Sema6a-PlxnA2 Signaling Negatively Regulates *rasl11b* to Maintain Proliferation of Retinal Precursor Cells during Zebrafish Eye Development

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Eye development in vertebrates is a complex process involving many developmental cues and signal transduction pathways. We have uncovered an important role for Semaphorin6a-PlexinA2 (Sema6a-PlxnA2) signaling in maintaining cellular cohesion and proliferation in developing zebrafish eye fields. However, the molecular mechanisms underlying these phenotypes are unknown. Microarray analysis found that upon morpholino-based knock-down of Sema6a or PlexinA2, there was a three-fold increase in the expression levels of the poorly-characterized small GTPase RasL11b. The present study used established cell lines and transgenic zebrafish to test the hypothesis that overexpressed RasL11b has a dominant-inhibitory effect on Ras-MAPK signaling and thereby contributes to the reduced retinal precursor cell proliferation observed in Sema6a or PlxnA2 morphant eye fields.