Brownian Dynamics Simulation of Actin-Polymerization-Driven Motility

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There is a type of biological motility, used in a form of cell crawling and by intracellular pathogens such as Listeria monocytogenes, that is driven not by motor proteins but by biological self-assembly of the protein actin. During this process, ATP hydrolysis and activation of the protein complex Arp2/3 drive actin self-assembly from monomers (G-actin) to branched networks of filaments (F-actin), thus providing the necessary thermodynamic free energy to push a bacterium or a cell forward. This driven, non-equilibrium self-assembly process is regulated by a cadre of proteins. It is now possible to drive a latex bead through a buffer solution containing only these proteins. Such beads travel through solution propelled by a dense branched actin network at their rear, demonstrating that non-equilibrium self-assembly of F-actin is sufficient to drive motility. We have constructed the first physically consistent Brownian dynamics simulation of actin-driven propulsion. The simulation leads to a self-assembled network that exerts forces on a model bead and pushes it with an average speed. This simulation approach is the first to observe a speed that varies non-monotonically with the concentration of branching proteins (Arp2/3), capping protein and depolymerization rate (Actin Depolymerization Factors), in accord with experimental observations. More importantly, the simulation shows that speed is independent of filament stiffness in contradiction to popular belief. Our results suggest a new interpretation of the origin of motility.