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Collective cell migration during inflammatory response[1] DI WU, KIMBERLY STROKA, HELIM ARANDA-ESPINOZA, Fischell Department of Bioengineering, University of Maryland — Wound scratch healing assays of endothelial cell monolayers is a simple model to study collective cell migration as a function of biological signals. A signal of particular interest is the immune response, which after initial wounding in vivo causes the release of various inflammatory factors such as tumor necrosis alpha (TNF-α). TNF-α is an innate inflammatory cytokine that can induce cell growth, cell necrosis, and change cell morphology. We studied the effects of TNF-α on collective cell migration using the wound healing assays and measured several migration metrics, such as rate of scratch closure, velocities of leading edge and bulk cells, closure index, and velocity correlation functions between migrating cells. We observed that TNF-α alters all migratory metrics as a function of the size of the scratch and TNF-α content. The changes observed in migration correlate with actin reorganization upon TNF-α exposure.

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