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Advance in Molecular Pathology of Pulmonary Hypertension

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Message from the Guest Editor

Pulmonary hypertension (PH) is a serious sequele of several systemic and genetic diseases, lung developmental and heart defects, and drug toxicity. PH is associated with inflammation, altered vascular relaxation responses, cellular phenotype changes, and cell proliferation and migration. The sustained cell proliferation and apoptosis resistance, abnormal angiogenesis, and dysregulated cellular metabolism observed in PH are reminiscent of cancer. The molecular mechanisms involved in the development and progression of PH is not yet fully understood. However, recent advances in the field have described several important aspects such as the deregulation of multiple genes and protein expressions, mitochondrial dysfunction, autophagic dysregulation in pulmonary endothelial and smooth muscle cells, and the endothelial–mesenchymal transition in PH. Mitochondrial dysfunction is thought to contribute to the metabolic alterations observed in PH. These responses promote the survival of proliferating cells. A number of these molecules function as a double-edged sword. They maintain homeostasis under normal conditions but facilitate cell proliferation and migration in the disease state.

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Message from the Editor-in-Chief

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