IN BRIEF

GROWTH FACTORS

Neurotrophins induce death of hippocampal neurons via the p75 receptor.

Friedman, W. J. *J. Neurosci.* **20**, 6340–6346 (2000).

The ability of nerve growth factor to elicit cell death by binding to its low-affinity receptor had been documented previously. Here Friedman extends this observation by showing that brain-derived neurotropic factor, and the neurotrophins 3 and 4 can also induce the death of hippocampal neurons in culture. This effect requires binding to the p75 receptor; it does not occur if the cells express the high-affinity neurotrophin receptors, and it seems to require Jun kinase activation. It will be of interest to see if a similar effect can be shown *in vivo*.

BRAIN REPAIR

Xenotransplantation of transgenic pig olfactory ensheathing cells promotes axonal regeneration in rat spinal cord.

Imaizumi, T. *et al. Nature Biotechnol.* **18**, 949–953 (2000).

The use of olfactory ensheathing cells (OEC) has become a promising tool to promote axon regeneration. Here the authors isolated OEC from transgenic pigs expressing a human complement inhibitory protein, and xenografted them to the rat spinal cord. The grafts promoted axon regeneration comparable to that observed for allografts, highlighting the potential of transgenic OEC for xenotransplantation and possible therapeutic use.

DEVELOPMENT

GATA2 is required for the generation of V2 interneurons.

Zhou, Y. *et al. Development* **127**, 3829–3838 (2000).

GATA2 is a transcription factor involved in haematopoiesis. GATA2 is also expressed in the developing brain but its role is unknown. The authors show that GATA2 is expressed in postmitotic V2 interneurons of the spinal cord, that this cell type is absent in *Gata2* knockout mice, and that the spinal cord expression of *Gata2* is controlled by a 190-base pair enhancer. As *Gata2* knockouts die early in development, selective deletion of this enhancer should lead to the elucidation of the functional role of V2 interneurons.

ATTENTION

Modulation of human visual cortex by crossmodal spatial attention.

Macaluso, E. *et al. Science* **289**, 1206–1208 (2000).

A touch to one hand can increase ipsilateral visual discrimination, a phenomenon thought to engage multimodal cortical regions. Using functional magnetic resonance imaging (fMRI), the authors find that the activity of a unimodal visual region — the lingual gyrus — is also increased if a visual stimulus to the right hemifield is accompanied by a touch to the right hand. The authors suggest that back-projections from multimodal parietal regions effectively connected to the visual cortex might be responsible for the crossmodal interactions in a unimodal cortex.

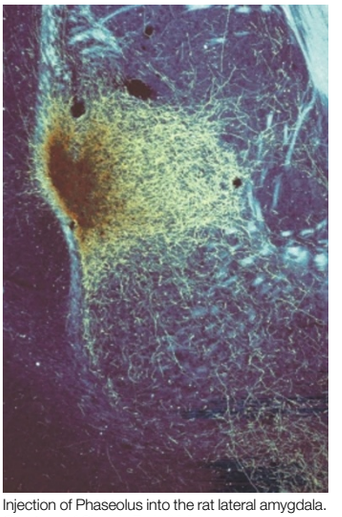
LEARNING AND MEMORY

Shaky memories in indelible ink

The conversion of a transient memory into its enduring version requires the synthesis of new proteins. Once it crosses that threshold, the memory has been consolidated, built to last. It has become ingrained in the brain, much like signing a document using indelible ink. This is, in a nutshell, one of those rare ideas in the learning and memory literature that has gained general acceptance over the years, becoming a beacon for a lot of the current research in the field. However, a report by Nader *et al.* in the 17 August issue of *Nature* indicates that the divide between ephemeral and unshakeable memories is not so unequivocal. These authors provide compelling evidence that the reactivation of a consolidated memory can return it to a labile state, susceptible again to the disruptive effect of protein synthesis inhibitors.

Nader *et al.* trained a group of rats to fear a tone by pairing it with an electric shock. After experiencing both stimuli together, rats tend to freeze in panic the next time they hear the tone alone. As the amygdala is known to be crucial in this form of memory, the authors infused the protein-synthesis inhibitor anisomycin directly into this brain structure at different times during the experiment. They observed that if they administered the inhibitor just after the retrieval of the memory (that is, immediately after the rats froze), their conditioned fear was erased — the following time the rats heard the tone they behaved as if they had never been exposed to the shock in the first place. This effect of anisomycin was observed only if the memory was actually retrieved. If the rats were not given the tone as a reminder, then the inhibitor was ineffective and the rats continued to freeze to the auditory stimulus.

The idea that stable memories can return to a labile state has pervaded the field of learning and memory gradually over the years. Early observations from experimental psychologists and from neurobiologists had already indicated that retrieval is a dynamic process during which new information merges with and modifies the representation of the past. The findings of Nader *et al.* have built upon those observations by tapping for the first time directly onto the brain site where the memory trace is thought to be stored. Although the actual mechanism whereby anisomycin exerts its action remains to be elucidated, the possibility of selectively erasing a memory by eliciting its recollection has profound implications. It will be of immediate interest to find out whether declarative memory tasks are equally susceptible to the withering action of retrieval.



Injection of Phaseolus into the rat lateral amygdala. Courtesy of Claudia Farb (NYU)

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References and links

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FURTHER READING Le Doux, J. E. Emotion circuits in the brain. *Annu. Rev. Neurosci.* **23**, 155–184 (2000)
WEB SITE J. E. Le Doux's laboratory