A Special Issue on the cell-specific roles of mitochondrial Ca^{2+} handling

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The importance of mitochondria for cellular function is universally acknowledged. It is therefore not surprising that there are plenty of excellent review articles (for example, 2) explaining the complex transport pathways that are essential for mitochondrial function. Very recently, major progress has occurred with regard to identification of the calcium influx and efflux machineries in the mitochondria [4]. The purpose of this Special Issue is—on the basis of the best and up-to-date available knowledge of the general mitochondrial characteristics—to describe and discuss the astonishing range of challenges that mitochondria face in different cell types and how these have been solved by evolution in a variety of ways.

The first three articles in this collection deal with general issues. Tullio Pozzan and his collaborators, who have done so much to reveal the physiological and pathological importance of mitochondrial Ca^{2+} uptake, review the latest developments in our understanding of mitochondrial Ca^{2+} homeostasis [12]. Damon Poburko and Nicolas Demaurex review the important topic of how the mitochondrial proton gradient is regulated by the cytosolic Ca^{2+} signals [13], whereas Anant Parekh and his colleagues deal with the crucially important topic of interactions between the mitochondria, the endoplasmic reticulum,

and the store-operated Ca^{2+} influx channels [1]. These issues are critical to the understanding of mitochondrial function in a number of specialized tissues.

The bulk of the articles in this Special Issue deal with mitochondrial functions under physiological and pathophysiological conditions in chromaffin cells [6], adrenal glomerulosa cells [14], smooth muscle cells [10], endothelial cells [8], neurons [5], pancreatic acinar cells [7, 11], hepatocytes [3], and cardiac myocytes [9]. These articles offer insights into some intriguing and partly unexpected relationships between aspects of mitochondrial function in cell types with very different functions. Thus, the ability of mitochondria to sequester Ca^{2+} also at low submicromolar cytosolic Ca^{2+} concentrations, identified in the endocrine aldosterone-secreting adrenal glomerulosa cells [14], turns out also to be of great importance for the epithelial pancreatic acinar cells, where the peripheral mitochondria only experience very modest rises in the local cytosolic Ca^{2+} concentration during store-operated Ca^{2+} entry, but nevertheless need to take up Ca^{2+} in order to activate the local ATP generation which is crucially needed to fuel the active Ca^{2+} uptake into the basal endoplasmic reticulum [11]. In stark contrast, the peripheral mitochondria in chromaffin cells experience large increases in the local cytosolic Ca^{2+} concentration during activation of voltage-activated Ca^{2+} channels, therefore only needing low-affinity mitochondrial Ca^{2+} uptake mechanisms [6]. Indeed, it might be undesirable—from the point of view of the physiological function of these cells—to have high-affinity mitochondrial Ca^{2+} uptake mechanisms. We hope that these, and many other insights into tissue-specific mitochondrial functions, will be helpful to all those interested in understanding how mitochondria can fuel the most vital processes in specialized cells, but nevertheless also play crucially important roles in a number of important disease processes.

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