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Investigating the Effects of Cholecalciferol on the Insulin Signaling Pathway Impairment Associated with A40/42 and Tau Hyper Phosphorylation in a Scopolamine-Induced Ad Rat Model

Marc Scherlinger*
The University of Sydney,
Department of chemistry, Australia.

Corresponding author:
Marc Melanoma Institute Australia,
Department of chemistry Australia

mscherli@bidmc.harvard.edu

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Introduction

Alzheimer's disease (AD) is a condition marked by memory loss, the presence of oligomers A, neuronal and synaptic loss, and insulin resistance. Recent preclinical and human studies suggested that insulin signalling dysfunction, which results in persistent insulin resistance, may contribute to the pathogenesis of Alzheimer's disease by mediating insulin signaling-associated proteins. The precise biological changes that occur in Alzheimer's disease, the different rates of progression in affected individuals, and how Alzheimer's disease can be slowed or prevented remain largely unknown. As of now, there are no long-term therapeutic or protective treatments for Alzheimer's disease. To delay and to treat the progression of Alzheimer's disease, a more effective and cost-effective treatment approach is required. A lack of vitamin D can result in cognitive impairment, such as poor memory and learning. The underlying problem, however,the goal of this study is to see how cholecalciferol affects A40/42 and tau hyperphosphorylation, which is associated with insulin signalling pathway dysfunction in a scopolamine-induced AD rat model.

How Cholecalciferol Affects A40/42 and Tau Hyperphosphorylation

40 years old male Sprague Dawley rats weighing 350 50 g and aged four months were randomly divided into five groups (n=8),the experiments were carried out in accordance with the Animal Ethic Protection Committee of the PAPRSB Institute of Health Sciences at University Brunei Darussalam in Brunei. the control group was given 0.9 percent saline (w/v) for 28 days in a row,Scopolamine (2.5 mg/kg) was administered intravenously to all groups as a disease inducer, for 28 days, the treatment groups were given Donepezil (5 mg/kg) and cholecalciferol (71.42 IU/kg) by oral gavage.

Scopolamine will get dissolved in 0.9 percent saline solution, and cholecalciferol in sunflower oil, Sunflower oil was administered orally via oral gavage to the Vehicle control group (in the same volume as the scopolamine-cholecalciferol-treated group. From day 1 to day 28 of compound and drug administration, a rectangular maze test and a locomotor activity test were performed for a behavioural study. Following compound/drug administration, each group's brain and blood were collected. Plasma was extracted. H&E staining and immunohistochemistry were performed on 5m thick brail slices. Following the manufacturer's instructions, plasma and brain lysate were used for ELISA studies for Human A(1–40) (N) assay kit (IBL, Gunma, Japan), Human A (1–42) assay kit (Immuno-biology) (IBL, Gunma, Japan), Amplex Red Glucose/ glucose Oxidase Assay Kit (Invitrogen), and the insulin assay Kit ELISA (Cloud-Cl The remaining lysate was immunoblotted for Akt, pAkt (Ser473), GLUT3 GLUT4, tau phosphorylation site at Ser396 (PHF13), Ser202/Thr205 (AT8), and A40/42 (MOAB2). A densitometry analysis was carried out.

Brain inflammation appears to have a dual function, acting as a neuroprotective factor during an acute response but becoming detrimental during a chronic response,Chronically activated microglia produce a wide range of proinflammatory and toxic byproducts, such as reactive oxygen species, nitric oxide, and cytokines. There is an increase in cerebral A deposits 1–3 weeks postinjury in deceased patients suffering from recent head trauma, and it has been shown that elevated levels of interleukin 1 (IL-1) are responsible for the increased APP production and A load Furthermore, increased IL-1 levels have been shown to increase the production of other cytokines, including IL-6, which has been shown to stimulate the activation of CDK5, a kinase known to hyperphosphorylate tau.

Downregulation

Downregulation of IRS-1, IRS-2, Akt, pAkt (Ser473), GLUT3 and GLUT4 expression may lead to impaired insulin signalling, which has been linked to the development of Alzheimer's disease. According to the findings, cholecalciferol treatment may increase the expression of insulin signalling proteins, thereby improving insulin signalling, as well as improving memory performance and lowering A40/42 and tau hyperphosphorylation levels. As a result,

cholecalciferol treatment may be a viable option for Alzheimer's disease treatment.

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Conflict of interest

There is no conflict disclosed in this article.