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IN BRIEF

SYNAPTIC PLASTICITY

Stacking up synaptic structure

The proximity of neurotransmitter-release sites to the corresponding postsynaptic receptors influences synaptic strength and plasticity, but the precise alignment between the two is poorly elucidated. Here, the density of the active-zone proteins Rab3-interacting molecule 1 and 2 (RIM1/2) at synapses between cultured hippocampal neurons was found to be aligned with areas of increased expression of postsynaptic density (PSD) proteins (PSD95 and GluA2 AMPA receptor subunit), forming a trans-synaptic 'nanocolumn'. Induction of long-term potentiation resulted in increased PSD95 density in postsynaptic nanoclusters, suggesting that reorganization of these nanocolumns plays a part in synaptic plasticity.

ORIGINAL ARTICLE Tang, A.-H. *et al.* A trans-synaptic nanocolumn aligns neurotransmitter release to receptors. *Nature* http://dx.doi.org/10.1038/nature19058 (2016)

LEARNING AND MEMORY

How synapses form memories

The synaptic remodelling that occurs during activity-dependent plasticity and that is thought to underlie memory formation is incompletely understood, but complement component 1 q subcompartment-like (C1qL) proteins have been reported to be involved. Here, C1qL3 was found to be expressed in an activity-dependent manner in a subset of basolateral amygdala neurons that project to the medial prefrontal cortex (mPFC). Compared with controls, conditional *C1ql3*-knockout mice had fewer excitatory synapses in mPFC subregions innervated by these neurons and impaired fear memory.

ORIGINAL ARTICLE Martinelli, D. C. *et al.* Expression of C1ql3 in discrete neuronal populations controls efferent synapse numbers and diverse behaviors. *Neuron* http://dx.doi.org/10.1016/j.neuron.2016.07.002 (2016)

NEUROPROTECTION

Power release

Damaged mitochondria can be released from neurons for uptake by glia for disposal. In this study, however, exposure of mice to focal cerebral ischaemia resulted in the release of functional mitochondria from astrocytes and their uptake into adjacent neurons, and these events were coincident with upregulation of the enzyme CD38 in astrocytes. Downregulation of this enzyme in astrocytes reduced both the number of astrocyte-derived mitochondria produced in response to ischaemia and their neuronal uptake. Ischaemia-induced neurological deficits were also increased, suggesting that these mitochondria are neuroprotective.

ORIGINAL ARTICLE Hayakawa, K. *et al.* Transfer of mitochondria from astrocytes to neurons after stroke. *Nature* **535**, 551–555 (2016)

GLIA

Motor learning with oligodendrocytes

Oligodendrocyte precursor cells (OPCs) are produced throughout life and are required for motor learning, but their role in this process is unclear. Within 2–3 hours of beginning a motor-learning task, mice lacking the transcription factor MYRF (myelin regulator factor) showed reduced levels of new oligodendrocytes and impaired learning. Wild-type mice trained in this task exhibited accelerated differentiation of OPCs, suggesting that OPC differentiation contributes to motor learning in this task.

ORIGINAL ARTICLE Xiao, L. *et al.* Rapid production of new oligodendrocytes is required in the earliest stages of motor-skill learning. *Nat. Neurosci.* http://dx.doi.org/10.1038/nn.4351 (2016)