**Abstract**

The retina is the light-sensitive tissue layer that lines inside of the eye that sends visual signals to the brain through the optic nerve. Eye injuries and certain eye diseases can damage retinal tissue and leads to blindness. Retinal regeneration refers to the recovery of vision in vertebrates that have suffered retinal lesions or retinal degeneration. Lower vertebrates, like zebrafish, shows the extensive regenerative capability of their retina. Mammals like mice have the same genes and pathways, but they lack such extensive regenerative potential. Müller glia is the cells from which all other retinal cell types are regenerated in zebrafish. These cells support the healthy functioning of neighboring neurons and hold the innate regenerative ability. Wnt signaling pathways play indispensable roles in cell fate specification, cellular proliferation, and differentiation. LiCl can inhibit GSK3β activity and stabilize free cytosolic β‐catenin efficiently, thereby behaving as an agonist of canonical Wnt signaling. Though lithium potently inhibits GSK-3 beta activity. It is not a general inhibitor of other protein kinases. Lithium shows neuroprotective nature against a wide variety of processes, including anticonvulsants and potassium deprivation. It is also known to promote the neuronal survival and axonal regeneration of retinal ganglionic cells through a Bcl-2-dependent mechanism in the rat model. This study was done to understand the variation in lithium-induced regenerative responses between mouse and zebrafish retina. BrdU staining was used to establish the proliferation status of retina both in terms of the number of cells and their localization. Gene expression patterns in both models were analyzed and compared. Overall this study gives new insights and better comparison of LiCl effect on injury responses between mouse and zebrafish retina models.