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Modulation of actomyosin-based vertex mechanics controls epithelial permeability

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Tricellular junctions (TCJs) at cell vertices are key sites that control paracellular transport as well as passage of migrating cells and pathogens. However, the mechanisms underlying TCJ remodeling are not clear. Here we analyze the roles of actomyosin dynamics during TCJ remodeling in Drosophila ovaries, where cell vertices open transiently to allow passage of yolk proteins through the follicle epithelium for uptake by the oocyte. We show that follicle cells comprise a unique organization of actomyosin filaments anchored end-on at TCJs. These structures are rapidly lost and actomyosin contractility is reduced before vertex opening, while F-actin is reassembled at vertices as they close. Consistent with these findings, we show that actin polymerization and myosin II activity are not required for vertex opening, but for closure, whereas stabilizing F-actin or constitutive activation of myosin II are sufficient to prevent vertex opening. Thus, actomyosin-based forces play distinct roles during vertex opening and closure, respectively. Our findings reveal how modulation of actomyosin-based vertex mechanics controls epithelial permeability, providing a framework for elucidating related processes, such as endothelial remodeling during leukocyte extravasation in vertebrates.

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