INTERCELLULAR INTERACTIONS PROMOTE COLLECTIVE BEHAVIOR IN BACTERIAL COLONIES AND DEVELOPING EPITHELIA

Abstract

by

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Collective behavior has been seen in several animal groups including a school of fish, a flock of birds, swarming bacteria, and even in epithelial tissues of Drosophila fruit fly during development. This self-organized motion, which is resulted from local interactions, leads to group behavior in time and size scales that are often larger than the scale of interactions between individual organisms. In this thesis, we study the impact of physical properties of individual bacteria and intercellular interactions on the swarming behavior, as well as the effect of mechanical interactions between epithelial cells on the tissue properties during development. Using a combination of computational modeling and experimentation, we explore the role that pili interactions play in expanding swarms of the pathogenic bacterium Pseudomonas aeruginosa in different environmental conditions. We find that pili interactions in a colony of wild-type P. aeruginosa promote a moderate rate of swarm expansion, and also enables bacteria to alter their movement to avoid toxic regions. Although the population of mutants that lack pili interactions acquire a relatively higher rate of swarm expansion compared to wild-type, they can not avoid the toxins by deviating their motion. Next, we extend a biologically calibrated computational model to investigate the mechanism that is used by a colony of swarming Myxobacteria to optimize the rate of making cell-cell connections and spreading protein within the population. This contact-mediated
signaling mechanism is necessary for bacterial self-organization during predation, fruiting body formation and genetic repair of damaged cells. We reveal that periodic reversals of motion direction with periods within the experimentally observed range, high flexibility of bacterial cells and a moderate level of strength of cell-cell adhesion are crucial for a colony of self-propelled rod-shaped bacteria to make a balance between the rate and the duration of cell-cell connections and spread protein within the population efficiently. Finally, we develop a detailed multi-scale biophysical model to study the dynamics of growth of epithelial tissues. During epithelial proliferation, in a process known as mitotic rounding, cells increase their size and roundness which is necessary for a robust segregation of chromosomes during division. Mitotic rounding coincides with several structural changes in a cell as it enters mitotic phase, including an increase in cytoplasmic pressure, increase in the polymerization of actomyosin at the cell cortex, and reduction of adhesion with neighboring cells. We investigate the impact of the structural changes on the epithelial mitotic rounding. Our results show that increase of cortical stiffness and reduction in cell-cell adhesion are the important factors that play role in increasing the cell roundness, and change in the cytoplasmic pressure is the primary factor that controls the cell size.