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Signaling Pathways Of Tissue Factor Expression In ***2022-03-20***

FITZGERALD HOBBS

Regulation of Tissue Responses: The TWEAK/Fn14 Pathway and other TNF/ TNFR Superfamily Members that Activate Noncanonical NFkB Signaling Frontiers Media SA
This book is a printed edition of the Special Issue "Antiphospholipid Antibodies and Syndrome" that was published in Antibodies
Elsevier Health Sciences
First published in 1943, Vitamins and Hormones is the longest-running serial published by Academic Press. In the early days of the Serial, the subjects of vitamins and hormones were quite distinct. The Editorial Board now reflects expertise in the field of hormone action, vitamin action, X-ray crystal structure, physiology, and enzyme mechanisms. Under the capable and qualified editorial leadership of Dr. Gerald Litwack, Vitamins and Hormones continues to publish cutting-edge reviews of interest to endocrinologists, biochemists, nutritionists, pharmacologists, cell biologists, and molecular biologists. Others interested in the structure and function of biologically active molecules like hormones and vitamins will, as always, turn to this series for comprehensive reviews by leading contributors to this and related disciplines. Vitamins are organic substances not naturally produced by the body that are necessary in trace amounts for normal physiologic and metabolic functioning. Hormones are biochemical substances produced in cells and tissues that cause a specific biological change or activity to occur elsewhere in the body Study of both vitamins and hormones is essential to our understanding of physiology
Targeting the Tissue Factor-Factor VIIa Signaling Pathway to Enhance Activity of MTOR Inhibitors in the Treatment of Breast Cancer CRC Press
Cell Surface GRP78, a New Paradigm in Signal Transduction Biology presents a new paradigm that has emerged in the past decade with the discovery that various intracellular proteins may acquire new functions as cell surface receptors. Two very prominent examples are ATP synthase and GRP78. While the role of cell surface ATP synthase has been reviewed in various books, this book directs its attention to the story of cell surface GRP78. Edited by the researcher who identified cell surface expression of the molecular chaperone GRP78 as a major factor in prostate cancer and other malignancies Presents an in-depth treatment of the biological underpinnings of GRP78 and its connection to disease Provides four-color illustrations that facilitate the narrative
ESC Textbook of Vascular Biology Springer Science & Business Media
Chronic inflammatory diseases such as rheumatoid arthritis, ankylosing spondylitis, multiple sclerosis, inflammatory bowel diseases, and others typically stimulate a systemic response of the entire body. This response has a uniform character in many diseases because common pathways are switched on. The uniform response regulates systemic energy and water provision. However, long-term application of this program leads to typical disease sequelae such as fatigue / depressive symptoms, sleep disturbances, anorexia, malnutrition, muscle wasting – cachexia, cachectic obesity, insulin resistance, dyslipidemia, alterations of steroid hormone axes, disturbances of the hypothalamic-pituitary-gonadal axis, elevated sympathetic tone, hypertension, volume expansion, decreased parasympathetic tone, inflammation–related anemia, bone loss, hypercoagulability, circadian rhythms of symptoms, and disease exacerbation by stress . The Origin of Chronic Inflammatory Systemic Diseases and Their Sequelae demonstrates concepts of neuroendocrine immunology, energy and water regulation, and evolutionary medicine in order to show that the uniform response that regulates systemic energy and water provision, has been positively selected for acute physiological responses and short-lived disease states, but is a misguided program in chronic inflammatory diseases and aging. Offers a broad conceptual framework with a strong clinical link, written in an easy to grasp style and demonstrating the link to aging research Describes the important principles derived from basic immunology that are used to explain pathogenesis of chronic inflammatory systemic diseases with a focus on autoimmunity Defines the bioenergetics and energy regulation of the body explaining common response pathways typical for systemic inflammation Makes use of evolutionary medicine theory to demonstrate the uniformity of the systemic response Explains the appearance of typical disease sequelae on the basis of the three pillars: neuroendocrine immunology, energy regulation, and evolutionary medicine theory Contains color figures and tables that explain the field to newcomers
Cell Signaling in Vascular Inflammation LAP Lambert Academic Publishing
Now in its Third Edition, this authoritative text continues to provide a comprehensive and systematic review of the biology, pathobiology, and clinical disorders of the hemostatic system. Its unique organization of the basic sciences coupled with clinical sections yields a user-friendly integrated text, and a reference tool that meets the needs of diverse investigators and clinicians of contemporary medicine for understanding the hemostatic system. New chapter topics covered in this edition include angiogenesis and vasculogenesis; hemorrhagic complications of antithrombotic therapy; interactions of coagulation and fibrinolytic proteins with the vessel wall; and less common thrombotic disorders.
A New Perspective in Epithelial Biology Springer Science & Business Media
Rationale: Tissue Factor (TF) is a transmembrane glycoprotein that canonically functions as the initiator of the coagulation cascade. Increased levels of TF have been associated with inflammatory airway diseases. Since lipopolysaccharide (LPS) is known to elicit and inflammatory response in airway epithelium, we hypothesized that airway epithelial cells release TF when exposed to LPS. Since TF aids in local wound healing, we also hypothesized that inhibition of TF would decrease NHBE growth. The specific aim of this work was to evaluate the effects of LPS exposure on TF production and release from airway epithelia and determine the signaling pathways involved. A secondary aim was

to evaluate the effects of TF inhibition on NHBE growth. Methods: Normal human bronchial epithelial cells were grown in submerged cell culture and exposed to LPS as well as several intracellular signaling pathway agonist and inhibitors. Measurements: Tissue Factor mRNA and protein were measured in culture media and cell lysate by reverse-transcriptase polymerize chain reaction and enzyme-linked immunosorbent assay, respectively. Signaling pathways were evaluated using selective agonists and inhibitors. Main results: TF protein levels increased nearly two-fold in cell media after exposure to LPS (p

Lahita's Systemic Lupus Erythematosus Elsevier

A consequence of rapid progress in the science of nutrigenomics and nutrigenetics is the substantial accumulation of data covering nutrienal modulation of gene expression at the cellular and subcellular levels. Current research is increasingly focused on the role of nutrition and diet in modifying oxidative damage in the progression of disease. Dietary Modulation of Cell Signaling Pathways reviews some of these findings, focusing on nutrient-gene interactions with particular emphasis on the intracellular signaling network. Explore a Pivotal Function for Maintaining Homeostasis The book addresses the dietary modulation of particular gene expression systems and highlights the underlying molecular and cellular mechanisms that involve upstream signaling molecules, such as kinases and transcription factors in the context of their therapeutic potential. It describes nutrients' actions on the activation of an antioxidant and inflammatory transcription factor and the induction of their target gene expression. Provides a Mechanistic Understanding of the Action of Dietary Components Comprehensively covering dietary modulation of cell signaling, leading experts provide information on state-of-the-art research in their own specialty. For those working in the fields of dietary components, molecular mechanisms, and health benefits, this book presents a useful tool for mechanistic understanding of the action of dietary components.

Signaling Pathways in Liver Diseases Lippincott Williams & Wilkins

Since publication of the First Edition in 1982, Hemostasis and Thrombosis has established itself as the pre-eminent book in the field of coagulation disorders. No other book is as inclusive in scope, with coverage of the field from the standpoint of both basic scientists and clinicians. This comprehensive resource details the essentials of bleeding and thrombotic disorders and the management of patients with these and related problems, and delivers the most up-to-date information on normal biochemistry and function of platelets or endothelial cells, as well as in-depth discussions of the pharmacology of anticoagulant, fibrinolytic, and hemostatic drugs. NEW to the Sixth Edition... • A new team of editors, each a leader in his field, assures you of fresh, authoritative perspectives. • Full color throughout • A companion website that offers full text online and an image bank. • A new introductory section of chapters on basic sciences as related to the field • Entirely new section on Hemostatic and Thrombotic Disorders Associated with Systemic Conditions includes material on pediatric patients, women's health issues, cancer, sickle cell disease, and other groups. • Overview chapters preceding each section address broad topics of general importance. This is the tablet version which does not include access to the supplemental content mentioned in the text.

Regulation of Signal Transduction in Human Cell Research John Wiley & Sons

Showcasing the expertise of top-tier specialists who contributed to the newly released guidelines for the care of thrombosis in cancer patients, this exciting guide was written and edited by members of the American Society of Clinical Oncology panel, (ASCO), on the prevention and treatment of cancer-associated thrombosis, among others, and provides

The Role of Tissue Factor in Canine Hemangiosarcoma Springer Science & Business Media

LPA is a component of oxidized low density lipoproteins (oxLDL) which has been shown to accumulate in human atherosclerotic plaques. Tissue factor (TF) is the principal initiator of blood coagulation. Tissue factor upregulation in atherosclerotic plaque can lead to undesirable vascular thrombosis. The generation of reactive oxygen species (ROS), which act as signaling molecules in the vascular system, is enhanced in response to injury and has been associated with a procoagulant state and the progression of atherosclerotic disease. Oxidative stress might contribute to the increased expression of pro atherosclerotic genes at sites of vascular injury, including TF. Little is known about the regulation of TF by LPA in smooth muscle cells (SMC) which is a major player in the process of atherosclerosis. Data generated by this study demonstrate that LPA markedly induces TF expression in rat aorta smooth muscle cells (RASMCs) and human aorta smooth muscle cells (HASMCs). The signaling pathways involved are multiple.

mTOR Pathway and mTOR Inhibitors in Cancer Therapy Springer Science & Business Media

"During this thesis project we uncovered a new and reciprocal link between genetic progression of glioblastoma multiforme (GBM) and activation of coagulation system effectors, notably the tissue factor (TF) pathway. GBM is a highly aggressive (grade IV) astrocytic primary brain tumor affecting both adults and children. Florid angiogenesis, intravascular and systemic thrombosis, pseudopalisading necrosis surrounding occluded vessels and cellular invasion are cellular hallmarks of this disease, in which epidermal growth factor receptor (EGFR) and its mutant (EGFRvIII) play a prominent oncogenic role. We have observed a close parallel between the expression levels of EGFR (Classical subtype of GBM) and TF, the procoagulant receptor for clotting factor VIIa, while analyzing gene expression data of 202 patients represented in The Cancer Genome Atlas (TCGA). This link was further substantiated through our analyses of EGFRvIII expressing human GBM cell lines that revealed that oncogenic EGFRvIII upregulates the expression of TF, coagulation factor VII (FVII) and protease activated receptors 1 and 2 (PAR-1/2). Moreover, we observed that signals generated by the TF/VIIa complex cooperated with EGFRvIII to regulate angiogenic factors (VEGF, IL8). Interestingly, experiments performed in vivo suggest that GBM xenograft aggressiveness can be diminished with the use of either an anticoagulant or anti-signaling antibodies, targeting the corresponding TF functions which suggests that both components of TF activity (coagulation and signaling) are important in tumor progression. Moreover, selective targeting of the host (mouse) TF reveals its independent, albeit modest, role in glioma tumorigenesis. Lastly, we observed that amidst TF-induced procoagulant, inflammatory and angiogenic responses in vivo, dormant glioma cells acquire mutational and epigenetic changes that propel their tumorigenic conversion. Thus, coagulation system represents a functionally important element in the GBM microenvironment, a property that could potentially be targeted using traditional and new anticoagulants." --

Inflammatory Tumor Immune Microenvironment: Molecular Mechanisms and Signaling Pathways in Cancer Progression and Metastasis Frontiers Media SA

This volume focuses on the relationship between the regulation of signal transduction and disease mechanisms, and discusses how the dysregulation of intracellular signals cause diseases, cell death, carcinogenesis, and other disorders. Growth, survival, transformation, and metabolic activities at the cellular level are

regulated by various intracellular signal transduction pathways. Sources that stimulate intracellular signals include intracellular stresses and signal regulators/modulators, as well as extracellular growth factors. Recent studies on signal transduction analysis using animal and human cell lines have revealed how the intracellular signals are regulated and why their dysregulation leads to pathological states such as tumorigenesis, metabolic diseases, cell death, and so on. This book highlights several important key molecules and intracellular signaling pathways such as microRNA, the TGF-beta signaling pathway, the Wnt signaling pathway and MET signaling pathway as topical and highly relevant issues in human cell research related to signal transduction. In addition to assessing the pathogenic role of these signaling pathways, it focuses on the molecular design of small molecule regulators/inhibitors of said pathways, one of the most important approaches in this area. This book offers a valuable guide, helping not only research scientists but also clinicians to understand how the dysregulation of intracellular signals leads to diseases.
Springer Science & Business Media
Atherosclerosis is the most significant cause of cardiovascular disease worldwide. Vascular biology is the key to understanding how atherosclerosis arises and operates. The ESC Textbook of Vascular Biology is a rich and clearly laid-out guide by leading European scientists providing comprehensive information on vascular physiology, disease, and research. The textbook covers molecular findings and novel targets within the speciality while also providing the basics of vascular biology and disease pathophysiology. It also covers the major changes in the diagnosis, prevention and treatment of atherosclerosis that have occurred in recent years, developments and recent breakthroughs in the field are specifically highlighted. The official publication of the ESC Working Group on Arthrosclerosis and Vascular Biology, this print edition comes with access to the online version on Oxford Medicine Online, for as long as the edition is published by Oxford University Press. By activating your unique access code, you can read and annotate the full text online, follow links from the references to primary research materials, and view, enlarge and download all the figures and tables. The textbook is also linked to the ESC's online learning platform (ESCel) and their core specialist training curriculum (ESC Core Curriculum). The textbook particularly appeals to vascular biologists, cardiologists, and other practising clinicians.
Managing for Outcome CRC Press
Tissue Factor Expression, Regulation, and Signaling in Human Airway Cells
CNS Cancer Springer
Tissue Factor (TF) is the cell surface receptor that activates coagulation by binding the serine protease coagulation factor Vila (Vila). The activation of the coagulation cascade leads to thrombin generation, fibrin formation and platelet activation which together aide tumor growth and metastasis. While the role of TF in metastasis through thrombin pathways is well established, evidence is increasing that TF may drive tumor development dependent on cell signaling pathways. A newly developed breast cancer model with a tetracycline regulated TF expression cassette shows TF enhances breast cancer tumor growth. This model will be useful to mechanisms by which TF enhances breast cancer progresssion. In this grant, we further evaluated the role of the TF cytoplasmic domain in breast cancer progresssion. We established tumor prone transgenic models in the C57B1/6 background and compared tumor development in TF cytoplasmic domain deleted mice with wild-type animals. Consistent with a recent report, we found that the C57B116 03-TAg model is unsuitable for studying breast cancer, because mice developed debilitating chondromatosis prior to the appearance of breast tumors. Experiments are ongoing to evaluate the role of the TF cytoplasmic domain in breast cancer development and progression to metastatic disease in the PyMT model.
Mechanical Stretch and Cytokines Springer Science & Business Media
Systemic lupus erythematosus (SLE), commonly called lupus, is a chronic autoimmune disorder that can affect virtually any organ of the body. In lupus, the body's immune system, which normally functions to protect against foreign invaders, becomes hyperactive, forming antibodies that attack normal tissues and organs, including the skin, joints, kidneys, brain, heart, lungs, and blood. Lupus is characterized by periods of illness, called flares, and periods of wellness or remission. Because its symptoms come and go and mimic those of other diseases, lupus is difficult to diagnose. There is no single laboratory test that can definitively prove that a person has the complex illness. To date, lupus has no known cause or cure. Early detection and treatment are the key to a better health outcome and can usually lessen the progression and severity of the disease. Anti-inflammatory drugs, antimalarials, and steroids (such as cortisone and others) are often used to treat lupus. Cytotoxic chemotherapies, like those used in the treatment of cancer, are also used to suppress the immune system in lupus patients. A new edition of this established and well-regarded reference combines basic science with clinical science to provide a translational medicine model. Systemic Lupus Erythematosus, Sixth Edition, is a useful reference for specialists in the diagnosis and management of patients with SLE, a tool for measurement of clinical activity for pharmaceutical development and basic research of the disease, and a reference work for hospital libraries. Completely updated, revised, and expanded with the most comprehensive and accessible reference on SLE for clinicians and scientists Full-color presentation throughout the book Provides the latest information available on diagnosis and treatment Incorporates an international panel of authors who are experts in their fields, with an emphasis on young, cutting-edge scientists and physicians
Models, Markers, Prognostic Factors, Targets, and Therapeutic Approaches Frontiers Media SA
Signal transduction comprises the intracellular biochemical signals which induce the appropriate cell response to an external stimulus. The players in signal transduction are diverse, from small molecules as first messengers, to proteins, receptors, transcription factors, among many others. The different signaling pathways and the crosstalk between them originates the unique signaling profile of every cell type in the human body. The cell signaling specificity depends on several aspects including protein composition, subcellular localization and complexes and gene promoters. This textbook provides a comprehensive overview of the specific signaling pathways on a variety of human tissues. This information can be of great value for health science researchers, professionals and students to understand key pathways for tissue-specific functions in the plethora of signals, signals receptors, transducers and effectors. Chapter 3 and 15 are available open access under a Creative Commons Attribution 4.0 International License via link.springer.com.
Cumulated Index Medicus Oxford University Press
The microcirculation is highly responsive to, and a vital participant in, the inflammatory response. All segments of the microvasculature (arterioles, capillaries, and venules) exhibit characteristic phenotypic changes during inflammation that appear to be directed toward enhancing the delivery of inflammatory cells to the injured/infected tissue, isolating the region from healthy tissue and the systemic circulation, and setting the stage for tissue repair and regeneration. The best

characterized responses of the microcirculation to inflammation include impaired vasomotor function, reduced capillary perfusion, adhesion of leukocytes and platelets, activation of the coagulation cascade, and enhanced thrombosis, increased vascular permeability, and an increase in the rate of proliferation of blood and lymphatic vessels. A variety of cells that normally circulate in blood (leukocytes, platelets) or reside within the vessel wall (endothelial cells, pericytes) or in the perivascular space (mast cells, macrophages) are activated in response to inflammation. The activation products and chemical mediators released from these cells act through different well-characterized signaling pathways to induce the phenotypic changes in microvessel function that accompany inflammation. Drugs that target a specific microvascular response to inflammation, such as leukocyte-endothelial cell adhesion or angiogenesis, have shown promise in both the preclinical and clinical studies of inflammatory disease. Future research efforts in this area will likely identify new avenues for therapeutic intervention in inflammation.

Recent Advances in Thrombosis and Hemostasis Academic Press

Head formation requires the well-orchestrated and harmonised development of various tissues and organs within the craniofacial complex. A big variety of signaling pathways are involved in this process by controlling cell proliferation, migration, differentiation, tissue morphogenesis, homeostasis and regeneration. Deregulation and malfunction of these signaling molecules may lead to mild or severe craniofacial pathologies. This eBook is a collection of articles dealing with a variety of important signals involved in the control of developmental and pathological events of craniofacial organs and tissues. These recent advances show the importance of signaling pathways in craniofacial physiology and pathology and generate important new knowledge aiming the development of new pharmaceutical products that mimic and/or block the actions of specific molecules.

Coordinated Interactions Between Growth Factor Receptor and Integrin Signaling Pathways in Breast Tissue-like Structure Biota Publishing

Cancers of the central nervous system are among the most lethal of human neoplasms. They are recalcitrant to even intensive multimodality therapies that include surgery, radiotherapy, and chemotherapy. Moreover, especially in children, the consequences of these therapies can itself be devastating and involve serious cognitive and developmental disorders. It is small wonder that such cancers have come under the intense scrutiny of each of the subspecialties of clinical care and investigation as well as attracting some of the best basic research scientists. Their joint efforts are gradually peeling away the mysteries surrounding the genesis and progression of these tumors and inroads are being steadily made into understanding why they resist therapies. This makes it an especially opportune time to assemble some of the best investigators in the field to review the ‘‘state of the art’’ in the various arenas that comprise the assault on CNS tumors. The breadth of this effort by the clinical and basic neuro-oncology community is quite simply amazing. To a large extent, it evolves from the knowledge of the human genome and its regulation that has been hard won over the past two decades.