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Mitochondria in Pulmonary Disease: From Molecular Mechanism to Medicine

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Message from the Guest Editors

In lung diseases, mitochondria have become the focus of attention owing to their role as a central hub for the integration and transformation of cellular signals, thereby regulating, e.g., inflammation, proliferation, apoptosis, and contraction. In addition, mitochondria act as primary sensors for hypoxia and allow for the availability of substrates that are utilized for ATP production. Mitochondrial release of reactive oxygen species, interference with intracellular calcium homeostasis, and modulation of metabolic intermediates play an important role in the development of various pathologies, e.g., pulmonary hypertension, emphysema, pulmonary fibrosis, and lung cancer, but also in physiological signaling processes such as hypoxia-dependent signaling and the activation of immune cells.

The aim of this Special Issue is to present clinical and experimental scientific reports that provide a deeper understanding of the physiological and pathological mitochondrial signal processes that allow for the development of new treatment strategies and the implementation of experimental concepts in clinical practice.

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