



REVIEW

The Axillary Bud's Decision Maker: How Strigolactones Dictate Plant Shoot Branching Patterns

Rui Liu¹, Fengjuan Lu¹, Guimin Tong¹, Han Wang² and Yingbi Xu^{1,*}

¹Department of Ecology, Hebei University of Environmental Engineering, Qinhuangdao, China

²Changli Institute of Pomology, Hebei Academy of Agriculture and Forestry Sciences, Qinhuangdao, China

*Corresponding Author: Yingbi Xu. Email: xuyingbi520@163.com

Received: 25 June 2026; Accepted: 10 September 2026; Published: 19 October 2026

ABSTRACT: Plant morphology is a fundamental horticultural trait that plays a critical role in plant growth and development. As a core determinant of plant morphology, shoot branching significantly influences light capture efficiency, nutrient utilization capacity, and stress tolerance. The developmental process of shoot branching begins with the formation of axillary buds, a process tightly coordinated by the shoot apical meristem (SAM) and axillary meristems (AM). Axillary buds outgrowth is regulated by complex interactions among genetic factors, photoperiod, environmental cues, and phytohormones such as auxin, cytokinin (CK), and strigolactones (SLs). As a class of carotenoid-derived terpenoid lactone phytohormones, SLs have been identified as key negative regulators of axillary buds outgrowth and have remained a prominent research focus in plant developmental biology over the past decades. This review systematically summarizes the core pathways of SLs biosynthesis and signal transduction and dissects the molecular basis underlying SLs-mediated control of axillary bud development and shoot branching patterning. We highlight the evolutionary conservation of core SLs biosynthetic and signaling components across diverse plant species, alongside their lineage-specific functional divergence. We further synthesize current evidence regarding the emerging, and often debated, roles of SLs in woody perennial species. Additionally, we dissect the crosstalk regulatory networks between SLs and other phytohormones, with a focus on their synergistic and antagonistic interactions. Finally, we outline key unresolved molecular questions, including the mechanistic basis of SLs transport, the PAT-independent regulatory pathway, and the tissue- and developmental stage-specific coordination of SLs signaling. We also critically evaluate the inherent experimental limitations of the widely used synthetic SLs analog GR24. Addressing these knowledge gaps is critical to deepening the mechanistic understanding of SLs-mediated branching regulation and unlocking translational applications for crop ideotype breeding.

KEYWORDS: Plant morphology; branching; phytohormones; strigolactones; hormonal crosstalk

1 Introduction

Shoot branching, a key component of plant morphology, exerts profound effects on plant growth, development progression, photosynthetic efficiency, and reproduction [1,2]. Divergent branching patterns reflect the adaptive strategies of plants to fluctuating environmental conditions [3]. Branching initiation depends on the formation of axillary buds, a process tightly linked to the shoot apical meristem (SAM) [4]. The SAM undergoes continuous development and differentiation, giving rise to new meristematic tissues known as axillary meristems (AM) [5]. The developmental trajectory of an axillary bud can be divided into three key stages, axillary meristem initiation, bud dormancy, and bud outgrowth [6]. During the initiation stage, axillary meristems are established from a subset of cells in the leaf axil, a process governed by a suite

of transcription factors including REVOLUTA, WUSCHEL, and SHOOT MERISTEMLESS [7]. Once formed, axillary buds may enter a dormant state, in which they remain metabolically active but cease further growth until dormancy is released. The transition from dormancy to outgrowth represents a critical developmental switch, controlled by a dynamic balance between growth-promoting and growth-inhibiting signals [8]. Branching is regulated by intricate crosstalk among transcriptional regulation, phytohormone homeostasis, and environmental signals [9,10]. Among plant hormones, auxin, strigolactones (SLs) and cytokinins (CKs) act as the major regulators governing plant shoot branching [11]. The different hormones interact and crosstalk within the branching regulatory network, thereby shaping the final phenotype [12].

Auxin is one of the earliest identified phytohormones known to regulate branching [13]. In *Arabidopsis thaliana*, auxin is mainly synthesized in the shoot apex via the TAA/YUCCA pathway [14]. It is subsequently transported downward through the stem to inhibit axillary bud outgrowth, thereby establishing the initial signal responsible for apical dominance [15]. This process plays an essential role in maintaining vertical growth and plant architectural development [16]. Regarding the mechanism of auxin inhibiting plant shoot branching, there are two different hypotheses: the canalization hypothesis and the second messenger hypothesis. The second messenger hypothesis posits that auxin exerts its regulatory function by modulating mobile signals in the stem. These signals act as second messengers to relay intracellular information, thereby directly regulating axillary bud activity [16]. CKs and SLs are the most widely accepted downstream second messengers of auxin, with their auxin-modulated biosynthesis indirectly controlling axillary bud outgrowth [17].

Cytokinin plays a pivotal role in plant growth and development, which relieves the auxin-established apical dominance by modulating the homeostasis among multiple endogenous phytohormones in plants, thereby promoting shoot branching. Studies in tobacco (*Nicotiana tabacum*) have revealed that overexpression of the *ISOPENTYL TRANSFERASE (IPT)* gene significantly elevates endogenous cytokinin levels. Along with the increase in cytokinin content, tobacco exhibits a marked attenuation of apical dominance and a concomitant increase in shoot branching number [18]. Conversely, *CYTOKININ OXIDASE/DEHYDROGENASEs (CKXs)* catalyze cytokinin degradation and overexpression of *CKXs* genes results in severe cytokinin deficiency, thereby leading to a significant reduction in shoot branching number [19].

SLs is a category of carotenoid-derived phytohormone, encompassing both natural products and synthetic analogues [20]. Beyond the well-characterized regulatory roles of auxin and CK in plant shoot branching, SLs, a class of carotenoid-derived terpenoid phytohormones, have been widely recognized as a core negative regulator of axillary bud outgrowth and shoot architecture formation [21]). Accumulating evidence has demonstrated that SLs do not function independently in branching control, but rather integrate into the core regulatory network of shoot branching via extensive, multi-level synergistic and antagonistic crosstalk with other endogenous phytohormones, which collectively fine-tune the initiation, activity and outgrowth of axillary buds to shape the final branching phenotype [22]. The most well-documented hormonal interaction for shoot branching regulation occurs between strigolactones (SLs) and auxin. These two hormones form a bidirectional regulatory loop to modulate this process. On one hand, auxin upregulates the expression of SLs biosynthetic genes, including *Carotenoid Cleavage Dioxygenase 7 (CCD7)* and *CCD8*, in an *AUXIN RESISTANCE1 (AXR1)*-dependent manner, in which SLs act as a second messenger to mediate auxin-dependent inhibition of shoot branching [23–25]. On the other hand, SLs repress the expression of PIN-FORMED (PIN) auxin efflux carriers, which disrupts polar auxin transport (PAT) and auxin canalization from axillary buds, thus suppressing bud activation and outgrowth [26,27]. Genetic evidence from pea mutants also supports an additional PAT-independent regulatory pathway for SLs-mediated branching inhibition [28]. In parallel, SLs and CK exhibit a well-established antagonistic relationship in the regulation

of axillary bud outgrowth, with bidirectional regulatory interplay occurring at the levels of hormone biosynthesis, long-distance root-to-shoot transport and catabolism [29]. SLs modulate CK accumulation in stems and axillary buds via regulating its root-to-shoot translocation and inducing the expression of CK-degrading *CYTOKININ OXIDASE/DEHYDROGENASE* (*CKX*) family genes, and the dynamic balance between SLs and CK in axillary buds has been identified as a key determinant of bud fate and branching phenotype [17,30], though the reverse regulatory mechanism of CK on SLs transport and signal transduction remains largely uncharacterized to date.

In addition to the well-established roles of auxin, cytokinin, and SLs in branching regulation, other phytohormones also contribute to this process. Gibberellins (GAs) are key promoters of stem elongation and overall vegetative growth [31]. Accumulating evidence indicates that GAs antagonize SLs-mediated branching inhibition, partly through the conserved GID1-DELLA signaling module and through shared downstream regulatory targets [32]. Brassinosteroids (BRs) regulate cell elongation and division, and recent studies have revealed that BR signaling converges with the SLs pathway at the *MAX2-D53/BES1* node. Specifically, the core BR signaling transcription factors *BRASSINAZOLE-RESISTANT 1* (*BZR1*) and *BRI1-EMS-SUPPRESSOR 1* (*BES1*) are targeted for ubiquitination and degradation by the key SLs signaling E3 ubiquitin ligase *MORE AXILLARY BRANCHING 2* (*MAX2*), thereby co-regulating the expression of the branching master regulator *BRC1* [33,34]. Abscisic acid (ABA), a classical stress hormone, is involved in maintaining bud dormancy and exhibits complex synergistic and antagonistic interactions with SLs at both biosynthetic and signaling levels, depending on species and growth conditions [35]. Notably, both hormones share a common carotenoid biosynthetic precursor, all-trans- β -carotene, which lays the material foundation for their crosstalk [36,37]. These hormonal pathways, together with auxin, CK, and SLs, form an integrated regulatory network that fine-tunes shoot branching in response to both internal developmental cues and external environmental inputs. Although these findings have laid a fundamental framework for understanding SLs-mediated multi-hormone crosstalk in branching regulation, the detailed molecular mechanisms and the core convergent regulatory nodes that integrate multiple hormonal signals remain to be fully elucidated.

Building on this foundational framework of SLs-mediated branching regulation, this review provides a comprehensive and systematic overview of the current state of SLs research in plant architectural control. We first summarize the core biosynthetic pathways and signal transduction cascades that underpin SLs functions in plant development. We then focus on dissecting the multi-level synergistic and antagonistic crosstalk between SLs and other major phytohormones in shoot branching regulation, with detailed elaboration of the established molecular mechanisms and key regulatory nodes of these hormonal interactions. Finally, we propose promising priority research directions and forward-looking perspectives in this field, aiming to provide a systematic theoretical reference for deciphering the holistic regulatory network of shoot branching, and to facilitate the molecular breeding of crop varieties with ideal plant architecture.

2 Biosynthetic Pathways and Key Genes of SLs

SLs is a class of terpenoid lactone hormone derived from carotenoids, which biosynthesis initiates via the methylerythritol phosphate (MEP) pathway in plant plastids, followed by a series of sequential enzymatic reactions to generate diverse SLs molecules [38]. The core and conserved biosynthetic steps of SLs are dominated by several key rate-limiting enzymes, which jointly drive the formation of the basic SLs skeleton, laying a molecular foundation for the subsequent structural diversification and functional differentiation of SLs.

The initial catalytic step of SLs biosynthesis is mediated by β -carotene isomerase *DWARF27* (*D27*), which specifically catalyzes the configurational transition of all-trans- β -carotene to 9-cis- β -carotene, laying the groundwork for the subsequent cleavage reaction [37]. The stereospecific substrate is then recognized and cleaved by *CCD7*, producing 9-cis-10'-apocarotenal and β -ionone as two primary cleavage products [39]. Among them, 9-cis-10'-apocarotenal is further processed by another key cleavage enzyme *CCD8*, undergoing oxidative cleavage and cyclization reactions to generate carlactone, the universal common precursor of all canonical and non-canonical SLs [40,41].

After the formation of the conserved precursor carlactone, species-specific downstream metabolic pathways drive the structural diversification of SLs, and more than 25 natural SLs molecules have been identified and isolated in plants to date [42]. Taking model plants as typical examples, distinct catalytic mechanisms exist in the downstream modification of carlactone. In *Arabidopsis*, the cytochrome P450 enzyme encoded by the *MORE AXILLARY BRANCHING 1* (*MAX1*) gene catalyzes carlactone to form carlactonoic acid (CLA), which is further modified by methyltransferase to generate methyl carlactonoate (MeCLA), the major active SLs component in *Arabidopsis* [43]. In rice (*Oryza sativa*), two homologous *MAX1* enzymes, namely *Os900* and *Os1400*, perform sequential catalytic reactions, ultimately synthesizing 4-deoxyorobanchol (4DO) and orobanchol, the two main canonical SLs products in rice [44] (Fig. 1).

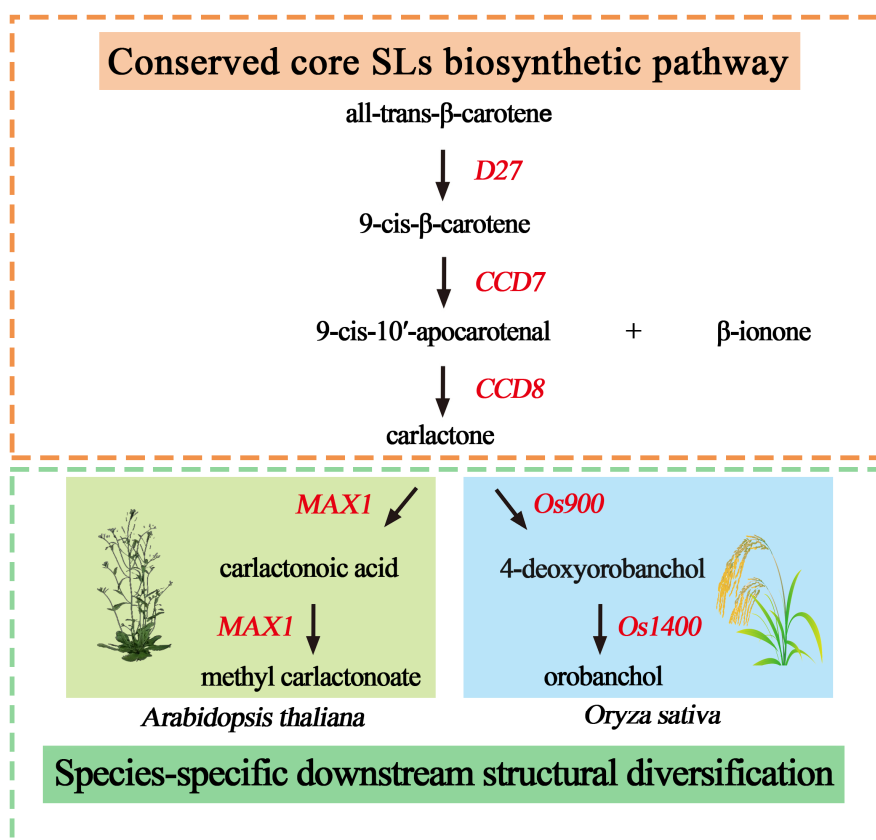


Figure 1: Schematic diagram of the SLs biosynthetic pathway in plants. The conserved core pathway generates the universal SLs precursor carlactone from all-trans- β -carotene through sequential catalysis by *D27*, *CCD7* and *CCD8*. Downstream species-specific structural diversification is illustrated with two model plants: *Arabidopsis thaliana* (*MAX1*-mediated MeCLA synthesis) and rice (*Os900*/*Os1400*-mediated orobanchol synthesis).

3 Signal Perception and Canonical Transduction Pathway of SLs

SLs exhibit an evolutionarily conserved signal transduction mechanism that centrally relies on the ubiquitin-26S proteasome system (UPS), as extensively characterized in the model plants *Arabidopsis thaliana* and *Oryza sativa* [45]. This pathway operates through the sequential activation of E1 ubiquitin-activating enzymes, E2 ubiquitin-conjugating enzymes, and E3 ubiquitin ligases to mediate the polyubiquitination modification and subsequent degradation of target proteins, thereby exerting transcriptional control over the expression of downstream target genes [46]. SLs signal perception is initiated by DWARF14 (D14), an α/β -fold hydrolase superfamily protein [47]. *D14* and its orthologs are well-established as the bona fide receptors of SLs, harboring an evolutionarily highly conserved Ser-His-Asp catalytic triad within their protein structure [48]. This receptor recognizes SLs molecules specifically via its hydrolase active center, thereby triggering the activation of downstream signal transduction cascades [49]. The binding of SLs to *D14* and the initiation of signaling are dependent on the specific hydrolysis of SLs molecules [50]. Notably, the SLs encompasses a structurally diverse group of natural compounds that can be classified into canonical and non-canonical SLs based on their chemical architecture. Canonical SLs, such as strigol, orobanchol, and sorgolactone, possess a complete ABCD ring system. By contrast, non-canonical SLs lack one or more of the A, B, or C rings but retain the enol ether-D ring moiety, which is essential for biological activity [51]. *D14* cleaves the enol ether bond of SLs, splitting the molecule into a tricyclic lactone (ABC ring) and a butenolide derivative (D-OH, the D-ring moiety) [52]. The D-ring moiety then forms a covalently linked intermediate molecule (CLIM) with *D14* via covalent bonding, which simultaneously drives a conformational switch of *D14* from an open state to a closed state, providing the structural basis for its subsequent interaction with downstream signaling components [53]. Collectively, these findings define the dual function of the *D14* receptor: it not only catalyzes the hydrolysis of SLs molecules, but also acts as the specific sensor for SLs signals [54].

However, a critical caveat of most functional studies on SLs signaling is that they have relied heavily on the synthetic SLs analog GR24, particularly its racemic mixture rac-GR24, which cannot fully recapitulate the authentic biological activity of endogenous SLs. Specifically, rac-GR24 contains multiple stereoisomers capable of activating both *D14* and *KAI2*, and this off-target cross-activation introduces confounding effects that compromise experimental data interpretation. *KAI2* is a paralogous α/β -hydrolase receptor that shares considerable structural similarity with *D14*, including a conserved Ser-His-Asp catalytic triad and an α/β -hydrolase fold [55,56]. However, its ligand-binding pocket is smaller and more constricted, enabling it to perceive karrikins and an unidentified endogenous ligand (*KAI2* ligand, KL) while excluding canonical strigolactones, which are too large to fit efficiently. Although the *KAI2* pathway converges with SLs signaling at the level of MAX2 and SMXL proteins, it regulates a distinct set of developmental processes and its role in shoot branching is generally considered minor or indirect compared with the *D14*-SLs pathway [51,57]. Nevertheless, altered shoot branching phenotypes have been reported in *KAI2* mutants or upon karrikin treatment, suggesting potential crosstalk or partial functional overlap between the two receptor pathways. Importantly, the unresolved identity of the endogenous KL has significant implications for the biological definition of SLs. If KL is structurally related to SLs, some activities previously attributed to SLs could instead be mediated by *KAI2*-KL signaling. Therefore, receptor-specific analogs, genetic separation of *D14* and *KAI2* functions, and identification of endogenous KL are essential to clarify the distinct and overlapping roles of these two signaling systems in shoot branching regulation.

Another indispensable core component for faithful SLs signal transduction is the leucine-rich repeat (LRR)-containing F-box family protein, encoded by MAX2 and its orthologs across land plants [58]. In the presence of SLs, the conformationally activated *D14* directly interacts with MAX2, which further

drives the assembly of the Skp1-Cullin-F-box (SCF) E3 ubiquitin ligase complex. The fully assembled SCF complex specifically recruits the core transcriptional repressors in the *SLs* signaling pathway: members of the *SUPPRESSOR OF MAX2 1-LIKE* (*SMXL*) family, namely *SMXL6/7/8* [59]. The SCF complex mediates the polyubiquitination of these *SMXLs* proteins, and the polyubiquitin-tagged *SMXLs* are subsequently recognized and degraded via the 26S proteasome pathway [60]. This degradation event relieves the transcriptional repression of *SL*-responsive target genes, thus enabling the execution of the biological regulatory functions of *SLs* [61]. In parallel, *SLs* can directly activate the transcription of *BRANCHED 1* (*BRC1*), *TEOSINTE BRANCHED 1* (*TCP1*) and *PRODUCTION OF ANTHOCYANIN PIGMENT 1* (*PAP1*), to coordinately regulate multiple biological processes including shoot branching, leaf development, and anthocyanin biosynthesis [62] (Fig. 2).

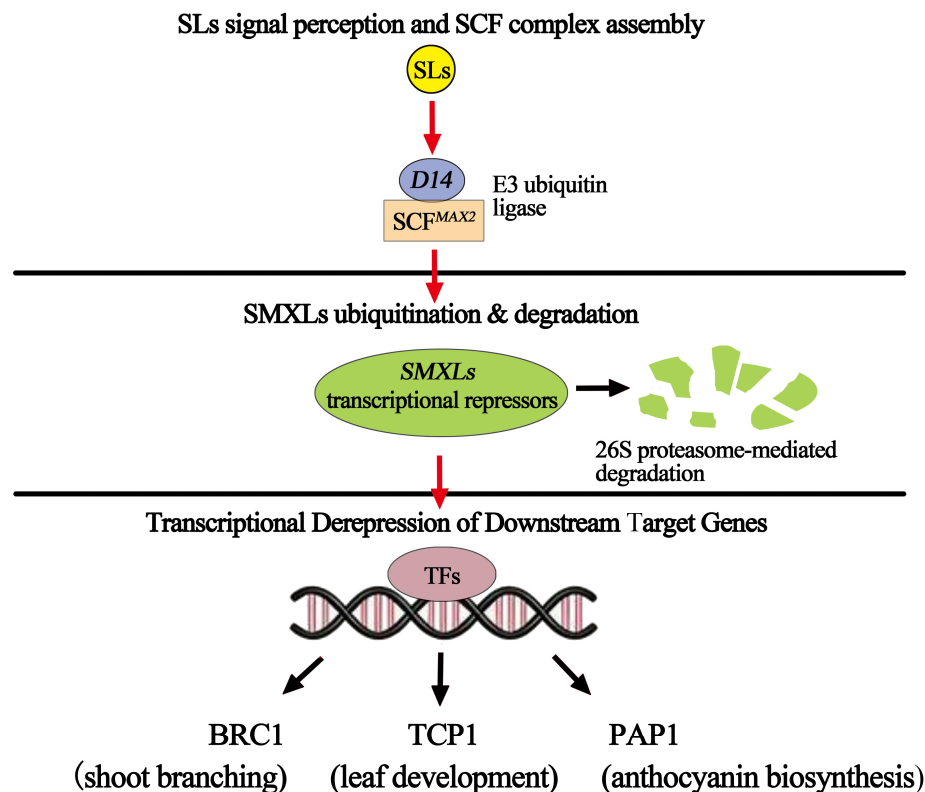


Figure 2: Schematic model of the core *SLs* signal transduction pathway in plants. *SLs* are perceived by the *D14* receptor, which recruits the SCF-MAX2-E3 ubiquitin ligase complex to trigger polyubiquitination and 26S proteasome-mediated degradation of *SMXL6/7/8* transcriptional repressors. This process releases transcriptional repression of downstream target genes *BRC1*, *TCP1* and *PAP1*, and regulates multiple biological processes such as shoot branching.

In *Arabidopsis*, *SMXL6*, *SMXL7*, and *SMXL8* function as co-orthologs of rice D53, redundantly repressing *SLs* signaling through the SCF-MAX2-mediated ubiquitination and 26S proteasome degradation pathway. *SMXL6* exerts a dual function as both a DNA-binding transcription factor and a transcriptional repressor, which interacts with MYB transcription factors via its C-terminal domain to repress downstream target genes, while also directly targeting the promoters of itself, *SMXL7*, and *SMXL8* through its N-terminal DNA-binding domain to auto-regulate transcription and maintain signaling homeostasis [62]. Beyond

this core framework, the *SMXLs* family exhibits functional redundancy, species-specific divergence, and additional non-canonical signaling modules (Table 1).

Table 1: Comparison of strigolactone signaling pathway gene functions among representative plant species.

Species	<i>D14</i>	<i>MAX2</i>	<i>SMXL</i>	Key Functional Outputs
<i>Arabidopsis thaliana</i>	<i>AtD14</i>	<i>AtMAX2</i>	<i>SMXL6/7/8</i>	Shoot branching inhibition; hypocotyl elongation; lateral root density
Rice (<i>Oryza sativa</i>)	<i>OsD14</i>	<i>OsMAX2</i>	<i>OsD53</i>	Tillering inhibition; mesocotyl elongation
Pea (<i>Pisum sativum</i>)	<i>PsD14</i>	<i>PsMAX2</i>	<i>PsSMXL7</i>	Shoot branching; nodulation
Soybean (<i>Glycine max</i>)	<i>GmD14</i>	<i>GmMAX2</i>	<i>GmSMXLs</i>	Shoot branching; rhizobia symbiosis; nodulation
Wheat (<i>Triticum aestivum</i>)	<i>TaD14</i>	<i>TaMAX2</i>	<i>TaSMXLs</i>	Regulates tillering; SLs biosynthesis genes upregulated in oligo-tillering mutants
Poplar (<i>Populus</i> spp.)	<i>PtD14</i>	<i>PtMAX2</i>	<i>PtSMXLs</i>	Shoot branching; stress responses
Apple (<i>Malus domestica</i>)	<i>MdD14</i>	<i>MdMAX2</i>	<i>MdSMXLs</i>	Branching architecture; anthocyanin biosynthesis; stress responses
Strawberry (<i>Fragaria</i> spp.)	<i>FaD14</i>	<i>FaMAX2</i>	<i>FaSMXLs</i>	Branch crown regulation; runner formation

4 Regulatory Roles of SLs in Shoot Branching Regulation

The core molecular framework of SLs biosynthesis and signal transduction, as well as its crosstalk with other phytohormones, collectively underpins the regulatory effects of SLs on plant shoot branching. As a core negative regulator of shoot branching, SLs exert precise control over the entire developmental process of axillary buds, modulate branching patterns across different plant taxa, and link environmental signals to plant architectural plasticity [63].

For the early stage of AM initiation, SLs act as a repressor to restrict the formation of new axillary meristems in leaf axils [64]. Early studies on SLs-deficient or signaling-insensitive mutants in *Arabidopsis* and rice, found that these mutants not only produce more outgrowing branches, but also form axillary buds in leaf axils that normally do not generate buds in wild-type plants [65,66]. This phenotype proves that SLs do not only function at the late stage of bud growth, but also limit the initiation of AMs at the early developmental stage. Recent studies further revealed that SLs regulate AM initiation through the *SMXLs*-dependent signaling pathway, by targeting key meristem regulatory genes including TCP family and WUSCHEL-related homeobox (WOX) family members [62]. For the late stage of axillary bud development, the well-characterized core function of SLs is to maintain axillary bud dormancy and inhibit its outgrowth [67]. When SLs biosynthesis or signaling is blocked, the inhibition on axillary buds is fully removed. Dormant buds are reactivated, and a large number of lateral branches grow out, forming a typical highly branched phenotype [68]. This regulatory effect is highly stable across most land plants, and is the most direct and prominent phenotypic effect of SLs on plant development. However, whether the individual *SMXL6/7/8* repressors exert stage-specific functions during these two developmental phases remains largely unresolved. Current evidence is derived almost exclusively from constitutive loss-of-function mutants, which simultaneously affect both AM initiation and bud outgrowth, making it difficult to assign distinct roles to specific *SMXLs* members at each stage. Moreover, tissue- or stage-specific manipulation of *SMXLs* activity has not been systematically performed, and it is unclear whether the same *SMXL* proteins regulate different downstream targets at initiation versus outgrowth.

The inhibitory role of SLs on shoot branching is evolutionarily conserved across land plants, but there are also species-specific functional divergences, which are closely related to the growth habits and breeding goals of different plants. In cereal crops such as rice, wheat and maize, SLs mainly regulate the formation of tillers, a special type of basal branching that directly determines grain yield [69–71]. In rice, SLs-deficient mutants produce a large number of ineffective tillers, leading to severe lodging and reduced yield, which has made the SLs pathway a key target for ideal plant architecture breeding in cereal crops [72]. Breeders can fine-tune tiller number by regulating the expression of SLs biosynthesis or signaling genes, to breed varieties with moderate tiller number, lodging resistance and high yield potential. In horticultural crops, SLs-mediated branching regulation has important application value in improving ornamental quality and simplifying cultivation management [73]. For example, in cut rose, the number of lateral branches directly determines the number of flowers [74]. Exogenous application of SLs analogs can inhibit excessive branching, while SLs biosynthesis inhibitors can promote branching to increase flower yield [63,73,75]. In grape, SLs regulate the growth of summer lateral shoots, which affect the ventilation and light transmission of the vineyard [76]. Regulating the SLs pathway can effectively reduce the workload of manual summer pruning. In tomato, SLs not only regulate the number of vegetative branches, but also affect the formation of reproductive branches, thus regulating final fruit yield [77]. In woody plants, the regulatory role of SLs on branching is more complex due to their perennial growth habit. It should be clarified that while the inhibitory effect of SLs on axillary bud outgrowth is well-documented in annual plants, relevant conclusions drawn from perennial woody species remain controversial and require further cross-species validation. Current studies on poplar and apple indicate that SLs still tend to act as branching inhibitors, but their regulatory effect is affected by the seasonal growth cycle and the apical dominance of perennial branches [78]. In poplar, overexpression of the SLs signaling gene *PtSMXL7* can significantly reduce the number of lateral branches, which is helpful for cultivating fast-growing timber varieties with straight trunks [79]. Given that the inhibitory role of SLs in shoot branching remains controversial in perennial woody species, conclusions derived from woody-plant materials should be interpreted with appropriate caution.

Plants can dynamically adjust their branching patterns according to environmental changes, to maximize resource utilization and improve stress tolerance. SLs act as a key signaling hub, linking external environmental cues to the internal regulation of shoot branching, and mediating the architectural plasticity of plants [80]. Nutrient availability is the most well-characterized environmental factor regulating SLs-mediated branching. When plants grow under low phosphorus or low nitrogen conditions, the biosynthesis of SLs in roots is significantly upregulated [81]. Elevated SLs are secreted into the rhizosphere to enhance arbuscular mycorrhizal symbiosis and nutrient uptake, while acropetally transported to shoots to suppress axillary bud outgrowth and reduce branching. This phenotypic adjustment reduces the nutrient consumption of excess branches, and helps plants adapt to low-nutrient environments.

Light conditions also regulate branching through the SLs pathway. Under shading conditions with a low red/far-red light ratio, which usually indicates intense inter-plant competition, plants inhibit lateral branching to maintain vertical growth for enhanced light capture [82]. Studies in cotton have shown that shading can upregulate the expression of SLs signaling gene *D14*, and enhance the inhibitory effect of SLs on axillary buds [83]. In addition, abiotic stresses such as drought and salt stress also regulate plant branching through the SLs pathway [84]. Under drought stress, SLs biosynthesis is induced, which inhibits excessive branching to reduce transpiration water loss, and improves the survival rate of plants under water deficit [85]. SLs signaling mutants usually show a drought-sensitive phenotype, which is partly due to their uncontrolled branching and increased water consumption [86]. Notably, most current studies on

environmental responses have been conducted under single stress treatments, and how the SLs pathway integrates multiple co-occurring environmental signals to precisely regulate shoot branching remains largely unclear.

Beyond shoot branching, SLs exert a broad spectrum of non-branching functions that are closely intertwined with plant growth and environmental adaptation. These include the regulation of root architecture, such as lateral root formation, root hair elongation, and primary root growth, as well as the promotion of arbuscular mycorrhizal symbiosis under nutrient-limiting conditions. SLs also participate in abiotic stress responses, including drought, salinity, and nutrient deficiency, and have been implicated in defense against pathogens and parasitic weeds [87]. Importantly, these non-branching functions are not isolated from branching control, they share common biosynthetic and signaling components and may compete for or redistribute resources, thereby indirectly influencing shoot branching outcomes. The functional connection between these processes and the core branching-regulatory role of SLs remains an important area for future investigation.

Taken together, SLs exert multi-level regulatory effects on plant shoot branching, from controlling the early initiation of axillary meristems to maintaining late bud dormancy, from conserved regulatory functions across species to mediating environmental adaptive plasticity (Fig. 3). These findings fully reveal the core role of SLs in plant architecture regulation, and lay a solid foundation for the agricultural application of the SLs pathway.

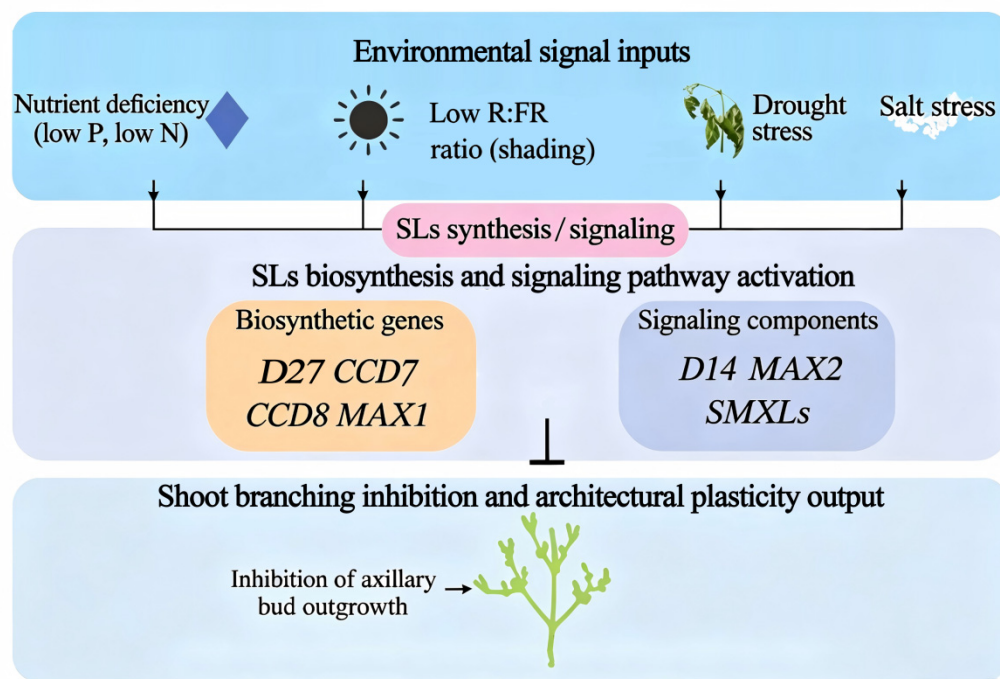


Figure 3: Schematic diagram of environmental regulation of SL-mediated shoot branching. Multiple environmental cues, including nutrient deficiency, low R:FR shading, drought and salt stress, converge to regulate the SLs biosynthesis and signaling pathway. Core biosynthetic genes and signaling components jointly mediate the inhibitory effect on shoot branching, and drive plant architectural plasticity in response to environmental changes.

5 Co-Regulatory Network of Strigolactones and Other Phytohormones in Shoot Branching

SLs do not independently regulate the entire process of shoot branching. Instead, they form a multi-level, finely tuned regulatory network with other endogenous phytohormones (Fig. 4). This network

integrates endogenous developmental signals and external environmental cues, and collectively determines the fate of axillary buds and the final branching phenotype of plants.

SLs share all-trans- β -carotene as a common biosynthetic precursor with ABA. Besides, SLs form a bidirectional regulatory loop with IAA, IAA promotes SLs biosynthesis, whereas SLs repress polar auxin transport. SLs antagonize CK primarily by promoting its degradation and inhibiting long-distance transport, though direct CK feedback on the SLs pathway remains poorly validated. GAs modulate endogenous SLs levels via the conserved GID1-DELLA cascade, while BRs and SLs converge on the shared signaling component MAX2 for coordinated regulation. Collectively, this multi-hormone network governs axillary bud development and shoot branching architecture.

Auxin is the core hormone that establishes plant apical dominance, and its regulatory effect on shoot branching is tightly linked to the SLs pathway [88]. The crosstalk between these two hormones forms a bidirectional regulatory loop, and they jointly modulate axillary bud outgrowth through two parallel and complementary regulatory branches, encompassing both a polar auxin transport (PAT)-dependent pathway and a PAT-independent inhibitory route.

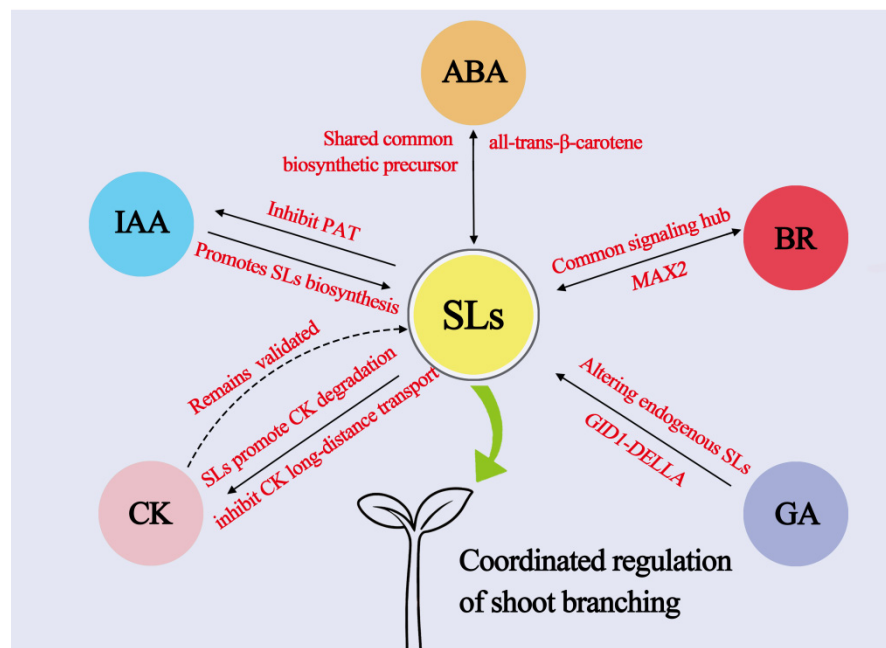


Figure 4: Schematic diagram of the crosstalk network between strigolactones (SLs) and other phytohormones in regulating shoot branching.

On one hand, auxin acts as an upstream signal to regulate SLs biosynthesis. Studies have confirmed that auxin can upregulate the expression of key SLs biosynthetic genes *CCD7* and *CCD8* in an *AUXIN RESISTANCE1* (*AXR1*)-dependent manner [23,25]. In this regulatory model, SLs act as a second messenger of auxin to mediate the inhibitory effect of auxin on axillary bud outgrowth. On the other hand, SLs can in turn regulate the polar auxin transport (PAT) system [27]. The activation and continuous growth of axillary buds depend on the establishment of a stable PAT stream from the bud to the main stem, a process called auxin canalization [89]. SLs can downregulate the expression of PIN-FORMED (*PIN*) auxin efflux carriers, the core components of the PAT system. Reduced *PIN* protein levels weaken the PAT stream, block auxin canalization from axillary buds, and finally inhibit bud outgrowth [26]. Notably, genetic studies in pea

have revealed that SLs can also inhibit axillary bud outgrowth through a PAT-independent pathway [28]. This means SLs regulate shoot branching through dual mechanisms, which together ensure the stability of their regulatory effect on branching. Beyond the dual regulatory mechanisms, SLs and their signaling components also employ dual long-distance transport modes to coordinate branching responses across the plant. Root-synthesized SLs can be transported acropetally to shoots, with evidence suggesting both xylem-mediated and phloem-mediated routes, although the relative contributions of each pathway remain debated. The perception and transduction of SL signals depend on its core receptor protein D14, an α/β -fold hydrolase that specifically binds SLs molecules and triggers downstream intracellular signaling cascades to suppress axillary bud outgrowth, more notably, this receptor protein itself has been shown to be mobile.

CK is the main promoting hormone for axillary bud outgrowth, and it acts antagonistically with SLs in regulating shoot branching [30]. Current evidence strongly supports SL-dependent regulation of CK homeostasis, whereas direct feedback from CK to SLs remains less established. On the one hand, SLs can induce the expression of *CYTOKININ OXIDASE/DEHYDROGENASE 9* (*CKX9*), which encodes a CK-degrading enzyme, thereby directly reducing CK accumulation in axillary buds [29]. On the other hand, SLs can regulate long-distance root-to-shoot CK transport, thus affecting CK content in aboveground tissues. In addition, SLs-deficient or SLs-signaling mutants in multiple plant species show altered CK biosynthesis and transport [90]. For example, tomato SLs-related mutants exhibit upregulated expression of the CK biosynthetic gene *SLIPT1* in stem nodes, which may compensate for reduced CK transport from roots [91], and the rice SLs-signaling *d53* mutant has significantly higher CK levels in the basal stem, which is associated with its high-tillering phenotype [92]. However, these mutant phenotypes and transcriptomic changes are largely correlative and primarily reflect regulation from SLs to CK; they do not by themselves establish direct CK-mediated feedback on SLs biosynthesis or signaling. Direct molecular evidence for CK-mediated regulation of the SL pathway remains limited. Therefore, although the dynamic balance between SLs and CK in axillary buds is a key determinant of bud fate, the reciprocal regulatory arm from CK to SL requires further direct experimental validation.

GAs are key regulators of plant vegetative growth, and emerging studies have revealed that they have crosstalk with SLs in regulating shoot branching, though the detailed regulatory network is not yet fully elucidated. Studies have confirmed that this regulatory process is mediated by the conserved GID1-DELLA signaling pathway, the core cascade of GA signal transduction [31]. This means the GA pathway can affect SL-mediated branching regulation by altering endogenous SLs levels in plants. Besides, SLs and GAs may share overlapping downstream regulatory pathways. Transcriptome analysis in *Arabidopsis* found that plants treated with both GA and the SL analog GR24 showed a superimposed transcriptional response in a set of shared target genes [32]. In addition, studies in rice found that both GA-deficient mutants with a high-tillering phenotype and SLs signaling mutants had upregulated expression of the same meristem regulatory gene *ORYZA SATIVA HOMEBOX 1* (*OSH1*) [93]. This suggests that the two hormones may have similar molecular mechanisms in regulating tillering and branching. However, there are also studies showing that GAs can antagonize some of the regulatory effects induced by SLs [94]. The specific mechanism of this antagonistic effect in shoot branching regulation still needs further experimental verification.

Brassinosteroids (BRs) are important regulators of plant growth and development [95], and their crosstalk with SLs in shoot branching regulation has been revealed in recent years. The core of this crosstalk is the convergence of the two hormone pathways on *MAX2*, the key signaling component of the SLs pathway. Studies have found that *MAX2* can directly interact with *BRASSINAZOLE-RESISTANT 1* (*BZR1*) and *BRI1-EMS-SUPPRESSOR 1* (*BES1*), the two core transcription factors in the BR signaling pathway [33,34].

SLs promote the ubiquitination and degradation of BES1/OsBZR1 via the *D14-DWARF 3 (D3)/MAX2* receptor complex, and the BES1/OsBZR1 and *DWARF 53 (D53)/SMXLs* act interdependently downstream of *SLs signaling* to co-repress the expression of BRC1/FC1, which are core switch genes governing axillary bud outgrowth [34]. This finding reveals that SLs and BRs co-regulate shoot branching by controlling the stability of the same set of core transcription factors, and provides a clear molecular mechanism for the crosstalk between the two hormones.

Absciscic acid (ABA) is the core hormone regulating plant stress response, and it also participates in the regulation of axillary bud dormancy and shoot branching [96]. Notably, SLs and ABA are both carotenoid-derived phytohormones, sharing all-trans- β -carotene as their common biosynthetic precursor [37]. This shared synthetic pathway lays the material foundation for their crosstalk. The crosstalk between SLs and ABA in shoot branching regulation shows both synergistic and antagonistic effects, depending on the plant species and growth conditions. On one hand, ABA can act upstream of the SLs pathway to regulate SLs biosynthesis and signal transduction. Studies have found that ABA-deficient plants have significantly reduced expression of SL biosynthetic genes and lower endogenous SLs content [97]. In addition, ABA can upregulate the expression of SLs-related genes to enhance plant stress tolerance, which is also linked to the regulation of shoot branching under stress conditions [98,99]. On the other hand, the two hormones also show antagonistic effects in regulating shoot branching. For example, studies in barley found that ABA promotes tiller formation by inhibiting the expression of SLs biosynthetic and signaling genes [97]. In addition, exogenous application of the SLs analog GR24 can inhibit ABA-induced physiological processes in some species, which also reflects the antagonistic relationship between the two hormones [36]. At present, the detailed molecular mechanism of the crosstalk between SLs and ABA in shoot branching regulation is still not fully elucidated. Further studies are needed to reveal the core regulatory nodes that link the two hormone pathways.

Taken together, SLs form a complex and precise co-regulatory network with auxin, cytokinin, gibberellins, brassinosteroids and absciscic acid. This network integrates multiple endogenous hormone signals, and links the regulation of shoot branching with plant growth, development and environmental adaptation. Although great progress has been made in the study of this regulatory network, there are still many unresolved key scientific questions, which will be the focus of future research in this field.

6 Discussion

This review systematically collates the research progress on strigolactones (SLs) in regulating plant shoot branching, covering the core biosynthetic pathway, canonical signal transduction cascade, direct regulatory effects on the full developmental cycle of axillary buds, and the multi-level crosstalk network with other major phytohormones. Collectively, the published studies in this field have built a foundational framework that defines SLs as a central negative regulator of shoot branching, and expanded our understanding of the molecular mechanisms underlying plant apical dominance and shoot architecture formation. Over the past decades, research on SLs-mediated branching regulation has advanced from initial phenotypic characterization of high-branching mutants, to the identification of core pathway components, and further to the dissection of regulatory networks integrating hormonal signals and environmental cues, laying a basic theoretical reference for follow-up research on the SLs pathway.

6.1 Phytohormone Crosstalk between SLs and Other Hormones in Shoot Branching Regulation

The crosstalk between SLs and other endogenous phytohormones is the core of the shoot branching regulatory network, and also a key focus of current research in this field. Existing studies have estab-

lished basic interaction models between SLs and several major branching-regulating hormones, but there are still many unresolved questions about the mechanistic details and *in vivo* functional relevance of these interactions.

The bidirectional crosstalk between SLs and auxin is explained by two classic models [100], one in which SLs act as auxin-downstream secondary messengers that move acropetally to directly inhibit buds [101], and another in which SLs suppress bud outgrowth via modulation of polar auxin transport [88]. Notably, the acropetal transport of SLs is not confined to a single vascular route. Physiological and tracer studies have revealed that SLs possess a dual translocation pathway. Root-synthesized SLs are primarily transported upward to the shoots via the xylem transpiration stream, which represents the major long-distance transport route for root-derived SLs [81]. In addition to xylem transport, the phloem participates in the systemic allocation of SLs signaling components. Grafting experiments revealed that *RMS3*, a pea ortholog of the SLs receptor *D14*, can be bi-directionally transported through the phloem to suppress shoot branching [102]. Nevertheless, it is important to highlight that this evidence originates from studies on the mobile SLs receptor protein rather than SLs molecules themselves, and the exact directionality of phloem transport of native SLs has yet to be fully clarified.

This distinction in transport routes holds critical implications for interpreting classical horticultural practices, such as bark scoring in fruit trees. The dominant effect of bark scoring is the physical disruption of the basipetal polar auxin transport stream originating from the apical meristem. This disruption has two interconnected consequences for SLs-mediated branching regulation. First, stem-localized SLs biosynthesis is transcriptionally induced by apically derived auxin transported basipetally via PAT [103]. Therefore, scoring immediately above the bud severs this auxin supply to the stem segment below the wound, thereby significantly reducing local SLs production around the axillary bud. Second, the PAT blockade causes a pronounced depletion of IAA in the bud itself, releasing the bud from auxin-mediated apical dominance. The effect likely overrides residual SLs-mediated inhibition that might persist from root-derived SLs arriving via the xylem. In addition, if the biologically effective SL signal for bud repression depends more on phloem-delivered SLs, then scoring, which severs phloem continuity, could further promote bud outgrowth by cutting off this supply pathway. However, this possibility remains speculative and awaits experimental verification. While the SLs-dual-transport framework provides a useful conceptual basis, the coordination mechanism between these two pathways *in vivo* remains largely unresolved. For example, it is still unclear whether these pathways operate in a tissue-specific or developmental stage-specific manner, and how they jointly maintain the stability of apical dominance under fluctuating growth conditions. Furthermore, the existence of a PAT-independent regulatory pathway for SLs has been confirmed by genetic evidence in pea [28], which supports the notion that SLs may employ a dual regulatory mechanism to control shoot branching, acting partially through the PAT system while also functioning via an auxin transport-independent route. However, the core molecular components and functional scope of this alternative pathway remain largely unknown, and its conservation across different plant species requires further investigation. Together, these unresolved questions highlight the need for future studies to dissect the spatiotemporal dynamics of SLs transport and signaling, and to integrate these distinct pathways into a unified model of branching regulation.

The antagonistic relationship between SLs and CK in axillary bud regulation has been widely confirmed, and the dynamic balance of these two hormones in axillary buds is considered a key determinant of bud fate. However, current research on this antagonistic interaction is highly unbalanced: most studies focus on the regulatory effects of SLs on CK biosynthesis, transport and degradation, while the reverse regulatory mechanism of CK on the SLs pathway is still poorly characterized. It is still unclear whether CK can directly

regulate the expression of SLs biosynthetic or signaling genes, or affect the long-distance transport of SLs, to participate in branching regulation. In addition, most existing findings are based on the observation of hormone content changes in whole stems or whole plants, and the dynamic changes of SLs and CK in single axillary buds during the transition from dormancy to activation have not been fully characterized, which limits our understanding of how this hormone balance directly determines bud fate.

For the crosstalk between SLs and other phytohormones including GA, BR and ABA, current research is still in the preliminary stage, and most findings are derived from phenotypic observation and transcriptomic correlation analysis in model plants. The direct molecular mechanisms linking these hormones to the SLs pathway in branching regulation are still not fully elucidated. For example, although transcriptomic studies have found overlapping target genes between SLs and GAs, the specific target genes that directly regulate axillary bud outgrowth have not been identified. For the crosstalk between SLs and BRs, the one confirmed convergent node is *MAX2*-mediated degradation of *BZR1/BES1* [34], but it is still unclear whether there are other regulatory nodes linking these two pathways. For SLs and ABA, which share a common biosynthetic precursor, it remains to be explored whether there is metabolic flux competition between the two hormone synthetic pathways, and how this potential competition coordinates the trade-off between plant branching growth and stress adaptation.

Notably, most existing studies focus on the pairwise interaction between SLs and a single hormone, while the *in vivo* regulation of shoot branching is the result of the integrated action of multiple endogenous hormones. How the SLs pathway acts as a hub to integrate signals from multiple hormones, and how the multi-hormone regulatory network responds to environmental changes to fine-tune branching plasticity, have not yet formed a complete and systematic model, which is a key direction for future research in this field.

6.2 Current Limitations and Unresolved Questions of SLs-Mediated Branching Regulation

Building on the unresolved questions surrounding hormone crosstalk outlined above, our understanding of SLs-mediated shoot branching regulation is further constrained by several fundamental controversies and critical knowledge gaps. In this section, we synthesize key ongoing debates and persisting uncertainties, and highlight priority open questions that warrant targeted future investigation. These issues can be grouped into four interrelated categories, cross-species conservation and functional divergence, unresolved mechanistic details within the core signalling cascade, methodological biases arising from synthetic SL analogues, and environmental signal integration.

First, the extent to which SLs function as universal inhibitors of shoot branching across plant lineages remains contentious. The canonical role of SLs in suppressing axillary bud outgrowth has been robustly validated in annual model species including *Arabidopsis thaliana*, rice, and tomato. However, both the magnitude and direction of branching responses to SLs exhibit substantial variation across species, tissue types, and developmental contexts. Notably, in woody perennial species such as poplar, a definitive role for SLs in branching control has yet to be unequivocally established [104]. Although the core *D14-MAX2-SMXL-UPS* module is deeply conserved across land plants, accumulating comparative studies reveal remarkable species-specific regulatory modulations. In early-diverging land plants, including bryophytes and lycophytes, the *KAI2*-dependent signalling pathway is thought to be ancestral and responds to as-yet-unidentified endogenous ligands, regulating developmental processes and symbiotic interactions, whereas *D14*-mediated perception of canonical strigolactones appears to have undergone subsequent neofunctionalization in vascular plants. This evolutionary framework cautions against assuming uniform SLs action across lineages. In eudicots, represented by pea and soybean, orthologs of *D14*, *MAX2*, and *SMXLs* retain canonical

SLs-triggered protein interaction and SCF-mediated repressor degradation, which governs shoot branching; in legumes, SL signalling is additionally integrated with nodulation and arbuscular mycorrhizal symbiosis pathways, further complicating the interpretation of branching-related phenotypes [92,105]. In monocot crops beyond rice, such as maize and wheat, *D14* homologs maintain conserved hydrolase activity, but their substrate preferences and downstream target genes have diverged, reflecting adaptive evolution to distinct developmental programs and environmental cues [106,107]. In line with this, *D14* orthologs across plant species display divergent substrate preferences for naturally occurring SLs stereoisomers [57]. In woody perennial fruit trees such as apple, SLs signalling regulates not only branching architecture but also stress responses and reproductive development, accompanied by lineage-specific gene expansion and subfunctionalization of the *SMXL* family [108]. In horticultural species like strawberry, SLs are involved in runner formation and flower senescence, revealing novel regulatory nodes not fully recapitulated in annual herbs [109]. Collectively, cross-species comparative evidence demonstrates that although the core SLs signal transduction machinery is evolutionarily conserved across land plants, its precise molecular regulatory nodes, functional outputs, and crosstalk with other hormone pathways are highly lineage-specific, shaped by species-specific gene duplication, functional diversification, and ecological adaptation. Accordingly, extrapolating findings from model species to agronomically important crops or woody perennials requires caution.

Second, several mechanistic details within the core SLs signalling cascade remain unresolved. Functional redundancy among *SMXL* paralogs remains incompletely characterized. Phylogenetic studies resolve *SMXL* proteins into four evolutionarily distinct clades, each bearing clade-specific motifs proposed to underpin signalling specificity between the SL and karrikin pathways [110]. Whereas *SMXL6/7/8* function in a largely redundant manner in *Arabidopsis*, rice *D53* and pea *PsSMXL7* appear to have evolved more dominant, non-redundant roles in branching regulation [92]. Whether such interspecific discrepancies reflect genuine species-specific regulatory wiring or methodological artefacts remains to be resolved through systematic cross-species comparative analyses. In addition, non-canonical signalling modules that bypass the *D14*–*MAX2*–*SMXL* axis are poorly understood, and their contribution to branching regulation is unclear. The cell-type-specific actions of SLs within axillary buds have not been systematically characterized, and it remains unknown whether SLs signalling functions in the bud itself, in the nodal vasculature, or in both tissues. Furthermore, the balance between functional specificity and redundancy of *SMXLs* family members in branching control is still not fully clarified, partly limited by the lack of tissue-specific mutants and the difficulty in distinguishing direct from indirect regulatory effects [111].

Third, a major methodological limitation is the pervasive reliance on synthetic SL analogues, particularly GR24, as proxies for endogenously produced SLs. To date, more than 25 naturally occurring SLs structures have been characterized [42], yet the majority of functional studies employ GR24, often as a racemic mixture. This introduces several experimental biases. GR24 enantiomers differ markedly in receptor selectivity: one enantiomer preferentially activates *D14*, whereas the other can activate *KAI2*, thereby confounding SLs-specific and karrikin-related responses. In addition, GR24 exhibits different metabolic stability, transport behaviour, and receptor-binding kinetics compared with endogenous SLs. Natural SLs differ substantially in stereochemistry, abundance, and tissue-specific distribution across species and developmental stages, and distinct SLs isoforms may exert discrete, non-redundant functions that GR24 cannot faithfully reproduce. Consequently, conclusions based solely on exogenous GR24 treatment may overestimate or misattribute SLs functions.

Fourth, the precise mechanisms by which environmental cues are transduced into endogenous SLs signals to modulate shoot branching plasticity remain only partially characterized. It is well established that

phosphate and nitrogen deficiencies strongly upregulate SLs biosynthesis [112], but the specific signalling cascades coupling these external nutrient stimuli to transcriptional regulation of SLs biosynthetic genes remain incompletely defined [46]. Recent work has identified *FAR-RED ELONGATED HYPOCOTYL 3 (FHY3)* and *FAR-RED IMPAIRED RESPONSE 1 (FAR1)* as key integrators of light and SLs signalling pathways [62], while the mechanistic links between shade avoidance responses and SLs-dependent branching regulation remain fragmentary. Beyond nutrient and light signals, the coordination between root development and shoot branching through SLs-mediated root-to-shoot communication remains an underexplored area. Although SLs are known to be synthesized predominantly in roots and transported acropetally, how root-derived SLs quantitatively and spatially coordinate with locally produced shoot signals to fine-tune branching is not well defined. Similarly, although SLs promote AM symbiosis in the rhizosphere, the functional connection between SLs-dependent mycorrhizal colonization and shoot branching regulation has not been directly examined. Under nutrient-limited conditions, elevated SLs levels simultaneously enhance AM symbiosis and suppress shoot branching; however, whether these two outputs are independently regulated or integrated through a common signaling logic, and how the carbon cost of maintaining AM symbiosis feeds back onto branching decisions, remain open questions. Furthermore, the contribution of growth–defense trade-offs to SLs-mediated branching control has received little attention. Defense activation often reallocates resources away from growth, and SLs have been implicated in both biotic stress responses and branching inhibition. Yet whether SLs serve as a molecular switch that prioritizes defense over branching under pathogen or herbivore attack, and how this trade-off is tuned in different ecological contexts, are poorly understood.

Although the core molecular machinery of SLs signalling is now well established, the field faces a pivotal challenge in unravelling the system-level complexity that governs branching phenotypes. The priority research directions should include systematic multi-species comparative analyses, direct genetic dissection of endogenous SLs function, and the development of advanced analytical tools to enable resolved dissection of SLs function.

6.3 Application Potential and Bottlenecks of the SLs Pathway in Crop Breeding and Trait Improvement

The SLs pathway has shown considerable application potential in crop breeding and plant architecture improvement, based on its core regulatory role in shoot branching. However, it is important to distinguish between results obtained from laboratory pot-grown assays and real-world field-application bottlenecks. Most current proof-of-concept studies are conducted under controlled conditions with small plant populations, whereas field deployment requires stable performance across variable environments, large-scale validation, and cost-effective delivery methods.

For cereal crops such as rice, wheat and maize, the SLs pathway is a potential target for ideal plant architecture breeding. Under controlled laboratory or pot conditions, fine-tuning the expression of SLs pathway genes has made it possible to create germplasm resources with moderate tiller number, strong lodging resistance and high density tolerance, which may contribute to the improvement of grain yield. Recent advances in CRISPR/Cas9-mediated genome editing have demonstrated the feasibility of this approach. In rice, targeted disruption of *CCD7*, a key gene in SLs biosynthesis, produced *ccd7* mutants with a striking increase in tillering and reduced plant height phenotype, which could be rescued by exogenous GR24 application [113]. More importantly, researchers carried out precise editing of the *D3* gene in the japonica rice cultivar DS. The *D3* gene acts as a positive regulator of plant height and inhibits tiller growth. This work produced compact lines with increased tillering capacity; these lines reached 73%–79% of the original plant height, bore roughly 20 panicles per plant versus around eight in the wild type, and exhibited

stronger resistance to bacterial blight without sacrificing total grain yield per plant [114]. The edited lines also exhibited earlier heading and stable, non-transgenic inheritance. Nevertheless, the field performance, yield stability, and agronomic suitability of these edited lines under diverse cultivation environments remain to be systematically evaluated. In wheat, CRISPR-mediated targeted mutagenesis has been harnessed to knock out yield-related negative regulators in elite cultivars, resulting in increased tiller number, spikelet number per spike, and thousand-grain weight, substantially enhancing grain yield per plant in experimental settings [115]. The wheat oligo-tillering mutant *ot1*, in which SLs biosynthesis genes including *D27* and *MAX1* are upregulated by 3.37- to 8.23-fold, further demonstrates the potential of modulating SLs pathway activity to control tiller number [116]. However, translating these findings to field-scale cultivation requires overcoming genotype-by-environment interactions and ensuring that branching modifications do not compromise other yield components.

For horticultural crops, SLs-based regulation can achieve precise control of branching number, reduce the growth of redundant lateral shoots, lower the workload of manual pruning, and improve the ventilation and light transmission of the canopy. In tomato, CRISPR/Cas9 knockout lines for four core SLs biosynthesis genes (*SID27*, *SLCCD7*, *SLCCD8*, *SIMAX1*) have been generated, revealing gene-specific effects on plant phenotype, such as markedly increased shoot branching and reduced plant height in *Slccd7* and *Slccd8* mutants, whereas *Slmax1* mutants exhibited altered reproductive development, and identifying *SID27* as a potential target for resistance to parasitic weeds [117]. Tomato *sb1*, *sb2*, and *sb3* mutants, which abolish SLs biosynthesis or impair SLs perception, exhibit increased shoot branching and provide valuable germplasm for breeding programs [77]. However, increased branching in these mutants is often associated with excessive vegetative growth and altered reproductive development under field-like conditions, underscoring the need for careful phenotypic evaluation beyond pot experiments. In grapevine, knockout of the *VvCCD8* gene has been shown to affect shoot branching, with knockout lines displaying increased lateral shoot number and altered branch angles, and studies are underway to characterize the role of SLs in regulating scion growth and branching in grafted grapevine [118].

For forest trees, regulating the SLs pathway may help to cultivate fast-growing timber varieties with straight trunks and fewer lateral branches, to improve wood yield and quality. While research in woody perennials remains limited, studies in apple have demonstrated that SLs regulate axillary bud growth through the transcription factor *SQUAMOSA PROMOTER BINDING PROTEIN-LIKE* (*SPL6*), which modulates ABA levels, identifying potential targets for breeding apple varieties with optimized branching and enhanced yield [67]. The SLs inhibitor Tis108 has been shown to improve shoot regeneration in apple, providing a chemical tool for manipulating SLs activity in woody species. In olive (*Olea europaea* L.), SLs biosynthesis and signaling genes have been identified and characterized, with potential applicability to breeding programs for other fruit crops [119]. However, the long generation times and low genetic transformation efficiency of woody plants remain major bottlenecks for functional validation and breeding application of SLs pathway genes in these species. Moreover, field evaluation of engineered woody plants is a long-term and resource-intensive process, and many reported effects remain at the level of in vitro or greenhouse observations. A particularly important concern for translational application is the pleiotropic nature of SLs pathway genes.

These examples across rice, wheat, tomato, grapevine, and woody perennials clearly show that the same SLs-pathway gene perturbation can produce markedly different phenotypic outcomes depending on the crop species. The molecular basis underlying such inter-crop phenotypic divergence is likely multi-layered. First, the degree of functional redundancy among SLs-pathway gene family members differs considerably across species. For instance, *SMXL6/7/8* in Arabidopsis act largely redundantly, whereas rice *D53* functions

as a dominant, non-redundant repressor, and pea PsSMXL7 similarly displays a more prominent role, so mutations in the same pathway can produce quantitatively distinct branching phenotypes. Second, the downstream target genes and their cis-regulatory landscapes have diverged among crops, meaning that the same SLs signal may regulate different sets of branching-related transcription factors. Third, the endogenous hormonal context varies between species, so perturbations of the SLs pathway can be buffered or amplified differently depending on the species-specific hormone network. These molecular determinants not only explain why editing the same gene can yield divergent phenotypes across crops, but also highlight the challenge of extrapolating results from one species to another.

The SLs pathway has significant pleiotropic effects [120]. It not only regulates shoot branching, but also participates in root development, stress resistance and rhizosphere symbiosis with arbuscular mycorrhizal fungi [121]. Complete knockout of core SLs pathway genes often leads to adverse agronomic traits, such as reduced stress resistance and impaired nutrient uptake capacity, which limits its direct application in breeding. This pleiotropy poses a substantial risk: mutations that improve shoot architecture may simultaneously compromise root function, abiotic stress tolerance, or beneficial microbial interactions, resulting in net yield penalties under field conditions. On the other hand, natural SLs have poor stability and are easily degraded in the field environment, while the production cost of synthetic SLs analogs with high activity is still high, which restricts their large-scale application in agricultural production. Despite these challenges, progress is being made. The synthetic SLs analog GR24 has been widely used to investigate stress tolerance mechanisms. For example, 10 μM GR24 treatment significantly enhanced salinity tolerance in wheat by modulating antioxidant enzyme activities, ion homeostasis, and stress-responsive gene expression [122]. Different methods of GR24 application, specifically foliar spray and application in residual irrigation water, have been shown to increase photosynthesis and yield in winter wheat under drought stress, suggesting that optimized delivery protocols could enable practical field application [85]. However, these studies are largely small-scale and short-term; the stability, cost-effectiveness, and environmental impact of chemical application under real farming conditions require further assessment.

Future research on SLs-mediated shoot branching regulation should move beyond descriptive directions and focus on specific mechanistic bottlenecks that currently limit our understanding. A central unresolved question is how SL signals are precisely decoded into the transcriptional regulatory networks downstream of SMXL proteins. Although *SMXL6/7/8* degradation is known to relieve transcriptional repression, the direct target genes, cell-type-specific regulatory logic, and temporal dynamics of this derepression remain poorly defined. It is unclear whether SMXL proteins act solely as transcriptional repressors or also as scaffolds that coordinate multi-protein complexes at specific genomic loci. Furthermore, *SMXL6* has been shown to bind DNA directly, yet the relationship between its DNA-binding activity and its repressor function in different developmental contexts is not fully understood. Resolving how the same SMXL proteins can elicit distinct transcriptional outputs during axillary meristem initiation versus bud outgrowth will require genome-wide mapping of SMXL occupancy, identification of interacting transcription factors, and functional validation using stage-specific genetic tools. Another key bottleneck lies in the non-canonical signaling modules that bypass or modulate the *D14-MAX2-SMXL* axis. The molecular identity of these alternative pathways, their contribution to branching regulation, and their interplay with canonical signaling remain largely unknown. Similarly, the cell-type-specific action of SLs within axillary buds, specifically whether SLs act primarily in the bud itself, in the nodal vasculature, or in both, remains unresolved, and this spatial information is essential for building accurate models of branching control. Addressing these questions will not only deepen our mechanistic understanding but also provide precise targets for genetic improvement of plant architecture.

In summary, as a key regulator of plant shoot branching, SLs have become a sustained research hotspot in plant developmental biology and crop molecular breeding. Although there are still many unresolved scientific questions and application bottlenecks in this field, with the continuous development of new technologies and the deepening of research, the regulatory mechanism of SLs will be further elucidated, and its application potential in agricultural production will be further explored. This review systematically sorts out the research context and core progress in this field, aiming to provide a reference for follow-up related research.

Acknowledgement: Not applicable.

Funding Statement: This work was supported by projects of the Hebei Natural Science Foundation, grant number C2025301048.

Author Contributions: Conceptualization, Rui Liu and Yingbi Xu; methodology, Fengjuan Lu; writing—original draft preparation, Rui Liu and Guimin Tong; writing—review and editing, Rui Liu and Han Wang; supervision, Yingbi Xu; funding acquisition, Han Wang. All authors reviewed and approved the final version of the manuscript.

Availability of Data and Materials: Data sharing does not apply to this article.

Ethics Approval: Not applicable.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Barbier FF, Dun EA, Kerr SC, Chabikwa TG, Beveridge CA. An update on the signals controlling shoot branching. *Trends Plant Sci.* 2019;24(3):220–36. [[CrossRef](#)].
2. Wang M, Pérez-García MD, Davière JM, Barbier F, Ogé L, Gentilhomme J, et al. Outgrowth of axillary bud in rose is controlled by sugar metabolism and signalling. *J Exp Bot.* 2021;72:3044–60. [[CrossRef](#)].
3. Xiong S, Wang Y, Chen Y, Shi X, Wu L. The sucrose regulation of plant shoot branching. *Horticulturae.* 2024;10(12):1348. [[CrossRef](#)].
4. Bowman JL, Eshed Y. Formation and maintenance of the shoot apical meristem. *Trends Plant Sci.* 2000;5(3):110–5. [[CrossRef](#)].
5. Bartušková A, Klimešová J. Reiteration in the short lived root-sprouting herb *Rorippa palustris*: Does the origin of buds matter? *Botany.* 2010;88(7):630–8. [[CrossRef](#)].
6. Wang Y, Jiao Y. Axillary meristem initiation—A way to branch out. *Curr Opin Plant Biol.* 2018;41:61–6. [[CrossRef](#)].
7. Yang F, Wang Q, Schmitz G, Müller D, Theres K. The bHLH protein ROX acts in concert with RAX1 and LAS to modulate axillary meristem formation in *Arabidopsis*. *Plant J.* 2012;71(1):61–70. [[CrossRef](#)].
8. Rameau C, Bertheloot J, Leduc N, Andrieu B, Foucher F, Sakr S. Multiple pathways regulate shoot branching. *Front Plant Sci.* 2014;5:741. [[CrossRef](#)].
9. Wen C, Zhao Q, Nie J, Liu G, Shen L, Cheng C, et al. Physiological controls of *Chrysanthemum* DgD27 gene expression in regulation of shoot branching. *Plant Cell Rep.* 2016;35(5):1053–70. [[CrossRef](#)].
10. Guo L, Plunkert M, Luo X, Liu Z. Developmental regulation of stolon and rhizome. *Curr Opin Plant Biol.* 2021;59:101970. [[CrossRef](#)].
11. Zhu J, Li X, Huang J, Wang L, Zheng Q, Li H, et al. Transcriptomics and plant hormone analysis reveal the mechanism of branching angle formation in tea plants (*Camellia sinensis*). *Int J Mol Sci.* 2025;26(2):604. [[CrossRef](#)].
12. Kotov AA, Kotova LM. Interaction of phytohormones in regulating the axillary bud growth in pea. *Russ J Plant Physiol.* 2018;65(5):628–41. [[CrossRef](#)].
13. Harrison CJ. Auxin transport in the evolution of branching forms. *New Phytol.* 2017;215(2):545–51. [[CrossRef](#)].
14. Mashiguchi K, Tanaka K, Sakai T, Sugawara S, Kawaide H, Natsume M, et al. The main auxin biosynthesis pathway in *Arabidopsis*. *Proc Natl Acad Sci U S A.* 2011;108(45):18512–7. [[CrossRef](#)].

15. Xie X, Qin G, Si P, Luo Z, Gao J, Chen X, et al. Analysis of *Nicotiana tabacum* PIN genes identifies NtPIN4 as a key regulator of axillary bud growth. *Physiol Plant*. 2017;160(2):222–39. [[CrossRef](#)].
16. Yang L, Zhu S, Xu J. Roles of auxin in the inhibition of shoot branching in ‘Dugan’ fir. *Tree Physiol*. 2022;42(7):1411–31. [[CrossRef](#)].
17. Zha M, Zhao Y, Wang Y, Chen B, Tan Z. Strigolactones and cytokinin interaction in buds in the control of rice tillering. *Front Plant Sci*. 2022;13:837136. [[CrossRef](#)].
18. Xu Y, Huang B. Transcriptional factors for stress signaling, oxidative protection, and protein modification in ipt-transgenic creeping bentgrass exposed to drought stress. *Environ Exp Bot*. 2017;144:49–60. [[CrossRef](#)].
19. Nguyen HN, Kambhampati S, Kisiala A, Seegobin M, Emery RJN. The soybean (*Glycine max* L.) cytokinin oxidase/dehydrogenase multigene family: Identification of natural variations for altered cytokinin content and seed yield. *Plant Direct*. 2021;5(2):e00308. [[CrossRef](#)].
20. Xu X, Liang Y, Feng G, Li S, Yang Z, Nie G, et al. A favorable natural variation in *CCD7* from orchardgrass confers enhanced tiller number. *Plant J*. 2025;121(2):e17200. [[CrossRef](#)].
21. Waters MT, Brewer PB, Bussell JD, Smith SM, Beveridge CA. The *Arabidopsis* ortholog of rice DWARF27 acts upstream of MAX1 in the control of plant development by strigolactones. *Plant Physiol*. 2012;159(3):1073–85. [[CrossRef](#)].
22. Dun EA, Brewer PB, Gillam EMJ, Beveridge CA. Strigolactones and shoot branching: What is the real hormone and how does it work? *Plant Cell Physiol*. 2023;64(9):967–83. [[CrossRef](#)].
23. Hayward A, Stirnberg P, Beveridge C, Leyser O. Interactions between auxin and strigolactone in shoot branching control. *Plant Physiol*. 2009;151(1):400–12. [[CrossRef](#)].
24. Li J, Chai M, Zhu X, Zhang X, Li H, Wang D, et al. Cloning and expression analysis of LoCCD8 during IAA-induced bulbils outgrowth in lily (Oriental Hybrid ‘Sorbonne’). *J Plant Physiol*. 2019;236:39–50. [[CrossRef](#)].
25. Yuan R, Mao Y, Zhang D, Wang S, Zhang H, Wu M, et al. The formation of rice Tillers and factors influencing it. *Agronomy*. 2024;14(12):2904. [[CrossRef](#)].
26. Zhang J, Mazur E, Balla J, Gallei M, Kalousek P, Medvedova Z, et al. Strigolactones inhibit auxin feedback on PIN-dependent auxin transport canalization. *Nat Commun*. 2020;11(1):3508. [[CrossRef](#)].
27. van Rongen M, Bennett T, Ticchiarelli F, Leyser O. Connective auxin transport contributes to strigolactone-mediated shoot branching control independent of the transcription factor BRC1. *PLoS Genet*. 2019;15(3):e1008023. [[CrossRef](#)].
28. Brewer PB, Dun EA, Gui R, Mason MG, Beveridge CA. Strigolactone inhibition of branching independent of polar auxin transport. *Plant Physiol*. 2015;168(4):1820–9. [[CrossRef](#)].
29. Duan J, Yu H, Yuan K, Liao Z, Meng X, Jing Y, et al. Strigolactone promotes cytokinin degradation through transcriptional activation of *CYTOKININ OXIDASE/DEHYDROGENASE 9* in rice. *Proc Natl Acad Sci U S A*. 2019;116(28):14319–24. [[CrossRef](#)].
30. Zhang W, Tao J, Chang Y, Wang D, Wu Y, Gu C, et al. Cytokinin catabolism and transport are involved in strigolactone-modulated rice tiller bud elongation fueled by phosphate and nitrogen supply. *Plant Physiol Biochem*. 2024;215:108982. [[CrossRef](#)].
31. Ito S, Nozoye T, Sasaki E, Imai M, Shiwa Y, Shibata-Hatta M, et al. Strigolactone regulates anthocyanin accumulation, acid phosphatases production and plant growth under low phosphate condition in *Arabidopsis*. *PLoS One*. 2015;10(3):e0119724. [[CrossRef](#)].
32. Lantzouni O, Klermund C, Schwechheimer C. Largely additive effects of gibberellin and strigolactone on gene expression in *Arabidopsis thaliana* seedlings. *Plant J*. 2017;92(5):924–38. [[CrossRef](#)].
33. Wang Y, Sun S, Zhu W, Jia K, Yang H, Wang X. Strigolactone/MAX2-induced degradation of brassinosteroid transcriptional effector BES1 regulates shoot branching. *Dev Cell*. 2013;27(6):681–8. [[CrossRef](#)].
34. Hu J, Sun S, Wang X. Regulation of shoot branching by strigolactones and brassinosteroids: Conserved and specific functions of *Arabidopsis* BES1 and rice BZR1. *Mol Plant*. 2020;13(6):808–10. [[CrossRef](#)].
35. Luo L, Takahashi M, Kameoka H, Qin R, Shiga T, Kanno Y, et al. Developmental analysis of the early steps in strigolactone-mediated axillary bud dormancy in rice. *Plant J*. 2019;97(6):1006–21. [[CrossRef](#)].
36. Liu X, Hu Q, Yan J, Sun K, Liang Y, Jia M, et al. ζ -carotene isomerase suppresses tillering in rice through the coordinated biosynthesis of strigolactone and abscisic acid. *Mol Plant*. 2020;13(12):1784–801. [[CrossRef](#)].

37. Yang Y, Abuauf H, Song S, Wang JY, Alagoz Y, Moreno JC, et al. The *Arabidopsis* D27-LIKE1 is a *cis/cis/trans*- β -carotene isomerase that contributes to Strigolactone biosynthesis and negatively impacts ABA level. *Plant J*. 2023;113(5):986–1003. [[CrossRef](#)].
38. Moreno JC, Mi J, Alagoz Y, Al-Babili S. Plant apocarotenoids: From retrograde signaling to interspecific communication. *Plant J*. 2021;105(2):351–75. [[CrossRef](#)].
39. Booker J, Auldridge M, Wills S, McCarty D, Klee H, Leyser O. MAX3/CCD7 is a carotenoid cleavage dioxygenase required for the synthesis of a novel plant signaling molecule. *Curr Biol*. 2004;14(14):1232–8. [[CrossRef](#)].
40. Cheng J, Luo S, Baldwin G, Cheng X, Baldwin IT, Li S. Strigolactone and karrikin signaling influence the recruitment of wild tobacco's root microbiome in the desert. *Agronomy*. 2025;15(1):44. [[CrossRef](#)].
41. Yi F, Song A, Cheng K, Liu J, Wang C, Shao L, et al. Strigolactones positively regulate *Verticillium* wilt resistance in cotton via crosstalk with other hormones. *Plant Physiol*. 2023;192(2):945–66. [[CrossRef](#)].
42. Chen GTE, Wang JY, Jamil M, Braguy J, Al-Babili S. 9-*cis*- β -Apo-10'-carotenal is the precursor of strigolactones in planta. *Planta*. 2022;256(5):88. [[CrossRef](#)].
43. Kuno M, Miyamoto A, Takano H, Homma M, Shiotani N, Uchida K, et al. CYP722A1-mediated 16-hydroxylation of carlactonoic acid regulates the floral transition in *Arabidopsis*. *Plant Cell Physiol*. 2025;66(4):645–57. [[CrossRef](#)].
44. Wang JY, Balakrishna A, Martínez C, Chen GTE, Sioud S, de Lera AR, et al. The rice orobanchol synthase catalyzes the hydroxylation of the noncanonical strigolactone methyl 4-oxo-carlactonoate. *New Phytol*. 2024;244(6):2121–6. [[CrossRef](#)].
45. Shen H, Zhu L, Bu QY, Huq E. MAX2 affects multiple hormones to promote photomorphogenesis. *Mol Plant*. 2012;5(3):750–62. [[CrossRef](#)].
46. Waters MT, Nelson DC. Karrikin perception and signalling. *New Phytol*. 2023;237(5):1525–41. [[CrossRef](#)].
47. Xu Y, Lv Z, Manzoor MA, Song LH, Wang MS, Wang L, et al. VvD14c-VvMAX2-VvLOB/VvLBD19 module is involved in the strigolactone-mediated regulation of grapevine root architecture. *Mol Hortic*. 2024;4(1):40. [[CrossRef](#)].
48. Kagiya M, Hirano Y, Mori T, Kim SY, Kyojuka J, Seto Y, et al. Structures of D14 and D14L in the strigolactone and karrikin signaling pathways. *Genes Cells*. 2013;18(2):147–60. [[CrossRef](#)].
49. Yao R, Ming Z, Yan L, Li S, Wang F, Ma S, et al. DWARF14 is a non-canonical hormone receptor for strigolactone. *Nature*. 2016;536(7617):469–73. [[CrossRef](#)].
50. Fiorilli V, Forgia M, de Saint Germain A, D'Arrigo G, Cornu D, Le Bris P, et al. A structural homologue of the plant receptor D14 mediates responses to strigolactones in the fungal phytopathogen *Cryphonectria parasitica*. *New Phytol*. 2022;234(3):1003–17. [[CrossRef](#)].
51. Yoneyama K, Xie X, Yoneyama K, Kisugi T, Nomura T, Nakatani Y, et al. Which are the major players, canonical or non-canonical strigolactones? *J Exp Bot*. 2018;69(9):2231–9. [[CrossRef](#)].
52. Liu Y, Rong C, Wang S, Wu L, Ding Y, Ding C. Absence of 4' methyl on the d-ring of strigolactone does not prevent inhibition of tillering in rice through activation of the strigolactone signaling pathway. *Plant Growth Regul*. 2022;98(3):499–509. [[CrossRef](#)].
53. Nakamura H, Asami T. Target sites for chemical regulation of strigolactone signaling. *Front Plant Sci*. 2014;5:623. [[CrossRef](#)].
54. Li X, Xie Z, Qin T, Zhan C, Jin L, Huang J. The *SLR1-OsMADS23-D14* module mediates the crosstalk between strigolactone and gibberellin signaling to control rice tillering. *New Phytol*. 2025;246(5):2137–54. [[CrossRef](#)].
55. Chevalier F, Nieminen K, Sánchez-Ferrero JC, Rodríguez ML, Chagoyen M, Hardtke CS, et al. Strigolactone promotes degradation of DWARF14, an α/β hydrolase essential for strigolactone signaling in *Arabidopsis*. *Plant Cell*. 2014;26(3):1134–50. [[CrossRef](#)].
56. Komatsu A, Fujibayashi M, Kumagai K, Suzuki H, Hata Y, Takebayashi Y, et al. KAI2-dependent signaling controls vegetative reproduction in *Marchantia polymorpha* through activation of LOG-mediated cytokinin synthesis. *Nat Commun*. 2025;16(1):1263. [[CrossRef](#)].
57. Flematti GR, Scaffidi A, Waters MT, Smith SM. Stereospecificity in strigolactone biosynthesis and perception. *Planta*. 2016;243(6):1361–73. [[CrossRef](#)].
58. Lopez-Obando M, de Villiers R, Hoffmann B, Ma LN, de Saint Germain A, Kossmann J, et al. *Physcomitrella patens* MAX2 characterization suggests an ancient role for this F-box protein in photomorphogenesis rather than strigolactone signalling. *New Phytol*. 2018;219(2):743–56. [[CrossRef](#)].

59. Lian Y, Lian C, Wang L, Li Z, Yuan G, Xuan L, et al. SUPPRESSOR OF MAX2 LIKE 6, 7, and 8 interact with DDB1 BINDING WD REPEAT DOMAIN HYPERSENSITIVE TO ABA DEFICIENT 1 to Regulate the Drought Tolerance and Target *SUCROSE nonfermenting 1 related protein kinase 2.3* to Absciscic Acid Response in *Arabidopsis*. *Biomolecules*. 2023;13(9):1406. [[CrossRef](#)].
60. Tal L, Palayam M, Ron M, Young A, Britt A, Shabek N. A conformational switch in the SCF-D3/MAX2 ubiquitin ligase facilitates strigolactone signalling. *Nat Plants*. 2022;8(5):561–73. [[CrossRef](#)].
61. Shen R, Ma X, Wang H. SMXL6/7/8: Dual-function transcriptional repressors of strigolactone signaling. *Mol Plant*. 2020;13(9):1244–6. [[CrossRef](#)].
62. Wang L, Wang B, Yu H, Guo H, Lin T, Kou L, et al. Transcriptional regulation of strigolactone signalling in *Arabidopsis*. *Nature*. 2020;583(7815):277–81. [[CrossRef](#)].
63. Liu R, Feng X, Yin X, Zhu P. Mining and identification of factors influencing multi-branch plasticity in ornamental kale. *Planta*. 2025;261(6):134. [[CrossRef](#)].
64. Janssen BJ, Snowden KC. Strigolactone and karrikin signal perception: Receptors, enzymes, or both? *Front Plant Sci*. 2012;3:296. [[CrossRef](#)].
65. Khalid MF, Shafqat W, Khan RI, Jawaid MZ, Hussain S, Saqib M, et al. Unveiling the resilience mechanism: Strigolactones as master regulators of plant responses to abiotic stresses. *Plant Stress*. 2024;12:100490. [[CrossRef](#)].
66. Boyer FD, de Saint Germain A, Pillot JP, Pouvreau JB, Chen VX, Ramos S, et al. Structure-activity relationship studies of strigolactone-related molecules for branching inhibition in garden pea: Molecule design for shoot branching. *Plant Physiol*. 2012;159(4):1524–44. [[CrossRef](#)].
67. Fan X, Li H, Xiong Y, Chen D, Zhang Y, Sun H, et al. Strigolactones inhibit axillary bud outgrowth through transcription factor MdSPL6-mediated regulation of ABA levels in apple. *Plant Physiol*. 2025;199(3):kiaf517. [[CrossRef](#)].
68. Kerr SC, Beveridge CA. IPA1: A direct target of SLs signaling. *Cell Res*. 2017;27(10):1191–2. [[CrossRef](#)].
69. Zhao B, Wu TT, Ma SS, Jiang DJ, Bie XM, Sui N, et al. TaD27-B gene controls the tiller number in hexaploid wheat. *Plant Biotechnol J*. 2020;18(2):513–25. [[CrossRef](#)].
70. Feng F, Guo X, Zhu X, Hu Y, Chen Y, Sun H, et al. OsPIN2 is involved in OsSPL14/17-inhibited tiller bud outgrowth in response to phosphate deficiency in rice. *Environ Exp Bot*. 2023;209:105297. [[CrossRef](#)].
71. Roesler K, Lu C, Thomas J, Xu Q, Vance P, Hou Z, et al. *Arabidopsis* carboxylesterase 20 binds strigolactone and increases branches and tillers when ectopically expressed in *Arabidopsis* and maize. *Front Plant Sci*. 2021;12:639401. [[CrossRef](#)].
72. Sang D, Chen D, Liu G, Liang Y, Huang L, Meng X, et al. Strigolactones regulate rice tiller angle by attenuating shoot gravitropism through inhibiting auxin biosynthesis. *Proc Natl Acad Sci U S A*. 2014;111(30):11199–204. [[CrossRef](#)].
73. Liang J, Zhao L, Challis R, Leyser O. Strigolactone regulation of shoot branching in *Chrysanthemum* (*Dendranthema grandiflorum*). *J Exp Bot*. 2010;61(11):3069–78. [[CrossRef](#)].
74. Yin X, Chen J, Ye Y, Zhu H, Li J, Zhang L, et al. Optimizing bent branch numbers improves transpiration and crop water productivity of cut rose (*Rosa hybrida*) in greenhouse. *Agric Water Manag*. 2024;296:108795. [[CrossRef](#)].
75. Dun EA, de Saint Germain A, Rameau C, Beveridge CA. Dynamics of strigolactone function and shoot branching responses in *Pisum sativum*. *Mol Plant*. 2013;6(1):128–40. [[CrossRef](#)].
76. Ren C, Guo Y, Kong J, Lecourieux F, Dai Z, Li S, et al. Knockout of VvCCD8 gene in grapevine affects shoot branching. *BMC Plant Biol*. 2020;20(1):47. [[CrossRef](#)].
77. Karniel U, Koch A, Bar Nun N, Zamir D, Hirschberg J. Tomato mutants reveal root and shoot strigolactone involvement in branching and broomrape resistance. *Plants*. 2024;13(11):1554. [[CrossRef](#)].
78. Muhr M, Prüfer N, Paulat M, Teichmann T. Knockdown of strigolactone biosynthesis genes in *Populus* affects BRANCHED1 expression and shoot architecture. *New Phytol*. 2016;212(3):613–26. [[CrossRef](#)].
79. Sun M, Wang D, Liu C, Liu Y, Niu M, Wang J, et al. Genome-wide identification and analysis of the SUPPRESSOR of MAX2 1-LIKE gene family and its interaction with DWARF14 in poplar. *BMC Plant Biol*. 2023;23(1):105. [[CrossRef](#)].
80. Chen L, Zhao Y, Xu S, Zhang Z, Xu Y, Zhang J, et al. OsMADS57 together with OsTB1 coordinates transcription of its target OsWRKY94 and D14 to switch its organogenesis to defense for cold adaptation in rice. *New Phytol*. 2018;218(1):219–31. [[CrossRef](#)].

81. Kohlen W, Charnikhova T, Liu Q, Bours R, Domagalska MA, Beguerie S, et al. Strigolactones are transported through the xylem and play a key role in shoot architectural response to phosphate deficiency in nonarbuscular mycorrhizal host *Arabidopsis*. *Plant Physiol.* 2011;155(2):974–87. [[CrossRef](#)].
82. Quijano A, Morandi EN. Leaf area reduction during the pod set period changes the photomorphogenic light balance and increases the pod number and yield in soybean canopies. *Field Crops Res.* 2023;303:109148. [[CrossRef](#)].
83. Li T, Dai J, Zhang Y, Kong X, Li C, Dong H. Topical shading substantially inhibits vegetative branching by altering leaf photosynthesis and hormone contents of cotton plants. *Field Crops Res.* 2019;238:18–26. [[CrossRef](#)].
84. Qiao Y, Lu W, Wang R, Nisa Z, Yu Y, Jin X, et al. Identification and expression analysis of strigolactone biosynthetic and signaling genes in response to salt and alkaline stresses in soybean (*Glycine max*). *DNA Cell Biol.* 2020;39(10):1850–61. [[CrossRef](#)].
85. Sedaghat M, Emam Y, Mokhtassi-Bidgoli A, Hazrati S, Lovisolo C, Visentin I, et al. The potential of the synthetic strigolactone analogue GR24 for the maintenance of photosynthesis and yield in winter wheat under drought: Investigations on the mechanisms of action and delivery modes. *Plants.* 2021;10(6):1223. [[CrossRef](#)].
86. Zheng X, Zhang J, Zhao M, Su Z, Li H, Wu J. Strigolactones, ROS and ABA regulate systemic salt-tolerance priming signals between dodder-connected tobacco plants. *Plant Cell Environ.* 2025;48(6):4370–84. [[CrossRef](#)].
87. Guan Y, Li L, Wang D, Zhou J, Qi W, Cheng Y, et al. Multifaceted functions of strigolactones in annual and perennial plants: Developmental regulation, phytohormone crosstalk and abiotic stresses. *Plant Stress.* 2025;16:100895. [[CrossRef](#)].
88. Bennett T, Sieberer T, Willett B, Booker J, Luschign C, Leyser O. The *Arabidopsis* MAX pathway controls shoot branching by regulating auxin transport. *Curr Biol.* 2006;16(6):553–63. [[CrossRef](#)].
89. He Y, He K, Mai J, Ou M, Chen L, Li Y, et al. Boron controls apical dominance in Pea (*Pisum sativum*) via promoting polar auxin transport. *Physiol Plant.* 2025;177:e70056. [[CrossRef](#)].
90. Peng Y, Jiang Y, He C, She M, Li M, Chen Q, et al. Exogenous GR24 inhibits strawberry tillering by affecting the phytohormone signaling and sugar metabolism pathways. *Agronomy.* 2023;13(12):3078. [[CrossRef](#)].
91. Chen S, Song X, Zheng Q, Liu Y, Yu J, Zhou Y, et al. The transcription factor SPL13 mediates strigolactone suppression of shoot branching by inhibiting cytokinin synthesis in *Solanum lycopersicum*. *J Exp Bot.* 2023;74(18):5722–35. [[CrossRef](#)].
92. Kerr SC, Patil SB, de Saint Germain A, Pillot JP, Saffar J, Ligerot Y, et al. Integration of the SMXL/D53 strigolactone signalling repressors in the model of shoot branching regulation in *Pisum sativum*. *Plant J.* 2021;107(6):1756–70. [[CrossRef](#)].
93. Su S, Hong J, Chen X, Zhang C, Chen M, Luo Z, et al. Gibberellins orchestrate panicle architecture mediated by DELLA-KNOX signalling in rice. *Plant Biotechnol J.* 2021;19(11):2304–18. [[CrossRef](#)].
94. Anfang M, Shani E. Transport mechanisms of plant hormones. *Curr Opin Plant Biol.* 2021;63:102055. [[CrossRef](#)].
95. Singh AP, Savaldi-Goldstein S. Growth control: Brassinosteroid activity gets context. *J Exp Bot.* 2015;66(4):1123–32. [[CrossRef](#)].
96. Liang T, Yu S, Pan Y, Wang J, Kay SA. The interplay between the circadian clock and abiotic stress responses mediated by ABF₃ and CCA1/LHY. *Proc Natl Acad Sci U S A.* 2024;121(7):e2316825121. [[CrossRef](#)].
97. Wang H, Chen W, Eggert K, Charnikhova T, Bouwmeester H, Schweizer P, et al. Absciscic acid influences tillering by modulation of strigolactones in barley. *J Exp Bot.* 2018;69(16):3883–98. [[CrossRef](#)].
98. Visentin I, Vitali M, Ferrero M, Zhang Y, Ruyter-Spira C, Novák O, et al. Low levels of strigolactones in roots as a component of the systemic signal of drought stress in tomato. *New Phytol.* 2016;212(4):954–63. [[CrossRef](#)].
99. Ren CG, Kong CC, Xie ZH. Role of absciscic acid in strigolactone-induced salt stress tolerance in arbuscular mycorrhizal *Sesbania cannabina* seedlings. *BMC Plant Biol.* 2018;18(1):74. [[CrossRef](#)].
100. Leyser O. The control of shoot branching: An example of plant information processing. *Plant Cell Environ.* 2009;32(6):694–703. [[CrossRef](#)].
101. Kapulnik Y, Resnick N, Mayzlish-Gati E, Kaplan Y, Wininger S, Hershenhorn J, et al. Strigolactones interact with ethylene and auxin in regulating root-hair elongation in *Arabidopsis*. *J Exp Bot.* 2011;62(8):2915–24. [[CrossRef](#)].
102. Kameoka H, Dun EA, Lopez-Obando M, Brewer PB, de Saint Germain A, Rameau C, et al. Phloem transport of the receptor DWARF14 protein is required for full function of strigolactones. *Plant Physiol.* 2016;172(3):1844–52. [[CrossRef](#)].

103. Kerr SC, de Saint Germain A, Dissanayanke IM, Mason MG, Dun EA, Beveridge CA, et al. Hormonal regulation of the BRC1-dependent strigolactone transcriptome involved in shoot branching responses. *bioRxiv*. 2020. [[CrossRef](#)].
104. Su C, Kokosza A, Xie X, Pěňčík A, Zhang Y, Raumonen P, et al. Tree architecture: A strigolactone-deficient mutant reveals a connection between branching order and auxin gradient along the tree stem. *Proc Natl Acad Sci U S A*. 2023;120(48):e2308587120. [[CrossRef](#)].
105. Ahmad MZ, Rehman NU, Yu S, Zhou Y, Haq BU, Wang J, et al. GmMAX2-D14 and-KAI interaction-mediated SL and KAR signaling play essential roles in soybean root nodulation. *Plant J*. 2020;101(2):334–51. [[CrossRef](#)].
106. Zhang C, Wang F, Jiao P, Liu J, Zhang H, Liu S, et al. The overexpression of *Zea mays* strigolactone receptor gene *D14* enhances drought resistance in *Arabidopsis thaliana* L. *Int J Mol Sci*. 2024;25(2):1327. [[CrossRef](#)].
107. Liu R, Hou J, Li H, Xu P, Zhang Z, Zhang X. Association of *TaD14-4D*, a gene involved in strigolactone signaling, with yield contributing traits in wheat. *Int J Mol Sci*. 2021;22(7):3748. [[CrossRef](#)].
108. Fan X, Li Y, Deng CH, Wang S, Wang Z, Wang Y, et al. Strigolactone regulates adventitious root formation via the MdSMXL7-MdWRKY6-MdBRC1 signaling cascade in apple. *Plant J*. 2023;113(4):772–86. [[CrossRef](#)].
109. Sun H, Zhang J, Li W, Wang Y, Zhang Z. FveDAD2 negatively regulates branch crowns by affecting abscisic acid metabolism through FveHB7 in woodland strawberry. *Hortic Res*. 2026;13(1):uhaf250. [[CrossRef](#)].
110. Moturu TR, Thula S, Singh RK, Nodzynski T, Vareková RS, Friml J, et al. Molecular evolution and diversification of the SMXL gene family. *J Exp Bot*. 2018;69(9):2367–78. [[CrossRef](#)].
111. Soundappan I, Bennett T, Morffy N, Liang Y, Stanga JP, Abbas A, et al. SMAX1-LIKE/D53 family members enable distinct MAX2-dependent responses to strigolactones and karrikins in *Arabidopsis*. *Plant Cell*. 2015;27(11):3143–59. [[CrossRef](#)].
112. Barbier FF, Cao D, Fichtner F, Weiste C, Perez-Garcia MD, Caradeuc M, et al. HEXOKINASE1 signalling promotes shoot branching and interacts with cytokinin and strigolactone pathways. *New Phytol*. 2021;231(3):1088–104. [[CrossRef](#)].
113. Butt H, Jamil M, Wang JY, Al-Babili S, Mahfouz M. Engineering plant architecture via CRISPR/Cas9-mediated alteration of strigolactone biosynthesis. *BMC Plant Biol*. 2018;18(1):174. [[CrossRef](#)].
114. He J, He ZW, Zhao YD, Huang YQ, Yi R, Chen PY, et al. CRISPR/Cas9-mediated editing of D3 gene reshapes rice architecture and enhances disease resistance. *Plant Horm*. 2025;1:e017.
115. Awan MJA, Amin I, Rasheed A, Saeed NA, Mansoor S. Knockout mutation in *TaD27* enhances number of productive tillers in hexaploid wheat. *Front Genome Ed*. 2024;6:1455761. [[CrossRef](#)].
116. Bai J, Guo H, Xiong H, Xie Y, Gu J, Zhao L, et al. Strigolactone and abscisic acid synthesis and signaling pathways are enhanced in the wheat oligo-tillering mutant *ot1*. *Mol Breed*. 2024;44(2):12. [[CrossRef](#)].
117. Lee J, Seo MG, Lim Y, Hong S, An JT, Jeong HY, et al. Modulating the strigolactone pathway to optimize tomato shoot branching for vertical farming. *J Integr Plant Biol*. 2026;68(1):113–29. [[CrossRef](#)].
118. Cochetel N, Météier E, Merlin I, Hévin C, Pouvreau JB, Coutos-Thévenot P, et al. Potential contribution of strigolactones in regulating scion growth and branching in grafted grapevine in response to nitrogen availability. *J Exp Bot*. 2018;69(16):4099–112. [[CrossRef](#)].
119. Özbilen A, Sezer F, Taşkın KM. Identification and expression of strigolactone biosynthesis and signaling genes and the *in vitro* effects of strigolactones in olive (*Olea europaea* L.). *Plant Direct*. 2024;8(2):e568. [[CrossRef](#)].
120. Marzec M, Melzer M. Regulation of root development and architecture by strigolactones under optimal and nutrient deficiency conditions. *Int J Mol Sci*. 2018;19(7):1887. [[CrossRef](#)].
121. Borghi L, Liu GW, Emonet A, Kretschmar T, Martinoia E. The importance of strigolactone transport regulation for symbiotic signaling and shoot branching. *Planta*. 2016;243(6):1351–60. [[CrossRef](#)].
122. Jariani P, Sabokdast M, Rajabi F, Naghavi MR, Dedicova B. Enhancing salinity tolerance in wheat: The role of synthetic Strigolactone (GR24) in modulating antioxidant enzyme activities, ion channels, and related gene expression in stress responses. *Sci Rep*. 2025;15(1):24216. [[CrossRef](#)].