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## RESCUING CONVERGENT EXTENSION AFTER INHIBITION OF AN AQUAPORIN (POSTER)

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Much is known about how aquaporins function within individual cells. Aquaporins are membrane protein channels that are permeable to water and a subset, aquaglyceroporins, are also permeable to glycerol. Little research has been conducted on how they contribute to larger processes such as gastrulation. Gastrulation organizes cells into germ layers, which will later form different body tissues. Convergent extension cell movements are critical in driving gastrulation. During convergent extension, cells that folded into the embryo at the dorsal lip of the blastopore 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, which we assay using Keller tissue explants from gastrula embryos. The aquaporin aqp2 and aquaglyceroporins aqp7 and aqp9 were cloned in order to conduct rescue experiments to determine whether it is the water or glycerol permeability of Aqp3b that functions in convergent extension. I have successfully cloned the aqp7 coding region, but errors made by Taq polymerase introduced mutations into the aqp2 and aqp9 sequences. I am still working to resolve these issues. In the meantime, I have begun to determine how Aqp3b interacts with noncanonical Wnt signaling, the primary signaling pathway involved in convergent extension, utilizing the same techniques. The embryos are injected with the aqp3b morpholino oligonucleotide, as well as the mRNA for a protein involved in noncanonical Wnt signaling. If convergent extension is rescued in the explants, then Aqp3b acts through noncanonical Wnt signaling.