

Glucose Role in Treatment of Alzheimers' Disease

Amos Gelbard*

Zefat Academics Kibbutz Eilon, Galil Maaravi, Israel

*Corresponding author: Amos Gelbard, Zefat Academics Kibbutz Eilon, Galil Maaravi, Israel, Tel: 1-800-344-544; E-mail: amosgelbard@gmail.com

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Commentary

Several researches have shown correlation between the advancement of Alzheimer's disease and higher blood Cortisol levels [1-3]. Cortisol supposedly impacts the function of alpha, beta and gamma secretases, enzymes responsible for the generation and modulation of amyloid β peptide [4].

The high cortisol levels evident in AD lead to malfunction in the peptide of amyloid β by these proteases and to excess amyloid buildup in the brain, within the neurons, and eventually to neuronal death, the main cause for the well-known symptoms of the disease [5].

Cortisol, known as "the stress hormone", is released by the adrenal glands in response to stress and low blood glucose concentration and its main objective is to stimulate a rise in said glucose levels.

It's plausible to assume down regulation of cortisol levels to be the desired effect necessary in order to contradict the advancement of the disease. One way to lower cortisol levels, through physical activity, is ineffective in most patients' age group.

However, If Cortisol's primary function is to compensate for low blood glucose level, it's at least logical to hypothesize that increased glucose levels in a patient's nutrition could have the same effect for which the body needs extra cortisol, therefore allowing it to release less Cortisol and avoid its harmful effect on AD patients.

If by increasing glucose dietary intake, the body would no longer be forced to release as much Cortisol and if cortisol is indeed responsible for the irregularity in secretase function that leads to amyloid beta

accumulation, it is very possible for such nutritional modification to be an effective treatment against Alzheimer's disease.

Previous studies have shown abnormalities in Alzheimer patients' glucose utilization and metabolism [6,7]. However, no clinical study has yet been made to test the exact effect of heightened Glucose nutrition or supplementation on Alzheimer patients.

Based on this logic and assumption, further research of glucose and its impact on Alzheimer's patients is recommended.

References

1. Dong H, Csernansky JG (2009) Effects of Stress and Stress Hormones on Amyloid β Protein and Plaque Deposition. *J Alzheimers Dis* 18: 459-469.
2. Csernansky JG, Dong H, Fagan AM, Wang L, Xiong C, et al. (2006) Plasma cortisol and progression of dementia in subjects with Alzheimer-type dementia. *Am J Psychiatry* 163: 2164-2169.
3. Swaab DF, Raadsheer FC, Endert E, Hofman MA, Kamphorst W, et al. (1994) Increased cortisol levels in aging and Alzheimer's disease in postmortem cerebrospinal fluid. *J Neuroendocrinol* 130: 1707-1712.
4. Strooper BD, Vassar R, Golde T (2010) The secretases: enzymes with therapeutic potential in Alzheimer disease. *Nat Rev Neurol* 6: 99-107.
5. Hardy J, Allsop D (1991) Amyloid deposition as the central event in the aetiology of Alzheimer's disease. *Trends Pharmacol Sci* 12: 383-388.
6. Hover S (1991) Abnormalities of glucose metabolism in Alzheimer's disease. *Ann N Y Acad Sci* 640: 53-58.
7. Chen Z, Zhong C (2013) Decoding Alzheimer's disease from perturbed cerebral glucose metabolism: implications for diagnostic and therapeutic strategies. *Progress in Neurobiology* 108: 21-43.