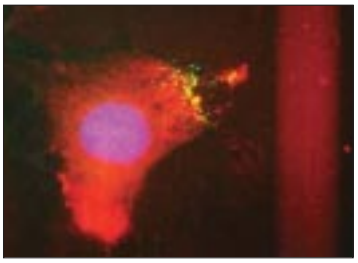
EDITORS' CHOICE

edited by Stella Hurtley

CELL BIOLOGY

A Quick Fix



Resealed fibroblast (red) binds antibodies against a lysosomal glycoprotein (green).

For an animal cell, rupture of the plasma membrane would be thought to imply certain death. However, in some instances repair is possible. Indeed it appears that mechanisms for rapid repair exist in most cell types.

Reddy *et al.* examined the process of plasma membrane repair in tissue culture cells wounded by scraping. Surprisingly, the repair process involved fusion of the cell's degradative organelles, the lysosomes, with the cell surface. Upon wounding, lysosomes fused with the plasma membrane, rapidly sealing the cell in a Ca^{2+}-triggered process. The mechanism of fusion involved the lysosomal form of synaptotagmin, SytVII, and could be inhibited by expressing the cytoplasmic C2A domain of Syt VII involved in Ca^{2+} sensing. In primary skin fibroblasts wounded by the contraction of their supporting collagen matrix, lysosomal exocytosis was also responsible for cell resealing. Thus, in addition to degrading ingested proteins, lysosomes also play a key role in plasma membrane repair by acting as Ca^{2+}-regulated secretory vesicles. — SMH

Cell **106**, 157 (2001).

MOLECULAR BIOLOGY

Remodeling p53

About half of all human cancers contain mutations in the *p53* tumor suppressor gene. In response to certain forms of cellular stress, the p53 protein induces cell cycle arrest by sequence-specific DNA binding and transcriptional activation of key target genes. Previous studies used purified DNA consensus sequences to produce a model in which p53 acquires DNA binding activity only after its carboxyl-terminal region is modified.

Using a chromatin-based assay that may better mimic the substrates p53 encounters in the cell nucleus, Espinosa and Emerson arrive at a very different model of how p53 regulates transcription. Studying the chromatin-assembled *p21/WAF1* promoter, they find that unmodified p53 does bind DNA/chromatin and requires the same carboxyl-terminal region that was previously thought to repress DNA binding. The chromatin-bound p53 recruits a histone acetyltransferase (p300) to the promoter, which then acetylates the p53-bound nucleosomes, perhaps facilitating interaction with other components of the transcriptional machinery. Whether p53 uses the same or different mechanisms to regulate expression of its many other target genes is an important question that remains to be investigated. — PAK

Mol. Cell **8**, 57 (2001).

GEOPHYSICS

Not Giving Physics the Slip

Great earthquakes (magnitude > 8) rupture over large distances in such a way that the amount of horizontal displacement (rupture length) between the two sides of the fault can be greater than the amount of vertical displacement (rupture width). Essentially, the rupture width cannot go any deeper than the base of the brittle crust, but the rupture length seems to be unlimited. This observation seems to defy physical principles of fracture mechanics observed in the laboratory.

Now, a study by Shaw and Scholz may resolve this apparent inconsistency. They modeled a great earthquake rupture in three dimensions with two layers of different frictional strength. Friction helps to determine how far a fault will slip; when friction decreases with increasing slip or slip rate, a fault will stick and slip to produce an earthquake, but if friction increases with increasing slip or slip rate, a fault will simply creep along. In simulations, the rupture length is related to a kinetic effect; slip pulses take a long time to build to their maximum intensity and then take a long time to dissipate. Thus, for a great earthquake, the slip pulses can move over long distances, and the maximum slip can occur far from the epicenter. Thus there is nothing fundamentally different about the physics of a small-to-large earthquake as compared with great earthquakes. — LR

Geophys. Res. Lett. **28**, 2991 (2001).

MICROBIOLOGY

Instant Death

Bacteriophages produce small proteins called holins, which, at a defined point in the infection cycle, punch holes in bacterial cell membranes. After the accumulation of a critical number of holin molecules in the cell membrane, oligomerization is triggered, causing cell lysis. Bacteria can be tethered to a substrate by antibodies directed against their flagella, and their cell bodies will then rotate. The rate of this rotation is proportional to the proton motif force (pmf) of the bacterial cell membrane and is a measure of membrane integrity.

Gruendling *et al.* saw that in phage-infected bacteria, rotation stops abruptly a few seconds before catastrophic cell lysis. Apparently, the pmf keeps the holins apart until the threshold concentration is reached, when they instantaneously clump to form a weak patch in the membrane. This collapses the pmf and causes membrane rupture. However, the holin's target in the membrane remains unknown, and the mechanism of action of these hugely diverse and ancient clock-like proteins remains elusive. — CA

Proc. Natl. Acad. Sci. U.S.A. **98**, 9348 (2001).

ASTROPHYSICS

Early Metal Production in the Universe

To understand the chemical evolution of the universe, astronomers use spectroscopy and photometry to determine the abundances of heavier elements (metals). Metals are created by stellar nucleosynthesis and supernovae, and their abundances can be used to estimate the amount and rate of star formation. Co, for example, is overabundant relative to Fe in metal-poor systems, such as Galactic bulge and thick-disk stars, that may have formed early in the universe. This overabundance may be produced by the fastest evolving stars.

Given the implications about star formation rates suggested by the overabundance of Co, Ellison *et al.* measured Co in damped Lyman alpha systems (DLAs). A DLA is a region of high hydrogen density that lies between a very distant bright quasar and the observer. In these very distant regions, Co is overabundant relative to Fe. Thus, this Co signature suggests that star forma-

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SOURCE: (IMAGE) NORMA ANDREWS, YALE