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SCIENTIFIC LITERATURE

EDITORIAL

Dietary Interventions reverse Insulin and Synaptic Plasticity defects linking to Diabetes and Neurodegenerative diseases

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ARTICLE INFO

Article history:
Received: 29 November 2016
Accepted: 05 June 2017
Published: 12 June 2017

Keywords:
SNAP25;
Insulin secretion;
Synaptic plasticity;
Diabetes;
Lipopolysaccharides;
Patulin

Citation this article: Martins IJ. Dietary Interventions reverse Insulin and Synaptic Plasticity defects linking to Diabetes and Neurodegenerative diseases. SL Nutr Metab. 2017; 1(1):111.

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EDITORIAL

In the developing world food and nutrition guidelines with relevance to nutrition and metabolism are required to reduce elevated cholesterol levels with relevance to diabetes, Parkinson's disease and Alzheimer's disease [1]. In diabetes and neurodegenerative diseases global Non Alcoholic Fatty Liver Disease (NAFLD) has become of major concern with the reduced hepatic metabolism of toxic compounds that increase with age to milligrams quantities of bacterial Lipopolysaccharides (LPS), mycotoxins and xenobiotics that induce neuron death. The increased brain content of these toxic compounds completely inactivate drug therapy with increased repetitive drug exposure to neurons and synpases [2-4]. In the global diabetes epidemic diabetes is expected to double by the year 2050 in the developing world and diets that reverse or stabilize neurodegenerationhave become relevant to synaptic plasticity and to prevent neuronal death [5]. These toxic compounds determine the survival of various species with relevance to greater toxicity to neurons and their synapses in diabetes when compared to toxic amyloid beta oligomers and alpha synuclein oligomers in Parkinson's disease and Alzheimer's disease [1].

In diabetes information processing is defective and related to defective interactions between neurons and synapses [6-9]. The toxic compounds that increase in the brain of diabetics induce post-translational modifications (Figure 1) between cholesterol interacting proteins [1] and are responsible for the poor synaptic connections with relevance to insulin resistance and impairments in synaptic plasticity. Defects in cholesterol transport in the brain are responsible defective information processing and synaptic plasticity in diabetes and neurodegenerative diseases. Interests in plasma LPS and patulin levels (Figure 1) in the developing world have attracted major concern with relevance to accelerated cross linking (intra and intermolecular crosslinks) between cholesterol interacting proteins [1] in cell membranes with relevance to cholesterol, alpha synuclein and amyloid beta metabolism (Figure 1). Cholesterol interacting proteins [1] include apolipoprotein AI (apo AI), apolipoprotein E (apo E), ATP binding cassette transport protein 1 (ABCA1), low density lipoprotein receptors (LDLr/LRP), G-coupled protein receptors (GPCR) [10-12], Phospholipid Transfer Protein (PLTP) and amyloid beta (Aβ).

Life changing scientific discoveries indicate that the nuclear receptor Sirtuin 1 (Sirt 1) is important to cholesterol homeostasis [13] and to prevent synaptic plasticity defects in diabetes, Parkinson's disease and Alzheimer's disease [6-9].

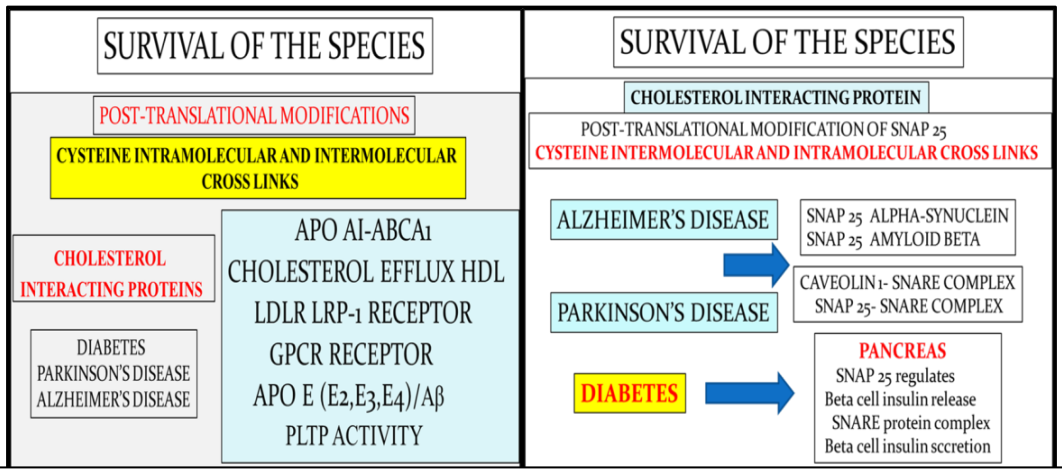

Figure 1: The poor survival rates of individuals in populations connected with increased global death rateshas raised concerns for individuals in the developing world with relevance to the current global diabetes epidemic. Hypercholesterolemia is a major defect and related to post-translational modifications of cholesterol interacting proteins (cysteine residues). LPS/Patulin are responsible for inactivation of cysteine residues in cholesterol interacting proteins (apo AI, apo E, GPCR, PLTP, LDLr/LRP, SNARE/caveolin 1) with relevance to diabetes (SNAP25 (cysteine-rich)/insulin release), Parkinson's disease(SNAP 25/alpha synuclein) and Alzheimer's disease(SNAP25/amyloid beta).

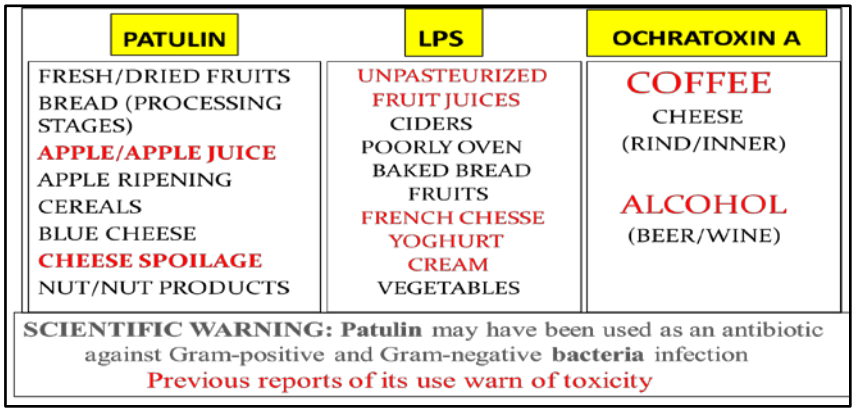

Figure 2: Blood tests for LPS and patulin in diabetic individuals in the developing world is required to determine the diagnosis of a miscible disease with relevance to diabetes and neurodegeneration. Nutritional intervention is required to measure LPS, Patulin and ochratoxin A in various foods to prevent ingestion of the end products that induce the miscible disease with relevance to mitochondrial dysfunction (NAFLD) and neuron apoptosis. In the developing world Patulin should not be ingested as an antibiotic with relevance to its poisonous effects (NAFLD) in various individuals.

Post-transcriptional dysregulation (via cysteine residues) that involve zinc defective Sirt 1/p53 interactions [1,14-19] effect caveolin 1 expression [20] that is important to cellular cholesterol homeostasis, insulin receptor transport and activity [21-24]. Interference of synatosomal associated protein 25 (SNAP25)-caveolin 1 interaction corrupt neuron synapse interactions (Figure 1) with implication of food ingestion that contain (LPS/Patulin) that inactivate cysteine residues essential for and SNAP25 membrane interactions [25,26] andSirt 1 function [11,12,27]. SNAP25 is involved in SNARE/caveolin 1 complex (Figure 1) with relevance to synaptic transmission in diabetes and neurodegenerative diseases [28-32].

Interference with SNAP25 and SNARE proteins in membranes now implicate SNAP25 to be involved with pancreatic beta cell insulin secretion (Figure 1) release with SNAP25a and SNAP25b now relevant to metabolic disease, synaptic plasticity and accelerated neurodegeneration [33-37]. . Nutritional interventions in the developing world need to be implemented to prevent a miscible disease with relevance to diabetes, Parkinson's disease and Alzheimer's disease. LPS levels should be carefully monitored in various foods such as

fruits, bread, apple/apple juice, cereals, cheese and nuts (Figure 2). Patulin levels should be assessed in yoghurt, cream, cheese, bread, fruits and unpasteurized fruit juices (Figure 2). In miscible diseases such as diabetes and neurodegenerative diseases ochratoxin levels should be assessed in coffee and alcohol to determine effects on neuron and mitochondria apoptosis. The plasma levels of LPS and patulin in the developing world (Figure 2) should be determined to prevent a miscible disease such as diabetes and neurodegeneration (synaptic plasticity defects). In the developing world low fat and very low carbohydrate diets are encouraged to prevent absorption of lipophilic LPS and patulin that are present in lipoproteins with toxic components transported to various tissues such as the pancreas (insulin secretin disorders), adipose tissue, liver (NAFLD) and brain (synaptic plasticity defects).

Conclusion

In the developing world the global diabetic epidemic is predicted to escalate over the next 30 years and a miscible disease with relevance to brain synaptic plasticity defects is expected to affect many of these diabetic individuals. Dietary interventions and nutritional research indicate that various food end productssuch as LPS and mycotoxins should not enter the blood plasma in diabetic individuals. LPS and patulin effect various cholesterol interacting proteins with effects on pancreatic insulin release and brain synaptic transmissions. Stabilization of this miscible disease with relevance to diabetes and neurodegneration involves low calorie nutrition that activates nuclear receptors relevant to the metabolism of LPS/mycotoxins with therapeutic activation of brain drug therapy in diabetic individuals in the developing world.

Acknowledgement

This work was supported by grants from Edith Cowan University, the McCusker Alzheimer's Research Foundation, and the National Health and Medical Research Council.

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