

1. Record Nr.	UNINA9910488713203321
Titolo	Targeting cellular signalling pathways in lung diseases // Kamal Dua [and four others], editors
Pubbl/distr/stampa	Gateway East, Singapore : , : Springer, , [2021] ©2021
ISBN	981-336-827-6
Descrizione fisica	1 online resource (921 pages)
Disciplina	616.2407
Soggetti	Lungs - Diseases - Molecular aspects
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia
Nota di contenuto	Intro -- Preface -- Acknowledgment -- Contents -- About the Editors -- 1: Introduction to Lung Diseases -- 1.1 Chronic Obstructive Pulmonary Disorder (COPD) -- 1.1.1 Definition -- 1.1.2 Epidemiology -- 1.1.3 Causes -- 1.1.4 Symptoms (Fig. 1.2) -- 1.1.5 Pathophysiology -- 1.1.6 Treatment -- 1.2 Asthma -- 1.2.1 Definition -- 1.2.2 Epidemiology -- 1.2.3 Causes (Fig. 1.3) -- 1.2.4 Symptoms (Fig. 1.4) -- 1.2.5 Pathophysiology -- 1.2.6 Types of Asthma [40] -- 1.2.7 Treatment -- 1.3 Lung Cancer -- 1.3.1 Classification -- 1.3.2 Epidemiology -- 1.3.3 Etiology and Risk Factors [45] (Fig. 1.6) -- 1.3.4 Sign and Symptoms -- 1.3.5 Diagnosis -- 1.3.6 Staging of Lung Cancer -- 1.3.7 Treatment [54] -- 1.4 Acute Respiratory Tract Infections -- 1.4.1 Upper Respiratory Tract Infections -- 1.4.1.1 Acute Pharyngitis -- 1.4.1.2 Acute Sinusitis -- 1.4.1.3 Acute Viral Laryngitis -- 1.4.2 Lower Respiratory Tract Infection -- 1.4.2.1 Pneumonia -- 1.4.2.2 Influenza -- 1.5 Idiopathic Pulmonary Fibrosis (IPF) -- 1.5.1 Epidemiology -- 1.5.2 Causes -- 1.5.3 Pathophysiology -- 1.5.4 Symptoms -- 1.5.5 Diagnosis -- 1.5.6 Treatment -- References -- 2: Targeting Molecular and Cellular Mechanisms in Asthma -- 2.1 Introduction -- 2.1.1 Rationale to the Study -- 2.1.2 Asthma: A Global Health Burden -- 2.1.3 Asthma: GINA Definition -- 2.2 Traditional Understanding and Parallel Non-specific Therapy -- 2.2.1 Major Types of Asthma -- 2.2.2 Allergens Are Major Inducers for Atopic Asthma -- 2.2.3 Pathophysiology -- 2.2.4 Symptomatic Therapy -- 2.3 Current

Understanding and Steps Towards Pathway-Specific Therapy(Fig. 2.1)
-- 2.3.1 Cells Involved in Asthma Pathogenesis and Respective Targets
-- 2.3.1.1 Airway Epithelium -- 2.3.1.2 Dendritic Cells -- Dendritic Cell Modulator as a Target -- 2.3.1.3 Lymphocytes -- 2.3.1.4 T Helper Type 2 (Th2) Cells -- Th2 Cytokines as Target.
2.3.1.5 B Lymphocytes and IgE -- 2.3.1.6 Mast Cells -- 2.3.2 Th2-High Eosinophilic Asthma Versus Th2-Low Neutrophilic Asthma -- 2.3.2.1 Eosinophilic Asthma/Th2-High Asthma -- 2.3.2.2 Neutrophilic Asthma -- 2.3.3 Asthma: A Complex Syndrome with Multiple Endotypes -- 2.4 Nitro-Oxidative Stress in Asthma Pathogenesis -- 2.4.1 Oxidative Stress -- 2.4.2 Mitochondrial Dysfunction in Asthma -- 2.4.3 Nitric Oxide (NO) -- 2.5 Airway Remodelling in Asthma: The Knowledge that Will Shape the Future Treatment -- 2.6 Non-pharmacological Intervention for Asthma: Bronchial Thermoplasty -- 2.7 Tailoring the Patient Needs Via `Personalised Approach` in Asthma -- 2.8 Conclusions and Future Perspectives -- References -- 3: Various Cellular and Molecular Axis Involved in the Pathogenesis of Asthma -- 3.1 Introduction -- 3.2 Epidemiology and Risk Factors -- 3.3 Granulocytes: As Predictors of Asthma -- 3.3.1 Basophils and Allergic Inflammation -- 3.3.1.1 Stimuli for Basophil Activation -- 3.3.1.2 Inflammatory Mediators of Basophils -- 3.3.1.3 Basophil Adhesion Molecules -- 3.3.2 Neutrophil Granule Contents -- 3.3.2.1 Mechanisms in Neutrophil Activation -- 3.3.2.2 Neutrophil in Airway Inflammation and its Signal Transduction Pathways -- 3.3.2.3 Neutrophil Apoptosis: The Resolution of Inflammation -- 3.3.3 Eosinophilic Asthma -- 3.3.3.1 Eosinophil-Derived Cytokines and Associated Bronchial Hyperresponsiveness -- 3.4 Fibroblasts and Myofibroblasts -- 3.4.1 Extracellular Matrix Production in the Airways -- 3.4.2 Fibroblast to Myofibroblast Transition -- 3.5 Pathological Signaling Pathways Involved in the Activation of Lung-Resident Macrophages and Dendritic Cells (DCs) -- 3.5.1 Macrophage Activation Pathways -- 3.5.1.1 IL-4/IL-13 Signaling Pathway -- 3.5.1.2 TNF- Signaling Pathway -- 3.5.1.3 TGF- Signaling Pathway -- 3.5.2 Reactive Oxygen and Nitrogen Species.
3.5.3 Antigen Presentation by Dendritic Cells and their Role in the Pathology of Asthma -- 3.6 Bronchial Epithelial Cells -- 3.6.1 Mechanisms of Endothelial Cell Activation -- 3.6.1.1 Cytokine Activation -- 3.6.1.2 Role of Epithelial Cells in Type 2-Driven and Non-type 2 Allergic Asthma -- 3.7 Airway Epithelial and Smooth Muscle Cells -- 3.7.1 Physiological Barrier -- 3.7.2 Airway Epithelial Cell-Derived Mediators -- 3.7.2.1 Nitric Oxide (NO) -- 3.7.2.2 Endothelin -- 3.7.2.3 Arachidonic Acid Metabolites -- 3.7.2.4 Inflammatory Cytokines -- 3.7.2.5 Cell Adhesion Molecules -- 3.7.2.6 Platelet-Activating Factor (PAF) -- 3.7.2.7 Tachykinin -- 3.7.2.8 Histamine -- 3.7.2.9 Adenosine -- 3.8 Role of Innate Immunity in Asthma -- 3.8.1 CD4+ and CD8+ T Cells in Asthma -- 3.8.2 Cellular Receptors of Innate Immunity in Asthma -- 3.8.3 Innate-like T Cells and Asthma -- 3.9 Conclusion -- References -- 4: Targeting Molecular and Cellular Mechanisms in Steroid-Resistant Asthma -- 4.1 Introduction -- 4.2 Inducers of Steroid Resistance in Asthma -- 4.3 Shedding Light on the Cellular Mechanisms Underlying the Glucocorticoid Receptor Signalling Pathway-Mediated Steroid Resis... -- 4.3.1 Neutrophils -- 4.3.2 Factors Inducing Airway Neutrophilia -- 4.3.3 Eosinophils -- 4.3.4 Group 2 Innate Lymphoid Cells (ILC-2) -- 4.3.5 T Helper Type 9 Cells (Th9 Cells) -- 4.3.6 CD8+ T Cells -- 4.4 Possible Molecular Mechanisms behind Steroid-Resistant (SR) Asthma (Fig. 4.1) -- 4.4.1 Bioavailability of Glucocorticoid Receptor (GR) and the Underlying Molecular Mechanism -- 4.4.2 IL-17A High and IFN- High Phenotypes -- 4.4.3 PI3K/HDAC

Signalling Pathway -- 4.4.4 p38 MAPK -- 4.4.5 NLRP3 Inflammasome -- 4.4.6 Lack of Autophagy -- 4.4.7 Other Key Mechanisms -- 4.5 Tailoring Steroid-Refractory Asthma: The Possible Diagnosis and Therapeutics -- 4.5.1 Immunosuppressive Drugs. 4.5.2 Biologic Therapies -- 4.5.3 Highly Potent Glucocorticoids -- 4.5.4 Vitamin D -- 4.5.5 Sulforaphane -- 4.6 Conclusions and Future Outlook -- References -- 5: Targeting Molecular and Cellular Mechanisms in Chronic Obstructive Pulmonary Disease -- 5.1 Introduction -- 5.2 Pathophysiology of COPD -- 5.2.1 Inflammatory Mediators and Cells -- 5.2.1.1 Neutrophils (NPHs) -- 5.2.1.2 Macrophages (MPs) -- 5.2.1.3 T-Lymphocyte -- Eosinophils (Eos) -- 5.2.1.4 Dendritic Cells (DCs) -- 5.2.1.5 Epithelial Cells (EP) -- 5.2.2 Protease and Antiprotease Imbalance -- 5.2.3 Oxidative Stress (OS) -- 5.3 Mediators Involved in COPD -- 5.3.1 Lipid Mediators -- 5.3.1.1 Prostaglandin -- 5.3.1.2 Thromboxane -- 5.3.1.3 Leukotrienes (LT) -- 5.3.1.4 Platelet-Activating Factor (PAF) -- 5.3.2 Peptide Mediators -- 5.3.2.1 Endothelins (ET) -- 5.3.2.2 Bradykinin (BK) -- 5.3.2.3 Tachykinins (TK) -- 5.3.2.4 Chemokines -- 5.3.2.5 Interleukin-8 (IL-8) -- 5.4 Potential Targets and Therapies for COPD -- 5.4.1 PDE (Phosphodiesterase) Inhibitors -- 5.4.2 Hypersecretion of Mucus -- 5.4.3 Bronchodilators -- 5.4.4 Glucocorticoids (Inhaled Corticosteroids) -- 5.4.5 Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) -- 5.4.5.1 CXCR2 Inhibitors -- 5.4.6 Phosphoinositide-3 Kinase Delta (PI3K) Inhibitor -- 5.4.7 Targeting Inflammatory Markers -- 5.4.8 Other COPD Treatments -- 5.5 Conclusion -- References -- 6: Probing the Cellular and Molecular Mechanisms Underlying in the Pathogenesis of Chronic Obstructive Pulmonary Disease -- 6.1 Introduction -- 6.2 Subtypes of COPD -- 6.2.1 Chronic Bronchitis -- 6.2.2 Emphysema -- 6.2.3 Small Airway Disease -- 6.3 Inflammatory Cells Involved in COPD -- 6.3.1 Epithelial Cells -- 6.3.2 Macrophages -- 6.3.3 Neutrophils -- 6.3.4 Eosinophil -- 6.3.5 Dendritic Cells -- 6.3.6 T-Lymphocytes -- 6.4 Inflammatory Mediators Involved in COPD. 6.4.1 Lipid Mediators -- 6.4.2 Pro-inflammatory Cytokines -- 6.4.3 Inflammasomes -- 6.4.4 Role of Reactive Oxygen Species (ROS) and Oxidative Stress in COPD -- 6.5 Conclusion -- References -- 7: Chronic Obstructive Pulmonary Disease: Molecular Basis of Pathogenesis and Targeted Therapeutic Approaches -- 7.1 Introduction -- 7.2 Epidemiology -- 7.3 Aetiology of Disease Progression -- 7.3.1 Inflammation -- 7.3.1.1 Neutrophils -- 7.3.1.2 Macrophages -- 7.3.1.3 T Lymphocytes -- 7.3.1.4 Eosinophils -- 7.3.1.5 Dendritic Cells -- 7.3.1.6 Epithelial Cells -- 7.3.2 Oxidative Stress -- 7.3.3 Genetic Predisposition -- 7.3.4 Epigenetics -- 7.3.5 Activity of Proteases -- 7.3.5.1 Neutrophil Wlastase -- 7.3.5.2 Cysteine Proteases -- 7.3.5.3 Matrix Metalloproteinases -- 7.4 Current Treatments and Their Drawbacks -- 7.4.1 Quit Smoking -- 7.4.2 Vaccination -- 7.4.3 Physical Activity -- 7.4.4 Pharmacological Treatment -- 7.4.5 Emerging Antioxidants for an Alternative Therapeutic Approach -- 7.4.6 Interventional Treatments -- 7.4.7 Oxygen and Ventilator Support -- 7.4.8 Comorbidity Treatment -- 7.5 Conclusion -- References -- 8: Exploring the 'Dormancy Activation Switch' in the Tumour Microenvironment for Metastatic Lung Cancer: The Possible Role of ... -- 8.1 Lung Cancer -- 8.2 Metastatic Niche in Different Sites -- 8.3 Lymph Node Metastasis -- 8.3.1 MAPK Signalling in Lymph Node Metastasis of Lung Cancer -- 8.4 Pleura Metastasis -- 8.4.1 CXCR4/CXCL12 Signalling and Pleura Metastasis in Lung Cancer -- 8.5 Liver Metastasis -- 8.5.1 ALK and Liver Metastasis in Lung Cancer -- 8.6 Brain Metastasis -- 8.6.1 Rho/ROCK Signalling and Brain Metastasis in Lung Cancer -- 8.6.2 PI3K/AKT Signalling and Brain Metastasis in

Lung Cancer -- 8.7 Bone Metastasis -- 8.7.1 RANK/RANKL Signalling
and Bone Metastasis in Lung Cancer -- 8.8 Controversial Role miRNA in
Lung Cancer Metastasis.
8.9 Concluding Remarks.
