2nd member in Alzheimer's toxic duo identified

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Like two unruly boys who need to be split up in class, a pair of protein molecules work together to speed up the toxic events of Alzheimer's disease. Researchers at the UT Health Science Center San Antonio today announced the discovery of the second molecule and said its identification could lead to drugs that disrupt the interaction, and thereby block or slow Alzheimer's onset or progression.

Alzheimer's disease is an irreversible, progressive brain disease marked by deterioration of nerve cells and eventual complete loss of cognitive functioning – thinking, memory and reason. Many Alzheimer's patients have brain lesions called amyloid plaques, which consist of protein fragments called amyloid-beta. Small aggregates of amyloid-beta are thought to contribute prominently to the degeneration of brain cells in Alzheimer's.

How genes are activated

The discovery involves an amyloid beta fragment called AICD. Scientists have known that AICD controls expression of genes that contribute to Alzheimer's, but how it did so was unclear – until now. "We discovered a protein molecule that communicates with AICD to turn on target genes," said Thomas G. Boyer, Ph.D., professor of molecular medicine at the Health Science Center. "We hope to exploit this knowledge to identify compounds or drugs that can disrupt these signals, leading to a novel and effective treatment for this disease."

Alzheimer's disease is the most common cause of dementia among older people, and estimates indicate that as many as 5.3 million Americans suffer from it. While several drugs approved by the U.S. Food and Drug Administration can temporarily slow worsening symptoms, no treatment is currently available to slow or stop the degeneration of nerve cells that lies at the root of the disease.

More information: The finding is in the Feb. 4 issue of *EMBO Reports*.

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