

# How toxic protein spreads in Alzheimer's disease

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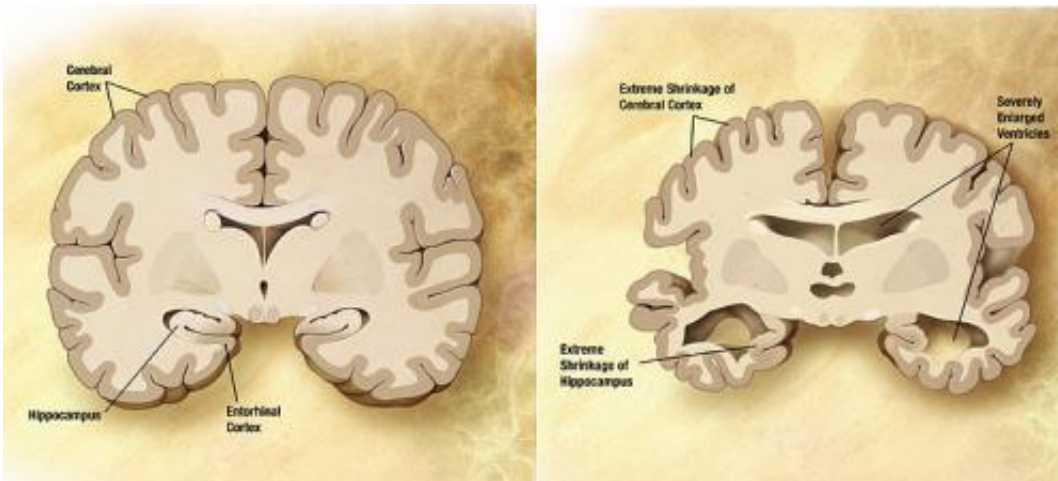


Diagram of the brain of a person with Alzheimer's Disease. Credit: Wikipedia/public domain.

Toxic versions of the protein tau are believed to cause death of neurons of the brain in Alzheimer's disease. A new study published in *Nature Communications* shows that the spread of toxic tau in the human brain in elderly individuals may occur via connected neurons. The researchers could see that beta-amyloid facilitates the spread of toxic tau.

The present study is a collaboration between Lund University in Sweden and McGill University in Canada, and provides information on how toxic tau spreads in the [human brain](#).

"Our research suggests that toxic tau may spread across different brain regions through direct neuronal connections, much like [infectious diseases](#) may spread to different cities through different transportation pathways. The spread is restricted during normal aging, but in Alzheimer's [disease](#) the spread may be facilitated by beta-amyloid, and likely leads to widespread neuronal death and eventually dementia," says lead author Jacob Vogel from McGill University.

"I think these findings have implications for therapies aiming at stopping the spread of tau and thereby halting the [disease progression](#) in Alzheimer's," says Oskar Hansson, professor of neurology at Lund University and co-lead investigator of the study.

There are two proteins that are known to be linked to Alzheimer's disease—beta-amyloid, which forms what is known as a plaque in the brain, and tau, which forms tangles within brain cells. Previous studies have linked the spread of toxic tau, in particular, to degeneration of the brain and symptoms such as memory impairment.

Intense research is ongoing to better understand how toxic tau spreads in the [brain](#), in order to develop new therapies that can stop the spread and thereby stop the disease. Ongoing clinical trials are currently evaluating whether antibodies developed to bind to tau might stop the disease.

"Our findings have implications for understanding the disease, but more importantly for the development of therapies against Alzheimer's, which are directed against either [beta-amyloid](#) or tau. Specifically, the results suggest that therapies that limit uptake of tau into the neurons or transportation or excretion of tau, could limit disease progression," says Oskar Hansson.

**More information:** undefined undefined et al. Spread of pathological tau proteins through communicating neurons in human Alzheimer's

disease, *Nature Communications* (2020). DOI: [10.1038/s41467-020-15701-2](https://doi.org/10.1038/s41467-020-15701-2)

Provided by Lund University

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