Glycomics & Lipidomics

Extended Abstract

Anti-aging genes regulate postprandial lipid metabolism with relevance to appetite, chronic disease and neurodegeneration

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Abstract

Interventions to the aging process involve early calorie restriction with appetite regulation connected to appropriate genetic mechanisms that involve mitochondrial biogenesis and DNA repair in cells. In the aging process as the anti-aging genes are suppressed as a result of transcriptional dysregulation chronic disease accelerates and is connected to insulin resistance and neurodegenerative. Interests in the gene-environment interaction indicate that the anti-aging gene Sirtuin 1 (Sirt 1) that regulates food intake has been repressed early in the aging process in various global populations. The connections between Sirt 1 and other anti-aging genes such as Klotho, p66shc (longevity protein) and Forkhead box proteins (FOXO1/FOXO3a) have been connected to lipid metabolism and alterations in these anti-aging genes regulate glucose, lipid and amyloid-beta metabolism. Appetite regulation by nutritional intervention is required early in life that involves Sirt 1 circadian clock gene expression with Sirt 1 maintenance of other cellular anti-aging genes involved in cell metabolism and apoptosis. Interests in anti-aging therapy with appetite regulation improves an individual's survival to metabolic disease induced by gene-environment interactions by maintenance of the anti-aging genes connected to the metabolism of cholesterol, bacterial lipopolysaccharides, drugs and xenobiotics. New discoveries in medicine are required to understand the importance of appetite regulation that is associated with the overconsumption of foodin Type 2 and Type 3 diabetes. Food restriction in diabetes is essential to maintain the hepatic metabolism of dietary fat with relevance to defective post-prandial lipid metabolism and to the global non alcoholic fatty liver disease (NAFLD) epidemic. Premature brain aging has become important with the development of Type 3 diabetesand Alzheimer's disease that is associated with repression of the anti-aging gene Sirtuin 1 (Sirt 1) relevant topost-prandial lipid metabolism, amyloid beta metabolism (peptide involved in amyloid beta plaques)and circadian rhythm abnormalities in the brain biological clock associated with the development of NAFLD.

Nutritional interventions such as very low carbohydrate diets have become important to diabetes to reverse defective post-prandial lipid and amyloid beta metabolism without atherogenic lipoprotein formation with the prevention of accelerated atherosclerosis in various communities. Western diets that are high in fat and glucose are linked to diabetes and NAFLD with anti-aging gene Sirt 1 transcriptional dysregulation in cell and tissues associated with, hyperglycemia, mitochondrial apoptosis and delayed hepatic fat and amyloid beta metabolism.

Dyslipidemia is one of the key risk factors for cardiovascular disease in diabetes. The management of dyslipidemia in diabetes continues to remain controversial and improvements in the characteristic diabetic dyslipidemia of high triglyceride and low HDL may not indicate that defective cell ontogeny is underway early in life. Diabetes and defective hepatic cell transcriptional programs induce delayed postprandial lipid metabolism associated with Western diets rich in fat and glucose. The clinical management of diabetes in the young and elderly now not only involves appetite regulation with calorie restricted diets that maintain the heat shock gene Sirt 1 expression but also careful core body temperature (37°C) to activate hepatic and brain Sirt 1. The biological active release of FGF21 is connected to Sirt 1 activation and glucose homeostasis with relevance to treatment of dyslipidemia, NAFLD, cardiovascular disease and neurodegenerative diseases. Consumption of Sirt 1 inhibitors such as alcohol, suramin and palmitic acid should be avoided to prevent defective liver and brain cell ontogeny in the young and the elderly to prevent hyperglycemia induced oxidative stress and myocardial infarction.

This work is partly presented at 2nd International Conference and Expo on Lipids: Metabolism, Nutrition & Health, October 03-05, 2016, Orlando, USA

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