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PhD 1993 University of Barcelona, Barcelona, Spain,
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Molecular Mechanism of Endocytosis

Current Research

Endocytosis is the process whereby cells internalize part of their own plasma membrane engulfing extracellular solutes. Different mechanisms of endocytosis serve fundamental cellular functions such as the immune response, the uptake of nutrients, the downregulation of hormone receptors or the regeneration of synaptic vesicles. The molecular mechanisms underlying some of these pathways are largely unknown. Our aim is to identify proteins required for the uptake step of endocytosis using the yeast *S.cerevisiae* as a model system. In yeast, endocytosis seems to be dependent on two actin-dependent motor systems: the unconventional myosins-I and the ARP2/3 actin-nucleating complex. Our results suggest that in response to intracellular signals myosins-I are recruited to the plasma membrane where they promote ARP2/3-dependent actin polymerization. Our current hypothesis postulates that a transient actin coat is built at the inner surface of the lipid bilayer. The myosin motor head might then help to remodel the coat and form the primary endocytic profile. Understanding in detail how myosins-I are recruited to the plasma membrane, what is the molecular machinery involved in myosin-I-induced actin polymerization and how is hydrolysis of ATP coupled to deformation of the lipid bilayer are our immediate research goals.

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Ph D student project

How are the yeast myosins-I recruited to the plasma membrane?

Myosins-I are ubiquitous actin-dependent molecular motors that bear a short tail that binds acidic phospholipids. A number of experiments demonstrate that myosins-I shuffle between the cytosol and specific subdomains of the plasma membrane where they fulfill their cellular task. What are the *cis* and *trans* elements required to achieve this particular distribution is unknown. We have established a novel reporter system to directly sense the plasma membrane/myosins-I interaction using a simple plate assay. With this tool in hands, we would like to perform genetic screens to isolate intragenic and extragenic mutations that disturb such interaction. Once putative *cis* and *trans* elements were identified, we would further analyze their role in determining the subcellular distribution of the myosins-I using immunofluorescence and biochemistry. Finally, the functional relevance of the elements identified would be tested by investigating their requirement for endocytosis.

Selected Publications

Research articles on the topic

Munn *et al.* (1995) *Mol Biol Cell* **6**, 1721-1742.

Geli and Riezman (1996) *Science*, **272**, 533-535.

Geli *et al.* (1998) *EMBO J.* **17**, 635-647.

Geli *et al.* (2000) *EMBO J.* **19**, 4281-91.

Review

Geli and Riezman (1998) *J. Cell Sci.* **111**, 1031-1037.

