

Elevated Insulin Levels Appear to Increase Levels of Inflammatory Markers and Beta-Amyloid, Which May Contribute to Alzheimer's Disease

CHICAGO, IL -- August 8, 2005 -- Moderately elevated levels of insulin increase the levels of inflammatory markers and beta-amyloid in plasma and in cerebrospinal fluid, and these markers may contribute to Alzheimer's disease, according to a new study posted online today from *Archives of Neurology*, one of the *JAMA/Archives journals*. The study will be published in the October print edition of the journal.

According to background information in the article, "conditions of insulin resistance and hyperinsulinemia are associated with elevated levels of inflammatory markers and increase the risk for Alzheimer disease (AD). Inflammation has been proposed as a key pathogenic factor for AD."

Mark A. Fishel, MD, from the University of Washington, Seattle, and colleagues, raised blood insulin levels (while maintaining normal blood sugar levels) in 16 healthy older adults ranging in age from 55 to 81 years, and then measured the changes in levels of inflammatory markers, modulators, and beta-amyloid (a protein associated with AD) in plasma and cerebrospinal fluid.

"Moderate peripheral hyperinsulinemia (increased levels of insulin) provoked striking increases in CNS (central nervous system) inflammatory markers," the authors report. "Our findings suggest that insulin-resistant conditions such as diabetes mellitus and hypertension may increase the risk for AD, in part through insulin-induced inflammation."

"Although this model has obvious relevance for diabetes mellitus, hyperinsulinemia and insulin resistance are widespread conditions that affect many nondiabetic adults with obesity, impaired glucose tolerance, cardiovascular disease, and hypertension. Our results provide a cautionary note for the current epidemic of such conditions, which, in the context of an aging population, may provoke a dramatic increase in the prevalence of AD. More encouragingly, greater understanding of insulin's role in AD pathogenesis may lead to novel and more effective strategies for treating, delaying, or even preventing this challenging disease," the authors conclude.

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REFERENCE:

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