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REVIEW

Strigolactones and their crosstalk with other phytohormones

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• **Background** Strigolactones (SLs) are a diverse class of butenolide-bearing phytohormones derived from the catabolism of carotenoids. They are associated with an increasing number of emerging regulatory roles in plant growth and development, including seed germination, root and shoot architecture patterning, nutrient acquisition, symbiotic and parasitic interactions, as well as mediation of plant responses to abiotic and biotic cues.
• **Scope** Here, we provide a concise overview of SL biosynthesis, signal transduction pathways and SL-mediated plant responses with a detailed discourse on the crosstalk(s) that exist between SLs/components of SL signalling and other phytohormones such as auxins, cytokinins, gibberellins, abscisic acid, ethylene, jasmonates and salicylic acid.
• **Conclusion** SLs elicit their control on physiological and morphological processes via a direct or indirect influence on the activities of other hormones and/or integrants of signalling cascades of other growth regulators. These, among many others, include modulation of hormone content, transport and distribution within plant tissues, interference with or complete dependence on downstream signal components of other phytohormones, as well as acting synergistically or antagonistically with other hormones to elicit plant responses. Although much has been done to evince the effects of SL interactions with other hormones at the cell and whole plant levels, research attention must be channelled towards elucidating the precise molecular events that underlie these processes. More especially in the case of abscisic acid, cytokinins, gibberellin, jasmonates and salicylic acid for which very little has been reported about their hormonal crosstalk with SLs.

Keywords: Abscisic acid, auxin, ethylene, cytokinins, D3/MAX2, D53/SMXL, gibberellins, GR24, jasmonates, salicylic acid, strigolactone signalling, strigolactone interactions.

INTRODUCTION

Biomolecules with butenolide moieties elicit a plethora of biological activities in plants. Their bioactivities range from acting as chemical cues or signal molecules for host–parasite/symbiont interactions (Akiyama *et al.*, 2005, 2010; Foo and Davies, 2011; De Cuyper *et al.*, 2015), stimulation and/or inhibition of germination in seeds (Van Staden *et al.*, 2004; Daws *et al.*, 2007; Soós *et al.*, 2012), mediation of stress responses in plants (Soós *et al.*, 2009; Cheng *et al.*, 2017; Haider *et al.*, 2018) to increasing seedling growth and vigour in plants (Light *et al.*, 2010). Karrikins and strigolactones (SLs) are two groups of butenolides that have garnered huge research attention due to their influence on key aspects of plant growth and development as well as ecological significance. While SLs are largely endogenous, being synthesized by tissues *in planta* and transported between tissues or exuded into the rhizosphere as components of root exudates, the karrikins (Fig. 1) are components of smoke that are derived from the combustion of plant materials (Van Staden *et al.*, 1995; Flematti *et al.*, 2004, 2013; Kulkarni *et al.*, 2010). Despite the marked similarities in bioactivities and

signal pathways of karrikins and SLs (Nelson *et al.*, 2011; Waters *et al.*, 2012*c*), the discovery of *kai2* mutants which are defective in karrikin perception but still retain their SL sensitivity and of *d14* mutants lacking response to SLs but still retain their sensitivity to karrikins confirm that plants can distinguish between these two molecular cues (Waters *et al.*, 2012*b*, *c*; Flematti *et al.*, 2013). Against the backdrop of extant and emerging experimental evidences karrikins and SLs are largely seen as distinct chemical cues with distinct roles in the regulation of plant growth and development, although experimental data exist that allude to the sharing of roles and signal pathways by both signal molecules (Li *et al.*, 2017).

Against the backdrop of experimental findings that suggest that SL bioactivities are mediated via a direct or indirect influence on the activities of other phytohormones, we provide brief highlights on the chemical nature, biosynthesis and signalling of SLs while focusing more on the interactions between SLs/elements of SL signal pathways and their influence(s) on the activities of other key phytohormones such as abscisic acid (ABA), auxins, cytokinins, ethylene, gibberellins, jasmonates and salicylic acid.
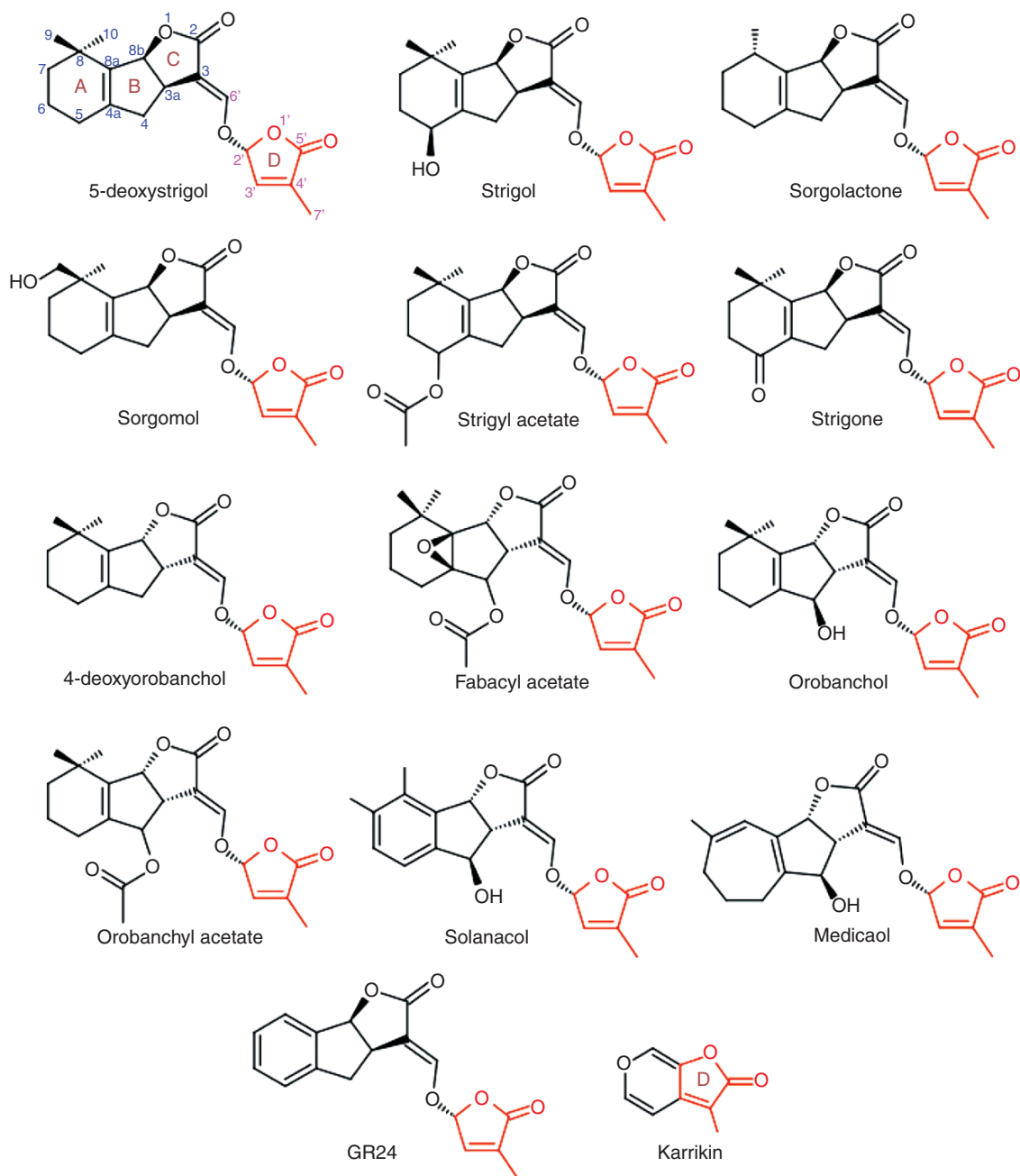
Fig. 1. Chemical structures of canonical strigolactones, karrikin and GR24 (a synthetic strigolactone).

Chemical nature of strigolactones

Endogenous SLs are grouped into two classes, canonical and non-canonical SLs, based on the presence or absence of a tricyclic lactone ring. Canonical SLs consist of a four-ring system designated A, B, C and D (see Fig. 1). The tricyclic lactone is linked to a butenolide moiety, the D ring, via an *enol–ether* bridge. As a result of functional group variability, A and B rings show maximum divergence, whereas C and D are highly conserved (Fig. 1). On the other hand, non-canonical SLs (Fig. 2) are very diverse in the structure of their ABC ring. Carlactone-type SLs possess the A and D rings as well as the *enol–ether* bridge but lack the B and C rings

(i.e. having an unclosed ring) (Fig. 2). Among others in this group are carlactone and its derivatives such as carlactonoic acid, 3-hydroxycarlactone, methyl carlactonoate (Alder *et al.*, 2012; Abe *et al.*, 2014; Seto *et al.*, 2014; Baz *et al.*, 2018), avenaol (Kim *et al.*, 2014) and zealactones (Charnikhova *et al.*, 2017; Xie *et al.*, 2019). Reports of non-canonical SLs which differ structurally from carlactone-type SLs, such as heliolactone (Ueno *et al.*, 2014), zeapyranolactone [with 4,4-dimethyltetrahydropyran-2-one as its A ring (Charnikhova *et al.*, 2018)] and lotuslactone (Xie *et al.*, 2019), suggest that biomolecules with SL activity accommodate a high level of structural plasticity in their ABC rings.
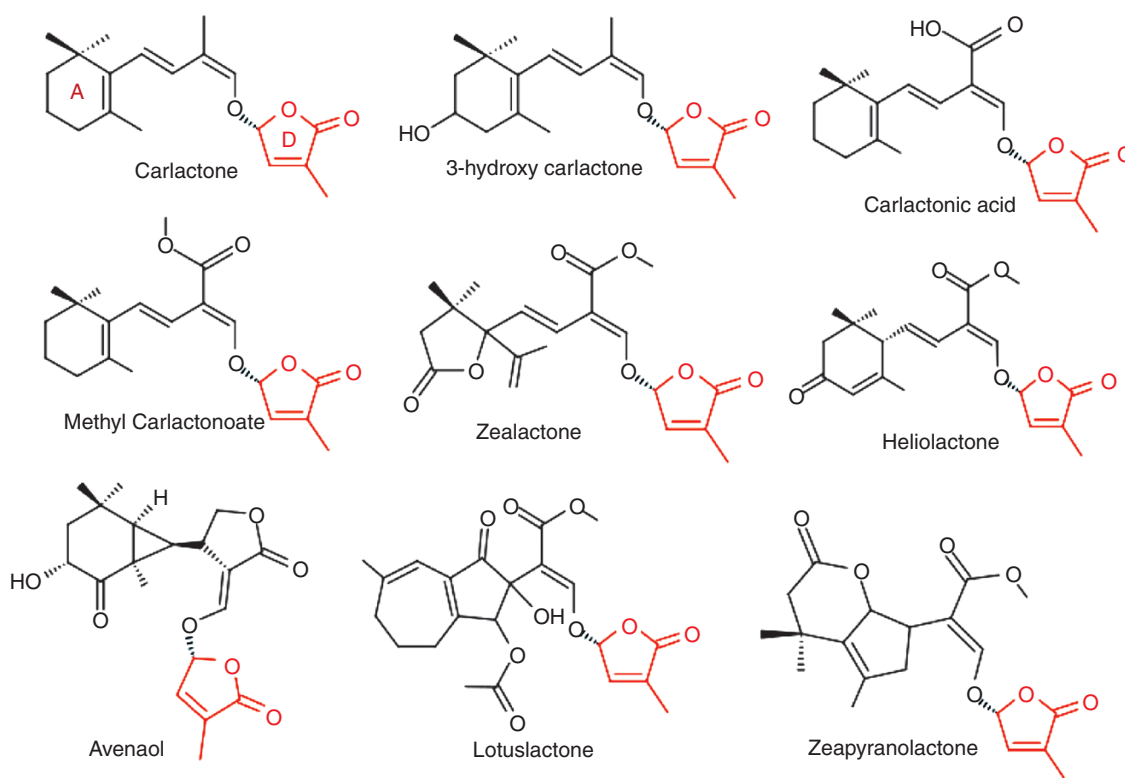
FIG. 2. Structures of non-canonical strigolactones.

With respect to the steric orientation of the BC ring junction, canonical SLs fall into either strigol or orobanchol classes. This stereochemistry has been shown to determine the functional specificity of specific SLs (Akiyama *et al.*, 2010; Nomura *et al.*, 2013). The C-ring in strigol-type SLs is in the β-orientation while in orobanchol-type SLs, it is in the α-orientation. Plants usually produce and transport one of either class of SLs, although both have been observed in some species (Xie *et al.*, 2013, 2016). Several studies including those on structure–activity relationships of SLs in stimulating germination in parasitic plants and hyphal branching in arbuscular mycorrhizal fungi (AMF) have established the butenolide moiety as the essential 'bioactiphore' for SL activity (Akiyama *et al.*, 2010; Kim *et al.*, 2010; Zwanenberg and Pospísil, 2013).

Biosynthesis of strigolactones

SLs are downstream products of carotenoid catabolism. SL biosynthesis occurs mainly in the root, although shoot tissues do produce SLs in lower quantities compared to the root but sufficient for shoot function in the absence of root-derived SLs (Domagalska and Leyser, 2011). For lucidity here, we mainly use rice and *Arabidopsis* gene nomenclature; for orthologues in other plants, see Table 1. The early dedicated steps in the biosynthesis of SLs lead to the production of carlactone, the key precursor of SLs (Seto *et al.*, 2014; Iseki *et al.*, 2017) (Fig. 3). Carlactone is derived from a series of reactions, including: the isomerization of *all-trans*-β-carotene C9–C10 double bonds by DWARF27 (D27) (Alder *et al.*, 2012; Waters *et al.*, 2012*a*; Bruno and Al-Babili, 2016) to form 9-*cis*-β-carotene; the stereospecific cleavage of 9-*cis*-β-carotene to yield 9-*cis*-β-*apo*-10′-carotenal and β-ionone catalysed by carotenoid cleavage dioxygenase 7 (CCD7) (Booker *et al.*, 2004; Bruno *et al.*, 2014); and the conversion of 9-*cis*-β-*apo*-10′-carotenal to carlactone by CCD8 in a combination of reactions which include oxygenation and functional group rearrangements (Alder *et al.*, 2012; Waters *et al.*, 2012*a*). The resulting carlactone is exported into the cytoplasm where it undergoes oxidation, ring closures and functional group modifications in a series of reactions catalysed by MORE AXILLARY GROWTH 1 (MAX1), a class of cytochrome P450 monooxygenases (Booker *et al.*, 2005; Seto *et al.*, 2014; Zhang *et al.*, 2014). Yoneyama *et al.* (2018) demonstrated that MAX1 first converts carlactone to carlactonoic acid which is then used to synthesize other SLs, and this reaction is conserved among plants. In rice, *Os*MAX1 (Os900) serves as a carlactone oxidase which converts carlactone via carlactonoic acid to 4-deoxyorobanchol, the precursor for the orobanchol class of SLs (Zhang *et al.*, 2014; Yoneyama *et al.*, 2018). Just like Os900, *Selaginella moellendorfii* MAX1 homologues, *Sm*-MAX1a and *Sm*-MAX1b, catalyse the conversion of carlactone to 4-deoxyorobanchol (Yoneyama *et al.*, 2018). On the other hand, MAX1 homologues of maize and rice, *Zm*-MAX1b and Os1400 respectively, which also oxidized carlactone to carlactonoic acid, synthesized orobanchol via hydroxylation of 4-deoxyorobanchol (Cardoso *et al.*, 2014; Zhang *et al.*, 2014; Yoneyama *et al.*, 2018). However, *At*-MAX1 only produced carlactonoic acid and its methyl ester from carlactone (Abe *et al.*, 2014; Yoneyama *et al.*, 2018). Brewer *et al.* (2016) identified *LATERAL BRANCHING OXIDOREDUCTASE*

TABLE 1. *Orthology of genes associated with strigolactone biosynthesis and signalling*

Oryza sativa	Arabidopsis thaliana	Petunia hybrida	Medicago truncatula	Striga hermonthica	Protein encoded and function.	Reference(s)
Strigolactone biosynthesis						
D27	At-D27	–	Mt-D27	–	β-carotene isomerase: Converts all-trans-β-carotene to 9-cis-β-carotene.	Liu *et al.* (2011); Alder *et al.* (2012); Waters *et al.* (2012*a*); Challis *et al.* (2013); Seto *et al.* (2014); van Zeijl *et al.* (2015); Bruno and Al-Babili (2016)
Os-CCD7/ D17/HTD1	MAX3	DAD3	Mt-CCD7	–	Carotenoid cleavage dioxygenase: Cleaves β-carotene to form an apo-carotenal.	Booker *et al.* (2004); Zou *et al.* (2006); Simons *et al.* (2007); Challis *et al.* (2013); Bruno *et al.* (2014); Lauressergues *et al.* (2015)
Os-CCD8/D10	MAX4	DAD1	Mt-CCD8	–	Carotenoid cleavage dioxygenase: Carlactone production.	Arite *et al.* (2007); Simons *et al.* (2007); Zhang *et al.* (2010); Challis *et al.* (2013); Lauressergues *et al.* (2015)
Os-MAX1 (Os900, Os1400, Os5100)	MAX1	–	Mt-MAX1 (Medtr3g104560)	–	Cytochrome P450 cytochrome 711 monooxygenase, carlactone oxidase and orobanchol synthase: Converts carlactone to canonical SLs and carlactone-type SLs.	Booker *et al.* (2005); Challis *et al.* (2013); Seto *et al.* (2014); Zhang *et al.* (2014); Yoneyama *et al.* (2018)
Os01g0935400	LBO	–	–	–	2-Oxoglutarate and Fe(II)-dependent dioxygenase: Converts methyl carlactone to a yet to be identified SL active compound.	Brewer *et al.* (2016)
Strigolactone signalling						
D3	MAX2/ ORE9	PhMAX2a, PhMAX2b	Mt-MAX2 (MTR_4g080020)	Sh-MAX2	F-box protein: Component of SCF ubiquitin ligase complex required for signal transduction.	Woo *et al.* (2001); Stirnberg *et al.* (2007); Challis *et al.* (2013); Zhao *et al.* (2015); Yao *et al.* (2017)
Os-D14/D88/ HTD2	AtD14	DAD2	Mt-D14	Sh-HTL7	α/β Hydrolase: SL perception and hydrolysis.	Simons *et al.* (2007); Gao *et al.* (2009); Liu *et al.* (2009); Hamiaux *et al.* (2012); Lauressergues *et al.* (2015); Tsuchiya *et al.* (2015); de Saint Germain *et al.* (2016); Marzec *et al.* (2016); Yao *et al.* (2017); Yao *et al.* (2018)
D53/ D53-LIKE	SMXL6, SMXL7, SMXL8	–	–	–	Class I Clp ATPase: Poly-ubiquitination and proteolytic targets of SL signalling. Transcriptional repressors of SL responses.	Jiang *et al.* (2013); Soundappan *et al.* (2015); Wang *et al.* (2015)

Abbreviations: CCD, CAROTENOID CLEAVAGE DIOXYGENASE; D, DWARF; DAD, DECREASED APICAL DOMINANCE; HTD, HIGH-TILLERING DWARF; HTL, HYPOSENSITIVE TO LIGHT; LBO, LATERAL BRANCHING OXIDOREDUCTASE; MAX, MORE AXILLARY GROWTH; ORE9, ORESARA9; SMXL, SUPPRESSOR OF MAX2 1-LIKE.

(*LBO*), a branching gene in *Arabidopsis* which encodes an oxidoreductase-like enzyme of the 2-oxoglutarate and iron (II)-dependent dioxygenase superfamily. LBO acts downstream of MAX1 to produce an SL-active compound which is yet to be chemically described.

Recent experimental data show that MAX1 is able to catalyse the biosynthesis of both canonical and non-canonical SLs from carlactone (Fig. 3) (Yoneyama *et al.*, 2018); and both carlactone and carlactonoic acid serve as precursors for the biosynthesis of orobanchol- and strigol-type SLs (Iseki *et al.*, 2017; Yoneyama *et al.*, 2018). Furthermore, the inability of cowpea to convert 4-deoxyorobanchol despite converting carlactone and carlactonoic acid into orobanchol and alectrol, and the fact that 5-deoxystrigol was not converted to strigol in
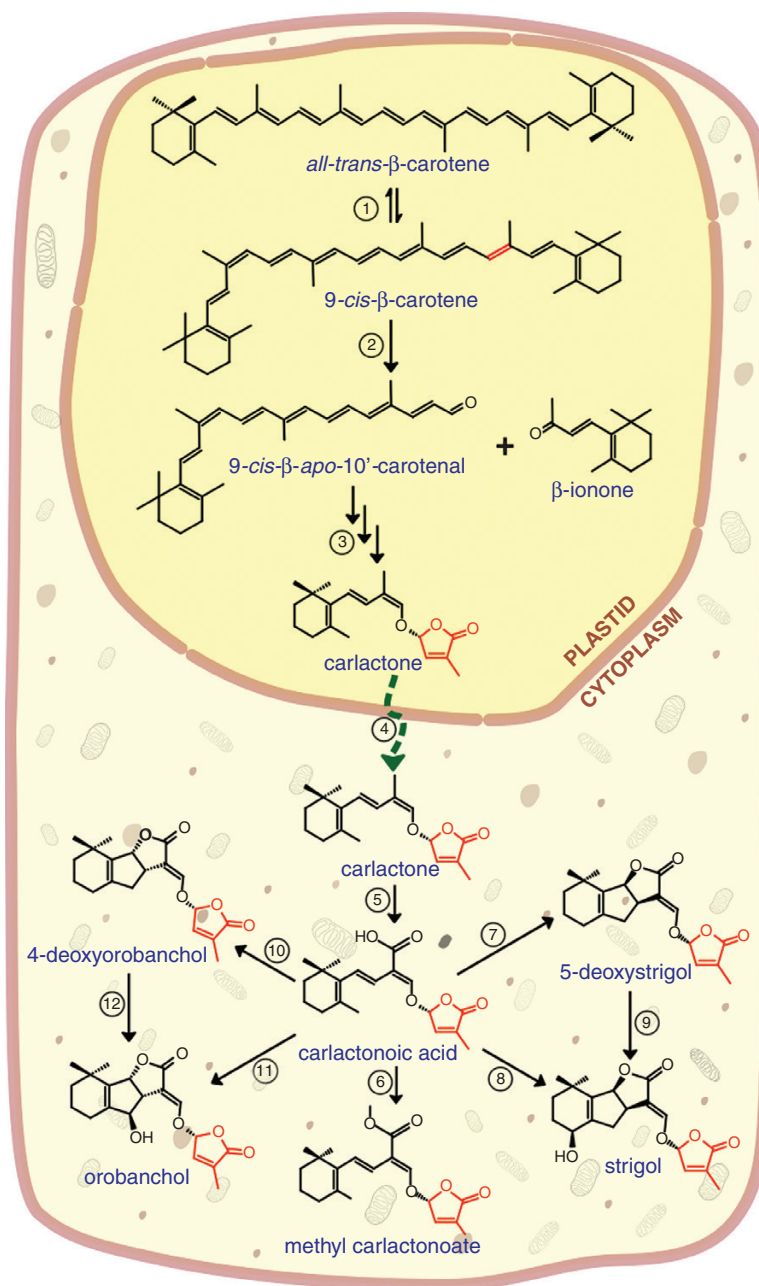
FIG. 3. Strigolactone biosynthetic pathway originating from β-carotene in plastids. ① D27 isomerise *all-trans*-β-carotene to form 9-*cis*-β-carotene; ② CCD7 cleavage of 9-*cis*-β-carotene to yield 9-*cis*-β-*apo*-10′-carotenal and β-ionone; ③ carlactone is produced from 9-*cis*-β-*apo*-10′-carotenal by CCD8; ④ carlactone is exported from the plastid into the cytoplasm; MAX1 catalyses the biosynthesis of ⑤ carlactonoic acid from carlactone and the conversion of carlactonoic acid to ⑥ methyl carlactonoate; ⑦ 5-deoxystrigol; ⑧ strigol directly as seen in moonseed; ⑨ strigol via 5-deoxystrigol as seen in cotton; ⑩ 4-deoxyorobanchol, ⑪ orobanchol directly in cowpea, and ⑫ orobanchol via 4-deoxyorobanchol as in rice.

moonseed (*Menispermum dauricum*) are clear indications that the biosynthesis of hydroxylated SLs does not always require a deoxy SL precursor (Iseki *et al.*, 2017).

Strigolactone transport

Aside those delegated to eliciting local actions within root tissues, there are two major destinations for root-derived SLs: the rhizosphere and the shoot. SLs released into the rhizosphere as part of the root exudate serve as cues to initiate mycorrhizae or nodule formation. They also induce the germination of seeds of root parasitic plants thus mediating such interactions. On the other hand, root-to-shoot transport of SLs serves as a major source of SLs in shoot tissues where they modulate diverse aspects of shoot growth and development. Shoot-ward directional transport of SLs and localized exudation into the rhizosphere is mediated by an ABC subtype G (ABCG)

class of transporter, PLEIOTROPIC DRUG RESISTANCE 1 (PDR1) (Kretzschmar *et al.*, 2012; Sasse *et al.*, 2015). Thus far, the only identified SL transporter is PDR1 from *Petunia axillaris* and *P. hybrida*. Over expression of PDR1 in *P. hybrida* upregulates SL biosynthesis while also influencing SL-related responses such as an increase in root biomass, auxin distribution and mycorrhization (Liu *et al.*, 2018). *Petunia axillaris* PDR1 (*Pa*-PDR1) exhibits a cell-type-specific polar and asymmetrical localization as it is localized on the apical membrane of hypodermal cells in the root apex but laterally localized on the outer membrane of hypodermal passage cells that occur above the root tip and also serve as entry point for AMF during mycorrhization (Sasse *et al.*, 2015). In stem tissues, *Ph*-PDR1 expression occurs in the vasculature and nodal tissues in close proximity to the lateral axils but is absent in dormant bud (Kretzschmar *et al.*, 2012). The expression of these transporters is upregulated by endogenous and exogenous cues that are known to elicit SL production and signalling such as auxin signals, limited inorganic phosphate (P_i) and AMF colonization (Kretzschmar *et al.*, 2012). Furthermore, interference with *Pa*-PDR1 function results in phenotypes that are associated with flawed SL production and signalling (Sasse *et al.*, 2015). The aforementioned observations from localization studies, in addition to impaired SL transport towards the shoot and exudation into the rhizosphere in *Pa-pdr1* mutants (Sasse *et al.*, 2015), as well as reduced SL levels in root exudates of *Ph-pdr1* mutants (Kretzschmar *et al.*, 2012) together establish the roles of PDR1 in SL transport.

In a bid to determine the root-to-shoot path of SL transport, Xie *et al.* (2015b) screened xylem saps from several plant species. SLs were not detected in any of the xylem saps although exogenous SLs applied to roots were detected in shoot tissues after 20 h of treatment. Their observations suggest a cell-to-cell movement of SLs (not via the xylem stream) from the root to the shoot. SL transport was further demonstrated to be a structure- and stereo-specific process (Xie *et al.*, 2016). The literature on the significance of SL transport and its regulation in the growth and development of plants as well as plant–microbe interactions is quite extensive and will not be discussed in detail here. Readers are encouraged to consult excellent recent reviews on the subject by Borghi *et al.* (2016) and Kameoka and Kyozuka (2018).

Strigolactone perception and signal transduction

SL signalling is a hormone-activated cascade of events that culminate in the poly-ubiquitination and proteolysis of specific target proteins as well as the hydrolytic inactivation of the hormone. This process is mediated by three main components: DWARF14 (D14), D3 and a Skp1-Cullin-F-box (SCF) E3 ubiquitin ligase complex. The prevailing SL signalling model (Fig. 4) posits that D14 – the SL receptor/perception protein – is activated by the binding of an SL molecule, and the ligand (SL)-bound D14 then forms a signalling complex with other signalling partners leading to the transduction of the hormonal signal and finally the hydrolysis of the bound SL at the *enol–ether* bridge to deactivate the hormone (Waters *et al.*, 2012b; Hamiaux *et al.*, 2012; Marzec *et al.*, 2016).
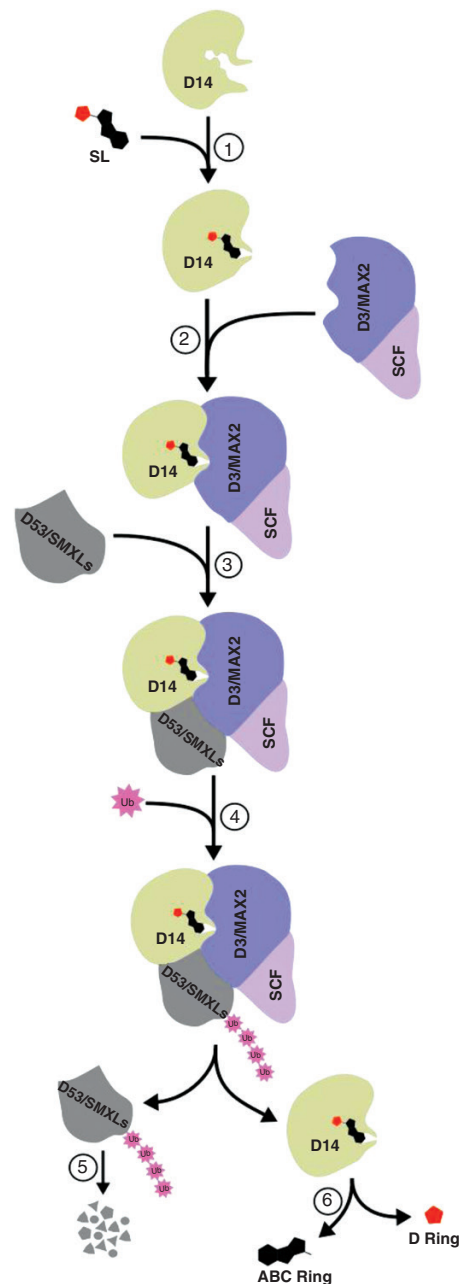

FIG. 4. A model of strigolactone perception and signal transduction pathway. ① SL binds in the catalytic cleft of D14 and induces a destabilization/conformational change in D14; ② SL-induced destabilization of D14 facilitates D14 interaction with SCF^D3; ③ SCF^D3-D14 recruits a target protein (e.g. D53 or SMXLs); ④ D53/SMXLs undergo polyubiquitination; ⑤ proteasomal degradation; ⑥ D14 deactivates the bound SL molecule by hydrolytic cleavage of the enol–ether bridge and releases the ABC and D rings.

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SL signal perception involves ligand (SL) docking in the catalytic cleft of D14, which results in conformational changes (open-to-closed state transition) in D14 (Yao *et al.*, 2016, 2018; Seto *et al.*, 2019). D14 belongs to the α/β serine hydrolase superfamily (Hamiaux *et al.*, 2012; Nakamura *et al.*, 2013; Tsuchiya *et al.*, 2015) and its hydrolytic activity is mediated by a serine–histidine–aspartate catalytic triad located within its active site (Hamiaux *et al.*, 2012; Zhao *et al.*, 2013). The

conformational changes induced in D14 by ligand binding are characteristic of a catalytically inactive state and are due to re-arrangement of the four top helices that constitute a V-shaped lid domain as well as an alteration in the shape of the catalytic triad due to a shift by the loop bearing the catalytic Asp residue (Zhao *et al.*, 2013; Yao *et al.*, 2016; Seto *et al.*, 2019).

Interactions/complex formation involving SL-bound D14, F-box protein and target proteins leading to the poly-ubiquitination and proteasomal degradation of target proteins are believed to underpin SL signal transduction. In its active state, D14 associates with D3 – the F-box protein component of the SCF E3 ubiquitin ligase complex (Stirnberg *et al.*, 2007; Zhao *et al.*, 2013, 2015) – and then a specific protein sub-strate is recruited for poly-ubiquitination and 26S proteasome-mediated degradation (Jiang *et al.*, 2013; Zhao *et al.*, 2015). The lid domain of D14 interacts with D3 (Zhao *et al.*, 2013) via a C-terminal α-helix of a leucine-rich repeat (LRR) domain on D3, with D14 catalytic cavity facing the LRR domain of D3 (Yao *et al.*, 2016; Shabek *et al.*, 2018). The SL-induced D14–D3 interaction further destabilizes D14 (Zhao *et al.*, 2015), but how this specifically bears on SL signal transduction is yet to be de-scribed in detail. According to Shabek *et al.* (2018), SCF$^{D3–D14}$ forms a stable ternary complex with a target protein (e.g. D53) via an ATPase domain on D53. The target protein undergoes proteasome-mediated degradation after poly-ubiquitination (Jiang *et al.*, 2013; Soundappan *et al.*, 2015; Wang *et al.*, 2015).

After SL signal transduction, D14 hydrolyses the bound SL molecule, thus inactivating it. The hydrolysis of SL is induced in order to restore the catalytic triad to its open state conformation (Seto *et al.*, 2019). A nucleophilic attack on the *enol–ether* bridge of the docked SL molecule separates the ABC ring from its D ring. The suggestions that D14 is a single-turnover enzyme and that SL hydrolysis and a SL-derived covalently linked intermediate molecule (CLIM) were central or necessary for SL signal transduction (de Saint Germain *et al.*, 2016; Yao *et al.*, 2016, 2018; Saeed *et al.*, 2017) have been subjects of serious debate. These views have been shown to be inconsistent with recent ex-perimental observations. First, Carlsson *et al.* (2018) – after re-analysing structural data including electron density maps calculated from coordinates of the crystallized SL-induced *At*-D14-D3-ASK1 complex reported by Yao *et al.* (2016) – concluded that what was previously thought to be bound to the active cleft of D14 was not CLIM but probably a compo-nent of the crystallization reagent. By monitoring the timing of *At*-D14 activation relative to SL hydrolysis, Seto *et al.* (2019) demonstrated that the intact SL molecule, not CLIM nor the hydrolysis products, triggers the conformational changes in *At*-D14 and SL signal transduction. Similarly, functional analysis of *At*-D14 catalytic triad mutants with reduced hydrolase activity showed that these mutants were still capable of SL signal transduction and complementation of the *At*-*d14* mutant phenotype despite lacking their hydro-lase activity (Seto *et al.*, 2019). In the same study, missense mutation of a highly conserved amino acid in *At*-D14 and *Os*-D14 significantly influenced SL signal transduction without affecting the hydrolytic function of D14. Together, these are clear indications that SL hydrolysis during SL signalling is not required for signal transduction but serves to deactivate the hormone.

SCF$^{D3–D14}$ target proteins such as SUPPRESSOR OF MAX2 1 (SMAX1) (Stanga *et al.*, 2013), D53 (Jiang *et al.*, 2013; Zhou *et al.*, 2013) and SUPPRESSOR OF MAX2-LIKE6–8 (SMXL6, SMXL7 and SMXL8) are known repressors of down-stream SL responses (Soundappan *et al.*, 2015; Wang *et al.*, 2015). Strigolactone-D14-D3 facilitated proteasomal degrad-ation of these repressors paves way for the activation of the hith-erto repressed transcription factors (TFs), which in turn leads to the transcriptional activation of associated genes. Examples of such TFs repressed by D53/SMXLs are BRANCHED1 (BRC1) (Soundappan *et al.*, 2015), TEOSINTE BRANCHED1 (TB1) (Liu *et al.*, 2017), IDEAL PLANT ARCHITECTURE1 (IPA1) (Song *et al.*, 2017) and SQUAMOSA PROMOTER BINDING PROTEIN-LIKE3/17 (SPL3/17) (Liu *et al.*, 2017). BRI1-EMS-SUPPRESSOR1 (BES1), a positive regulator in brassinosteroid signalling, was suggested to be targeted for degradation via SCF^{MAX2} (Wang *et al.*, 2013), although a con-trasting observation was recently reported by Bennett *et al.* (2016). In addition, D53/SMXL interaction with the TOPLESS (TPL)/TPL-RELATED (TPR) family of transcriptional repres-sors via Ethylene-responsive element-binding factor-associated Amphiphilic Repression (EAR) motifs also suggests a putative role for SL signalling in TPL/TPR-mediated regulation of gene expression (Jiang *et al.*, 2013; Soundappan *et al.*, 2015; Wang *et al.*, 2015; Ma *et al.*, 2017).

Major strigolactone-mediated responses in plants

SLs mediate the regulation of a broad group of developmental processes in plants (Fig. 5). Some of the major responses in plants elicited by SLs are highlighted below.

Germination stimulants for seeds of root parasitic plants. The first SL bioactivity to be demonstrated was the induction of seed germination in root parasitic plants. Strigol, strigyl acetate and sorgolactone act as germination stimulants for *Striga* species. These compounds are secreted in root exudates of cotton, maize and rice, among others (Cook *et al.*, 1972; Hauck *et al.*, 1992). Alectrol and orobanchol were identified as germination stimu-lants for *Alectra vogelii* (Müller *et al.*, 1992) and *Orobanche* species respectively, both of which parasitize some temperate crops (Yokota *et al.*, 1998). Brun *et al.* (2018) provide an exten-sive review on this aspect of SL bioactivity.

Strigolactones as chemical cues for plant–microbe symbi-otic interactions. Another early discovery is SL-induced hy-phal branching in AMF. Akiyama *et al.* (2005) identified 5-deoxystrigol in root exudates of *Lotus japonicus* as a cue that triggered hyphal branching. Other SLs like sorgolactone, strigol, orobanchol (Akiyama *et al.*, 2010) as well as carlactone and its derivatives (Mori *et al.*, 2016) also induced hyphal branching. In addition to the induction of hyphal branching, the roles of SLs in AM symbiosis are evident from the following observations. First, the production of short-chain chitin oligomers, a group of mycor-rhizal factors that are believed to elicit the symbiotic signals ne-cessary for fungal root colonization in plants, is enhanced in AMF by GR24 (Genre *et al.*, 2013). Second, fungal metabolism – evident as increased ATP production/respiration and mitochon-drial division, up-regulation of the expression of mitochondrial genes as well as proteins associated with other cell components
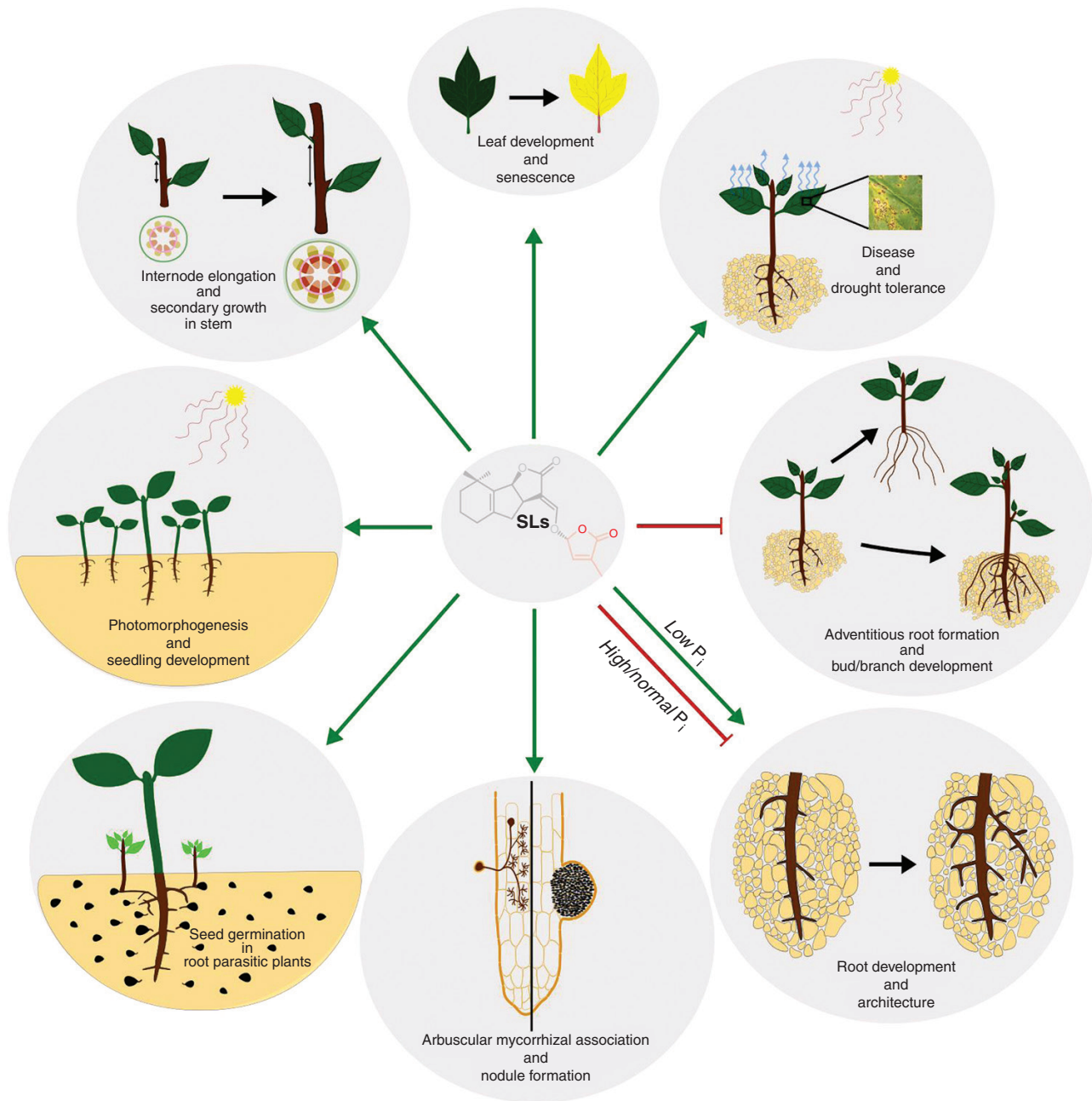
FIG. 5. Major growth and developmental processes mediated by strigolactones in plants. ━━▶, stimulatory effect/regulation; ━━┫, inhibitory effect/regulation.

– is also induced by SLs (reviewed by Lanfranco *et al.*, 2018). Finally, in addition to serving as molecular cues and their effects on the mycosymbiont in the stages just before fungal colonization of the root, SLs also perform endogenous roles within the root during mycorrhizal development. This is evident from the fact that SL-signalling mutants show defective mycorrhizal phenotypes. For instance, *rms4* mutants (defective in SL perception but having relatively normal SL content) have significantly reduced mycorrhizal colonization compared to wild-type (WT) plants (Foo *et al.*, 2013). In a similar manner, *Sl-IAA27*-silenced tomato lines with undetectable SL content showed mycorrhizal defects characterized by a decrease in infection frequency and

arbuscule abundance (Guillotin *et al.*, 2017). Furthermore, AMF-inoculated *d3-1* rice mutants showed defects in the early stages of AM symbiosis and an almost abolished expression of AM-inducible genes (Yoshida *et al.*, 2012). Despite these observations, the endogenous roles of SLs in mycorrhizal development remain largely elusive. It is of note that SL production/exudation does not translate into an ability to form AM associations as non-AM host plants also produce SLs that induce AM activities and exogenous supply of SLs does not result in AM formation in non-host plants (Mori *et al.*, 2016).

The early stages of root nodule organogenesis are characterized by an exchange of chemical signals between the bacterial

symbiont and the host plant. Chemical cues exuded into the rhizosphere by the host plant trigger the expression of bacterial nodulation genes which direct the production of signal molecules such as nodulation (Nod) factors which when perceived by the host plant elicit nodule organogenesis such as root hair curling, infection thread formation, and the attendant division of inner cortical and pericycle cells to form nodule meristem. SL-deficient mutants form fewer nodules, and treatment with GR24 increased nodule number in both mutants and WT plants (Foo and Davies, 2011; Foo *et al.*, 2013; De Cuyper *et al.*, 2015). Unlike in AM symbiosis, SL response mutants displayed enhanced nodulation with increased nodule number (Foo *et al.*, 2013). Attempts to establish how SLs feature in nodulation revealed that Nod factors induce rapid expression of SL biosynthesis genes (Liu *et al.*, 2011; van Zeijl *et al.*, 2015) and SL actions are limited to the formation of infection threads and the expression of some genes that are induced by the Nod factor signal cascade (McAdam *et al.*, 2017).

Regulation of photomorphogenesis. How SLs feature in photomorphogenesis is still emerging, with conflicting reports of SL influences in this aspect of plant development. Shen *et al.* (2012) showed that MAX2 interacts with gibberellin and ABA signalling to mediate light-induced seed germination and CONSTITUTIVE PHOTOMORPHOGENESIS 1 (COP1) to regulate photomorphogenesis, but SLs were not involved. Similarly, seedling photomorphogenic responses were not affected by strigolactone deficiency in *Arabidopsis* (Nelson *et al.*, 2011) or in pea (Urquhart *et al.*, 2015). Despite this evidence, recent studies indicate that SLs are active in photomorphogenesis. For instance, SLs regulate hypocotyl elongation via several processes that require the actions of cryptochrome, phytochrome (Jia *et al.*, 2014) and STH7/BBX20, a member of the B-box zinc-finger protein family, which are known downstream effectors of photomorphogenesis (Thussagunpanit *et al.*, 2017).

Regulation of shoot branching and plant architecture. In shaping the overall structure of plants, SLs control bud activation/branching, secondary growth and root morphology. SL-deficient and signalling mutants exhibit excessive shoot branching, and direct application of SL to axillary buds fully inhibits bud development/branching (Braun *et al.*, 2012; Dun *et al.*, 2013). The influence of SLs on bud development is predominantly mediated via either or both of these two mechanisms (reviewed by Waters *et al.*, 2017). One involves SL-induced direct up-regulation of a broad group of conserved negative regulators of shoot branching such as *BRC1* (Dun *et al.*, 2013), its orthologue in rice, *FINE CULM1* (FC1) (Xu *et al.*, 2015) and *IPA1* (Song *et al.*, 2017). In addition, *BES1* (a gene that encodes a positive regulator of shoot branching) undergoes SL-SCF^MAX2-induced proteasomal degradation (Wang *et al.*, 2013). The other mechanism is auxin dependent and involves SL-induced modulation of polar auxin transport (PAT)/canalization (see discussion below).

In rice, *FC1* acts downstream of SLs to suppress tillering given that *fc1* mutants are insensitive to high doses of GR24 when compared to WT plants with respect to bud growth and tillering. However, unlike *BRC1*, *FC1* expression is not induced by GR24 treatment (Minakuchi *et al.*, 2010), but rather FC1 (reported as *Os*-TB1) perturbs the suppression of D14 transcription by *Os*-MADS57, a TF of the MADS-domain family, by binding to *Os*-MADS57 (Guo *et al.*, 2013). This shows that the interactions between SL signal elements and the BRC1 family of TFs is complex and requires further investigations to develop a lucid picture of how they function to regulate shoot branching.

To promote internode/stem elongation and secondary growth, SLs stimulate cell division/increase in cell number (de Saint Germain *et al.*, 2013) and cambial activity within the stem (Agusti *et al.*, 2011). In addition to shoot elongation, leaf margin serrations in *Medicago truncatula* are also controlled by SLs in a process that appears to be dependent on auxin transport (Lauressergues *et al.*, 2015). Leaf senescence, which marks the last stage of leaf development, is also regulated by SLs (Yamada *et al.*, 2014). Leaves of the *oresara9* (*ore9*) mutant of *Arabidopsis* exhibit delayed onset of senescence. *At-ORE9* is identical to *At-MAX2* (Woo *et al.*, 2001; Stirnberg *et al.* 2007). Leaf senescence is accelerated under phosphate limiting conditions and in the presence of exogenous SL (Yamada *et al.*, 2014; Ueda and Kusaba, 2015; Tian *et al.*, 2018).

SLs mediate the tuning of crucial aspects of root architecture. This is evident in: the restoration of normal primary root length in SL mutants of *Arabidopsis* by GR24 (Ruyter-Spira *et al.*, 2011); GR24-induced suppression of adventitious root (AR) formation in *Arabidopsis* and pea (Rasmussen *et al.*, 2012) while promoting crown root growth and AR elongation in rice (Arite *et al.*, 2012; Sun *et al.*, 2014); the repression of lateral root (LR) formation (Kapulnik *et al.*, 2011*a*, *b*; Ruyter-Spira *et al.*, 2011, De Cuyper *et al.*, 2015); and the regulation of root hair elongation (Kapulnik *et al.*, 2011*a*, *b*). Sun *et al.* (2016) and Marzec and Melzer (2018) provide detailed reviews of SLs as regulators of root development.

Proteoid (cluster) roots, a unique morphological adaptation of roots to limiting nutrient conditions, are found in certain species of families such as Casuarinacea, Fabaceae and Myricacea but are almost ubiquitous among Proteaceae (Watt and Evans, 1999; Shane and Lambers, 2005). The development of this particular root structure has been linked to limiting phosphate conditions and is driven by phosphate supply (Neumann *et al.*, 2000; Felderer *et al.*, 2015). Interestingly, SL biosynthesis is induced in plants under low phosphate conditions (Yoneyama *et al.*, 2007; Koltai 2013). Despite the aforementioned, and that SLs feature prominently in the regulation of root architecture, experimental data on the role of SLs in proteoid root development are still lacking.

Mediation of plant tolerance to nutrient deficiency. Under limited nutrient conditions, especially in phosphate- and nitrogen-deficient soils, plants increase SL biosynthesis which becomes evident as an increase in SL content of root exudates (Yoneyama *et al.*, 2007, 2012; Koltai, 2013, 2015). This is significant because high levels of SLs facilitate root branching (Ruyter-Spira *et al.*, 2011) while also increasing the molecular signal content of root exudates for the establishment of symbiotic associations with AMF. Under limiting P_i conditions, Mayzlish-Gati *et al.* (2012) found both SL-deficient (*max4-1*) and SL-insensitive (*max2-1*) mutants had reduced root hair densities. Also, treatment with GR24 increased root hair density in *max4-1* to similar levels as in the WT but did not significantly alter root hair densities in *max2-1*. These processes promote efficient mobilization of limited nutrients in soils (Akiyama

et al., 2010; Kapulnik *et al.*, 2011a; Liu *et al.*, 2018). SLs may also modulate P_i utilization in addition to P_i acquisition in plants under limited P_i. Czarnecki *et al.* (2013) suggested that SL-induced suppression of shoot branching in *Arabidopsis* in response to P_i deficiency may be a means to reduce P_i utilization. SLs also regulate plant responses to nitrogen supply. Both SL-deficient (*d10* and *d27*) and SL-insensitive (*d3*) mutants of rice lack the root responses to low P_i and nitrogen concentrations observed in the WTs (Sun *et al.*, 2014). In addition, GR24 treatments compensate for the reduced responses in SL-deficient mutants but not in the SL-insensitive mutants. Genetic evidence for the role of SLs in plant responses to nitrogen deficiency was also provided by Ito *et al.* (2016). They reported an alteration in the expression of *MAX3* and *MAX4* under nitrogen-deficient conditions.

Mediation of plant responses to abiotic and biotic stressors. The role of SLs in plant responses to abiotic stress cues still requires more research attention. However, available data portray SLs as positive regulators of abiotic stress responses. Stomatal closure, a process that fosters drought tolerance by reducing water loss via transpiration, is induced by SLs (Lv *et al.*, 2018; Zhang *et al.*, 2018). Similarly, SL-deficient and SL-responsive mutants exhibited hypersensitivity to drought (Bu *et al.*, 2014; Ha *et al.*, 2014; Li *et al.*, 2016) with shoots receiving little SL from the root behaving as if they were under mild drought stress even when supplied with enough water (Visentin *et al.*, 2016). Furthermore, treatment with exogenous SLs rescued drought-sensitivity in SL-deficient mutants but not in signalling mutants (Ha *et al.*, 2014) while also enhancing drought tolerance in WTs. Under salinity stress, the actions of ABA in ameliorating the deleterious effects of salinity was seen to be dependent on SL biosynthesis and signalling (Ren *et al.*, 2018).

In establishing the roles of SLs in the regulation of plant responses to biotic stressors, Cheng *et al.* (2017) showed that SL-deficient tomato plants displayed increased susceptibility to *Phelipanche ramosa* infection and rapid development of the parasite. A report by Torres-Vera *et al.* (2014) also indicated that SL-deficient tomato mutants, *Sl-ccd8*, were susceptible to foliar fungal pathogens, *Botrytis cinerea* and *Alternaria alternata*, and this was found to be coupled to a reduction in the concentrations of defence-related hormones such as jasmonates, salicylic acid and ABA.

Strigolactone interactions with other phytohormones

Strigolactone crosstalk with auxin signalling. Endogenous auxins act in concert with other hormones, either synergistically or antagonistically, to regulate biological processes in plants (Naseem *et al.*, 2015; Liu *et al.*, 2017; Leyser, 2018). Several studies have evinced links between auxin and SL signalling. Auxin influence on SL signalling is largely via modulating SL biosynthesis, which in turn may act as a second messenger in eliciting some auxin-dependent responses. The expression of CCD7- and CCD8-encoding genes are up-regulated by auxin to exert control on shoot branching (Zou *et al.*, 2006; Arite *et al.*, 2007). Hayward *et al.* (2009) found these to be an AUXIN RESISTANCE1 (AXR1)-dependent process. Experimental data are lacking on the role(s) of auxin in SL flux and transport.

In regulating shoot branching, SLs interfere with PAT/distribution and, in effect, auxin canalization (Bennett *et al.*, 2006; Ruyter-Spira *et al.*, 2011; Sun *et al.*, 2014) (Fig. 6). Auxin flux/canalization is a significant factor in bud activation and outgrowth (Prusinkiewicz *et al.*, 2009; Balla *et al.*, 2011, 2016). SL-mediated down-regulation of the expression of PIN-FORMED (PIN) proteins, a family of transporters responsible for auxin influx and efflux from cells, and their polarized localization on the plasma membrane (Crawford *et al.*, 2010; Shinohara *et al.*, 2013; Hu *et al.*, 2018) dampens auxin sink strength of the PAT stream, thus hampering auxin export from buds and canalization, which ultimately results in the repression of bud development. However, reports that diminished auxin transport in pea had little inhibitory effects on bud outgrowth in SL-deficient mutants and the ability of SLs to inhibit bud outgrowth in pea plants with impaired auxin transport (Brewer *et al.*, 2015) indicate that although PAT/canalization may be used in SL-mediated modulation of branching, SLs can also act independent of this mechanism.

Experimental observations have revealed inconsistencies in the two models of SL action in bud activation. Among these are: the uncoupling of the *TB1* sub-network from SL signalling in maize (Guan *et al.*, 2012); the insensitivity of *FC1* expression to GR24 (Minakuchi *et al.*, 2010); buds lacking *BRC1* expression remaining inhibited and being sensitive to inhibition by SLs, buds with high *BRC1* transcripts being active (Seale *et al.*, 2017); and conflicting reports of SL influence on auxin transport and canalization mentioned above. All these call for a rethink of these models with a view to developing one which takes these limitations into consideration. The way forward may lie in the events that occur upstream, in parallel with and/or downstream of BRC1/FC1/TB1 activities. Auxin canalization and the repression of branching factors may be necessary for bud activation/development, and some other mechanisms which may be under the influence of these two hormones may determine whether an activated bud proceeds to develop into a branch. Research efforts tailored towards investigating how nutrient partitioning affects development among competing buds will also provide progress in illuminating this aspect of plant development. For instance, sucrose features significantly in apical dominance, promoting bud release and down-regulation of *BRC1* (Mason *et al.*, 2014), so how SL and other key hormonal signals are integrated with sugar signals during bud activation should be a subject for further investigation.

The regulation of secondary growth by SLs occurs by positively modulating interfascicular cambial activity (Agusti *et al.*, 2011). The slight induction of cambium-like cell division in *max2* plants by GR24 treatment, which contrasts with the complete insensitivity observed in other processes, suggests other factors act in parallel with MAX2-dependent SL signalling to affect secondary growth (Agusti *et al.*, 2011). In addition, observations made using *max* and auxin response mutants suggest SLs affect secondary growth directly, independent of auxin accumulation, and they act downstream of auxin. This then raises the question of how SLs elicit cambial activity and secondary growth if auxin-induced cambial activity is not employed.

As with shoot branching, SLs interact with auxin to control root development by modulating auxin sensitivity (Mayzlish-Gati *et al.*, 2012), PAT from shoot to root (Sun *et al.*, 2014) and
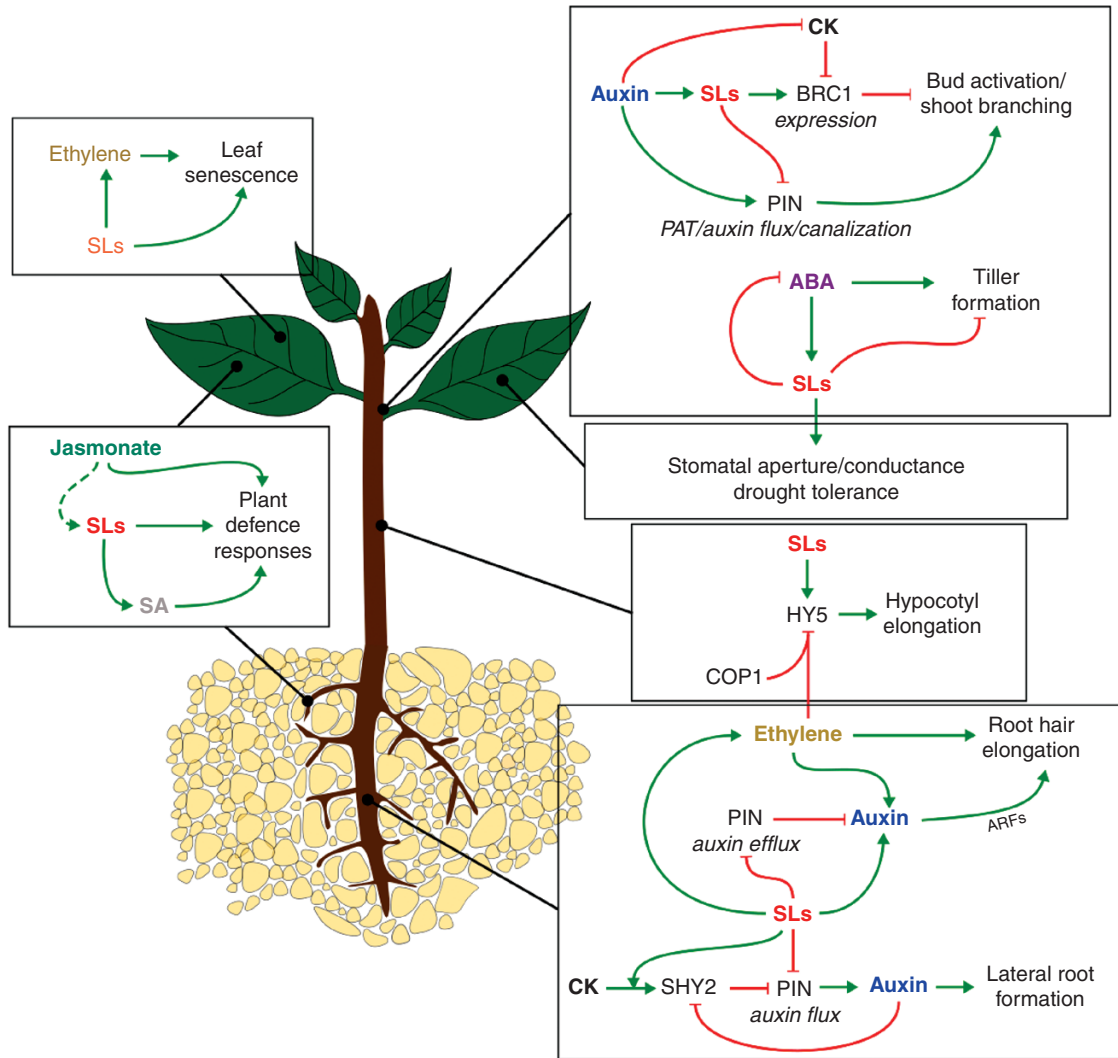
FIG. 6. Strigolactone interactions with abscisic acid, auxin, cytokinin, ethylene and salicylic acid. Abbreviations: ABA, abscisic acid; BRC1, BRANCHED1; CK, cytokinin; COP1, CONSTITUTIVE PHOTOMORPHOGENESIS1; HY5, ELONGATED HYPOCOTYL5; PDR1/6, PLEIOTROPIC DRUG RESISTANCE1/6; PIN, PIN-FORMED; SHY2, SHORT HYPOCOTYL2; SA, salicylic acid; SLs, strigolactones. ➡, stimulatory effect/regulation; ➡ (dashed), possible stimulatory effect; ⊣, inhibitory effect/regulation.

auxin flux within root tissues (Koren *et al.*, 2013; Kumar *et al.*, 2015). Here, auxin signal modules act downstream of SLs. The early steps in LR formation – which includes the priming of pericycle cells in the basal meristem to specify them for LR initiation, transition from founder cells to LR initiation and primordium formation, and the development of LR primordium until LR emergence – are all driven by local auxin gradients and response maxima (reviewed by Olatunji *et al.*, 2017). Since GR24 have been shown to affect the polarization and localization of PIN proteins as well as LR-forming potential (Ruyter-Spira *et al.*, 2011; Pandya-Kumar *et al.*, 2014; Kumar *et al.*, 2015), SLs probably influence these processes by modulating local auxin flux and homeostasis that is required to establish the auxin response maxima.

Similarly, the inhibitory effects of SLs on auxin efflux via SLs modulation of PIN activities also result in the accumulation of auxin within primary root meristem cells. This promotes cell division and number, expansion of meristem and

transition zone sizes and consequently primary root length (Ruyter-Spira *et al.*, 2011). Similar crosstalk between SLs and auxin occurs in the regulation of root hair development where SLs induce an increase in auxin accumulation/levels within epidermal cells by modulating auxin efflux (Koltai *et al.*, 2010) to promote root hair elongation. ROOT HAIR DEFECTIVE 6-LIKE 4 (RSL4) is an auxin-responsive TF which positively regulates root hair formation by controlling genes associated with root hair morphogenesis and is suggested to be an integrator of internal and external cues (Yi *et al.*, 2010). P$_i$ deficiency significantly enhances RSL4 synthesis and half-life (Datta *et al.*, 2015). Against these backgrounds, it is possible that RSL4 might serve as a nexus for SL-auxin crosstalk in modulating root hair development. Experimental data suggest ethylene features prominently in SL-auxin crosstalk in the regulation of root hair growth (Kapulnik *et al.*, 2011b) (see section on *Strigolactone crosstalk with ethylene signalling*).

In regulating AR formation, SLs and auxin elicit opposing effects. The role of auxin in promoting AR formation and the inhibition of the same by SLs have been shown experimentally. In a bid to unravel the nature of SL–auxin crosstalk in AR development, Rasmussen *et al.* (2012) discovered that GR24 can partially revert the stimulatory effects of auxin on AR formation while auxin increased the number of ARs in *max* mutants of *Arabidopsis* and pea. Of note is the fact that the inhibitory effects of SLs occurred even in the presence of elevated auxin content. These findings suggest both hormones act independently and SL suppression of AR formation might not be mediated by limiting local auxin build-up. In a rather contrasting manner, rice mutants flawed in SL biosynthesis and signalling exhibited reduced AR formation, and GR24 treatment increased AR number in SL-deficient mutants but not in signalling mutants (Sun *et al.*, 2015). Modulation of PAT also seemed to be employed to regulate AR formation in rice. Taken together, it appears SL–auxin crosstalk in this process is a complex one and the actions of SLs in regulating AR formation might be species-dependent. Further investigations of this process in other species and the effects of SL signals on the expression and stability of downstream targets of auxin signals involved in AR development, such as ADVENTITIOUS ROOTLESS 1 (an auxin-responsive factor involved in auxin-mediated cell dedifferentiation and AR formation; Liu *et al.*, 2005), will be invaluable in drawing valid conclusions on SL–auxin interactions in AR development.

Plant P_i status is critical in SLs' influence on root development and architecture. Under normal/high P_i levels, LR development is inhibited but stimulated under P_i-limiting conditions (Ruyter-Spira *et al.*, 2011; Mayzlish-Gati *et al.*, 2012; Jiang *et al.*, 2015). Although it is clear that SLs serve to translate ambient signals into growth cues in the root, the precise mechanism that enables SLs to delineate their influence under normal P_i levels from those under P_i-limiting conditions is yet to be fully described. A possible avenue for exploration is how SLs influence ethylene signalling under P_i status because ethylene is known to stall auxin-driven LR development (Lewis *et al.*, 2011). In addition, the molecular and/or genetic events that relay P_i status cues to induce SL biosynthesis and signalling await full characterization.

The hormonal interactions between auxin and SLs during AM and rhizobial symbiosis are not well characterized. Experimental observations by Foo (2013) suggested that auxin regulates the early stages of AM symbiosis by modulating SL levels. Recently, the expression of *Sl-IAA27* – a downstream component/repressor of auxin signalling – was shown to be up-regulated by AM colonization and *Sl-IAA27*-silencing adversely affected AM colonization (Guillotin *et al.*, 2017). Interestingly, aside the down-regulation of genes involved in SL biosynthesis in *Sl-IAA27*-silenced plants, treatment with GR24 complemented the mycorrhizal defect by increasing infection frequency and arbuscule abundance. These demonstrate clearly the existence of a yet-to-be identified hormonal signal link between auxin and SLs in mycorrhizal development. The signal channel(s) by which *Sl-IAA27*-induced SL production and signalling influence arbuscule development also remains to be elucidated.

Computer modelling and experimental data have established the crucial role of auxin accumulation in nodule organogenesis (reviewed by Kohlen *et al.*, 2017). Given the impact of SLs in modulating auxin transport and flux, it is possible that SLs might elicit some influence on nodule development via this channel.

Strigolactone crosstalk with cytokinin signalling. The interactions between cytokinin and SLs seem to vary with physiological processes. They act independent of each other in adventitious rooting (Rasmussen *et al.*, 2012), antagonistically in bud activation and shoot branching (Dun *et al.*, 2012; Xu *et al.*, 2015; Manandhar *et al.*, 2018), but synergistically in the regulation of LR development (Jiang *et al.*, 2015). Both hormones regulate expression of the other's biosynthesis genes (Dun *et al.*, 2012; Zhang *et al.*, 2010). While SL-associated genes have been shown to mediate cytokinin biosynthesis and export from the root (Beveridge, 2000; Dun *et al.*, 2012), reports on cytokinin influence on SL transport are lacking.

In mediating bud activation/shoot branching, SLs and cytokinin interact directly in buds with their actions converging at the transcriptional regulation of *BRC1* in *Arabidopsis* and pea (Aguilar-Martinez *et al.*, 2007; Braun *et al.*, 2012; Dun *et al.*, 2012) and *FC1* in rice (Xu *et al.*, 2015) (Fig. 6). Given the above, any factor that influences SLs and cytokinin homeostasis in the bud will determine bud development. This is where auxin features; its induction of SL biosynthesis but inhibition of cytokinin biosynthesis in the root ensures more SLs get to the shoot and repress branching. To shed more light on cytokinin–SL crosstalk in regulating bud development, the influence(s), if any, of SLs/SL signals on downstream cytokinin signal components/pathways involved in bud activation must be elucidated. In resolving the role of sucrose mentioned above, other crucial questions to be investigated include: does sucrose supply affect SLs and/or cytokinin homeostasis in the bud? If yes, what are the molecular mechanisms that drive this process and at what point in this cascade of events is the sucrose signal integrated?

On the other hand, cytokinin signalling components are required in GR24-induced suppression of LR development (Jiang *et al.*, 2015). The repression of LR development by GR24 under sufficient P_i, by down-regulating the expression of PIN genes, is mediated by AHK3, ARR1 and ARR12 via *SHORT HYPOCOTYL2* (*SHY2*). Because auxin transport is required to develop an auxin gradient, which is vital for the induction of LR initiation from pericycle founder cells, SHY2 repression of PIN activity stalls LR formation (Goh *et al.*, 2012). A loss-of-function mutation in *SHY2* results in an insensitivity to GR24 with respect to LR development (Koren *et al.*, 2013). SHY2 is also central to the balancing of auxin and cytokinin signalling, with cytokinin promoting its expression and auxin promoting its degradation (Dello Ioio *et al.*, 2008; Moubayidin *et al.*, 2010). It is clear that SHY2 acts as a nexus for the integration of auxin, cytokinin and SL signalling to regulate LR development in P_i-sufficient conditions, although whether SLs use this same channel and crosstalk to promote LR development under P_i-limiting conditions is yet to be determined.

The relative rate of cell division to cell differentiation is a key driver of growth in root meristem. Growth, fostered by cell division, is repressed when the rates of cell division and differentiation reach a balance after final meristem size is attained. Cytokinin promotes cell differentiation and hence reduces meristem size (Dello Ioio *et al.*, 2007, 2008) while GR24

promotes an increase in meristem cell number and size (Ruyter-Spira *et al.*, 2011; Koren *et al.*, 2013). As with LR development, SHY2 could be a converging point for the antagonistic interplay between cytokinin and SLs in regulating primary root growth. This is supported by observations such as the insensitivity of a loss-of-function mutant, *shy2-31*, to GR24 with respect to meristem cell number/size (Koren *et al.*, 2013); the induction of *SHY2* transcript expression by cytokinin (Dello Ioio *et al.*, 2008; Moubayidin *et al.*, 2010); and the reduced sensitivity of *shy2-31* and *max2* to cytokinin (Koren *et al.*, 2013). However, how both hormones use a single channel to elicit the repression of LR formation but induce opposing effects during primary root growth needs further investigation.

Strigolactone crosstalk with abscisic acid signalling. With an increase in experimental data suggesting an active role for SLs in the mediation of plant responses and resilience to abiotic and biotic stressors, it is expected that SLs might interact, either directly or indirectly, with ABA in the regulation of adaptive stress responses in plants. So far, three highpoints can be inferred from available data with regard to ABA–SL interactions. First, ABA acts upstream of SLs and SL production/signalling is required and modulated to elicit ABA-mediated responses. In support of this view are the observations that the expression of SL biosynthesis genes and SL content are significantly lowered in ABA-deficient plants (López- Ráez *et al.*, 2010; Wang *et al.*, 2018); SL-deficient and signalling mutants are hypersensitive to drought stress, are less sensitive to exogenous ABA (Ha *et al.*, 2014; Visentin *et al.*, 2016) and exhibit reduced stomatal sensitivity to ABA (Bu *et al.*, 2014; Liu *et al.*, 2015; Lv *et al.*, 2018); an increase in stomatal aperture and water loss caused by infection-induced increases in ABA levels were not seen in SL-deficient lines (Cheng *et al.*, 2017); and, finally, ABA up-regulated the expression of SL production and signalling genes to mediate tolerance to salt stress in *Sesbania cannabina*, but was only able to induce partial and transient increases in salt tolerance of plants treated with an inhibitor of SL synthesis (Ren *et al.*, 2018). Second, SL and ABA interactions are antagonistic in regulating certain aspects such as shoot architecture and fruit ripening. ABA suppresses the expression of SL biosynthesis and signalling genes to enhance tiller formation (Wang *et al.*, 2018). On the other hand, rather than promoting ABA action, exogenous application of GR24 markedly inhibited ABA-induced accumulation of sugars and anthocyanins in grape berries (Ferrero *et al.*, 2018). Finally, in addition to being a requirement for ABA-mediated responses, SLs may also serve as a feedback channel in modulating ABA signalling. This is evident from reports that treatment with GR24 resulted in the reduction of endogenous ABA concentrations: GR24 inhibited the stress-induced increase in ABA levels, repressed the transcriptional activation of ABA biosynthetic genes in *Lotus japonicas* root (Liu *et al.*, 2015) and up-regulated the expression of ABA catabolic genes but not biosynthetic genes (Ferrero *et al.*, 2018). Also in support of this view is the observed high ABA content in SL-deficient and SL-insensitive mutant rice plants with greater tolerance to drought (Haider *et al.*, 2018).

Despite all the above, some experimental observations suggest inconsistencies. For instance, SL-deficient mutants did not display the rapid water loss and hypersensitivity to ABA and drought observed in SL-signalling mutants, thus suggesting

MAX2 signalling, but not SLs, is involved in drought responses (Bu *et al.*, 2014). These allude to the existence of a complex interaction between SLs, ABA and their downstream signalling components that only further extensive investigations would unravel. Given that the expression and accumulation of ELONGATED HYPOCOTYL 5 (HY5) (a negative TF and an established integrator of light and ABA responses; Chen *et al.*, 2008) is under the control of SLs (Jia *et al.*, 2014), it is possible that HY5 might be one of the missing links in ABA–SL crosstalk. To further determine if in fact SLs serve as feedback modulators of ABA actions, the idea that SL signals might interfere and dampen ABA activity by influencing Type 2C protein phosphatases and SNF1-related protein kinases, key components of ABA signalling, is worth investigating.

Strigolactone crosstalk with gibberellin signalling. Although still largely unclear, SL–gibberellin crosstalk is implied by some experimental observations. For instance, Lantzouni *et al.* (2017) reported an additive transcriptional change in an overlapping set of genes in response to gibberellin and GR24. Their data also suggested an antagonistic effect by gibberellin on SL-induced up-regulation of some ABC-type transporters. Furthermore, *in silico* analysis showed that SL biosynthesis is under gibberellin regulation (Marzec and Muszynska, 2015) which was later demonstrated experimentally to be mediated via a GID1-DELLA signal pathway (Ito *et al.*, 2017). In the control of branching and tillering, the observations that *ORYZA SATIVA HOMEOBOX 1* (*OSH1*) expression is up-regulated in highly branched gibberellin-deficient mutants (Lo *et al.*, 2008) and SL-signalling mutants (Gao *et al.*, 2009) suggests the sharing of a similar molecular mechanism by SLs and gibberellin, although this has yet to be demonstrated experimentally.

Attempts have been made to find a direct molecular link between downstream components of gibberellin signalling and those of SLs. For instance, D14 interacts with SLENDER RICE1 (SLR1) – a DELLA protein which is a downstream signal component and repressor of gibberellin signalling – in an SL-dependent manner (Nakamura *et al.*, 2013). Despite this, SL-induced degradation of SLR1 is yet to be demonstrated experimentally and the functional relevance/downstream effect(s) of this interaction are still largely unknown. Recent attempts at finding a molecular nexus between SL and gibberellin signalling have yielded data that support a functional independence between both hormones. For instance, the stability (Bennett *et al.*, 2016) and accumulation (Lantzouni *et al.*, 2017) of DELLA proteins were not affected by SL signalling. Experimental data are lacking regarding the influence of SL signals on the stability and transcriptional regulation of vital components of gibberellin signalling such as GIBBERELLIN INSENSITIVE DWARF1 and 2 (GID1 and 2), GIBBERELLIC ACID INSENSITIVE (GAI), SLEEPY1 (SLY1) and SNEEZY (SNE) (Nelson and Steber, 2016). Further supporting a functional diversification for both hormones is the report that SLs stimulate the elongation of internodes independent of gibberellin by increasing cell number and not cell length (de Saint Germain *et al.*, 2013). Against the backdrop of the above, it is likely that SL and gibberellin crosstalk is limited, or might not be mediated by direct interactions between signalling components, and inhibition of SL synthesis by gibberellin serves only to modulate SL content. Whatever the case may be, further

investigations are required to reveal the true nature of SL–gibberellin crosstalk.

Strigolactone crosstalk with ethylene signalling. Ethylene signals are involved in the control of some growth and developmental processes that are also regulated by SLs. Among these are hypocotyl growth, root hair elongation, seed germination and leaf senescence (Fig. 6). During hypocotyl development, the COP1–HY5 complex acts as an integrator of light and hormonal signalling in regulating hypocotyl growth. In light, SLs inhibit hypocotyl elongation by up-regulating HY5 expression in a MAX2-dependent process (Jia *et al.*, 2014) while ethylene enhances COP1-mediated degradation of HY5 to promote hypocotyl elongation (Yu *et al.*, 2013). Together, these observations suggest that an antagonistic interaction between SLs and ethylene signalling might exist in regulating hypocotyl growth. Because hypocotyl growth is rapidly arrested in light, an important question is how SLs override the promoting effects of ethylene on hypocotyl growth in light.

SL control of root hair elongation is also dependent on ethylene signalling. This is evident as ethylene signalling mutants, *At-ein* and *At-etr*, displayed reduced sensitivity to GR24 treatment and blockage of ethylene production completely eliminated SL effects on root hair elongation (Kapulnik *et al.*, 2011*b*). Furthermore, GR24 treatment enhanced *At-ACS2* transcription, a gene involved in ethylene biosynthesis (Kapulnik *et al.*, 2011*b*). Observations from the same study also suggest SLs use ethylene, which may in turn recruit auxin signalling, thus implying the existence of a three-way hormonal crosstalk in the control of root hair development. Nonetheless, the report that a transcriptional complex – consisting of an ethylene-activated factor and a positive regulator of hair cells – co-activates a hair length-determining gene (ROOT HAIR DEFECTIVE 6-LIKE) and other root hair genes (Feng *et al.*, 2017) shows that ethylene can act independently and does not always require auxin signalling to influence root hair development.

Further demonstrating a crosstalk between SLs and ethylene, an early report indicated that SLs induced the biosynthesis of ethylene in the seeds of *Striga* prior to germination (Sugimoto *et al.*, 2003). In promoting leaf senescence, SLs also acted by activating ethylene-mediated senescence signals, although ethylene-independent pathways may be employed (Ueda and Kusaba, 2015). Taken together, in addition to suggesting that ethylene acts downstream of SLs, the above shows that SL-mediated responses that require ethylene signalling are elicited by modulating ethylene content in tissues. Also supporting the view that ethylene may act downstream of SLs is ethylene control over AR initiation sites in *Arabidopsis* hypocotyl, which is independent of SLs (Rasmussen *et al.*, 2017). Regardless of these, the precise molecular channels used by SLs to elicit the transcriptional control of ethylene production remains undescribed. Because light is a potent regulator of ethylene biosynthesis – via processes that are mediated by phytochrome/phytochrome interacting factors (reviewed by Zdarska *et al.*, 2015) – and given that some SL responses are elicited via cryptochrome and phytochrome (Jia *et al.*, 2014), the idea that SLs might modulate ethylene signalling in a phytochrome-dependent manner is worth investigating.

Again, as with gibberellins, describing a lucid and complete model of SL–ethylene crosstalk in regulating developmental processes in plant is still beyond reach with extant experimental data. Providing answers on how D14/MAX2 signalling and D53/SMXL activity affect key ethylene signalling components and ethylene-responsive TFs and vice versa will be invaluable in understanding the role of SL–ethylene hormonal and signalling crosstalk(s) in processes such as root development, fruit ripening and senescence.

Strigolactone crosstalk with jasmonate signalling. Jasmonates, a group of plant oxylipins, mediate a range of physiological processes during vegetative growth, secondary metabolism, plant–insect and plant–pathogen interactions, wounding, etc. The nature of SL–jasmonate crosstalk cannot be clearly described at this point due to limited experimental data. However, the report that jasmonic acid (JA) content and the expression of a jasmonate-dependent gene, *PINII* (a gene responsible for tomato resistance to *Botrytis cinerea*) were reduced in an SL-deficient tomato mutant (*Sl-ccd8*) (Torres-Vera *et al.*, 2014) points to a connection between SLs and jasmonates in disease tolerance. Further alluding to this link is the fact that methyl jasmonate, a plant defence signalling molecule, exerts some influence on *Nicotiana tabacum PDR6* (*Nt-PDR6*), which is an orthologue of the SL transporter gene, *Ph-PDR1* (Xie *et al.*, 2015a). Although strongly induced by phosphate starvation and also involved in the regulation of shoot branching (Xie *et al.*, 2015a), *Nt-PDR6* has not been reported to be involved in SL transport. A rather inconsistent observation was reported recently in which GR24 supply did not affect JA accumulation in WT plants, *At-max1* and *At-max2*, nor did inoculation with *Mucor* sp. (Rozpądek *et al.*, 2018). Together, these observations are not sufficient to draw valid conclusions on the nature of SL–jasmonate crosstalk, but the fact that both hormones elicit responses in similar developmental processes – such as mesocotyl elongation, senescence and plant–microbe interactions – offer some indications that SL–jasmonate crosstalk is likely to feature actively in these processes and cannot be completely ruled out.

Strigolactone crosstalk with salicylic acid signalling. Salicylic acid (SA) features prominently in plant perception and defence against pathogens as well as tolerance to abiotic stressors (Herrera-Vásquez *et al.*, 2015; Khan *et al.*, 2015; Prodhan *et al.*, 2018). Its control in this aspect is largely because of its influence on the reactive oxygen species (ROS) status of plants (Herrera-Vásquez *et al.*, 2015). SA controls drought tolerance (Askari and Ehsanzadeh, 2015), senescence (Ji *et al.*, 2016) and stomatal closure/conductance (Prodhan *et al.*, 2018), all of which are under SL regulation. Thus far, SA–SL interactions have only been demonstrated in plant–endophytic fungus interactions where GR24 induced the accumulation of SA while an SL signalling mutant, *max2*, had decreased SA concentrations (Rozpądek *et al.*, 2018). This is an indication that SLs may influence plant defence responses to infection by inducing SA signalling. Whether SLs elicit SA accumulation by a direct transcriptional control of SA biosynthetic genes or by inducing oxidative outburst/ROS (which are known to induce SA production) remains to be determined. Given the recent reports of SL actions in ABA-induced stomatal closure under drought conditions (Ha *et al.*, 2014; Visentin *et al.*, 2016) and senescence

(Yamada *et al.*, 2014; Ueda and Kusaba, 2015; Tian *et al.*, 2018), exploring SL–SA crosstalk in these aspects may provide invaluable tools for breeding programmes aimed at developing drought-tolerant crops with increased fruit shelf life.

PERSPECTIVES

Among many other facts, the interactions (both directly and indirectly) between chemically diverse phytohormones in the modulation of developmental processes in plants have been consistently established by experimental observations over the years. From the *sui generis* physiological processes that mark the germination stages of seed development to vegetative growth, flowering and setting of seeds, maturation of fruits and senescence, hormonal interactions form an integral circuit of the molecular signals that give shape to developmental patterns of plants in response to internal and external cues. Despite the significant progress in understanding hormonal interactions at the molecular (Fig. 6), cellular and whole plant levels, much still needs to be done with respect to SL crosstalk(s) with other phytohormones.

For instance, the actions of SLs in several processes such as its influence on cell division appear to be tissue-specific given that SLs stall cell proliferation in axillary meristems via BRC1 to inhibit branching while promoting cambial activity/cell division to enhance stem elongation and growth. While much has been done to understand SL crosstalk(s) with other key hormonal players in bud activation and branching, the precise molecular channel(s) by which SLs delineate their influences on internode elongation and secondary growth from their actions during bud activation remains to be unravelled. A possible way forward is to look into selective interactions between SL signals and TFs as well as hormones associated with both processes and into how SL signalling achieves this selectivity if found to be present. Given the central role of cytokinins as regulators of cambial activity, SL–cytokinin interactions in modulating cambial activity might also hold the answer to the aforementioned. In addition, SL actions in root secondary growth also remain uncharacterized.

The pivotal role of ABA, jasmonates and SA hormonal actions in enabling plants cope with limiting and stressful ambient conditions cannot be over emphasized. In addition, with the emerging roles of SLs in this aspect of plant development, it is imperative to characterize the crosstalk between SLs and these hormones to develop a model of the circuit of hormonal actions that drive plants' innate response and defence mechanisms against biotic and abiotic stress in their environment.

Furthermore, meaningful progress can also be achieved by studies tailored towards the identification and characterization of SL-responsive MYB class proteins and how they feature in SL signalling and responses. This group of transcriptional regulators feature prominently as downstream targets of hormonal signals and key modulators in a myriad of growth and developmental processes in plants via their active recognition and binding to specific DNA targets and the modulation of the expression of such target genes.

Finally, the impact of epigenetic cues and associated regulatory mechanisms on the interactions between SL signals and those of other phytohormones as well as their evolution during plant adaptation to new environments require much research attention. These are crucial if SL-mediated plant responses are to be fully understood in the face of dynamic ambient conditions, as is being experienced with the current change in global weather and climate patterns and the rapidly evolving microbial communities within the rhizosphere.

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