IN BRIEF

MEMBRANE TRAFFICKING

The COG complex interacts directly with syntaxin 6 and positively regulates endosome-to-TGN retrograde transport

Laufman, O., Hong, W. & Lev, S. *J. Cell Biol.* **194**, 459–472 (2011)

The conserved oligomeric Golgi (COG) membrane-tethering complex is essential for the transport of vesicles between different stacks of the *trans*-Golgi network (TGN), and there have been some indications that it also affects endosome to TGN trafficking during vesicle recycling. Lev and colleagues now demonstrate that the COG complex has a direct role in this retrograde transport, through an interaction between the COG6 subunit and the target membrane SNARE syntaxin 6 (STX6) that promotes endosome–TGN vesicle fusion. When COG6 is lost, transport from endosomes to the TGN is disrupted and STX6 is degraded. The authors propose that, by coordinating endosome–TGN and intra-Golgi transport, the COG tethering complex may help to ensure Golgi maintenance.

UBIQUITYLATION

Alternative ubiquitin activation/conjugation cascades interact with N-end rule ubiquitin ligases to control degradation of RGS proteins

Lee, P. C. W. *et al. Mol. Cell* **43**, 392–405 (2011)

UBA6 is an alternative E1 ubiquitin-activating enzyme to the canonical E1, UBA1, and it interacts with the E2 USE1. However, the E3 ligase that they work with was unknown. Lee *et al.* found that USE1 interacts with the UBR family of E3s, which function in the N-end rule pathway. Interestingly, both USE1 and UBE2 (the E2 for UBA1) interacted with the UBR2 RING domain, and this mediated Lys48-linked ubiquitylation, indicating a role in protein turnover. Moreover, the best-characterized N-end rule substrates, regulator of G protein signalling (RGS) proteins, were found to be targeted by both UBA1–UBE2 and UBA6–USE1. However, the two pathways are not fully redundant, as they act on distinct pools of RGS proteins. So, UBA6–USE1 and the canonical UBA1–UBE2 pathway interact with N-end rule E3 ligases and can work in parallel to promote N-end rule substrate degradation.

SMALL RNAS

piRNA production requires heterochromatin formation in *Drosophila*

Rangan, P. *et al. Curr. Biol.* 4 Aug 2011 (doi:10.1016/j.cub.2011.06.057)

PIWI-interacting RNAs (piRNAs) are transcribed from heterochromatic clusters and protect the genome by silencing transposable elements, which is important in germline development. The processes controlling piRNA transcription are not well understood. To investigate whether heterochromatin formation is involved in transposable element regulation during oogenesis, Rangan *et al.* studied the distribution of the heterochromatic mark histone H3 Lys9 trimethylation (H3K9me3) during germline stem cell (GSC) differentiation. Total H3K9me3 increased during GSC differentiation and was enriched at piRNA clusters. Mutation of SETDB1, which catalyses H3K9me3, caused a reduction in piRNA levels and a decrease in piRNA precursor transcription, suggesting that SETDB1 is required for piRNA transcription, rather than acting elsewhere in the piRNA pathway. Furthermore, loss of SETDB1 caused increased transposable element expression and a block in GSC differentiation. Thus, heterochromatin formation mediated by SETDB1 may protect the germline by activating piRNA transcription.