

The Role of PGC1-1a in Modulating Oxidative Stress, Cardiovascular Risk Factors, and Cellular Metabolism in Cardiomyocytes

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Received September 1, 2020 Accepted September 20, 2020 Published online September 30, 2020



Dear Editor,

Oxidative stress is characterized with an imbalanced and excessive production of free radicals. To scavenge these radicals, the activity of antioxidant defense mechanism is critical. Oxidative stress is considered to be a main pathogenic process in a variety of disorders, particularly cardiovascular diseases.

In addition, obesity is a rising challenge in today's world, and is closely related to immobility and unhealthy diets. Obesity has been recognized in association with various diseases, including neuronal (movement or cognitive), neoplasms, cardiovascular, and metabolic abnormalities.¹

Hydrogen peroxide is an oxidative mediator contributing to oxidative stress and the associated biological toxicity. This agent has a role in releasing Ca^{2+} from internal cellular storage (sarcoplasmic reticulum) mediated via ryanodine receptors, inducing cardiomyocyte damage. Thus, inactivation of antioxidant defensive mechanisms or excessive production of reactive oxygen species can lead to oxidative stress.^{1,2}

Mortality from cardiovascular diseases is influenced by the presence of metabolic syndromes (e.g. obesity, hypertension, and diseases associated with excess body weight). The incidence of metabolic diseases is rising in many countries around the globe, including Asia. This necessitates taking measures to identify and control risk factors of metabolic syndrome to be able to manage and prevent other chronic conditions, such as cardiovascular diseases and diabetes.

During high intensity interval training (HIIT), studies have shown that peroxisome proliferator-activated receptor-gamma coactivator (PGC-1 α) is an important player in mitochondrial biogenesis. In fact, the beneficial roles of aerobic function can be in part related to such

changes in the biology of mitochondria. PGC-1 α is an important nuclear receptor, which interacts with multiple transcription factors in regulating gene expression during cell signaling processes. Environmental factors, particularly physical activity and exercise, can induce PGC-1 α and other related molecules in the family. These molecules then execute a variety of roles in regulating cellular energy expenditure, glucose, and lipid metabolism. They are also supposed to be involved in the pathogenesis of such diseases as diabetes, neuronal degeneration, cardiac diseases, and obesity.

The expression of PGC-1 α gene is increased, mediated via phosphate-dependent PGC-1 α activation pathways (through calmodulin-dependent kinases activated by secondary messengers of calcium and cAMP), following HIIT and exercise, which subsequently induce the genes and proteins involved in mitochondrial respiratory chain reactions. Subsequently, elevated PGC-1 α expression can augment the number and function of mitochondria and cellular metabolism in cardiomyocytes, and help to fight oxidative stress in these cells.³

Ethical Approval

Not applicable.

Conflict of Interest Disclosure

There is no conflict of interests in this study.

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Please cite this article as follows: Ahmadi A. The role of PGC1-1a in modulating oxidative stress, cardiovascular risk factors, and cellular metabolism in cardiomyocytes . *Int J Basic Sci Med.* 2020;5(3):66-67 doi:10.34172/ijbms.2020.13.