

Link found between chronic inflammation and risk for Alzheimer's disease

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While it is widely shown that possessing the ApoE4 Provided by Boston University School of Medicine

gene is the major genetic risk factor of Alzheimer's disease (AD), not all ApoE4 carriers develop AD. For the first time, researchers at Boston University School of Medicine (BUSM) have shown that ApoE4 linked with chronic inflammation dramatically increases the risk for AD. This can be detected by sequential measurements of Creactive protein, a common clinical test which can be could be done routinely in a clinical setting.

"Finding out what mediating factors for ApoE4 increase AD risk is important for developing intervention and prevention of the disease," explained corresponding author Wendy Qiu, MD, Ph.D., associate professor of psychiatry and pharmacology & experimental therapeutics at BUSM. "Since many elders have chronic low-grade inflammation after suffering from common diseases like cardiovascular diseases, diabetes, pneumonia and <u>urinary tract infection</u>, or after having surgeries, rigorously treating chronic systemic inflammation in ApoE4 carriers could be effective for prevention of Alzheimer's dementia."

Using data from the Framingham Heart Study which includes more than 3,000 human subjects, the researchers studied patients with the ApoE4 gene and those with and without chronic low-grade inflammation defined by sequential C-reactive protein measurements. They found carriers of ApoE4 with chronic low-grade inflammation, was more strongly related to onset of dementia as well as AD as compared to ApoE4 carriers without inflammation.

Qiu believes that without chronic low-grade inflammation there could be no difference of Alzheimer's risk between ApoE4 and non-ApoE4 carriers and that anti-inflammatory treatments could be effective for AD prevention.

The findings currently appear in *JAMA Network Open*.



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