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DEVELOPMENT AT A GLANCE

Strigolactone biosynthesis and signaling in plant development

Mauricio Lopez-Obando[1], Yasmine Ligerot[1,2], Sandrine Bonhomme[1], François-Didier Boyer[1,3] and Catherine Rameau[1,*]

ABSTRACT

Strigolactones (SLs), first identified for their role in parasitic and symbiotic interactions in the rhizosphere, constitute the most recently discovered group of plant hormones. They are best known for their role in shoot branching but, more recently, roles for SLs in other aspects of plant development have emerged. In the last five years, insights into the SL biosynthetic pathway have also been revealed and several key components of the SL signaling pathway have been identified. Here, and in the accompanying poster, we summarize our current understanding of the SL pathway and discuss how this pathway regulates plant development.

[1]Institut Jean-Pierre Bourgin, INRA, AgroParisTech, CNRS, Université Paris-Saclay, RD10, 78026 Versailles Cedex, France. [2]Université Paris Sud, Orsay Cedex F-91405, France. [3]Institut de Chimie des Substances Naturelles, CNRS UPR2301, Univ. Paris-Sud, Université Paris-Saclay, 1 av. de la Terrasse, F-91198 Gif-sur-Yvette, France.

*Author for correspondence (catherine.rameau@versailles.inra.fr)

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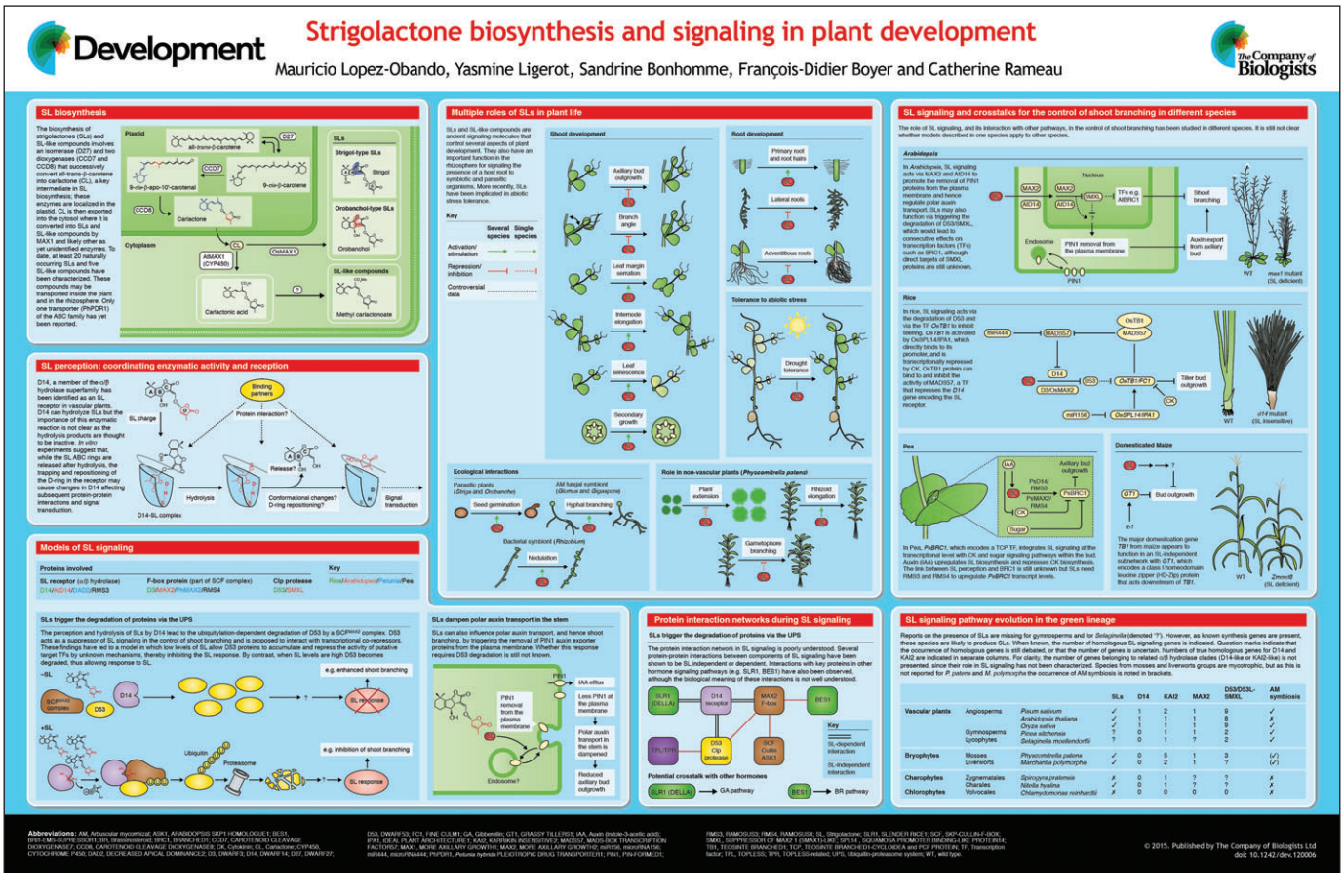
Introduction

Strigolactones (SLs) are evolutionarily ancient plant signaling molecules that play roles in diverse organisms [such as bryophytes, angiosperms and arbuscular mycorrhizal (AM) fungi] and in several aspects of plant development. The name ‘strigolactone’ comes from the first identified role of these compounds as stimulants of seed germination in species of the parasitic weed *Striga* (Cook et al., 1966) and from their lactone ring-containing chemical structure. In 2008, SLs were shown to play a role in repressing shoot branching and hence were included in the list of plant hormones that modulate plant development. These early studies showed that SL-deficient mutants are highly branched and that application of the synthetic SL GR24 inhibits axillary bud outgrowth (Gomez-Roldan et al., 2008; Umehara et al., 2008). Since then, novel functions of SLs in plant development have continuously been identified (Brewer et al., 2013; Ruyter-Spira et al., 2013). Furthermore, parts of the SL biosynthesis pathway have recently been deciphered, with the



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discovery of carlactone (CL) as a key intermediate for SL biosynthesis as well as of novel bioactive SL-like compounds (Abe et al., 2014; Al-Babili and Bouwmeester, 2015; Seto et al., 2014). Receptors for SLs, and some of their downstream effectors, have also been identified (Hamiaux et al., 2012; Xiong et al., 2014).

Here, we summarize our current understanding of the SL biosynthesis and signaling pathways, we describe the different functions of SLs during plant development, and we discuss the evolutionary origin of SL signaling. Models of SL signaling and crosstalk with other plant signaling pathways in the context of shoot branching are also presented.

Chemical structure and biosynthesis of natural SLs

The structural core of SLs is a tricyclic lactone (containing rings that are referred to as ABC), with different carbon A-ring sizes and substitution patterns on AB-rings. This core is connected via an enol ether bridge to an invariable α,β-unsaturated furanone moiety (termed the D-ring). To date, at least 20 naturally occurring SLs have been identified and characterized in root exudates of various land plants (Al-Babili and Bouwmeester, 2015; Tokunaga et al., 2015). They can be separated into two types – strigol and orobanchol – according to the stereochemistry of the B–C-ring junction, with both having a conserved *R*-configuration at the C-2′ position. The bioactiphore responsible for the various SL bioactivities resides within the region that connects the D-ring to the core, which can differ according to SL type (Zwanenburg and Pospíšil, 2013).

Recent studies have provided some insights into the SL biosynthesis pathway. SLs are synthesized from the key precursor CL, which is derived from all-trans β-carotene via the action of an isomerase (D27) and two carotenoid cleavage dioxygenases (CCD7 and CCD8). These steps of the SL biosynthesis pathway take place in the plastid and the resulting CL is then exported into the cytoplasm. The subsequent steps involve CL oxidation, further ring closures and functionalizations involving members of the CYP711 family (MAX1) (Zhang et al., 2014), eventually giving rise to SLs and SL-like compounds. In *Arabidopsis*, the unique enzyme MAX1 is able to transform CL to carlactonoic acid, which is further transformed into the SL-like compound methyl carlactonoate (MeCLA) by an unknown enzyme (Abe et al., 2014). Other SL-like compounds, with a CL-type structure lacking the canonical ABC-rings (Kim et al., 2014; Ueno et al., 2014), have been discovered in different plants, highlighting the structural diversity of this class of compounds. Once synthesized, all of these compounds may be transported within the plant and in the rhizosphere. PhPDR1, a member of the ABC family, has been identified as a potential SL transporter (Kretzschmar et al., 2012; Sasse et al., 2015).

SL biosynthesis, which occurs mainly in roots but also in the stem, is tightly regulated (Al-Babili and Bouwmeester, 2015). Phosphate starvation, for example, strongly induces SL biosynthesis (López-Ráez et al., 2008). In addition, high levels of SL biosynthesis gene transcripts (in particular *CCD7* and *CCD8*) in SL-deficient and SL-insensitive mutants in several species indicate that there is feedback regulation of SL biosynthesis (Hayward et al., 2009; Proust et al., 2011).

SL perception: coordinating enzymatic activity and reception

Using genetic approaches, genes encoding SL receptors have been identified in several vascular land plants, including petunia (where the receptor is named DAD2), rice (D14) and *Arabidopsis* (AtD14) (Arite et al., 2009; Hamiaux et al., 2012; Waters et al., 2012). These proteins belong to a clade of the α/β-hydrolase enzyme superfamily that includes DWARF14 (D14) and GID1, which is a gibberellin receptor that has lost its enzymatic activity. It was shown, using *in vitro* enzymatic studies, that D14 proteins can hydrolyze the synthetic SL GR24 into inactive ABC- and D-ring parts (Hamiaux et al., 2012; Seto and Yamaguchi, 2014; Xiong et al., 2014). Importantly, it was shown that the SL-like molecule MeCLA can also interact *in vitro* with the AtD14 SL receptor and be hydrolyzed, despite not having the canonical four-ring structure (Abe et al., 2014). The enzymatic activity of D14 proteins is mediated by a conserved catalytic triad, Ser-His-Asp (S-H-D), that has hydrolase activity. Additionally, the catalytic triad seems to be important for the biological response of D14 proteins since mutated proteins, at least those harboring mutations at the Ser residue, cannot complement the *d14* mutant branching phenotype, as shown in petunia (Hamiaux et al., 2012).

The mechanism of SL reception by D14 is still not well understood; in particular, it is unclear whether SL hydrolysis by the receptor is of importance as the hydrolysis products have been shown to be biologically inactive. However, it has been proposed that, following hydrolysis, the D-OH part of SLs forms a complex with D14 thereby allowing the recruitment of binding partners (de Saint Germain et al., 2013; Nakamura et al., 2013).

It should be noted that the D14 SL receptor clade is closely related to the KARRIKIN INSENSITIVE 2/HYPOSENSITIVE TO LIGHT (KAI2/HTL) clade. In *Arabidopsis*, KAI2 is able to perceive butenolide-containing rings, including the smoke-derived karrikin (KAR) compounds, which, similar to SLs, have a lactone D-ring (Guo et al., 2013b; Smith and Li, 2014; Waters et al., 2012). Interestingly, the SL and KAR pathways modulate plant development in distinct ways but both require the F-box protein MAX2 (D3) to mediate their responses (Nelson et al., 2011).

SL signaling: the role of UPS-dependent protein degradation

Most plant hormone signaling pathways involve the targeting of proteins for degradation through the ubiquitin-proteasome system (UPS) (Kelley and Estelle, 2012). There are strong arguments to suggest that the UPS is also involved in SL signaling (Bennett and Leyser, 2014). In particular, the F-box protein MAX2, which is part of a SKP1–CULLIN–F-BOX (SCF) ubiquitin ligase protein complex, appears to play a key role in mediating SL-triggered protein degradation (Stirnberg et al., 2007; Zhao et al., 2014). Recently, the protein D53 was identified in rice and shown to be targeted for degradation after SL treatment; this degradation was not observed when the proteasome inhibitor MG132 was used (Jiang et al., 2013; Zhou et al., 2013). The rice *d53* semi-dominant mutant, which expresses a mutated protein that is resistant to degradation by SL treatment, is SL insensitive and shows high tillering/branching (Jiang et al., 2013; Zhou et al., 2013). Furthermore, it was shown that a reduction in *D53* expression in *d3* and *d14* mutant backgrounds can restore a non-branched wild-type phenotype (Jiang et al., 2013; Zhou et al., 2013). Together, these data indicate that D53 acts as a suppressor of SL signaling in the control of shoot branching (Jiang et al., 2013; Zhou et al., 2013).

This idea led to a model in which MAX2 interacts with D14 in an SL-dependent manner, and this leads to the ubiquitylation-dependent degradation of D53 by the SCF^{MAX2} complex. However, nothing is currently known about the subsequent effects of D53 degradation and how this protein acts to suppress SL signaling. D53 belongs to a small family of proteins [SMAX1-like (SMXL)] that show some homology with class I CIp ATPase enzymes (Stanga et al., 2013). The presence of potential ethylene-responsive element binding factor-associated amphiphilic repression (EAR) motifs in D53 and its ability to interact with

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topless-related (TPR) proteins, which are known transcriptional co-repressors, suggest that a D53-TPR complex could repress the transcription of downstream targets (Bennett and Leyser, 2014; Jiang et al., 2013; Smith and Li, 2014), but this has not yet been demonstrated. Other proteins that are subject to SL-triggered degradation, or other transcription factors that lie directly downstream of D53/SMXL proteins, are still unknown (Smith and Li, 2014). BES1, a positive regulator in the brassinosteroid signaling pathway, can also be targeted for degradation via SCF^{MAX2}, although SL is not needed for a BES1-MAX2 interaction (Wang et al., 2013). An SL-dependent interaction between SLR1, a rice gibberellin signaling repressor (DELLA protein), and D14 has also been shown but the biological significance of this interaction is not yet understood (Nakamura et al., 2013).

Models for SL signaling in the control of shoot branching

SLs are best known for their role in repressing shoot branching, and two mechanisms have been proposed to explain this role. In rice and pea, SLs were shown to act via their effects on the TCP transcription factor OsTB1/PsBRC1 to repress axillary bud outgrowth (Braun et al., 2012; Minakuchi et al., 2010). This transcription factor acts as a key integrator of several other pathways, such as the cytokinin pathway and the recently proposed sucrose signaling pathway in pea (Mason et al., 2014; Rameau et al., 2015). Interestingly, the maize ortholog (*TB1*) of the gene encoding this transcription factor seems to act independently of SLs to repress shoot branching (Guan et al., 2012). In rice, other transcription factors, such as MADS57 and IPA1/OsSPL14, that are involved in shoot branching have also been connected to key components of the SL signaling pathway, but whether these various transcription factors mediate SL signaling, and if or how they lie downstream of the D14-D3-D53 axis, are still not clear (Guo et al., 2013a; Lu et al., 2013).

Because SL-deficient mutants are more branched than *brc1* mutants (Braun et al., 2012; Minakuchi et al., 2010), there is very likely a BRC1-independent effect of SLs on shoot branching. Indeed, in *Arabidopsis* a non-transcriptional mechanism relies on SLs triggering the rapid removal of the auxin efflux carrier PIN-FORMED 1 (PIN1) from the plasma membrane of stem xylem parenchyma cells (Shinohara et al., 2013). This effect of SLs would increase competition between buds to export auxin into the main auxin stream, based on an auxin transport canalization-dependent mechanism (Shinohara et al., 2013; Waldie et al., 2014).

Whether both of these mechanisms regulate shoot branching or act at different stages of bud outgrowth is still debated. Moreover, whether these downstream targets of SL signaling are dependent on the UPS-mediated degradation of D53 remains to be clarified.

Key developmental roles for SL signaling

SLs control numerous other aspects of plant development. Pea, *Arabidopsis*, rice and petunia mutants with defects in SL biosynthesis or SL responses were first identified based on their increased shoot branching phenotypes and their dwarfism (Beveridge et al., 1996; Ishikawa et al., 2005; Napoli, 1996; Stirnberg et al., 2002). Less obvious phenotypes, such as reduced secondary growth, delay in leaf senescence or modified root architecture, were later identified (Brewer et al., 2013; Ueda and Kusaba, 2015; Yamada et al., 2014). SLs can also modulate tolerance to abiotic stresses (drought) (Ha et al., 2014). Direct or indirect roles for SLs in biotic stress-related responses have been suggested to act via crosstalk with other hormones (Al-Babili and Bouwmeester, 2015; Brewer et al., 2013; Stes et al., 2015). Thus, like other plant hormones, SLs can modulate multiple aspects of plant growth and development, either independently or via interactions with other hormonal and environmental pathways. The observed diversity of D53-like/SMXL proteins may contribute to the multiple processes controlled by SLs in plant development.

The origin and evolution of SL signaling

Studies have shown that species of the fresh water algae *Nitella* (Charales) are able to synthesize SLs (Delaux et al., 2012), suggesting that the SL pathway originated prior to the diversification of land plants (embryophytes). Since Charales do not establish symbiosis with AM fungi, it has been proposed that SLs first played a hormonal role during rhizoid elongation and were later recruited for symbiotic interactions (Delaux et al., 2012). In the moss *Physcomitrella patens*, SLs regulate protonema filament extension (Proust et al., 2011) as well as leafy shoot branching (Coudert et al., 2015). Furthermore, although SLs are detected in basal plants (Delaux et al., 2012), the KAI2/HTL clade appeared before the D14 clade, suggesting that D14 proteins might have been later selected as SL receptors during land plant evolution for novel developmental processes. Intriguingly, a high number of *KAI2*/*HTL* genes are present in the *P. patens* genome compared with angiosperms but also with *Selaginella* and *Marchantia* (Delaux et al., 2012). A similar *KAI2*/*HTL* gene expansion is found in parasitic plants (Conn et al., 2015; Tsuchiya et al., 2015). Interestingly, in these species, it was suggested that some of these KAI2/HTL homologs could be SL receptors (Conn et al., 2015). It should be noted that, despite SLs being detected in basal plants, the SL signaling pathway is poorly described in these plants. Recently, it was shown that KAI2/HTL homologs of *Selaginella* and *Marchantia* do not complement *Arabidopsis d14* mutant phenotypes, nor some phenotypes of *Arabidopsis kai2* (Waters et al., 2015). This leaves open the question of SL receptor identity in basal plants.

Perspectives

Despite significant progress, many key questions regarding SL biosynthesis, perception and signaling remain to be answered. The enzymatic activity of the SL receptor has been conserved during evolution, indicating that it plays an essential function, but this function is puzzling as the hydrolysis products (the ABC and D-OH parts) are known to be inactive. Is this enzymatic function of the SL receptor essential for SL reception and signaling? Does it play an important role in SL homeostasis? Furthermore, if SL perception truly involves an SL degradation process, are there other mechanisms of SL inactivation? The link between SL perception and downstream responses is also unclear. Although some downstream transcription factors have been identified, it is often noted that very few genes are transcriptionally regulated after SL application, at least over a short time frame, compared with other plant hormones (Smith and Li, 2014; Waldie et al., 2014), suggesting that non-transcriptional mechanisms might also be important in mediating the response to SL. Further investigation into both transcriptional and non-transcriptional responses and their importance will be key.

Understanding the molecular events acting downstream of D53/SMXL proteins will also be essential. In particular, it is unknown whether other post-translational modifications, such as phosphorylation and/or glycosylation, are required for the regulation of these downstream targets (Chen et al., 2014). The protein-protein interaction network in SL signaling also appears to be quite complex, and further understanding of these interactions might help to explain the observed crosstalk between the SL pathway and other plant signaling pathways. There is no doubt that the coming years will bring answers to the key questions in this exciting field.

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Competing interests
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