Roles of the actin cross-linking proteins transgelin and fimbrin in promotion of cortical actin patches in fission yeast

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The internalization of endocytic vesicles at cortical actin patches, a critical life process, is driven by a force producing motor protein called myosin-I (Myo1p in fission yeast). Myo1p consist of three domains (head, neck, and tail regions) that all contribute to the functionality of the protein. The energy released by ATP hydrolysis in the head region propagates a conformational change that is amplified by the neck region and ultimately drives displacement of actin filaments bound by the head leading to force production.
At the 2010 SRC I presented work that suggested a role for Cam2p (a light chain of Myo1p that binds at the neck region) in maintaining Myo1p cellular levels and ensuring maximum force production by Myo1p. This URECA! project followed up that work by focusing on the roles of two actin filament cross-linkers found at fission yeast cortical actin patches (fimbrin/Fim1p and transgelin/Stg1p). Both cross-linkers are highly conserved, suggesting a critical role in the function of actin patches and Myo1p. Using epi-fluorescent microscopy the levels, intensity, and lifetime of Myo1p at endocytic patches were compared in wild-type, *fim1Δ*, and *stg1Δ* cells. Loss of transgelin had no effect on the dynamics of Myo1p. However, loss of fimbrin increased the intensity and lifetime of Myo1p at cortical actin patches. Since defects in fission yeast (or budding yeast) myosin-I function are known to lead to increased myosin-I patch signals and lifetimes, we propose that cross-linking of actin filaments by fimbrin promotes the role of Myo1p at endocytic patches. Future studies will look at Myo1p patch dynamics in a *fim1Δ stg1Δ* double mutant to test whether transgelin can substitute for fimbrin in regulating Myo1p function at endocytic patches.

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