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# The diabetic effects in Alzheimer disease: The role of insulin and β amyloid peptide

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## Abstract

Insulin resistance, hyperinsulinemia, glucose intolerance and products related to insulin metabolism can affect the amyloid cascade and collaborate in precipitation of the Alzheimer dementia picture or aggravate it, in early or old age, regardless of development of type 2 diabetes. The changes described in pathological studies and molecular research, classify two types of mechanism involved with cognitive impairment in these cases: one related to cerebrovascular events due the action of vascular risk factors, and the other controversial, non-cerebrovascular, from the interaction of insulin with the Aβ in the entorhinal cortex and hippocampus, as well as its direct action modulating NMDA receptors and neurotransmitters as acetylcholine and norepinephrine, not directly related to the glucose modulation. Using the literature review, and clinical observation of cases, we emphasize the role of insulin in the central nervous system and its participation in amyloidogenesis process in the evolution of Alzheimer disease. We bring into discussion the main mechanisms contributing to chronic peripheral hyperinsulinemia, which determines the sensitizing of insulin receptors in the blood-brain barrier (with insulin receptors down regulation) and decreased insulin uptake, causing a state of central hipoinsulinism, triggering interference mainly in the process of the Aβ regulation and inflammation, of the Aβ degradation transported to periphery and decreased of the Aβ clearance, among others, increasing synaptic toxicity, implying in clinical and anatomic changes in favor of the Alzheimer dementia.

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