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Scientists study link between amyloid beta peptide levels and Alzheimer's disease

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The effects of the bacterial endotoxin lipopolysaccharide (LPS) has been found to elevate amyloid beta (Aβ) peptide levels in the brain, leading to short-term deficits in learning.

"We have found that after several injections with LPS toxin, (seven consecutive days of LPS administration) mice showed significant elevation in Aβ levels in their brains," said Michael Chumley, assistant professor of biology at Texas Christian University in Fort Worth. "This elevation corresponds with a decreased ability to learn simple tasks."

Scientists have long thought that inflammation might stimulate the onset of Alzheimer's or other cognitive diseases. It is well known that one of the things that cause inflammation in the brain is Aβ plaques, which are produced in response to inflammation. This leads to a self-perpetuating inflammation, where the product of inflammation leads to more inflammation. Scientists continue to research just how these Aβ plaques cause inflammation that leads to diminished brain function.

"What is unique in our study is that we initiated the inflammation in the body of the animal and not in the brain, and it lead to both an elevation of Aβ in the brain and learning deficits," said Chumley. "Our next step will be to determine if it is the Aβ produced in response to the inflammation that is the cause of the short-term memory problems."

"In over 90% of all Alzheimer's disease cases, the so called sporadic AD, the cause is unknown. It is likely that many factors, including genetic,

environmental, etc., play a role in the onset of AD. We believe that peripheral inflammation (inflammation outside of the brain and spinal cord), may also be one of these factors," said Chumley.

This research was published in *Journal of Behavioural Brain Research* in January 2012.

Provided by Texas Christian University in Fort Worth

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