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IN BRIEF

DEVELOPMENT

DNA methylation and female puberty

The onset of puberty is known to be regulated by a complex network of genes; however, our understanding lacks mechanistic details. In this paper, the authors found that puberty in rats is preceded by increased promoter DNA methylation and downregulation of two Polycomb genes in the hypothalamus. These Polycomb genes were found to repress the puberty-activating gene *Kiss1*, and inhibition of DNA methylation resulted in their sustained expression and pubertal failure. Hence, the controlled repression of Polycomb genes in the hypothalamus is an epigenetic mechanism for regulating the timing of puberty.

ORIGINAL RESEARCH PAPER Lomniczi, A. *et al.* Epigenetic control of female puberty. *Nature Neurosci.* 27 Jan 2013 (doi:10.1038/nn.3319)

EPIGENETICS

Epigenomics road map

Here, histone modification maps for 29 human tissues and cell types, including stem cells, were obtained by chromatin immunoprecipitation followed by high-throughput sequencing (ChIP–seq). The authors used these maps to identify enhancers and other distal regulatory elements to an unprecedented scale across different developmental stages. By following changes in chromatin states during differentiation, combined with known correlations between these states and nuclear architecture, they found that developmental specification is associated with progressive chromatin restriction and alterations to nuclear architecture. The study is a valuable resource for understanding the role of chromatin in cellular phenotypes.

ORIGINAL RESEARCH PAPER Zhu, J. *et al.* Genome-wide chromatin state transitions associated with developmental and environmental cues. *Cell* **152**, 642–654 (2013)

GENOMICS

Finding Mr Anonymous

There is ongoing debate regarding the extent to which anonymous human sequence data can reveal donor identities. Given that paternal inheritance results in an association between short tandem repeats on the Y chromosome (Y-STRs) and surnames, Gymrek *et al.* showed that Y-STR haplotypes from 911 men could be used to deduce their known surname with a ~12% success rate. Furthermore, they found that Y-STR haplotypes, derived from personal whole-genome sequences, could be combined with associated demographic data to identify the individual participant in some cases. Crucially, all of the data analysed are freely available from public web resources.

ORIGINAL RESEARCH PAPER Gymrek, M. *et al.* Identifying personal genomes by surname inference. *Science* **339**, 321–324 (2013)

SMALL RNAS

A tumour-suppressive tRNA fragment

Although some tRNAs can be processed into other small RNAs, the functions of these fragments are generally poorly defined. Maute *et al.* showed that the tRNA-derived fragment CU1276 exhibits various properties of a functional microRNA and is downregulated in B cell lymphoma relative to normal B cells. CU1276 was also found to repress the expression of replication protein A1 (RPA1) as a potential tumour suppressive mechanism to limit cell proliferation and modulate the DNA damage response.

ORIGINAL RESEARCH PAPER Maute, R. L. *et al.* tRNA-derived microRNA modulates proliferation and the DNA damage response and is down-regulated in B cell lymphoma. *Proc. Natl Acad. Sci. USA* **110**, 1404–1409 (2013)