Title:	Molecular mechanisms underlying Zebrafish retina regeneration: insights from Hippo signaling
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Abstract:	Unlike higher vertebrates, Zebrafish possess remarkable regenerative response in retina, driven by Muller Glia (MG), the only glial cell type in retina. But the exact mechanism of molecular interplay that orchestrate de-differentiation, proliferation and re-differentiation still remains elusive. Previously, it was reported that Wnt signalling plays key role in retina regeneration of zebrafish and β-catenin stabilization was shown to be one of the key factors driving proliferation. Following injury, in a cascade of signaling events many regeneration- associated genes like ascl1a, lin28a, mycb, mmp9, hdac1, her4.1, insm1a, and cytokines like il- 11b were reported to be regulated. Although the roles of many genetic and epigenetic regulators have been identified in retina regeneration, that of developmentally important Hippo signaling pathway remains under- explored. In this study, we found components of Hippo signaling like yap1 to be up-regulated very early during the de-differentiation phase and inhibiting nuclear localization regulates proliferation differently at different phases of regeneration. This study also shows the possible interplay of Hippo signaling with Wnt signaling as revealed by a reduction in stabilized β-catenin in Yap1 inhibited background. Further, we also aimed towards establishing a mechanistic involvement of different regeneration-associated genes like mmp9 and lin28a, and Hdac1 in mediating the effects of Hippo signaling in zebrafish retina regeneration. We also showed that the reduction in proliferation upon continuous inhibition of nuclear localization of Yap1 is reversible in nature. Finally we also tried to find out possible signaling pathways or molecules which might be regulating yap1 and subsequently the Hippo signaling pathway. Our study provides new insights in to the molecular interplay during retina regeneration and provides potential targets for stimulating mammalian Muller Glia to regenerate following retinal damage.
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