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There are no translations available.

Latest on Learning & Memory: Some of the Most Recent Research & Discoveries

Study Examines Memory, Learning and Aging

In a recent Psychology and Aging study funded by the National Institute on Aging, Dunlosky and colleagues examined whether aging affects metacomprehension, or the ability to judge your own comprehension and learning of text materials.

They found that while judgments for both younger and older adults were significantly related to how easily they could process the information, the difference between the two groups did not differ significantly. Thus, aging does not seem to affect people’s ability to judge their own learning of text materials.

These findings support the idea that if people have the ability to self-evaluate their learning process accurately, they also can regulate their learning more effectively. To help healthy adults aged 60-75 years old, Dunlosky and colleagues have created a memory intervention program that helps them learn more quickly and competently using self-evaluation tools.

Discovery of Cell Linked to Learning and Memory

Queensland Brain Institute (QBI) neuroscientists at The University of Queensland have discovered a fundamental component of the process that regulates memory formation.

QBI Director Professor Perry Bartlett said the discovery explains, for the first time, how new nerve cells form in an area of the brain associated with learning and memory – which is known to deteriorate in people with stroke and dementia.

"The hippocampus is a region of the brain involved in important brain functions such as learning and memory and loss of neuronal production in the hippocampus is associated with a range of neurodegenerative conditions, and is particularly evident in ageing dementia." Professor Bartlett said.

"Surprisingly, however, studies have so far failed to identify a resident stem cell population in the hippocampus that's capable of providing the renewable source of these essential nerve cells."

Research by Professor Bartlett and his QBI colleague Dr Tara Walker – which features in this week's Journal of Neuroscience (May 14/08) – has identified the resident stem cell in the hippocampus and, even more importantly, has discovered how it can be activated to produce new neurons.

According to Dr Walker, an understanding of the activation process should enable the development of therapeutics that can stimulate the production of new neurons and reverse or prevent the cognitive decline that occurs during ageing dementia.

It has long been known that learning stimulates this production; not only in the hippocampus but in other parts of the brain. But we have to redefine 'learning' as a particular process that must be done in a particular way. Trying to hammer facts and figures into the brain top down will not be effective (indeed, this can slow down other abilities such as imagination and creativity). You can find out more about the process of learning, brain plasticity, and how to get the best performance out of your brain in the tutorials.

Genetic Enhancement of Learning and Memory.

Hebb's rule (1949) states that learning and memory are based on modifications of synaptic strength among neurons that are simultaneously active. This implies that enhanced synaptic coincidence detection would lead to better learning and memory. If the NMDA (N-methyl-D-aspartate) receptor, a synaptic coincidence detector, acts as a graded switch for memory formation, enhanced signal detection by NMDA receptors should enhance learning and memory. Here we show that overexpression of NMDA receptor 2B (NR2B) in the forebrains of transgenic mice leads to enhanced activation of NMDA receptors, facilitating synaptic potentiation in response to stimulation at 10-100 Hz. These mice exhibit superior ability in learning and memory in various behavioural tasks, showing that NR2B is critical in gating the age-dependent threshold for plasticity and memory formation. NMDA-receptor-dependent modifications of synaptic efficacy, therefore, represent a unifying mechanism for associative learning and memory. Our results suggest that genetic enhancement of mental and cognitive attributes such as intelligence and memory in mammals is feasibleAMPA receptor trafficking at excitatory synapses.

Excitatory synapses in the CNS release glutamate, which acts primarily on two sides of ionotropic receptors: AMPA receptors and NMDA receptors. AMPA receptors mediate the postsynaptic depolarization that initiates neuronal firing, whereas NMDA receptors initiate synaptic plasticity. Recent studies have emphasized that distinct mechanisms control synaptic expression of these two receptor classes. Whereas NMDA receptor proteins are relatively fixed, AMPA receptors cycle synaptic membranes on and off. A large family of interacting proteins regulates AMPA receptor turnover at synapses and thereby influences synaptic strength. Furthermore, neuronal activity controls synaptic AMPA receptor trafficking, and this dynamic process plays a key role in the synaptic plasticity that is thought to underlie aspects of learning and memory.

Molecular Motor Tied to Memory

Scientists believe that recording memories involves a process, called long-term potentiation (LTP), that enhances communication between pairs of neurons. Neurons communicate by releasing neurotransmitters that stimulate receptors on their neighbors, and LTP triggers more receptors to accumulate at the receiving cell's membrane--making it more sensitive to incoming messages.

Previous research had suggested that actin and myosin, two proteins with key roles in muscle contraction, might play a role in clustering receptors in neurons. To investigate this possibility, Michael Ehlers, a neurobiologist at Duke University Medical Center in Durham, North Carolina, and his colleagues used time-lapse imaging and biochemical methods to examine brain slices from rats. These experiments revealed what happens after an incoming signal triggers a rush of calcium into a neuron. The calcium activates a myosin protein called myosin Vb, prompting it to latch onto packets of receptors stored deeper in cells and drag them out to the neuron's signaling site, where the receptors can receive neurotransmitters and contribute to LTP.

To make sure that myosin Vb was indeed the motor that made the learning process possible, Ehlers and his colleagues chemically inhibited myosin Vb in neurons. The cells were incapable of generating LTP, the researchers report today in Cell. "We were surprised that one motor molecule could account for the bulk of membrane trafficking events," says Ehlers. "It may in fact be a motor that makes memories."

"What they've done is connect a lot of dots," says Marie Wooten, a neurobiologist who studies cell signaling at Auburn University in Alabama. The paper shows step by step how neurons move receptors to their outer membrane during LTP, Wooten says. Bettina Winckler, a neuroscientist at the University of Virginia, Charlottesville, agrees. "This paper is like a jewel," says Winckler--or, perhaps, a well-engineered motor. "It all fits everything together so beautifully," she says.

Sources

(memory, learning & aging)

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