A model for myosin anchored actin protrusions

Daniel Bahr[1] and Ludger Santen[1]

[1]*Theoretical Physics Department, University of Saarland, Saarbrücken, Germany*

Filopodia are usually described as being driven by the lamellipodium, arising through ‚convergent elongation', which states that several filaments converge near the membrane to form a Λ-precursor elongating into a fully-sized protrusion [1]. Still there is also evidence that linear actin filaments, that build the core of filopodia can also be nucleated de novo by Formin and that the formation of lamellipodia and filopodia are functionally separable as cells can show protrusions with no apparent sign of a lamellipodium [2].
In order to form a protrusion, filaments need to overcome the force of the membrane pushing them back. To do so there must be a physical connection to the cell's cytoskeleton. Based on studies that show that actin protrusions can be affected by Blebbistatin, a myosin II inhibitor, we introduce a model that proposes this connection to be mediated by non-muscle myosin II. We investigate, whether the processive nature of this molecular motor can account for the statistical properties of protrusions we found in HMEC-1 cells.

[1] A. Mogilner and B. Rubinstein. The physics of filopodial protrusion. Biophysical Journal, 89(2):782–795 (2005).
[2] A. Steffen, J. Faix, G. Resch, J. Linkner, J. Wehland, J. Small, K. Rottner, and T. Stradal. Filopodia formation in the absence of functional wave- and arp2/3- complexes. Molecular Biology of the Cell, 17(6):2581–2591 (2006).