Uncovering potential pathway to slowing Alzheimer's

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If we can overcome the loss of a process in the brain called "RNA editing," we may be able to slow the progress of Alzheimer's disease and other synaptic disorders, a new study has shown.

RNA editing is a genetic mechanism that modifies proteins essential in the connection between nerve cells in the brain, called synapses. RNA editing is deregulated in the brains of people with Alzheimer's disease, but whether this can cause disease is unknown.

In this study, the scientific team at the University of Technology Sydney Centre for Neuroscience & Regenerative Medicine (CNRM) replicated this deregulated process in the brains of mice, and discovered it led to the loss of synapses, as occurs in Alzheimer's.

The findings, published in the journal *Molecular Brain*, could have implications for a new way forward for ultimately treating Alzheimer's disease, says Professor Bryce Vissel, senior author of the study.

"Understanding mechanisms leading to synapses loss is essential to understand how patients suffering from Alzheimer's disease start losing their memory capacities and how to prevent this from happening," Professor Vissel says.

"Many scientists consider that Alzheimer's results from the build-up of a substance called amyloid in the brain. Consequently, they've focused their studies on removing amyloid. However, the most important event is actually the loss of connections between nerve cells called synapses which are known to be essential for memory formation.

"Our study is extremely important because we now have shown a mechanism that can lead to loss of synapses as occurs in Alzheimer's disease."

Dr. Gary Morris, a scientist who contributed to the study, says that because "synapses are important for learning, the loss of these synapses leads to memory loss."

"Our study suggests that if we can overcome the loss of RNA editing in the brain, we may potentially be able to slow the disease."

Professor Vissel says the team's next step is to see if they can rescue synapses and memory deficits in Alzheimer's disease by overcoming the loss of RNA editing in the Alzheimer's brain.

"We have good reason to think that this could ultimately be a highly beneficial approach for solving Alzheimer's and potentially other neurodegenerative diseases such as Parkinson's."

More information: Lyndsey M. Konen et al. A new mouse line with reduced GluA2 Q/R site RNA editing exhibits loss of dendritic spines, hippocampal CA1-neuron loss, learning and memory impairments and NMDA receptor-independent seizure vulnerability, *Molecular Brain* (2020). DOI: 10.1186/s13041-020-0545-1

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