The ability to memorize our daily life depends on a brain region called the hippocampus. Patients with damage to this region are unable to form new long-term memories of day-to-day events. Given its importance, the hippocampus has been the subject of a tremendous amount of research aimed at understanding how the hippocampal neuronal network supports memory. Writing on page 272 of this issue, Donato and colleagues provide insight into how recent experience can alter hippocampal memory functions. The main contribution of this work is the identification of bidirectional, experience-dependent modifications in a subset of hippocampal neurons that are associated with enhanced or impaired memory performance.

Neurons in the hippocampal network can be divided into two main categories: excitatory and inhibitory. Excitatory neurons are the main carriers of information and connect the hippocampal network with other brain regions. But their activity is not left unchecked: excitatory neurons are tightly regulated by the release of the neurotransmitter GABA from inhibitory neurons. In fact, it has been suggested that many of the extraordinary computational processes implemented in neuronal networks originate from the action and diversity of inhibitory neurons.

Donato et al. investigated the effect of previous experience on a group of inhibitory neurons that express the calcium-buffering protein parvalbumin. Although this group constitutes only some 5% of the hippocampal neuronal population, it is crucial for sustaining several types of rhythmic neural activity in the brain and has been implicated in numerous cognitive processes.

The authors assigned mice to one of two experimental conditions. One group of animals was housed in an enriched environment rather than the standard conditions common to most laboratories. The second group was subjected to contextual fear conditioning, which involves placing the animals in an environment where

**Figure 1 | Experience and alteration of parvalbumin-expressing neurons.** a, Donato et al. propose that an enriched environment and spatial learning can increase the number of inhibitory synaptic terminals (red) that make contact with parvalbumin-expressing neurons, leading to low parvalbumin expression. This affects hippocampal excitatory neurons downstream, resulting in enhanced memory. Excitatory synaptic terminals are shown in blue. The pink synaptic terminal is inhibitory, and expresses low levels of parvalbumin and the enzyme GAD67. b, By contrast, contextual fear conditioning and completion of learning increase the number of excitatory synaptic terminals that make contact with parvalbumin-expressing neurons, which is associated with high parvalbumin expression and, consequently, reduced recognition memory at the level of hippocampal neurons.
in parvalbumin expression, wherein contextual fear conditioning caused the opposite (Fig. 1). Intriguingly, parvalbumin expression was strongly correlated with that of the enzyme GAD67, which in inhibitory neurons transforms the amino acid glutamate into GABA. This effect of recent experience on parvalbumin levels seemed to be restricted to basket cells, a subgroup of parvalbumin-expressing neurons that makes synaptic connections predominantly onto the cell body of excitatory and other inhibitory neurons. Donato and co-workers propose that the change in parvalbumin expression resulted from a shift in the ratio of excitatory to inhibitory synaptic inputs onto basket cells.

The most surprising finding of this work is that experience-dependent changes in parvalbumin expression coincide with changes in memory functions. Mice with low parvalbumin expression following exposure to an enriched environment showed enhanced object-recognition memory, whereas those with high parvalbumin expression after fear conditioning displayed impaired memory. To gather further evidence of a role for parvalbumin expression level in memory, the authors used a pharmacogenetic approach to manipulate the activity of cells expressing parvalbumin. Inhibiting or activating parvalbumin-expressing neurons led to a decrease or increase in parvalbumin expression levels, respectively. These manipulations were associated with the expected behavioural outcome, such that experimentally controlled parvalbumin expression was negatively correlated with object-recognition memory.

Donato et al. also assessed parvalbumin expression levels during the course of spatial learning. They found that, in a navigation task, parvalbumin expression was low during the learning phase of the task, but shifted to high once learning was completed. Again, low and high parvalbumin expression were associated with enhanced and reduced object-recognition memory, respectively. These learning-related changes in parvalbumin expression were also accompanied by a modification of the synaptic inputs onto basket cells.

One of the most pressing questions in light of this work is how neuronal activity in the hippocampal network changes with variations in parvalbumin expression. Future studies should address the consequences of enriched environments and contextual fear conditioning on the activity of parvalbumin-expressing neurons within the hippocampal network. The activation of parvalbumin-expressing basket cells during high and low expression of parvalbumin could also be investigated in brain sections by measuring the response of these neurons after stimulation of input pathways. Given the anatomical synaptic alterations presented in this study, one expects modifications of the excitatory and inhibitory synaptic currents during high and low expression states of parvalbumin, respectively. Moreover, changes in the calcium-buffering capacity and in GAD67 levels should affect the synaptic output of parvalbumin-expressing neurons.

A second avenue that deserves further investigation relates to the link between recent experience and parvalbumin expression. Donato and co-workers suggest that the balance of excitatory and inhibitory synapses onto parvalbumin neurons is a key factor governing the expression of this protein. But what determines these changes in synaptic connectivity in the first place? Are they the result of specific activity patterns occurring in the hippocampus or are they instead controlled by neuromodulatory signals originating outside the hippocampus? Answers to these questions would complete the picture of how recent experience modulates learning and memory by altering the configuration of the parvalbumin-expressing neuronal network.

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At room temperature, iron atoms behave as identical sub-nanometre-sized bar magnets. Solid iron's crystal structure is such that if a piece of the metal — say, a nail — is placed in a magnetic field, the atomic magnets, which will tend to align along the field, will maintain their induced orientation even when the field is removed. The nail itself will thus become a big bar magnet (Fig. 1a). As a result, materials that hold magnetic ordering in the absence of an applied magnetic field are named after iron and termed ferromagnetic, or just magnetic for short. On page 237 of this issue, Mertelj et al. report the observation of

Figure 1 | Bar magnets and ferrofluids. a. In ferromagnetic iron, the magnetic moments of iron atoms, here illustrated as tiny bar magnets, maintain their mutual south–north orientation in the absence of a magnetic field to form a bigger, permanent bar magnet. Its magnetic moment is shown by the arrow. b. Ferrofluids, suspensions of sub-micrometre-sized magnetic particles, respond strongly to an applied magnetic field (B), forcing the fluid to form dramatic spikes (c) as a way of filling its volume with field. c. Photograph of a ferrofluid on a reflective glass plate and subjected to a strong magnetic field.

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