

Abstract

Retina regeneration is the process of endogenous repairing of damaged retina. This regeneration process depends upon a single retinal glial cell type in the retina- the Müller glial cells. Müller glial cells are present in invertebrates and mammalian retina. Zebrafish, which naturally regenerates its damaged retina and visual functions, attains this property through the interplay of various signaling pathways such as Wnt-, Notch-, Mapk-erk, Jak-stat etc. These pathways turn on and regulate crucial process of reprogramming and proliferation of Müller glial cells. Microarray analysis has shown the differential expression of thousands of genes associated with retina regeneration as compared to a normal retina. c-fos oncogene was earlier reported to be responsible for tumor progression and oncogenic transformation. In the context of cancer, c-fos is upregulated by various signaling pathways including Mapk-Erk, Jak-Stat etc. and they are induced rapidly after stimulation. c-fos or any of the Fos family member is not studied in the context of retina regeneration in zebrafish. c-myc, an oncogene is important for Müller glial cell reprogramming and proliferation and studies have shown the regulation of c-fos and c-myc by growth factors. In the context of Chick retinal regeneration, Hedgehog signaling has been shown to regulate c-fos. All these indicate a possible role of c-fos and fosl1 during retina regeneration. This study is basically trying to see the various characteristics of c-fos and fosl1 including spatio-temporal expression profile, regulatory pathways, regulatory function etc. by employing cellular and molecular based techniques like time course studies, in situ hybridization etc. and essential pharmacological and Morpholino mediated knockdown approach.