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Fructose made in the brain could be a mechanism driving Alzheimer's disease

by CU Anschutz Medical Campus



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New research released from the University of Colorado Anschutz Medical Campus proposes that Alzheimer's disease may be driven by the overactivation of fructose made in the brain.

The study was published in the *Frontiers in Aging Neuroscience* and outlined the hypothesis that Alzheimer's disease is driven largely by Western culture that has resulted in excessive

fructose metabolism in the brain.

The paper brought together an interdisciplinary team of neurologists, neuroscientists and experts on sugar metabolism, and presents evidence from extensive data and research conducted in Alzheimer's disease that links high fructose levels in the brain to the disease. It also helps explain associations, such as why diabetes and obesity are associated with an [increased risk](#) for Alzheimer's disease.

"In essence, we propose that Alzheimer's disease is a modern disease driven by changes in dietary lifestyle in which fructose can disrupt cerebral metabolism and [neuronal function](#)," said author Richard Johnson, MD, professor at the University of Colorado School of Medicine on the CU Anschutz Medical Campus.

Johnson outlines data that showcases the overactivation of cerebral fructose metabolism that can drive Alzheimer's disease. The source of fructose is largely from endogenous production in the brain. Thus, the reduction in mitochondrial energy production is hampered by neuronal glycolysis that is inadequate, resulting in progressive loss of cerebral energy levels required for neurons to remain functional and viable.

"By outlining consistent evidence, we're hoping to inspire researchers to continue exploring the relationship between fructose in the brain and Alzheimer's disease. New treatments aimed at inhibiting intracerebral fructose metabolism could provide a novel way to prevent and treat this disease," Johnson adds.

In one of the scenarios outlined by Johnson and his collaborators, glucose hypometabolism increased [oxidative stress](#), and a progressive loss of mitochondria occurred, leading eventually to neuronal dysfunction and death. In this scenario, the [amyloid plaques](#) and [neurofibrillary tangles](#) are part of the inflammatory response and participate in injury, but are not the central factors driving the disease. Johnson mentions that theoretically, inhibiting enzymes in the brain that are involved in fructose production or metabolism might provide novel ways to prevent and treat Alzheimer's disease.

More information: Richard J. Johnson et al, Cerebral Fructose Metabolism as a Potential Mechanism Driving Alzheimer's Disease, *Frontiers in Aging Neuroscience* (2020). DOI: [10.3389/fnagi.2020.560865](https://doi.org/10.3389/fnagi.2020.560865)

Provided by [CU Anschutz Medical Campus](#)

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