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IN BRIEF

DEVELOPMENT

UNC-6/Netrin induces neuronal asymmetry and defines the site of axon formation.

Adler, C. E. *et al. Nature Neurosci.* **9**, 511–518 (2006)

A defining characteristic of neurons is the ability to form long neurites, but the mechanisms leading to neuronal asymmetry and process elongation are poorly understood. Evidence that guidance information might direct the earliest stages of asymmetric neuronal growth comes from observations that neurons usually extend their first stable process in the exact direction that will be taken by the eventual axon. Adler and colleagues show that netrin (UNC-6) and its receptor UNC-40, which are known for their role in growth cone guidance, are also active at early developmental stages to generate, maintain and orient asymmetry in *Caenorhabditis elegans* HSN neurons. As the axon forms, UNC-40 and another protein, MIG-10, become localized to the ventral side of HSN neurons, where they mark the leading edge of the growing neuron that defines the site of axon formation.

NEUROTECHNIQUES

Nano neuro knitting: peptide nanofiber scaffold for brain repair and axon regeneration with functional return of vision.

Ellis-Behnke, R. G. *et al. Proc. Natl Acad. Sci. USA* **103**, 5054–5059 (2006)

Ellis-Behnke and co-workers have combined nanotechnology and biomedicine to stimulate axonal regeneration following injury in the mammalian visual system. The authors report that a self-assembling peptide nanofibre scaffold (SAPNS) permitted regenerated axons to reconnect to target tissues with sufficient density for functional return of vision in hamsters with severed optic tracts. Because SAPNSs can be broken down into component L-amino acids, are synthetic and free of contaminants, and are immunologically inert, they represent a viable technology for tissue repair and treatment of CNS trauma.

AGEING

Early and simultaneous emergence of multiple hippocampal biomarkers of aging is mediated by Ca^{2+}-induced Ca^{2+} release.

Gant, J. C. *et al. J. Neurosci.* **26**, 3482–3490 (2006)

Numerous Ca^{2+}-related electrophysiological processes in the hippocampus are dysregulated with age. These include processes involved in the Ca^{2+}-dependent slow afterhyperpolarization, spike accommodation, Ca^{2+} action potential and whole-cell Ca^{2+} currents. L-type voltage-gated Ca^{2+} channel activity is also increased with age. These findings suggest a possible common mechanism of Ca^{2+} dysregulation in age-related cognitive impairment. Now Gant *et al.* provide evidence that changes in Ca^{2+}-induced Ca^{2+} release from ryanodine receptors on the endoplasmic reticulum underpin Ca^{2+} dysregulation in hippocampal neurons during ageing.