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Regulation of microtubule mechanics during epithelial morphogenesis

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Coordinated rearrangements of cytoskeletal structures govern cell and tissue morphogenesis. In contrast to actin-based forces, our knowledge of microtubule (MT)-based forces is still limited. In recent years, we have shown that the Ft-PCP signaling pathway globally patterns the MT cytoskeleton to coordinate local cell behavior during *Drosophila* wing morphogenesis. Interestingly, in *ft-PCP* mutant animals, where MT alignment is disrupted, cells and tissue fail to elongate, suggesting an interdependence of these processes. Furthermore, we have shown that physical forces based on the global patterning of MTs contribute to cell mechanics. However, the mechanisms regulating MT organization and patterning during tissue remodeling are not understood. We recently showed that the formation of non-centrosomal MTs in wing cells depends on MT minus-end binding protein Patronin (CAMSAP in vertebrates). Consistently, depletion of Patronin leads to cell and tissue elongation defects.

Importantly, MT-based mechanical properties should be adaptable to environmental forces that vary considerably across tissue types to be functionally appropriate. Strikingly, our analysis revealed that non-centrosomal MTs in wing cells have a mean diameter of 29 nm, substantially larger than the diameter of canonical 13 protofilament MTs (~25nm). This is significant because the increased diameter of the tubular architecture caused by the increased number of protofilaments dramatically increases flexural rigidity, thus providing an alternative mechanism for cells to regulate MT stiffness and adapt to cell mechanics. Furthermore, as the formation of non-centrosomal MTs in wing cells depends on Patronin, our data suggest that Patronin regulates MT lattice diameter by regulating the number of MT protofilaments. Together, these results provide the molecular basis to explain how regulating MT properties and organization controls cell mechanics during tissue remodeling.

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