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Seed-to-seedling transition: chloroplasts on a Polycomb leash?

The establishment of a photoautotrophic seedling involves complex developmental and metabolic reprogramming, mediated by chromatin remodelling and orchestrated changes in gene expression. Similar to other major developmental transitions in plants, the seed-to-seedling transition requires the activity of Polycomb Repressive Complexes (PRCs) - evolutionarily conserved chromatin-modifying protein complexes that establish facultative heterochromatin at repressed genes. The role of PRCs in the seed-to- transition has previously been associated with the repression of the embryo maturation programme represented by the "LAFL" (LEC1, ABI3, FUS3, LEC2) transcription factors. Recently, however, a more complex picture is emerging implementing Polycomb repression in developmental, as well as metabolic, reprogramming during the developmental transition.

Our work focuses on understanding the functions of the H3K27me3 histone methyltransferase PRC2 in orchestrating the seed-to-seedling transition, with an emphasis on primary metabolism and greening. We have recently revealed a role for PRC2 in seedling establishment that is independent of LAFL suppression, but influences appropriate responses to ambient light during photomorphogenesis. Using transcriptome and H3K27me3 distribution profiles of source and sink tissues at different developmental time points, we identified genes involved in primary metabolism, chloroplast development and operational control that are subject to PRC2-dependent transcriptional repression. Due to defects in chloroplast morphology and function in PRC2 mutants, we focused on chloroplast biogenesis to demonstrate the involvement of PRC2 repression in the transcription of nuclear-encoded photosynthesis-associated genes. Our results suggest that PRC2 may be an important component of organelle-nucleus signalling that orchestrates metabolic and developmental cues. These and other recent findings extend our understanding of PRC2 function beyond its well-studied effects on developmental gene regulatory networks and raise interesting questions about the interplay between metabolic and developmental cell identity.

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