HIGHLIGHTS from *MBoC*

The Editorial Board of *Molecular Biology of the Cell* has highlighted the following articles from the April 1 and April 15, 2010, 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.

Evasion of Endoplasmic Reticulum Surveillance Makes Wsc1p an Obligate Substrate of Golgi Quality Control

Songyu Wang and Davis T.W. Ng

The lack of ERAD determinants and a powerful ER exit signal mediate Wsc1p's evasion of ER folding surveillance systems. As a consequence, Wsc1p traffics to Golgi apparatus whether folded or not, making it an obligate substrate of Golgi quality control.

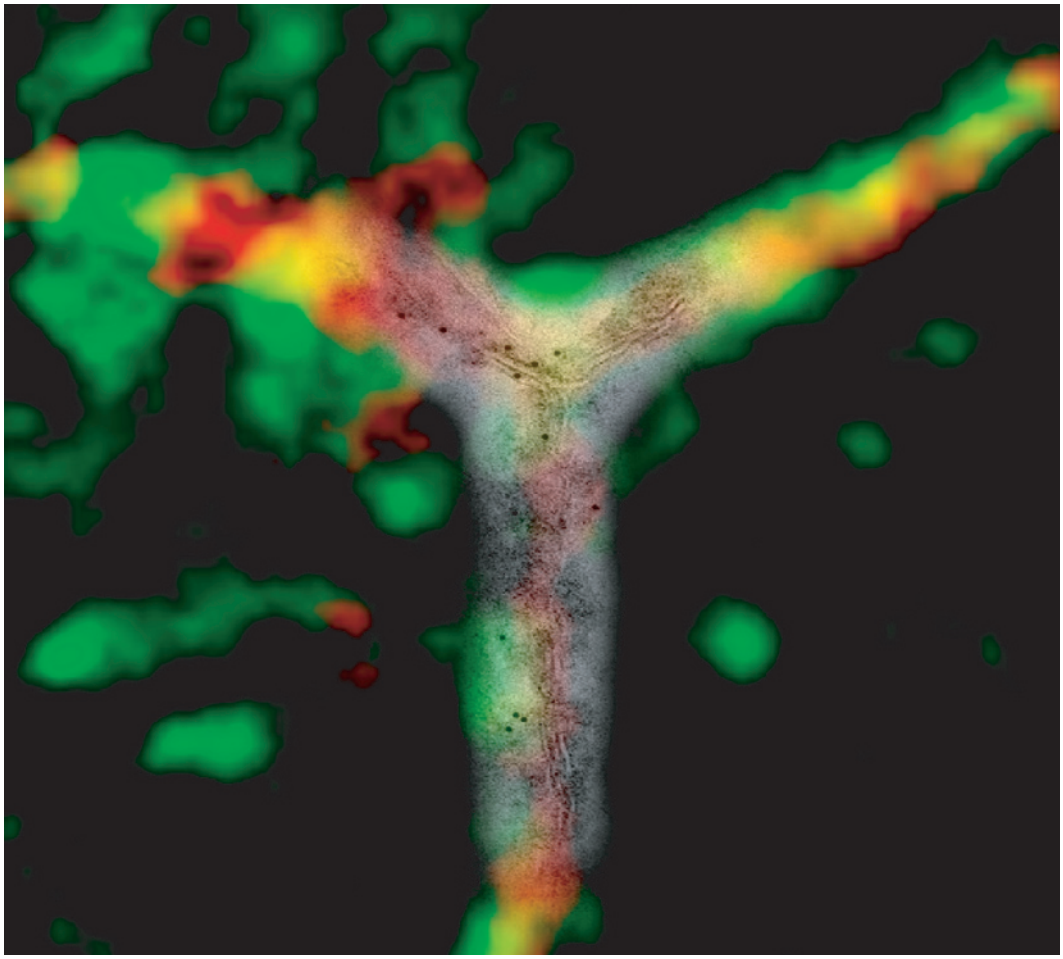
Mol. Biol. Cell 21 (7), 1153–1165

Tight Junction–associated MARVEL Proteins MarvelD3, Tricellulin, and Occludin Have Distinct but Overlapping Functions

David R. Raleigh, Amanda M. Marchiando, Yong Zhang, Le Shen, Hiroyuki Sasaki, Yingmin Wang, Manyuan Long, and Jerrold R. Turner

This study identifies and characterizes marvelD3, a novel tight junction protein that contains a conserved MARVEL domain. Analyses using phylogenetic, expression profiling, microscopic, and functional approaches show that marvelD3, occludin, and tricellulin are related and have distinct but overlapping functions at the tight junction.

Mol. Biol. Cell 21 (7), 1200–1213



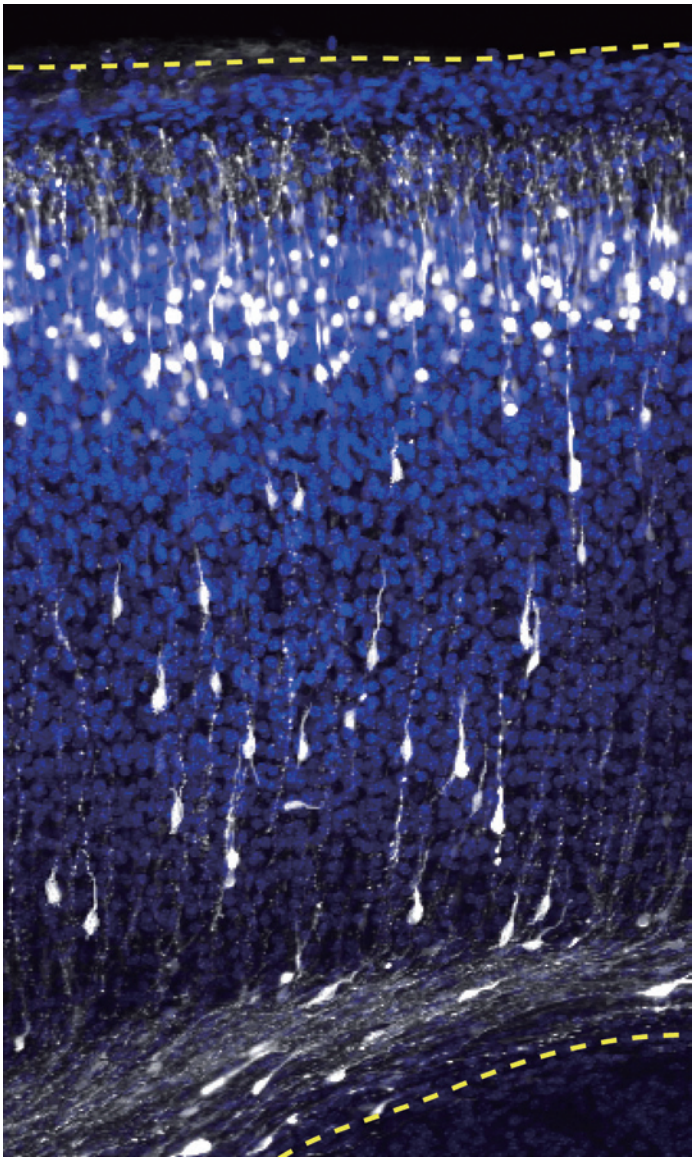
In a merged immunofluorescence and transmission electron microscopy image of the tight junction between mouse jejunal enterocytes, actin is shown in red and the novel tight junction protein MarvelD3 is shown in green; MarvelD3 is also labeled with gold-conjugated antibodies. (Image: David R. Raleigh, Amanda M. Marchiando, Hiroyuki Sasaki, *and* Jerrold R. Turner*)*

Initial Polarized Bud Growth by Endocytic Recycling in the Absence of Actin Cable–dependent Vesicle Transport in Yeast
Takaharu Yamamoto, Junko Mochida, Jun Kadota, Miyoko Takeda, Erfei Bi, and Kazuma Tanaka

Budding yeast mutants in assembly of actin cables, which are thought to be the only actin structures essential for budding, still could form a small bud. Mutations in actin patch endocytic machineries/endocytic recycling factors inhibited this budding, suggesting a mechanism that promotes polarized growth by local recycling of endocytic vesicles.
Mol. Biol. Cell 21 (7), 1237–1252

LRPPRC and SLIRP Interact in a Ribonucleoprotein Complex That Regulates Posttranscriptional Gene Expression in Mitochondria
Florin Sasarman, Catherine Brunel-Guitton, Hana Antonicka, Timothy Wai, Eric A. Shoubridge; LSFC Consortium

The PPR family protein LRPPRC is implicated in the French Canadian variant of Leigh syndrome, a fatal neurodegenerative disease. LRPPRC functions in posttranscriptional mitochondrial gene expression as part of an RNP complex with SLIRP, a stem-loop RNA-binding protein, to regulate the stability and handling of mature mRNAs.
Mol. Biol. Cell 21 (8), 1315–1323



Many Septin14–deficient mouse neuronal cells show abnormal positioning during corticogenesis. (Image: Tomoyasu Shinoda*)*

Septin 14 Is Involved in Cortical Neuronal Migration via Interaction with Septin 4
Tomoyasu Shinoda, Hidenori Ito, Kaori Sudo, Ikuko Iwamoto, Rika Morishita, and Koh-ichi Nagata

Septins are a family of conserved GTP/GDP-binding proteins implicated in a variety of cellular functions. We found that knockdown of Septin 14 or Septin 4 resulted in inhibition of cortical neuronal migration and defective leading process formation. These results suggest a novel function of septin in cortical development.
Mol. Biol. Cell 21 (8), 1324–1334 ■

MBoC Selected Articles

A volume of selected articles from *Molecular Biology of the Cell* (*MBoC*) is available as a PDF file at www.ascb.org/newsfiles/MBoC_Selected_Articles.pdf. The 32-page collection was distributed at the 2009 ASCB Annual Meeting and includes an editorial by David Drubin, a retrospective by David Botstein, essays by 2009 ASCB Award winners, and the 2009 *MBoC* Paper of the Year. ■