



COMMENTARY

Mitochondrial Dynamics and Cardiovascular Risk: An Insight at the Molecular Level

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Mitochondria are cellular organelles; whose function goes beyond generating adenosine triphosphate (ATP) [1]. They are part of the machinery involved in the production of substances related to iron, sulfur, and heme group, involved in lipid metabolism, calcium homeostasis, heat generation, cell signaling, stem cell pathway determination, and apoptotic processes [2]. A vast variety of pathologies have been related to mitochondrial deficit, within these, cardiovascular diseases. Mitochondrial dysfunction is a physiological process that goes hand in hand with aging, however, the modifiable cardiovascular risk factors are described today (overweight/obesity, smoking, alcohol consumption, consumption of psychoactive substances, sedentary lifestyle, high-calorie diet, among others) overwhelmingly accelerate this process [3]. Proteomics is a field of molecular and biochemical biology that evaluates the structure and function of proteins [4], and this has allowed great advances in the compression of mitochondrial dynamics.

Mitochondrial dynamics integrate concepts such as mitochondrial movement through the cytoskeleton, changes in mitochondrial morphology, distribution, and connectivity, which influence mitochondrial fusion and fission [5]. The disruption between the balance of fusion and fission events produces cellular dysfunction with subsequent apoptosis, which stimulates the activation of fibrogenic repair pathways, constituting the first step in the loss of organic functionality and genesis of many diseases, such as neurodegenerative disorders, type 2 diabetes mellitus and heart failure [6].

From a physiological perspective, it is understandable to expect mitochondrial functional deterioration to be directly proportional to human aging, which makes the cardiovascular system more vulnerable to cellular stress, and thus explains the evolution of subclinical/clinical cardiovascular diseases in older ages [7]. Finally, cardiovascular deterioration is manifested as the loss of the ventricular contractile capacity, with the subsequent development of hypertrophy, which leads to a decrease in ventricular distension and prolongation of the diastole; vascular rigidity, and endothelial dysfunction [8].

The heart specifically, high bioenergetic demanding organ, and more than 90% of the ATP needed to perform its functions properly, is produced by mitochondria [9]. The adult cardiomyocyte has about 6000 mitochondria, and these constitute up to 40% of the cellular volume. In the cardiomyocyte, there are three types of mitochondria, perinuclear, subsarcolemmal, and interfibrillar, which are interconnected through fusion and fission proteins [9]. These organelles' strict organizations allows the adequate substance flow regulating the cellular process, which controls the balance of reactive oxygen species, cell motility, and specifically in the heart, heart rate since they are involved in cellular calcium circulation [10]. That is why mitochondrial dysfunction marked by the presence of modifiable risk factors, affect white organs such as the heart and the endothelium in such a way since they fragment the mitochondrial metabolic pathways' integrity and induce adaptive mechanisms resulting from cell destruction, resulting in arrhythmias,



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atherosclerosis, and alteration in cardiac morphology [11].

Mitophagy is a specialized form of autophagy, where mitochondria are selectively degraded and recycled. Mitophagy and mitochondrial dynamics are unique in the heart [12]. The decrease in the mitochondrial division is strongly associated with an increase in mitophagy as a defense mechanism, resulting in a substantial and irreparable loss of cellular and molecular regulation machinery. This suggests that the balance between the processes of fusion and division, are more important than either of these two individually [13], and can be the objective of research for future therapeutic targets, which seek to slow the process of cell deterioration, not only in the heart but in a variety of organs.

The evidence shows that mitochondrial dynamics is an indispensable process in cell physiology, being its dysfunction, responsible for the metabolic alteration, cell death, and disease genesis. From a cardiovascular perspective, it is a very wide field of research where you can look for answers about these unknown processes, and generate future therapeutic targets that allow reducing cardiovascular risk, or even, avoid accelerating the process of vascular aging, and with this, increase life expectancy and improve the quality of life of humans.

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Conflict of Interest

None.

Authors Contribution

All authors have contributed for this manuscript.

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