Alzheimer's disease is associated with removal of the synaptic protein ADAM10

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Alzheimer's disease is characterized by the accumulation of neurotoxic β-amyloid peptide (A-beta). ADAM10, a protein that resides in the neural synapses, has previously been shown to prevent the formation of A-beta.

In this issue of the *Journal of Clinical Investigation*, Monica Di Luca and colleagues at the University of Milan in Milan, Italy, report that ADAM10 is removed from synapses through association with the protein AP2.

Strikingly, the association between ADAM10 and AP2 was increased in human brain homogenates from Alzheimer's disease (AD) patients compared to healthy controls.

Neuronal activity was shown to influence the level and activity of ADAM10 in synapses and its association with AP2.

These studies identify pathological mechanisms associated with AD that control the localization of proteins at the synapse.

More information: Endocytosis of synaptic ADAM10 in neuronal plasticity and Alzheimer's disease *J Clin Invest*. doi:10.1172/JCI65401

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