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Control of seed germination in the shade

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Abbreviations: SCBA, seed coat bedding assay; ABA, abscisic acid; GA, gibberellic acid; phyA, phytochrome A; phyB, phytochrome B

Seeds consist of encapsulated plant embryos in a highly resistant and desiccated state. Seeds enhance plant fitness by allowing plants to withhold their growth until the appearance of appropriate environmental conditions. Thereupon, seeds germinate, allowing the resumption of the plant's life cycle. However, protection and propagation is not expected per se to confer fitness without germination control mechanisms avoiding potentially fatal starts such as germination in an inappropriate season or in the face of sudden environmental changes in light or water quality. It is therefore not surprising that multiple levels of germination control mechanisms have appeared during plant evolution. The first level, present in most plant species, including in the model plant Arabidopsis, is that of seed dormancy where the germination of newly produced seeds is blocked even under favorable germination conditions (i.e., presence of adequate water and light). Seeds must therefore first lose their dormancy in order to germinate. Dormancy is progressively lost over time (after-ripening) or following a cold treatment under wet conditions (stratification). Once seeds become post-dormant (i.e., able to germinate when exposed to adequate germination conditions), a second level of germination control unfolds, where the pace of germination is adjusted in response to the specific physical parameters of the seed's environment.

In Arabidopsis, as in many plant species, the seed coat is essential to prevent dormant seed germination, since its removal triggers embryonic growth.[1] The Arabidopsis seed coat consists of an outer layer of dead tissue, the testa, and a single cell layer of living endosperm tissue surrounding the embryo (**Fig. 1**). To study the germination-repressive activity of the seed coat, we developed a seed coat bedding assay (SCBA), where the growth of dissected embryos laid on a bed of dissected seed coats is monitored, allowing combinatorial use of seed coat and embryo material of different genetic or ecotype origins.[2] The SCBA therefore allows dissecting genetically in vitro the signaling pathways underlying the seed coat's germination-repressive activities. We could show that, upon dormant seed imbibition, the endosperm continuously synthesizes and releases abscisic acid (ABA), a growth-repressing hormone, toward the embryo, thus blocking germination. Thus, the first level of germination control appears to be extra-embryonic and "delocalized" to the endosperm.

We recently explored whether the endosperm is also involved to control seed germination in non-dormant seeds by considering how Arabidopsis seeds respond to the shade of neighboring plants.[3] Plants detect canopy light by measuring the relative intensity of far red (FR) light and red (R) light through light photoreceptors called phytochromes.[4] Unlike sunlight, canopy light (unfavorable for photosynthesis) is poor in red light relative to FR light. Remarkably, a pulse of FR light inhibits seed germination early upon seed imbibition, whereas later on it will stimulate seed germination.[5-7] These paradoxical responses may reflect that it is advantageous for a plant to repress germination upon seed imbibition in the shade of other plants. However, it is most certainly disadvantageous to prevent germination indefinitely in the shade.[8]

This developmental switch is controlled by phytochromes A (phyA) and B (phyB). FR light exerts opposite effects on their signaling activities: FR activates phyA, while it inactivates phyB. Early upon seed imbibition, phyB inactivation by a pulse of FR light prevents the synthesis of gibberellic acid (GA), a growth hormone negatively regulating ABA synthesis.[9,10] As a result, ABA accumulates and germination is blocked. Later upon seed imbibition (i.e., about 2 d), a second FR pulse will activate phyA, thus triggering GA synthesis, which, in turn, will downregulate ABA levels and allow seed germination.

phyB accumulates in dry seeds, and its levels remain roughly constant upon seed imbibition. In contrast, phyA protein levels are very low in dry seeds and increase progressively upon seed imbibition.[7] It was therefore proposed that phyB-dependent responses dominate over phyA-dependent responses early upon seed imbibition by virtue of phyB's higher accumulation.[7] However, we observed that seed coat removal of seeds treated with an early FR pulse led to embryonic growth, unlike intact seeds. Thus, despite phyB inactivation by FR light, embryos may germinate provided the seed coat is removed, directly suggesting that the endosperm is releasing ABA, as in dormant seeds, in response to an early FR light pulse.

Using the SCBA, we showed that phyB and phyA signal in the endosperm and embryo, respectively. phyB inactivation in the endosperm by a first FR pulse prevents GA synthesis, which leads to ABA synthesis and release toward the embryo (**Fig. 1**). In turn, endospermic ABA overrides phyA-dependent stimulation of GA

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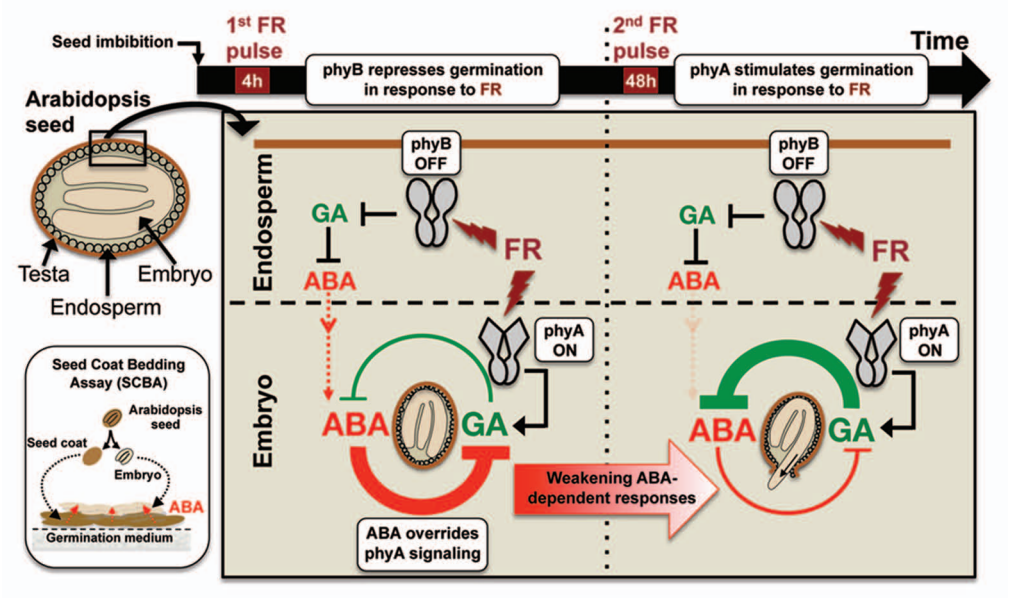

Figure 1. Model describing the control of seed germination under the canopy (enriched in far red light). Far red light blocks germination early upon seed imbibition and stimulates germination later on. Active phytochromes promote GA synthesis. GA negatively controls ABA levels and vice versa. ABA represses germination. Overtime, a weakening of ABA-dependent responses facilitates FR- and phyA-dependent GA synthesis thus leading to germination. The inset on the lower left corner describes the procedure to assemble a seed coat bedding assay.

synthesis in the embryo. Thus, GA and ABA act to negatively regulate each other's levels. The developmental switch leading to FR-dependent stimulation of phyA-dependent germination is best explained by a gradual decrease over time in ABA-dependent responses rather than increased phyA protein levels during imbibition (**Fig. 1**). Thus, the developmental switch is a switch from ABA dominance over GA early upon seed imbibition to later GA dominance over ABA in seeds imbibed for a few days (**Fig. 1**). Light-dependent control of germination is not the apanage of Arabidopsis. It remains to be explored whether this developmental switch and the particular role of the seed coat occur in a similar manner for the light-dependent control of seed germination in different plant species.

References

1. Finch-Savage WE, et al. New Phytol 2006; 171:501-23; PMID:16866955; http://dx.doi.org/10.1111/j.1469-8137.2006.01787.x.
2. Lee KP, et al. Proc Natl Acad Sci USA 2010; 107:19108-13; PMID:20956298; http://dx.doi.org/10.1073/pnas.1012896107.
3. Lee KP, et al. Genes Dev 2012; 26:1984-96; PMID:22948663; http://dx.doi.org/10.1101/gad.194266.112.
4. Kami C, et al. Curr Top Dev Biol 2010; 91:29-66; PMID:20705178; http://dx.doi.org/10.1016/S0070-2153(10)91002-8.
5. Reed JW, et al. Plant Physiol 1994; 104:1139-49; PMID:12232154.
6. Shinomura T, et al. Plant Physiol 1994; 104:363-71; PMID:12232088.
7. Shinomura T, et al. Proc Natl Acad Sci USA 1996; 93:8129-33; PMID:8755615; http://dx.doi.org/10.1073/pnas.93.15.8129.
8. Mathews S. Mol Ecol 2006; 15:3483-503; PMID:17032252; http://dx.doi.org/10.1111/j.1365-294X.2006.03051.x.
9. Piskurewicz U, et al. Plant Cell 2008; 20:2729-45; PMID:18941053; http://dx.doi.org/10.1105/tpc.108.061515.
10. Piskurewicz U, et al. EMBO J 2009; 28:2259-71; PMID:19556968; http://dx.doi.org/10.1038/emboj.2009.170.