Dual controls for seed germination

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Seed germination is tightly regulated so that it only occurs in optimal environmental conditions; for root parasitic plants, this is the presence of potential host plant revealed by strigolactone exudates. New research shows that, unexpectedly, this response to strigolactone bypasses the core gibberellin-dependent pathway for germination.

46 words

Main text

Germination: it's a risky business. Emerging from their hardened shelter, each seedling has only one chance to establish before their stockpiled resources are exhausted. It's no wonder then, that germination is one of the most tightly regulated events in the life of a seed plant. Seeds continually monitor their environment, waiting for exactly the right set of conditions before venturing forth. For most plants, some combination of water, nutrients and sunlight nicely fits the bill; for fire-following species, evidence of a conflagration is also much appreciated. But for holoparasitic plants in the Orobanchaceae, which include the crop pests of the *Striga* genus, the overriding concern is to find and exploit a host plant. Coiled like a serpent ready to strike, *Striga* seeds are superbly adapted to sniff out the scent of neighbouring plants, and to germinate when their prey strays too near. But how are these distinct germination cues integrated to produce a coherent response? New research from Bunswick *et al.* in this issue sheds light on this important question.

The key germination stimuli for most root parasitic plants are strigolactones, small molecules present in root exudates[1]. Plants exude strigolactones primarily to attract symbiotic fungi, but in doing so, advertise their presence to these pernicious parasites[2]. Previous work has elegantly demonstrated that members of the Orobanchaceae use receptors of the HTL/KAI2 α/β hydrolase family to perceive exogenous strigolactones[3]. Intriguingly, in most flowering plants, members of the HTL/KAI2 family promote germination in response to perception of smoke-derived 'karrikin' molecules, and not strigolactones[4]. Even more intriguingly, the closely related D14 family of α/β hydrolases do act as receptors for endogenous strigolactones in most flowering plants[4]. Thus, parasitic plants seem to have 're-evolved' a

D14-like function within the HTL/KAI2 pathway, to tie germination to the presence of neighbouring plants, rather than their likely absence[5]. Typically, discourse on germination revolves around the interplay of two hormones, abscisic acid and gibberellin; the former inhibiting, the latter promoting germination[6]. Ultimately, germination is understood to occur when gibberellin levels become high enough to trigger degradation of the growth-repressing DELLA proteins, allowing the dramatic and sustained growth of the seedling[3]. So how does perception of karrikins and strigolactones relate to this core germination machinery?

The study from Bunswick *et al*. addresses this question, with exciting and unexpected results. Since studies in *Striga* are technically difficult, the authors utilised *Arabidopsis thaliana* as a model to investigate how *Striga hermonthica* HTL receptors regulate seed germination in response to strigolactones. By expressing six different *S. hermonthica* HTL proteins in Arabidopsis, they showed that the strigolactone analogue $GR24_{rac}$ could induce seed germination, even in the complete absence of gibberellin or gibberellin-sensitivity. This substitution was germination-specific, as other gibberellin-deficient mutant phenotypes were not rescued, and was also independent of endogenous Arabidopsis strigolactone receptors. Gibberellin-mediated germination in Arabidopsis occurs through degradation of DELLA repressors, in particular RGL2[8]. Using a RGL2-GFP reporter protein fusion, the authors showed RGL2 is not degraded when ShHTL7 is activated by $GR24_{rac}$, but the seeds were still capable of germinating. Thus, strigolactone-induced germination appears to bypass DELLA-mediated germination inhibition altogether.

The endogenous Arabidopsis HTL/KAI2 pathway also functions in germination, but only plays a minor role under rather specific circumstances[4,9]. Arabidopsis HTL/KAI2 signals through activation of the F-box protein MAX2, promoting degradation of the SMAX1 repressor protein[1]. The authors observed that, even if ShHTL7 is present, $GR24_{rac}$ cannot induce germination in a *max2-1* loss-of-function mutant background, strongly implying that ShHTL7 acts through the canonical MAX2/SMAX1 pathway. Indeed, the authors also found that loss of SMAX1 was sufficient to allow germination in gibberellin-depleted seeds, further suggesting HTL/KAI2 pathway activation and resultant SMAX1 inactivation can circumvent gibberellin signalling. That the MAX2/SMAX1 pathway can trigger germination independent of gibberellin in ShHTL7 transgenic lines was surprising, since karrikin treatment, which should inactivate SMAX1, cannot rescue germination in gibberellin-deficient wild-type *Arabidopsis*[10]. The authors thus hypothesised that native seed HTL/KAI2 levels are insufficient for SMAX1 suppression. Consistent with this, they found that HTL/KAI2 is poorly expressed during early germination of *Arabidopsis*, and that ectopic HTL/KAI2 expression in seed permitted much stronger karrikin-induced germination under gibberellin depletion.

Furthermore, the authors showed that gibberellin depletion inhibited increased HTL/KAI2 expression during germination, suggesting that gibberellin normally promotes HTL/KAI2 expression, in effect turning on a second checkpoint for germination.

The exciting work of *Bunswick et al*. suggests there are at least two independent pathways (DELLA and SMAX1) inhibiting seed germination, and that overcoming either may be sufficient for germination – although the synergistic activation of both pathways undoubtedly promotes a better response. Given the traditional centrality of gibberellin in germination research, this is a very intriguing finding indeed. This study also demonstrates that 'differential wiring' of the two germination pathways provides plants with the opportunity to alter the balance of inputs required for germination (Figure 1). This work provides an exciting advance in understanding germination, while opening up new avenues of study. For instance, does DELLA and SMAX1 function converge on the same downstream regulatory pathways? How do other plants with non-canonical germination, such as fire-followers, use these two pathways to regulate their germination? Perhaps most pertinently, how can we use this new knowledge to inhibit the germination of pests such as Striga, to reduce their devastating impact on harvests in the developing world? Ultimately, improved knowledge of the pathways regulating germination should also allow us to refine germination in crop plants, tailoring them for future climatic conditions.

896 words

Author Contributions

DCM and TB wrote this article.

Competing Interests

The authors declare they have no competing interests.

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Figure Legends

Figure 1. The interplay of multiple pathways regulating germination.

A) In Arabidopsis, the gibberellin pathway is the predominant germination signal, causing inactivation of DELLA proteins to lift germination repression. This also promotes expression of HTL/KAI2, allowing the endogenous “KAI2 ligand” to activate the HTL/KAI2 pathway, causing inactivation of SMAX1 and further lifting germination repression. B) In fire followers, activation of both the gibberellin pathway and the HTL/KAI2 pathway (the latter with karrikins derived from smoke) are required to lift germination repression, through degradation of DELLA proteins and SMAX1, respectively. C) In root parasitic plants, activation of the HTL pathway by host-derived strigolactones and subsequent inactivation of SMAX1 is the sole germination stimulus. The function of the gibberellin pathway in these plants is unknown.