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IN BRIEF

MICROBIOLOGY

The positioning of cytoplasmic protein clusters in bacteria.

Thompson, S. R. *et al. Proc. Natl Acad. Sci. USA* **103**, 8209–8214 (2006)

A subset of *Rhodobacter sphaeroides* chemotaxis proteins is known to form discrete clusters in the bacterial cytoplasm. This paper shows that the precise positioning of the clusters is determined by the number of clusters in the cell. It also shows that PpfA, which shows homology to DNA-partitioning factors, is required for the correct positioning of the chemotaxis-protein clusters. The authors speculate that bacteria might have a mechanism that ensures the proper segregation of cytoplasmic protein clusters following cell division.

PHAGOCYTOSIS

C. elegans dynamin mediates the signaling of phagocytic receptor CED-1 for the engulfment and degradation of apoptotic cells.

Yu, X. *et al. Dev. Cell* **10**, 743–757 (2006)

Yu *et al.* identified 14 mutants of the *Caenorhabditis elegans* dynamin gene *dyn-1* that are defective in the removal of apoptotic cells. Epistasis studies established that DYN-1 functions in the phagocytic signalling pathway that also contains the phagocytic receptor CED-1. Endosomes fail to cluster at the site of engulfment in *dyn-1* mutants and abnormal vesicles accumulate, which led the authors to propose that DYN-1 might recruit vesicles to the site of engulfment, and that DYN-1 promotes engulfment through a function other than endocytosis.

TUMOUR SUPPRESSOR

A short mitochondrial form of p19^{ARF} induces autophagy and caspase-independent cell death.

Reef, S. *et al. Mol. Cell* **22**, 463–475 (2006)

The ARF tumour suppressor protein has both p53-dependent and p53-independent functions. Reef *et al.* now describe a short mitochondrial isoform of ARF (smARF) in mouse and in human and shed light on the p53-independent role of ARF. This isoform is normally present at low levels but it is upregulated in response to viral and cellular oncogenes. Overexpression of smARF causes p53- and BCL2-family-independent depletion of the mitochondrial membrane potential, and induces autophagy and caspase-independent cell death. The authors speculate that this mitochondrial pathway might provide a back-up mechanism in circumstances when p53- and/or caspase-dependent cell-death pathways are non-functional.

AGEING

Elimination of damaged proteins during differentiation of embryonic stem cells.

Hernebring, M. *et al. Proc. Natl Acad. Sci. USA* **103**, 7700–7705 (2006)

Cellular proteins become increasingly damaged with age, for example by protein carbonylation and by the formation of advanced glycation end products (AGEs). The authors found that undifferentiated mouse embryonic stem (ES) cells contain high levels of carbonyls and AGEs. However, both types of damage were eliminated during ES-cell differentiation, which coincided with a boost in 20S proteasomal activity. These findings indicate that a 'rejuvenation' process occurs during early embryonic development that involves the elimination of damaged proteins.