BiophysTO Lunchtime Talks

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Dynamic force patterns promote collective cell migration during embryonic wound repair

An outstanding question in cell biology is how cells coordinate their movements during tissue assembly and repair, and also in metastatic disease. A common feature of cells that move collectively is the reorganization of cytoskeletal proteins that mediate force generation. During embryonic wound repair, for instance, actin and the motor protein non-muscle myosin II become polarized in the cells adjacent to the wound, forming a supracellular, cable-like structure around the wound whose contraction promotes cohesive cell migration and rapid wound closure. We showed that, in Drosophila embryos, the actomyosin cable that drives wound closure displays a heterogeneous pattern, with regions of high and low actomyosin density. Mutants in which the heterogeneous actomyosin distribution at the wound margin is lost display significantly slower wound closure. However, the mechanisms by which a non-uniform distribution of cytoskeletal proteins favours rapid wound repair are unknown. We are using *in vivo* quantitative imaging, biophysical manipulations, and *in silico* modelling to investigate how the distribution of cytoskeletal molecules affects collective cell migration during wound closure in Drosophila embryos. Our studies will reveal the physical and molecular mechanisms that cells use to move collectively, which could facilitate the identification of therapeutic targets to promote or inhibit cell migration in disease.

Host: Dr. Anton Zilman

(Refreshments and pizza will be provided)

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Thursday, December 3, 2015 – 12:00 pm, noon
Davenport Room, Chemistry Building
(and via streaming to Davis Building 4001 UTM)