Autoimmune disease and mitochondrial dysfunction in chronic diseases

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Defective anti-aging genes are responsible for programmed cell death in chronic disease.

Editorial

Anti-aging genes such as Klotho, FOXO 3a, p66shc and Sirtuin 1 (Sirt 1) are connected to various chronic diseases and repression of Sirt 1 is involved with the transcriptional dysregulation of other anti-aging genes (Klotho, p66shc, FOXO1/FOXO3a) that leads to abnormal regulation of glucose, lipid and amyloid beta metabolism associated with programmed cell death in various cell and tissues [1]. Anti-aging genes and their connections to autoimmune disease [2-5] and chronic diseases has attracted interest with relevance to irreversible programmed cell death in many tissues. Dietary effects with Sirt 1 downregulation [1] accelerate disease progression with autonomous disease connected to a defective immune system in various global communities [6].

Mitophagy has become of major concern to various chronic diseases [7-9] with accelerated mitochondrial apoptosis connected to the heat shock gene Sirt 1 [10]. Sirt 1 is involved in mitochondrial biogenesis and its connections to the other anti-aging genes via deacetylation of the transcription factor p53 have become important to mitochondrial biogenesis versus mitochondrial apoptosis. p53 is critical to the maintenance of the immune system [11,12] and its role mitochondrial function is connected to the various anti-aging genes. Sirt 1 is now connected to autoimmune disease [6] and its regulation of p53 and the anti-aging genes (FIGURE 1) may be the primary defect with irreversible consequences to cell biology and programmed cell death involved with the many chronic diseases in the developing and developed world.

The origin of chronic disease that involve the autoimmune process and abnormal immune responses are connected to mitochondrial disease and have raised concerns with relevance to irreversible processes that may now be involved in non alcoholic fatty liver disease (NAFLD), obesity, diabetes, multiple organ disease syndrome and neurodegenerative diseases (FIGURE 1). Analysis of plasma heat shock proteins (HSP) is now essential to avoid uncontrolled immune reactions connected to the repression of the heat shock gene Sirt 1 that is responsible for HSP metabolism [13]. In the normal aging process HSP accumulate with autoimmune reactions and mitophagy connected to the progression of age related diseases [13-17]. The relevance of abnormal core body temperature [13] has become of major concern to the early origins of chronic disease with the critical maintenance of body temperature that implicates that anti-aging gene Sirt 1 in core body temperature and immune system regulation. The irreversible induction of HSP mediated programmed cell death should be avoided in various chronic diseases with careful maintenance of the suprachiasmatic nucleus [13] in the brain that is involved with core body temperature regulation [14,18] in many neurodegenerative diseases and connected to peripheral organ...
The alterations in the light/dark cycle [19,20] may be the primary factor involved in the induction of HSP alterations connected to autoimmune disease and mitophagy. Anti-aging genes and their connections to autoimmune disease mitophagy now identify Sirt 1 to be defective with increased HSP levels involved in autoimmune disease and mitophagy connected to irreversible programmed cell death in many organ diseases in global populations.

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References