

Dietary Composition Modulates Memory Performance in Rats: Role of B-Amyloid 1-42 and Oxidative Stress

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Abstract

Novel evidence showed that the high fructose consumption impaired cognitive abilities and disrupted the insulin signaling. High-fat diet consumption also caused learning impairment. Learning and memory deficit were induced by β -amyloid peptide 1-42 was documented in recent studies. Based on this assumption, the aim of the present study was to test the hypothesis that diet would modulate β -amyloid 1-42 deposition in the brain, which may be implicated in causing memory deficit. The study was conducted on 30 adult male albino rats, aged 12-14 months, ranging in body weight from 150–200 g, divided into 3 groups (n=10), each was given one of 3 different diets: group I given Control diet (CD) consisted of standard chow pellets, group II given high unsaturated-fat diet (HUFd) and group III given high carbohydrate diet (HCD), for the entire test period which lasted for 8 weeks. On the last day of the eight weeks, memory training through water maze test was done. 24 hours later, memory testing was performed after which rats were allowed to fast for 12 hours before being sacrificed. Blood obtained from the jugular vein was collected and plasma was separated for measuring fasting blood glucose and plasma lipids, hippocampus was homogenized and stored for biochemical determinations of β -amyloid 1-42, and superoxide dismutase activity in all groups.



Biography:

Maha Deif is the faculty of Medicine currently working in Alexander University. So far I have published two papers on Neurology.



Speaker Publications:

1. “Wang C, Yang X-M, Zhuo Y-Y, Zhou H, Lin H-B, Cheng Y-F, et al. The phosphodiesterase-4 inhibitor rolipram reverses A β -induced cognitive impairment and neuroinflammatory and apoptotic responses in rats. *Int J Neuropsychopharmacol.* 2012;15:749–66.
2. “Hritcu L, Noumedem J, Cioanca O, Hancianu M, Kuete V, Mihasan M. Methanolic extract of Piper nigrum fruits improves memory impairment by decreasing brain oxidative stress in amyloid beta(1–42) rat model of Alzheimer’s disease. *Cell Mol Neurobiol.* 2014;34:437–49.
3. “Suh Y-H, Checler F. Amyloid precursor protein, presenilins, and α -synuclein: molecular pathogenesis and pharmacological applications in Alzheimer’s disease. *Pharmacol Rev.* 2002;54:469–525.
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5. “Ferretti L, McCurry S, Logsdon R, Gibbons L, Teri L. Anxiety and Alzheimer’s disease. *J Geriatr Psychiatry Neurol.* 2001;14:52–8.

[9th International Conference on Brain Disorders & Therapeutics](#); August 21-22, 2020.

Abstract Citation:

Maha M Deif, Dietary composition modulates memory performance in rats: Role of B-amyloid 1-42 and oxidative stress, *Brain Disorders 2020*, 9th International Conference on Brain Disorders & Therapeutics; August 21-22, 2020.

<https://braindisorders.neuroconferences.com/abstract/2020/dietary-composition-modulates-memory-performance-in-rats-role-of---amyloid-1-42-and-oxidative-stress>