Contents

**PERSPECTIVE**

Tricellular junctions: how to build junctions at the TRICkiest points of epithelial cells  
Tomohito Higashi and Ann L. Miller  
2023–2034

**BRIEF REPORT**

Spindle assembly checkpoint signaling and sister chromatid cohesion are disrupted by HPV E6-mediated transformation  
Hazheen K. Shirnekhi, Erin P. Kelley, Jennifer G. DeLuca, and Jacob A. Herman  
2035–2041

HPV-transforming protein E6 weakens both SAC activity and sister chromatid cohesion. These processes largely function normally and result in chromosome segregation errors only when mitosis is delayed by the presence of one to four unaligned, pole-associated chromosomes. This mechanism of aneuploidy is not observed in response to oncogenic signaling.

**ARTICLES**

**Biosynthesis and Biodegradation**

Temporal regulation of epithelium formation mediated by FoxA, MKLP1, MgcRacGAP, and PAR-6  
Stephen E. Von Stetina, Jennifer Liang, Georgios Marnellos, and Susan E. Mango  
2042–2065

During embryo morphogenesis, minor epithelia are generated after, and then form bridges between, major epithelia (e.g., epidermis and gut). In *Caenorhabditis elegans*, this delay is regulated by four proteins that control production and localization of polarity proteins: the pioneer factor PHA-4/FoxA, kinesin ZEN-4/MKLP1, its partner CYK-4/MgcRacGAP, and PAR-6.

Substrate binding by the yeast Hsp110 nucleotide exchange factor and molecular chaperone Sse1 is not obligate for its biological activities  
Veronica M. Garcia, Nadinath B. Nillegoda, Bernd Bukau, and Kevin A. Morano  
2066–2075

Hsp110 functions as both a nucleotide exchange factor and a protein molecular chaperone. A novel yeast Hsp110 mutant reveals that the ability to bind substrate proteins is not required for Hsp110 to support proteostasis under normal conditions but may enhance growth under chronic thermal stress.

**A Highlights from MBoC Selection**

Transmembrane helix hydrophobicity is an energetic barrier during the retrotranslocation of integral membrane ERAD substrates  
2076–2090

Cdc48/p97 provides the energy to retrotranslocate integral membrane ERAD substrates. A series of ERAD substrates with increasingly hydrophobic transmembrane helices is used to show that retrotranslocation efficiency inversely correlates with hydrophobicity.

**Cell Biology of Disease**

Leishmania donovani restricts mitochondrial dynamics to enhance miRNP stability and target RNA repression in host macrophages  
Yogaditya Chakrabarty and Suvendra N. Bhattacharyya  
2091–2105

The miRNA complex with Argonaute protein recognizes and down-regulates target mRNA. The role of interorganellar interactions in controlling miRNA activity in mammalian macrophages infected with *Leishmania* is explored.
Tumor susceptibility gene 101 regulates predisposition to apoptosis via ESCRT machinery accessory proteins

Zenia Kaul and Oishee Chakrabarti
2106–2122
The ESCRT-I protein TSG101 can mitigate endoplasmic reticulum stress–mediated apoptosis involving caspase 4/12 caused by MGRN1 depletion, probably contributing to neurodegeneration. TSG101 associates with ALIX to prevent predisposition to apoptosis, whereas ALIX–ALG-2 interaction favors a cell death phenotype.

Cell Cycle

NEK7 is required for G1 progression and procentriole formation

Akshari Gupta, Yuki Tsuchiya, Midori Ohta, Gen Shiratsuchi, and Daiju Kitagawa
2123–2134
As cells exit mitosis, the decision to commit to the next cell cycle is made during G1. Not only DNA replication, but also centriole duplication is initiated as cells enter the S-phase. The kinase NEK7 is required for the timely regulation of G1 progression, S-phase entry, and procentriole formation.

Cell Physiology

Spatial analysis of Cdc42 activity reveals a role for plasma membrane–associated Cdc42 in centrosome regulation

Kari A. Herrington, Andrew L. Trinh, Carolyn Dang, Ellen O'Shaughnessy, Klaus M. Hahn, Enrico Gratton, Michelle A. Digman, and Christine Sütterlin
2135–2145
A Cdc42 biosensor and the phasor approach to FLIM-FRET are used to determine Cdc42 activity at specific subcellular locations, revealing new regulatory principles and functions of this small GTPase.

Membrane Trafficking

SEC23B is required for pancreatic acinar cell function in adult mice

2146–2154
Inactivation of Sec23b exclusively in the pancreatic acinar cells of adult mice results in loss of pancreatic mass, with evidence of cell loss, degeneration of exocrine cells (with smaller-than-normal zymogen granules and ER dilation), ER stress, and increased pancreatic cell apoptosis.