IN BRIEF

ORGANELLE DYNAMICS

Cell starvation through proteasome block

Proteasome inhibition is lethal, and there is strong interest in the underlying mechanisms of this. In addition to degrading proteins, the proteasome has a role in amino acid recycling, so Suraweera *et al.* asked whether this might cause lethality following proteasome inhibition. In yeast, mammalian cells and *Drosophila melanogaster*, amino acid supplementation following proteasome inhibition rescued viability; however, it did not rescue proteasome function, indicating that amino acid starvation is the underlying cause of lethality. Moreover, amino acid scarcity resulting from proteasome inhibition signals the induction of autophagy, which attempts to replenish the amino acid pool and to restore homeostasis. The authors propose that lethality following proteasome inhibition may be due to the accumulation of proteasome substrates and the sequestration of amino acids that would otherwise be recycled.

ORIGINAL RESEARCH PAPER Suraweera, A. *et al.* Failure of amino acid homeostasis causes cell death following proteasome inhibition. *Mol. Cell* 6 Sep 2012 (doi:10.1016/j.molcel.2012.08.003)

CELL SIGNALLING

Mediating hormonal crosstalk

Small-molecule hormones, including brassinosteroids (BRs) and gibberellins (GAs), influence plant growth and development. Although previous studies suggested that crosstalk occurs between the BR and GA signalling pathways, the mechanisms by which GAs and BRs coordinate to control growth were unclear. Here, Li *et al.* report that in *Arabidopsis thaliana* BZR1 (a transcription factor that is activated by BR signalling) interacts with REPRESSOR OF GA1-3 (RGA), a member of the DELLA family of transcriptional regulators, which inhibit the GA signalling pathway. Overexpression of RGA lacking the DELLA domain resulted in a reduction in BZR1, suggesting that DELLAs affect the levels of BZR1. Furthermore, reporter gene assays indicated that BZR1 and RGA antagonize each other's transcriptional activity. Notably, this paper highlights the role of DELLAs as mediators of signalling crosstalk between BRs and GAs in controlling cell elongation and growth.

ORIGINAL RESEARCH PAPER Li, Q.-F. *et al.* An interaction between BZR1 and DELLAs mediates direct signaling crosstalk between brassinosteroids and gibberellins in *Arabidopsis*. *Sci. Signal.* 2 Oct 2012 (doi:10.1126/scisignal.2002908)

MEMBRANE DYNAMICS

ER connections boost phagocytosis

Phagocytosis is important for the uptake of nutrients and for the removal of pathogens and cell debris by the immune system. High Ca^{2+} levels at the phagosome boost phagocytosis, but the mechanisms driving these Ca^{2+} increases are unclear. Here Nunes *et al.* show that stromal interaction molecule 1 (STIM1), an endoplasmic reticulum (ER) Ca^{2+} sensor that interacts with and activates plasma membrane Ca^{2+} channels, promotes the formation of tight phagosome–ER junctions in mouse fibroblasts and neutrophils, and that this generates localized Ca^{2+} rises. Furthermore, they find that signalling-deficient STIM1 can still promote the recruitment of the ER to the phagosome but cannot induce Ca^{2+} hotspots and phagocytosis. This suggests that STIM1 signals the opening of phagosomal Ca^{2+} channels and also sheds light on the functional significance of ER remodelling during phagocytosis.

ORIGINAL RESEARCH PAPER Nunes, P. *et al.* STIM1 juxtaposes ER to phagosomes, generating Ca^{2+} hotspots that boost phagocytosis. *Curr. Biol.* 4 Oct 2012 (doi:10.1016/j.cub.2012.08.049)