
Preface

Inflammation has been described as the basis of many pathologies of human disease. When one considers the updated signs of inflammation, they would be vasodilation, cell migration, and, in the case of chronic inflammation, cell proliferation, often with an underlying autoimmune basis. Generally, inflammation may be divided into acute, chronic, and autoimmune, although the editors believe that most, if not all, chronic states are often the result of an autoimmune response to an endogenous antigen. Thus, a proper understanding of the inflammatory basis may provide clues to new therapeutic targets not only in classical inflammatory diseases, but atherosclerosis, cancer, and ischemic heart disease as well.

The lack of advances in classical inflammatory diseases, such as rheumatoid arthritis, may in part arise from a failure to classify the disease into different forms. That different forms exist is exemplified in patients with differing responses to existing antiinflammatory drugs, ranging from nonresponders to very positive responders for a particular nonsteroidal antiinflammatory drug (NSAID). Though researchers have progressively unraveled the mechanisms, the story is far from complete. It should also be noted that the inflammatory response is part of the innate immune response, or to use John Hunter's words in 1795, "inflammation is a salutary response." That may be applied in particular to the defensive response to invading microorganisms.

Because of the large multidisciplinary scope of inflammation research, it is inevitable that a protocols collection such as *Inflammation Protocols* will represent a limited selection of the more important tools for studying inflammation. The editors have therefore focused this volume on those methods that they believe are most likely to be applicable to investigations of potential new antiinflammatory drugs in active target areas for R&D such as transcription factors, adhesion molecules, cyclooxygenase-2 (COX-2) inhibitors, nitric oxide synthases, and metalloproteinases. Some of the experimental protocols (especially the in vitro ones) described in this book are generic, in the sense that they are applicable to the study of many different inflammatory diseases, whereas others attempt to model particular human inflammatory diseases or particular aspects of inflammation.

Inflammation Protocols has been divided into three sections: (1) in vitro systems for studying aspects of inflammation, (2) in vivo models, and (3) relevant pharmacodynamic measurements for the assessment of antiinflammatory compounds. Each section opens with one or two introductory chapters that attempt to provide an overview, or at least viewpoints, of the significance of the methods in that section of the book.

There is no doubt that we will have introduced a particular “flavor” to the book that might not be to everyone’s taste—inflammation researchers are of course notorious for having their own favorite cell or inflammatory cascade. Perhaps a second volume will eventually be needed to produce a wider coverage of inflammation protocols, but the editors must recoup their energies before contemplating that prospect. Despite this, we hope that the present volume will provide a unique and contemporary collection of methods that will be useful to both the established experimenter and newcomers to the field of inflammation.

Finally, we are very grateful indeed to the contributing authors, all of them leading researchers within their respective fields. We would also like to thank the series editor, John M. Walker, for his efficient help in reviewing the manuscripts. Most of all, we would like to express our appreciation to Lin Wells, who has somehow managed to maintain a high level of administrative organization within the project, despite the disruptive behavior of the Editors and some of the Contributors!

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<http://www.springer.com/978-0-89603-970-4>

Inflammation Protocols

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2003, XII, 380 p., Hardcover

ISBN: 978-0-89603-970-4

A product of Humana Press