Title:	The morphogen Decapentaplegic employs a two-tier mechanism to activate target retinal determining genes during ectopic eye formation in Drosophila
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Abstract:	Understanding the role of morphogen in activating its target genes, otherwise epigenetically repressed, during change in cell fate specification is a very fascinating yet relatively unexplored domain. Our in vivo loss-of-function genetic analyses reveal that specifically during ectopic eye formation, the morphogen Decapentaplegic (Dpp), in conjunction with the canonical signaling responsible for transcriptional activation of retinal determining (RD) genes, triggers another signaling cascade. Involving dTak1 and JNK, this pathway down-regulates the expression of polycomb group of genes to do away with their repressive role on RD genes. Upon genetic inactivation of members of this newly identified pathway, the canonical Dpp signaling fails to trigger RD gene expression beyond a threshold, critical for ectopic photoreceptor differentiation. Moreover, the drop in ectopic RD gene expression and subsequent reduction in ectopic photoreceptor differentiation resulting from inactivation of dTak1 can be rescued by down-regulating the expression of polycomb group of genes. Our results unravel an otherwise unknown role of morphogen in coordinating simultaneous transcriptional activation and de-repression of target genes implicating its importance in cellular plasticity.
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