

IMAGERY

Imagine...

Our ability to imagine or to recall images into our 'mind's eye' has played a significant role in the evolution and development of human culture by freeing us from the confines of the immediate present to allow us to plan, speculate and reflect. John Lennon even wrote a song to encourage it. The neural basis of visual recall, and whether it relies on the same mechanisms as visual perception, has long been a topic of interest to neuroscientists and psychologists.

Several recent studies have explored this issue using a variety of experimental procedures. Kreiman, Koch and Fried recorded the activity of neurons in various areas of the medial temporal lobes while subjects either viewed or imagined previously viewed visual images. This experiment capitalized on a rare opportunity to monitor the firing patterns of neurons in the human brain. The subjects were all patients with pharmacologically intractable epilepsy who were implanted with chronic electrodes to localize the seizure foci in preparation for surgical intervention. Single neurons in the hippocampus, amygdala, entorhinal cortex and parahippocampal gyrus selectively altered their firing rates depending on the stimulus the subjects were imagining. Three types of selective neuron were observed. Some neurons were selective for visual input but not imagery, others were selective only during visual recall. Perhaps the most interesting neurons were those that responded during both vision and imagery. Most of these neurons had identical selectivity — if they responded to pictures of faces but not pictures of animals, they also fired when the subject imagined faces but not when they imagined animals. Although it is still unclear what the activity in these neuronal populations represents, these data do reveal a neural correlate of volitional visual imagery at the single cell level and suggest a common substrate for the processing of visual information and visual recall.



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These results are broadly consistent with a recent event-related-fMRI study by Wheeler, Petersen and Buckner in which normal subjects learned a set of visual and auditory items, each of which was paired with a descriptive label. For example, the label DOG was paired with an image of a dog for half of the subjects and the sound of a dog barking for the others. Subsequently, scanning was performed while the subjects were presented with either the images or sounds, or were asked to recall the appropriate images or sounds to mind in response to the presentation of the label. The results indicate that the areas of the brain involved in late rather than early processing of visual or auditory information were transiently active during vivid visual or auditory recall, respectively.

These studies and others are now beginning to probe the neural basis of imagery and perception at several different levels. The consistent finding is that some of the same information processing pathways involved in the perception of sensory information are also involved during retrieval of that information for use in imagery.

Peter Collins

References and links

ORIGINAL RESEARCH PAPERS Kreiman, G. *et al.* Imagery neurons in the human brain. *Nature* **408**, 357–361 (2000) | Wheeler, M. *et al.* Memories echo: vivid remembering reactivates sensory-specific cortex. *Proc. Natl Acad. Sci. USA* **97**, 11125–11129 (2000)
 FURTHER READING Kreiman, G. *et al.* Category-specific visual responses of single neurons in the human medial temporal lobe. *Nature Neurosci.* **3**, 946–953 (2000) | Kosslyn, S. M. *Image and the Brain* (MIT Press, Cambridge, Massachusetts, 1994)
 WEB SITES Itzhak Fried | Christof Koch | Randy Buckner

IN BRIEF

SYNAPTIC PHYSIOLOGY

Dendritic release of glutamate suppresses synaptic inhibition of pyramidal neurons in rat neocortex.

Zilberter, Y. *J. Physiol.* **528**, 489–496 (2000)

Dendritic release of GABA can inhibit presynaptic glutamate release between pyramidal cells and bitufted interneurons in the neocortex. The story has now come full circle, as Zilberter shows that glutamate released from pyramidal cell dendrites can inhibit GABA release from fast-spiking interneurons by activation of presynaptic metabotropic glutamate receptors. As both examples of retrograde signalling would tend to favour excitation over inhibition in the cortex, it is possible that alterations of this process might contribute to the generation of epileptic activity.

ADDICTION

Reduced operant ethanol self-administration and *in vivo* mesolimbic dopamine responses to ethanol in PKC ϵ -deficient mice.

Olive, M. F. *et al. Eur. J. Neurosci.* **12**, 4131–4140 (2000)

The authors explored the molecular mechanisms of ethanol reinforcement by studying the effects of a null mutation in PKC ϵ . Mice lacking PKC ϵ showed reduced ethanol self-administration. In addition, dopamine levels in the nucleus accumbens of the mutant mice were not increased by the acute administration of ethanol. These findings raise the possibility that selective PKC ϵ antagonists could be used to treat alcohol abuse.

MEMORY

Auditory fear conditioning increases CS-elicited spike firing in lateral amygdala neurons even after extensive overtraining.

Maren, S. *Eur. J. Neurosci.* **12**, 4047–4054 (2000)

The basolateral amygdala (BLA) is involved in the acquisition and storage of fear conditioning, but it is not clear whether BLA is a temporary or a permanent storage site. Maren overtrained rats in a fear-conditioning task and recorded action potential firing in the BLA upon presentation of the conditioned stimulus. Increase in firing commonly seen after conditioning also occurred in the overtrained animals, indicating that the BLA is indeed a long-lasting storage site of fear conditioning.

NEURODEGENERATION

Binding of disease-associated prion protein to plasminogen.

Fischer, M. B. *et al. Nature* **408**, 479–483 (2000)

The structural discrimination between PrP^C and its abnormal variant PrP^{Sc} has remained problematic. The authors show that plasminogen can distinguish between the two proteins by binding selectively to PrP^{Sc}, and have defined some of the structural determinants that are important for binding. Plasminogen might therefore be useful for the diagnosis of prion-related diseases.