# REGIONAL CENTRE FOR BIOTECHNOLOGY

## Seminar series

# Structural Epigenetic regulation of KNOX loci in differentiating *Arabidopsis* leaves


**Mukesh Lodha, PhD**

Cold Spring Harbor Laboratory,
USA


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**Seminar Room**

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## Abstract

In both plants and animals, transcriptional regulation of gene expression is central to the control of stem cell homeostasis. The stable but reversible silencing of genes that drive differentiation is a defining property of stem cells, whereas repression of key stem cell factors is required to allow cellular differentiation and the progression through normal development. In plants, the careful balance between stem cell propagation and differentiation is attained in part through the precise spatiotemporal regulation of the class I KNOTTED1-like homeobox (KNOX) genes. These genes promote stem cell activity and their expression marks indeterminate cells within shoot meristems. The stable repression of KNOX activity in determinate lateral organs involves distinct and possibly overlapping cellular memory systems. In *Arabidopsis*, silencing of the *KNOX* genes *BREVIPEDICELLUS* (*BP*) and *KNAT2* in the developing leaf is mediated by *ASYMMETRIC LEAVES 1* (*AS1*) and *ASYMMETRIC LEAVES 2* (*AS2)*. These DNA-binding proteins form a complex that binds the promoters of *BP* and *KNAT2* and recruits chromatin-remodeling factors to maintain these genes in a somatically heritable silenced state throughout leaf development. We have shown that Polycomb repressive complex 2 (PRC2) is one of these complexes. In normal leaves, levels of H3K27me3 at *BP* and *KNAT2* are enriched but, in *as1* and *as2*, H3K27me3 levels at these *KNOX* loci are dramatically reduced. Loss of this PRC2 signature is correlated with reduced PRC1 occupancy at *BP* and *KNAT2.* Moreover, the PRC2 component CURLY LEAF localizes to the KNOX promoters in wild type leaves but not in leaves of *as1* and *as2* mutants. These data identify AS1 and AS2 as upstream components and possible recruitment factors for PRC2 in the KNOX silencing pathway. Bimolecular fluorescence complementation (BiFC) and protein immunoprecipitation assays were used to investigate possible direct interactions between AS1, AS2 and PRC2 components. These experiments revealed multiple physical interactions for both AS1 and AS2 with core subunits of PRC2. Further, AS1-AS2 binding sites can recruit PRC2 independent of genomic location. These data demonstrate that cellular differentiation is achieved via an epigenetic mechanism in which the AS1-AS2 complex serves to recruit Polycomb group proteins to pluripotency factors to stably suppress their expression throughout organ differentiation.