IN BRIEF

AUTOPHAGY

Eating up damaged lysosomes

Autophagosomes engulf cytosol or damaged organelles and deliver them to lysosomes to be degraded and released as nutrients. Hung *et al.* show here that damaged lysosomes themselves are degraded in this way. They utilized a photosensitizer that is targeted to lysosomes by endocytosis; its light-mediated activation triggers the formation of reactive oxygen species, and thus lysosomal membrane permeabilization (LMP), with spatio-temporal precision. Imaging experiments following LMP in HeLa cells revealed that tagged versions of ubiquitin, the selective autophagy adaptor p62 and the late autophagosome marker LC3 accumulated in the illuminated (and thus damaged) region of lysosomes. The authors also showed that the autophagic structures that are triggered by LMP become mature autolysosomes. Thus, they propose that damaged lysosomes are removed by a type of organelle-specific autophagy that they term lysophagy.

ORIGINAL RESEARCH PAPER Hung, Y.-H. *et al.* Spatiotemporally controlled induction of autophagy-mediated lysosome turnover. *Nature Commun.* http://dx.doi.org/10.1038/ncomms3111 (2013)

PROTEIN METABOLISM

Cytosolic aggregates impair nuclear degradation

Polyglutamine (polyQ)-expanded proteins form cytoplasmic aggregates that interfere with cellular protein quality control systems. Park *et al.* studied how polyQ proteins disrupt the clearance of misfolded proteins by the ubiquitin–proteasome system. They found that the degradation of a misfolded mutant version of cytosolic carboxypeptidase fused to GFP (CG*), which is normally degraded in the nucleus, was inhibited by co-expression of polyQ proteins, and that CG* accumulated in the cytoplasm. A quantitative interactome analysis revealed that polyQ aggregates sequester the yeast Hsp40 chaperone Sis1p (and one of its mammalian homologues, DNAJB1), and that this causes impaired CG* degradation. Importantly, the authors show that the function of Sis1p is to transport CG* from the cytoplasm to the nucleus for degradation.

ORIGINAL RESEARCH PAPER Park, S.-H. *et al.* PolyQ proteins interfere with nuclear degradation of cytosolic proteins by sequestering the Sis1p chaperone. *Cell* **154**, 134–145 (2013)

CHROMATIN

Lysosomes help process chromatin in senescence

Cellular senescence involves chromatin remodelling, and Ivanov *et al.* show here that an autophagy–lysosome pathway contributes to this. They observed more cytoplasmic chromatin fragments (CCFs) in senescent cells than in proliferating cells; these were positive for the DNA damage marker γH2A.X and the heterochromatic histone mark H3K27me3 (trimethylated Lys27 of histone H3). Using time-lapse imaging they observed that CCFs associated with these marks entered the cytoplasm by 'blebbing' off the cell nucleus and that the integrity of the nuclear envelope was compromised in senescent cells. The authors also found that CCFs partially colocalize with autophagy markers and that the level of a lysosomal protease (cathepsin L) that cleaves histone H3 to produce histone H3cs1 was increased in senescent cells, along with H3cs1 levels. The histone content of senescent cells was also reduced in a lysosome-dependent manner. The authors conclude that "autophagy and lysosomes contribute to the proteolytic processing of histones in senescence."

ORIGINAL RESEARCH PAPER Ivanov, A. et al. Lysosome-mediated processing of chromatin in senescence. *J. Cell Biol.* http://dx.doi.org/10.1083/jcb.201212110 (2013)

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