

IN BRIEF

SYNAPTIC PHYSIOLOGY**Neuroigin and neurexin go retro**

Acetylcholine (ACh) release at the *Caenorhabditis elegans* neuromuscular junction is inhibited by a retrograde signal that is induced by inactivation of microRNA 1, but the nature of the signal was unknown. The authors show that retrograde signalling requires presynaptic expression of neuroigin 1 (*nlg1*) and postsynaptic expression of neurexin 1 (*nrx1*). The retrograde signal inhibits exocytosis of distal synaptic vesicles, causing ACh release to be fast- and short-lasting rather than slow and prolonged. Mutations in *NLG* and *NRX* have been associated with autism, suggesting that prolonged synaptic responses may contribute to the autism phenotype.

ORIGINAL RESEARCH PAPER Hu, Z. et al. Neurexin and neuroigin mediate retrograde synaptic inhibition in *C. elegans*. *Science* 2 Aug 2012 (doi:10.1126/science.1224896)

TECHNIQUES**Optogenetic control in monkey brains**

Despite advances in optogenetic control of distinct neurons in rodents, applying optogenetics in monkeys has been challenging. Here, the authors report that optogenetic stimulation of neurons in the arcuate sulcus of two rhesus monkeys during a saccade task induced changes in eye movement. Simultaneous functional MRI (fMRI) showed that optogenetic stimulation induced consistent changes in fMRI activity in functional networks, indicating that optogenetic stimulation can be used to alter local as well as network activity.

ORIGINAL RESEARCH PAPER Gerits, A. et al. Optogenetically induced behavioral and functional network changes in primates. *Curr. Biol.* 26 Jul 2012 (doi: 10.1016/j.cub.2012.07.023)

REWARD**Serotonin promotes patience**

Activation of serotonin neurons in the raphe nucleus has been associated with waiting for delayed rewards in rats, but whether this reflects a causal relationship was not known. Here, the authors used a navigation task in which rats had to remain in a fixed posture at a reward site to receive a food or water reward. Inhibition of serotonin neuron activity in the dorsal raphe nucleus caused rats to fail to wait for a reward if the delay was long, whereas it had no effect under conditions in which the delay was short. The inhibition did not affect cognitive or motor functions. These findings suggest that activation of serotonin neurons is required for waiting for delayed rewards.

ORIGINAL RESEARCH PAPER Miyazaki, K. W., Miyazaki, K. & Doya, K. Activation of dorsal raphe serotonin neurons is necessary for waiting for delayed rewards. *J. Neurosci.* **32**, 10451–10457 (2012)

PSYCHIATRIC DISORDERS**Improving responses to antipsychotics**

About 30% of patients with schizophrenia do not respond to antipsychotic drugs. The authors showed that chronic administration of atypical antipsychotics downregulates metabotropic glutamate 2 receptor (*mGlu2*) expression in the frontal cortex in mice and in patients with schizophrenia, and this was due to a serotonin receptor 2A-dependent increase in histone deacetylase 2 (*Hdac2*) expression and HDAC2 binding to the *mGlu2* promoter. Chronic administration of HDAC inhibitors improved the effects of antipsychotic drugs in a mouse model of schizophrenia, pointing to their potential clinical use in treatment-resistant schizophrenia.

ORIGINAL RESEARCH PAPER Kurita, M. et al. HDAC2 regulates atypical antipsychotic responses through the modulation of *mGlu2* promoter activity. *Nature Neurosci.* 5 Aug 2012 (doi:10.1038/nrn.3181)