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IN BRIEF

MUTATION

Linking transcription and genome instability

Two papers reveal details of how transcription can jeopardize genome integrity. Helmrich *et al.* showed that transcription of large human genes takes longer than one cell cycle, resulting in inevitable collisions between transcription and replication machineries; the sites of collision are associated with the formation of RNA–DNA hybrids and genomic instability. The importance of RNA–DNA hybrids is also shown by Wahba *et al.*, who found increased hybrid formation and chromosomal instability in yeast mutants that are defective in RNA biogenesis.

ORIGINAL RESEARCH PAPERS Helmrich, A. *et al.* Collisions between replication and transcription complexes cause common fragile site instability at the longest human genes. *Mol. Cell.* **44**, 966–977 (2011) | Wahba, L. *et al.* RNase H and multiple RNA biogenesis factors cooperate to prevent RNA:DNA hybrids from generating genome instability. *Mol. Cell.* **44**, 978–988 (2011)

BIOINFORMATICS

Data compression facilitates genome assembly

As genome sequence data sets continue to grow, there is a pressing need to develop accurate yet memory-efficient means of assembling genomes *de novo*. Using new computational tools, the authors assembled a human genome using less than 64 gigabytes of memory. A compression algorithm stores the reads efficiently by taking advantage of redundancy between them; the compressed reads are then error-corrected and assembled by String Graph Assembler, which is a new algorithm that is easily parallelizable.

ORIGINAL RESEARCH PAPER Simpson, J. T. & Durbin, R. Efficient *de novo* assembly of large genomes using compressed data structures. *Genome Res.* 7 Dec 2011 (doi:10.1101/gr.126953.111)

DIFFERENTIATION

Asymmetry caused by replication-coupled chromatin assembly

The mechanistic details of how asymmetric cell divisions result in differential gene expression in daughter cells are poorly characterized. Working in *Caenorhabditis elegans*, Nakano *et al.* found that the M1 motor neuron cell identity, which results from asymmetric cell division, is abolished by a histone H3 mutation that disrupts nucleosome assembly or by deficiencies in homologues of chromatin assembly factor 1 (CAF1) and proliferating cell nuclear antigen (PCNA). These data suggest a model whereby replication-coupled asymmetric chromatin assembly differentially regulates the expression of as of yet uncharacterized cell fate genes in daughter cells.

ORIGINAL RESEARCH PAPER Nakano, S., Stillman, B. & Horvitz, H. R. Replication-coupled chromatin assembly generates a neuronal bilateral asymmetry in *C. elegans*. *Cell* **147**, 1525–1536 (2011)

DEVELOPMENT

Role found for DNMT3A in somatic stem cells

The authors highlight a previously unknown role for the *de novo* DNA methyltransferase DNMT3A in somatic stem cells and provide mechanistic insights into the role of DNMT3A in haematopoietic stem cell (HSC) function. Mice in which *Dnmt3a* was conditionally ablated in HSCs show a decline in the differentiation potential of HSCs and a bias towards HSC self-renewal. DNA methylation and transcriptional profiling of *Dnmt3a*-null HSCs is consistent with hypomethylation being associated with the upregulation of multipotency genes.

ORIGINAL RESEARCH PAPER Challen, G. A. *et al.* Dnmt3a is essential for hematopoietic stem cell differentiation. *Nature Genet.* **44**, 23–31 (2012)