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REVIEW

Current perspectives on shoot branching regulation

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Abstract Shoot branching is regulated by the complex interactions among hormones, development, and environmental factors. Recent studies into the regulatory mechanisms of shoot branching have focused on strigolactones, which is a new area of investigation in shoot branching regulation. Elucidation of the function of the *D53* gene has allowed exploration of detailed mechanisms of action of strigolactones in regulating shoot branching. In addition, the recent discovery that sucrose is key for axillary bud release has challenged the established auxin theory, in which auxin is the principal agent in the control of apical dominance. These developments increase our understanding of branching control and indicate that regulation of shoot branching involves a complex network. Here, we first summarize advances in the systematic regulatory network of plant shoot branching based on current information. Then we describe recent developments in the synthesis and signal transduction of strigolactones. Based on these considerations, we further summarize the plant shoot branching regulatory network, including long distance systemic signals and local gene activity mediated by strigolactones following perception of external environmental signals, such as shading, in order to provide a comprehensive overview of plant shoot branching.

Keywords apical dominance, decapitation, shade, shoot branching, strigolactones, sugar demand

1 Introduction

1.1 Significance of plant shoot branching

Branching characteristics, including the number of lateral branches, their length and location on the stem, determine plant architecture. Plant architecture is an important trait of crop and horticultural plants. Branching is a highly plastic process, suggesting that plants can integrate signals from endogenous hormones and external environmental during the developmental process and adjust their branching characteristics to adapt to these environmental changes[1–3]. Thus, branching is jointly regulated by complex interactions among hormones, development, and environmental factors.

1.2 Stages of lateral branch development

Generally, plant lateral branch development consists of two phases: the initiation of meristems in the axil and elongation of the axillary bud[4]. Axillary meristems are first initiated in axils, and then form axillary buds. Axillary buds then enter a transitional phase (dormancy or growth). Axillary buds in the transitional phase are mainly regulated by developmental processes, hormonal factors and environmental factors. Buds either choose to continue in a dormant state or grow to form lateral branches. Axillary buds in dormancy can be activated into growth by the interaction between internal developmental signals and external environmental factors. Likewise, growing axillary buds can be transformed into dormant buds by internal and external factors[4,5]. Therefore, the final number of branches depends on the number of axillary meristems and activity of the axillary buds. The plasticity of shoot branching depend on axillary buds activity (i.e., elongation of the axillary bud)[6]. Therefore, the theoretical model of branching regulation was based on the activity regulation of axillary buds.

In this review, systematic regulatory networks of branching and its advances are summarized based on existing knowledge. Recent progress on the synthesis and signal transduction of strigolactones are discussed as well. Based on this, we summarize the shoot branching regulatory network involved in long distance systemic signaling and local gene activity, mediated by strigolactones after plants receive external environment signals.

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2 Shoot branching regulatory networks

2.1 Long-distance signals of systemic regulatory networks: auxin, strigolactone, and cytokinin are the main hormones that regulate shoot branching

Plants have evolved a unique transport system to generate a variety of signaling molecules that are transferred between various tissues and organs. This coordinates the developmental processes of plants, including shoot branching. As small-molecule organic compounds, plant hormones are vital in this process[7]. Currently, the well-characterized hormones that are involved in the shoot branching regulation over long distances include auxin, strigolactones and cytokinin. Auxin indirectly inhibits branching by regulating the activity of a second messenger and by competing with the auxin transport channel. Strigolactones also regulates shoot branching by two mechanisms: by weakening auxin transport and by transcriptional activation of *BRANCHED 1* (*BRC1*). Cytokinins promote shoot branching and display the interaction with auxin and strigolactones. Thus, plant shoot branching is not regulated by a single hormone that functions independently, but by complicated interactions among more members.

Auxins are produced in young leaves at the shoot apex[8] and are then transported downward through the polar auxin transport (PAT) stream to inhibit branching. During this process, PIN-FORMED (PIN) protein functions as an output vector of auxin that controls the direction of the PAT stream[9,10]. Cytokinin is produced mainly in roots and is then transported upwards through the xylem and actively regulates shoot branching. Strigolactones are synthesized both in roots and stems and are thought to be transported in the xylem to the plant apex, thereby inhibiting shoot branching[11–13].

The three hormones mentioned above interact with each other to form a complex system that regulates shoot branching. However, there is currently no ideal model to explain the entire branching phenomenon in plants. The auxin polar transport and second messenger models have been used to explain the regulation of shoot branching. However, the recent findings also show that sucrose is key to the process of axillary bud release, which expanded our understanding of apical dominance control. In this review, we propose a third regulation hypothesis in which sucrose regulates the activity of axillary buds.

Therefore, the following three hypotheses can be proposed to explain long-distance signaling and regulation of shoot branching.

2.1.1 Auxin transport canalisation-based model

The auxin transport canalisation-based model suggests that the establishment of auxin output channels is essential for activating axillary buds. This process is regulated by PAT in the main stem[6,13,14]. This hypothesis is based on the positive correlation between the activity state of buds and the PAT stream in bud shoots. In this model, the shoot apex or buds function as an auxin source and roots as an auxin sink. Auxin is transported to the roots through the auxin transport channel in the main stem. Bud activation occurs when the output of auxin from the bud increases. After removing the tip of the plant (decapitation), the output of auxin in activated buds increases rapidly. This upregulation of auxin export is accompanied by gradual polarized accumulation of PIN-FORMED 1 (PIN1, auxin efflux protein)[15]. According to this theory, the various auxin sources will compete for establishing the auxin transport channel with the main stem. The export of auxin from activated buds will reduce the transport capacity of the main stem, thereby preventing the output of auxin in other buds[6] and inhibiting the activity of axillary buds[16–19]. During this process, PIN proteins are important. With an accumulation of PIN proteins in cells, auxin flux is gradually canalised into cell files. These cells then differentiate into vascular tissue and transport auxin[6,20]. PIN1-expressing cell files build up the relationships between the PAT stream and axillary buds, and ultimately establish the connection channel between the bud and main stem. Using a computer model, Przemyslaw et al.[21] further demonstrated that auxin transport acts as a switch to control bud activation.

In this hypothesis, strigolactones are thought to inhibit shoot branching by regulating auxin polar transport. Strigolactones systematically suppresses PAT stream channels by reducing the accumulation of PIN proteins in the cell membrane. This decreases the competitiveness of buds for the auxin sink on the main stem and inhibits the activation of buds. Support for this theory is follows. In strigolactones synthesis mutants, a large number of branching phenotypes are often accompanied by high levels of PIN1 protein accumulation, high capacity of auxin transport, and a high concentration of auxin accumulation in the main stem[17,21]. In *Arabidopsis* and rice strigolactones mutants (more axillary growth (*max*) mutants), *max* mutants show resistance to the inhibitory effect on axially buds generated by apical application of auxin and this resistance is not dependent on the signal transduction pathway mediated by *AXR1*(auxin resistance 1). In contrast, *max* mutants showed increased auxin transport capacity. At the same time, the phenotype restoration of mutants would caused PIN1 decreasing and less PAT capacity. The above results indicate that a large number of branching phenotypes in the strigolactones mutant are closely associated with enhancement of auxin transport ability[17,19,22]. Crawford et al.[23] used *max* mutants to perform further studies and found that both the exogenous synthetic strigolactone (SL) analog GR24 and endogenous strigolactones synthesis could inhibit auxin transport. In addition, inhibition of branching by GR24

was functional only when there was an auxin source in the main stem, which means there is a source of auxin competition. GR24 can also reduce the accumulation of PIN1 in xylem parenchyma cells[23]. In chrysanthemum, GR24 did not affect the growth of lateral buds on one-bud stem segments[24]. However, when GR24 and NAA were used at the same time, they strongly inhibited the growth of lateral buds, which was more effective than using NAA or GR24 alone[24]. In the presence of two lateral buds, GR24 inhibited growth of only the bottom one, but had no effect on growth of the top lateral bud. However, using GR24 and NAA simultaneously strongly inhibited the growth of single and double buds[24]. Thus it showed that the ability of strigolactones to inhibit chrysanthemum lateral bud outgrowth depends on the presence of a competitive auxin source[24]. Shinohara et al.[25] used computer models to simulate the effect of strigolactones in the process of regulating auxin transport and subsequently shoot branching. They demonstrated that strigolactones regulate shoot branching by rapidly adjusting auxin transport, which provides strong evidence for the auxin polar transport hypothesis. Further studies indicate that strigolactones can promote or inhibit shoot branching through rapidly modulating auxin transport by triggering rapid depletion of the PIN1 protein from the plasma membrane of xylem parenchyma cells in the stem[25].

In the auxin transport canalisation-based model, cytokinins are thought to function in branching regulation by regulating auxin synthesis and transportation. There is significant support for this hypothesis.

Firstly, applying cytokinin and auxin to the top bud at the same time can inhibit auxin transport to the axillary bud[26–28]. Other studies show that cytokinin can directly induce auxin synthesis[29]. Secondly, the application of exogenous BAP to dormant buds rapidly increases the expression of auxin-carrier coding genes *PsAUX1* and *PsPIN1* of pea plants (*Pisum sativum* L.)[30]. In addition, cytokinin treatment rapidly leads to polarized deposion of PsPIN1 protein[30]. These findings support the auxin transport channel hypothesis, where competition between auxin sources in the polar auxin transport stream channel in the main stem regulates activity of the axillary bud. In addition, strigolactones regulate shoot branching by adjusting this competitive process and cytokinin regulates branching by adjusting auxin synthesis and transportation.

The auxin polar transport hypothesis explains this shoot branching phenomenon. However, the following phenomena are difficult to explain based on this hypothesis for the reasons given below.

Firstly, Brewer et al.[31] found that in peas, after removing the tip of a pea plant, exogenous strigolactones could still inhibit bud outgrowth. In the *Arabidopsis* auxin response mutants *axr1* and *tir1-q*, exogenous strigolactones can reduce branching in *Arabidopsis*. Therefore, it can restore the mutant branching phenotype to wild-type levels. In the main stem of both wild type (WT) and strigolactones synthesis mutants of *Arabidopsis* and pea, both WT and mutants can transport not only their own synthetic endogenous auxin but also excess exogenous auxin. This indicates that the primary reason for branches decrease is not auxin transport capacity[31].

Secondly, Renton et al.[32] used a simple compartment model to detect patterns of carrier-dependent auxin transport and signaling. Their results showed that the main stem has the ability to transport a large amount of additional auxin. Furthermore, auxin depletion in stems was far slower than the initiation of bud outgrowth after decapitation in pea. Thus, the depletion of auxin is less likely to be responsible for causing initial bud release.

Applying auxin transport inhibitors to the main stem tissue upper bud does not necessarily stimulate bud outgrowth[33,34]. This further supports the suggestion that there are other messengers participant in auxin indirect function on inhibiting buds outgrowth. The second messenger hypothesis discussed below can explain this phenomenon.

2.1.2 Second messenger hypothesis

According to the second messenger hypothesis, auxin cannot directly act on buds, but rather regulates an upward transport signal that moves directly into the bud to regulate the activity of axillary buds. Cytokinin and strigolactone are considered two potential second messengers[6,35].

The main evidence to support this alternative mechanism is as follows.

Firstly, buds of numerous strigolactones mutant are resistant to the inhibition effect produced by applying auxin to the shoot apex[31,36], indicating that strigolactones are required during this inhibition process.

Secondly, auxin regulates strigolactones production. Compared with *Arabidopsis* WT, the *max4-1* (more axillary growth (*max*) mutants) mutant showed weaker inhibitory bud outgrowth effects when auxin was applied to the apex using a split plate assay. Removing the source of auxin using decapitation significantly decreased *RMS1* (ramosus mutants, *MAX4* homologous genes) transcription in peas within 6 h, but *RMS1* transcription was significantly upregulated after the application of exogenous auxin (IAA). In *Arabidopsis*, although the increase in *MAX4* transcription in stems were not observed within 6 h, using both RT-PCR and GUS reporter lines, after applying auxin in the apex the GUS expression level was significantly upregulated in GUS reporter lines when they were placed in an auxin-containing medium. Furthermore, when grafting *max4* mutant scions onto wild-type rootstocks, the mutant showed a branching phenotype restored to the wild-type level. These results indicated that auxin may function by regulating a signal that can move directly into the axillary buds by upregulating the expression of *RMS1/MAX4*, a signal dependent on *RMS1/MAX4* to inhibit shoot branching[37]. Subsequently, Foo et al.[38] demonstrated

that the application of auxin to the apex rapidly increases the expression of *RMS1* in pea nodal tissue. In rice, auxin could also upregulate the accumulation of *D10* (DWARF 10) mRNA[18] and expression of *HTD1* (HIGH TILLERING DWARF 1) (Encoding CAROTENOID CLEAVAGE DIOXYGENASE (CCD7))[39]. After strigolactones were shown to be a plant shoot branching inhibitor, Brewer et al.[31] explored their function and showed that exogenous strigolactones could inhibit bud outgrowth of pea. Strigolactones applied to decapitated peas could also inhibit bud outgrowth[31]. Meanwhile, researchers found that strigolactones applied to the *Arabidopsis* auxin response mutant could decrease its branch number, indicating that auxin may promote apical dominance mediated by strigolactones[31]. After the application of strigolactones to the tiny buds of pea strigolactones mutant and decapitated pea, in both cases they quickly stopped growing. However, when using *N*-1-naphthylphthalamic acid (NPA, an auxin transport inhibitor) treatment, the tiny buds slowly stopped growing after several days. Furthermore, stems of strigolactones systemic mutants and WT in peas and *Arabidopsis* could transport their own endogenous and exogenous auxin. This demonstrates that the main factor restricting branching is not only auxin transport capacity. Hayward et al.[36] used the *axr1* mutant combined with qRT-PCR and found that the expression of *MAX3* and *MAX4* in shoots was upregulated by auxin; an upregulation dependent on *AXR1*. After NPA processing, expression levels decreased. In addition, *MAX3* and *MAX4* downregulation, which is associated with auxin, can activate branching. In chrysanthemum, it was also found that exogenous auxin increases expression of the strigolactones synthesis gene *DgCCD8* in roots and stem[24]. Dun et al.[40] showed that the major site perceiving strigolactones is axillary buds and not the adjacent stipules. The continuous application of strigolactones to each axillary bud on the main stem internode could completely inhibit branching. This inhibitory effect of strigolactones on early bud growth did not require inhibitory signals originating from other growing buds or the shoot apex. Meanwhile, the inhibition of bud growth by application of GR24 did not depend on auxin from the apex. These results indicated that strigolactones act downstream of auxin to inhibit bud outgrowth and can function as a second messenger for auxin.

In addition to strigolactones, cytokinin is also a second messenger of auxin, as supported by the following observations.

Cytokinin can be transported upward through xylem sap and enter the axillary bud to promote axillary bud outgrowth. Also, direct application of cytokinin to the axillary buds can promote bud outgrowth. This effect is also observed in the presence of an auxin source in the tip bud[27,41]. Finally, auxin can negatively regulate cytokinin synthesis. In *Arabidopsis*, auxin inhibits cytokinin synthesis through the auxin-signaling pathway, which is mediated by *AXR1*[42]. In pea, auxin can inhibit local cytokinin the production in nodal stems by negatively regulating *PsIPT* (cytokinin synthetic gene) expression[43].

The above results suggest that cytokinin is a second messenger for auxin in shoot branching regulation.

2.1.3 Nutritional and hormonal hypothesis

For more than a century, the auxin theory has been considered an established, core theory of maintaining apical dominance of plants. When considering the auxin transport channel hypothesis or second messenger hypothesis, auxin is central.

However, Morris et al.[33] performed an experiment in pea, monitoring dynamic changes in auxin level, auxin transport and axillary bud growth after decapitation, and applying NPA to the top bud. They found that initial bud growth occurs prior to changes in IAA concentration or transport in surrounding stem tissue, and that it is not prevented by an acropetal supply of exogenous auxin after decapitation. Therefore, they proposed that there is no relationship between the dynamic changes in auxin with initial growth of axillary bud after decapitation. In addition, they showed that depletion caused by girdling, defoliation or NPA application is not sufficient to trigger bud outgrowth in wild-type plants[34]. Applying auxin to decapitated plants could not inhibit bud outgrowth in the 10 species tested. Auxin also had no effect on Coleus or *Arabidopsis* grown in a greenhouse[44]. Furthermore, Renton et al.[32] used a model to study the relationship between auxin depletion and bud release, and found that auxin depletion is slower than the initial release of bud outgrowth after decapitation. Thus, during bud outgrowth there is no direct correlation between changes in auxin content and changes in polar auxin transport capacity in the main stem. Therefore, auxin loss is unlikely to be the initial cause of bud outgrowth. In addition, merely relying on auxin alone cannot prevent the initial release of dormant buds, indicating that other factors may be involved in this process.

The above studies suggested that decapitation might cause bud release by triggering a mechanism that is not dependent on auxin.

Morris et al.[33] previously used peas to study the relationship between auxin content, transport rate and bud release, and found that auxin did not have a significant effect on the early bud growth of pea. Furthermore, girdling the lower part of the pea stem did not trigger bud release below the girdling region. This suggests that decreases in inhibitory signals, which originate from the top of the stem (including auxin depletion), are not sufficient for promoting bud release. In contrast, girdling the decapitated plants could prevent bud release, suggesting that there must a specific signal produced above the girdling cuts ring to control bud release. Furthermore, auxin in main stems cannot move into the axillary bud and

applying auxin to decapitated plants does not always prevent bud release. These results suggest that axillary buds are released by a positive shoot-derived signal and that this signal might be something other than auxin. However, the nature of this signal needed to be resolved.

This new signal was not identified until a recent study by Mason et al.[45]. They performed an in-depth study regarding the positive shoot-derived signal in pea. They found that the process of bud release of axillary buds occurred earlier than the changes in auxin concentration after decapitation. Meanwhile, this type of bud release was not affected on auxin application in the apical region. In contrast to auxin, sucrose is rapidly redistributed by long-distance transport within 24 h after decapitation and accumulates in axillary buds. This change is closely associated with bud release. In addition, artificially increasing the level of sucrose in plants can rapidly promote axillary bud release, along with the downregulation of *BRC1*. Overall, this study showed that an enhancement in sugar supply is both necessary and sufficient for suppressed buds to be released from apical dominance. Based on the above findings, Mason et al.[45] proposed that apical dominance is controlled jointly by sucrose and hormone. In his hypothesis, the sugar demand of the shoot apex is critical to maintaining apical dominance. In addition, the shoot tip's demand for sugars inhibits axillary bud outgrowth by limiting the amount of sugar translocated to those buds. When the accumulation of sucrose reaches a threshold in the axillary buds, its translocation becomes limited. The process of bud release activation is independent of the auxin status in the main stem. Auxin mainly affects the elongation process after the bud is activated, and functions in determining which buds will continue to grow out in the later stage.

Characterization of the role of sucrose in bud release has expanded understanding of shoot branching, but how the hormone signals integrate with sucrose signals to regulate branching remains unclear.

2.2 Biosynthesis, transport and signal transduction of strigolactones

In recent years, research on branching has generated great interest following the discovery of the role of strigolactones in branching. Within a few years, a large number of genes related to the biosynthesis of and signal transduction by strigolactones were identified. Recent studies have provided novel insights into strigolactones biosynthesis and signal transduction; the strigolactones biosynthetic pathway is well-understood and the signal transduction network framework has been established[6,13,46,47]. However, significant gaps in our knowledge remain. For example, the function of strigolactones in plant development and the detailed regulatory mechanism of strigolactones in shoot branching remains unclear. Recently, the function of strigolactones in shoot branching has been widely reviewed[1,48–57]. However, few comprehensive reviews are summarized on strigolactones transduction with D53, DWARF 14 (D14) and DWARF 3 (D3) as central components. However, the development of strigolactones research has been rapid, especially since the discovery of *D53*, which is involved in the crux pathway between strigolactones and branching. The primary objective of this review is to synthesize recent research with historical studies and previous reviews, identifying knowledge gaps that still exist in strigolactones synthesis and transport. The interactions between genes central to strigolactones transduction pathways, particularly based on recent strigolactones studies, are emphasized and a comprehensive branching regulation network connecting strigolactones with *D14*, *D3*, and *D53* is described.

2.2.1 Strigolactones biosynthesis

Seto et al.[55] have reviewed strigolactones biosynthesis in detail. Here, we summarize the strigolactones biosynthesis process and discuss current problems. Strigolactones are mainly produced in plant roots and close to the shoot axis[37,58], and they are synthesized via the carotenoid pathway. Under the action of a carotenoid isomerase D27 (homolog of *AtD27*) (a carotenoid isomerase, DWARF27 (D27)), all-*trans*-β-carotene is transformed into 9-*cis*-β-carotene[59,60]. Then under the action of carotenoid cleavage dioxygenases (CCD7), 9-*cis*-β-apo-10′-carotenal is generated. Again, under the action of CCD8, active precursor strigolactones carlactone (CL) is generated[61–63]. These biosynthetic pathway genes have been identified, but the process from CL to strigolactones remains unclear. At this time, the only gene known to be involved in this process is *MAX1*, which encodes a class-III cytochrome P450 monooxygenase. Currently, it has been found that in *Arabidopsis*, *MAX1* facilitates conversion of CL to mature strigolactone 5-deoxystrigol (Fig. 1).

However, further experiments are required to clarify its biochemical action[63,69]. In *Nicotiana benthamiana*, a gene homologous to *MAX1* has been identified, which encodes a subfamily member of cytochrome P450 CYP711 that acts as a CL oxidase to stereo-selectively convert CL into ent-2′-*epi*-5-deoxystrigol (involving B-C lactone ring formation), which is the presumed precursor of rice strigolactones[68].

Numerous studies have shown that not only these key enzymes, but also some other genes may be important, or there may even be other strigolactones synthetic pathways. For example, tomato *Sl-ORT1* is involved in the synthesis of strigolactones[70], and in *Arabidopsis AtPPD5* is thought to be involved in strigolactones synthesis, but the specific features of these genes require further study[71]. Furthermore, symbiotic GRAS-type transcription factors *NSP1* and *NSP2* are important in the strigolactones synthetic pathways of *Medicago truncatula* and rice[72].

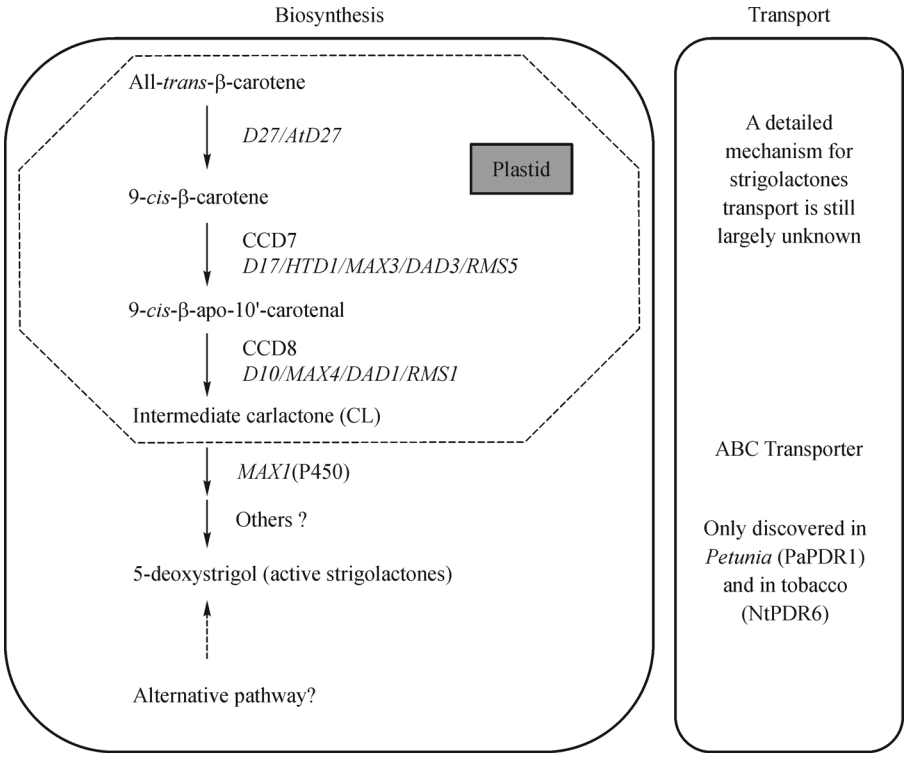

Fig. 1 Strigolactones biosynthesis and transport pathway. Several CCD enzymes have been found in various species. Orthologous CCD7, MAX3 in *Arabidopsis thaliana*[61], D17/HTD1 in *Oryza sativa*[64], RMS5 in *Pisum sativum*[65], DAD3 in *Petunia hybrida*[66]; Orthologous CCD8, D10 in *O. sativa*[18], MAX4 in *A. thaliana*[37], RMS1 in *P. sativum*[37], DgCCD8 in *Chrysanthemum*[24] and DAD1 in *P. hybrida*[67]. A gene homologous to *MAX1* has been identified in *Nicotiana benthamiana*[68].

The strigolactones synthesis pathway has not been fully characterized and the following issues remain to be addressed. Firstly, how is CL converted to active strigolactones? Secondly, are there other possible synthetic pathways for strigolactones? Finally, what are the factors that regulate strigolactones synthesis?

2.2.2 Transport of strigolactones

In the second messenger hypothesis, strigolactones are transported to axillary buds and act as a second messenger for auxin. Since strigolactones are considered to be produce in root tips and close to shoot axis[37,58], this suggests that characterizing strigolactones transport mechanisms in plants will increase our understanding of the mechanism whereby they regulate shoot branching. Grafting experiments have demonstrated that shoot-derived strigolactones are sufficient to regulate bud outgrowth, but strigolactones are also transported from the root to the shoot[11,73]. In addition, in mycorrhizal fungi, it is also reported that strigolactones are exported from the root to the shoot[73]. These facts suggest that the mechanisms of strigolactones transport are multiple and complex. However, it is still unclear how strigolactones transport.

Recent models show that *D3*, *D14* and *D53* are key factors in strigolactones signaling. The above three enzymes regulate strigolactones through the ubiquitination degradation pathway, which is formed by interactions among *D3*, *D14* and *D53*; these interactions are activated by strigolactones. Therefore, only when these factors are found together in the plant can the strigolactones response occur. *D53* is widely distributed and expressed in the rice vasculature in roots, shoots, leaves, leaf sheaths, nodes, internodes and young panicles, and preferentially in the parenchyma cells surrounding the xylem. *D14* is expressed in axillary buds, root vasculature and carpels. The location of *D14* expression is identical to *D3*[74–76]. The similar expression pattern of *D14*, *D3* and *D53* suggests that interactions among these three proteins are possible. However, the distribution of strigolactones in plants is not ubiquitous, and strigolactones are considered to be produced in root tips and close to the shoot axes, and are transported upward in xylem sap[11,37,58,77]. However, *D14* is not expressed in the xylem. Besides, grafting experiments of wild-type *Arabidopsis* and *d14-2/seto5*, *max2-1* and *max4-1* show that *D14* regulates branching not by being transported from a long distance, but by acting locally[76]. However, some locations where *D14* and *D3* are highly expressed are not connected with the vasculature, such as axillary buds. In addition, the synthesis of strigolactones is regulated by negative feedback[78].

Therefore, a mechanism likely exists to coordinate the biosynthesis and transport of strigolactones in the plant to prevent the accumulation of excessive strigolactones by blocking biosynthesis. The question that arises is how are strigolactones released from producing cells and transferred to their destination where they have their effect? Currently, some researchers speculate that a cellular transport system may be involved in the transport of strigolactones to dormant buds[79]. The first discovered transporter of strigolactones is *Petunia axillaries* PLEIOTROPIC DRUG RESISTANCE 1 (PaPDR1), which is required for regulating the development of arbuscular mycorrhizae and axillary branches by acting as a cellular strigolactone exporter[79]. The recent report by Sasse et al.[73] further demonstrates that *PaPDR1* exhibits a cell-type-specific asymmetric localization in root tips and in the hypodermal passage cells. And the polar localization of *PaPDR1* mediates directional (shootward) strigolactones transport as well as localized exudation into the soil. Meanwhile, the study also suggested that the localization of *PaPDR1* is determined by strigolactones and auxin. Another novel *Nicotiana tabacum* ATP binding cassette (ABC) transporter *NtPDR6* was discovered in tobacco, which might function as a strigolactones transporter to regulate shoot branching[80] (Fig. 1). It seems that the transportation of strigolactones from biosynthetic to target tissues is regulated not only by endogenous and exogenous signals, but also by the interactions between strigolactones and auxin biosynthesis, and possibly other hormonal networks. Whether, and exactly how, root and shoot-derived strigolactones interact in the plant and how these interactions are integrated and modulated by transport processes still need further study.

2.2.3 Signal transduction of strigolactones

Strigolactones perception[55] and signal transduction[54] have been discussed in previous reviews. Here the focus is on summarizing the latest regulatory network with D3, D14 and D53 at the core, especially based on our recent understanding of D53 in strigolactones signaling and the discovery of a negative feedback loop in the strigolactones pathway.

Recent advances in our understanding of strigolactones signal transduction, particularly the clarification of the function of D53[46,47], have allowed the core signal transduction pathways of D3, D14 and D53 to be established. *D14* encodes a protein of the α/β hydrolase fold family, which is considered to be the receptor for strigolactones[81–84]. *D3* encodes a Leu-rich repeat F-box protein and plays a role in strigolactones perception[16]. *D53* encodes a class I Clp ATPase protein, which is a substrate of the SCF-D3 ubiquitination complex. D53 can interact with D14 and D3 to form a complex, acting as a repressor in strigolactones signaling and, therefore, D53 is a positive regulator of branching. The detailed mechanisms are strigolactones-dependent. When strigolactones are present, D14 first senses the strigolactones signal and then forms a D14-SCF-D3 complex with SCF-D3 to promote the ubiquitination of D53. The complex then becomes polyubiquitinated for proteasome-dependent degradation. When D53 is degraded, the positive effects on branching are relieved so that the strigolactones signal can be transmitted and the downstream target genes respond and inhibit branching. When there are no strigolactones, or they are present at low concentration, D53 cannot be degraded, and ultimately promotes shoot branching[46,47]. In the strigolactones pathway, there is also a negative feedback regulation mechanism. Strigolactones can also induce and promote the rapid degradation of D14, and this response depends on the function of *MAX2* (which has homology to D3). This negative feedback loop will weaken strigolactones signal perception and effectively limits the duration and intensity of strigolactones signaling[76]. Conversely, strigolactones can regulate its concentration by regulating its own synthesis gene at the transcriptional level. Also, strigolactones can facilitate the degradation of its own receptors to maintain the balance of the strigolactones signal. If strigolactones can directly contribute to the degradation of its own receptor D14, then this mechanism may reduce the efficiency of strigolactones. Based on these results, we considered whether there are mechanisms that can prevent strigolactones degradation of D14. We speculate that, when other SCF-MAX2 degradable substrates are present, strigolactones preferentially promotes SCF-MAX2 degradation of other substrates; D14 will promote or possibly be involved in this degradation process, thus promoting the degradation of other substrates, after which strigolactones can have its effect. After the substrate is exhausted, strigolactones will stimulate SCF-MAX2 to degrade D14, thereby reducing the level of strigolactones signal. Furthermore, a mechanism or factor that depends on strigolactones to maintain D14 stability may exist. In the presence of such factors, D14 will not be degraded by strigolactones, but when such factors lose their function, D14 will be degraded. D53 can be considered such a factor. In the presence of strigolactones, D14 can promote the degradation of D53 by interacting with SCF-MAX2. After D53 is exhausted, strigolactones will gradually degrade D14. However, an in-depth study of this phenomenon would increase our understanding of the strigolactones signaling pathways and their internal balancing.

Since the function of D53 has been characterized, the basic framework of strigolactones signal transduction has been established (Fig. 2), but we know relatively little about the events that occur after the D53-D14-SCFD3 complex detects strigolactones. In particular, the strigolactones target genes or direct effect genes have not been identified. Previous reports have indicated that strigolactones can regulate branching by (1) regulating *BRC1* (the *BRC1* pathway), similar to the second messenger hypoth-

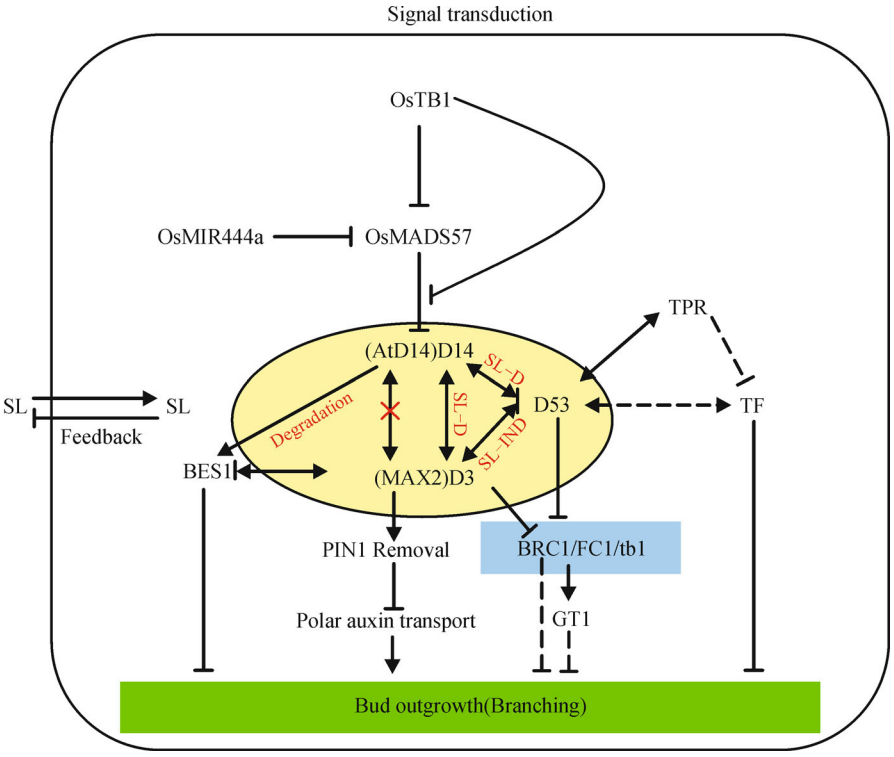

Fig. 2 The strigolactones signal transduction pathway and the control of bud outgrowth. At high concentrations of strigolactones, strigolactones perception by D14 promotes the interaction between D14 and D3 (MAX2 in *Arabidopsis*) with D53, and subsequent degradation of D53 by the ubiquitin proteasome system. This leads to the propagation of the strigolactones signal and plays a role through the *BRC1* pathway or auxin transport pathway (possibly a third pathway present in *Arabidopsis* mediated by *BES1*), or some other unknown pathway. At low concentrations of strigolactones, D53 may interact with TPR and repress *BRC1*, thus blocking the strigolactones signal and finally promoting bud outgrowth. SL, strigolactone; SL-D, SL dependent; SL-IND, SL independent. AtD14 and MAX2 cannot interact with each other.

esis, or (2) regulating auxin transport, similar to the auxin transport canalization-based model.

In the *BRC1* pathway, *BRC1* expression is induced at the transcriptional level by strigolactones. GR24 can upregulate expression of *BRC1* and its homologous gene, and in *max* mutants the *BRC1* mRNA level is reduced, indicating that *BRC1* is downstream of the strigolactones signal[78,85–87]. *FC1* was previously reported as *OsTB1*, an ortholog of maize teosinte branched (*tb1*)[88,89]. However, in rice and maize, GR24 treatment does not induce *FC1* mRNA expression changes and in an SL-defective mutant (*d* mutant), *FC1* expression does not significantly decrease. In a *d3* mutant, *FC1* overexpression can only partially restore the branching phenotype of the *d3* mutant. The above results indicate that *FC1* and strigolactones might be not be fully independent in regulating branching[18,86,88,90]. However, the different branching phenotypes of the pea *rms1* mutant and *Psbrc1* mutant indicate that strigolactones may regulate branching not only through the *BRC1* pathway but potentially also through other mechanisms. In this hypotheses, strigolactones inhibition of branching requires the presence of a competitive source of auxin and strigolactones can reduce the level of PIN1 in xylem parenchyma cells[23,24]. The *max* mutants often showed greater stem auxin transport capacity along with higher accumulation of PIN1 in stems compared with the wild-type[17]. Recent studies have shown that strigolactones can promote or inhibit shoot branching by triggering rapid depletion of auxin efflux protein PIN1 from the plasma membrane[25]. These results demonstrate that strigolactones regulates branching by regulating auxin transport. However, the role of strigolactones in PIN1 protein depletion remains unclear. Waldie et al.[53] proposed one hypothetical mechanism for strigolactones action in which a protein that promotes PIN1 endocytosis is sequestered in the nucleus by a protein that is targeted for degradation in a strigolactones, D14 and MAX2-dependent manner.

Although it has been shown that strigolactones inhibited branching by regulating *BRC1* expression and by affecting the accumulation of PIN1 protein at the plasma membrane, the detailed mechanism remains unclear. In particular, the target genes of strigolactones key signal components (D3, D14 and D53) or genes that can directly interact with these key signal components have rarely been found.

The identification of *Oryza sativa* MADS-box transcription factor 57 (*OsMADS57*) in rice provided additional information about the complexity of strigolactones in regulating branching. Guo et al.[91] found that *OsMADS57* could regulate tillering by interacting with *OsTB1* and *D14*. *OsMADS57* can suppress the expression of *D14* by directly targeting site 1 [CTTTAAAAAG] and site 2 [CATTAAAAAG] CArG *cis*-elements in the *D14* promoter. Meanwhile, *OsMADS57* was shown to be capable of interacting directly with *OsTB1* and this reduced *OsMADS57* and inhibited *D14* transcription. In addition, the *OsMADS57* gene is a target of *miR444a*. These results indicate that *OsMIR444a*-regulates *OsMADS57* and *OsMADS57* may play a role in the strigolactones signaling pathway and control tillering by interacting with OsTB1/FC1 proteins, mediated by D14, establishing the link between *OsMIR444a*, *OsMADS57*, *OsTB1*, *D14* and tillering and showing that it is not the sole mechanisms for *BRC1 to* act downstream of strigolactones to regulate shoot branching in the strigolactones pathway.

In *Arabidopsis*, *BES1* (brassinosteroid transcriptional effector) promotes shoot branching. BES1 interacts with MAX2 and be degraded by the MAX2-dependent pathway, with AtD14 and strigolactones promoting this degradation. Further studies demonstrated that BES1 acts downstream of MAX2 and promotes branching by acting as a negative regulator of the strigolactones signal[92]. However, the relationship between *BRC1* and *BES1* has not been examined. It is important to determine whether *BRC1* and *BES1* function in the same pathway to regulate shoot branching by genetic approaches.

2.3 Integration of systemic and local signals in regulation of shoot branching

Shoot branching is controlled by endogenous and environmental stimuli. Numerous studies have explored regulatory networks in branching, which involve long-distance systemic signaling mediated by hormones and environmental stimuli. The hormonal regulation network has been established and a number of genes involved in this long-distance systemic regulation have been identified. However, our understanding of the regulation mechanism within axillary buds remains limited. Furthermore, little is known about the molecular cascade of environmental stimuli that regulating branching presently. What is the detailed mechanism of hormone regulatory networks mediating environmental stimuli and eventually regulating the axillary bud?

2.3.1 Local regulation — *BRC1* is a key player in the integration of branching within axillary bud

BRC1 is the only discovered transcription factor that acts as an integrator of branching signals within axillary buds. This gene was first discovered in maize[88], then in *Arabidopsis* (*BRC1* and *BRC2*)[85], sorghum[93], wheat[94], peas[87], tomato[95], chrysanthemum[96] and rice (*FC1*)[86], and is functionally conserved. *BRC1* is expressed in developing buds where it arrests bud development[85].

2.3.2 *BRC1* responds to hormonal stimulation

Auxin cannot directly regulate the transcription of *BRC1*, but *BRC1* is essential in controlling apical dominance[85]. Also, *BRC1* is induced by strigolactones and cytokinin. In the axillary bud of peas, GR24 can induce *PsBRC1* upregulation and cytokinin induces *PsBRC1* downregulation[87]. Cytokinin regulates *PsBRC1* at the transcriptional level, independent of strigolactones[87]. In rice, GR24 treatment does not significantly induce *FC1* mRNA expression, suggesting that transcriptional induction may not be the mechanism by which strigolactones affect *FC1* functioning[86].

BRC1 (*FC1*) acts downstream of the strigolactones shoot branching regulatory pathways and reductions in *BRC1* mRNA levels in *max* mutants and the induction of *BRC1* expression by strigolactones suggest that *BRC1* functions downstream of the strigolactones signal. *FC1* certainly functions as a regulator of tiller outgrowth downstream of the strigolactones signal and is required for strigolactones to inhibit tiller outgrowth[78,85–87].

Finally, *BRC1* is the common target of strigolactones and cytokinin. The study by Dun et al.[97] showed that strigolactones and cytokinin could act directly in buds to control their outgrowth. Combined treatment with BA (cytokinin) and GR24 resulted in an intermediate level of *PsBRC1* expression, indicative of the antagonistic action of strigolactones and cytokinin in control of bud outgrowth. Cytokinin may converge at a common point in the bud outgrowth regulatory pathway.

However, some evidence suggested that *FC1* is not dependent on strigolactones. In rice, GR24 treatment does not significantly induce *FC1* mRNA expression, indicating that *FC1* may be partially dependent on the strigolactones approach to regulate branching[86]. Different from *BRC1* in peas and *Arabidopsis* that acts downstream of strigolactones and can be induced by strigolactones, *FC1* is not induced in rice seedlings by strigolactones treatment nor is its expression reduced in strigolactones-deficient mutants of rice. These results indicate that *FC1* expression may be less directly coupled to strigolactones signaling in rice[90].

2.3.3 Environmental signal regulation — importance of light quality in regulation of shoot branching

Light is an important environmental signal for plant morphogenesis, while light quality (the ratio of red to far red, R: FR) is an important environmental factor regulating

shoot branching[98]. Low R:FR often triggers a series of growth changes in plants, such as a reduction in the number of branches, collectively known as the shade avoidance syndrome[99]. The majority of studies on the regulation of light on shoot branching have focused on R:FR.

2.3.3.1 *BRC1* integrates light quality signals to regulate axillary bud activity

Kebrom et al.[93] first hypothesized that active phyB (Pfr) suppresses expression of the *SbTB1* gene, thereby inducing bud outgrowth, whereas environmental conditions that inactivate phyB increase the expression of *SbTB1*, thereby suppressing bud outgrowth in response to light signals[93,100]. In *Arabidopsis*, when under high-density planting conditions, the *BRC1* expression level increased in axillary buds; meanwhile, *BRC1* is essential for inhibition of bud outgrowth under high-density planting conditions[85]. González-Grandío et al.[101] explored the mechanism of *BRC1* in promoting bud dormancy in response to low R:FR in *Arabidopsis* and showed that *BRC1* can respond to such changes in light quality. Meanwhile, *BRC1* transcription is negatively controlled by *PHYB* and *BRC1* may respond to the R:FR signal through the *PHYB* pathway. This study also showed that *BRC1* is required for branch suppression in response to shade. Transcriptome data show that, in the shade, a large number of cell cycle- and ribosome-related genes are downregulated. The promoter region of downregulated genes is rich in TCP binding sites. Using *BRC1* estradiol-inducible lines further showed that some downregulated cell cycle-related genes are regulated by *BRC1*. The above results demonstrate the association between *BRC1* with light quality and branching regulation.

2.3.3.2 Strigolactones involvement in the shade response process

Plants detect R:FR changes through phytochrome B (PHYB). Inactive PHYB tends to inhibit axillary bud outgrowth and reduce the number of branches[93,102]. Under high R:FR, the phyB mutant showed reduced branching. However, at high R:FR ratios, the phenotype of *max2phyB* double mutants and *max4phyB* double mutants are the same as strigolactones mutants, showing a high branching phenotype and thereby inhibiting the less branching phenotype of *phyB*. These results indicate that strigolactones might act downstream of the *PHYB* pathway and strigolactones are required for the response to R:FR changes[102]. The studies further showed that *AXR1, BRC1, BRC2, MAX2* and *MAX4* are required in the branching regulation influenced by phytochrome. *Max2* participates in hypocotyl elongation mediated by *PHYB*[74] and *BRC1* and *BRC2* participate in the branching regulation response mediated by phytochrome through different pathways[101,102]. Tao et al.[103] proposed the following hypothesis concerning the relationship between *PHYB* mediated signals in relation to R:FR changes and strigolactones. Taking into account the fact that under shade conditions, auxin content will increase and that auxin positively regulates strigolactones synthesis, it is possible that at high R:FR, strigolactones synthesis may be suppressed by *PHYB*, while at low R:FR, this inhibition is released and strigolactones content increases and contributes to the shade response phenotype.

This study connects environment stimulation (decaptiation and shade) with hormone (strigolactones, auxin, cytokinin) and local regulation (*BRC1)*. Based on this, we describe a network that integrates the quality signal, hormones and local factors to regulate shooting branching (Fig. 3), but more details of the mechanisms require further study.

Figure 3a based on the auxin transport channel hypothesis. In this model, the output of auxin from axillary bud to the main stem is an important factor allowing axillary bud outgrowth and this process is negatively regulated by the PAT stream in the stem. In this model, cytokinin (CK) exerts its effect on branching by affecting the biosynthesis of auxin and negatively regulating the PAT stream in the stem. Meanwhile, strigolactone (SL) inhibits the PAT stream in the main stem by decreasing the accumulation of PIN proteins in the cell membrane. After decapitation, sugar demand in the shoot tip disappears and sugars are rapidly redistributed over large distances and accumulated in axillary buds within a specific frame. When the sugar content of the axillary buds passes a threshold, the buds are released. Following auxin export in axillary buds at special positions, the inhibitory effect of *BRC1* is reduced or absent and the cell cycle machinery will continue to function, stimulating the special position axillary bud to grow. In contrast, when auxin in axillary buds cannot be transported, the cell cycle will be in a stable stationary state due to the action of a *TB1*-like (*BRC1*) gene, so that the axillary buds will not grow.

Figure 3b based on the second messenger hypothesis. The model is based on the hypothesis that cytokinin can modulate TCP transcription factors, while TCP transcription factors can regulate the cell cycle and the activity of axillary buds. In this model, auxin regulates branching by affecting the synthesis of cytokinin and strigolactones, which can enter the axillary bud. In axillary buds, cytokinin regulates the cell cycle machinery by regulating TCP transcription factors, such as inhibiting *TB1*-like (*BRC1*) expression. Strigolactones inhibits bud outgrowth by promoting *BRC1* expression through other mechanisms.

In both models, shade regulates branching by regulating auxin biosynthesis and through the auxin-signaling pathway mediated by *AXR1*. The third mechanism is the *PHYB* pathway, mediated by the strigolactones pathway. It is

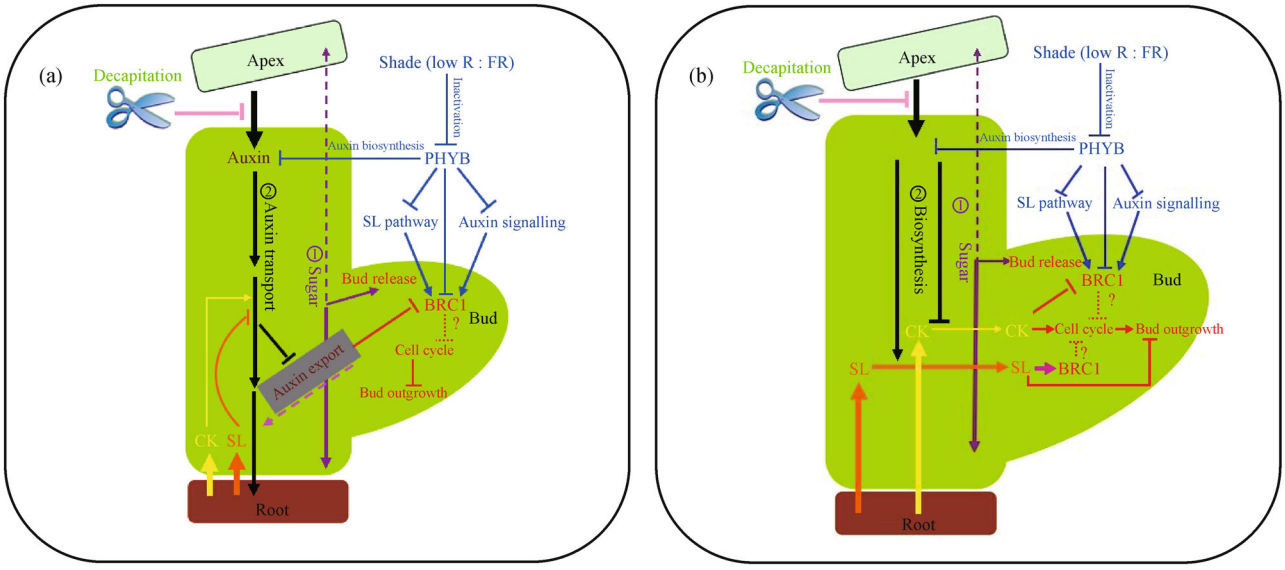

Fig. 3 Schematic representation of the systemic and local branching regulatory network model based on the hormonal and sugar control of branching. In these models, sugars are predominately responsible for the initial bud release and hormones determine bud outgrowth.

thought that shade inhibits bud outgrowth mediated by *PHYB*, but requires the full involvement of auxin, strigolactones signals and *BRC1* activity. Whether shade can affect sucrose (e.g., sucrose signaling, sucrose activity, or sucrose effects) remains unknown.

3 Perspectives and conclusions

The studies in strigolactones has greatly increased our knowledge of the mechanism of shoot branching. At the present, the main outline and content of the strigolactones synthetic pathway, and signal transduction network have been established. However, studies regarding strigolactones transport *in vivo* and its significance are limited. Numerous genes that participate in strigolactones signal transduction have been identified. However, the target genes regulated by strigolactones, especially the direct target remain unknown. Inside axillary buds, the connection between strigolactones and *BRC1* has been established. However, it remains unclear whether *BRC1* is the direct target of the strigolactones signal and the mechanisms of interaction require further study. In addition, although *BRC1* is known to be important in the regulation of branching in many species, several key questions still need to be addressed. Firstly, how does *BRC1* activate its downstream components to regulate branching? Secondly, whether the regulation mechanism of *BRC1* in shoot branching is different in various species? Identifying the target genes of *BRC1* is an interesting issue for future studies. In addition, it is important to explore how the hormone signals discussed above is integrated with the sucrose signal to regulate branching.

Overall, much progress has been made in understanding shoot branching regulatory mechanism. However, in horticultural plants, many significant branching phenomena occur that do not occur in model plants. Meawhile, there are many agricultural techniques used in horticultural cultivation for regulating branching, such as pruning, disbudding, branch-bending, layering, grafting and peeling. Can these phenomena be fully explained by the existing branching theory? How can we use the branching mechanism formed in model plants to guide horticultural cultivation? Answering these questions and researching the conservation or diversity of branching pathways in multiple horticultural plants are worthy undertakings for horticultural scientists in the future.

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This article is a review and does not contain any studies with human or animal subjects performed by any of the authors.

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