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IN BRIEF

EPIGENETICS

Dynamics of replication-independent histone turnover in budding yeast.

Dion, M. F. *et al.* Science **315**,1405–1408 (2007)

Histone replacement marks the boundaries of *cis*-regulatory domains.

Mito, Y., Henikoff, J. G. & Henikoff, S. Science **315**,1408–1411 (2007)

Characterizing the dynamics of chromatin remodelling is crucial to understand chromatin function. Two teams measured the levels of histone turnover, at high resolution, in budding yeast and fruitflies and found a huge variation in histone-replacement rate across the genomes of both organisms. Histone turnover is higher at promoters, rather than in coding regions, where it could transiently expose DNA *cis*-regulatory elements to other diffusible factors, providing the potential for a rapid and dynamic regulation. Rapid histone turnover was also found at chromatin boundaries, where it could serve to prevent the spreading of chromatin states and functionally delimit different domains.

TECHNOLOGY

Targeted gene addition into a specified location in the human genome using designed zinc finger nucleases.

Moehle, E. A. *et al. Proc. Natl Acad. Sci. USA* **104**, 3055–3060 (2007)

This study presents a new strategy for the insertion of ectopic DNA sequences in specific genomic locations. Engineered zinc finger nucleases (ZFNs) were used to induce DNA double-strand breaks (DSBs) at specific loci in human cells. The subsequent homology-directed repair of the DSBs was then allowed to occur in the presence of an extrachromosomal DNA donor, which carried the ectopic sequence of interest that was to be inserted flanked by two locus-specific homology arms. ZFNs guaranteed the site-specific integration of DNA fragments — of up to 8 kb — with high frequency, no need for selection and no increase in random integration.

CANCER GENETICS

A virus causes cancer by inducing massive chromosomal instability through cell fusion.

Duelli, D. M. *et al. Curr. Biol.* **17**, 431–437 (2007)

Chromosomal instability (CIN) is a hallmark of many human tumours, and tumours are often associated with viral infection. This work provides a new link between these two observations, showing that viruses can be tumorigenic by inducing cell fusion, which leads to CIN. The authors used the Mason–Pfizer monkey virus to fuse oncogene-expressing human fibroblasts, and showed through karyotype analyses that these hybrid cells were highly aneuploid, had many numerical and structural chromosomal aberrations and were tumorigenic in mice. As many viruses that infect human cells are fusogenic, preventing infection or cell fusion in infected individuals might help to reduce cancer incidence.