Rescuing Convergent Extension after Inhibition of an Aquaporin

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Much is known about the function of aquaporins within individual cells. Aquaporins are membrane protein channels that are permeable to water and a subset, the aquaglyceroporins, are also permeable to glycerol. Little research has been conducted on how they contribute to larger processes such as gastrulation. Gastrulation organizes embryos into germ layers, which will later form different body tissues. Convergent extension cell movements are critical for driving gastrulation. During convergent extension, cells fold into the embryo at the dorsal lip of the blastopore and then merge to help form the long body axis. An aquaglyceroporin, aqp3b, is expressed during convergent extension. When it is inhibited using a morpholino oligonucleotide, convergent extension does not occur properly. Since this process is difficult to manipulate in whole embryos, I explant and culture the dorsal lip of the blastopore region of embryos, which then undergoes convergent extension by growing long and narrow protrusions. When aqp3b is inhibited, these protrusions do not develop. My project focuses on rescuing the convergent extension defects caused by inhibiting. If rescue methods are successful, explants will form a long and narrow protrusion as observed in control embryos. For these experiments, 4-cell embryos are injected into the dorsal blastomeres and explants are cut at early gastrula stage. So far, I have achieved 25-35% convergent extension in control explants. I plan to achieve 80% convergent extension and will then begin the rescue experiments.