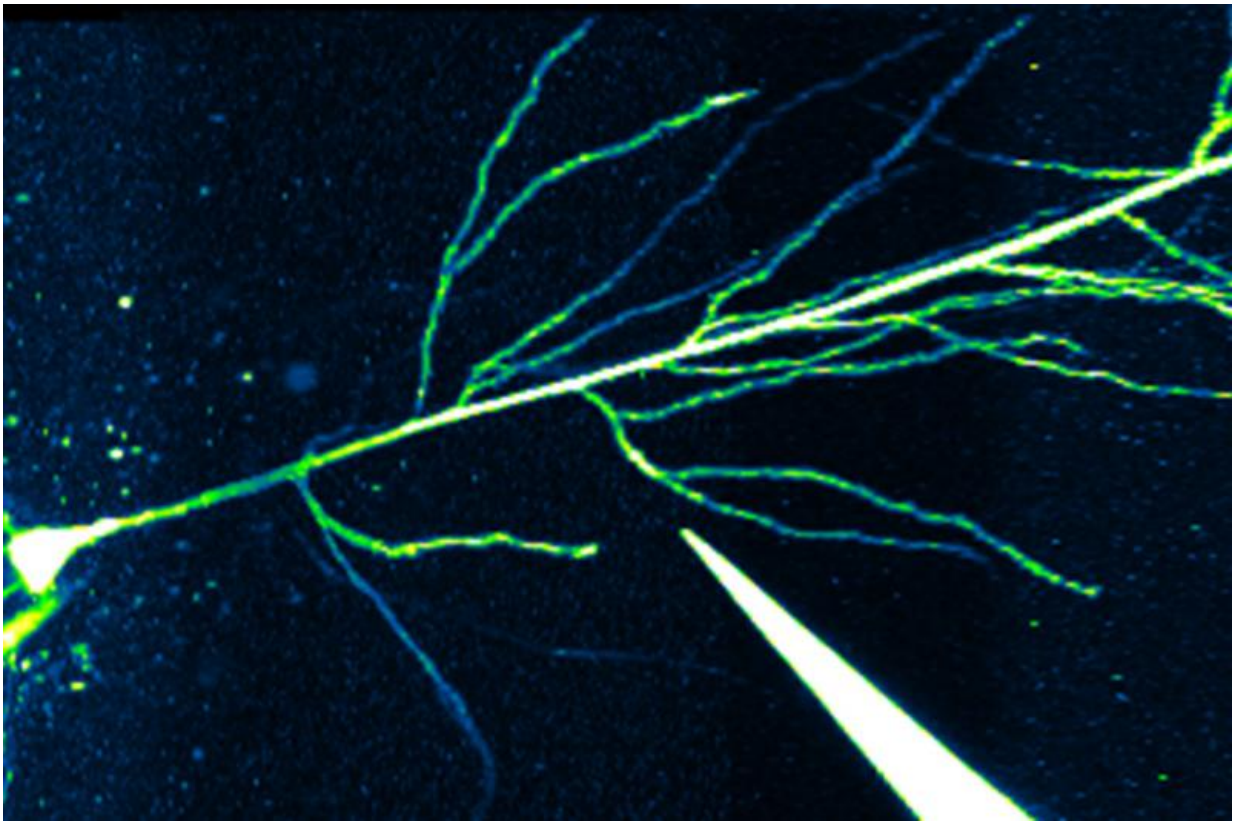


How drugs can help your brain encode memories

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Medical researchers at the University of Bristol have uncovered a fundamental mechanism that explains the interaction between brain state and the neural triggers responsible for learning. The discoveries, made

by researchers in collaboration with the pharmaceutical company Eli Lilly & Co., could lead to new ways of boosting cognitive function to counteract the effects of diseases such as Alzheimer's, as well as enhancing memory in healthy people.

The findings, published today in the journals *Nature Communications* and *Cerebral Cortex*, identify fundamental processes occurring at the microscopic connections between nerve cells, which involve an exquisite regulation of calcium ions by the neurotransmitter acetylcholine.

This neurotransmitter is released in the brain during learning and is critical for the acquisition of new memories. Currently, the only effective treatment for the symptoms of cognitive or memory impairment seen in diseases such as Alzheimer's is through the use of drugs that boost the amount of acetylcholine release.

Lead researcher Dr Jack Mellor, from Bristol's Centre for Synaptic Plasticity, said: 'These findings are about the fundamental processes that occur in the brain during the encoding of memory and how they may be regulated by brain state or drugs for the treatment of dementia. They are not going to revolutionise the treatment of Alzheimer's disease or other forms of memory impairment overnight, but given the priority for research into aging and dementia at national and international levels, we expect many more advances in the next few years.'

Dr Mellor added: 'From a therapeutic point of view, these studies suggest certain drugs that act on specific acetylcholine receptors may be highly attractive as potential treatments for dementia and other cognitive disorders. Currently, the only effective treatments for patients with Alzheimer's disease are drugs that boost the effectiveness of naturally-released acetylcholine. We have shown that mimicking the effect of acetylcholine at specific receptors facilitates changes in the strength of connections between nerve cells. This has the potential to be beneficial

for patients suffering from Alzheimer's disease or schizophrenia."

More information: Cezar M. Tigaret et al. Coordinated activation of distinct Ca²⁺ sources and metabotropic glutamate receptors encodes Hebbian synaptic plasticity, *Nature Communications* (2016). DOI: [10.1038/NCOMMS10289](https://doi.org/10.1038/NCOMMS10289)

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Facilitation of Long-Term Potentiation by Muscarinic M1 Receptors Is Mediated by Inhibition of SK Channels. *Neuron*. DOI: [dx.doi.org/10.1016/j.neuron.2011.02.030](https://doi.org/10.1016/j.neuron.2011.02.030)

Provided by University of Bristol

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