HIGHLIGHTS from *MBoC*

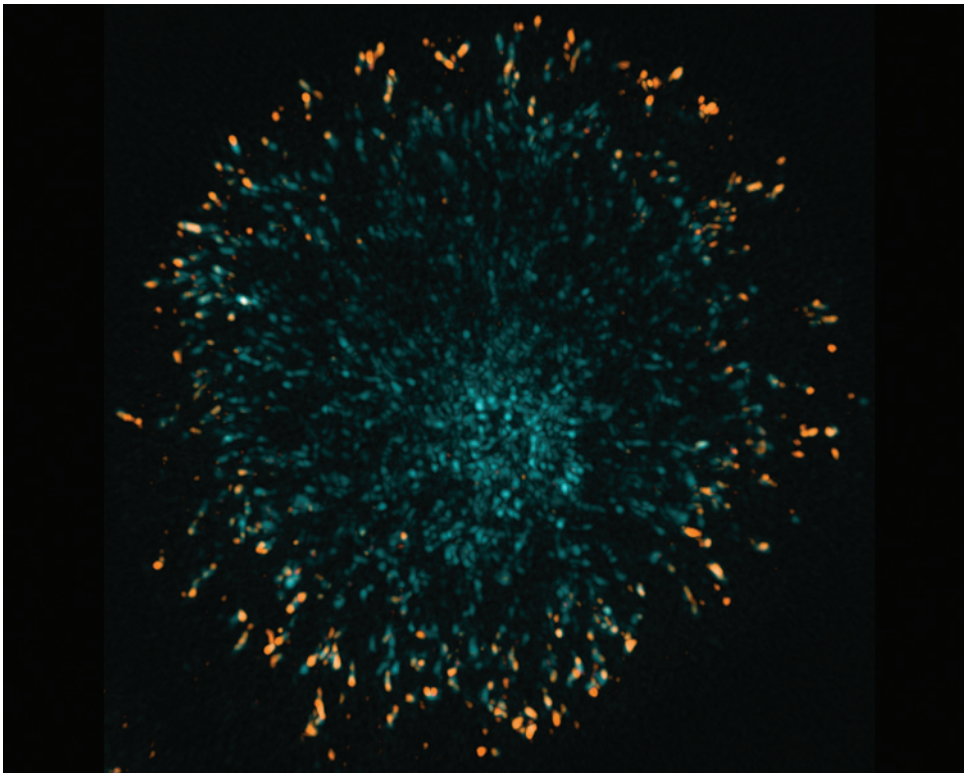
The Editorial Board of *Molecular Biology of the Cell* has highlighted the following articles from the September 2011 issues. From among the many fine articles in the journal, the Board selects for these Highlights articles that are of broad interest and significantly advance knowledge or provide new concepts or approaches that extend our understanding.

Zwint-1 is a novel Aurora B substrate required for the assembly of a dynein-binding platform on kinetochores
J. M. Kasuboski, J. R. Bader, P. S. Vaughan, S. B. F. Tauhata, M. Winding, M. A. Morrissey, M. V. Joyce, W. Boggess, L. Vos, G. K. Chan, E. H. Hinchcliffe, and K. T. Vaughan

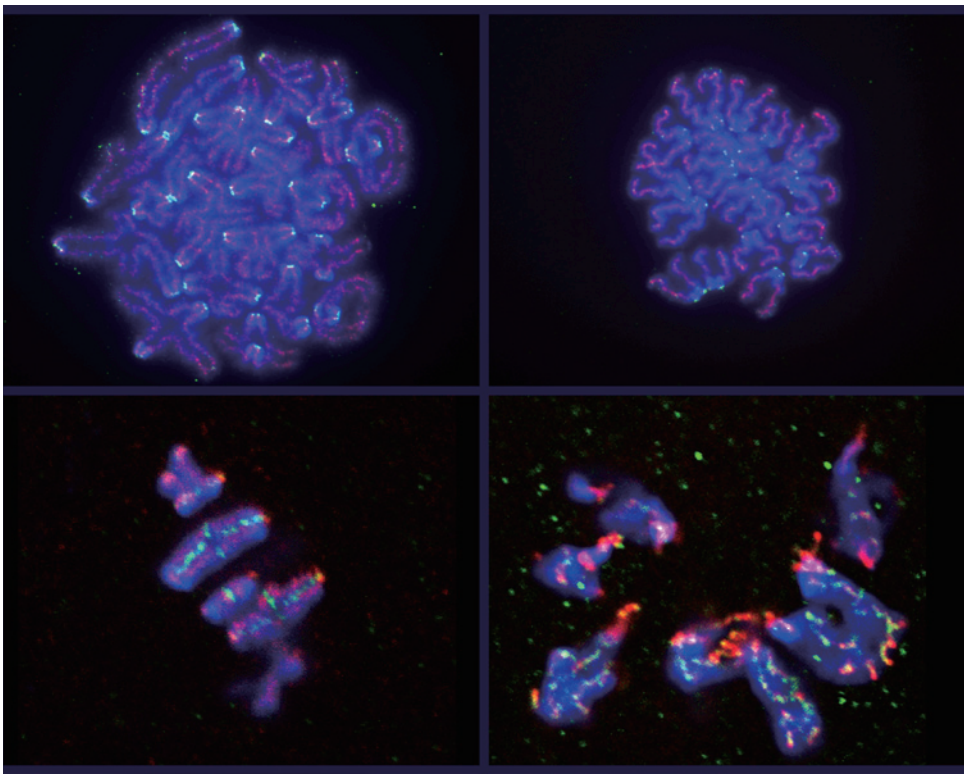
This study identifies zwint-1 as a novel substrate for AurB during mitosis. Phosphorylation is required for outer kinetochore assembly during prometaphase. However, zwint-1 dephosphorylation is required at metaphase for checkpoint silencing.
Mol. Biol. Cell 22 (18), 3318–3330

Glucose depletion inhibits translation initiation via eIF4A loss and subsequent 48S preinitiation complex accumulation, while the pentose phosphate pathway is coordinately up-regulated
L. M. Castelli, J. Lui, S. G. Campbell, W. Rowe, L. A. H. Zeef, L. E. A. Holmes, N. P. Hoyle, J. Bone, J. N. Selley, P. F. G. Sims, and M. P. Ashe

The mechanism and consequences of the translational inhibition caused by glucose depletion in yeast are characterized. eIF4A is lost from the preinitiation complex, and the pentose phosphate pathway is translationally up-regulated, allowing an efficient transition to the new conditions.
Mol. Biol. Cell 22 (18), 3379–3393



A HeLa cell treated with the kinesin-5 inhibitor monastrol to create asters that are enriched with microtubules that extend past the chromosomes toward the cortex. Whereas the plus-end microtubule binding protein EB1 (cyan) is found on all microtubule plus ends, Kif18B (orange) is highly enriched on microtubules near the cortex. See Mol. Biol. Cell *22 (17), 3070–3080. (Image:* Amber Yount, James Powers, *and* Claire Walczak, *Indiana University)*



Condensin II (red) and centromere proteins (green) are labeled in bivalent chromosomes (upper left panel) and univalent chromosomes (upper right panel) prepared from metaphase I and metaphase II mouse oocytes, respectively. In metaphase I oocytes (lower left panel) and metaphase I-like oocytes that had been injected with antibody against structural maintenance of chromosomes core subunit 2 (SMC2; lower right panel), SMC2 (condensin), REC8 (cohesin), and DNA appear in red, green, and blue, respectively. Condensin I localizes around centromeric regions, whereas condensin II is concentrated onto chromatid axes. See Mol. Biol. Cell *22 (18) 3465–3477. (Image:* Jibak Lee, *RIKEN Advanced Science Institute [currently at Kobe University])*

Kinesin molecular motor Eg5 functions during polypeptide synthesis
K. M. Bartoli, J. Jakovljevic, J. L. Woolford, Jr., and W. S. Saunders

The microtubule motor Eg5 is well known for its functions during mitosis. It is shown that during interphase, Eg5 associates with ribosomes and is required for efficient protein synthesis.
Mol. Biol. Cell 22 (18), 3420–3430

The fission yeast pleckstrin homology domain protein Spo7 is essential for initiation of forespore membrane assembly and spore morphogenesis
M. Nakamura-Kubo, A. Hirata, C. Shimoda, and T. Nakamura

Assembly of the forespore membrane (FSM) initiates at the spindle pole body (SPB), and the leading edge of the FSM is a critical factor in the proper shaping of the FSM. We report a novel SPB component, Spo7. Our study suggests that Spo7 coordinates formation of the leading edge and initiation of FSM assembly, thereby accomplishing accurate FSM formation.
Mol. Biol. Cell 22 (18), 3442–3455

Membrane aberrancy and unfolded proteins activate the endoplasmic reticulum stress sensor Ire1 in different ways
T. Promlek, Y. Ishiwata-Kimata, M. Shido, M. Sakuramoto, K. Kohno, and Y. Kimata

In contrast to the classical model, in which unfolded proteins accumulated in the endoplasmic reticulum trigger the unfolded-protein response (UPR), we show that membrane aberrancy also evokes this protective cellular event. This finding may explain UPR activation under various physiological conditions.
Mol. Biol. Cell 22 (18), 3520–3532 ■