Genetic Interaction Between gpr125 and knypek in Convergence and Extension Movements

Songyuan Geng
Washington University in St. Louis

Follow this and additional works at: https://openscholarship.wustl.edu/wuurd_vol12

Recommended Citation
https://openscholarship.wustl.edu/wuurd_vol12/66

This Abstracts A-I is brought to you for free and open access by the Washington University Undergraduate Research Digest at Washington University Open Scholarship. It has been accepted for inclusion in Volume 12 by an authorized administrator of Washington University Open Scholarship. For more information, please contact digital@wumail.wustl.edu.
Genetic Interaction between gpr125 and knypek in Convergence and Extension Movements

Songyuan Geng

Mentor: Lillianna Solnica-Krezel

Vertebrate gastrulation and formation of the embryonic body plan relies on polarized cell behaviors to drive convergence and extension movements (C&E). Wnt/Planar Cell Polarity (Wnt/PCP) pathway components, which function was initially described in Drosophila epithelial planar polarity have been identified as essential during gastrulation cell movements. Zebrafish embryos carrying mutations in core Wnt/PCP genes present characteristic phenotypes with shorter and wider body axes, a consequence of perturbed C&E movements. The Solnica-Krezel lab has identified Gpr125, an adhesion G protein-coupled receptor, as a modulator of the Wnt/PCP signaling. Zebrafish embryos carrying mutations in gpr125 gene present characteristic phenotypes with shorter and wider body axes, a probable consequence of perturbed C&E movements. This is reminiscent of the Wnt/PCP pathway components mutant phenotype, for example: knypek (kny).

In this experiment, we are trying to determine if there is a genetic interaction between the gpr125 and kny gene to have a better understanding of the gpr125 role during C&E. Based on previous data showing that gpr125 has a genetic interaction with trilobite and scribble1 mutants, both components of the PCP pathway, our hypothesis is the gpr125 mutant gene will affect kny mutant phenotype. The first stage of the experiment was carried out by incrossing the double heterozygous fish (gpr125+/- and kny+/-). A stronger kny phenotype was observed in several clutches. Subsequently, the embryos were pooled according to their phenotype (length of the body axis). The different classes of the embryos were then subjected for genotyping to determine the presence of the mutant gpr125 allele in the stronger kny mutant category.