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Genetic Interaction between *gpr125* and *knypek* in Convergence and Extension Movements

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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 initial 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.