Lamelipodia to Finger-like Protrusion: *In-vitro*, *In-silico* and in Silicon

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During cell migration regulated assembly of actin takes place at the cell leading edge. This process generates different types of cellular protrusions such as Lamellipodia and Filopodia. While in Lamellipodia actin is organized in a dense branched actin network generated by the Actin related protein 2/3 complex (Arp2/3 complex), in the filopodia, actin filaments are organized parallelly into bundles by fascin, an actin bundling protein. While most of the cells generate both types of structures, some cells form exclusively Lamellipodia, while others, are dominated by filopodia. Up to date, it is not fully understood how cells "choose" the appropriate type of structure and what drives the transition from Lamellipodia and filopodia. In addition, quantitative information on the role of Arp2/3 complex and fascin on the transition from lamellipodia to filopodia is still lacking. Here we use an *in-vitro* reconstituted model system to obtain quantitative information on the role of VCA/Arp2/3 complex and fascin on the assembly of actin in bulk. We show that the presence of VCA (activator of the Apr2/3 complex) and Arp2/3 results in spontaneous formation of diffused aster-like structures. In the presence of fascin these asters transition first into stars with bundles of actin filaments growing from the surface. At even higher fascin concentration we opbserve the formation of 'network of bundles'. We find that the transition from asters to stars by fascin is facilitated while reducing the density of the original Arp2/3 network. A phase diagram describing the different types of structures formed as a function of Arp2/3 and fascin concentration is also given. Some new concepts for the study of filopodial dynamics are presented in a new microfluidic device.