DOWNLOAD PDF MEMBRANE RECEPTORS, CHANNELS, AND TRANSPORTERS IN PULMONARY CIRCULATION

Chapter 1 : calendrierdelascience.com | Membrane Receptors, Channels and Transporters in Pulmonary C

Membrane Receptors, Channels and Transporters in Pulmonary Circulation is divided into six parts. Part 1 (Ion Channels in the Pulmonary Vasculature: Basics and New Findings) is designated for basic knowledge and recent findings in the research field of ion channels in pulmonary circulation.

Research Interests The Stevens lab studies the endothelium, with a particular interest in endothelial cell heterogeneity. Considerable work in the past 10 years has demonstrated that pulmonary artery, capillary and vein endothelial cells are highly specialized in order to perform the physiological functions that are relevant to their vascular location. The mechanisms responsible for establishing such heterogeneity are still poorly understood, and are the focus of our ongoing work. Endothelium forms a semi-permeable barrier that separates blood from underlying tissue. Strength of this barrier, and the nature of cell-cell adhesion, changes dramatically from the pulmonary artery to the capillaries, where capillary endothelial cells form an especially tight barrier. During the course of inflammation, endothelial cells transiently decrease cell-cell adhesion and form intercellular gaps. Calcium entry through ion channels on the plasma membrane is an important signal that triggers cytoskeletal reorganization and promotes gap formation. Our group has worked to identify calcium channels that provide the calcium source responsible for inter-endothelial cell gap formation. We have found that proteins belonging to the canonical transient receptor potential family of proteins TRPC contribute subunits to this channel. TRPC4, in particular, is linked directly to the cytoskeleton. TRPC4 binds to protein 4. Ongoing studies suggest that this linkage establishes a privileged niche, where calcium entering through the channel serves as a catalyst to reorganize peripheral actin and enable intercellular gap formation. Hormones such as epinephrine increase endothelial cAMP, and therefore have anti-inflammatory actions. In contrast to the anti-inflammatory actions of epinephrine, pathogenic bacteria, such as Pseudomonas aeruginosa, have evolved mechanisms to utilize adenylyl cyclase toxins to generate cAMP and disrupt the endothelial cell barrier. Once in the host cell, exoY binds a mammalian cofactor and becomes an active enzyme, generating cAMP. Unlike the cAMP that is synthesized by the host cell, which is primarily produced at the plasma membrane, exoY generates cAMP in the cytosol. This cytosolic cAMP does not strengthen the cortical actin rim, but rather, disassembles microtubules leading to intercellular gap formation. Hence, studies on the function of exoY have revealed previously unappreciated mechanisms of cAMP signal transduction, cytoskeletal regulation of endothelial cell barrier, and mechanisms of bacterial pathogenesis. It will be important to continue these studies, to better understand how exoY becomes localized to target microtubule structures, and how the toxin serves to impact bacterial dissemination and pathogenicity. Mechanisms regulating endothelial cell barrier function. Pulmonary vasoconstriction induced by Gq agonists. Is there a role for store operated calcium entry? Essential control of an endothelial cell ISOC by the spectrin membrane skeleton. Endothelial cell phenotypes in heart, lung and blood diseases. Hydraulic conductance of segmental endothelial phenotypes in pulmonary circulation. Dominant regulation of inter-endothelial cell gap formation by the calcium-inhibited type 6 adenylyl cyclase. Bronchial endothelial cell phenotypes and the form: Pulmonary microvascular and arterial endothelial cells differ in their responses to ICAM-1 ligation. Norwood N and Stevens T. Coordinate regulation of membrane cAMP by calcium inhibited adenylyl cyclase type 6 and phosphodiesterase type 4 activities. On the endothelial cell ISOC. Shear stress increases expression of a KATP channel in rat pulmonary microvascular endothelial cells. Structural and functional characteristics of lung macro- and microvascular endothelial cell phenotypes. CpG DNA-mediated immune response in pulmonary endothelial cells. Gebb SA and Stevens T. On lung endothelial cell heterogeneity. Activated leukocyte cell adhesion molecule in breast cancer: The paired-related homeobox gene transcription factor Prx1 is required for lung vascularization and normal alveolar development. Paradoxical cAMP-induced lung endothelial hyperpermeability revealed by P. Stat3 activity is required for centrosome duplication in Chinese hamster ovary cells. Sayner S and Stevens T. Adenylyl cyclase and cAMP regulation of the endothelial barrier.

DOWNLOAD PDF MEMBRANE RECEPTORS, CHANNELS, AND TRANSPORTERS IN PULMONARY CIRCULATION

Perspectives on Lung Endothelial Barrier Function. Heterogeneity of Lung Endothelial Cells. Calcium inhibited adenylyl cyclase AC6 controls endothelial cell barrier function. Proinflammatory Signaling Mechanisms in the Pulmonary Circulation. Ion Channels in the Pulmonary Vasculature. Taylor and Francis, Boca Raton, Florida pg. Taylor and Francis, Boca Raton, Florida, pg. Encyclopedia of the Microvasculature. Molecular and cellular determinants of lung endothelial cell heterogeneity. Pulmonary circulation and pulmonary hypertension. Endothelial Cells in Health and Disease. Informa Healthcare, pages , Cyclic GMP-specific phosphodiesterase 5 regulates cell growth and apoptosis in pulmonary endothelial cells. Perspectives, translational research, and letters to the editor. Downregulation of endothelin-1 farnesoid X receptor in vascular endothelial cells. Soluble adenylyl cyclase reveals the significance of cAMP compartmentation on pulmonary microvascular endothelial cell barrier. Activated leukocyte cell adhesion molecule is a component of the endothelial junction involved in transendothelial monocyte migration. Soluble adenylate cyclase reveals the significance of compartmentalized cAMP on endothelial cell barrier function. Biochemical Society Transactions, Hydraulic conductance of pulmonary microvascular and macrovascular endothelial cell monolayers. Cioffi D and Stevens T. Regulation of endothelial cell barrier function by store operated calcium entry. Cell-surface protein disulfide isomerase is required for transnitrosation of metallothionein by S-nitroso-albumin in intact rat pulmonary vascular endothelial cells. Microheterogeneity of lung endothelium. Aird, Cambridge University Press, pages , Phenotypic heterogeneity in lung capillary and extra - alveolar endothelial cells. Increased extra-alveolar endothelial permeability is sufficient to decrease compliance. Spectrin-anchored phosphodiesterase 4D4 restricts cAMP from disrupting microtubules and inducing endothelial cell gap formation. Stevens T, Gillespie MN. The hyperproliferative endothelial cell phenotype in idiopathic pulmonary arterial hypertension. Microtubule motors regulate ISOC activation necessary to increase endothelial cell permeability. Regulatory role for nucleosome assembly protein-1 in the proliferative and vasculogenic phenotype of pulmonary endothelium. Lung microvascular endothelium is enriched with progenitor cells that exhibit rapid vasculogenic capacity. Hydroxyurea attenuates activated neutrophil-mediated sickle erythrocyte membrane phosphatidylserine exposure and adhesion to pulmonary vascular endothelium. Heterogeneity of barrier function in the lung reflects diversity in endothelial cell junctions. Type 5 phosphodiesterase expression is a critical determinant of the endothelial cell angiogenic phenotype. Prasain N and Stevens T. The actin cytoskeleton in endothelial phenotypes. Soluble adenylyl cyclase-dependent microtubule disassembly reveals a novel mechanism of endothelial cell retraction. Development and pathology of pulmonary hypertension. TRPing on the lung endothelium. Studies on the structure and function of the calcium selective store operated calcium entry current. Ward and Jason X. Yuan, Humana Press, In Press.

DOWNLOAD PDF MEMBRANE RECEPTORS, CHANNELS, AND TRANSPORTERS IN PULMONARY CIRCULATION

Chapter 2 : Receptor tyrosine kinase inhibitors in rodent pulmonary hypertension. â€" Northwestern Schola

Part IV (Receptors and Signaling Cascades in Pulmonary Arterial Hypertension) consists of five chapters devoted to the role of bone morphogenetic protein receptors, Notch receptors, serotonin.

Received Mar 21; Accepted Apr 5. This article has been cited by other articles in PMC. The pulmonary circulation is a low-pressure, low-resistance, highly compliant vasculature. In contrast to the systemic circulation, it is not primarily regulated by a central nervous control mechanism. The regulation of resting membrane potential due to ion channels is of integral importance in the physiology and pathophysiology of the pulmonary vasculature. Redox-driven ion conductance changes initiated by direct oxidation, nitration, and S-nitrosylation of the cysteine thiols and indirect phosphorylation of the threonine and serine residues directly affect pulmonary vascular tone. Molecular mechanisms of changes in ion channel conductance, especially the identification of the sites of action, are still not fully elucidated. Further investigation of the interaction between redox status and ion channel gating, especially the physiological significance of S-glutathionylation and S-nitrosylation, could result in a better understanding of the physiological and pathophysiological importance of these mediators in general and the implications of such modifications in cellular functions and related diseases and their importance for targeted treatment strategies. The interaction between the air, the lungs, and the blood has fascinated philosophers and physiologists for centuries. In , an Oxford physician, Richard Lower, reported that it was exposure to air in the lungs that caused the change from dark venous blood to bright arterial blood. However, more than another years elapsed before Bradford and Dean described constriction of the pulmonary arteries PA caused by asphyxia The suggestion that redox changes might play a role in the mechanism of HPV was initially made in 9 and focused on ion channels in 4. Besides ROS, small signaling chemical species such as nitric oxide NO , carbon monoxide CO , and hydrogen sulfide H2S molecules participate in the regulation of pulmonary vascular function. Independent of transporters, membrane receptors, or second messenger systems, they freely diffuse through cell membranes and elicit various responses. Oxidative stress is postulated to play a prominent role in the etiology of vascular and ventricular dysfunction that is associated with cardiovascular disease 53 , , , In the pulmonary vasculature and in the heart, changes in the ROS levels result in changes in the redox state of proteins, some of which can be reversed. In part, reversible protein oxidation involves the free thiol -SH side chain of cysteine residues that can undergo a number of redox-mediated molecular modifications which may elicit positive or negative changes in protein function 34 , Although redox-target specification is, no doubt, dictated by the innate susceptibility of the target, additional measures are in place within the cell to prevent accumulation of ROS to toxic levels. The role of redox regulation in the pulmonary circulation emerged, in part, because the pulmonary vasculature constricts in response to hypoxia, while the ductus arteriosus DA and systemic vessels, such as the renal arteries, dilate. It is possible that redox control of ion channels might provide a mechanism which could explain these disparate responses. The fact that the role of redox signaling in the control of ion channels and tone in the PA is not yet agreed probably derives from the variety of ROS generated and methods of their inactivation, variation in ion channel expression in different vessels and different stages of maturation, and the use of a number of different experimental techniques. ROS can have different physiologic and pathologic roles, and the overlap of these effects can cause confusion. In this regard, when exogenous ROS or anti-oxidant enzymes are added to cells in culture, attention should be paid as to whether the concentrations used and the sites of administration are physiological or pathological. Furthermore, since ion channels and calcium-handling proteins act as effectors, differences in the ion channels expressed in resistance and conduit vessels and in fetal and adult vessels also have to be considered. Consequently, much of the work discussed in this review is relevant to our understanding of the interaction of redox status, ion channel control, and tone in the pulmonary vasculature.

Chapter 3 : Grover Conference on Pulmonary Vascular Pathobiology â€" University of Arizona

Membrane Receptors, Channels and Transporters in Pulmonary Circulation is a proceeding of the Grover Conference (Lost Valley Ranch and Conference Center, Sedalia, Colorado; September ,), which provided a forum for experts in the fields of those receptors, channels and transporters that have been identified as playing key roles in the physiology and pathophysiology of the pulmonary circulation.

Please click button to get transmembrane transporters book now. This site is like a library, you could find million book here by using search box in the widget. A must-have far-reaching text that provides readers with a state-of-the-art molecule update on transmembrane transporters, focusing on the methodological approaches currently employed to better understand how transporters work and how they can be used in cutting edge therapies. Each chapter begins with an overview of the importnat biological questions presently being considered in their field, then presents scientific approaches to address these questions. In explaining approaches, the authors cover bench-top protocols, conceptual frameworks, data obtained, and pitfalls common to the techniques. Membrane Receptors, Channels and Transporters in Pulmonary Circulation is a proceeding of the Grover Conference Lost Valley Ranch and Conference Center, Sedalia, Colorado; September , , which provided a forum for experts in the fields of those receptors, channels and transporters that have been identified as playing key roles in the physiology and pathophysiology of the pulmonary circulation. The book rigorously addresses: The overall goal was to explore the mechanisms by which specific receptors, channels and transporters contribute to pulmonary vascular function in both health and disease, and how this knowledge may lead to novel interventions in lung dysplasia, pulmonary edema, lung injury, and pulmonary and systemic hypertension to reduce and prevent death from lung disease. Part 1 Ion Channels in the Pulmonary Vasculature: Basics and New Findings is designated for basic knowledge and recent findings in the research field of ion channels in pulmonary circulation. Basics and New Findings is composed of five chapters that are exclusively designed to discuss the role of a recently identified family of cation channels, transient receptor potential TRP channels, in the regulation of pulmonary vascular tone and arterial structure. Part III Pathogenic Role of Ion Channels in Pulmonary Vascular Disease includes four chapters that discuss how abnormal function and expression of various ion channels contribute to changes in cell functions and the development of pulmonary hypertension. Part IV Receptors and Signaling Cascades in Pulmonary Arterial Hypertension consists of five chapters devoted to the role of bone morphogenetic protein receptors, Notch receptors, serotonin receptors, Rho kinase and vascular endothelial growth factor receptors in the development of pulmonary arterial hypertension. Part V Receptors and Transporters: Role in Cell Function and Hypoxic Pulmonary Vasoconstriction includes four chapters designed to illustrate the potential mechanisms involved in oxygen sensing and hypoxia-induced pulmonary vasoconstriction and hypertension. Part VI Targeting Ion Channels and Membrane Receptors in Developing Novel Therapeutic Approaches for Pulmonary Vascular Disease consists five chapters which discuss the translational research involving on membrane receptors, channels and transporters, including their potential as novel drug targets. We hope that Membrane Receptors, Channels and Transporters in Pulmonary Circulation will allow readers to foster new concepts and new collaborations and cooperations among investigators so as to further understand the role of receptors, channels and transporters in lung pathophysiology. The ultimate goal is to identify new mechanisms of disease, as well as new therapeutic targets for pulmonary vascular diseases. An additional outcome should be enhanced understanding of the role of these entities in systemic vascular pathophysiology, since the conference will include researchers and clinicians with interests in both pulmonary and systemic circulations. Because progress in the field of transporters has been extraordinary, this volume will focus on recent advances in our understanding of the structure, function, physiology, and molecular biology of membrane transporters. There will be an emphasis on transporters as molecular targets for drug delivery and disposition in the body. Every cell and organism faces the problem of spaces, made up of the two leaflets of the lipid generating a confined

space in which metabolic bilayer. A failure of any of lites, ions, proteins, and signals across its bor der. Evolution has solved the problem by these proteins may have dramatic con se generating lipid membranes that contain trans quences for ceH function. In recent years much porters, ion channels, and receptors. In eukary attention has been paid to diseases resulting otic cells, this problem is exacerbated by the from nonfunctional ion channels "chan presence of multiple organelles, which are con nelopathies". Not surprisingly, many of these fined spaces in their own right. Even the lipid diseases affect the excitability of cells.

Chapter 4 : pulmonary circulation | Download eBook pdf, epub, tuebl, mobi

Read "Membrane Receptors, Channels and Transporters in Pulmonary Circulation" by with Rakuten Kobo. Membrane Receptors, Channels and Transporters in Pulmonary Circulation is a proceeding of the Grover Conference (Lo.

Chapter 5 : Membrane Receptors, Channels and Transporters in Pulmonary Circulation | Ebook | Ellibs Eb

Membrane Receptors, Channels and Transporters in Pulmonary Circulation is a proceeding of the Grover Conference (Lost Valley Ranch and Conference Center, Sedalia, Colorado; September ,), which provided a forum for experts in the fields of those receptors, channels and transporters that.

Chapter 6 : Redox Regulation of Ion Channels in the Pulmonary Circulation

Table of contents. 1. The Role of Ion Channels in Hypoxic Pulmonary Vasoconstriction E. Kenneth Weir, JÃ©sus A. Cabrera, Saswati Mahapatra, Douglas A. Peterson, Zhigang Hong.

Chapter 7 : transmembrane transporters | Download eBook PDF/EPUB

Membrane Receptors, Channels and Transporters in Pulmonary Circulation by Jason X -J Yuan (Editor), Jeremy P T Ward (Editor) starting at. Membrane Receptors, Channels and Transporters in Pulmonary Circulation has 0 available edition to buy at Alibris.