IN BRIEF

GLIA

TNFα controls glutamatergic gliotransmission in the hippocampal dentate gyrus

Santello, M., Bezzi, P. & Volterra, A. *Neuron* **69**, 988–1001 (2011)

Ca^{2+}-dependent glutamate release from astrocytes is thought to regulate synaptic transmission. TNFα controls this process in cultured astrocytes, but the underlying mechanisms were not known. The authors showed that in hippocampal slices from *TNFα*$^{-/-}$ mice, activation of purinergic P2Y1 receptors increased intracellular Ca^{2+} in astrocytes without inducing synaptic modulation. The absence of synaptic modulation was due to altered vesicle docking resulting in slowed glutamate exocytosis from astrocytes. These effects could be normalized by adding low concentrations of TNFα. Thus, TNFα regulates gliotransmission by controlling astrocytic glutamate release.

REPAIR

Proteoglycan-specific molecular switch for RPTPσ clustering and neuronal extension

Coles, C. H. *et al. Science* 31 March 2011 (doi:10.1126/science.1200840)

Heparan and chondroitine sulphate proteoglycans (HSPGs and CSPGs) promote and inhibit axon extension, respectively. In dorsal root ganglion (DRG) neurons, both effects required receptor protein tyrosine phosphatase-σ (RPTPσ), and HSPGs and CSPGs bound to the same RPTPσ glucosaminoglycan binding site. Only HSPGs induced receptor oligomerization, and this was inhibited by CSPGs. In DRG cultures, exogenous CSPG blocked the outgrowth promoting effect of exogenous HSPGs. These data suggest that the HSPG to CSPG ratio determines whether RPTPσ forms growth promoting clusters, and point to new strategies for repair after nerve injury.

NEUROLOGICAL DISORDERS

Single-neuron dynamics in human focal epilepsy

Truccolo, W. *et al. Nature Neurosci.* 27 March 2011 (doi:10.1038/nn.2782)

Electroencephalogram recordings have suggested that epileptic seizures arise from hypersynchronous neuronal firing in large neuronal populations; however, the activity of single neurons during a seizure has never been recorded in humans. The authors used microelectrode arrays to record from hundreds of neurons before and during seizures in human patients. They discovered that neuronal firing patterns are in fact heterogeneous during seizure induction and spread, becoming homogeneous as a seizure ends. These findings have implications for our understanding of epilepsy mechanisms and the development of seizure prediction and intervention methods.

SENSORY SYSTEMS

Loss-of-function mutations in sodium channel $Na_v1.7$ cause anosmia

Weiss, J. *et al. Nature* 23 March 2011 (doi:10.1038/nature09975)

The genetic basis of congenital anosmia (the inability to sense odours) is largely unknown. Here, the authors showed that three individuals who are unable to sense pain as a result of mutations in the sodium channel $Na_v1.7$ are also unable to detect odours. Likewise, transgenic mice lacking $Na_v1.7$ expression in olfactory sensory neurons could not detect odours or respond appropriately in odour-driven behavioural tests. This provides new information on the molecular basis of odour detection and a link between the senses of smell and pain.