Geriatric medicine and heat shock gene therapy in global populations

Ian J. Martins
Edith Cowan University

Follow this and additional works at: https://ro.ecu.edu.au/ecuworkspost2013

Part of the Medicine and Health Sciences Commons

This Editorial is posted at Research Online.
https://ro.ecu.edu.au/ecuworkspost2013/3422
Geriatric Medicine and Heat Shock Gene Therapy in Global Populations

Martins IJ1,2,3*

1Centre of Excellence in Alzheimer’s Disease Research and Care, School of Medical Sciences, Edith Cowan University, Australia
2School of Psychiatry and Clinical Neurosciences, The University of Western Australia, Australia
3McCusker Alzheimer’s Research Foundation, Hollywood Medical Centre, Australia

*Corresponding author: Ian Martins, School of Medical Sciences, Edith Cowan University, 270 Joondalup Drive, Joondalup, Western Australia 6027, Australia, Tel: +61863042574; Email: i.martins@ecu.edu.au

Copyright: © 2016 Ian Martins. This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source.

Original Submission
Received: December 06, 2016
Accepted: December 15, 2016
Published: December 22, 2016

Open Peer Review Status: Editorials, news items, analysis articles, and features do not undergo external peer review.

How to cite this article: Ian Martins. Geriatric Medicine and Heat Shock Gene Therapy in Global Populations. Curr Updates Gerontol. (2016) 1: 2.1
Recent interests in the role of Sirt 1 as a heat shock gene in deacetylase) that targets various transcription factors involved in the nicotinamide adenine dinucleotide dependent class III histone deacetylase 1 (Sirt 1) to be defective in these populations [5]. Sirt 1 is a transcription repressor of HSF1 [3,10,11]. Geriatrics and brain temperature regulation may be defective with relevance to Sirt 1 gene repression by LPS [12] and induction of NAFLD with increased transport to LPS to the brain [5].

Major interests in geriatric medicine has accelerated with diet and lifestyles changes that may stabilize mitochondrial apoptosis [9] and organ diseases in these communities. In the developing world increased plasma LPS levels have raised alarm with relevance to thermoregulation disorders [7] connected to mitochondrial apoptosis relevant to NAFLD, myocardial infarction and various organ diseases [6]. Sirt 1 and the heat shock response involve the transcription factor p53/PGC1 alpha, various heat shock proteins and the heat shock transcription factor 1 important to neuron survival and insulin receptor pathways [7]. Sirt 1 regulation of HSF1 is via PGC1 alpha that is a direct transcription repressor of HSF1 [3,10,11]. Geriatrics and brain temperature regulation may be defective with relevance to Sirt 1 gene repression by LPS [12] and induction of NAFLD with increased transport to LPS to the brain [5].

Diets that contain fat may be metabolized rapidly in individuals (<65 years) compared with geriatrics with thermoregulation defects and defective fat metabolism [8,13]. Consumption of fats such as palm oil (palmitic acid rich) and virgin coconut oil (saturated fatty acids) [7] that are solid (20-24°C) may be sensitive to abnormal body temperature dysregulation with the induction of NAFLD (Figure 1) versus the consumption of olive oil (monounsaturated) that is liquid at a temperature (4°C). Dietary fat reduction restrict LPS absorption with relevance to Sirt 1/p53 interactions that are essential for uncoupling protein 1 (UCP1) expression [14] with Sirt 1 activators important to activation of thermogenesis related genes (PTEN, UCP1) [15].

In geriatrics the discovery of the heat shock gene Sirt 1 [3] has become important with relevance to the use of thermoregulation drugs that maintain the thermoregulatory set points in geriatrics [16]. Other drugs for depression and psychosis [17,18] may be inactivated with relevance to thermoregulation disorders with increased transport to the brain relevant to defective insulin therapy [19,20] that determines social interaction and behaviour . In geriatrics Sirt 1 is responsible for appetite regulation [6] and its loss from the suprachiasmatic nucleus in the hypothalamus of geriatrics result not only in central neural thermoregulatory dysregulation, circadian rhythm abnormalities but also loss appetite control (anorexia nervosa) [21]. Caffeine consumption [22] in geriatrics without NAFLD needs to be carefully determined to activate hepatic mitochondrial function that may improve post-prandial lipid metabolism (Figure 1) after consumption of meals that contain fat. However global Type 3 diabetes and neurodegenerative diseases may be irreversible with relevance to long term caffeine consumption.
in geriatrics. Caffeine may be relevant to accelerated neurodegeneration and critical changes to diet, lifestyles and thermoregulation are required to prevent caffeine induced neurodegeneration. Geriatrics and links to NAFLD [8] now indicate defective caffeine metabolism [23]. Caffeine has been used to improve mitochondrial thermogenesis [24,25] but with NAFLD defective caffeine metabolism over years increases CNS caffeine transport with relevance to p53 mediated mitochondrial death relevant to neuron apoptosis [26,27].

Conclusion

The global geriatric population by the year 2060 is expected to markedly increase and global death rate in geriatric individuals is predicted to rise sharply and associated with mitochondrial apoptosis in geriatric individuals with Type 3/Type 2 diabetes, NAFLD and neurodegenerative disease. The heat shock gene Sirt 1 is critical to geriatric medicine with relevance to appetite regulation, thermoregulation disorders and defective post-prandial lipid metabolism. Fat consumption such as palm oil/coconut oil should be carefully evaluated before consumption in geriatric individuals (thermoregulation disorders) with relevance to delayed metabolism of these fats (solid at body temperature) and the induction of NAFLD. Diet, drug therapy and lifestyle changes are a critical component for thermoregulatory adaptations that allow reversal of accelerated aging in geriatric individuals with the prevention of programmed cell death.

Acknowledgements

This work was supported by grants from Edith Cowan University, the McCusker Alzheimer’s Research Foundation and the National Health and Medical Research Council.

References


