

## ROLE OF GIBBERELLIN AND *PICKLE* IN DETERMINATION OF DEVELOPMENTAL IDENTITY

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### ABSTRACT

The ability of gibberellin (GA) to promote developmental transitions such as germination or flowering is well known. Similarly, it is also well appreciated that acquisition of a new developmental state is accompanied by the expression of a new suite of genes that typify that state. Much remains to be learned regarding the regulatory mechanisms by which this change in gene expression is achieved, particularly with respect to repression.

*PICKLE* (*PKL*) encodes an ATP-dependent chromatin remodeling factor that acts to turn off genes that are expressed in developing seeds. *pkl* plants continue to express embryonic traits after germination. This ectopic expression of embryonic traits is dramatically evidenced by the ability of *pkl* seedlings to generate an altered primary root – referred to as the “pickle root” phenotype based on its visual appearance – in which many seed-specific programs continue to be expressed, including accumulation of seed storage compounds such as triacylglycerol, and the ability to generate somatic embryos. Microarray analysis reveals that many genes associated with seed development – including the *LEAFY COTYLEDON* (*LEC*) class of master regulators – fail to be repressed during germination of *pkl* seeds. Use of an inducible version of *PKL* confirms that *PKL* acts during germination to repress expression of these genes.

In addition to playing a critical role in regulation of gene expression, *PKL* is also involved in gibberellin (GA) signaling. GA plays a profound role in the development of higher plants and is well known for its ability to promote processes such as germination of seeds, shoot elongation, and flowering. Much remains to be learned, however, regarding the factors and mechanisms by which GA signal transduction takes place. GA appears to act through two separate pathways to influence the ability of *PKL* to repress embryonic traits. GA acts via an uncharacterized response pathway to repress expression of the pickle root phenotype in *pkl* seedlings. In addition, *pkl* plants express the phenotypic hallmarks of a plant defective in GA signal transduction, suggesting that *PKL* itself codes for a component of a GA signal transduction pathway. Based on these and other observations, we propose that GA acts through both a *PKL*-dependent and a *PKL*-independent pathway to repress expression of embryonic traits during germination.

Reciprocal shift studies reveal that GA, like *PKL*, acts during germination to prevent expression of embryonic traits in germinating seedlings. We used Affymetrix ATH1 microarrays to examine the contribution of *PKL* and GA to gene expression during germination. The results of this analysis substantially clarify the respective roles of these two factors in repression of embryonic traits.

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