Inflammatory disease of the lung vascular bed is a major cause of morbidity and mortality in both adult and pediatric age groups. A particularly devastating consequence of lung inflammation is lung injury, which alone accounts for 150,000 cases annually in the United States, and carries a mortality rate of more than 30%. Yet, in the teaching about lung inflammation and in its application to clinical practice, the importance of vascular biology has been somewhat neglected.

Lung inflammation results from the defensive responses of pulmonary vascular cells to pathogenic stimuli. The responses arise through signal transduction mechanisms, which constitute sequences of intracellular events that lead up to specific cellular responses. Secondary effects of such responses precipitate all of the phenotypic features of lung inflammatory disease, including vascular hyperpermeability, white cell accumulation, and vascular remodeling. An understanding of signal transduction pathways in lung vascular cells is therefore required not only to explain the processes of lung inflammation, but also to develop new therapeutic strategies to combat inflammatory lung diseases.

Although great strides have been made in the science of cell signaling, much of this understanding is derived from nonlung cells. Consequently, the understanding is often of tangential relevance to lung vascular biology. The lung’s unique position at the systemic interface with the environment arms it with a sensitive immune defense capability, a physiologically protective feature that also carries significant pathological risk. This and other special features of the pulmonary circulation compel a studied and specific consideration of signal transduction processes in the context of lung vascular disease. It is my hope that Cell Signaling in Vascular Inflammation will foster better awareness of these phenomena.

My intention in assembling these chapters has been to cut across disciplines to bring together a broad-based presentation of inflammatory challenge, both in the initial phases of the inflammatory response as also in the more prolonged phase of genomic involvement. The chapters comprise a comprehensive survey of signaling processes. Hence, the book will be useful to a broad spectrum of readers, including advanced students of lung biology, investigators seeking new research directions, and clinicians and scientists involved in lung inflammation and its management.

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Jahar Bhattacharya, MBBS, DPhil
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