Gibberellins (GAs) and abscisic acid (ABA) affect plant development in an opposite manner. GA is generally considered a growth-promoting hormone, whereas ABA signaling, triggered in response to stress, counteracts the GA effects and restricts growth under suboptimal conditions (Vanstraelen and Benkova, 2012). To integrate the intrinsic and external cues, ABA and GA need to antagonize each other’s actions. For example, in Arabidopsis (Arabidopsis thaliana), ABA inhibits GA functions by reducing GA biosynthesis, which results in stabilization of DELLA proteins, the GA repressors (Zentella et al., 2007). In turn, it was shown in rice (Oryza sativa) that GA antagonizes ABA signaling by enhancing the degradation of ABA receptors (Lin et al., 2015).

The GA-mediated inhibition of ABA signaling in rice requires an activating component of the APC/C TE E3 ubiquitin ligase complex, TILLER ENHANCER (TE; Lin et al., 2015). TE, by the physical interaction with ABA receptors OsPYL/RCARs, recruits them to the APC/C TE complex, which subsequently targets them for proteasomal degradation. ABA activates SnRK2 protein kinases to phosphorylate TE, which releases OsPYL/RCARs from the interaction with TE and thus prevents their degradation. To antagonize ABA, increased GA levels reduce SnRK2 protein abundance and as a result promote OsPYL/RCAR protein decay (Lin et al., 2015).

TE was initially identified as a negative regulator of tiller development (Lin et al., 2012). Recent work of Lin et al. (2020) demonstrates that APC/C TE is also a hub for ABA-GA antagonistic control of both root growth and shoot branching in rice. The authors showed that overexpression of TE resulted in a dramatic decrease in root length, indicating that APC/C TE regulates root growth. Lin et al. (2020) further investigated the involvement of ABA and GA in APC/C TE-mediated root growth.

Low levels of GA stimulated root meristem (RM) cell proliferation and as consequence enhanced root growth. By contrast, higher GA levels restricted RM cell division, resulting in root growth inhibition. Only low ABA doses enhanced root growth, but this enhancement was suppressed by elevated TE expression. In the shoot, GA repressed whereas ABA induced tillering, yet the latter was restrained by TE overexpression. Together, this shows that ABA and GA antagonism in root growth and shoot branching regulation is mediated by the APC/C TE complex (Lin et al., 2020).

APC/C TE has previously been shown to mediate the decay of MOC1, a GRAS family transcription factor (TF) that induces tillering (Lin et al., 2012). Here, the authors screened sequences of known root growth regulators for a TE recognition motif and identified another GRAS TF, SHORTROOT1 (OsSHR1), as an APC/C TE substrate. Silencing OsSHR1 reduced RM cell division and as consequence suppressed root growth, similarly to GA treatment (Lin et al., 2020).

APC/C TE-mediated degradation of OsSHR1 and MOC1 was induced by GA, but ABA was able to suppress this effect. GA enhanced the physical interaction of TE with OsSHR1 and MOC1 and thus their recruitment to the APC/C TE complex. Subsequent proteolysis of OsSHR1 and MOC1 diminished the activity of root and auxillary meristems. In the presence of ABA, SnRK2 protein kinases phosphorylate TE, which decreases TE affinity to its substrates (Lin et al., 2015). By this mechanism, ABA prevents OsSHR1 and MOC1 from entering the APC/C TE complex, stabilizing their protein levels and thus meristematic activity.

Lin et al. (2020) present a model in which antagonistic action of ABA and GA integrates growth and development with external conditions via a SnRK2-APC/C TE signaling module (see figure). Interestingly, this SnRK2-APC/C TE hub mediates opposing effects of ABA and GA in both directions. GA can inhibit ABA actions via ABA receptor degradation (Lin et al., 2015), while ABA can antagonize GA signaling by stabilizing the levels of growth-related TFs (Lin et al., 2020). Both pathways rely on modulation of SnRK2-mediated phosphorylation of TE. Moreover, the SnRK2-APC/C TE module emerges as a
critical mechanism regulating overall rice plant architecture, in both aerial and underground organs, as well as a putative dial for fine-tuning hormone-mediated growth.

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