sato and Mori 2001). This may provide a convenient framework in which to consider multiple branching inputs, where factors such as light, nutrient availability, the age and position of the bud and the plant's developmental stage may determine the ability of individual buds to respond to other long-distance signals such as auxin, cytokinin and strigolactone.

### Hormone interactions and functions

Strigolactones are carotenoid-derived terpenoid lactones first characterised as germination stimulants of the parasitic plant genera *Striga* and *Orobanchae*, and as hyphal branching regulators in arbuscular mycorrhizal associations (Cook et al. 1972; Matusova et al. 2005; Akiyama et al. 2005; Humphrey and Beale 2006; López-Ráez et al. 2008). Strigolactones, or a derivative thereof, have recently been identified as the long-distance branching inhibitor regulated by the *RMS* (*RAMOSUS*) pathway in pea, *MAX* (*MORE AXILLARY GROWTH*) pathway in *Arabidopsis*, and *D/HTD* (*DWARF/HIGH TILLERING DWARF*)

(Shalit et al. 2009). Auxin acts to independently up-regulate strigolactones and down-regulate cytokinin level. Auxin and strigolactones inhibit the progression to growth while cytokinins promote this progression. If a bud is induced to grow (3), auxin transported from the bud into the PATS acts to feedback up-regulate strigolactone synthesis and attract nutrient import. Depending on nutritional status and hormonal interactions a growing bud will enter into sustained growth (4) and can inhibit the growth of additional buds by correlative inhibition (Morris 1977; Li and Bangerth 1999)

pathway in rice (Gomez-Roldan et al. 2008; Umehara et al. 2008). Mutants for these pathways that are defective in the production, but not the perception, of the branching inhibitor have reduced strigolactone levels. Furthermore, the increased branching in the synthesis mutants, but not the perception mutants, can be repressed by addition of the strigolactone analogue, GR24 (Gomez-Roldan et al. 2008; Umehara et al. 2008). As small, potent molecules required for communication within the plant body and between plants and the environment, strigolactones therefore fit the bill as a new class of phytohormone (Klee 2008; Leyser 2008; Dun et al. 2009).

The hormones auxin and cytokinin are also essential components of the network controlling branching in plants. Auxin transported downwards from the shoot tip in the polar auxin transport stream (PATS) indirectly represses bud outgrowth, while cytokinin promotes cell division and differentiation and bud outgrowth (reviewed in Kyojuka 2007; Shimizu-Sato et al. 2009). A number of studies have begun to elucidate how these hormones and strigolactones may specifically interact to regulate bud dormancy.

Auxin has been shown to promote the expression of strigolactone biosynthesis genes encoded by *MAX3* and *MAX4* in *Arabidopsis*, *RMS5* and *RMS1* in pea, and *HTD1* and *D10* in rice (Foo et al. 2005; Bainbridge et al. 2005; Johnson et al. 2006; Zou et al. 2006; Arite et al. 2007; Hayward et al. 2009). In *Arabidopsis*, this regulation is dependent on *AXR1* (*AUXIN RESISTANT1*) (Bainbridge

et al. 2005; Hayward et al. 2009). AXR1 is required for the optimal activity of SCF (Skp1-Cul1/Cdc53-F-box) E3 ubiquitin ligase complexes including the auxin receptor SCF<sup>TIR1</sup> (TRANSPORT INHIBITOR1), and auxin binding to SCF<sup>TIR1</sup> induces the degradation of Aux/IAA transcriptional repressors (reviewed in Mockaitis and Estelle 2008). In *Arabidopsis*, recent data has provided evidence that this transcriptional interaction between auxin and strigolactone synthesis genes is required, at least in part, for auxin-mediated branching inhibition. The auxin response mutant *bodenlos*, which has a lesion in domain II of the Aux/IAA transcriptional repressor IAA12, has very low *MAX3* and *MAX4* expression and increased branching that is dependent on strigolactone supply (Hayward et al. 2009). The increased branching in additional auxin response mutants can also be suppressed by strigolactone application (Brewer et al. 2009).

While the above studies demonstrate auxin may regulate strigolactone levels, this interaction is not sufficient to completely explain auxin-mediated branching inhibition (Bennett et al. 2006; Hayward et al. 2009), which also likely involves auxin-mediated repression of cytokinin levels (reviewed in Shimizu-Sato et al. 2009). In pea stems, auxin inhibits the expression of *ISOPENTENYL TRANSFERASE (IPT)* cytokinin biosynthesis genes and promotes the expression of a *CYTOKININ OXIDASE (CKX)* gene involved in cytokinin breakdown (Tanaka et al. 2006; Shimizu-Sato et al. 2009). In *Arabidopsis*, cytokinin levels are negatively regulated by auxin in an AXR1-dependent manner (Nordström et al. 2004). Thus a transcriptional interaction via the SCF<sup>TIR1</sup> E3 ubiquitin ligase signalling cascade may control both strigolactone and cytokinin levels. On the other hand, a functional interaction between strigolactones and cytokinin is yet to be clarified and evidence so far suggests they may act partly independently, as discussed further below (Bennett et al. 2006; Ongaro et al. 2008; Ferguson and Beveridge 2009; Leyser 2009). Nonetheless, it is known that low strigolactone signalling in the shoot leads to decreased cytokinin content in the root xylem sap (Foo et al. 2007) and, in *Arabidopsis*, exogenous cytokinin can prevent the up-regulation of strigolactone synthesis gene expression by auxin in the root (Bainbridge et al. 2005).

Currently, the mechanism of strigolactone action in branching inhibition is uncertain. Strigolactone perception or signal transduction requires localised action of an F-box protein within axillary buds or the adjacent nodal stem, encoded by *MAX2* in *Arabidopsis*, *RMS4* in pea and *D3* in rice (Stirnberg et al. 2002, 2007; Ishikawa et al. 2005; Johnson et al. 2006; Gomez-Roldan et al. 2008; Umehara et al. 2008). Therefore, strigolactone perception via an SCF complex, similarly to auxin and SCF<sup>TIR1</sup>, may target additional downstream branching regulators for ubiquitination and degradation by the 26S proteasome (Stirnberg

et al. 2007). In *Arabidopsis*, a gene thought to act downstream of the *MAX* pathway is *BRANCHED1* or *TEOSINTE BRANCHED-LIKE1 (BRC1 or TBL1)*, identified based on its similarity to *TEOSINTE BRANCHED1 (TBI)* in maize and its orthologue, *OsTBI/FC1 (FINE CULM1)*, in rice (Doebley et al. 1997; Hu et al. 2003; Takeda et al. 2003; Aguilar-Martínez et al. 2007). *TBI* encodes a member of the TCP (TB1, CYCLOIDEA, PCF domain) transcription factor family, and TB1 is required in buds to inhibit outgrowth. In *Arabidopsis*, *BRC1* expression is down-regulated in *max* mutant buds, and double mutants for *brc1* and the *max* genes show similar branching phenotypes as the single mutants, suggesting they act on the same pathway (Aguilar-Martínez et al. 2007; Finlayson 2007). *BRC1* is also thought to act partly independently of cytokinin to control branching (Aguilar-Martínez et al. 2007), consistent with some distinct aspects of strigolactone- and cytokinin-mediated branching control (Leyser 2009). In rice, however, evidence so far suggests that *FC1* may act independently from strigolactone, as *FC1* expression is not altered in strigolactone pathway mutants (Arite et al. 2007).

Two hypotheses have been presented for the ultimate output of strigolactone signalling. The first is that strigolactones modulate the ability for buds to establish an auxin flow into the PATS of the primary stem (Bennett et al. 2006; reviewed in Ongaro and Leyser 2008). This is based on the premise that axillary meristems require auxin-transporting vascular connections to the main stem vasculature, canalised by auxin flow and driven by source-sink relationships, in order to function and grow (reviewed in Ongaro and Leyser 2008; Leyser 2009). Accordingly, auxin transport and/or level is increased in strigolactone pathway mutants of *Arabidopsis* and rice, and where tested, branching in these mutants can be reduced by applying polar auxin transport inhibitors (Bennett et al. 2006; Arite et al. 2007; Lin et al. 2009). The second hypothesis for strigolactone action builds upon the auxin regulation of strigolactone synthesis and suggests that strigolactones function downstream of auxin to inhibit branching as second messengers (reviewed in Dun et al. 2009). In accordance with this hypothesis, GR24 applied to buds can inhibit bud outgrowth in auxin-depleted stems of decapitated pea plants (Brewer et al. 2009). Here, auxin transport from buds is proposed to be important for continued growth, rather than the initial release of buds, and the increased auxin content often observed in the mutants is suggested to be a result of bud outgrowth and/or feedback up-regulation (Dun et al. 2006, 2009).

Auxin and strigolactone may also interact to control branching as part of a feedback process. Recently it was demonstrated in *Arabidopsis* shoots that the increased auxin content of strigolactone pathway mutants acts to feedback up-regulate the expression levels of strigolactone

synthesis genes (Hayward et al. 2009). Auxin and strigolactone are therefore likely to be linked in a feedback loop, where low strigolactone leads to increased auxin content, which in turn up-regulates strigolactone synthesis (Hayward et al. 2009; Fig. 1).

### Developmental and positional effects

The position or age of a bud along the stem can determine its ability to grow out. During vegetative development, bud outgrowth tends to occur in an acropetal wave before shifting to a basipetal wave during the reproductive phase (McSteen and Leyser 2005). This pattern of branch development broadly defines three distinct zones in the main stem: (1) an upper zone, where increased bud outgrowth accompanies flowering, (2) a middle zone directly beneath the upper zone where bud outgrowth is typically reduced, and (3) a basal zone (Weberling 1989; Napoli et al. 1999). In pea, the bud at node 2 within the basal zone is particularly responsive to outgrowth following the release of apical dominance by decapitation or girdling (discussed below; Ferguson and Beveridge 2009). Buds at these basal nodes are also very responsive to photoperiod, having a greater tendency for outgrowth under short days (Beveridge et al. 2003). Cytokinin application to buds at the first four nodes only promotes branching in nodes 1 and 2 in intact plants; decapitation is additionally required to trigger the outgrowth at the upper nodes (King and Van Staden 1988). Buds at lower nodes, particularly node 2 may possess particularly well-established vascular connections (Ferguson and Beveridge 2009).

Auxin is one candidate for mediating some of these positional effects (reviewed in McSteen and Leyser 2005). As described above, it has been proposed that vascular connectivity and the ability of buds to plumb into the PATS is critical for outgrowth (Ongaro and Leyser 2008; Ongaro et al. 2008). The maintenance of bud outgrowth also appears to correlate with the ability of buds to synthesise auxin *de novo* and export it into the stem, even in cases where buds already have well-developed vascular connections (Morris 1977; Morris and Johnson 1990; Li and Bangerth 1999). Furthermore, auxin movement may also determine the ability of a bud to attract nutrients to support sustained outgrowth (Davies and Wareing 1965; Phillips 1968; Jiang et al. 2001; Yang et al. 2007; Fig. 1).

Three-dimensional positioning can also influence axillary branching. When plants are placed horizontally, bud outgrowth occurs along the upper stem, and when plants are completely inverted, the most basal bud grows out (Cline and Riley 1984; Prasad and Cline 1985). Likewise, when only the upper part of the main stem is bent downwards, the axillary bud at the node closest to the bend point

is induced to grow. This response is likely to be mediated by gravity because the agravitropic *weeping* (*we*) mutants in Japanese morning glory (*Pharbitis nil*) exhibit a reduced lateral outgrowth response of buds to stem bending (Kitazawa et al. 2008). This gravity-induced release of axillary buds appears to act independently of auxin and cytokinin, and possibly independently of strigolactone, as auxin and cytokinin levels do not change in accordance with shoot bending. The *we* mutants also possess a normal decapitation response that can be rescued by auxin application. A gravity-mediated mechanism for bud outgrowth further highlights the ability of plants to adapt their growth pattern in order to cope with changing conditions.

### Correlative inhibition

For branching to be optimised in the context of a growing, healthy shoot system, it must be carefully modulated. In branching terms, correlative inhibition refers to the ability of one branch to suppress the growth of other axillary shoots, and this has been associated with the active transport of auxin from the growing branch (Morris 1977; Li and Bangerth 1999). In pea, where more than one dormant bud may exist at each node, the largest bud will generally be induced to grow, suppressing the growth of the accessory buds (reviewed in Shimizu-Sato and Mori 2001). In *rms* mutants, growing basal axillary buds can inhibit the outgrowth of aerial buds and accessory buds in the same node, suggesting correlative inhibition acts at least partly independently from the strigolactone pathway (Morris et al. 2005; Ferguson and Beveridge 2009). Using a two-branched system in *Arabidopsis*, correlative inhibition was found to comprise both *MAX*-pathway dependent and independent components that can be transmitted unilaterally across the stem (Ongaro et al. 2008). Interestingly, double mutants for the aforementioned *rms* genes and *rms2*, which is not yet cloned but causes increased xylem cytokinin content, display outgrowth of previously inhibited accessory buds compared to single *rms* mutants (Beveridge et al. 1997; Murfet and Symons 2000a, b), suggesting the *RMS2* gene product and/or cytokinin may also be involved in correlative inhibition independent from strigolactones.

### Decapitation

Activities which cause loss of the shoot apex, such as pruning and herbivory, can lead to the activation of dormant axillary buds, enabling continued shoot growth and reproduction. This is particularly important in species that show a strong apical dominance phenotype (where lateral shoot growth is normally suppressed). Loss of auxin produced by

these apices is central to this response, resulting in increased sink strength of the main stem PATS for bud-derived auxin, reduced strigolactone production and increased cytokinin biosynthesis and levels (Sachs and Thimann 1964, 1967; Bangerth 1994; Nordström et al. 2004; Tanaka et al. 2006; Brewer et al. 2009; Hayward et al. 2009). Nonetheless, in pea, the outgrowth of the bud at node 2 is rapidly induced by decapitation in a manner that does not correlate with the reductions in auxin level and strigolactone synthesis gene expression in the stem (Morris et al. 2005; Ferguson and Beveridge 2009). Decapitation itself may trigger a physical signal (such as an electrochemical pulse), which is transmitted along the main stem to buds at every node to induce an initial growth response (Morris et al. 2005; Ferguson and Beveridge 2009). The enhanced ability of the bud at node 2 to respond to this signal and subsequently grow may be due to its well-developed vascular connectivity. Such connections are essential for meristems to export auxin and receive nutrients, as previously described. Indeed, physical re-directions in assimilates, minerals, endogenous cytokinins and water flow from the shoot apex to axillary buds occur rapidly after loss of the shoot apex, as well as alterations in photosynthate supply and source-sink relations (Husain and Linck 1966; Stahlberg and Cosgrove 1992; Turnbull et al. 1997). In addition, cytokinin is metabolised in buds soon after decapitation (Mader et al. 2003).

Further evidence for an auxin-independent effect of decapitation on bud outgrowth is that branching at certain nodes of pea is not always induced when stem auxin levels are depleted by girdling or inhibiting polar auxin transport, unless plants are simultaneously decapitated (Morris et al. 2005; Ferguson and Beveridge 2009). For example, buds at node 2 of pea remain dormant even when the plant is girdled in the internode directly above (Ferguson and Beveridge 2009). In these girdled plants, reduced branching responses are associated with low expression of *IPT* cytokinin synthesis genes in the vicinity of the bud, suggesting that the regulation of cytokinin by factors independent from polar auxin transport could be an additional determining factor for bud outgrowth (Ferguson and Beveridge 2009). Accordingly, cytokinin application to these buds restores outgrowth (Ferguson and Beveridge 2009). In pea *rms* mutants and in the rice *d10* mutant, treatments that disrupt the flow of auxin from the shoot tip cause an additive branching response (Beveridge 2000; Arite et al. 2007; Ferguson and Beveridge 2009). Again this suggests that auxin inhibits branching via strigolactone-dependent and independent pathways. Buds in different locations may also be controlled by partially distinct genetic pathways, as shown in the monocot, foxtail millet (*Setaria italica*), where the branches produced in the basal nodes are under separate control to those produced in the upper nodes (Doust et al. 2004; Doust 2007).

Despite an initial growth response following decapitation, the sustained growth of buds in pea and many species is reliably inhibited by exogenous auxin applied to the decapitation site (Thimann and Skoog 1933, 1934; Cline 1996; Cline et al. 2001). In flowering *Arabidopsis*, however, auxin cannot prevent the outgrowth of buds following decapitation except in the *avr3* increased auxin-response mutant and in isolated cauline nodes lacking a root system (Cline 1996, 2000; Cline et al. 2001; Ouellet et al. 2001). In these isolated nodes, basally supplied cytokinin can counteract the inhibitory effect of apically supplied auxin on bud outgrowth (Chatfield et al. 2000). Perhaps in different species, differences in the relative levels or influence (response) of branching hormones in the context of diverse developmental programs and environmental signals may be a source of varied branching responses. In species with short life-cycles such as *Arabidopsis*, a strong response to decapitation after the transition to reproductive growth may be particularly important for the completion of the life-cycle.

## Light

The length and quality of light that plants are exposed to can alter their branching patterns. As mentioned above, under short day conditions, branching is increased at basal nodes in pea (Beveridge et al. 2003). Similar branching patterns are also observed in *Arabidopsis* and petunia (*Petunia hybrida*) (Grbić and Bleecker 2000; Stirnberg et al. 2002; Snowden and Napoli 2003). Unlike WT plants, intact *die neutralis* (*dne*) mutants in pea lack the ability to produce basal branches under short days, suggesting that the *DNE* gene product is probably required to mediate photoperiod responsiveness in basal axillary buds. The branching pattern of *rms* mutants is dependent on photoperiod, as the basal branching habit of *rms1* and *rms2* mutants under short days is shifted towards a relatively more aerial pattern of branching under long days (Beveridge et al. 2003). Reduced outgrowth of buds at basal nodes under long days is also observed in decapitated WT plants, suggesting a similar pathway mediates this photoperiod response (Beveridge et al. 2003).

The ratio of red to far-red light (R:FR) is critical in the light reception response. The relative amount of FR light is greatly enhanced when light conditions are low (i.e. canopy shading), or when light is reflected (i.e. crowding or increased plant density). High FR levels alter phytochrome activity and initiate light responses, particularly phytochrome B, which acts in the shade response (Robson et al. 1993). As a consequence, the plant's resources are invested into growth of the main shoot axis, including the hypocotyl, at the expense of bud outgrowth and leaf

growth (Cerdán and Chory 2003; Casal et al. 2004; Doust 2007; Kebrom and Brutnell 2007; Carabelli et al. 2007).

Recent work has demonstrated interactions and overlap between strigolactone- and light-mediated branching control. In *Arabidopsis*, the *MAX2* F-box gene has been identified independently in three mutant screens for delayed leaf senescence, increased branching and photomorphogenesis regulation, respectively (Woo et al. 2001; Stirnberg et al. 2002; Shen et al. 2007). In rice, the D3 F-box is also involved in leaf senescence (Yan et al. 2007). The pleiotropic phenotypes of *max2* mutants are indicative of multiple roles for *MAX2* in plant development and consistent with a possible function for the nuclear-localised F-box in regulating multiple signalling cascades (Stirnberg et al. 2007). *MAX2* expression is induced by light in WT seedlings, and *max2* mutant seedlings are hyposensitive to R and FR light in a phytochrome-dependent manner (Shen et al. 2007). However, *MAX2* and *phyB* appear to have antagonistic roles in older plants in branching regulation, as *phyB* mutants display small reductions in branch number and *MAX2* is epistatic to *phyB* in double mutants which possess a *max2* increased-branching phenotype (Reed et al. 1993; Shen et al. 2007). These findings suggest *MAX2* could play different roles in the plant depending on developmental stage.

In *Sorghum bicolor*, increased apical dominance in a *phyB* mutant is accompanied by increased expression of the *TBI* orthologue *SbTBI* (Kebrom et al. 2006). Thus, low R:FR ratios leading to inactive *phyB* (or non-functional *phyB* in a mutant), results in maintenance of *SbTBI* expression and bud outgrowth is inhibited. Further to this, expression of *BRC1* in *Arabidopsis* is increased under crowded conditions, though it is not known for certain if the crowding effect is specifically related to reduced R:FR ratios (Aguilar-Martínez et al. 2007). Together, these data suggest *TBI* could be an important integrator of light and strigolactone to control bud dormancy in some species.

Another point of cross-talk between light and branching could potentially occur via cytokinin catabolism. As part of shade avoidance response, the arrest of leaf growth is also known to rely on the up-regulation of certain *CKX* genes under low R:FR ratios (Carabelli et al. 2007). It is possible that a low R:FR ratio might also restrict branching in a similar way by maintaining levels of *CKX* enzymes. As reviewed in Leyser (2009), much of this relationship between light quality and branching may be mediated by affects of light on auxin fluxes and auxin response.

## Flowering

In most annual/model species, the amount of vegetative branching is typically decreased under environmental

conditions conducive to flowering, while delayed flowering promotes branching (e.g., Beveridge et al. 2003). In *Arabidopsis*, while growth under long days promotes flowering and results in less overall rosette branching, rosette bud outgrowth is usually not induced until the switch to flowering. This is possibly due to reduced auxin production by, and increased distance from, the floral apex (McSteen and Leyser 2005).

In monocots and dicots, overexpression or high expression of *TERMINAL FLOWER1* (*TFL1*) usually leads to delayed flowering and increased lateral branching, while *tfl1* mutants exhibit early flowering and little to no branching (Ratcliffe et al. 1998; Jensen et al. 2001; Nakagawa et al. 2002; Foucher et al. 2003). In tomato, *TFL* and *FLOWERING LOCUS T* (*FT*) homologues are considered important regulators of vegetative development in their own right (Shalit et al. 2009). The aforementioned circadian clock photoperiod response gene *DNE* in pea may act via regulation of *FT* to enhance basal branching under short days (Liew et al. 2009). In sweet pea, where floral induction occurs vary late in development (even in early day-neutral flowering lines) assimilate partitioning and basal branching is regulated by the photoperiod response pathway well before flowering has been induced at the shoot apical meristem (Beveridge et al. 1992). In perennial plants, *FT* has been strongly implicated in growth cessation and bud endodormancy (reviewed by Horvath 2009). Consequently, evidence from a broad range of species supports the notion that florigen, mediated by *FT*, can be considered as a general plant hormone affecting vegetative and reproductive development.

Strigolactone mutants do not possess major pleiotropic effects on flowering time or flower number. However, the *dad1-1* mutant in petunia flowers later than WT, *dad2-1* and *dad3* mutants (Napoli 1996; Snowden and Napoli 2003). Nevertheless, interactions between flowering and axillary branching involving florigen, strigolactone and other chemical hormones are beginning to emerge. Overexpression of a gene promoting the vegetative-to-flowering transition, *TaVRN1*, encoding a MADS-box transcription factor, has been shown to increase axillary bud outgrowth, as well as *MAX4* expression (Adam et al. 2007). This raises the possibility that a feedback mechanism(s) may act to coordinate the strigolactone branching pathway with inflorescence development. Recently it was suggested that interactions between auxin and florigen may coordinate sympodial branching, axillary bud release and flowering in tomato (*Solanum lycopersicon* L.). Mutations in the tomato *TFL1* homologue *SELF-PRUNING* (*SP*) gene can be mimicked by the PATS inhibitor TIBA (2,3,5-triiodobenzoic acid) and *sp* mutants possess reduced apical dominance (Pnueli et al. 2001; Shalit et al. 2009). A possible model for the regulated release of axillary buds to form

sympodial branches is that a particular ratio between the gene products of *SP* (an inhibitor of florigen) and *SINGLE FLOWER TRUSS (SFT)* (florigen) arrests the PATS in localised manner, leading to outgrowth of an adjacent axillary meristem (discussed in Shalit et al. 2009).

Depleted gibberellin and auxin content underlie the reduced internode lengths and increased branching at upper nodes of *gigas-1 (gi-1)* late flowering pea plants under long days compared to short days (Beveridge et al. 2001). These *gi-1* plants likely carry a mutation in one of the *FLOWERING LOCUS T-LIKE (FTL)* genes in pea (Weller et al. 2009) and exhibit photoperiod-dependent delayed flowering which under long-days is accompanied by a reiterative reduction in vegetative growth commencing at about the time where flower development occurs in WT (Taylor and Murfet 1994; Beveridge et al. 2001; Hecht et al. 2005). In contrast with the gibberellin flowering pathway in *Arabidopsis*, gibberellin application to pea can only restore the vegetative phenotype back to WT and has no effect on flowering (Beveridge and Murfet 1996).

## Nutrients

Nutrient status can undoubtedly affect the ability of shoots to produce lateral branches. As strigolactone acts as a shoot branching inhibitor and a promoter of hyphal branching in mycorrhizal associations, it is possible strigolactone could provide the plant with a means for balancing nutrient levels, particularly phosphorus, with shoot growth (Akiyama et al. 2005; Gomez-Roldan et al. 2008; Umehara et al. 2008; Leyser 2009). Indeed, in rice, tomato and red clover, phosphorous starvation can increase the production of strigolactones (Yoneyama et al. 2007; López-Ráez et al. 2009; Umehara et al. 2008). Finer regulation of branching might also be achieved in combination with auxin and cytokinin, which exhibit nutrient dependent responses either in movement, accumulation or levels (for review, see Rubio et al. 2009). As discussed above, auxin flow in the PATS may influence the movement of nutrients within the plant. Auxin transport can act as a sink for phosphorus (Davies and Wareing 1965). Nitrogen availability is critical for cytokinin synthesis and nitrate has been shown to up-regulate cytokinin levels and *IPT* gene expression (Takei et al. 2004; Miyawaki et al. 2004). Boron deficiency in pea causes bud outgrowth, probably due to reduced auxin transport out of the apex (Wang et al. 2006). Defoliation of plants cannot inhibit branching in plants triggered by decapitation, though branch length is reduced (Ferguson and Beveridge 2009). This suggests nutrients, specifically carbon sources, could limit the sustained outgrowth of buds but not the initial release of bud dormancy.

## Conclusions and perspectives

Like many developmental processes, shoot branching is under multifactorial regulatory control. Even with the discovery of strigolactones, the complexities and number of interactions means we are still far from a solid understanding of branching control in plants. Moreover, in some cases, the hormone interactions are in themselves multiple. For example, auxin may affect strigolactone levels, and strigolactones may affect auxin levels in a feedback role, or more directly, as strigolactone function may involve localised auxin transport. In addition to strigolactone and auxin, cytokinin, nutrient supply and the interaction with light, photoperiod and the induction of flowering are all important. It is likely that branching is induced by changes in different pathways under different conditions and at different developmental stages and that the relative importance of each may vary in different species. Elucidation of the strigolactone signal transduction pathway and the mechanisms of crosstalk in biosynthesis and signalling are likely to yield major progress in understanding branching control in the future. However, the discovery of strigolactones already provides new avenues to consider agricultural and horticultural opportunities such as suppressing an undesirable vegetative flush after pruning.

**Acknowledgments** We thank the Australian Research Council for grant funding and the Australian Postgraduate Award scheme for funding to AH and TW.

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