

Insulin Resistance Increases Risk for Alzheimer's Disease



An imaging study finds a strong association between insulin resistance and lower brain glucose metabolism in a group of late middle-age adults at risk for Alzheimer's disease (AD). The findings are published online in JAMA Neurology.

Insulin resistance, broadly defined as reduced tissue responsiveness to the action of insulin, is common in people who are obese, prediabetic or have Type 2 diabetes. Insulin has been increasingly recognised as playing an important role in the brain, according to the study background.

Barbara B. Bendlin, PhD, of the University of Wisconsin School of Medicine and Public Health, and colleagues examined 150 cognitively normal, late middle-age adults (average age nearly 61) from the Wisconsin Registry for Alzheimer's Prevention (WRAP) study, a general community sample enriched for AD parental history. The subjects underwent cognitive testing, a fasting blood draw and fludeoxyglucose F 18-labelled positron emission tomography (PET) of the brain.

Of the 150 participants, 108 (72 percent) were women, 103 (68.7 percent) had a parental history of AD, 61 (40.7 percent) had an APOE ε4 allele and seven (4.7 percent) had type 2 diabetes.

Dr. Bendlin's team found that insulin resistance was associated with lower global glucose metabolism and lower regional glucose metabolism across large portions of the brain in the frontal, lateral, parietal, lateral temporal and medial temporal lobes. Lower glucose metabolism in the left medial temporal lobe was related to worse performance in immediate memory, according to researchers.

The study is the first to look at insulin resistance in late middle-aged people, identify a pattern of decreased blood sugar use related to AD and link that to memory decline.

"The prevalence of AD continues to grow, and midlife may be a critical period for initiating treatments aimed at preventing or delaying the onset of AD. Accumulating evidence suggests that treatments targeting mechanisms involved in insulin signalling may affect central glucose metabolism and should be investigated in the context of presymptomatic AD," the authors conclude.

Source: [JAMA](#)

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