

BOOK REVIEWS

Cell Signaling in Vascular Inflammation

Edited by J. Bhattacharya

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This book is largely a collection of papers that were presented at an American Heart Association sponsored symposium. Although the title of the book broadly refers to “vascular” inflammation, the objective is to educate the reader in “lung inflammation”. This is stated in the Preface by the editor. For those interested in “lung biology”, this book is relevant. However, it is a collection of articles, such as protein modifications by reactive nitrogen species and signalling by mitochondria to general endothelial cell barrier function (chapters 7, 9, 14 and 20, respectively), which are written by experts. Most of the chapters are well illustrated with diagrams and flow charts and are generously referenced. Some of the chapters provide rather general reviews of a particular signalling pathway, relying on the reader to make the connection to the vasculature. This may not necessarily constitute a weakness, but it demonstrates how little lung- and lung vessel-specific knowledge has been accommodated.

Two of the chapters deserve to be highlighted. The first is by Wolfgang Kuebler, who summarises elegantly, actually exploring intact integrated lungs, the pro-inflammatory effects of hydrostatic stress. He convincingly demonstrates pulmonary vascular pressure and how stretch activates endothelial cells, which in turn secrete the contents of their Weibel-Palade bodies, resulting in a pro-inflammatory intravascular milieu. The real significance of the hydrostatic pressure/inflammation concept developed by W. Kuebler may be the transformed pulmonary microcirculation in the millions of patients worldwide suffering from chronic congestive heart failure.

The second chapter is by Aron Fischer, who suggests a new paradigm to explain the response of the lung vessels to ischaemia, simply as a response of the endothelium to cease shear stress, whereby depolarisation of endothelial cells leads to inactivation of K_{apt} channels and activation of the NADPH oxidoreductase. It is being suggested that the K_{apt} channel may serve as a flow sensor. Interestingly, if one reads Kuebler’s and Fisher’s chapters back-to-back, one may come to the conclusion that shear stress is part of the secret of the health or disease of perfused organs, with too little or too much shear stress causing inflammation.

While some parts of this book are more enjoyable to read than others, an opportunity has been missed to critically define the elements of “lung vascular inflammation”, which cannot be conceptualised without involvement of the immune system (both innate and adaptive), and to synthesise the many thousands of parts of signalling components. As we move forward, the task of integration of information becomes harder, but all-important. After all, just because one can show activation of a particular signalling cascade in isolated vascular cells *in vitro* does not mean that the cascade actually plays a pathogenetically important role. Yet, even a cursory glance over the individual chapters of this book leaves the impression that lung vascular biology might actually be important.

N.F. Voelkel
USA