WEB WATCH

Ch, ch, ch, ch, changes…

Remember watching those old, low-budget comedy shows in which an actor approaches a pedestrian and asks for directions to a coffee shop? While the pedestrian attempts to help, two people pass between the actor and the pedestrian carrying a large door. During this brief interruption, the actor is replaced by another actor, unbeknown to the pedestrian. But even though the two actors look quite different and may have different voices and clothing, the pedestrian somehow fails to notice that they are speaking to a different person after the door has passed in front of them. This and related phenomena are examples of 'change blindness' and are more common than you might think. Just think of all the road accidents in which the driver claims that the pedestrian 'just appeared from nowhere'. Change blindness has been studied experimentally for several years and seems to occur when the changes to a scene that normally produce a motion signal coincide with another event that disrupts that motion signal. Under these conditions, observers are often blind to surprisingly large changes in the visual scene.

The Change Detection Research Database, maintained by Daniel Simons at Harvard University, is a welcome addition to this area. At present, it contains information provided by around 50 scientists working on change detection, and provides a clear and accessible demonstration of change blindness and related attentional phenomena. Useful features include an alphabetical list of change detection references, a list of researchers in this area, and links to web sites providing demonstrations and related links created by the leaders in the field. The demonstrations provided by Ron Rensink are particularly fun — but beware, you may never leave this site!

Peter Collins

DEVELOPMENT

Out of the loop and on the move

The decision of a neuroepithelial cell to stop dividing is so tightly coordinated with the start of both its migration and its differentiation into a neuron that all of these processes can be thought of as a continuum. The fact is, however, that the control of the cell cycle and the acquisition of a neuronal phenotype depend on largely different subsets of molecules that need to be effectively coordinated for the successful development of the nervous system. But how does the cell harmonize these two processes? Although some intrinsic and extrinsic factors have been proposed to regulate cell cycle in neuroepithelial cells, their actual identity remains poorly understood. Dubreuil *et al.* have addressed this question by asking whether the transcription factor Phox2b, which is known to participate in the differentiation of motor neurons, can also induce their precursors to stop dividing.

Phox2b is expressed in the dividing neuroepithelial cells and its absence results in a reduction in the number of postmitotic motor neuron precursors in the hindbrain. The authors found that this reduction was not related to increased cell death or reduced proliferation but to the generation of fewer postmitotic neurons in the ventricular zone. Is this defect related to abnormalities in the cell cycle? Dubreuil *et al.* misexpressed *Phox2b* and found an increase in the number of cells expressing early markers of postmitotic precursors, a smaller number of mitotic cells in the transfected area, and the reduced expression of a cycling cell marker. In addition, *Phox2b* expression caused more cells to migrate out of the ventricular zone.

These, and previous findings on the role of Phox2b, indicate that this transcription factor may act as a

MEMORY

Cross talk

It is well known that human long-term memory is not a unitary system. Evidence from various approaches indicate that an explicit memory system that supports the ability to consciously remember a past experience is disrupted by damage to the medial temporal lobe. In contrast, repetition priming — the non-conscious facilitation of processing that results from implicit memory for previous stimulus presentation — is relatively intact following medial temporal lobe damage. Very little is known about the interactions between these systems. A recent paper in the *Journal of Cognitive Neuroscience* by Wagner, Maril and Schacter provides intriguing evidence of 'crosstalk' between these two systems.

The authors investigated the novel and somewhat counter-intuitive hypothesis that priming may hinder new episodic learning. This hypothesis is based on neuroimaging data demonstrating that greater activation of regions of inferior prefrontal cortex during episodic encoding is associated with superior long-term retrieval, and that priming results in reduced activation in the same brain regions during subsequent re-encoding of the primed stimulus.

On day one of the experiment, subjects incidentally encoded a list of novel words by reading the words on a screen and making a judgement as to whether the word was concrete (chair) or abstract (love). The next day a shorter list of novel words was presented and incidentally encoded before the subjects were scanned using fMRI while they incidentally encoded a third set of words. The key variable during the scan was that some of the words were from the first list (re-encoding with long-lag between presentations), some were from the second list (re-encoding with short-lag between presentations) and some were novel. Varying the lag in this way facilitated a comparison between different levels of priming during re-encoding, and a subsequent explicit memory test. The latter was performed two days later in the form of an old/new explicit recognition memory test for all of the previously seen words.