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Cell mechanics and non-genetic developmental defects[1]

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Genetic mutations are not the only, nor necessarily the most prevalent route to misregulation of morphogenesis. In fact, remarkably specific developmental defects can be caused by non-specific environmental stress – e.g., heat shock, anoxia or chemical exposure. I will discuss one example from *Drosophila* embryos in which the funneling from broad-spectrum insult to specific developmental defects can be traced to cell and tissue mechanics. In particular, I will discuss heat shocks applied to *Drosophila* embryos at the onset of gastrulation. These lead to common developmental defects in head involution and germband retraction – the latter phenocopying U-shaped mutants. Although these heat shocks induce a wide range of transient effects – on protein synthesis, cytoskeletal structures, and the cell cycle – morphogenetic movements resume after heat shock and proceed nearly normally for several hours. Then, four to ten hours after heat shock, dramatic holes open between cells in the amnioserosa, disrupting the integrity of this monolayer epithelium. The presence of holes in the amnioserosa at this stage (germband extension) is highly correlated with later defects in retraction of the germband – a tissue adjacent to the amnioserosa. This observation begs two questions: (1) how does heat-shock of the entire embryo lead to mechanical disruption of this specific tissue; and (2) how does this mechanical disruption lead to morphogenetic defects in adjacent tissues? Using a combination of quantitative live imaging, laser-microsurgery, FRAP and computational models, we find answers to both questions in the underlying cell- and tissue-level mechanics.

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