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IN BRIEF

CYTOSKELETON

Asymmetric CLASP-dependent nucleation of noncentrosomal microtubules at the *trans*-Golgi network.

Efimov, A. *et al. Dev. Cell* **12**, 917–930 (2007)

Although most microtubules (MTs) are known to be formed by the centrosome, it has been unclear where non-centrosomal MTs originate from. As suggested previously by *in vitro* studies, Efimov *et al.* now show with fluorescent labelling that non-centrosomal MTs can originate from the Golgi apparatus. Specifically, the authors' work suggests that these MTs originate in a γ-tubulin-dependent manner from the *cis*-Golgi and are then recruited to the *trans*-Golgi network (TGN), where further growth is dependent on CLASPs, TGN-localized MT-binding proteins. Unlike the radial MTs that extend from the centrosome, these Golgi-derived MTs are asymmetrical and preferentially orientate towards the leading edge in polarized and motile cells. These findings implicate Golgi-derived MTs in efficient cell migration.

STEM CELLS

DNA repair is limiting for haematopoietic stem cells during ageing.

Nijnik, A. *et al. Nature* **447**, 686–690 (2007)

Deficiencies in DNA damage repair limit the function of haematopoietic stem cells with age.

Rossi, D. J. *et al. Nature* **447**, 725–729 (2007)

A principal mechanism of ageing is thought to be the inability to maintain tissue homeostasis owing to the accumulation of DNA damage in stem cells. Having discovered a mouse strain with a hypomorphic mutation in LIG4, a non-homologous end-joining (NHEJ) DNA-repair protein, Nijnik and colleagues found that inefficient NHEJ resulted in progressive loss of bone-marrow-derived stem-cell populations, as manifested through loss of erythropoiesis, a feature of normal ageing. Rossi *et al.* examined the consequences of deficiencies in nucleotide-excision repair, telomere maintenance and NHEJ in haematopoietic stem cells. Although deficiencies in these pathways did not affect the sizes of the stem-cell reserve populations, they did cause stem cells to lose their capacity to self-renew and proliferate following stress. These studies suggest that inherited and genetic factors that increase the levels of DNA damage might be key determinants of stem-cell capacity and, therefore, ageing.

CELL CYCLE

Maximal chromosome compaction occurs by axial shortening in anaphase and depends on Aurora kinase.

Mora-Bermúdez, F. *et al. Nature Cell Biol.* 10 June 2007 (doi:10.1038/ncb1606)

During cell division, chromosome compaction begins in prometaphase and must be complete before genetic material is segregated. Now, for the first time, Ellenberg and colleagues have monitored chromosome compaction in real time using time-lapse confocal microscopy. Contrary to expectations that chromosomes are most compact in metaphase, compaction continues after sister-chromatid segregation and reaches its maximum in late anaphase. The authors propose that this additional compaction minimizes segregation errors by removing chromosome arms from the cytokinetic plane, thereby preventing the cleavage furrow from damaging them. Aurora B and dynamic microtubules are required for compaction during anaphase.