How AQP3B Influences Convergent Extension Through Noncanonical WNT Signaling

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Aquaporin-3b, Aqp3b, is an aquaglyceroporin, a membrane water channel, that is present during gastrulation and various other stages of embryonic development. Gastrulation organizes cells into germ layers, which will later form different body tissues. During gastrulation, cells fold into the embryo, then merge by convergent extension to form the long body axis. These cell movements are regulated by noncanonical Wnt signaling, an intercellular signaling pathway that controls the migration and polarity of tissues. When Aqp3b is inhibited using a morpholino oligonucleotide (MO), convergent extension does not occur properly, suggesting a link between Aqp3b and noncanonical Wnt signaling. To assay these defects, we use the Keller tissue explanting method to observe convergent extension. Our goal is to determine which Wnt signaling pathway(s) are influenced by Aqp3b. We conducted rescue experiments by inhibiting Aqp3b with morpholino oligonucleotides and co-injecting an RNA or DNA construct of several proteins involved in Wnt signaling. Successful rescue with Dvl1ΔDix and Dvl2ΔDix constructs indicated that Aqp3b is involved in noncanonical Wnt signaling, since DvlΔDix acts in all noncanonical Wnt signaling. Further, Aqp3b acts through the Wnt/Ca2+ subpathway, indicated by rescue by PKC and PMA, and through a branch of the Wnt/PCP pathway, indicated by successful rescue with RhoA but not with Rac1. Aqp3b does not directly affect the Wnt/Ror2 pathway. In conclusion, I have demonstrated that the ability of Aqp3b to influence convergent extension is dependent on noncanonical Wnt signaling, specifically the Wnt/Ca2+ pathway and the RhoA branch of Wnt/PCP pathway.