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Spotlight

Strigolactones and Gibberellins: A New Couple in the Phytohormone World?

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Strigolactones (SLs) and gibberellins (GAs) are plant hormones that share some unique aspects of their perception and signalling pathways. Recent discoveries indicate that these two phytohormones may act together in processes of plant development and that SL biosynthesis is regulated by GAs.

SLs are a carotenoid-derived group of plant growth and development regulators that have been identified as phytohormones. They are involved in the coordination of plant growth via the regulation of shoot branching and the development of the root system [1] and also function as signalling molecules in the communication between plants and their symbiotic and pathogenic partners [2]. In recent years, specific steps of SL action have been elucidated; however, knowledge concerning the interaction between SLs and phytohormones other than abscisic acid, auxin, or cytokinin remains meagre [1]. This spotlight article summarises the discoveries that have revealed possible interactions between SLs and GAs in the control of shoot branching and have additionally indicated that the two phytohormones may regulate each other.

GAs are phytohormones that regulate various aspects of plant development, including shoot and root growth, leaf morphogenesis, germination dormancy, seed production, and flowering [3]. The biosynthesis of GAs and their signalling pathway are well known and since recent findings indicate that SL perception and signalling is similar to that described for GAs, it is postulated that knowledge about GAs may also be applicable in SL biology [4]. In the literature there are also suggestions that these two phytohormones are involved in the same processes during plant development. Analysis of the rice (*Oryza sativa*) semidwarf mutants *GIBBERELLIN OXIDASE5*, *6*, and *9* (*OsGAox5*, *OsGAox6*, and *OsGAox9*), which are disturbed in GA biosynthesis, revealed a more branched shoot phenotype similar to SL mutants. Additionally, treatment with 5 μM of biologically active GA (GA_3) represses tillering in *OsGAox6* and the wild-type. It was shown that GA regulates the number of tillers via the activity of *TEOSINTE BRANCHED1* (*OsTB1*) and *ORYZA SATIVA HOMEOBOX1* (*OSH1*), which are involved in tiller bud outgrowth. The expression of these genes was elevated in GA-deficiency mutants and repressed by GA_3 treatment [5]. Also, in the highly branched rice SL-signalling mutant (*OsD14*, *DWARF14*), the expression of *OSH1* was upregulated [6], which may indicate that the two phytohormones share the mechanism for tillering control in rice. Unfortunately, there are no data in the literature concerning the expression of *OSH1* in other SL mutants, and additional investigations will be necessary to confirm that hypothesis.

Subsequently, it has been shown that SLs induce the interaction between the SL receptor DWARF14 (D14) and SLENDER1 (SLR1), a representative of DELLA proteins that negatively regulates GA signalling [7]. This indicates crosstalk between SLs and GAs because SLR1 might be degraded in a SL-dependent manner, similar to the way in which it occurs in the GA signalling pathway where binding of the GA receptor GIBBERELLIC-ACID INSENSITIVE1 (GID1) to the GA molecule stimulates the interaction of the GID1 and DELLA proteins. The subsequent interaction of GID1–DELLA with the Skp1–Cullin–F-box protein (SCF) complex) results in polyubiquitination of DELLA and its degradation through the 26S proteasome (Figure 1A). However, mutants of *Arabidopsis thaliana* lacking all DELLA protein activity or expressing stabilised versions of DELLA proteins share only some of the phenotypic features described for SL mutants and a higher number of branches was not observed in mutants [8]. It was also shown that the second member of the DELLA proteins −REPRESSOR OF GA1-3 (RGA) – is not degraded by D14 in a SL-dependent manner [8]. It remains possible, however, that SLs may regulate only some aspects of the plant phenotype via degradation of specific DELLA proteins. Direct evidence for this mechanism is still lacking and it cannot be excluded that some DELLA proteins are not degraded but still recognised by a D14-containing complex.

Recent studies on rice and *Lotus japonicus* have shown that the biosynthesis of SLs is negatively regulated by treatment with bioactive forms of GAs (GA_1, GA_3, and GA_4) [9]. Tanginbozu, a rice GA-biosynthesis mutant, displayed elevated levels of SLs, corresponding with its semidwarf phenotype and increased number of tillers. Although elevated levels of SLs can be suppressed by treatment with bioactive GAs, this is not the case in the GA-insensitive mutants *gid1-3* and *gid2-2*. Interestingly, in another GA-insensitive mutant, *slr1-5*, endogenous SLs were undetecTable Since SLR1 is a repressor of the GA signal, which is degraded in a GA-dependent manner, it is postulated that production of SLs might be regulated via the activity of DELLA proteins. There is evidence that GAs regulate SL biosynthesis independently from SL signalling, because in the rice SL-insensitive mutants *d3-1* and *d14-1* treatment with GA_3 reduces the level of endogenous SLs [9]. An additional indication for a GA influence on SL biosynthesis came from the *in silico* analysis of the promoter region of *A. thaliana* and rice genes involved in this process. Promoter regions of four rice genes encoding

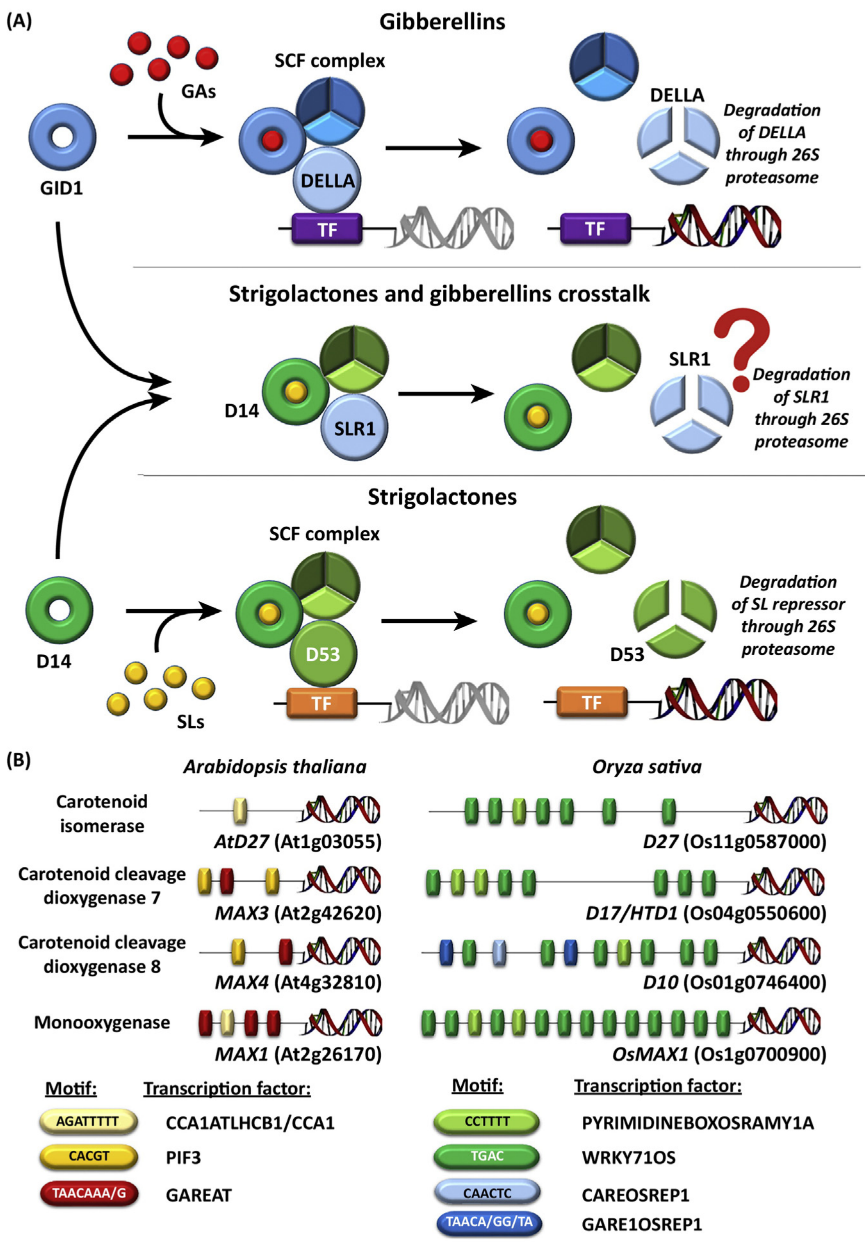

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Figure 1. Gibberellin (GA) and Strigolactone (SL) Signalling Pathways in Plants and Motifs Recognised by GA-Related Transcription Factors (TFs) in the Promoter Region of SL-Biosynthesis Genes. (A) The signalling pathways of GAs and SLs share some similarities, such as receptors [GIBBERELLIC-ACID INSENSITIVE1 (GID1) for GAs and DWARF14 (D14) for SLs] that belong to the α/β hydrolases and degradation of repressors [DELLA/SLENDER1 (SLR1) for GAs and DWARF53 (D53) for SLs] via the 26S proteasome. Because in rice D14 is able to bind SRL1 [7], one of the DELLA proteins, in a SL-dependent manner, crosstalk between SLs and GAs could be postulated, but there is no evidence that SLR1 is degraded in a SL-dependent manner. (B) Distribution of motifs recognised by GA-dependent transcription factors in 1000-bp promoter regions of *Arabidopsis thaliana* and rice SL-biosynthesis genes (according to [10]). MAX, more axillary growth; SCF complex, Skp1–Cullin–F-box protein complex.

enzymes from the SL biosynthesis pathway contained multiple motifs recognised by transcription factors (TFs) from the WRKY71OS family [10] (Figure 1C). This family of TFs are transcriptional repressors of the GA signalling pathway [11].

Data available in expression databases confirm that treatment with 10 μM GA_3 decreases the expression of rice SL-biosynthesis genes after 15, 30, and 60 min whereas a lower concentration of GA_3 (50 nM) decreased the expression of SL-biosynthesis genes for up to 24 h [10].

Interestingly, the rice GA-biosynthesis mutant was insensitive to treatment with a synthetic analogue of SLs (GR24) whereas the wild type responds to that treatment with inhibition of the second tiller bud outgrowth in 2-week-old seedlings [9]. This indicates that shoot branching is probably regulated by SLs in cooperation with GAs. This hypothesis needs to be confirmed by analysis of other SL and GA mutants in combination with a detailed investigation of the hormone status of growing/inhibited axillary buds. Currently, it is also known that, in some aspects of plant development, SLs may act independently from Gas; for example, during promotion of internode elongation in *Pisum sativum* [12].

While studies in rice seem to indicate an interaction between SLs and GAs, results obtained for *A. thaliana* are more ambiguous. *A. thaliana* promoter regions of SL-biosynthesis genes contain fewer motifs recognised by GA-dependent TFs. Among these TFs, only transcriptional activators were characterised, such as CCA1ATLHCB1/CCA1, GAREAT, or PIF3 (Figure 1C). Microarray data have shown that treatment with GA_3 resulted in varied expression of *A. thaliana* SL-biosynthesis genes [10], but it has to be considered that plant responses to hormone treatment might be dose dependent in many cases. Unfortunately, neither the effect of GA treatment on SL levels in *A. thaliana* nor hormone content in SL or GA mutants has been investigated. Final confirmation of the crosstalk between SLs and GAs awaits further analysis of the hormone status in different genetic backgrounds and the interactions of the SL receptor with single DELLA proteins. It also has to be considered that interactions between SLs and GAs might be modulated during plant growth or by environmental conditions or might be restricted to a specific aspect of plant development. So far there is an indication that at some stages of plant development SLs and GAs may act together and that in rice the biosynthesis of SLs is controlled by GAs. Considering the highly conserved mechanisms of perception of and signalling by SLs and GAs in plants, it is tempting to speculate that these two phytohormones may act together in both monocots and dicots.

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Spotlight

Type-B *ARABIDOPSIS* RESPONSE REGULATORs Directly Activate *WUSCHEL*

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The *WUSCHEL* (*WUS*) gene is necessary for the maintenance of stem cells in the shoot apical meristem. Four recent reports show that cytokinin responsive type-B *ARABIDOPSIS* RESPONSE REGULATORs (ARRs) directly activate *WUS* expression, providing a long-awaited explanation for how cytokinin influences the maintenance of the stem cell niche.

WUS Is a Key Regulator of Shoot meristem Development

The maintenance of the stem cell niche in the shoot apical meristem (SAM) depends on the action of the homeobox-containing gene *WUSCHEL* (*WUS*). Loss of *WUS* activity in the SAM as well as in axillary meristems eliminates shoot development, while overexpression of *WUS* promotes ectopic shoot growth [1]. Similarly, *WUS* is required to promote shoot regeneration from tissue culture [2,3]. During regeneration, *WUS* is *de novo* activated and *WUS*-expressing cells mark the shoot progenitor region [3]. *WUS* is expressed specifically in a small group of cells just beneath the SAM, and movement of the WUS protein to overlying SAM cells regulates a number of genes that in turn function to maintain the domain of *WUS* expression [4]. These include several type-A *ARABIDOPSIS* RESPONSE REGULATORs (ARRs) that negatively regulate cytokinin responses and meristem function [5]. This cytokinin–WUS feedback loop is critical for normal shoot meristem development. However, there has been a major gap in understanding how cytokinin signaling activates *WUS* expression.

Type-B ARRs Mediate Cytokinin Signaling to *WUS*

Arabidopsis (*Arabidopsis thaliana*) also possesses type-B ARRs that mediate primary cytokinin responses and promote cytokinin-induced gene expression [6]. Mutations in several type-B *ARR*s *(ARR1*, *ARR2*, *ARR10,* and *ARR12)* result in defects in shoot regeneration and axillary meristem development, implicating them in the regulation of meristem maintenance [2,3,7]. Four different groups have now shown that type-B ARRs bind directly to the *WUS* promoter and activate *WUS* expression [1–3,8].

Genetic analysis suggests that the *WUS* expression requires the function of type-B ARRs during shoot regeneration [2,3]. Indeed, expression of type-B *ARR*s can be observed 3 days prior to that of *WUS* in shoot regeneration studies, consistent with a model in which *WUS* acts downstream of type-B *ARR*s [3]. Supporting this idea, overexpression of *WUS* can restore the shoot regeneration capacity of an *arr1 arr12* double mutant [2]. Various approaches, including chromatin immunoprecipitation (ChIP), were used to show that type-B ARRs directly bind to the *WUS* promoter [2,3]. An unbiased approach using ChIP-seq technology also identified *WUS* as a direct target for ARR10 [8].

The activation of *WUS* by type-B ARRs requires the function of HD-ZIP III genes;