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Pulmonary Vascular Remodeling: Cellular and Molecular Mechanisms

Guest Editor:

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Deadline for manuscript submissions:
closed (15 July 2020)

Message from the Guest Editor

Dear Colleagues,

Pulmonary hypertension (PH) can manifest in its standalone idiopathic form or be associated with chronic lung disease, where even a mild elevation of pulmonary arterial pressure is associated with poor prognosis. The current consensus is that vascular remodeling arises from a dysfunctional endothelium and the perturbed crosstalk between other resident structural cell types, including pericytes, smooth muscle cells, and fibroblasts. Recruited inflammatory cells can actively affect remodeling by releasing potent signaling molecules such as growth factors, cytokines, and enzymes and thereby alter vascular homeostasis. However, many of the mechansims that govern cell accumulation or mediate cellular cross-talk are still unidentified. Therefore, delineating this cross-talk and communication between diverse cell types and involved signaling processess is crucial to better understanding remodeling and bring us towards more targeted therapies, which can be specifically applied in different forms of PH. This Special Issue focuses on multiple aspects that govern vascular remodeling, and especially the interaction between different resident cell types and immune cells.

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Message from the Editorial Board

Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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