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Perspective

Appetite Control and Nutrigenomic Diets are Connected to Immune Regulation and Diabetes Prevention

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Dietary interventions in diabetes have become of critical importance to stabilize insulin resistance, non-alcoholic fatty liver disease (NAFLD), synaptic plasticity defects and neurodegenerative diseases [1-6]. Nutritional research in diabetics (Type 3 or Type 2) to control appetite dysregulation [7] has promoted research into food and nutrition guidelines to allow appetite control to prevent insulin resistance that is connected to the global burden of disease. Appetite control and healthy dietary interventions in diabetes has failed to correct the adipocyte-liver interaction defect [8] with relevance to the induction of NAFLD that is projected to effect 40% of the global population by the year 2050.

Dietary interventions to reverse the defective adipocyte tissue-liver interaction may involve appetite control with food restriction essential to reverse defective hepatic metabolism of ingested dietary fat [9]. In the global burden of disease connections between diet and the immune system [10] has become of critical importance (Figure 1) with primary immune dysregulation related to the defective hepatic fat metabolism [8]. Low calorie diets to reverse defective adipose tissue-liver interactions will decrease adipocyte release of toxic inflammatory agents such adipocytokines [11,12] that are toxic to liver that induce NAFLD [13,14] and uncontrolled diabetes.

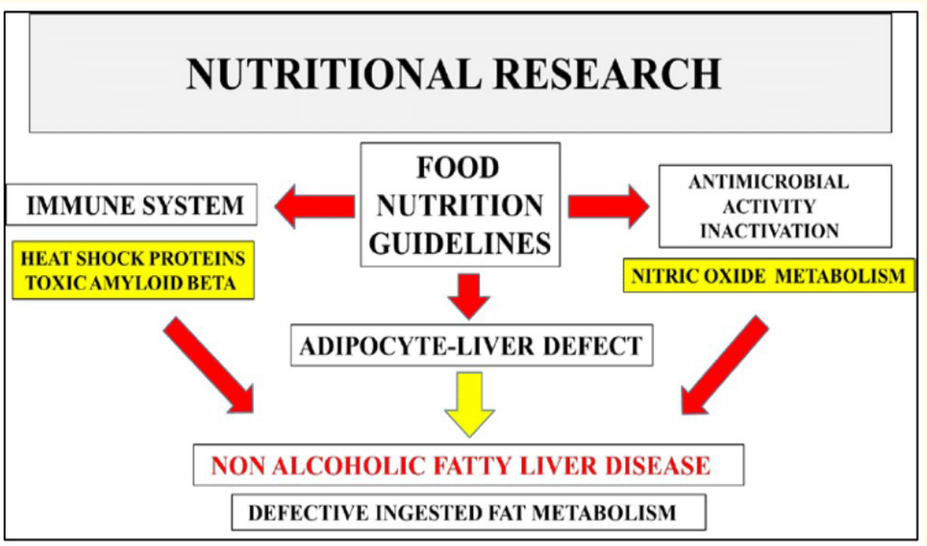

Figure 1: *Nutrigenomic diets are essential to prevent activation of the immune system that is associated with the adipocyte-liver defect and involved in the induction of NAFLD. Nutrition guidelines are essential to maintain nitric oxide regulation of antimicrobial activity/autoimmune disease with relevance to NAFLD and the metabolism of dietary fat.*

Diets that regulate appetite control the rapid plasma metabolism of heat shock protein (HSP) and toxic amyloid beta proteins [15,16]. These toxic proteins require rapid hepatic metabolism to prevent natural killer cells activity [17] involved with defective hepatic fat metabolism (Figure 1). Stress and diet that interfere with nitric oxide metabolism inactivate antimicrobial activity [18] essential for protection against microorganisms and activate natural killer cell activity involved with programmed cell death [19]. Defective adipocyte-liver interactions that induce NAFLD [8,17] are associated with the gene Sirtuin 1 that is defective in NAFLD in rodents and man [9,20]. Sirtuin 1 is associated with the regulation of food intake and its knockout in rodents is connected to NAFLD [9]. Nutritional regulation (low calorie) of Sirtuin 1 is essential to maintain the immune system [21], antimicrobial activity [18] with rapid metabolism of HSP, amyloid beta and nitric oxide relevant to the global burden of disease. In the developing world antibiotic resistance and inactive cell antimicrobial activity has raised concerns with relevance to accelerated NAFLD, diabetes and defective antimicrobial drug therapy.

Mitophagy is now connected to the global chronic disease with the defective immune system involved in mitochondrial apoptosis [22,23]. Sirtuin 1 is important to appetite and mitochondrial biogenesis [24] with its dysregulation related to defective immune system, nitric oxide dyshomeostasis and mitochondrial apoptosis. Diets that control immunometabolism [25,26] regulate the adipocyte-liver interaction with reversal of NAFLD. Food and nutrition guidelines in the developing world need to be revised [27] with relevance to the defective heat shock gene Sirt 1 [15,21] that is related to mitophagy, NAFLD and diabetes. Handling and processing of food in the developing world [15] needs attention to prevent Sirtuin 1 repression with immune cell defects related to autoimmune disease and mitophagy [28].

Conclusion

Appetite control with relevance to immunometabolism has become critical to the treatment of NAFLD and diabetes. Nutritional diets that contain activators to maintain immunometabolism and prevent mitophagy has become important to nutritional research. Appetite control or food restriction is required to maintain the heat shock gene that regulates heat shock proteins, amyloid beta and nitric oxide metabolism that are connected to natural killer cell activity, mitophagy and autoimmune disease in diabetes. Nutrition and antimicrobial activity in diabetes needs re-evaluation with relevance to antibiotic resistance and the failure of antimicrobial drugs.

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