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Current Opinion in Plant Biology

The age of multiplexity: recruitment and interactions of Polycomb complexes in plants

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Polycomb group (PcG) proteins form distinct complexes that modify chromatin by histone H3 methylation and H2A mono-ubiquitination leading to chromatin compaction and epigenetic repression of target genes. A network of PcG protein complexes, associated partners and antagonistically acting chromatin modifiers is essential to regulate developmental transitions and cell fate in all multicellular eukaryotes. In this review, we discuss insights on the subfunctionalization of PcG complexes and their modes of recruitment to target sites based on data from the model organism *Arabidopsis thaliana*.

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Current Opinion in Plant Biology 2016, **29**:169–178

This review comes from a themed issue on **Growth and development**

Edited by **Doris Wagner** and **Dolf Weijers**

http://dx.doi.org/10.1016/j.pbi.2015.11.010

Introduction

PcG protein complexes play a crucial role in the development of multicelluar organisms including plants [1,2]. Trimethylated lysine 27 of histone H3 (H3K27me3), H2A mono-ubiquitinated at a PKKT consensus motif (H2Aub), and chromatin compaction are widely accepted as their core functional readouts. In the classic model, Polycomb Repressive Complex (PRC) 2 is recruited to target genes allowing its SET-domain component of the Enhancer of zeste (E(z)) type to deposit H3K27me3, which recruits a second complex, PRC1, *via* the interaction of a H3K27me3-binding chromodomain component [1,2]. H2Aub is then deposited by RING-RAWUL twin domain proteins of the RING1 and BMI1 type which are present in PRC1 [1,2].

Recent data from plant and animal models suggest that this textbook view of the PcG mechanism may be too linear. First, several new mechanisms of PcG-recruitment were identified suggesting that the order of events can be reversed so that PRC1 acts upstream of PRC2 [3••,4••]. This more circular view of the pathway is also indicated by the discovery of direct interactions between components of PRC1 and PRC2 [5•]. In the following, we will place some of the recent results in the context of the multi-layered and complex PcG network described in *Arabidopsis thaliana* (Arabidopsis).

Defects in PcG repression: how bad does it get?

Arabidopsis PcG genes occur in small families, allowing combinatorial assembly of complexes with different activities, partners and target preference, as well as differential temporal and spatial distribution. The partial redundancy between paralogs allows genetic analysis of non-lethal phenotypes, which is both a blessing and a curse given that extreme phenotypic pleiotropy makes proper genetic analysis near impossible [1,6]. Nevertheless, the combined efforts of many groups link PcG mutants or mutant combinations with key targets during specific developmental stages (Table 1).

H3K27me3 derives from the activity of the SET domain proteins CURLY LEAF (CLF), SWINGER (SWN), or MEDEA (MEA). MEA is at the heart of a gametophyte/endosperm-specific PRC2 that also encompasses the Suppressor of zeste 12 (Su(z)12)-related C2H2-domain protein FERTILIZATION INDEPENDENT SEED 2 (FIS2) and the 7-blade-WD40-propeller protein FERTILIZATION INDEPENDENT ENDOSPERM (FIE), present in gametophyte, developing endosperm and sporophyte. Deleterious mutations in *FIS2*, *MEA* or *FIE* lead to embryo abortion caused by overproliferation and delayed/abolished cellularization of the endosperm [7]. Gametophytic and sporophytic PRC2 complexes are also distinct due to differences in protein primary sequence since overexpression of *SWN* or *MEA* fails to suppress the *clf* mutant phenotype of upward leaf curling and early flowering [8].

Approximately 10–15% of all genes are covered by H3K27me3 in both gametophyte/endosperm and sporophyte, but the sets are quite distinct [9,10]. PcG-target genes in the endosperm include many transposable elements (TEs), which are heterochromatic and H3K27me3-depleted in the sporophyte [9,11]. H3K27me3 could protect the developing endosperm from the activation of TEs by global DNA de-methylation in the central cell of the female gametophyte [12]. However, embryo abortion is likely not caused by TE deregulation but by upregulation of other direct FIS2-PRC2 targets such as

Table 1

***A. thaliana* PRCs, mutants mirroring the function of a PRC, affected target genes putatively causal for phenotype, chromatin change in mutants and developmental phenotype**

Mutants	Interaction modules	Confirmed and putative causal target genes	Chromatin change	Phenotype
	PRC2			
fis, mea, fie	FIS2-MSI1-FIE-MEA/SWN	*AGL62* and *PHE1*	Reduction of H3K27me3	Central cell division without fertilisation, endosperm overproliferation, failed endosperm cellularization, embryo arrest (endosperm/ embryo phenotype)
msi1	FIS2-MSI1-FIE-MEA/SWN	unknown		Endosperm/embryo phenotype + more
emf2	EMF2-MSI1-FIE-CLF/SWN	*FLC, FT, AG, SEP3*	Reduction of H3K27me3	Early flowering and upward leaf curling
vrn2	VRN2-MSI1-FIE-CLF/SWN	*FLC*	Reduction of H3K27me3	Failed maintanance of FLC repression
clf	VRN2/EMF2-MSI1-FIE-CLF	*FLC, FT, AG, SEP3*	Reduction of H3K27me3	Mild early flowering and upward leaf curling
clf swn	VRN2/EMF2-MSI1-FIE-CLF/SWN	unknown	Loss of H3K27me3	PcG-callus
	PRC1			
atbmi1ab (weak)	AtBMI1A/AtBMI1B-PRC1-like	*miR156* and *FLC*	Reduction of H2Aub and reduction of H3K27me3	Late flowering
atbmi1ab (strong)	AtBMI1A/AtBMI1B-PRC1-like	*LEC* genes, *ABI3* and *WUS*	Loss of H2Aub	Defective seed maturation, PcG-callus
atbmi1abc	AtBMI1A/AtBMI1B/ AtBMI1C-PRC1-like	*LEC* genes, *ABI3* and *WUS*	Loss of H2Aub	Defective seed maturation, embryo without root, PcG-callus
atbmi1ab al6 al7	AtRING1-AtBMI1-LHP1-AL6/AL7	*ABI3* and *DOG1*	Gain of H3K27me3 and loss of H3K4me3	Impaired seed germination
atring1a	AtRING1A-CLF-LHP1	*MAF4* and *MAF5*	Reduction of H3K27me3	Late flowering
atring1ab (weak)	AtRING1A/ AtRING1B-LHP1	*KNOX* genes	Reduction of H3K27me3	Enlarged apical meristem, fasciated stem
atring1ab (strong)	AtRING1A/ AtRING1B-LHP1	unknown	Reduction of H3K27me3	PcG-callus
lhp1	LHP1-PRC1-like	*FT, AG* and *SEP3*	Reduction of H3K27me3	Early flowering and downward leaf curling
emf1	EMF1-PRC1-like	*AG, AP3* and *miR172*	Reduction of H3K27me3 and impaired chromatin compaction (?)	Extreme early flowering
jmj14	EMF1-LHP1-JMJ14	*FT*	Reduction of H3K27me3	Early flowering

genes encoding for the interacting MADS-domain factors *PHERES 1 (PHE1)* and *AGL62* [13,14•]. Mutation of either compensates for the loss of FIS2-PRC2 activity and at least partially rescues seed development [13,14•,15].

Rescue of embryo development has also been achieved by adding a compensatory mutation to *fie* mutants that alleviates the defect of endosperm overproliferation by removing the paternal contribution [16]. Although *FIE* is encoded by a single copy gene, a substantial proportion of rescued seeds develop normal embryos leading to the astonishing conclusion that PcG function is not essential for embryogenesis *per se* [17]. Post-germinative growth of *fie* plants results in the formation of an amorphous cell cluster that randomly starts forming embryo and leaf-like structures without ever completing organ development (PcG-callus), a phenotype also observed in *clf swn* double mutants [8]. Likewise, strong double mutants of PRC1 components *AtBMI1a* and *AtBMI1b* or weak triple mutants including the mostly gametophyte-specific third gene, *AtBMI1c*, develop a PcG-callus [3••,18]. Strong *atbmi1abc* triple mutants show embryo abortion/endosperm phenotypes similar to *fie* [3••]. Segregating siblings of *atbmi1* double and triple mutant combinations form classes of either strongly or only weakly affected homozygous individuals. The lack of full phenotypic penetrance is likely due to the available *atbmi1b* allele, which is a knock-down mutation. The occurrence of phenotypic classes illustrates that PcG-mediated repression is crucial at discrete steps of development, when switching takes place between major developmental programs.

MSI1 — Jack of all trades?

The animal 7-blade-WD40-propeller protein Nurf55/ Rbap48 binds histones H3 and H4 [19]. The Arabidopsis