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 (GA3) represses tillering in OsGAox6 and the wild-type. It was shown that GA regulates the number of tillers via the activity of TESOSINTE BRANCHED1 (OsTB1) and Oryza Sativa HOMEODOMAIN-BOX1 (OSH1), which are involved in tiller bud outgrowth. The expression of these genes was elevated in GA-deficiency mutants and repressed by GA3 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 SLEN- DER1 (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 (GA1, GA3, and GA4) [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, sfr1-5, 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 GA3 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
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 GA₃ decreases the expression of rice SL biosynthesis genes after 15, 30, and 60 min whereas a lower concentration of GA₃ (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 CCA1ATLHC81/CCA1, GAREAT, or PIF3 (Figure 1C). Microarray data have shown that treatment with GA₃ 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 signaling 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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References


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 ARRs (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 ARRs 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 ARRs [3]. Supporting this idea, overexpression of WUS can restore the shoot regeneration capacity of an arr1 arr2 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;