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Abstract for an Invited Paper for the 4CF17 Meeting of the American Physical Society

## Unexpected parallelisms: From swimming bacteria to wound healing and cancer metastasis<sup>1</sup> CHARLES WOLGEMUTH, University of Arizona

Over 20 years ago, Neil Mendelson observed whirls and jets in dense colonies of *Bacillus subtilis*. This organized collective motion has since been shown to arise whenever swimming bacteria are at sufficient density. Under appropriate conditions, hydrodynamic effects drive the alignment of nearby bacteria, but dipole-distributed forces from the bacteria destabilize the system and cause the formation of transient vortices and jets. When your skin gets cut, one of the first processes is reepithelialization. The top living layer of your skin, the epithelium, heals itself via the crawling of cells over the wounded region. Experiments have shown that this process involves elaborate coordinated cell motions that include whirling vortices. Are the similarities in these two disparate systems coincidence? Or is similar physics driving these analogous motions? Here I will discuss our attempts to construct mathematical models for these two systems that are grounded in the basic behaviour of the single cells that generate the motions. An intriguing connection is that both swimming bacteria and crawling epithelial cells exert dipole-distributed forces on their surroundings. Using experiments to test these models has led to some unexpected results. For example, it has been shown that while confined suspensions of B. subtilis form a single, stable, counter-rotating vortex, confined E. coli instead forms micro-spin cycles, a persistent periodically reversing vortex. What defines the marked difference between the collective dynamics of these two flagellated swimmers? In addition, in epithelial cells, perturbations that slow isolated cells are found to dramatically increase collective migration. I will show that our models naturally predict these behaviours and can quantitatively match our experimental data. I will conclude by arguing for a biophysical examination of the transition to metastasis in cancer and discuss how our epithelial cell model may provide insights that are currently obscured by traditional genomic and proteomic methodologies.

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