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Multifaceted actions of Polycomb repressive complex 2 during embryogenesis

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Throughout embryogenesis, Polycomb repressive complex 2 (PRC2) silences transcription via its histone-modifying activity to establish and maintain distinct gene expression that drives cellular plasticity and identity. However, the spatial and temporal dynamics of PRC2 during development and its molecular mechanisms that control proper embryogenesis remain largely unknown. This is especially crucial as aberrant expression of PRC2 has been frequently reported in various diseases, including cancers.

We implemented a mouse embryonic stem cell-based model, termed trunk-like structure (TLS), to recapitulate multiple developmental events of gastrulating embryos, including somitogenesis and neural tube formation. By combining a protein degradation strategy that allows acute depletion of the PRC2 complex in a time-course manner, we showed early onset of PRC2 depletion led to failed symmetry breaking, primitive streak formation, and axial elongation. This suggested that PRC2 activity is instrumental in cellular responses to WNT signaling. Intriguingly, transient restoration of PRC2 during WNT activation could partially rescue these defective phenotypes.

Finally, we are applying low-input Cut&Tag coupled with RNA sequencing to understand the causality between perturbed epigenetic marks and developmental defects. Ultimately, we aim to unveil PRC2-mediated regulation of molecular and cellular processes during development.

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