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Joint Event on

Down Syndrome, Autism, Brain Disorders & Therapeutics

October 21-22, 2019 | Tokyo, Japan

Investigation of long-term potentiation- and depression-induced Tau phosphorylation in rats with starch based sugar

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Objective: A lot of evidence supports the hypothesis that the mechanism of memory trace formation at the cellular level is Long-Term Potentiation (LTP) and Long-Term Depression (LTD). LTP and LTD are initiated postsynaptically by the activation of N-Methyl-D-Aspartate (NMDA) receptors (NMDARs), resulting in Ca2+ influx and the subsequent activation of several kinases. Studies in which experimental animals were fed with fructose for a long time showed that insulin resistance occurred and this was associated with poor performance in hippocampus dependent learning. We therefore wanted to study that LTP/LTD-related modifications of Tau phosphorylation could be, or not be, changed with High-Fructose Corn Syrup consumption (HFCS).

Methods: The study was performed on 60 (100±15 grams; 20/group) 21-day old male Wistar Albino rats obtained from Erciyes University Experimental Animal Research Center. On the 21st day, the male rats leaving their mothers are fed with unrestricted standard rat chow and tap water, HFCS solution (8%; 0.24 Kcal/mL) or sucrose solution (10%, 0.4 Kcal/ mL) for at least 21 days. The field potentials were recorded from the right dentate gyrus with stimulation of the right medial perforant path. LTP and LTD were induced by High and Low Frequency Stimulation (HFS and LFS), respectively and total and phosphorylated forms of Tau was measured in the hippocampus at least 60 minutes after induction of plasticity.

Results: The input/output curves of the study groups did not differ ($P>0.05$). After 1 hour of induction, LTD was 92±8% and 90±5% of the pre-LFS value in the control and sucrose groups, while LTP was 121±4% and 119±5%, respectively. There was no statistical significance between these groups ($P>0.05$). In the group fed with HFCS, the LFS and HFS did not induce LTP or LTD responses. In control and sucrose groups, it was found that the induced LTD was accompanied by spike potentiation (154±8% and 128±15%, respectively) and this potentiation was not observed in the HFCS group ($P<0.01$). Western blot experiments indicated that Tau protein was hyperphosphorylated at ser416 epitope upon LTP but rather hypophosphorylated at thr231 epitope upon LTP in the whole hippocampus of HFCS-fed rats. These changes concomitantly occurred with notable alterations in the levels of total Tau.

Conclusion: These findings suggest that high fructose-containing diets may disrupt the balance between two forms of synaptic plasticity and thus adversely affect learning processes. Epitope specific Tau phosphorylation had been emphasized for Alzheimer's disease-like learning deficit due to feeding with HFSC.

Biography

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