Modelling Actin-based Cellular Motility

Juliane Zimmermann, Mihaela Enculescu, Martin Falcke

Max-Delbrück-Center for Molecular Medicine Berlin, Mathematical Cell Physiology

Cell crawling is a vital process, playing an essential role in embryonic development, wound healing and immune response, but is also responsible for cancer metastasis. The driving phenomenon of cell motility is the protrusion of the cell's plasma membrane and the formation of a thin flat structure at the cell front, called lamellipodium, by means of actin filaments. Steadily binding monomers at one end and loosing monomers at the other end, those biopolymers show a highly dynamic behaviour on different spatial scales.

We combine microscopic and macroscopic models of the actin network in the lamellipodium. In a microscopic description, we model the formation of a lamellipodium by calculating the force which the single semiflexible actin filaments exert on the membrane. This force is crucially dependent on the length of the fluctuating filaments. When analyzing the dynamics of the filaments' lengths, we obtain stationary and oscillatory movements of the plasma membrane. In the bulk of the lamellipodium, actin filaments are cross-linked thereby forming a dense network also called "actin gel". This part can be described in a continuum approximation. The theory of the "active polar gel" captures not only the visco-elasticity of the actin network, but also its polarity due to alignment of the actin filaments into the direction of movement and active contractions created by myosin motors. Our microscopic model then serves as a boundary condition to solve the hydrodynamic equations of the active polar gel and calculate the flow velocity of the gel that determines the absolute velocity of the cell.

As we extend our model to two dimensions and regard the lateral extension of the lamellipodium, we find the spreading of different wave forms along the plasma membrane. Those morphodynamic patterns are in good agreement with experimental results.