Title:	Non-canonical Decapentaplegic Signaling Activates Matrix Metalloproteinase 1 To Restrict Hedgehog Activity and Limit Ectopic Eye Differentiation in Drosophila
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Abstract:	One of the pertinent issues associated with cellular plasticity is to understand how the delicate balance between the determined state of cells and the extent to which they can transdetermine is maintained. Employing the well-established model of generating ectopic eyes in developing wing discs of Drosophila by ectopic eyeless expression, we provide evidence for the genetic basis of this mechanism. By both loss-of-function and gain-of-function genetic analyses, we demonstrate that Matrix metalloproteinase 1 (Mmp1) plays an important role in regulating the extent of ectopic ommatidial differentiation. Transcriptional activation of ectopic Mmp1 by the morphogen Decapentaplegic (Dpp) is not triggered by its canonical signaling pathway that involves Mad. Rather, Dpp activates an alternate cascade involving dTak1 and JNK, to induce ectopic Mmp1 expression. Mutational analyses reveal that Mmp1 negatively regulates ectopic eye differentiation by restricting the rate of proliferation and the levels of expression of retinal determining genes dachshund and eyes absent. This is primarily achieved by restricting the range of Hh signaling. Importantly, the increase in proliferation and up-regulation of target retinal determining genes, as observed upon attenuating Mmp1 activity, get significantly rescued when ectopic eyes are generated in wing discs of hh heterozygous mutants. In conjunction with the previously established instructive and permissive roles of Dpp in facilitating ectopic eye differentiation in wing discs, the outcome of this study sheds light on a mechanism by which Dpp plays a dual role in modulating the delicate balance between the determined state of cells and the extent they can transdetermine.
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