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Activation of brain glucose metabolism ameliorating cognitive impairment in APP/PS1 transgenic mice by electroacupuncture

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Abstract

An essential feature of Alzheimer's disease (AD) is implicated in brain energy metabolic impairment that is considered underlying pathogenesis of cognitive impairment. Therefore, therapeutic interventions to allay cognitive deficits that target energy metabolism may be an efficacy strategy in AD. In this study, we found that electroacupuncture (EA) at the DU20 acupoint obviously increased glucose metabolism in specific brain regions such as cortex, hippocampus, cingulate gyrus, basal forebrain septum, brain stem, and cerebellum in APP/PS1 transgenic mice by animal ¹⁸F-Fluoro-2-deoxy-D-Glucose (¹⁸F-FDG)/ positron emission tomography (PET) imaging, accompanied by cognitive improvements in the spatial reference learning and memory and memory flexibility and novel object recognition performances. Further evidence shown energy metabolism occurred in neurons or non-neuronal cells of the cortex and hippocampus in terms of the co-location of GLUT3/NeuN and GLUT1/GFAP. Simultaneously, metabolic homeostatic factors were critical for glucose metabolism, including phosphorylated adenosine monophosphate-activated protein kinase (AMPK) and AKT serine/threonine kinase. Furthermore, EA-induced phosphorylated AMPK and AKT inhibited the phosphorylation level of the mammalian target of rapamycin (mTOR) to decrease the accumulation of amyloid-beta (A β) in the cortex and hippocampus. These findings are concluded that EA is a potential therapeutic target for delaying memory decline and A β deposition of AD. The AMPK and AKT are implicated in the

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