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IN BRIEF

ADDICTION

Curtailing reward

Histone deacetylases (HDACs), which regulate transcription, seem to modulate cocaine reward behaviours, but the underlying mechanisms are unclear. A new study shows that in rodent striatal neurons, cocaine and cyclic AMP signalling, which is upregulated by cocaine, induce HDAC5 nuclear translocation. Such translocation is dependent on transient dephosphorylation of Ser279 in the HDAC5 nuclear localization signal. Mice overexpressing Ser279-non-phosphorylatable HDAC5 in the nucleus accumbens show impaired development of cocaine-induced reward behaviour in a cocaine-conditioned place preference assay. Thus, HDAC5 activity seems to limit the rewarding effects of cocaine, possibly via transcriptional effects.

ORIGINAL RESEARCH PAPER Taniguchi, M. *et al.* Histone deacetylase 5 limits cocaine reward through cAMP-induced nuclear import. *Neuron* **73**, 108–120 (2012)

CEREBRAL CORTEX

Whisking up a change in state

In mammals, quiet wakefulness and active behaviour are associated with synchronized and desynchronized patterns, respectively, of spontaneous cortical activity (termed cortical states). The mechanisms controlling cortical states are poorly understood. Poulet *et al.* showed that active whisking behaviour in mice, which is associated with a desynchronized state in the barrel cortex, is linked to increased thalamic activity, and that thalamic inactivation blocks the desynchronized state during whisking. In animals in quiet wakefulness, optogenetic-stimulated thalamic firing could induce a desynchronized cortical state in the barrel cortex. These findings suggest that thalamic activity may drive cortical desynchronization.

ORIGINAL RESEARCH PAPER Poulet, J. F. A. *et al.* Thalamic control of cortical states. *Nature Neurosci.* 22 Jan 2012 (doi:10.1038/nn.3035)

NEURODEGENERATION

Alternative neuronal loss

Although various RNA-processing proteins are implicated in neurodegenerative mechanisms, there is no firm evidence that defective RNA splicing causes neuronal loss. The pre-messenger RNA splicing machinery includes several uridine-rich small nuclear RNAs (U-snRNAs). Here, Jia *et al.* show that in mice, a mutation in *Rnu2-8*, which is one of several U2 snRNA genes, causes progressive neurodegeneration and ataxia. This neuronal loss follows a distinct spatiotemporal pattern and is associated with defects in alternative splicing. Thus, impaired RNA processing and, specifically, mutations in U-snRNA genes can have profound effects on neuronal viability.

ORIGINAL RESEARCH PAPER Jia, Y. *et al.* Mutation of a U2 snRNA gene causes global disruption of alternative splicing and neurodegeneration. *Cell* **148**, 296–308 (2012)

GENE EXPRESSION

Transcriptional mapping

How the transcriptomes of various cell types in a specific brain area differ is unclear. Here, Siegert *et al.* created an atlas of cell type transcriptomes for the mouse retina and found that each cell type was associated with the expression of a specific set of genes, including a specific set of transcription factor genes. This atlas may be useful in various ways, including the mapping of disease-associated genes to specific retinal cell types, providing insight into disease pathophysiology.

ORIGINAL RESEARCH PAPER Siegert, S. *et al.* Transcriptional code and disease map for adult retinal cell types. *Nature Neurosci.* 22 Jan 2012 (doi:10.1038/nn.3032)