

2nd International Conference and Exhibition on

Pharmacology and Ethnopharmacology

May 02-04, 2016 Chicago, USA



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Anti-aging genes improve NAFLD and type 3 diabetes in global populations

In global communities humans have become more susceptible to adiposity compared with other species with the increased development of overweight individuals, non alcoholic fatty liver disease (NAFLD) and Type 3 diabetes. Drugs that improve insulin resistance, dyslipidemia and the metabolic syndrome with long term treatment have not been successful as a result of the increased susceptibility to NAFLD with removal of various anti-obese drugs from the market. The increased cell senescence in diabetes has been associated with the limited ability of cells to divide with indication of telomere shortening and genomic instability of cells that is connected to cell suicide. Lipoaspirates from obese individuals allow assessment of anti-aging genes relevant to mitochondrial biogenesis and effective drug therapy will be determined by non consumption of inhibitors of anti-aging genes (drugs) and consumption of healthy low calorie diets that activate adipocyte anti-aging genes. Defective anti-aging genes in adipocytes are linked to mitochondrial apoptosis in obesity and indicate that these genes are associated with defective hepatic drug clearance and metabolism. Interests in the global epidemic in obesity and diabetes involve anti-aging therapies that stabilize accelerated aging and delay chronic diseases. New drug development needs to be carefully interpreted in relation to nutritional intake with drug safety concerns/adverse effects relevant to adipogenesis and NAFLD in obesity.

Biography

Ian James Martins is a reviewer for various journals and was appointed as the Chief Editor for Scientific and Academic Publishing (2013/2014). Research Gate's analysis of his publication stats places the RG score higher than 93% of the international researchers. He has been conferred with the Richard Kuhn Research Award-2015 Endocrinology and Metabolism.

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