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# IN BRIEF

## CENTROMERES

The CENP-H–I complex is required for the efficient incorporation of newly synthesized CENP-A into centromeres.

Okada, M. *et al. Nature Cell Biol.* **8**, 446–457 (2006)

The human CENP-A centromeric nucleosome-associated complex.

Foltz, D. *et al. Nature Cell Biol.* **8**, 458–469 (2006)

Two groups report the identification of a multiprotein complex that is associated with histone variant CENP-A-containing centromeric chromatin. This complex, named NAC (nucleosome-associated complex), comprises known and new kinetochore proteins. Foltz *et al.* show that NAC recruits a second complex, CAD (CENP-A distal), that contains other novel kinetochore proteins. Disruption of NAC caused defective chromosome alignment and segregation, but did not affect checkpoint signalling. Okada *et al.* provide evidence that NAC components are required for the efficient incorporation of CENP-A into centromeric chromatin.

## CELL DEATH

Caspase-9 holoenzyme is a specific and optimal procaspase-3 processing machine.

Yin, Q. *et al. Mol. Cell* **22**, 259–268 (2006)

The apoptosome activates caspase-9 by dimerization.

Pop, C. *et al. Mol. Cell* **22**, 269–275 (2006)

In the intrinsic apoptotic pathway, the apoptosome complex recruits and activates caspase-9, which subsequently activates effector caspases, such as caspase-3, that execute apoptosis. Two papers now address a long-standing debate on how the apoptosome activates caspase-9. Yin *et al.* showed that a dimeric caspase-9 construct had greater enzymatic activity than the caspase-9 holoenzyme, which indicates that dimerization is important for its activation. Also, the apoptosome enhances the affinity of caspase-9 for procaspase-3. The findings of Pop *et al.* also favour the dimerization model. They showed that a hybrid-caspase construct that consists of caspase-8 and the recruitment domain of caspase-9 was sufficient to allow caspase-9-based recruitment to the apoptosome.

## AUTOPHAGY

Loss of autophagy in the central nervous system causes neurodegeneration in mice.

Komatsu, M. *et al. Nature* 19 Apr 2006 (doi:10.1038/nature04723)

Suppression of basal autophagy in neural cells causes neurodegenerative disease in mice.

Hara, T. *et al. Nature* 19 Apr 2006 (doi:10.1038/nature04724)

Autophagy is a protein-degradation process that is induced by starvation and generates nutrients for survival. Both studies now implicate the loss of autophagy in neurodegeneration. Mice that lacked either the *Atg7* (autophagy-related-7) or *Atg5* genes specifically in neural cells showed neurological defects and an accumulation of cytoplasmic proteins in inclusion bodies. These findings indicate that autophagy is required for the normal turnover of proteins to avoid the accumulation of abnormal proteins that can disrupt neural function.