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Magnesium deficiency and induction of NAFLD and Type 3 diabetes in Australasia

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EDITORIAL

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Chronic diseases in Australasia[1] have attracted interest by medical researchers with relevance to the gene-environment interaction that indicates that genes under nutritional regulation have malfunctioned with the induction of organ suicide. The individuals in these communities develop appetite disorders[2] with consumption of excess food that leads to metabolic dysfunction, non-alcoholic fatty liver disease (NAFLD) and neurodegeneration. The organ that has central control of other organs is the brain and with brain neurotoxicity peripheral glucose levels increase and induce oxidative stress that leads to organ disease and brain disorders such as Type 3 diabetes[3,4] relevant to the apoptosis of cells in various tissues and organs.

The role of anti-aging genes in Type 3 diabetes[2] has become of central interest to maintain mitochondrial functions and the identification of longevity genes that determine their function is critical to the prevention of chronic diseases in the developing and developed world. Mitochondria in neurons become unstable with neuron apoptosis[5–7] associated with accelerated aging. Neurons within the brain that regulate appetite become altered with altered gene expression and posttranscriptional regulation closely connected to overeating, defective post-prandial lipid metabolism and chronic diseases.

The Australasian association may indicate that the developing world (South East Asia, Australian country towns, farms, communes) population may be at greater risk for chronic diseases such as NAFLD relevant to mitochondrial apoptosis and insulin resistance. In the developing world plasma bacterial lipopolysaccharides (LPS) levels have been shown to be increased with the major concern for antibiotic resistance in these communities.[8] Increased access to food (high fat/cholesterol) with LPS leads to induction of epigenetic alterations that are associated with lipid and glucose dyshomeostasis linked to oxidative stress, insulin resistance and NAFLD.[9]

Diets that are rich in fat release intestinal lipoproteins that contain LPS for transport of LPS from the intestine to the blood plasma[10] (Figure 1). Magnesium and bacterial LPS levels are connected and as LPS levels rise magnesium levels decrease with the induction of organ suicide closely connected to magnesium deficiency, albumin levels and an absent peripheral sink amyloid beta clearance pathway.[11] Magnesium deficiency may induce hypercholesterolemia, hyperglycemia, NAFLD and Type 3 diabetes. LPS may induce magnesium deficiency, inactivate hepatic amyloid beta clearance[11] and repress the anti-aging gene Sirtuin 1[12] (Figure 1) that may

predispose developing world individuals to emergency acute myocardial infarction.[13,14]

Healthy fat consumption may need to be reduced with LPS liver transformation relevant to delayed hepatic fat metabolism.[15] The amount of fat consumed[16] in individuals with LPS may need to be carefully modified to allow suprachiasmatic nucleus timing for peripheral hepatic amyloid beta (Figure 1) and glucose metabolism. Elevated LPS levels indicate that the tests for normal cholesterol levels should be reassessed with the risk for heart attacks emergent with LPS induced amyloid toxic oligomers.[11,17] The connections between LPS and magnesium deficiency indicate repression of the anti-aging genes Sirtuin 1[12] (magnesium activator) to be involved in brain insulin resistance[3,4] and cardiovascular disease.[18–22]

Developing and developed Australasia and the relevance of the global Aluminium (Al) industry has increased in recent years[23] and indicate that plasma Al levels should be measured with relevance to the increased Al levels in food.[23] Al may bind to membrane lipid binding sites sensitive to amyloid beta metabolism and interfere with zinc and magnesium membrane lipid interactions. Zinc deficiency has been reported in the developing world and LPS may induce zinc deficiency[17] and with Al intake (>3–12mg/day) may further promote amyloid beta aggregation[24,25] with acute inflammation relevant to myocardial infarction.

Induction of NAFLD and Type 3 diabetes in Australasia may be prevented by consumption of nutritional diets[26] that do not allow consumption of LPS and patulin involved in the induction of Type 3 diabetes. Magnesium supplements should be consumed by individuals with LPS and in individuals with extended exercise that induces magnesium deficiency and NAFLD. Fat consumption should not allow lipophilic xenbiotics/drugs[27,28] to enter the liver or brain that may corrupt brain glucose sensing[29] that determine peripheral glucose and lipid metabolism. LPS and induction of NAFLD has become of major concern in various Australasian communities with personal hygiene and food quality that may determine LPS induction of cardiovascular disease and various chronic diseases.

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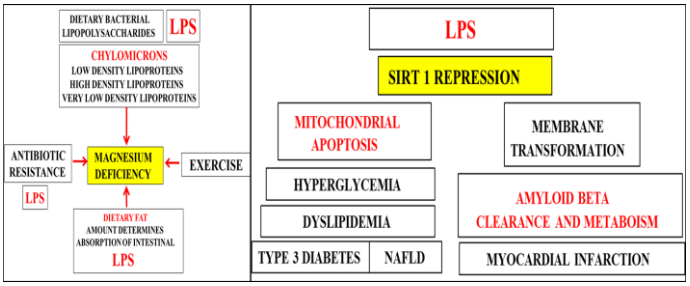
Figure 1: The increased plasma LPS levels in developing world individuals may be related to increased LPS fat transport from the intestine or antibiotic resistance. LPS induces magnesium deficiency with relevance to toxic amyloid beta oligomer formation with risk for myocardial infarction. Neuron amyloid beta transport to the liver for metabolism may be corrupted by LPS induced magnesium deficiency. The amount of fat in the diet may determine brain LPS and magnesium levels with relevance to the severity of insulin resistance and Type 3 diabetes



PEER REVIEW

Peer reviewed.

CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

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