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**A computational study of amoeboid motility in 3D: Role of extracellular matrix geometry, cell deformability and cell-matrix adhesion<sup>1</sup>**

PROSENJIT BAGCHI, ERIC CAMPBELL, Rutgers University — Cells exhibiting amoeboid locomotion are abundant within the human body, as immune cells, epithelial cells, neuronal cells, embryonic cells, and even metastatic cancer cells migrate using the amoeboid phenotype. Amoeboid locomotion is accomplished through the use of pseudopods, or cylindrical membrane extensions which protrude, bifurcate, and retract dynamically, resulting in a net cell displacement. The modeling of amoeboid locomotion is a complex and multiscale process, where large cell deformation, protein biochemistry, and both cytosolic and extracellular fluid interactions must be considered. Furthermore, cells are often confined inside the extracellular matrix (ECM), a porous, fluid-filled medium. Adhesive interactions between the cell and underlying substrate add further layers of complexity. In this work, we present a 3D computational model of amoeboid migration in various ECM geometries. Our model couples a fluid/structure interaction for extreme cell deformation, a pseudopod generating activator-inhibitor system, cytoplasmic and extracellular fluid motion, and a fully resolved extracellular matrix. Simulation results show a strong coupling between cell deformability, matrix geometry and cell-ECM adhesion providing valuable on the mechanics of amoeboid migration.

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