Elucidating the Relationship Between *sox4a* and *sox4b* in the Zebrafish Eye

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Abnormal development of the vertebrate eye can result in long-term defects, one of which is ocular coloboma. Ocular coloboma is a congenital malformation that occurs when the choroid fissure fails to close during embryonic development. It can result in visual impairment and is also associated with other morphological defects like micropthalmia and cataracts. SOX4 is a transcription factor that has been shown to be associated with ocular coloboma. SOX4 belongs to the SoxC subfamily of proteins, characterized by the presence of a high mobility group (HMG) domain-containing Sry-box, that allows the proteins to bind to DNA, and a transactivation domain that allows the proteins to function as a transcription activator. In previous studies, morpholinos were used to knock down the expression of Sox4 in zebrafish. This resulted in coloboma, micropthalmia, and fewer rod photoreceptors. These morphant phenotypes were supported by preliminary data observed from individual CRISPR genetic mutants in *sox4a* and *sox4b*. However, the mutants have a less severe phenotype than the morphants and previous attempts to make a double mutant were not successful. To determine whether mutation of one *sox4* co-orthologue sensitizes the zebrafish to loss of the second co-orthologue, we injected a low dose of *sox4b* morpholino into *sox4a* mutant embryos, and compared the penetrance and severity of the resulting ocular phenotypes to control injected and single mutant embryos. The data presented here suggest that a low dose of *sox4b* morpholino does increase the penetrance and severity of some of the microphthalmia phenotype.