

New findings suggest new ways to diagnose and treat Alzheimer's

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A team of researchers at UMass Lowell has found a new mechanism by which a key protein associated with Alzheimer's disease can spread within the human brain.

The research, led by UMass Lowell biological sciences professor Garth Hall, gives new hope that the disease may someday be cured. It provides a new explanation of how the protein tau, a normal human protein that becomes toxic in Alzheimer's patients, can appear in their [cerebrospinal fluid](#) (CSF). The findings of the team from the UMass Lowell Center for Cellular Neuroscience and [Neurodegeneration](#) Research appear in the February issue of the *Journal of Alzheimer's Disease*.

"My team has discovered two different ways in which tau is secreted by neurons, or [brain cells](#)," said Hall, who has spent more than 20 years studying Alzheimer's on the cellular level using larval sea lampreys as a model system. "This might explain how tau-containing lesions seem to propagate between adjacent, interconnected parts of the brain during the development of the disease."

Until very recently, it was universally assumed by scientists that tau is never secreted from or transferred between neurons, and that CSF-tau only appears after many neurons have died and irreversible harm has been done to the [brain](#).

"That tau secretion can occur via two distinct mechanisms strongly indicates that it is biologically 'real' and is not just [tau protein](#) leaking out

of dead neurons," said Hall. "The fact that it occurs in a pattern that reproduces what is seen in the CSF of Alzheimer's patients holds out hope that patients in early stages of the disease might someday be cured. If we can distinguish secreted tau from tau that is released from dying neurons in CSF samples, then maybe we can diagnose Alzheimer's in time to stop the disease before the [neurons](#) die."

Provided by IOS Press

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