1. Record Nr. UNINA9910227350203321

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Titolo	Mitochondria: Hubs of Cellular Signaling, Energetics and Redox Balance
Pubbl/distr/stampa	Frontiers Media SA, 2017
Descrizione fisica	1 electronic resource (228 p.)
Collana	Frontiers Research Topics
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia

Sommario/riassunto

Poised at the convergence of most catabolic and anabolic pathways, mitochondria are the center of heterotrophic aerobic life, representing a hub in the overall metabolic network of cells. The energetic functions performed by mitochondria face the unavoidable redox hurdle of handling huge amounts of oxygen while keeping its own as well as the cellular redox environment under control. Reactive oxygen species (ROS) are produced in the respiratory chain as a result of the energy supplying function of mitochondria. Originally considered an unavoidable by-product of oxidative phosphorylation, ROS have become crucial signaling molecules when their levels are kept within physiological range. This occurs when their production and scavenging are balanced within mitochondria and cells. Mitochondria-generated hydrogen peroxide can act as a signaling molecule within mitochondria or in the cytoplasm, affecting multiple networks that control, for example, cell cycle, stress response, cell migration and adhesion, energy metabolism, redox balance, cell contraction, and ion channels. However, under pathophysiological conditions, excessive ROS levels can happen due to either overproduction, overwhelming of antioxidant defenses, or both. Under oxidative stress, detrimental effects of ROS include oxidation of protein, lipids, and nucleic acids; mitochondrial depolarization and calcium overload; and cell-wide oscillations mediated by ROS-induced ROS release mechanisms. Mitochondrial dysfunction is central in the pathogenesis of numerous human

maladies including cardiomyopathies and neurodegeneration. Diseases characterized by altered nutrient metabolism, such as diabetes and cancer, exhibit elevated ROS levels. These may contribute to pathogenesis by increasing DNA mutation, affecting regulatory signaling and transcription, and promoting inflammation. Under metabolic stress, several ionic channels present in the inner and outer mitochondrial membranes can have pro-life and -death effects. In the present E-book, based on the Frontiers Research Topic entitled: "Mitochondria: Hubs of cellular signaling, energetics and redox balance", we address one of the fundamental questions that the field of ROS biology faces today: how do mitochondria accomplish a reliable energy provision and at the same time keep ROS levels within physiological, non-harming, limits but crucial for cellular signaling function? Additionally, and within the perspective of mitochondria as signaling-energetic hubs in the extensive cellular metabolic network, we ask how can their collective dynamics scale from the subcellular to the cellular, tissue and organ levels to affect function in health and disease.