The architecture and mechanics of ezrin-linked minimal actin cortices

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The actin cortex is a thin cross-linked network attached to the plasma membrane, being responsible for the cell's shape during migration, division and growth. Direct linkage between the plasma membrane and the actin cortex is controlled by ezrin, a member of the ezrin-radixin-moesin protein family. To become fully functional, ezrin switches from a “dormant” to an active state controlled by binding to the lipid $PI(4,5)P_2$ and phosphorylation of a conserved threonine residue [1,2]. In a reductionist approach, we have created minimal actin cortices on a supported 1-palmitoyl-2-oleoyl-*sn*-glycero-3-phosphocholine (POPC) bilayer doped with the receptor lipid phosphatidylinositol(4,5)-bisphosphate ($PtdIns(4,5)P_2$) to which a constitutively active mutant of ezrin was bound. With this approach, we can modulate the individual components of the membrane, its linkage as well as the actin network including cross-linkers and myosin. By means of fluorescence microscopy, we were able to relate the F-actin architecture to the number of PIP_2/ezrin binding sites at the membrane interface [3] and could resolve the impact of cross-linkers such as fascin and α-actinin on the F-actin structure [4]. Dynamic changes of the network were observed in the presence of myosin motors. Bead tracking microrheology on the membrane attached actin network provided further information about the viscoelastic properties [3]. Our results demonstrate that ezrin serves as a dynamic cross-linker for the actin cortex attached to a lipid bilayer.

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