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Team discovers a way to potentially slow down Alzheimer's disease

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Researchers at the University of Texas Medical Branch at Galveston have discovered a way to potentially halt the progression of dementia caused by accumulation of a protein known as tau.

Normally, tau protein is involved in microtubule formation, which acts as a brain cell's transportation system for carrying nutrients in and waste out. In the absence of tau protein, brain cells become dysfunctional and eventually die.

In many forms of dementia, such as Alzheimer's disease and chronic traumatic encephalopathy caused by multiple concussions, the tau protein starts behaving badly and instead of performing its normal cellular functions, it begins accumulating and interfering with cell-to-cell communications.

Without the ability of brain cells to receive signals, they become severely dysfunctional and if enough of them die in a given area of the brain, the result is cognitive impairment, which means difficulty in planning tasks and remembering things. This accumulation of tau results in the formation of tau oligomers (oligo- meaning "many"), the toxic form of tau protein.

Scientists believe that if you can get rid of this toxic oligomeric tau protein, you can potentially stop the spread of tau-related dementia. The trick is to remove the toxic oligomeric tau without also removing the normal, functional tau protein.

Researchers at UTMB did just that and their findings, just published in the *Journal of Neuroscience*, demonstrated that treatment with their tau oligomer-specific monoclonal antibody, called TOMA, in experiments involving a rodent model of tauopathy (tau-related dementia) improved locomotor function and performance on memory tests.

The TOMA antibody sticks to the oligomeric tau so it can no longer interfere with cell-to-cell communication, but leaves the native tau protein intact.

"This is significant because this research describes a very promising vaccination strategy for Alzheimer's disease, which could prevent memory loss from occurring later in life. No safety concerns were detected in mice receiving this treatment, but more research is needed to confirm the efficacy and safety of immunization in other animals and in humans," said UTMB neurology professor Rakez Kayed, member of the Sealy Center for Vaccine Development and senior author of this study.

Recently, this group also found the TOMA antibody effective in halting the spread of toxic tau aggregates in a paper published in the Journal of Alzheimer's Diseases.

Diana Castillo-Carranza, postdoctoral scientist in the Kayed lab and lead author of both studies, said, "New evidence suggests that in Alzheimer's disease the pathology is spread through the brain from one area to another by oligomeric tau. Here we are showing that TOMA antibody has the ability to protect the brain from tau toxic aggregates."

Provided by University of Texas Medical Branch at Galveston