



## Short Communication

### Vangl2 regulates membrane-protrusive activity in migrating gastrula cells

Anna Love and Jason Jessen

Our lab works to determine the mechanism whereby the planar cell polarity (PCP) protein Vang-Like 2 (Vangl2) regulates cell migration during embryogenesis. We focus on the gastrula stage of zebrafish development as the cells naturally undergo PCP-dependent migration. Loss of Vangl2 in trilobite mutant embryos results in a strong convergence and extension phenotype characterized by shortened and broadened body axes. Here, both ectodermal and mesodermal cell populations fail to polarize. Previous data established migrating vangl2 mutant cells lack directionality and meander compared to wild type. We have also shown vangl2 mutants have increased matrix metalloproteinase activity and decreased fibronectin extracellular matrix (ECM). We hypothesize defective cell-ECM interactions underlie the

vangl2 mutant phenotype. Using time-lapse confocal imaging, we have now analyzed the membrane protrusive activity of ectodermal cells from wild-type and PCP mutant embryos. Our current data suggest vangl2 mutant ectodermal cells exhibit increased membrane protrusive activity and have significantly fewer polarized protrusions. Our data suggest filopodia are concentrated at the trailing edge in wild-type cells, while vangl2 mutant cells have more filopodia at the leading edge. We also found that vangl2 mutant ectodermal cells have reduced directness compared to wild type. To determine the requirement for fibronectin during protrusion formation, we used morpholinos to knockdown fibronectin protein expression in wild-type embryos. The data showed that fibronectin morpholino injected cells exhibited increased formation of non-polarized membrane protrusions similar to vangl2 mutant cells, suggesting defective cell-ECM interactions contributing to at least a portion of the mutant phenotype. Our preliminary studies suggest decreased Vangl2 protein localization to filopodia and larger membrane protrusions. Together, our data suggest a model whereby Vangl2-dependent regulation of cell-ECM interactions is required to suppress inappropriate proper membrane protrusive activity.

**Note :** This work is partially presented at Annual Congress on Cell Science, Stem Cell Research & Pharmacological Regenerative Medicine during November 29-30, 2017 in Atlanta, USA

#### Author Affiliations

Middle Tennessee State University, USA