ISSN: 2690-3164

Martinez et al. Int Arch Vasc Med 2021, 4:011
DOI: 10.23937/2690-3164/1710011
Volume 4 | Issue 1
Open Access

International Archives of
Vascular Medicine

COMMENTARY

Mitochondrial Dysfunction and Insulin Resistance: Topic of High Interest in Research

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Insulin resistance is a condition derived from the disorder in the use of substrates for metabolic activities [1]. It can be caused by chronic hyperinsulinemia, inflammation, and long-term use of corticosteroids. *In vivo* and *in vitro* models have revealed that this process is secondary to the excessive production of mitochondrial oxidants, a product of mitochondrial dysfunction, even in cases where mitochondrial respiration is conserved [2]. Insulin increases the number of type 4 glucose transporters (GLUT4) on the cell surface, via the phosphatidylinositol 3-kinase/Akt (PI3K/Akt) signaling pathway, producing the appropriate intra-cellular glucose transport for use [3]. This process is affected in insulin resistance, becoming a potential risk factor for chronic non-communicable diseases such as type 2 Diabetes Mellitus, Obesity, Coronary Heart Disease, Non-alcoholic Fatty Liver Disease, Atherosclerosis, among many others [4].

Taking into account that these diseases have a very high global prevalence, leading to high morbidity and mortality rates, in addition to having high economic costs for therapeutic expenses, incapacitation, and disability [5]; the clinical, cellular, molecular and genetic study, has become a priority in the area of biomedical research, to find the metabolic pathways involved in these pathophysiological mechanisms, which facilitate the design of diagnostic tools and therapeutic targets, to optimize the approach and improve the survival and quality of life of these patients [6].

The relationship between mitochondrial dysfunction and insulin resistance has been a topic of research interest in the biomedical area that has grown considerably during the last decade, a product of technological advances that have made it possible to go further in cellular and molecular evaluation [7]. To the extent that it is clearly described that this pathological process is generated liver cells, skeletal muscle, brain, adipose tissue, among others [4]. In the clinical context, it is essential to know the systemic impact of insulin resistance, since it allows establishing a more accurate diagnosis and prognosis on the real situation of those individuals at risk. In those patients who have isolated insulin resistance, significant changes have been found regarding the accumulation of fatty acids in liver and muscle tissue, inflammatory markers, microbiota, epigenetic interaction, and response to endoplasmic reticulum stress [8-10]. Besides, since mitochondrial dysfunction is the product of mitochondrial defense mechanisms, which include changes in distribution, fusion, fission, and specific activation of internal pathways [11], ultimately leading to mitophagy, the decrease in the number of mitochondria, and consequently of the cellular lesion by an increase of reactive oxygen species is presumed, since there is an alteration within the system of regulation of oxidants/antioxidants, the dysfunction is systemic [7,12].

Promising studies have been made, where it has been possible to evaluate the activity of various substances

Citation: Martinez IL, Llinás DT, Rivera JA, Valverde JJ, Berrio MC (2021) Mitochondrial Dysfunction and Insulin Resistance: Topic of High Interest in Research. Int Arch Vasc Med 4:011. doi.org/10.23937/2690-3164/1710011

Accepted: December 04, 2021: **Published:** December 06, 2021

against the cell lesion produced by insulin resistance, these studies show that it is possible to suppress oxidative pathways, preventing and significantly controlling damage to tissues primarily affected [13,14]. It is necessary to design and carry out studies of the best quality, which evaluate the effectiveness of substances related to mitochondrial and cellular protection, that promise to be therapeutic targets that not only serve for cardiometabolic diseases but, that also impact on conditions where no association has even been found.

Financial Support

None.

Conflict of Interest

None.

Authors Contribution

All authors have contributed for this manuscript.

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