Li Zuo, J Pulm Respir Med 2016, 6:5(Suppl)
http://dx.doi.org/10.4172/2161-105X.C1.015

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Reactive Oxygen Species: from Respiratory muscle to the Heart

Reactive oxygen species (ROS) play critical roles in biological systems. ROS formation is closely associated with infection, inflammation, ethanol toxicity, cell death, and cell signaling cascades. Respiratory and cardiac muscles have been shown to generate considerable ROS under various conditions. In this study, we tested the hypothesis that increased ROS production in diaphragmatic and heart muscles is associated with exposure to heat stress, hypoxia and ultra-fine particle exposure, all of which may play a role in muscle function and regulatory activity.

Results demonstrate: 1) ROS formation is a natural response of the diaphragm to heat exposure; 2) ROS release is not directly linked to NADPH oxidase, mitochondria, or anion channels in diaphragm in heat stress; 3) Nitric oxide synthase, lipoxygenase, and phospholipase A2 are all associated with ROS release in respiratory muscle; 4) Hydrogen peroxide is largely induced by acute hypoxia in respiratory muscle; 5) Particle exposure causes marked dysfunction of isolated cardiomyocytes via ROS formation. These results suggested that ROS profoundly affects skeletal and cardiac muscle physiology in various stress models.

Biography

Li Zuo obtained his PhD at Ohio State University (OSU). Currently, he is an Assistant Professor and Director of the Molecular Physiology and Rehabilitation Research Lab at OSU Medical center. He authored and editted over 140 original papers, abstracts, book chapters, review articles and edited publications. He is serving as associate editor for Frontiers in Physiology (the 2016 #1 most cited open-access journal in Physiology with an impact factor of 4.031). He also earned recognition as a fellow by American College of Sports Medicine (ACSM) in 2015 and won S&R Foundation Ryuji Ueno Award, the largest American Physiological Society (APS) award in 2016.

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