Investigating the Role of Innexins and Calcium Regulation of a Highly Conserved Mechanism for Axial Patterning in the Drosophila Embryo

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Morphogen gradients determine distinct cell fate within tissues, and it has previously been thought that the cells affected by this determination are static. However, cells usually move during development, complicating this process. Two morphogens have the greatest effects: decapentaplegic (dpp), which attracts cells toward the dorsal midline, and dorsal (dl), stalls them ventrally. Another gene, frazzled (fra), is the main downstream effector regulated by these morphogens that constrict cells and also provides the mechanical force that is necessary to draw them dorsally, while also modulating the gradients of both dpp and dl. This force is provided by protecting E-Cadherin (E-Cad) from cleavage, i.e. allowing it to be expressed. E-Cad then acts as the actual protein causing cell movement and constriction or expansion. It is suggested that gap junction proteins (which are formed by Innexins (INX)) may play a large role, as they regulate Ca2+, which forms a gradient across the embryo as well. Additionally, Ca2+ seems to increase dpp activation in late-stage embryos as well as physically interact with E-Cad. The three innexin genes relevant are ogre, inx2, and inx3, which form gap junctions that regulate both intercellular and extracellular calcium flux. Of these, ogre is the gene most prominently looked at in the experiments. The effect of ogre on calcium concentration throughout the cell then directly affects the morphogen gradient across the cell, thereby affecting cell fate.

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