

Potential Alzheimer's medication shows promise in mouse model of neurodegenerative disease

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Memory loss and other cognitive symptoms of Alzheimer's disease are attributed, in part, to the degeneration of acetylcholine-producing neurons. Acetylcholinesterase inhibitors are a common treatment for patients with Alzheimer's; however, in spite of their clinical benefits, these non-selective medications are also associated with numerous adverse effects. It has been hypothesized that more selective targeting of acetylcholine signaling may reduce the side effects associated with current Alzheimer's medications, but it's not known whether improving selectivity could decrease the treatment's efficacy.

This month in the *JCI*, work led by Andrew Tobin at the University of Leicester tested two drugs that specifically target the M1 muscarinic acetylcholine receptor in a [mouse model](#) of neurodegeneration and discovered that the treatments had promising effects.

The mouse model showed many hallmarks of human Alzheimer's disease, including [memory deficits](#) and progressive hippocampal neuron degeneration.

Treatment with the M1-selective medications reversed memory deficits and profoundly extended the lifespan of the diseased mice. These findings support the concept that more specific drugs can be effective in treating the [cognitive symptoms](#) of neurodegenerative disease.

Future work is needed to determine whether these potential medications are effective in humans, and whether the improved targeting actually leads to fewer side effects.

More information: Sophie J. Bradley et al, M1 muscarinic allosteric modulators slow prion neurodegeneration and restore memory loss, *Journal of Clinical Investigation* (2016). [DOI: 10.1172/JCI87526](https://doi.org/10.1172/JCI87526)

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