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Chronic Brain Glucose Deficits Modulates Neuropathology and Dementia: Implication For Alzheimer's Disease

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In recent years, growing experimental evidence has suggested a direct association between altered glucose metabolism, brain function and neurodegeneration. Together with human studies, the investigations using experimental models have all convened on a common final point: dysregulated brain glucose levels and impaired energy metabolism in the brain are not only a clinical feature of Alzheimer's disease (AD) but also important contributors to its pathogenesis. Some evidence suggested that brain glucose deficits can influence amyloid beta levels in vivo but no data are available on the effect that this condition might have the development of tau neurofibrillary tangles and cognitive functions, the other two most important faetures of AD pathophysiology. In this paper we investigated the effect of chronic brain glucose deficits and energy dysregulation on memory and learning, synaptic function as well as the development of tau neuropathology in a model of tauopathy. Compared with controls, a condition of glucose deprivation and chronic brain energy deficiency resulted in significant memory deficits, impaired synaptic function, increased tau phosphorylation and neuronal cell death via apoptosis. Our studies demonstrate that reduced glucose availability in the central nervous system promotes directly the development of memory impairments, tau neuropathology, synaptic dysfunction, and neuronal cell death. Since restoring brain glucose levels and metabolism could afford the opportunity to positively influence the entire AD phenotype, it should be considered as viable therapeutic approach for this disease and related dementias.

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