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IN BRIEF

PREFRONTAL CORTEX

Down in front

Disruption of nicotinic acetylcholine receptor (nAChR) signalling in the prefrontal cortex has been suggested to underlie cognitive deficits in schizophrenia. In this study, mice expressing a variant of human nAChR subunit α5 implicated in schizophrenia showed impaired sociability and sensorimotor gating. Two-photon calcium imaging revealed that these mice have lower pyramidal neuron activity in layers 2/3 of the prelimbic cortex, and this activity was normalized by chronic nicotine infusion, suggesting nicotine could have potential in treating cognitive symptoms of schizophrenia.

ORIGINAL ARTICLE Koukouli, F. et al. Nicotine reverses hypofrontality in animal models of addiction and schizophrenia. *Nat. Med.* http://dx.doi.org/10.1038/nm.4274 (2017)

NEURODEGENERATIVE DISEASE

Losing the way

Individuals with Alzheimer disease (AD) often have difficulties with spatial navigation, which depends on the entorhinal cortex (EC), but whether tau aggregates affect the EC in AD is unclear. Fu et al. studied mice expressing aggregable human tau in the hippocampal formation (EC-tau mice). Compared with controls, old EC-tau mice (aged ≥30 months) showed spatial-memory deficits in the Morris water maze and reduced grid-cell firing and periodicity. Moreover, tau aggregation and cell death in old EC-tau mice specifically affected excitatory, but not inhibitory, neurons in the medial EC. Thus, tau aggregates may impair spatial memory in AD by affecting EC network activity.

ORIGINAL ARTICLE Fu, H. *et al.* Tau pathology induces excitatory neuron loss, grid cell dysfunction, and spatial memory deficits reminiscent of early Alzheimer's disease. *Neuron* http://dx.doi.org/10.1016/j.neuron.2016.12.023 (2017)

BEHAVIOURAL NEUROSCIENCE

Right on (social) cue

The neural circuitry mediating the ability to produce learned responses to social cues is unknown. Here, 'observer' rats watched 'demonstrator' rats that received footshocks paired with a tone; with social learning, observers learned to freeze in response to the tone. Chemogenetic inhibition of lateral amygdala (LA) neurons projecting to the medial amygdala (MeA) reduced social learning. Rats lacking neurexin 1 (a model of autism spectrum disorder (ASD)) showed disrupted LA→MeA transmission and social learning deficits that were rescued by chemogenetic activation of the MeA. LA→MeA neurons therefore regulate social learning and may be disrupted in ASD.

ORIGINAL ARTICLE Twining, R. C. *et al.* An intra-amygdala circuit specifically regulates social fear learning. *Nat. Neurosci.* http://dx.doi.org/10.1038/nn.4481 (2017)

CELL BIOLOGY OF THE NEURON

Adding fuel to the firing

Neuronal activity promotes glycolysis at terminals to sustain synaptic vesicle (SV) recycling; however, the underlying mechanisms are not known. Ashrafi *et al.* expressed a pHluorin-tagged variant of the glucose transporter GLUT4 in rat hippocampal neurons and showed that, with sustained firing of action potentials, GLUT4 is exocytosed to the cell membrane from endosomes that are distinct from SVs. Moreover, GLUT4 translocation is necessary for SV endocytosis and recycling, and relies on activation of AMP kinase. Thus, GLUT4 provides metabolic support during sustained synaptic activity.

ORIGINAL ARTICLE Ashrafi, G. *et al.* GLUT4 mobilization supports energetic demands of active synapses. *Neuron* http://dx.doi.org/10.1016/j.neuron.2016.12.020 (2017)