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Polycomb repression complex 2 is required for the maintenance of retinal progenitor cells and balanced retinal differentiation

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Abstract:

Polycomb repressive complexes maintain transcriptional repression of genes encoding crucial developmental regulators through chromatin modification. Here we investigated the role of Polycomb repressive complex 2 (PRC2) in retinal development by inactivating its key components *Eed* and *Ezh2*. Conditional deletion of *Ezh2* resulted in a partial loss of PRC2 function and accelerated differentiation of Müller glial cells. In contrast, inactivation of *Eed* led to the ablation of PRC2 function at early postnatal stage. Cell proliferation was reduced and retinal progenitor cells were significantly decreased in this mutant, which subsequently caused depletion of Müller glia, bipolar, and rod photoreceptor cells, primarily generated from postnatal retinal progenitor cells. Interestingly, the proportion of amacrine cells was dramatically increased at postnatal stages in the *Eed*-deficient retina. In accordance, multiple transcription factors controlling amacrine cell differentiation were upregulated. Furthermore,