

IN BRIEF

➤ NUCLEAR TRANSPORT**Nuclear import of an intact preassembled proteasome particle**

Savulescu, A. F. *et al. Mol. Biol. Cell* 2 Feb 2011 (doi:10.1091/mbc.E10-07-0595)

Proteasomes localize to many cellular compartments, but the details of this dynamic distribution, and the factors that regulate it, were largely unknown. This study investigated the nuclear targeting of the proteasome using *Xenopus laevis* egg extracts. In addition to the 20S core particle of the proteasome and the 26S holoenzyme (made up of 20S and two 19S), they report the existence of a complex (which they term 20S+) that is made up of 20S and at least two 19S regulatory complex components (rpn1 and rpn2). In contrast to 20S and 26S, which accumulated in the nuclear periphery, 20S+ was imported into the nucleus through the nuclear pore complex in a Ran-independent manner. The authors propose that the 20S+ complex facilitates the nuclear accumulation of proteasomal subunits, which may then associate with independently imported 19S components to form a nuclear pool of 26S holoenzyme.

➤ POST-TRANSLATIONAL MODIFICATION**Role of the ubiquitin-like protein Urm1 as a noncanonical lysine-directed protein modifier**

Van der Veen, A. G. *et al. Proc. Natl Acad. Sci. USA* **108**, 1763–1770 (2011)

Although ubiquitin has an established role in post-translational control, it is less clear whether this holds true for other members of the ubiquitin protein family. There have been hints that ubiquitin-related modifier 1 (URM1), which donates a sulphur group during tRNA thiolation, may also modify other proteins. Van der Veen *et al.* now show that URM1 is indeed conjugated to several proteins, including components of the URM1 pathway itself, in response to oxidative stress in mammalian cells and *Saccharomyces cerevisiae*, and that this process shows several parallels with ubiquitylation. URM1 targets Lys residues of substrate proteins, and this requires a thioester intermediate and formation of a covalent peptide bond between URM1 and the target protein. Thus, the authors speculate that “during conditions of oxidative stress, the role of Urm1 as a posttranslational modifier may become increasingly important.”

➤ MEMBRANE TRAFFICKING**Cells respond to mechanical stress by rapid disassembly of caveolae**

Sinha, B. *et al. Cell* **144**, 402–413 (2011)

Caveolae have been implicated in many processes, such as endocytosis, cell signalling and lipid metabolism, but these cup-shaped membrane invaginations now seem to have another function: buffering sudden and acute surges in membrane tension in response to mechanical stress. Sinha *et al.* show that acute mechanical stress (induced by hypo-osmotic shock or membrane stretching) causes caveolae to undergo rapid flattening, thereby releasing free caveolins and disappearing from the cell surface. This flattening is passive, requiring neither actin nor ATP, and functions to buffer the increase in membrane tension. By contrast, the reassembly of new caveolae is slower and requires ATP and actin. Interestingly, myotubes from patients with a hereditary form of muscular dystrophy, which show markedly decreased cell-surface levels of caveolin 3, are more susceptible to membrane rupture when exposed to hypo-osmotic shock.