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Mitochondrial dysfunction and reactive oxygen species in diseases of cardiac and skeletal muscles

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Functional and structural abnormalities of mitochondria are involved in the majority of cardiac and skeletal muscle pathologies. Indeed, mitochondrial dysfunction is causally related to both contractile impairment and loss of viability. Consequently, interventions aimed at maintaining mitochondrial function elicit a significant protection against various pathological conditions, especially in the case of injury induced by ischemia and reperfusion.

These concepts will be reviewed highlighting the role of reactive oxygen species (ROS) that are both a consequence and a cause of mitochondrial derangements. In this respect, the various mitochondrial sources of ROS will be discussed focusing on monoamine oxidases. On the other hand, among the many targets of ROS that are involved in myocyte injury, the role of oxidative stress in causing both the opening of the permeability transition pore and contractile alterations will be reviewed.

Finally, while the presentation will focus mostly on pathological conditions, room will also be given to the physiological roles of ROS and their involvement in endogenous self-defense mechanisms. In particular, the role of mitochondrial Ca^{2+} uptake will be analyzed as a process involved in both cell death and cardiac protection.

Short biography

Prof. FABIO DI LISA

Fabio Di Lisa has provided significant contributions elucidating the role of mitochondrial dysfunction in cardiac diseases. He found that the mitochondrial membrane potential is maintained during anoxia using ATP produced by glycolysis, so that mitochondria changes from ATP producers into avid ATP utilizers. By developing methods to study the PTP in isolated cells and intact hearts Prof. Di Lisa characterized the occurrence of transient and prolonged openings demonstrating that the latter modality is involved in cell death. In addition, PTP opening was causally related to NAD depletion and loss of viability induced by reperfusion. Regarding oxidative alterations, Prof. Di Lisa demonstrated that the oxidation of myofibrillar proteins correlates linearly with contractile impairment. This relationship has been extended to muscular dystrophy. Concomitantly, he demonstrated the relevance of monoamine oxidases (MAO) as a relevant source of reactive species in cardiac and muscular diseases.

Positions and Honors (chronological order)

1980-1992 Researcher, National Council of Research (CNR)
1987 NATO scholarship (Michigan State University)
1991-1992 Visiting Associate (NIH)
1992-1995 Associate Professor, University of Catania, Faculty of Medicine
1995-2002 Associate Professor, University of Padova, Faculty of Pharmacy
2002- present Full Professor, University of Padova, Faculty of Pharmacy
2003- 2007; 2013- present Chairman of the PhD course in Biochemistry and Biophysics, University of Padova
2002-2005 President Elect, European Section of the International Society for Heart Research
2005- 2008 President, European Section of the International Society for Heart Research
2007 Fellow of the International Society for Heart Research
2014 Keith Reimer Award of the International Society for Heart Research

Editorial activities

Editorial Board member: Journal of Biological Chemistry (2003-2008; 2013-); Cardiovascular Research (2003 -); Journal of Molecular and Cellular Cardiology (2004 -); Basic Research in Cardiology (2003 -)
Ad hoc reviewer: American Journal of Physiology, Cardiovascular Research, Circulation Research, Biochimica et Biophysica Acta, FEBS Letters.

Biblometric index

- H-index: 51 (Google Scholar)

- Total citations: 9394

Relevant publications

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