



Title : Mechanosensitive binding of p120-Catenin regulates E-Cadherin

turnover and Viscoelastic behavior of Epithelial Tissues

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Abstract:

Studying how epithelia respond to mechanical stresses is key to understanding tissue shape changes during morphogenesis. Here, we study the viscoelastic properties of the *Drosophila* wing epithelium during pupal morphogenesis, by quantifying mechanical stress and cell shape as a function of time. We find a delay of 8 hours between maximal tissue stress and maximal cell elongation indicating a viscoelastic deformation of the tissue. We show that this viscoelastic behaviour emerges from the mechanosensitivity of endocytic E-Cadherin turnover. The increase in E-Cadherin turnover in response to stress is mediated by mechanosensitive relocalization of the E-Cadherin binding protein p120 Catenin from cell junctions to cytoplasm. Mechanosensitivity of E-Cadherin turnover is lost in p120 mutant wings, where E-Cadherin turnover is constitutively high. In this mutant, the relationship between mechanical stress and cell elongation is altered. Cells deform more rapidly in response to stress, indicating a lower viscosity. Unlike wild type, p120 mutant cells deform rapidly, reverting to their original shape when stress is relaxed. Taken together, our findings reveal that p120-dependent mechanosensitive E-Cadherin turnover regulates viscoelastic behaviour of epithelial tissues, allowing mechanical stresses to generate stable cell shape changes during development.

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