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

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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.

Biography

Ian James Martins is a reviewer for various journals and was appointed as the Chief Editor for Scientific and Academic Publishing (2013/2014). He is a Fellow at Edith Cowan University/Honorary Senior Fellow of University Western Australia.

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