J Neurol Neurophysiol 2018, Volume 9
DOI: 10.4172/2155-9562-C8-083

In vivo modulation of transferrin receptor protein-1 by a complex vitamin molecule reverses Alzheimer's-type pathology

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Metal ions are crucial for neurochemical signaling, while perturbations in their regulation are associated with neurodegenerative processes in Alzheimer's disease (AD). Hypothesizing that metal chelators, anti-oxidants and anti-inflammatory agents could improve disease outcomes via modulation of transferrin receptor protein-1 (TfP-1)-a metal ion regulator, the efficacy of a complex vitamin supplement (CVS) formulated with B-vitamins and ascorbic acid in reversing AD type neurodegeneration was investigated. Eight-weeks-old Wistar rats were assigned into five groups (n=8), including controls and those administered CVS orally for two weeks before or after aluminum chloride ($AlCl_3$)-induced neurotoxicity. Rats were assessed for standard behavioral functions after which prefrontal cortex (PFC), hippocampus and amygdala were prepared for spectrophotometry, histology, histochemistry and immunohistochemistry. Obtained data showed that CVS significantly reversed reduction of exploratory/working memory, frontal-dependent motor deficits, cognitive decline, memory dysfunction and anxiety in rats. These findings correlated with CVS-dependent modulation of TfP-1 expression within studied brain regions that were accompanied by significant reversal of neural oxidative stress in expressed superoxide dismutase, nitric oxide, catalase, glutathione peroxidase and malondialdehyde. Through modulation of TfP-1, CVS inhibited neural bioenergetics dysfunction as increased labeling of glucokinase within PFC and hippocampus (but not amygdala) correlated with increased glucose-6-phosphate dehydrogenase and decreased lactate dehydrogenase expressions. These further relates to inhibition of overexpressed acetyl cholinesterase and increased total protein synthesis. H and E and Nissl staining of thin sections corroborated roles of CVS in reversing $AlCl_3$-induced AD-like pathology and were accompanied by related changes in astrocytes and neuro-filaments (cytoskeleton) immunohistochemical analyses. Summarily, this study showed that modulation of TfP-1 overexpression by CVS restores normal neurochemical signaling and inhibits AD-like cellular hypertrophy.

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