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Loss of Sox4 Impacts Early Ocular Development

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Abstract Text

SoxC transcription factors are highly conserved among vertebrates and have pleiotropic functions during embryonic development. Our lab has previously demonstrated that SoxC factors regulate choroid fissure closure in the zebrafish eye. Zebrafish contain two copies of the SoxC transcription factor *sox4*, *sox4a* and *sox4b*. To further explore the role of *sox4a/b* during eye development, we generated zebrafish *sox4a* and *sox4b* mutants.

Methods: All animal procedures were performed in accordance with guidelines established by the ARVO Statement for the Use of Animals in Ophthalmic and Vision Research. The *sox4a* and *sox4b* genetic mutant lines were generated by CRISPR/Cas9 using two single strand guide RNAs targeting deletion of the HMG domain for each gene. Amplification of the coding sequence by PCR was used to identify organisms with a large deletion in either *sox4a* or *sox4b*. Ocular morphogenesis was visualized using light microscopy and the Rx3:GFP transgenic line by lightsheet microscopy. Images obtained by lightsheet microscopy were analyzed with the software ARIVIS. qPCR was used to examine mRNA levels of *sox4a/b* and other genes of interest.

Results: Maternal zygotic mutants for both sox4a and sox4b displayed microphthalmia. Sox4a/b mutants display an eyefield with reduced volume and transgene expression, which were apparent as early as the single somite stage. Additionally, it appears that the shape of the eyefield may be abnormal, this may be due to a delay in ocular morphogenesis or due to differences in the patterning of the eyefield.

Conclusions: These data suggest that *sox4a*/*b* are required for proper

ocular morphogenesis in the zebrafish.

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