

Neural Mechanisms of Memory Updating

In collaboration with Attila Sik (School of Clinical and Experimental Medicine, University of Birmingham), we have been awarded a 3 year grant from the Biotechnology and Biological Sciences Research Council: "The neural mechanisms of memory updating".

Memories can be updated with new information, but little is known about the underlying mechanisms behind this process. New research has revealed a simple, yet powerful, approach for demonstrating memory updating. First, humans or experimental animals are taught that a stimulus predicts a fearful outcome. This fear memory is then updated by first reminding the subject to reactivate the memory, and then updating the memory by teaching the subject that the stimulus is now safe. This approach exploits the process of memory reconsolidation, which restabilises the reactivated memory, by introducing new information that is thought to become integrated into the restabilising memory. We will be studying the mechanisms by which this integration process takes place.

Effectively updating a fearful memory may well be useful in the treatment of anxiety disorders. However, it is currently not clear how we can ensure that the memory reminders will successfully reactivate the memory and trigger the reconsolidation process. The current project will also be testing the possibility that we can pharmacologically enhance memory reactivation in order to update memories that would otherwise not be modifiable.

News

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The first outcomes of this grant have recently been published in **Frontiers in Behavioral Neuroscience** (<http://journal.frontiersin.org/Journal/10.3389/fnbeh.2014.00144/abstract>). In this paper, we show that it is possible to stimulate contextual fear memories to destabilise even under behavioural conditions that do not normally engage memory reconsolidation.

This can be achieved by activating cannabinoid CB1 receptors in the dorsal hippocampus. We also show that it is not necessary to actually express the fear memory in order to allow it to destabilise. Instead the trace of the memory that exists in the hippocampus needs to be brought back to an active state and then can be destabilised and/or expressed.

These findings suggest that problematic fear memories (e.g. in posttraumatic stress disorder) might be beneficially manipulated in a reliable manner using pharmacological tools to ensure that the memory destabilises, perhaps even while preventing any adverse effects of recalling the traumatic memory.