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Characterization of Nebl in zebrafish eye development

Semaphorin ligands and their Plexin receptors are known to be a necessary factor of cell migration and repulsive axon guidance during development (Zhou et al., 2008). More so, it has been demonstrated that Semaphorin6A-PlexinA2 signaling is a key factor in eye cell cohesion during bilateral migration of the eye fields (Ebert et al., submitted). If either of these components are lost, ligand or receptor, the migrating eye is subject to decreased tissue stability. When a microarray was run of Semaphorin6A and PlexinA2 deficient zebrafish (*Danio rerio)* embryos, several novel proteins, including Nebulette (nebl), were found to be up-regulated. Nebulette was up-regulated 4 fold compared to control embryos. My hypothesis is that nebl is negatively transcriptionally regulated by PlexinA2 signaling. This would mean that loss of PlexinA2 promotes an increase in expression of nebl, causing cell cohesion loss in the developing eye. Using Zebrafish as a model organism, we have determined the nebl genome sequence, fashioned primers specific to this sequence and, through the use of polymerase chain reaction and reverse transcriptase, constructed an antisense RNA *in situ* probe. Images of *in situ* hybridization lend evidence that nebl mRNA is expressed in the eye, suggesting that nebl can be regulated by PlexinA2 signaling. Future work on this project will include generating and injecting a full-length zebrafish nebl construct to phenocopy PlexinA2 morphants, and double knockdown of PlexinA2 and nebl to rescue the phenotypic effects.