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Abstract:	Tissue remodelling is a process ubiquitous in nature, showing up in processes from wound healing to early morphogenesis. During this process of remodelling, inter-cellular junctions must shrink to allow cell shape changes. For this thesis, we focus on the experimentally observed germ band extension in the Drosophila Embryo. This is known to be initiated by the active shrinkage of the vertical junctions. Experiments have also observed that this shrinkage happens in a ratchet-like manner. The steps of the ratchet are controlled by the density of molecular motors (Myosin II and E-Cadherin clusters) with noisy dynamics at the cell junction. There is a delicate interplay of the force generating Myosin and the force sensing E-Cadherin. We attempt to model this using a Myosin-II driven stochastic ratchet. The cell junction length is modeled analogous to an active spring. We assume that the rest length of the spring changes with each step, acting as the ratcheting mechanism and preventing backlash errors in the junction length. We construct non dimensional difference equations for the dynamics and attempt to understand the dynamics of the junction length. We find conditions for 'good' and 'bad' ratchets in our system. We also attempt to study the effects of offsets and lags between the cadherin and myosin dynamics on the junction length.
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