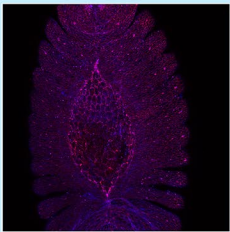




## ***HALLE RONK***

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Wild-type *Drosophila* embryo during dorsal closure  
(red: Canoe, blue: DE-cadherin)

### **Characterizing the role of Canoe as a cell junction-cytoskeletal linker protein during *Drosophila* morphogenesis**

Adherens junctions (AJs) connect epithelial cells to one another and connect the plasma membrane to the actomyosin cytoskeleton; this organization translates contractility to neighboring cells and preserves tissue integrity during morphogenesis. It is not known exactly how the AJs are organized. The current model postulates that actin cables bind directly to unknown linker proteins, which bind to  $\alpha$ -catenin, which binds to  $\beta$ -catenin, which binds to the transmembrane cadherin. We examined whether Canoe acts as a junction linker protein by testing its role in maintaining epithelial integrity, which is an indicator of junctional integrity. We used immunofluorescence and confocal microscopy to examine *Drosophila melanogaster* embryos during dorsal closure. In wild-type embryos, Canoe is enriched at the leading edge epidermis and at tricellular junctions along the lateral epidermis. This enrichment pattern aligns closely with the location of actin cables and myosin II heavy chain. We used RNA interference in conjunction with the Gal4-UAS system to reduce *canoe* function. Loss of *canoe* caused cells along the lateral epidermis to become highly variable in shape, suggesting that translation of contractility to AJs occurred unevenly among the cell population. Furthermore, the number and regularity of puncta of Enabled, an actin assembly and elongation factor usually enriched at AJs, decreased across the leading edge. These results support the hypothesis that Canoe acts as a cytoskeletal-junction linker protein, playing an essential role in regulating epithelial sheet integrity and contractility embryogenesis. Understanding these complex interactions can provide insight into the mechanisms of wound healing and neural tube closure in humans and guide the creation of embryonic defect prevention therapies.