Special Issue

Pathogenesis of Pulmonary Arterial Hypertension: Differences in Molecular Mechanisms Between Neonatal and Adult Pulmonary Hypertension

Message from the Guest Editor

Pulmonary arterial hypertension (PAH) is a syndrome with multiple etiological agents. It is a pathology of great complexity as it progressively induces a sustained increase in pulmonary arterial pressure. In addition, its etiological origins at different ages in humans are even more diverse; in the neonatal period, the pulmonary arteries show a decrease in the thickness of the inefficient arterial wall and increased vasoconstrictor reactivity, whereas in adults, the gradual thickening of the arterial wall is observed. Molecular research on PAH aims to unravel its underlying mechanisms and identify possible therapeutic targets.

Recent studies highlight the fundamental role of genetic mutations, epigenetic modifications, and variations within signaling pathways involved in PAH's pathogenesis. Sex-associated changes may also significantly influence PAH.

Classical signaling pathways associated with vascular reactivity and remodeling are essential for maintaining vascular integrity and efficient function. The dysregulation of these pathways results in vasoconstriction and the proliferation of endothelial and smooth muscle cells, which are characteristic of PAH.

Guest Editor

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