## Organelle morphogenesis, targeting, and distribution

Jeremy G. Carltona,b,\* and Benoît Kornmannc,\*

<sup>a</sup>Division of Cancer Studies, Guy's Hospital, King's College London, London SE1 1UL, UK; <sup>b</sup>The Francis Crick Institute, London NW1 1AT, UK; <sup>c</sup>Institute of Biochemistry, ETH Zürich, Zürich 8093, Switzerland

The Minisymposium on "Organelle Morphogenesis, Targeting, and Distribution" covered mechanisms governing organelle identity, explored organellar contacts and interactions with the cytoskeleton, examined new data suggesting how cells and organelles are intrinsically capable of "measuring," and finally considered how some of these structures are remodeled during cell division.

Alina Guna (Hegde laboratory, Medical Research Council Laboratory of Molecular Biology) presented an elegant in vitro membrane protein insertion assay allowing her to delineate a new pathway of membrane protein insertion into the endoplasmic reticulum (ER) involving the EMC complex. This pathway operates in parallel to the established GET targeting complex for the insertion of low-to-moderately hydrophobic tail-anchored membrane proteins into the ER (Guna et al., 2018).

Chris Obara (Lippincott-Schwartz laboratory, Janelia) presented his recent work on ER dynamics and showed the amazing ability of this organelle to explore the entire cytoplasmic volume. Moreover, by analyzing single molecule dynamics of select ER proteins, Chris showed how motility was slowed at sites of contact with mitochondria, suggesting that these proteins pause to shake hands with binding partners on the other organelles.

Dan Zhang (Temasek Laboratories, National University of Singapore) showed beautifully how the cortical ER in *Schizosaccharomyces pombe* was cleared to allow exocytotic vesicles to fuse with the plasma membrane. Mechanisms underlying this clearance are unknown but raise important questions regarding how ER-plasma membrane contacts must be remodeled to allow vesicular trafficking events to occur (Ng et al., 2018).

DOI:10.1091/mbc.E17-11-0681

Molecular Biology of the Cell Volume 29 Page 692

MBoC is pleased to publish this summary of the Minisymposium on "Organelle Morphogenesis, Targeting, and Distribution," held at the 2017 ASCB EMBO Meeting, Philadelphia, PA, December 5, 2017.

\*Address correspondence to: Jeremy G Carlton (jeremy.carlton@kcl.ac.uk) and Benoît Kornmann (benoit.kornmann@bc.biol.ethz.ch).

© 2018 Carlton and Kornmann. This article is distributed by The American Society for Cell Biology under license from the author(s). Two months after publication it is available to the public under an Attribution–Noncommercial–Share Alike 3.0 Unported Creative Commons License (http://creativecommons.org/licenses/by-nc-sa/3.0).

"ASCB", "The American Society for Cell Biology"," and "Molecular Biology of the Cell" are registered trademarks of The American Society for Cell Biology.

**Ting-Sung Hsieh** (Liou laboratory, UT Southwestern) showed lovely superresolution data of ER–plasma membrane contacts. Intriguingly, these contacts were elongated in shape, forming  $80 \times 120$  nm oblongs on average. Ting-Sung showed how, rather than regulating their formation, the actin cytoskeleton actually acted to maintain the stability and restrict the lateral mobility of these contacts, which has important implications for our understanding of how the cytoskeleton controls plasma membrane microdomains (Hsieh et al., 2017).

Patricia Bassereau (Institut Curie) presented a beautiful analysis of ezrin localization to filopodia, microvilli, and the concave face of trans-epithelial tunnels. Using an elegant combination of biophysics and microscopy, she described how pulled nanotubes from an immobilized giant unilamellar vesicle created a topologically precise negative curvature environment to model filopodial enrichment. Patricia showed how the ezrin-interacting iBAR domain from IRSp53 acted as a sensor of negative curvature within the nanotube and acted to localize ezrin to this surface to couple membrane binding to the cytoskeleton.

Guillaume Cordier (Lakadamyali laboratory, Institute of Photonics Sciences and University of Pennsylvania) presented quantitative superresolution data regarding motor protein organization on organelles. Guillaume found that, rather than acting in isolation, dynein, kinesin-1, and kinesin-2 clustered into mini-teams on lysosomes, with a second level of organization being the observed clustering of these teams themselves. These findings raise some intriguing questions in terms of the motor protein makeup of these teams, mechanisms of team formation, and the effects that teams have on transport dynamics.

Nat Hendel (Marshall laboratory, University of California, San Francisco) looked to explore whether cells are intrinsically capable of "measuring." Cilia growth depends on the kinesin-mediated transport of tubulin to the cilium tip. Kinesin then diffuses back to the cell body. Taking computational approaches, Nat concluded that the slow diffusion along long cilia was limiting the pool of available kinesin at the cilium base, possibly preventing further growth. This provided a satisfying demonstration that autoregulation of a cellular structure can be achieved using diffusion time as a proxy for length measurement (Hendel and Marshall, 2017; Hendel et al., 2018).

**Benoît Kornmann** (ETH Zurich) presented his latest work on describing how physical forces acting on mitochondria (be that collision from *Shigella* nanotorpedoes or compressive force due to growth on vintage records!) acted to induce mitochondrial fission. Fission occurred at these sites of compression due to recruitment of compression-sensitive adaptors that acted to recruit the dynamin-like GTPase Drp1 to effect mitochondrial fission (Helle et al., 2017).

Arpan Rai (Pelkmans lab, University of Zurich) closed the session by describing how the dual-specificity tyrosine phosphorylation regulated kinase, DYRK3, acts as a mitotic dissolvase of membraneless organelles. DYRK3's activity was regulated by the cell cycle as levels of this kinase dropped upon entering G1, presumably allowing the regeneration of these organelles upon mitotic exit. These exciting data add to our understanding of how membrane-less organelles are regulated during cell division.

692 | J. G. Carlton and B. Kornmann Molecular Biology of the Cell

## **REFERENCES**

- Guna A, Volkmar N, Christianson JC, Hegde RS (2018). The ER membrane protein complex is a transmembrane domain insertase. Science 359, 470–473.
- Helle SCJ, Feng Q, Aebersold MJ, Hirt L, Grüter RR, Vahid A, Sirianni A, Mostowy S, Snedeker JG, Šarić A, et al. (2017). Mechanical force induces mitochondrial fission. eLife 6, e30292.
- Hendel NL, Marshall WF (2017). Flagellar length control can be achieved through a simple diffusion-based mechanism. Biophys J 112, 432a.
- Hendel NL, Thomson M, Marshall WF (2018). Diffusion as a ruler: modeling kinesin diffusion as a length sensor for intraflagellar transport. Biophys J 114, 663–674.
- Hsieh T-S, Chen Y-J, Chang C-L, Lee W-R, Liou J (2017). Cortical actin contributes to spatial organization of ER-PM junctions. Mol Biol Cell 28, 3171–3180.
- Ng AYE, Ng AQE, Zhang D (2018). ER-PM contacts restrict exocytic sites for polarized morphogenesis. Curr Biol 28, 146–153.

Volume 29 March 15, 2018 Organelle morphogenesis | **693**