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Endocrinology and Diabetology 2018 - Cortisol as the cause for several diseases and Folic Acid and Vitamin B12 as possible therapeutic targets

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Abstract

Background: This research first hypothesis is that Cortisol, the hormone in charge of sugar transfusion to the bloodstream to handle stressful situations is what's causing Alzheimer's disease because high calibers of this hormone for protracted periods of time lead to deterioration of Secretases alpha beta and gamma, in charge of the peptide of beta amyloid within the neurons. The 2nd hypothesis is that Diabetics consumption of glucose additionally stems from wanting to deter hyper cortisol secretion. Methods: This research is a review of many studies exhibiting correlation between Alzheimer's disease and Diabetes as well as other illnesses (Hypertension, Cushing Syndrome and Schizophrenia) with High Cortisol Levels, as well as low Vitamin B12 and Folic Acid Levels and high calibers of Homocysteine. Results: In Alzheimer's Diseases, Diabetes, Hypertension, Schizophrenia and Cushing Syndrome, High Cortisol Levels as well as Low Folic Acid and Vitamin B12 levels are conspicuous Conclusions: This proves true the hypotheses brought in the background section regarding both AD and Diabetes to stem from Cortisol secretion and rudimentally from dealing with stress and stressful everyday lives. What these finding suggest is that a possible way of downgrading Cortisol Levels and obviating or even treating verbalized diseases is by maintaining a salubrious diet of folic acid and vitamin b12 and that deficiency of these vitamins could possibly not only cause the above diseases but additionally to be the cause of stress itself.

Keywords

Cortisol, Alzheimer's disease, Diabetes, Folic Acid, Folate, Vitamin B12

Back Ground

Alzheimer's disease is defined by accumulation of beta amyloid plaques within the neurons of the encephalon. The enzymes responsible for the peptide of amyloid beta and accordingly of this malfunction are called alpha, beta and gamma secretases. The deterioration of their activity possibly stems from exorbitant levels of cortisol secretion through long periods of time aevident in Alzheimer's Disease Cortisol, "The Stress Hormone" is engendered by the encephalon to promote transfer of sugar to the blood streamin"fight or flight" situations.I first hypothesized that one way to downplay Cortisol secretion is by ascending of blood glucose levels through alimentation and consequently compensate for the desideratum in Cortisol, if you maintain high blood glucose levels, the desideratum for Cortisol subsides, but this was challenged by Alzheimer's occurrence inType2DiabetesPatients,then reached a conclusion that diabetes could be a behavioral reaction to high Cortisol levels, for the same logic of answering Cortisol secretion with Glucose ,Irealized that my initial conception of answering high cortisol secretion with glucose consumption could be the underlying cause for Diabetes. As I endeavored to cerebrate what could remedy high Cortisol Levels, not by duplication of its role, but by itself, I found numerous studies that show low folate (folic acid) levels and not just high cortisol levels to directly correlate with Alzheimer's, Higher Folate intake to truncate risk of Alzheimer's and Folic Acid treatment to manufacture amendment on all Important parameters in AD patients.

High cortisol and low folate are the only routine blood tests prognosticating probable Alzheimer's disease after age 75, Serum total homocysteine levels were significantly higher and serum folate and vitamin B12 levels were lower in patients with dementia of Alzheimer type and patients with histologically corroborated AD than in controls .Low blood levels of folate and vitamin B12, and ascended homocysteine (tHcy) levels were associated with AD. Low blood folate and raised homocysteine concentrations are associated with poor cognitive function. Folic acid supplementation ameliorates cognitive function.This diminutive pilot study examined the effect of folic acid supplementation on incipiently diagnosed patients with AD. Folic acid amended cognition and markers of inflammation. Study findings have suggested a sodality between Alzheimer's disease

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(AD) risk and several vitamins and have notionally theorized about their utilization as preventive agents. Here, we examine whether total intake (intake from diet plus supplements) of antioxidant vitamins (E, C, carotenoids) and B vitamins (folate, B6, and B12) is associated with a minimized risk of AD…After a mean follow-up of 9.3 years, AD developed in 57 participants. Higher intake of folate (RR, 0.41; 95% confidence interval [CI], 0.22 to 0.76), vitamin E (RR, 0.56; 95% CI, 0.30 to 1.06), and vitamin B6 (RR, 0.41; 95% CI, 0.20 to 0.84) were associated individually with a decremented risk of AD after adjusting for age, gender, inculcation, and caloric intake

When these 3 vitamins were analyzed together, only total intake of folate at or above the RDA(RR, 0.45; 95% CI, 0.21 to 0.97) was associated with a paramount decremented risk of AD. No sodality was found between total intake of vitamins C, carotenoids, or vitamin B12 and jeopardize of AD.This study's objective was to explore the sodalities of low serum levels of vitamin B12 and folate with AD occurrence. When utilizing B12 ≤150pmol/L and folate ≤10 nmol/L to define low calibers, compared with people with mundane levels of both vitamins, subjects with low calibers of B12 or folate had twice higher risks of developing AD (relative risk [RR] = 2.1, 95% CI = 1.2 to 3.5). These sodalities were even more vigorous in subjects with good baseline cognition (RR = 3.1, 95% CI = 1.1 to 8.4). Homogeneous relative risks of AD were found in subjects with low calibers of B12 or folate and among those with both vitamins at low calibers.

In type 2 diabetic subjects, hypothalmic-pituitary-adrenal activity is enhanced in patients with diabetes complications and the degree of cortisol secretion is cognate to the presence and number of diabetes complications.Short-term oral folic acid supplementation significantly enhances endothelial function in type 2 diabetic patients Data implicatively insinuate that folate can be habituated to ameliorate nitric oxide status and to recuperate endothelial dysfunction in patients with Type II diabetes.Our results provide a vigorous rationale for the initiation of studies that investigate whether supplementation with folic acid obviates future cardiovascular events in this patient group. Average Tardy night salivary cortisol levels (nmol/l) in diabetics were significantly higher than in non-diabetics.

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