IN BRIEF

AGEING

Re-evaluating oxidative damage in ageing

Damaging reactive oxygen species (ROS) are thought to be drivers of mitochondrial mutagenesis and ageing. However, thorough investigation of mitochondrial DNA (mtDNA) mutations has been hampered by a lack of methods to accurately detect such mutations, which are rare. Using a recently developed, highly specific and accurate method called duplex sequencing, the authors assessed mtDNA mutations from brain tissues of old and young individuals. Surprisingly, they found that although the total number of mutations increases with age, there is no increase in mutations that are characteristic of ROS damage, which suggests a rethink of the role of ROS in theories of ageing.

ORIGINAL RESEARCH PAPER Kennedy, S. R. *et al.* Ultra-sensitive sequencing reveals an age-related increase in somatic mitochondrial mutations that are inconsistent with oxidative damage. *PLoS Genet.* **9**, e1003794 (2013)

EPIGENETICS

Transgenerational effects of folate defects

This study reveals transgenerational epigenetic effects of genetic defects in folate metabolism. Mutation of the methionine synthase reductase gene (*Mtrr*) in mice resulted in both congenital malformations and growth defects in offspring; these defects mimic the effects of inadequate folate consumption during pregnancy. The congenital defects persisted, independently of the maternal environment, for five generations in progeny that did not carry the initial mutation, which seems to be due to epigenetic inheritance. Conversely, the growth defects were found to be due to the altered maternal environment, specifically adverse effects of *Mtrr* deficiency on the uterine environment.

ORIGINAL RESEARCH PAPER Padmanabhan, N. *et al.* Mutation in folate metabolism causes epigenetic instability and transgenerational effects on development. *Cell* **155**, 81–93 (2013)

EVOLUTION

Resolving Darwin's dilemma

The near-simultaneous appearance of most modern animal body plans in the Cambrian explosion suggests a brief interval of rapid phenotypic and genetic evolution, which Darwin believed were too fast to be explained by natural selection. Lee *et al.* have now inferred the rates of these innovations by applying phylogenetic clock methods to the arthropods during this period. The rates of genetic and phenotypic evolution were shown to be ~4 times and ~5.5 times faster, respectively, than all subsequent parts of the Phanerozoic era — consistent with natural selection during the Cambrian explosion.

ORIGINAL RESEARCH PAPER Lee, S. Y. *et al.* Rates of phenotypic and genomic evolution during the Cambrian explosion. *Curr. Biol.* http://dx.doi.org/10.1016/j.cub.1013.07.055 (2013)

DNA DAMAGE

H3.3 recovers transcription

The recovery of transcription after genotoxic stress is crucial for cells. Here, the authors show that the histone chaperone HIRA deposits histone H3.3 to sites of ultraviolet C irradiation before these sites are repaired, in a manner that is dependent on local chromatin ubiquitylation events after such damage. This early function of HIRA is required for transcriptional activation after repair, and the authors propose that it acts as a chromatin 'bookmark' for transcription recovery.

ORIGINAL RESEARCH PAPER Adam, S., Polo, S. E. & Almouzni, G. Transcription recovery after DNA damage requires chromatin priming by the H3.3 histone chaperone HIRA. *Cell* **155**, 94–106 (2013)