A phosphoinositide-based model of actin waves in frustrated phagocytosis

Marco A. Avila Ponce de Leon
University of Minnesota - Twin Cities, ponce018@umn.edu

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Phagocytosis is a complex process by which phagocytes such as lymphocytes or macrophages engulf and destroy foreign bodies called pathogens in a tissue. The process is triggered by the detection of antibodies that trigger signaling mechanisms that control the changes of the cellular cytoskeleton needed for engulfment of the pathogen. A mathematical model of the entire process would be extremely complicated, because the signaling and cytoskeletal changes produce large mechanical deformations of the cell. Recent experiments have used a confinement technique that leads to a process called frustrated phagocytosis, in which the membrane does not deform, but rather, signaling triggers actin waves that propagate along the boundary of the cell. This eliminates the large-scale deformations and facilitates modeling of the wave dynamics. Herein we develop a model of the actin dynamics observed in frustrated phagocytosis and show that it can replicate the experimental observations. We identify the key components that control the actin waves and make a number of experimentally-testable predictions. In particular, we predict that diffusion coefficients of membrane-bound species must be larger behind the wavefront to replicate the internal structure of the waves. Our model is a first step toward a more complete model of phagocytosis, and provides insights into circular dorsal ruffles as well.