Abstract

Retinal cells degenerate in a variety of different diseases like diabetic retinopathy, glaucoma, age-related macular degeneration. Unfortunately, mammals are incapable of regenerating retinal cell types. Mammals exhibit a limited propensity for regeneration in tissues and organs like skin, skeletal muscle and liver but CNS (central nervous system) do not regenerate in mammals. The retina is a part of CNS and zebrafish (Danio rerio) can regenerate most of its organs like heart, liver, fin, including retina which makes zebrafish a suitable model for studying tissue regeneration. Zebrafish act as a unique model system to understand the molecular mechanism involved in retina regeneration. The fish and mammalian retina structure are similar and composed of identical cell types with conserved function. Müller glial cells have an essential role in zebrafish retina regeneration. Upon injury, Müller glial cells undergo reprogramming and divide asymmetrically into multipotent progenitor cells, and these progenitor cells give rise to all retinal cell types. Retina regeneration requires many growth factors and signaling cascades like transforming growth factor(TGF-β), Sonic Hedgehog(SHH), and WNT signaling pathways. These pathways are involved in EMT(epithelial-to-mesenchymal transition). EMT is a critical step in development and regeneration. The crosstalk of these signaling pathways in EMT are sophisticated and remain explored. In this project, we have investigated the effect of YAP nuclear inhibition on zebs and cadherins and crosstalk of zebs and cadherins with HDACs and klf4 in early dedifferentiation phase, and we showed that zebs and cadherins could have an essential role in zebrafish retina regeneration since EMT can be closely similar to dedifferentiation and proliferation.