Study indicates that the hippocampus mediates cognitive decline in Huntington's disease

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Huntington's disease is a neurodegenerative disorder that results in involuntary spastic movement and loss control of voluntary motor function. Patients also exhibit a decline in cognitive ability. The defects in learning and memory associated with Huntington's disease have been ascribed to pathology in the frontal lobe of the brain.

A new study in the *Journal of Clinical Investigation* suggests that alterations in the hippocampus contribute to memory dysfunction in Huntington's disease.

Silvia Gines and colleagues at the University of Barcelona found increased expression of a protein, $p75^{NTR}$, in the hippocampus of Huntington's disease patients and mouse models of this disorder. $p75^{NTR}$ is known to reduce dendritic spine density, which is associated with memory and learning defects. Reduction of $p75^{NTR}$ in mouse models of Huntington's disease prevented the cognitive decline and maintained spine density.

Moreover, over expression of $p75^{NTR}$ in the hippocampus of wild type mice recapitulated memory and learning defects associated with Huntington's disease.

The results of this study recognize a potential role of the hippocampus in the development of Huntington's disease.

More information: Neurotrophin receptor p75NTR mediates Huntington's disease–associated synaptic and memory dysfunction, *J Clin Invest*. DOI: 10.1172/JCI74809