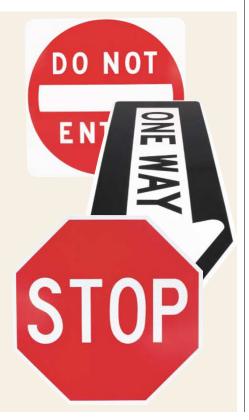
case, the reduced size of the hippocampus is probably conducive to an exaggerated hormonal and behavioural response to stress. This scenario would also explain why only a few people develop the disorder, despite the fact that many are exposed to the same trauma.

In the end, the relationship between stress and hippocampal size is probably a two-way street. In other words, the egg (stress) often comes first, but sometimes the chicken (a reduced hippocampus) has precedence. This should be taken as a sign that it is time to drop rhetorical conundrums about chickens and eggs in the face of evidence that nature and environment, brain and society, can often not be dissociated. After all, chicken and egg are one and the same animal.

Suzana Herculano-Houzel, Universidade Federal do Rio de Janeiro Comparison de Compari

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LEARNING AND MEMORY

Overcoming our fears

Animals — including humans can be conditioned to fear a neutral stimulus, such as a tone, if it is repeatedly paired with something unpleasant, such as a mild shock. Presenting the tone without the shock can diminish this conditioned fear; this process is called extinction. Since Pavlov's original conditioning studies, a key question has been whether this extinction process erases the memory for the original fear conditioning, or instead lays down a new memory that inhibits the fear. New research by Milad and Quirk provides evidence for the latter hypothesis and suggests that extinction memory might be controlled by the prefrontal cortex (PFC), a brain region long thought to be involved in executive control.

In this study, rats were first conditioned to show a fear response ('freezing') to a tone. Next, the rats were given extinction trials, during which the freezing responses diminished. Finally, they were tested for recall of extinction. The activity of neurons in the medial PFC was recorded during each of these stages. Neuronal firing in the PFC increased in response to the tone, but only during the recall of extinction. Furthermore, the rats

that showed the lowest levels of freezing (the best extinction) also exhibited the highest levels of PFC firing in response to the tones.

Electrical stimulation of the PFC during tone presentations reduced freezing in animals that did not experience extinction trials. In other words, artificially activating the PFC imitated extinction. PFC stimulation could also hasten the appearance of the extinction response in animals that received extinction training. Together with previous results showing that destruction of the PFC blocks extinction memory, but not the original conditioning or the learning of extinction, the data provide evidence for a role of the PFC in the storage of a new extinction memory that inhibits or replaces the original fear memory.

The authors propose that, during extinction training, neural inputs to the PFC from subcortical areas are enhanced, aiding in the formation and consolidation of extinction memory. Subsequent





exposure to the previously feared stimulus triggers increased neural activity in the PFC and suppresses the original fear response. So, new extinction memories stored in the PFC inhibit fearful memories that probably reside in subcortical structures such as the amygdala, and dampen the expression of fear-related behaviours. Clinically, the formation of extinction memory might provide a mechanism for alleviating fears that are associated with trauma or phobias. These results provide support for the development of strategies to treat phobias that focus on the PFC.

> Anne Marie Brady, Albany Medical College

S References and links

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