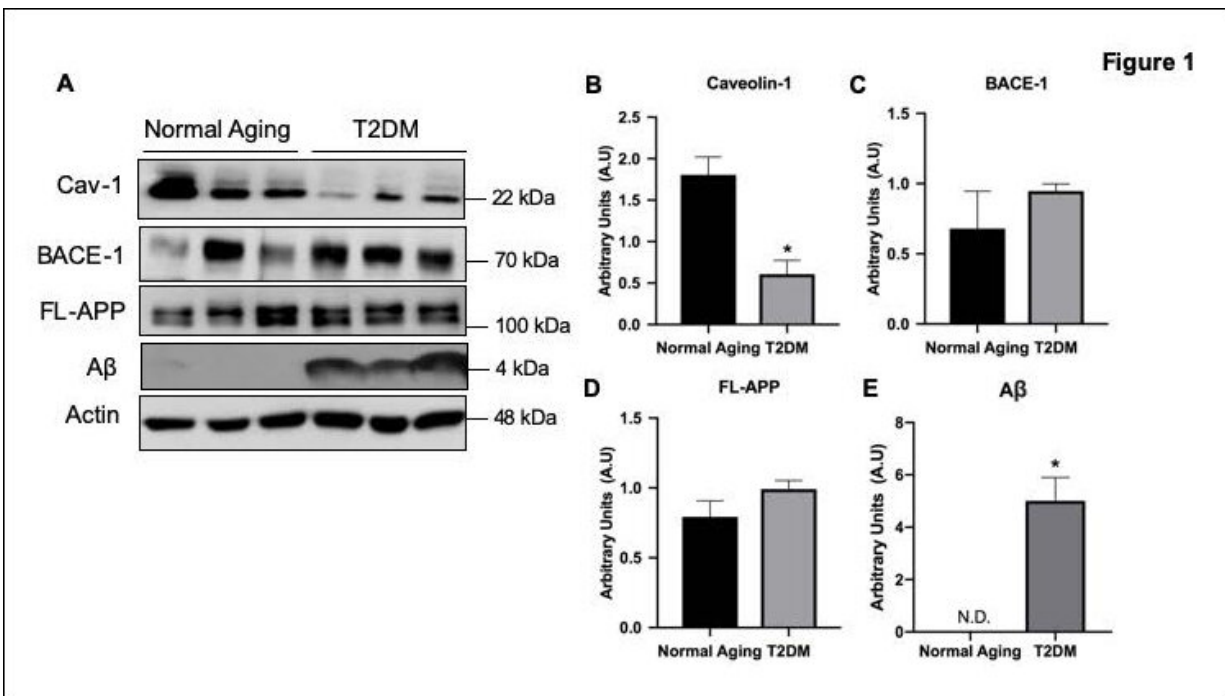


Potential target for diabetes-associated Alzheimer's disease

September 16 2019



Depletion of Caveolin-1 in Type-2 Diabetes Model Induces Alzheimer's disease Pathology Precursors Credit: Bonds et al., *JNeurosci*(2019)

Researchers have identified a protein that may contribute to the progression of Alzheimer's disease pathology in type-2 diabetes, reports a new study of male mice and human brain tissue. The research, published in *JNeurosci*, could have implications for future drug development.

The cause of sporadic, late onset Alzheimer's disease is unknown. However, [type-2-diabetes](#) is associated with an increased Alzheimer's risk, which may provide a clue to its origin.

Bonds et al. examined the relationship between diabetes and Alzheimer's disease and found that a protein called caveolin-1 (Cav-1) is depleted in the temporal lobe of humans with diabetes and in a diabetic mouse model.

Depletion of Cav-1 causes the upregulation of amyloid precursor protein and b-amyloid levels.

Importantly, restoring Cav-1 levels in mice reduced Alzheimer's pathology and improved learning and [memory deficits](#), revealing a potential mechanism responsible for the increased risk of Alzheimer's disease in this population.

More information: Depletion of Caveolin-1 in Type-2 Diabetes Model Induces Alzheimer's disease Pathology Precursors, *JNeurosci* (2019). [DOI: 10.1523/JNEUROSCI.0730-19.2019](https://doi.org/10.1523/JNEUROSCI.0730-19.2019)

Provided by Society for Neuroscience

Citation: Potential target for diabetes-associated Alzheimer's disease (2019, September 16) retrieved 22 April 2023 from <https://medicalxpress.com/news/2019-09-potential-diabetes-associated-alzheimer-disease.html>

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