

Study provides potential explanation for mechanisms of associative memory

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Researchers from the University of Bristol have discovered that a chemical compound in the brain can weaken the synaptic connections between neurons in a region of the brain important for the formation of long-term memories. The findings, published today in the *Journal of Neuroscience*, may also provide a potential explanation for the loss of memory associated with Alzheimer's.

Acetylcholine, a neurotransmitter, is released in the brain and is known to play an important role in normal brain functions such as sleep, attention, and learning and memory. Until now the mechanisms by which this transmitter controls such processes were not well understood.

The findings, led by researchers from the University's MRC Centre for Synaptic Plasticity in the School of Physiology and Pharmacology, highlight the mechanisms by which acetylcholine controls communication between neurons located in the prefrontal cortex and may help in understanding how higher cognitive processing is controlled in this important brain area.

Professor of Cellular Neuroscience, Zafar Bashir and his team have demonstrated how electrical stimulation of the prefrontal cortex leads to the release of acetylcholine from synaptic terminals and the subsequent weakening of synaptic connections between neurons.

When acetylcholine is released it binds to specific receptors and starts a molecular cascade which triggers physiological alterations in how

prefrontal [cortical neurons](#) are 'wired' together. The findings suggest that the persistent weakening of [synaptic connections](#) between neurons induced by the endogenous release of acetylcholine in the prefrontal cortex may underlie the formation of new associative memories.

The authors speculate that the memory impairments associated with Alzheimer's dementia may result, in part, from a loss of synaptic plasticity in the prefrontal cortex related to the depletion of brain acetylcholine that occurs in the disease.

Dr Douglas Caruana, who carried out the experiments, said: "Disruptions in cholinergic signaling in the [prefrontal cortex](#) are known to affect how the brain encodes lasting associations between objects and places, and a depletion of brain acetylcholine levels in the cortex is a classic hallmark of Alzheimer's dementia'."

Professor Bashir added: "Acetylcholinesterase inhibitors are the most widely used medication to treat individuals with Alzheimer's dementia and the enhancement of synaptic plasticity by acetylcholinesterase inhibition that we now demonstrate may be a way in which these drugs provide clinical efficacy."

More information: The research, entitled 'Induction of activity-dependent LTD requires muscarinic receptor activation in medial prefrontal cortex' by Professor Zafar Bashir, Dr Clea Warburton and Dr Douglas Caruana is published in the *Journal of Neuroscience*.

Provided by University of Bristol

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