In This Issue

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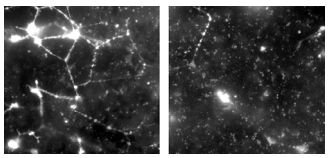
A ribonuclease helps the ER get in shape



ER membranes form an extensive tubular network in control extract (left) but are dispersed in extract lacking XendoU (right).

Schwarz and Blower reveal that a calcium-regulated ribonuclease promotes the formation of tubular ER networks.

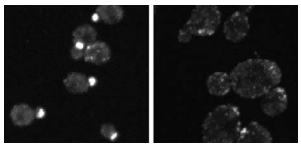
By triggering a wave of intracellular calcium, fertilization induces dramatic changes in the internal organization and protein expression pattern of oocytes. Schwarz and Blower discovered that increased calcium levels activate a ribonuclease called XendoU in *Xenopus* egg extracts. XendoU doesn't appear to degrade any specific RNAs in response to calcium. Instead, the researchers found that depleting the ribonuclease delayed nuclear envelope assembly and restricted the formation of tubular ER networks as the extracts exited meiosis. These membrane organization defects could be rescued by the addition of wild-type XendoU but not by catalytically dead versions of the enzyme.

ER membranes are covered with RNAs and associated ribosomes. Schwarz and Blower determined that a portion of XendoU localized to the ER, where it promoted the release of RNA and ribosomal proteins into the cytosol. This activity stimulated the fusion of ER membranes into a dense, tubular network, possibly by giving the membrane fusion machinery a clear space in which to operate.

Knocking down the human homologues of XendoU also altered ER morphology in HeLa cells. Senior author Michael Blower now wants to investigate the ribonuclease's function in vivo, particularly in tissues where calcium signaling plays a major role, such as in muscles and neurons.

Schwarz, D.S., and M.D. Blower. 2014. *J. Cell Biol.* http://dx.doi.org/10.1083/jcb.201406037.

Bem1p directs vesicular traffic



In the presence of the actin-depolymerizing drug Latrunculin A, Exo70p localizes to the bud tip in wild-type cells (left) but is mislocalized in cells lacking Bem1p (right).

The polarity protein Bem1p recruits the exocyst subunit Exo70p to the site of polarized exocytosis, Liu and Novick reveal.

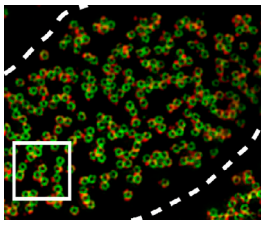
Budding yeast direct secretory vesicles to the sites of polarized bud growth, where they are tethered to the cell cortex by an octameric complex called the exocyst. The exocyst assembles on vesicles as they move along actin cables into the bud, but two of its subunits—Exo70p and Sec3p—are also recruited directly to the sites of exocytosis by an actin-independent mechanism. Sec3p is recruited by members of the Rho GTPase family, including the polarity determinant Cdc42p, and the phospholipid PI(4,5)P_2. Although Exo70p also binds to PI(4,5)P_2, the mechanism underlying its recruitment to exocytic sites remains unclear.

Liu and Novick found that the polarity determinant Bem1p—a scaffold protein that binds to both Cdc42p and its activating protein Cdc24p—was required for the actin-independent localization of Exo70p to polarized exocytic sites. Bem1p and Exo70p colocalized throughout the cell cycle and bound directly to one another in vitro. The researchers identified point mutations in Exo70p that specifically disrupted its interaction with Bem1p without affecting the protein's association with other known binding partners. These mutations disrupted Exo70p's actin-independent localization to exocytic sites, which, when combined with mutations in other secretory proteins, inhibited cell growth.

A combination of Bem1p and PI(4,5)P_2 therefore recruits Exo70p to exocytic sites. Senior author Peter Novick now wants to investigate how the small GTPase Rho3p—another protein that binds to Exo70p—contributes to the exocyst subunit's function.

Liu, D., and P. Novick. 2014. *J. Cell Biol.* http://dx.doi.org/10.1083/jcb.201404122.

Chibby's function in the dock



Structured illumination microscopy shows rings of Chibby (green) and CEP164 (red) at the distal appendages of basal bodies in multiciliated tracheal cells.

Burke et al. describe how the coiled-coil protein Chibby promotes the formation of a membranous cap that helps centrioles dock with the plasma membrane during ciliogenesis.

Centrioles, also known as basal bodies, dock with the plasma membrane to nucleate the axonemal microtubules of both primary and motile cilia. Mice lacking the centriolar protein Chibby suffer chronic respiratory infections because their airway epithelial cells fail to form enough motile cilia to clear the respiratory tract of mucus and debris. The cells' centrioles are unable to efficiently dock with the apical plasma membrane during ciliogenesis, but Chibby's precise role in the process is unclear.

Burke et al. used super-resolution and electron microscopy to localize Chibby to the distal appendages of tracheal cell centrioles. Chibby was recruited to these appendages by CEP164, a protein that tethers small, Golgi-derived vesicles to centrioles during ciliogenesis. These vesicles subsequently merge into a large, membranous cap called the ciliary vesicle, which is proposed to fuse with the plasma membrane and anchor the centriole at the cell surface. Centrioles lacking Chibby either failed to recruit small vesicles or were unable to merge them into a larger ciliary membrane structure, explaining their inability to dock with the apical membrane.

Chibby bound to the guanine nucleotide exchange factor Rabin8, enhancing this protein's association with CEP164. Rabin8 activates the small GTPase Rab8 to promote ciliary vesicle assembly. Senior author Ken-Ichi Takemaru now wants to characterize the roles of other Chibby-interacting proteins in ciliary vesicle formation and basal body docking.

Burke, M.C., et al. 2014. *J. Cell Biol.* http://dx.doi.org/10.1083/jcb.201406140.

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