

mice, the authors studied NMJs that contained one input from each labelled neuron. Interestingly, they found that if the 'cyan' input won the competition against a 'yellow' axon at one NMJ, it also beat the yellow input at all the other NMJs that were co-innervated by these two neurons. However, this did not mean that the outcome for these two axons would be the same against all competitors, and by bringing a third neuron into the equation, the authors could establish a competitive hierarchy.

So what gives a motor neuron the competitive edge? In a second study, Buffelli and colleagues showed that synaptic efficacy might be a defining factor. They reduced neurotransmission in some motor neurons by conditionally knocking out the gene for choline acetyltransferase (*ChAT*). In the absence of competition, the *ChAT*-negative axons could readily make synapses with muscle fibres, but their inputs were always eliminated when they were pitted against wild-type inputs.

In addition, Kasthuri and Lichtman showed that the ranking of a motor neuron in the competitive

hierarchy was inversely proportional to the size of its axonal tree. Taken together with the findings of Buffelli *et al.*, this might imply that each neuron has a finite supply of neurotransmitter, which is spread more thinly as the size of the motor unit increases.

These findings highlight the benefits of looking at the bigger picture to make sense of events at individual synapses. Manipulations such as knocking out the *ChAT* gene create abnormally large imbalances in synaptic efficacy, and it remains to be seen whether natural variations in neurotransmission are sufficient to drive competition at the NMJ, or whether it requires additional factors that are globally distributed in motor neurons.

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References and links

ORIGINAL RESEARCH PAPERS Kasthuri, N. & Lichtman, J. W. The role of neuronal identity in synaptic competition. *Nature* **424**, 426–430 (2003) | Buffelli, M. *et al.* Genetic evidence that relative synaptic efficacy biases the outcome of synaptic competition. *Nature* **424**, 430–434 (2003) **FURTHER READING** Sanes, J. R. & Lichtman, J. W. Development of the vertebrate neuromuscular junction. *Annu. Rev. Neurosci.* **22**, 389–442 (1999) | Sanes, J. R. & Lichtman, J. W. Induction, assembly, maturation and maintenance of a postsynaptic apparatus. *Nature Rev. Neurosci.* **2**, 791–805 (2001)

early in the visual pathway, although more experiments will be needed to support this idea. More importantly, the data indicate that mnemonic problems are not the only feature of the preclinical stage of Alzheimer's disease, but that attentional deficits, which might have diagnostic implications, are also present.

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References and links

ORIGINAL RESEARCH PAPER Perry, R. J. & Hodges, J. R. Dissociation between top-down attentional control and the time course of visual attention as measured by attentional dwell time in patients with mild cognitive impairment. *Eur. J. Neurosci.* **18**, 221–226 (2003) **FURTHER READING** Corbetta, M. & Shulman, G. L. Control of goal-directed and stimulus-driven attention in the brain. *Nature Rev. Neurosci.* **3**, 201–215 (2002)

IN BRIEF

ION CHANNELS

Atomic proximity between S4 segment and pore domain in Shaker potassium channels.

Lainé, M. *et al. Neuron* **39**, 467–481 (2003)

Crystallographic data on the structure of the bacterial channel KvAP indicated that S4 — the voltage-sensing domain — is at the periphery of the channel and moves through the membrane in response to voltage changes. Here, Lainé *et al.* present a different model for Shaker K⁺ channels; on the basis of disulphide-bond formation between engineered pairs of cysteines and molecular modelling, they argue that S4 is located between pore domains.

NEURODEGENERATIVE DISEASE

Neurodegeneration and defective neurotransmission in a *Caenorhabditis elegans* model of tauopathy.

Kraemer, B. C. *et al. Proc. Natl Acad. Sci. USA* 18 Jul 2003 (doi:10.1073/pnas.1533448100)

Kraemer *et al.* created a new transgenic model of tau-induced neurodegeneration by expressing wild-type and mutant forms of human tau in *C. elegans*. The expression of wild-type tau led to uncoordinated locomotor behaviour, accumulation of tau and neurodegeneration. These phenotypes were exacerbated in worms that expressed the mutant protein.

NEUROIMMUNOLOGY

Polyamines play a critical role in the control of the innate immune response in the mouse central nervous system.

Soulet, D. & Rivest, S. *J. Cell Biol.* **162**, 257–268 (2003)

The authors found a link between polyamines and the innate immune response in the nervous system. Challenging mice with systemic lipopolysaccharide increased the neuronal and glial levels of ornithine decarboxylase — the rate-limiting enzyme for the synthesis of polyamines. This treatment also elicited an increase in the production of pro-inflammatory cytokines, which was abolished by inhibiting polyamine synthesis. Similarly, the inhibition of polyamine synthesis prevented neuronal death in a mouse model of innate immune reactivity in the brain.

NEUROPHYSIOLOGY

Passive transport disrupts directional path integration by rat head direction cells.

Stackman, R. W. *et al. J. Neurophysiol.* 30 July 2003 (doi:10.1152/jn.00346.2003)

Head-direction cells discharge when the head of a rat points in a preferred direction. Here, the authors manipulated different interoceptive cues to test their importance for maintaining the preferred firing direction under conditions in which external cues were unavailable. Altering proprioceptive cues by passively transporting the rat between locations elicited the most significant shift of the preferred direction, highlighting the relevance of proprioception for spatial navigation under conditions that require path integration.