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Sp21-120

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Funding Source:
MARC/IMSD - NIH-funded Maximizing Access to Research Careers/ Initiative for Maximizing Student Diversity

Evaluating Neuronal Migration in Celsr1 and Wnt5a Double Mutants

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Defective neuronal migration during development can contribute to several brain disorders, including epilepsy. The goal is to understand the mechanisms of neuronal migration to help remedy these human brain disorders. Since the migration pathways of the Facial Branchiomotor (FBM) neurons are well-studied and an evolutionarily conserved process, this system will be used to examine phenotypes. The current model proposes that the function of the chemoattractant Wnt5a is blocked by the membrane receptor Celsr1 to prevent inappropriate rostral migration. Previous studies with *Wnt*-soaked beads showed that excess *Wnt5a* can induce rostral migration. In addition, *Celsr1* knockout mutants exhibited a rostral migration phenotype, suggestive of a role for Celsr1 in suppressing chemoattractant activity. To further test the model, both the *Celsr1* and *Wnt5a g*enes will be knocked out, and the migration phenotype in embryos' hindbrains will be examined. Double heterozygous *Celsr1+/KO Wnt5a+/KO* mouse lines have been generated, and preliminary findings are consistent with the proposed model.