J Bioenerg Biomembr (2016) 48:97
DOI 10.1007/s10863-014-9596-2

Introduction to the special issue of the journal of bioenergetics and biomembranes: "mitochondrial lipids: essential roles in physiology and disease."

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Published online: 23 December 2014

Mitochondria are the most densely membraneous organelles in the cell. For this reason the lipids that comprise these membranes have an outsized impact on the numerous functions of this versatile intracellular particle. In this issue of the Journal of Bioenergetics and Biomembranes we focus on mitochondrial lipids in both the normal and pathophysiological states: their synthesis, metabolism, function, and vulnerability to misregulation and damage. Mitochondria produce ATP and other metabolic intermediates, conduct lipid metabolism, serve as a calcium store, act as signaling nexus for apoptosis, and compartmentalize an ever-increasing list of signaling pathways, among many other functions. All of these depend on the two membranes of mitochondria to function precisely and thus there is a heavy reliance on the appropriate lipid composition of the inner and outer mitochondrial membranes. It is no surprise that the ancient prokaryotic origin of mitochondria is reflected in the unusual lipid composition of those membranes, enriched in the four acyl-chained phospholipid cardiolipin and dearth of cholesterol. In this Issue Edgard M. Mejia and Grant M. Hatch (University of Manitoba) provide a guide to the diversity of lipids that make up the inner and outer membranes of mitochondria and touch on the functions that are dependent on those lipids. The unique role that cardiolipin plays in mitochondrial function is explored by Cunqi Ye, Ph.D., Zheni Shen, and Miriam L Greenberg (Wayne State University), the specificity of which is illuminated in part by the consequences of Barth syndrome, in which a defect in cardiolipin fatty acid remodeling has catastrophic consequences. Lena Böttinger, Lars Ellenrieder, and Thomas Becker (University of Freiberg) delve into how the lipid composition of the inner and outer membranes impacts on the astonishingly efficient and precise processes that accomplish the import of proteins into the multiple mitochondrial compartments. Although mitochondria are traditionally thought of as a cholesterol-poor organelle, Laura A. Martin, Barry E. Kennedy, and Barbara Karten (Dalhousie University) explore how cholesterol is imported into mitochondria not only for the well-characterized generation of steroidal cholesterol metabolites, but also for the normal function of the mitochondrial membranes. Sphingolipids, comprising a potent lipid signaling family, are not traditionally thought of being important in mitochondrial function. But the emerging understanding of the life and death-determining functions of sphingolipids in mitochondria is addressed by Gauri A. Patwardhan, Levi J. Beverly and Leah J. Siskind (University of Louisville). They describe that sphingolipids, such as the ceramides and sphingosines, have profound physiological roles in triggering the apoptotic process emanating from mitochondria. Edward D. Hall has devoted his career to understanding how lipid oxidation in neuronal injury lies at the center of the pathogenic consequences of injury in these tissues. Along with his colleagues Juan A. Wang, Jeffrey M. Bosken and Indrapal N. Singh (University of Kentucky), Dr. Hall summarizes years of work on the mechanism and consequences of lipid peroxidation in mitochondria resulting from ischemia and spinal cord injury.

Evolution has pushed the role mitochondria far past the early symbiotic relationship of the mitochondrial ancestor in producing ATP. Each new mitochondrial function capitalizes on the compartmental nature of this organelle and thus is intimately dependent on the proper function of the membranes. The articles in this issue are designed to illustrate how the mitochondrial lipids have adapted to and become incorporated into the mechanisms driving these additional roles of mitochondria. These changes are reflected in the pathological consequences of alterations in mitochondrial lipid composition and highlight how an understanding of these lipids opens opportunities to intervene in the generation of pathological states.

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