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A Model for Actin Nucleation, Polymerization and Branching Based on the “Sidetrack” Polymerization Pathway. R. Reutzel, C. Yoshioka, L. Govindasamy, E.G. Yarmola, M. Agbandje-McKenna, M.R. Bubb, R. McKenna, Dept. of Biochem. and Mol. Bio., Univ. of Florida, Gainesville, FL.

Cell motility mediated by the actin cytoskeleton is involved in fundamental physiological processes like cell motility, as well as pathological mechanisms such as tumour invasion. Through extensive self-associations and interactions with actin binding proteins, G-actin nucleates into F-actin to mediate these events. The mechanism of nucleation and filament elongation has been exhaustively studied using many biophysical and biochemical techniques but the process is still not well understood. Studies with highly motile cells have suggested that an anti-parallel actin dimer may mediate nucleation, polymerization and branching with pathological implications in these disease states. There is also a widespread belief that nucleation is spawned from an actin trimer complex at the leading edge of mobile cells. Here we present a model for actin polymerization based on the structures of actin dimers and trimers in three crystal systems that supports the “sidetrack” polymerization pathway.

Reutzel, R. et al. Actin crystal dynamics: structural implications for F-actin nucleation, polymerization, and branching mediated by the anti-parallel dimer. (2003) *J. Struct Biol.*, 146(3), 191-201