Cell Research (2017) 27:1191-1192.

www.nature.com/cr

RESEARCH HIGHLIGHT

IPA1: a direct target of SL signaling

Cell Research (2017) **27**:1191-1192. doi:10.1038/cr.2017.114; published online 8 September 2017

The plant hormone strigolactone (SL) is important for many processes in plants, but its molecular mode of action has been difficult to elucidate. A new discovery has identified the SPL transcription factor, IPA1, as a crucial component directly involved in SL signaling.

Strigolactones (SLs) are a group of carotenoid-derived terpenoid lactones first studied for their involvement in the germination of parasitic weeds and the promotion of hyphal branching in arbuscular mycorrhizal (AM) fungi [1]. Since being classified as a plant hormone for their role in the inhibition of shoot branching, SLs have been found to be involved in many other processes including, but not limited to, the regulation of root growth, secondary vascular growth, and leaf senescence. In recent years, many advances have been made in elucidating the SL biosynthetic and response pathways, however, signaling pathways acting downstream of SL perception remain unresolved [1].

The perception of SL involves an α/β hydrolase and an SCF complex which, in the presence of SL, target the DWARF53 (D53) protein for polyubiquitination and degradation by the 26S proteasome [1]. In the absence of SL, D53 is predicted to inhibit transcriptional activation of genes in partnership with TOPLESS-related proteins [1]. Few potential targets of D53 transcriptional repression have been reported, questioning this classical hormone signaling view of the function of D53 and raising the possibility that SL signaling might act predominantly via direct protein regulation. For example, localization of the PIN1 protein, a transporter of the plant hormone auxin, is rapidly modified by SL and this does not require protein synthesis [1].

In the search for transcriptional repression targets of D53, Song *et al*. [2] have now identified that the transcriptional activation activity of the transcription factor IDEAL PLANT ARCHITECTURE1 (IPA1) is affected by D53. IPA1, otherwise known as OsSPL14, is a member of the SQUAMOSA PROMOTER BINDING PROTEIN-LIKE (SPL) family of plant-specific transcription factors [3]. IPA1 has previously been implicated in the negative regulation of shoot branching, also called tillering in monocots; rice mutants overexpressing *IPA1* exhibit a reduced shoot branching phenotype while *ipa1* loss-of-function mutants have increased shoot branching [3]. These phenotypes are consistent with IPA1 being repressed by D53, which is further supported by the observation that enhanced shoot branching in the *ipa1* lines is not suppressed by SL treatment [2].

These observations led Song *et al*. [2] to test the potential involvement of IPA1 downstream of SL. They found that the SL degradation target D53 could physically interact with IPA1 both *in vitro* and *in vivo*. This interaction with D53 prevented the transcriptional activation activity of IPA1 in a dose-dependent manner (Figure 1). In rice, D53 is one of the only known transcriptional targets of SL [4, 5], and D53 prevented IPA1 from upregulating *D53* expression [2] (Figure 1). By showing evidence of a mechanism via which SL signaling transcriptionally regulates downstream target genes, this work has enabled description of a continuous SL signaling pathway from perception to gene regulation (Figure 1).

One important next step is to find other transcriptional targets of IPA1 downstream of SL; previous ChIP-seq analyses have discovered thousands of potential IPA1-binding sites [6] and yet only a few transcriptional targets of SL have been identified to date. FINE CULM1 (FC1), TEOSINTE BRANCHED1 (TB1) and BRANCHED 1 (BRC1) are well established in several species as homologous transcription factors that suppress shoot branching [7]. *TB1* has been shown to be a direct transcriptional target of IPA1 in rice [6] (Figure 1). Consistent with this, tb1 enhances branching in the *ipa1-1D* gain of function mutant [6]. A role for BRC1 downstream of SL has been implied in pea and *Arabidopsis* where *BRC1* expression is upregulated by SL, and SL treatment cannot inhibit shoot branching in the *brc1* mutant [7]. However, transcriptional activation of *TB1* by SL has not been observed in rice and indeed, *TB1* expression is not always anti-correlated with shoot branching in rice SL mutants [1]. Further work will need to confirm whether SL can regulate *TB1* gene expression in bud-specific tissues of rice, and whether this regulation requires D53 and IPA1.

Further support for a shoot branching control module including *IPA1*-related *SPL* genes and *TB1* comes from a recent study in wheat [8]. TaD53 physically interacts with two SPL proteins TaSPL17, the wheat homologue of IPA1, and TaSPL3. TaSPL3 and TaSPL17 transcriptionally activate *TaTB1* expression, and further investigations using only

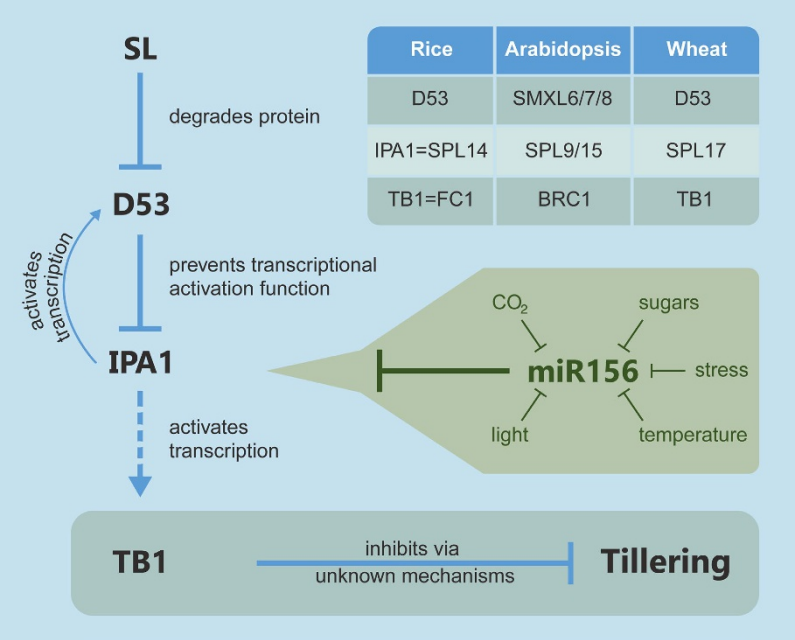

Figure 1 Simplified rice-centric model of SL signaling in the regulation of shoot branching/tillering. SL degrades the D53 protein in rice [4, 5] and the related SMXL6, SMXL7 and SMXL8 proteins in *Arabidopsis* [1]. D53 directly prevents the transcriptional activation function of IPA1 (OsSPL14) in rice [2] and the related SPL3 and SPL17 proteins in wheat [8]; D53 prevents IPA1 upregulation of *D53* gene expression in rice [2], and TaSPL3 upregulation of *TaTB1* gene expression in wheat [8]. IPA1 regulation of *TB1* gene expression in rice has also been confirmed [6] even though SL regulation of *TB1* has not been reported in rice [1]. TB1 can then inhibit shoot branching/tillering via unknown mechanisms [7]. miR156 integrates many environmental and endogenous signals [11] and post-transcriptionally regulates *IPA1* gene expression [3].

TaSPL3 found that TaD53 could prevent TaSPL3 upregulation of *TaTB1* gene expression. Genetic and SL response studies are required to confirm this pathway in wheat.

Many *SPL* genes, including *IPA1*, are post-transcriptionally suppressed by miR156 in a highly conserved regulatory pathway [3]. Consistent with this, overexpressing *miR156* results in reduced expression of *IPA1* and increased shoot branching, while reducing *miR156* expression has opposing effects [9]. It is likely that not all the shoot branching effects of OsmiR156 are mediated by IPA1 because *OsmiR156* overexpression lines respond to SL [10], whereas *ipa1* lines do not. The IPA1-independent effects of miR156 on shoot branching may be mediated by other SPLs such as OsSPL7 and OsSPL17 whose mutants also display altered shoot branching phenotypes [9].

In addition to regulating shoot branching, miR156/SPLs have roles in many diverse functions such as phase change, leaf development, flower structure, fruit maturation, nodulation, immunity, and response to environmental stimuli [3]. This raises the possibility that SPLs may be involved in SL regulation of developmental processes other than shoot branching. Furthermore, the miR156/SPL regulatory hub is also a key target of many external and internal signals including CO_2, sugar, temperature, light and different stresses [11] (Figure 1). Many of these signals also regulate *TB1* and its homologues [7], raising the possibility that miR156/SPL is a key integrator of external signals that influence SL signaling.

The original identification of IPA1 was prompted by the search for an ideal plant architecture in rice; the *ipa1-1D* gain-of-function mutant has improved grain yield with reduced shoot branching, increased plant height and larger panicles [11]. Numerous papers have since reported the importance of being able to manipulate expression of *SPL* genes and their transcriptional targets to further improve ideal plant architecture in rice varieties [11]. Integrating a classical hormone pathway with the mobile miR156, IPA1 enables fine-tuning of plant phenotype in accordance with the environment (Figure 1) and hence provides a module of much interest for the improvement of yield in crops.

Stephanie C Kerr[1],
Christine A Beveridge[1]

[1]*School of Biological Sciences, The University of Queensland, Brisbane, Australia*
Correspondence: Stephanie C Kerr[a],
Christine A Beveridge[b]
[a]E-mail: stephanie.kerr1@uqconnect.edu.au
[b]E-mail: c.beveridge@uq.edu.au

References

1 Waters MT, Gutjahr C, Bennett T, *et al*. *Annu Rev Plant Biol* 2017; **68**:291-322.
2 Song X, Lu Z, Yu H, *et al*. *Cell Res* 2017; **27**:1127-1141.
3 Wang L, Zhang QF. *Trends Plant Sci* 2017; **22**:643-646.
4 Jiang L, Liu X, Xiong G, *et al*. *Nature* 2013; **504**:401-405.
5 Zhou F, Lin Q, Zhu L, *et al*. *Nature* 2013; **504**:406-410.
6 Lu Z, Yu H, Xiong G, *et al*. *Plant Cell* 2013; **25**:3743-3759.
7 Rameau C, Bertheloot J, Leduc N, *et al*. *Front Plant Sci* 2015; **5**:741.
8 Liu J, Cheng XL, Liu P, *et al*. *Plant Physiol* 2017; **174**:1931-1948.
9 Wang L, Sun SY, Jin JY, *et al*. *Proc Natl Acad Sci USA* 2015; **112**:15504-15509.
10 Luo L, Li W, Miura K, *et al*. *Plant Cell Physiol* 2012; **53**:1793-1801.
11 Wang H, Wang H. *Mol Plant* 2015; **8**:677-688.