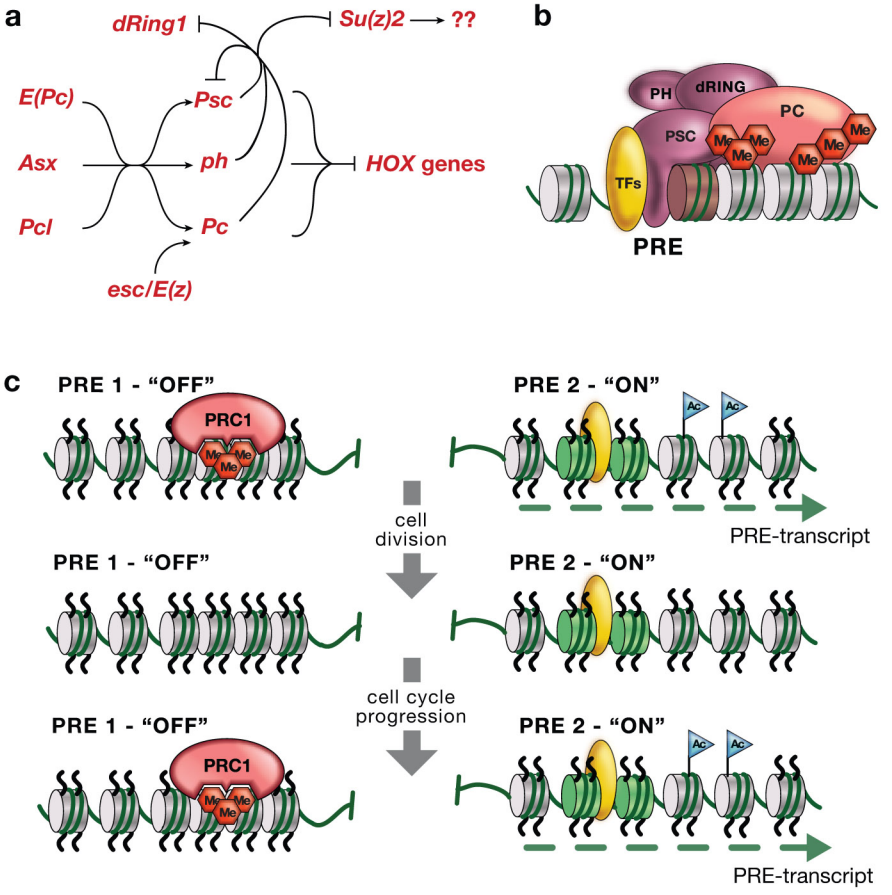

Figure 7. PRC1 Regulation and Function during Cell Division

(*a*) Cross-regulatory interactions among the PcG genes, as suggested from genetic evidence. *E(Pc)*, *Pcl*, and *Asx* are positive regulators of the core PRC1 members acting upstream. PRC2 members *Esc* and *E(z)* act as positive regulators of *Pc* transcription. A negative feedback by core PRC1 members on *Psc* and *dRing1*, as well as on *Su(z)2*, is observed. The fine-tuning of gene product level is probably required for well-balanced processes based on chemical equilibrium. (*b*) Sequence-specific transcription factors (TF) tether components of PRC1 to a PRE. A stable silencing complex requires anchoring of PRC1 via the chromodomain of PC to neighboring methylated histone tails. (*c*) Possible model for how differential gene expression states can be inherited. The process of intergenic transcription places positive epigenetic marks (e.g., acetylated histone tails, histone variants) at PREs that control active genes (PRE 2). All other PREs are silenced by default (PRE 1). During DNA replication and mitosis, only the positive epigenetic signal needs to be transmitted to the daughter cells, ensuring that in the next interphase intergenic transcription is restarted at PRE 2 before default silencing is reestablished at all other PREs. (*a*, Adapted from Ali and Bender 2004.)