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Regulation of Core Body Temperature and the Immune System Determines Species Longevity

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Abstract

The anti-aging gene Sirtuin 1 has now major relevance to genetics and the fields of pharmacology, toxicology, neuroscience, immunology, biochemistry and cell/molecular biology. Advances in anti-aging therapy are now essential to prevent mitochondrial apoptosis to promote longevity with the prevention of accelerated ageing. Calorie restriction that maintains the anti-aging genes changes the core body temperature and promotes species longevity. Stress and calorie consumption are sensitive to Sirt 1 function with relevance heat shock protein 70 metabolism and mitochondrial biogenesis. Sirt 1 regulation of the circadian rhythm mediates melatonin effects on core body temperature regulation and immune responses. Diet and fat are essential factors that determine species longevity with relevance to heat shock gene regulation and mitochondrial disease in animals and man. Strenuous exercise to activate the cellular heat shock gene in animals and man should be carefully controlled to prevent magnesium deficiency with relevance to immune disorders and mitophagy.

Keywords

Core Body Temperature; Mitochondria; Heat Shock Gene; Transcriptional Dysregulation; Sirtuin 1; Immune System, Anti-Aging; Diet; Longevity; Species; Melatonin; Fat; Magnesium; Circadian Rhythm; Heat Shock Protein

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Editorial

Anti-aging genes have major relevance to genetics and the fields of pharmacology, toxicology, neuroscience, immunology, biochemistry, cell and molecular biology. The gene-environment interaction identifies the low calorie gene Sirtuin 1 (Sirt 1) to be disrupted with effects on transcriptional regulation of other anti-aging [1,2] and responsive genes that are sensitive to accelerated mitochondrial apoptosis [3-5]. Sirt 1 defects lead to defective xenobiotic metabolism with effects on mitophagy [6] and corruption of the nuclear-mitochondria interaction [1] that determines species longevity with relevance to senescence and the universality of aging in man and various species [7]. Advances in anti-aging diets are required to maintain the critical breakthroughs in the fields of pharmacology [8] and immunology [9] that are essential for mitochondrial biogenesis linked to core body temperature and the prevention of Type 3 diabetes, accelerated ageing and neurodegenerative disease (Figure 1).

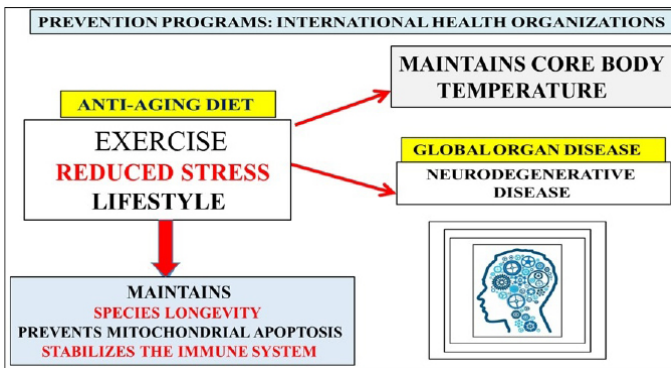


Figure 1: Genetics and genomics now identify the anti-aging gene Sirt 1 to be essential for life to maintain core body temperature and species longevity. Calorie restriction activates Sirt 1 but with the aging process Sirt 1 becomes repressed in animals and man with mitochondrial apoptosis and programmed cell death. Nutritional interventions and lifestyle changes stabilize the immune system with the prevention of global organ disease and neurodegeneration.

Research findings have reported that temperature variations in organisms have marked changes in metabolism with higher temperatures associated with increased ageing [10,11]. The observation that diets with calorie restriction change core body temperature has led to explanations for differences in species longevity [12]. In man and animals the circadian rhythm is critical to maintain body temperature and implicates Sirt 1 in suprachiasmatic nucleus (SCN) regulation [1,13] with relevance to the circadian rhythm, core body temperature [14,15] and the immune system [16]. Temperature dysregulation involves other anti-aging genes but identifies Sirt 1 as the heat shock gene [17] that is involved in the circadian regulation of heat shock proteins (HSP) 60,70 and 90 [18-20]. HSP are now linked to obesity, cardiovascular disease, adiposity, Type 3 diabetes (20,21) and Alzheimer's disease [22-25]. Stress and calorie consumption are sensitive to hepatic HSP 60,70 and 90 metabolism with relevance to Sirt 1 and its involvement in HSP 70 metabolism and mitochondrial biogenesis [26-31]. Heat shock gene

dysregulation is now important to pharmacology [8] with relevance to HSP induced mitochondrial apoptosis and not drug induced mitochondrial apoptosis [32].

Core body temperature regulation in man and various species implicates the pineal gland melatonin (Figure 2) as essential for the thermoregulatory effects in the hypothalamus and the periphery [33-35]. Sirt 1 regulates the SCN [1,13] in the hypothalamus with effects on melatonin secretion [36,37] relevant to Type 3 diabetes [20,21] in man and premature aging in animals. Sirt 1 regulates the circadian rhythm and mediates melatonin effects [35] on the SCN and core body temperature regulation [15,38,39]. Melatonin and its effects on the species longevity involves the immune system because of its essential role in the prevention of exacerbated immune responses [40,41] and its effect on Sirt 1 in immune and autimmunity regulation [9,16].

Diet and fat [42] are essential factors (Figure 2) that may determine species longevity with relevance to heat shock gene regulation and mitochondrial disease in animals and man. Appetite control is critical for mitochondrial biogenesis [43] but heat shock gene dysregulation may inactivate magnesium/Sirt 1 interactions with relevance to melatonin formation essential for mitochondrial biogenesis in mammals [44-46]. Melatonin reacts with peroxynitrite and nitric oxide to reduced toxicity to cells [47,48] and peroxynitrite can be referred to as Sirt 1 modulator [49] with relevance to Sirt 1's involvement in SCN regulation of nitric oxide [50] with relevance to core body temperature and gene expression [51,52]. Strenuous exercise to activate the cellular heat shock gene in animals and man should be carefully controlled to prevent magnesium deficiency with relevance to nitric oxide dysregulation and mitophagy [53-57].

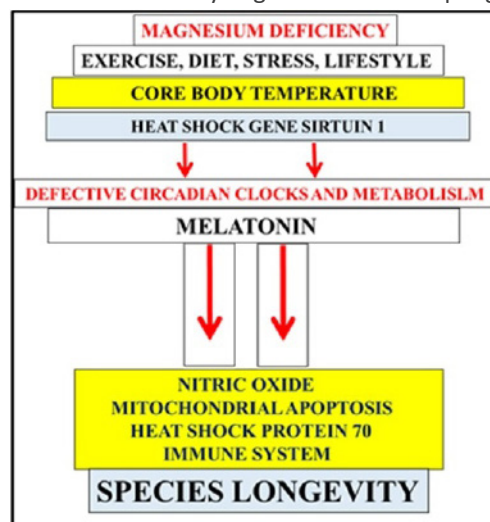


Figure 2: The discovery of the heat shock gene Sirt 1 indicates that diet, stress and lifestyle are critical factors that regulate melatonin (pineal gland) levels with relevance to core body temperature and species longevity. Heat/cold stress, intense exercise with high calorie diets inactivate the heat shock gene (Sirt 1) in animals and man with immune system imbalances, elevated HSP 70, nitric oxide dysregulation and magnesium deficiency connected to mitophagy.

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Conclusion

In developed and developing world the global epidemic for chronic disease may include many species such as animals and man. Western diets and environmental changes disrupt anti-aging processes that determine species survival and are responsible for malfunction in anti-aging genes with relevance to global non alcoholic fatty liver disease, obesity and diabetes (Type 2 and Type 3) epidemic. Advances in anti-aging therapy identify Sirt 1 as critical to breakthroughs in the fields of genomics, pharmacology and immunology essential for longevity and the prevention of mitochondrial apoptosis linked to accelerated ageing and Type 3 diabetes. Unhealthy diets and lifestyle changes prevent Sirt 1 effects on body temperature regulation with low melatonin levels and immune dysfunction associated with reduced species longevity. Dietary fat and appetite control are essential factors that determine species survival relevant to core body temperature and mitochondrial disease in animals and man. Strenuous exercise to activate cellular anti-aging processes in animals and man should be carefully controlled to prevent magnesium deficiency that induce immune changes related to mitophagy and cell death in various species.

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