NEUROANATOMY

Fast-track vision

The responses of dopaminergic neurons in the midbrain to visual stimuli — particularly those that predict reward — has come under scrutiny recently, but the pathway by which visual information reaches these neurons is unclear. Comoli *et al*. have found a direct projection from a primary visual area, the superior colliculus, to the substantia nigra that could be responsible for the very fast responses of the dopamine neurons.

Previous clues to the existence of such a pathway came from the finding that chemical stimulation of the superior colliculus could cause correlated bursting in midbrain dopaminergic nuclei. Also, cortical response latencies to visual stimuli are longer than those of dopaminergic neurons, making it unlikely that the responses of these neurons are mediated by input from the visual cortex.

Comoli and colleagues used anterograde tracing to investigate whether there was a direct link between the superior colliculus and the midbrain dopaminergic nuclei. They injected tracers into the superior colliculus and found that they were transported specifically to the dopaminergic part of the substantia nigra and to the ventral tegmental area. Electron microscopy confirmed that the labelled fibres formed synapses, apparently onto both dopaminergic and non-dopaminergic neurons in these regions. Interestingly, the synapses were both symmetric (probably excitatory) and asymmetric (probably inhibitory), and these two populations of synapses might arise from different groups of neurons in the superior colliculus.

The authors went on to investigate the electrophysiological properties of the pathway. Both the superior colliculus and the substantia nigra showed short-latency responses to visual input, with the superior colliculus responding more quickly. Pharmacological inhibition or disinhibition of the superior colliculus decreased or increased, respectively, the size of the evoked response in the substantia nigra. Finally, removal of the superficial layers of the superior colliculus abolished visual evoked responses in the substantia nigra, whereas removal of the visual cortex did not.

These findings support the idea that a direct projection from the superior colliculus carries short-latency visual input to the dopaminergic areas of the ventral midbrain. However, it remains to be seen whether this pathway can mediate specific responses of dopaminergic neurons to reward-related stimuli. To do so, it would need to distinguish reward-predicting stimuli from other salient or unpredictable events — even before the eyes have moved to localize the event.

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References and links

ORIGINAL RESEARCH PAPER Comoli, E. *et al.* A direct projection from superior colliculus to substantia nigra for detecting salient visual events. *Nature Neurosci.* **6**, 974–980 (2003)

IN BRIEF

SENSORY SYSTEMS

Trans-synaptic shift in anion gradient in spinal lamina I neurons as a mechanism of neuropathic pain.

Coull, J. A. M. *et al*. *Nature* **424**, 938–942 (2003)

By chronically constricting the sciatic nerve, the authors induced neuropathic pain in the rat and identified a reduction in expression of the K^+–Cl^- transporter KCC2 as the underlying mechanism. This trans-synaptic reduction led to disruption of Cl^- homeostasis, and caused GABA (γ-aminobutyric acid)- and glycine-mediated neurotransmission to become excitatory instead of inhibitory, increasing the excitability of spinal neurons. Moreover, reducing the function of KCC2 in intact rats led to a reduction in the nociceptive threshold.

SYSTEMS NEUROSCIENCE

Amphetamine or cocaine limits the ability of later experience to promote structural plasticity in the neocortex and nucleus accumbens.

Kolb, B. *et al*. *Proc. Natl Acad. Sci. USA* **100**, 10523–10528 (2003)

Do plastic changes induced by drugs of abuse interfere with the ability of experience to elicit similar changes? The authors repeatedly treated rats with amphetamine or cocaine, and found that the drugs limited the ability of an enriched environment to alter dendritic structure. This interference might relate to the cognitive deficits that are associated with drug abuse.

PSYCHIATRIC DISORDERS

Abnormal neural synchrony in schizophrenia.

Spencer, K. M. *et al*. *J. Neurosci.* **23**, 7407–7411 (2003)

According to this electroencephalographic study, schizophrenia is associated with disturbances of neuronal synchronization in the gamma-frequency range. The authors asked patients to perform a task that required feature binding, and found that both the amplitude and the phase synchronization of gamma oscillations was impaired, raising the possibility that such an impairment might be related to the cognitive disturbances that characterize the disease.

NEUROLOGICAL DISEASES

A peptide inhibitor of c-Jun N-terminal kinase protects against excitotoxicity and cerebral ischemia.

Borsello, T. *et al*. *Nature Med.* **9**, 1180–1186 (2003)

The authors used a peptide inhibitor to target c-Jun N-terminal kinase (JNK), which is activated as part of an excitotoxic cascade, in two animal models of cerebral ischaemia. Intracerebroventricular and systemic administration of the peptide as late as 6 h after the ischaemic episode significantly reduced brain damage and activation of the JNK pathway. These results indicate that JNK is a valid drug target for anti-ischaemic drugs, and highlight the potential of the peptide inhibitor as therapeutic agent.