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Rethinking Glucose Role in Alzheimer's Disease Based on the Disease Incidence in Diabetes Type 2 Patients

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Commentary

In a previous essay [1], I explained why Alzheimer's Disease (AD) is a result of high cortisol levels, [2-4] that through time cause defects in the enzymes responsible to the proper peptide of beta-amyloid-alpha, beta and gamma secretases-and consequently to the accumulation of beta-amyloid within the neurons which results in Alzheimer's Disease [1].

In the said paper, I suggested that due to Cortisol's role-releasing Glucose to the bloodstream in order to stimulate the body to handle stressful situations-that a possible treatment to AD would be by enhancement of Glucose intake, through nutrition or food supplementation.

This theory is challenged by the relatively high occurrence of AD and Dementia in Diabetics. Diabetes is a medical condition that stems from high Glucosic nutrition, in which too much glucose in the bloodstream leads to its appearance in the patient's urine-the traditional hallmark of Diabetes.

Two hormones working in collaboration are responsible for transferring Glucose from the bloodstream to the cells-Insulin-and from the cells to the bloodstream-glucagon.

In Diabetes, the body inhibits Insulin secretion in order to enable weight loss and the disposal of Glucose through the bloodstream to the urine. This process requires Glucagon but also the production of cortisol, which has the same effect of transferring glucose from the body to the bloodstream [5]. It's not clear if the higher cortisol secretion is a hallmark of Diabetes [6] or just a side effect of insulin treatment to the disease. Some Diabetic treatments do not promote AD appearance but actually reduce its occurrence, in these cases cortisol levels are also normal [7]. If we assume that cortisol has many of the same responsibilities and methods as glucagon in transferring glucose from the body cells to the bloodstream, it makes sense to suggest not glucose but glucagon as a possible substance to replace the need for cortisol and downgrade its levels [5].

Coincidentally, a hormone called glucagon-like peptide 1 (GLP-1) has recently been tested with very promising results as a possible treatment to AD [8-10]. This coincides with the above logic regarding glucagon related substances possibly playing a role in downgrading cortisol levels and possibly having a positive influence in treatment of Alzheimer's Disease.

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