Title:	Profiling of Inflammatory Mediators During Regeneration in Adult Zebrafish Retina.
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Abstract:	Retinal injuries, caused due to various reasons, are great burden and it often leads to serious consequences due to permanent tissue damage and life-long impairments. The human CNS possesses very low regenerative capacity and there are no therapies available to cure disorders that impact the normal function of the CNS. Retinal injury is accompanied by strong neuroinflammation, including accumulation of immune cells, activation of microglia (immune cells of the CNS), secretion of various inflammatory-mediators (pro-inflammatory and anti-inflammatory cytokines in case of mammals secreted by two distinct M1, M2 activated microglial subtypes). In case of humans, neuroinflammation persist over years contributing to chronic scar formation and is hence regarded as a major determental process that is considered as the cause of low regenerative capacity. In contrast, zebrafish is known to successfully regenerate injured tissues including retina after the lesion. Importantly, the neuroinflammatory response is required for the injury induced MG proliferation, and regenerative neurogenesis in the zebrafish. However, identification of specific inflammatory-mediators involved in this process remained obscure. Hence, this study was involved in the identification of the molecular and cellular composition of the neuroinflammatory response upon light lesion in the adult zebrafish retina. Additionally, the role of NF-kB signalling in case of inflammation has been known for many years. Hence, some preliminary experiments were performed to decipher the role of NF-kB signalling in regeneration during the proliferation of MG and how this signalling pathway impacts MG proliferation in the presence and absence of microglia.
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