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New Targets in Inflammation The Search for Anti-Inflammatory Drugs *Improved Non-Steroid Anti-Inflammatory Drugs: COX-2 Enzyme Inhibitors* COX-2 Inhibitors *Kinase Targets and Inhibitors in Inflammation, 2007* Novel Cytokine Inhibitors Role of Natural Compounds in Inflammation and Inflammatory-Related Diseases Metalloproteinases as Targets for Anti-Inflammatory Drugs Proteases and Their Receptors in Inflammation High Throughput Screening for Novel Anti-Inflammatories *Trends in COX-2 Inhibitor Research* Proteinases and Their Inhibitors in Inflammation Novel Inhibitors of Leukotrienes Immunomodulatory Roles of Tryptophan Metabolites in Inflammation and Cancer New Perspectives in Anti-inflammatory Therapies Studies on Interaction of INOS and Cox-2 Inhibitors in Inflammation and Pain Progress in Inflammation Research and Therapy In Vivo Models of Inflammation *Cancer and Inflammation* Inflammatory Processes: Anti-Inflammatory Drugs in Asthma Targeting the IL-17 Pathway in Inflammatory Disorders TNF-alpha Inhibitors

Advances in Inflammation Research Novel Cytokine Inhibitors Cancer and Inflammation *Inflammation and Rheumatic Diseases* Interplay Between Neutrophils and Proteinase Inhibitors in Inflammation Inducible Enzymes in the Inflammatory Response Inflammation Eicosanoids and Other Bioactive Lipids in Cancer, Inflammation, and Radiation Injury, 5 Drug-Induced Liver Injury *Angiogenesis in Inflammation: Mechanisms and Clinical Correlates Essentials of Cox-2 Inhibitors in the Management of Pain and Inflammation* Targeting Developmental Pathways in Inflammation and Disease JAK inhibition in autoimmune and inflammatory diseases Inflammation in the Pathogenesis of Chronic Diseases Inflammation, 4 Volume Set Inflammation Protocols Essentials of Cox-2 Inhibitors in the Management of Pain and Inflammation

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This book provides an overview of the discovery and structure of IL-17, including its pathogenesis and role in chronic inflammation and autoimmunity. To capture the latest developments and product approvals the book also discusses the therapeutic advances and looks at emerging therapies targeting the IL-17 pathway. IL-17 is a pro-inflammatory cytokine that has a key role in inflammation, autoimmunity, and host defense in a number of inflammatory disorders such as rheumatoid arthritis, psoriatic arthritis and psoriasis, ankylosing spondylitis, multiple sclerosis, and inflammatory bowel disease. The discovery of the IL-17-Th17 pathway has seen exciting development in the field of immunology and inflammation research, which has led to a number of recent regulatory approvals. In 1971, Vane proposed that the mechanism of action of the aspirin-like drugs was through their inhibition of prostaglandin biosynthesis. Since then, there has been intense interest in the interaction between this diverse

group of inhibitors and the enzyme known as cyclooxygenase (COX). It exists in two isoforms, COX-l and COX-2 (discovered some 5 years ago). Over the last two decades several new drugs have reached the market based on COX-l enzyme screens. Elucidation of the three-dimensional structure of COX-l has provided a new understanding for the actions of COX inhibitors. The constitutive isoform of COX, COX-l has clear physiological functions. Its activation leads, for instance, to the production of prostacyclin which when released by the endothelium is anti-thrombogenic and anti-atherosclerotic, and in the gastric mucosa is cyto protective. COX-l also generates prostaglandins in the kidney, where they help to maintain blood flow and promote natriuresis. The inducible isoform, COX-2, was discovered through its activity being increased in a number of cells by pro inflammatory stimuli. A year or so later, COX-2 was identified as a distinct isoform encoded by a different gene from COX-I. COX-2 is induced by inflammatory stimuli and by cytokines in migratory and other cells. Thus the anti-inflammatory actions of non-steroid anti-inflammatory drugs (NSAIDs) may be due to the inhibition of COX-2, whereas the unwanted side-effects such as irritation of the stomach lining and toxic effects on the kidney are due to inhibition of the constitutive enzyme, COX-I. This much-needed text develops current knowledge on the mechanisms of angiogenesis at the molecular and cellular levels as they relate to inflammation, including acute and chronic inflammation, neurogenic initiation, and

the role of the multiple cellular components that comprise inflammation. The volume brings together experts in each of these fields to link the molecular and cellular processes in angiogenesis to those of inflammation and disease, culminating in a discourse on areas for future therapies. Drug-Induced Liver Injury, Volume 85, the newest volume in the Advances in Pharmacology series, presents a variety of chapters from the best authors in the field. Chapters in this new release include Cell death mechanisms in DILI, Mitochondria in DILI, Primary hepatocytes and their cultures for the testing of drug-induced liver injury, MetaHeps an alternate approach to identify IDILI, Autophagy and DILI, Biomarkers and DILI, Regeneration and DILI, Drug-induced liver injury in obesity and nonalcoholic fatty liver disease, Mechanisms of Idiosyncratic Drug-Induced Liver Injury, the Evaluation and Treatment of Acetaminophen Toxicity, and much more. Includes the authority and expertise of leading contributors in pharmacology Presents the latest release in the Advances in Pharmacology series Whereas G protein-coupled and nuclear receptors have been in the focus of the pharmaceutical industry since decades, protein kinases became interesting drug targets more recently. Several protein kinase inhibitors are well established for the treatment of tumors in the meantime, many more are in clinical development. Inside into the biology of protein kinases and progress in technologies to target these molecules, however, fostered the discovery and development of

protein kinase inhibitors for the treatment of inflammation, too. They are particular promising since they are key factors in signal transduction with major impact for initiating, propagation and regulation of immunological responses. In addition, they are well drugable by small molecular weight inhibitors, opening the chance to discover oral bioavailable drugs. When considering kinases as targets in inflammation, however, there are several issues too. These are mainly the selectivity of both the target and the inhibitor and the associated side effect profile of corresponding drugs. In contrast to application in oncology where efficacy is more or less the most important criteria, a high safety profile is key for the chronic treatment of usual non-life treating inflammatory and autoimmune diseases. Having said this, however, clearly better drugs with a favorable therapeutic index (effect / side effect ratio) are needed for these indications, too. Thus, to be considered as a really promising drug target the protein kinase of interest should be significantly involved in the inflammatory process - otherwise insufficient efficacy and potency would have to be expected - without being involved in other physiological processes of essential biological importance or a very selective expression pattern. The same is true with the specifity of the inhibitors itself. So a compound hitting just one other kinase a little may not be acceptable, depending on the function of these off target molecule. Consequently, a solid target identification and validation is needed before lead identification and optimization with the

goal of achieving high specifity is started. Despite all these challenges major progress has been made and the first promising kinase inhibitors are moving into the clinic for anti-inflammatory therapy. There are many more on the horizon and it s pretty likely that protein kinase inhibitors will enrich the spectrum of therapy for chronic inflammatory and autoimmune diseases. Within this issue an overview is given on recent progress in the field of protein kinase inhibitors for the treatment of inflammation addressing the opportunities and chances on one hand and the issues and hurdles on the other hand. We are very pleased that we were able to convince real experts to contribute to this hot topic issue. Starting with some general points the recent findings are issued and should enabled the reader to keep up with this fast developing field that is of substantial interest for basic and clinical orientated scientists on one hand and academia as well as pharmaceutical industry on the other hand. All chapters in this book have been published before as articles in a hot topic issue of Anti-Inflammatory & Anti-Allergy Agents in Medicinal Chemistry Volume 6, Number 1, February 2007 and are reprinted here with permission by Bentham Science Publishers Ltd. This eBook is a collection of articles from a Frontiers Research Topic. Frontiers Research Topics are very popular trademarks of the Frontiers Journals Series: they are collections of at least ten articles, all centered on a particular subject. With their unique mix of varied contributions from Original Research to Review Articles, Frontiers

Research Topics unify the most influential researchers, the latest key findings and historical advances in a hot research area! Find out more on how to host your own Frontiers Research Topic or contribute to one as an author by contacting the Frontiers Editorial Office: frontiersin.org/about/contact. This volume describes recent research in the field of metalloproteinases (a family of enzymes that can catalyze tissue degradation), in particular their participation in autoimmune diseases such as rheumatoid arthritis, reviewing the latest developments in metalloproteinase inhibitor design and the current status of clinical candidates. This volume is intended not only for those active in research into metalloproteinases but also for those with an interest in inflammatory diseases. Thus it addresses both academic and industrial researchers. COX-2 inhibitors are important drugs with analgesic and anti-inflammatory effects. The discovery of COX-2, the evolution of drug development in this field and the implications of these developments in patient therapy are topics of this volume. This book presents both pre-clinical and clinical information and is important for clinicians interested in the latest information about this class of drugs, for researchers and for students in the field. For some years, international guidelines on the management of asthma have stressed the importance of early intervention with anti-inflammatory drugs to prevent acute asthma exacerbations and to avert chronic inflammatory damage to the airway.

Introduced by a broad survey of the targets for anti-inflammatory drugs, this book proceeds to bring together the most recent research into the mechanisms and clinical benefits of presently available anti-inflammatory treatments including corticosteroids, cromones, and theophylline. For the first time, these drugs are discussed alongside the newly introduced leukotriene modifier drugs, and in the context of frontline research into anti-inflammatory drugs of the future. Leading authorities in their fields evaluate the prospects of novel anti-IgE agents and cytokine antagonists, and examine recent developments in immunosuppressant compounds, protease inhibitors, and selective phosphodiesterase inhibitors. This book is an up-to-date and authoritative survey which will be invaluable to university and pharmaceutical company researchers working on anti-asthma and anti-allergy drugs and to respiratory physicians keeping abreast of developments in their specialty. This volume provides a comprehensive overview of the development, pharmacology, efficacy, and safety of the currently available TNF-alpha inhibitors. It is the first volume that summarizes this material for all available TNF-alpha inhibitors. Elevated levels of TNF-alpha have been demonstrated in Crohn's disease, psoriasis, psoriatic arthritis, and rheumatoid arthritis, suggesting a role for TNF-alpha in their pathogenesis. The most recent preclinical and clinical data is presented in this book. The papers reported here will contribute to

proposing new insights into the mechanisms of several conditions, as well as suggesting new diagnostic alternatives and therapeutic targets in widespread pathologies such inflammation and inflammatory-based diseases. The discovery of the new is, as always, anchored in recourse to the old. Cytokines have become established as key mediators of the signs and symptoms of inflammatory diseases such as arthritis, dermatitis, asthma and multiple sclerosis. Furthermore, they are involved in the cascade of events leading to cardiovascular shock and are major regulators of the function of immune cells. This book reviews recent advances in the development of new anti-inflammatory drugs. It addresses different therapeutic intervention possibilities for new drugs, such as the cellular source of cytokines, specific receptors which induce cytokine synthesis, intracellular regulators of cytokine gene induction and expression, secretion and activation of cytokines, cytokine receptors and signalling pathways from these receptors. Accordingly, experts were drawn from different backgrounds including academic research institutes, the pharmaceutical industry and clinical pharmacology. In each area, the opportunities for drug development are highlighted and, where possible, clinical data is reviewed. The purpose of this volume in the series Progress in Inflammation Research is to pro vide the biomedical researcher with a description of the state of the art of the devel opment and use of animal models of diseases with components of inflammation. Par ticularly

highlighted are those models which can serve as in vivo correlates of diseases most commonly targeted for therapeutic intervention. The format is designed with the laboratory in mind; thus it provides detailed descriptions of the methodologies and uses of the most significant models. Also, new approaches to the development of future models in selected therapeutic areas have been highlighted. While emphasis is on the newest models, new information broadening our understanding of several well-known models of proven clinical utility is included. In addition, we have pro vided coverage of transgenic and gene transfer technologies which will undoubtedly serve as tools for many future approaches. Provocative comments on the cutting edge and future directions are meant to stimulate new thinking. Of course, it is important to recognize that the experimental use of animals for human benefit carries with it a solemn responsibility for the welfare of these animals. The reader is referred to the section on current regulations governing animal use which addresses this concern. To fulfill our purpose, the content is organized according to therapeutic areas with the associated models arranged in subcategories of each therapeutic area. How are cancer and inflammation interrelated mechanistically and clinically? Though extensive literature exists on the topic "Cancer and Inflammation", there are relatively few texts that have truly integrated the two in spite of the many common mechanisms shared by their processes. Certainly, areas such as cytokines, growth factors,

proliferation, signal transduction and angiogenesis, for example, are found in both. Yet, the dynamics of how these common mechanisms are maybe interrelated in the pathologies of the two is not widely covered. Such coverage, as presented in this volume, may help further understanding and bring new approaches to therapeutics. The first section of the book discusses inflammatory mechanisms, studied in cellular and animal studies. The second part concentrates on clinical studies with antiinflammatory drugs in cancer treatment. The volume is written for biomedical researchers in the health care industry and in academia who are working in these areas. Many new antileukotriene drugs are now marketed as antiasthma drugs and represent the first new drugs in this field since the 1970s. This book covers the steps that have led to the discovery and development of these new drugs and offers detailed descriptions of their clinical applications. The review chapters on the main aspects of basic and applied leukotriene research are written by leading specialists in the field, and the volume takes a new approach in presenting information of particular interest to both scientists and clinicians in the fields of asthma, inflammation and allergic diseases. In November 1998 many of the key leaders of new drug discovery for inflammatory diseases gathered at Hershey, Pennsylvania for the 9th International Conference of the Inflammation Research Association. The Conference was held over a five day period and provided a stimulating environment for the open

exchange of important advances in basic inflammation research as well as new drug discovery and development. This book encompasses some of the highlights of several presentations made at the Conference. It contains some of the latest and important developments in the field of inflammation research. Topics include the status of eotaxin and chemokines in asthma and allergy, signal transduction and regulation of diverse mediators such as the JNK group of MAP kinases, TNF and IL-1 signaling of NF-kB as well as regulators of AP-1, macrophage metalloproteinases, lymphotoxin and further insights into the role of MCP-1 in disease. Also discussed are drug targets in rheumatoid and osteoarthritis, fibrotic diseases,... Combinatorial chemistry in conjunction with High Throughput Screening (HTS) is revolutionizing the drug discovery process. Yet, we have much to learn about the integration of these powerful techniques with information from genomics, proteomics, computation and pharmacokinetics before dramatic increases in the drug discovery/development processes can be achieved. The chapters in this book represent the state of the art regarding the integration of combinatorial chemistry and HTS in connection with anti-inflammatory targets. Obviously, there is much work to be done beyond what is described in this text, nevertheless, it should set the stage for creative thinking among scientists of many disciplines for the accomplishment of our ultimate goals in treating inflammatory diseases. Cox-2 Inhibitors are newly developed drugs for

inflammation that selectively block the Cox-2 enzyme. Blocking this enzyme impedes the production of the chemical messengers (prostaglandins) that cause the pain and swelling of arthritis inflammation. Cox-2 inhibitors are a new class of non-steroidal anti-inflammatory drugs (NSAIDS). Because they selectively block the Cox-2 enzyme and not the Cox-1 enzyme, these drugs are uniquely different from traditional NSAIDS. This book explores new research in the field. This volume represents a collection of contributions from the 6th International Conference on Eicosanoids and Other Bioactive Lipids in Cancer, Inflammation, and Related Diseases held in Boston from September 12-15, 1999. The mission of this meeting was to bring together senior and junior investigators to both announce and examine their recent advancements in cutting-edge research on the roles and actions of lipid mediators and their impact in human physiology and disease pathogenesis. The meeting focused on new concepts in these areas of interest to both clinicians and researchers. The program included several outstanding plenary lectures and presentations by leading experts in the fields of cancer and inflammation. In addition, the Boston meeting presented three Young Investigator awards, one in each of the major focus areas. The meeting was exciting and proved to be very memorable. The program was developed with an emphasis on recent advances in molecular and of lipid mediators relevant in cellular mechanisims involved in the formation and actions inflammation

and cancer. Plenary lectures were presented by Prof. Bengt Sammuelsson (Karolinska Institute, Stockholm; 1982 Nobel Laureate in Physiology or Medicine) and Prof. E. 1. Corey (Harvard University; 1990 Nobel Laureate in Chemistry). Both of these plenary lectures were held on Day 1, which set an exciting tone for this meeting. Immediately following these plenary lectures, three simultaneous breakout sessions were held, one of inflammation, a second on cancer and synthesis of novel inhibitors, and a third on enzymes-lipoxygenases/cyclooxygenases and inhibitors.

Perspectives on Anti-Inflammatory Drugs

Inflammation is a very complicated process of interrelated events and cas cades that does not allow for an easily defined, focused attack for drug discovery. It is evident from years of research and development that certain classes of compounds (e.g., NSAIDs, steroids, and so on) have had a meas ure of success in alleviating pain and even dampening cellularlhormonal mechanisms involved in the process. Clear, mechanism-related therapies (e.g., for arthritis) and targeted drugs (e.g., for transplantation) have not been available in the past and, in reality, research in inflammation has re lied on more phenomenological approaches for resolving symptoms or on blatant cytoreductive approaches in cases like organ transplantation. In the last decade, approaches that have revealed novel cellular pathways in which intervention is possible for lymphocyte regulation (for example, cyclosporine and FK506) and small molecular weight mediators

(e.g., leu kotriene inhibitors) are now either standard therapy or will be in a short time. These latter approaches have been the result of research from the 1970s up to the present. 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 inflam- tion, cell proliferation, often with an underlying autoimmune basis. Gen- ally, inflammation may be divided into acute, chronic, and autoimmune, - though 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 therap- tic 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 rh- matoid arthritis, may in part arise from a failure to classify the disease into different forms. That different forms exist is exemplified in patients with d- fering responses to existing antiinflammatory drugs, ranging from nonresponders to very positive responders for a particular nonsteroidal an- inflammatory drug (NSAID). Though researchers have progressively unr- eled 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 micro- ganisms. For the past 100 years the mainstay of therapy for rheumatoid arthritis (RA) has been aspirin or other drugs of the non-steroid anti-inflammatory group. In 1971 Vane pro posed that both the beneficial and toxic actions of these drugs was through inhibition of prostaglandin synthesis. The recent discovery that prostaglandins responsible for pain and other symptoms at inflammatory foci are synthesized by an inducible cyclooxygenase (COX-2) that is encoded by a gene distinct from that of the consti tutive enzyme (COX-I) provided a new target for therapy of RA. A drug that would selectively inhibit COX-2 would hopefully produce the symptomatic benefit provided by existing NSAIDs without the gastrointestinal and renal toxicity due to the inhibition of COX-I. Drugs selective for COX-2 are now available. Experimental studies have shown them to be effective with minimal toxicity, and in clinical trials gastric and renal toxicities are less. Highly selective COX-2 inhibitors, perhaps designed with knowledge of the crystal structures of COX-I and COX-2, are also available. Other experimental studies, including those in animals lacking effective genes for COX-lor COX-2 and in experimental carcinomas, suggest there is still much to be learned of the pathophysiological functions of these enzymes. The inflammatory response is a complex reaction involving many mediators that derive from white blood cells, endothelial cells and other tissues. Preliminary data have revealed that

inhibitors of the cytokines and adhesion molecules that play a crucial role in the migration of white cells to inflammatory sites may be useful in RA. Chronic inflammation predisposes to some forms of cancer and the host response to malignant disease shows several parallels with inflammation and wound healing. The cells involved in inflammation are detected in a range of common cancers, together with the inflammatory cytokines and members of the chemokine ligand/receptor systems. Neutralization or deletion of the gene for some inflammatory cytokines confers resistance to tumour induction and experimental metastasis. Over-expression of such cytokines in tumour cells may enhance malignant potential. Certain chemokines are likely to subvert antitumour immunity by favouring development of ineffective Type 2 responses. Tumour cells may even utilize chemokine receptors in homing to lymph nodes and other organs. Thus, the cells, cytokines and chemokines found in tumours are more likely to contribute to tumour growth, progression and immunosuppression than they are to mount an effective host antitumour response. This book draws together contributions from an international group of scientists and clinicians from diverse disciplines, ranging from epidemiology to immunology, cell biology, molecular oncology, molecular medicine and pharmacology to debate these and related issues. Topics covered include the epidemiological links between cancer and inflammation, the parallels between inflammation and cancer, the role

of inflammation in cancer, inflammatory genes as risk factors for cancer initiation and progression, inflammation and cancer angiogenesis, and preventative and therapeutic strategies. Related Novartis Foundation symposia: 252 Generation and Effector Functions of Regulatory Lymphocytes Chair: Jean-François Bach 254 Immunoinformatics: Bioinformatic Strategies for Better Understanding of Immune Function Chair: Hans-Georg Rammensee In this book, a worldwide panel of leading experts discuss the role of inflammation in the pathogenesis of major chronic diseases and the current controversy regarding risk versus benefit of selective cyclooxygenase-2 (COX-2) inhibitors. The authors provide exciting and enlightening perspectives on COX-2 and related molecular targets in the future of medicine, including historical perspectives. Throughout the centuries, inflammation has been considered as a disease in itself. This misconception arose from the inability to distinguish between inflammatory changes and the insults which induce them. The understanding of the distinction between the genesis of inflammation and the tissue reactions that follow is attributed to JOHN HUNTER, who, at the end of the 18th century, substantially contributed to the analysis of inflammation in objective terms. Today, however, we are still trying to find explanations for Celsus' Signs in terms of structural and functional changes occurring in the inflamed tissue. There are drugs which modulate these signs but, without a detailed knowledge of the basic physiopathological events, it

is impossible to understand their mechanism of action. Notwithstanding, the effects of anti inflammatory drugs provided new knowledge of the relevance of the signs and symptoms to the sequence of biochemical and morphological changes occurring in inflammation. When we accepted the invitation to edit a Handbook on Inflammation and Anti Inflammatory Drugs, we were aware of the magnitude of the task. We knew the impossibility of covering the whole field in detail, especially taking into account the rapid accumulation of experimental knowledge which would, in all likelihood, overtake the process of publication. Proteases are everywhere from prokaryotes to eukaryotes, from virus to bacteria and in all human tissues, playing a role in many biological functions. Among these functions, the inflammatory reaction is of particular interest. In inflamed tissues, proteases can have a microbial and/or host origin and are involved not only in tissue remodeling, but also in specific signaling to resident or inflammatory cells, thereby contributing to the innate immune response. This volume presents all advances in our knowledge of the role proteases and their inhibitors play in various diseases associated with inflammatory response. Mechanisms involved in protease signaling to cells are presented, and the different types of proteases that are present at inflammatory sites and their effects on the course of inflammation are discussed. Finally, the evidence for considering proteases and their receptors as potential molecular targets for therapeutic interventions in the treatment of

inflammatory diseases is discussed in the context of specific organ inflammatory pathologies (the lung, gastrointestinal tract, skin, joints, etc.). This volume will be of great value to all those researchers in the area of the inflam matory response, notably academics, clinicians and members of the pharmaceutical industry. The book has in the main been restricted to three inducible enzymes, namely nitric oxide synthase (iNOS), cyclooxygenase (COX-2) and hemeoxygenase (HO-l), although matrix metalloproteinases, xanthine oxidoreductase and tissue transgluta minases are reviewed. The modulation of these enzymes is viewed as possible novel therapeutic advances in the area of inflammation and also cancer. The latter topic may well be the subject of a further book. It will be interesting to observe the progress of such new therapies in the next decade. Already some of these enzyme modulators have been approved for the treat ment of inflammatory disease, as evidenced by the new families of COX-2 inhibitors. We believe such advances will herald a series of new and exciting agents to be included in the clinician's armamentarium in the constant struggle against inflammatory disease. The editors wish to thank all contributors to this volume on inducible enzymes. It should however be stressed that the views expressed by the authors are personal and do not necessarily reflect those of the editors. Indeed, the reader may find con flicting statements in a number of the chapters. We believe that this is entirely appro priate as this volume reflects the latest work in a

rapidly developing area. The leading reference on this topic of increasing medical relevance is unique in offering unparalleled coverage. The editors are among the most respected researchers in inflammation worldwide and here have put together a prestigious team of contributors. Starting with the molecular basis of inflammation, from cytokines via the innate immune system to the different kinds of inflammatory cells, they continue with the function of inflammation in infectious disease before devoting a large section to the relationship between inflammation and chronic diseases. The book concludes with wound and tissue healing and options for therapeutic interventions. A must have for clinicians and biomedical researchers alike. This book is a useful guide for students, physicians in continuing education and practitioners who want to keep abreast of the latest developments in rheumatology. The chapters on the pathogenesis of rheumatoid arthritis and the biochemistry of inflammation present the latest research results in this field and are illustrated by a wealth of charts and tables. There follows practical and critical information on all drug groups that are currently used in the treatment of inflammatory rheumatic diseases and osteoarthritis. (Midwest).

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