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SHARP HOLLAND

Mitochondrial Diseases Createspace Independent Pub
Progress in the field of genetics is moving faster and demonstrating accomplishments unlike ever before. Genes marking for specific diseases and methods in gene therapy are evolving rapidly and being incorporated into daily patient care. Ethical issues are under constant debate by politicians, journalists, and laymen. All health care providers need to stay informed on the research, the applicability to patient management, and the moral issues involved. Case Studies in Genes and Disease tackles all these issues for those who need it most: busy clinicians who daily see patients needing to know how advances in genetic research and therapy affect their health. Written for practitioners who are not geneticists, it does not presume an expert's familiarity with the subject. From fundamentals to specific diseases to "the dark side" of genetics, Case Studies in Genes and Disease will educate, intrigue, and astound you.
Mitochondrial Function Springer Science & Business Media
Radiations, or Evolution in Action We have just celebrated the “Darwin Year” with the double anniversary of his 200th birthday and 150th year of his masterpiece, “On the Origin of Species by means of Natural Selection”. In this work, Darwin established the factual evidence of biological evolution, that species change over time, and that new organisms arise by the splitting of ancestral forms into two or more descendant species. However, above all, Darwin provided the mechanisms by arguing convincingly that it is by natural selection – as well as by sexual selection (as he later

added) – that organisms adapt to their environment. The many discoveries since then have essentially con?rmed and strengthened Darwin’s central theses, with latest evidence, for example, from molecular genetics, revealing the evolutionary relationships of all life forms through one shared history of descent from a common ancestor. We have also come a long way to progressively understand more on how new species actually originate, i. e. on speciation which remained Darwin’s “mystery of m- teries”, as noted in one of his earliest transmutation notebooks. Since speciation is the underlying mechanism for radiations, it is the ultimate causation for the biological diversity of life that surrounds us.

Mitochondrial Dynamic Abnormalities in Alzheimer's Diease IOS Press

Alzheimer's Disease (AD) is the most common cause of dementia leading to progressive memory loss and neurodegeneration in the hippocampus and frontal cortex. While it has been shown that mitochondrial dysfunction is an early and prominent feature in the progression of AD, it is unclear whether mitochondrial dysfunction itself can lead to neurodegeneration in AD-affected brain regions. Evidence has already suggested that various mitochondrial dynamic proteins (DLP1, OPA1, Mfn1, Mfn2, Fis1) are altered in AD and that there is an imbalance of mitochondrial fission and fusion yet there is a knowledge gap of whether this altered dynamics leads to neurodegeneration and what mechanisms lead to altered mitochondrial proteins in AD. To answer these questions, we created an Mfn2 conditional knockout mouse to recapitulate mitochondrial fragmentation phenotype in the hippocampus and frontal cortex. We found that indeed loss of mitochondrial fusion leads to mitochondrial morphological and bioenergetics abnormalities. These early changes lead to a series of events including oxidative stress, inflammation, and microtubule abnormalities that precede neurodegeneration. To understand the underlying mechanisms leading to loss of important mitochondrial dynamic proteins in AD, we treated primary neurons with amyloid-beta derived diffusible ligands to mimic AD and we found that the loss of DLP1 and Mfn2 is attributed to the calcium-activated protease, calpain. We also found that calpain specifically cleaves DLP1 leading the appearance of several cleavage fragments in both AD transgenic mice as well as AD patient brains. Altogether, these studies show that loss of mitochondrial dynamics could lead to neurodegeneration and that the loss of mitochondrial dynamic proteins in AD could be through the activity of calcium-activated proteases such as calpain.

Cancer as a Metabolic Disease Springer Science & Business Media

Mitochondrial Case StudiesAcademic Press

Oxidative Stress in Vertebrates and Invertebrates CRC Press

This interactive clinical textbook takes a system- and case-based approach in understanding mitochondrial disorders in clinical practice.

Mitochondrial Dysfunction Caused by Drugs and Environmental Toxicants Academic Press

This book addresses the therapeutic strategies to target mitochondrial metabolism in diseases where the function of that organelle is compromised, and it discusses the effective strategies used to create mitochondrial-targeted agents that can

become commercially available drug delivery platforms. The consistent growth of research focused in understanding the multifaceted role of mitochondria in cellular metabolism, controlling pathways related with cell death, and ionic/redox regulation has extended the research of mitochondrial chemical-biological interactions to include various pharmacological and toxicological applications. Not only does the book extensively cover basic mitochondrial physiology, but it also links the molecular interactions within these pathways to a variety of diseases. It is one of the first books to combine state-of-the-art reviews regarding basic mitochondrial biology, the role of mitochondrial alterations in different diseases, and the importance of that organelle as a target for pharmacological and non-pharmacological interventions to improve human health. The different chapters highlight the chemical-biological linkages of the mitochondria in context with drug development and clinical applications.

Maternal Control of Development in Vertebrates Academic Press

Most strokes are attributed to atherosclerosis of neck and intracranial arteries, brain embolism from the heart, and penetrating artery disease; these are discussed in detail in many other books. This compendium fills an important niche by providing authoritative discussions on the other, less common causes of stroke, including various forms of angiitis, coagulation disorders, infective, paraneoplastic and metabolic disorders that may be associated with stroke, and a number of rare syndromes such as Eales disease and Fabry's disease. This new edition contains detailed, up-to-date information about the nature, diagnosis, and treatment of those relatively uncommon types of cerebrovascular disease that cause strokes. It is therefore a unique scientific and clinical resource that provides a useful reference to help physicians diagnose and treat stroke patients who do not fit well into the usual clinical categories. New chapters include stroke in patients with Lyme disease, scleroderma, Cogan's syndrome, Chagas' disease, and HIV.

Targeting Mitochondrial Pathways in Obesity and Type 2 Diabetes National Academies Press

It was once assumed that mitochondrial diseases were rare and that few people were affected. As knowledge has grown about these organelles and their function, it became clear that mitochondrial malfunction could be linked to several chronic diseases. Diabetes has been associated with DNA mutation and can cause mutation itself. This text discusses findings involving the effects of disease on mitochondrial number, mitogenesis, and the base sequence of mitochondrial DNA. Experts discuss their study of mitochondria and what happens when it malfunctions. This book also explores the idea that mutated mitochondrial DNA can result in disease, and vice versa.

Complementary Therapies for the Body, Mind and Soul John Wiley & Sons

Complementary Therapies (CT) refers to the practices, products, or health systems that are outside the realm of conventional medicine, used to treat disease or to promote health and well-being. Defining CT is difficult, because the field is very broad and constantly changing. The title of this book includes the words body, mind, and soul. The body and the mind (and their reciprocal relations) have been extensively studied scientifically. What about the soul? The book brings some points about this new

ground in CT. We hope you find in the present work the sincere desire to collaborate with the dissemination of knowledge. May this book be useful and pleasant to you.

Alzheimer's Disease John Wiley & Sons

The explosion of insights in the field of metabolic disease has shed new light on diagnostic as well as treatment options. 'Inherited Metabolic Disease – A Clinical Approach' is written with a reader-friendly consistent structure. It helps the reader to find the information in an easily accessible and rapid way when needed. Starting with an overview of the major groups of metabolic disorders it includes algorithms with questions and answers as well as numerous graphs, metabolic pathways, and an expanded index. Clinical and diagnostic details with a system and symptom based are given to facilitate an efficient and yet complete diagnostic work-up of individual patients. Further, it offers helpful advice for emergency situations, such as hypoglycemia, hyperammonemia, lactic acidosis or acute encephalopathy. Five different indices allow a quick but complete orientation for common important constellations. Last but not least, it has an appendix with a guide to rapid differential diagnosis of signs and symptoms and when not to suspect metabolic disease. It will help physicians to diagnose patients they may otherwise fail to diagnose and to reduce unnecessary referrals. For metabolic and genetic specialists especially the indices will be helpful as a quick look when being called for advice. It has all it needs to become a gold standard defining the clinical practice in this field.

Diagnosis and Management of Mitochondrial Disorders Springer Science & Business Media

Mitochondria are subcellular organelles evolved by the endosymbiosis of bacteria with eukaryotic cells. They are the main source of ATP in the cell and engaged in other aspects of cell metabolism and cell function, including the regulation of ion homeostasis, cell growth, redox status, and cell signaling. Due to their central role in cell life and death, mitochondria are also involved in the pathogenesis and progression of human diseases/conditions, including neurodegenerative and cardiovascular disorders, cancer, diabetes, inflammation, and aging. However, despite the increasing number of studies, precise mechanisms whereby mitochondria are involved in the regulation of basic physiological functions, as well as their role in the cell under pathophysiological conditions, remain unknown. A lack of in-depth knowledge of the regulatory mechanisms of mitochondrial metabolism and function, as well as interplay between the factors that transform the organelle from its role in pro-survival to pro-death, have hindered the development of new mitochondria-targeted pharmacological and conditional approaches for the treatment of human diseases. This book highlights the latest achievements in elucidating the role of mitochondria under physiological conditions, in various cell/animal models of human diseases, and in patients.

Mitochondrial Medicine MDPI

Mitochondria are critical to the survival of cells, therefore, it is not surprising that abnormalities in mitochondrial function may lead to human disease. This book concentrates on the biology and pathology of mitochondria, covering some ot the important basic science features of the biology of mitochondria. It then moves on to discuss the breadth of human diseases related to

mitochondrial dysfunction, including Parkinson's disease, Amyotrophic Lateral Sclerosis (ALS), and Alzheimer's disease. * Provides comprehensive coverage of basic science and clinical features of mitochondrial dysfunction * Presents detailed analysis of "hot" topics in mitochondrial function and neurodegenerative diseases * Includes outstanding list of contributing authors

Mitochondrial Dysfunction in Ageing and Diseases CRC Press

Developed as a one-stop reference source for drug safety and toxicology professionals, this book explains why mitochondrial failure is a crucial step in drug toxicity and how it can be avoided. • Covers both basic science and applied technology / methods • Allows readers to understand the basis of mitochondrial function, the preclinical assessments used, and what they reveal about drug effects • Contains both in vitro and in vivo methods for analysis, including practical screening approaches for drug discovery and development • Adds coverage about mitochondrial toxicity underlying organ injury, clinical reports on drug classes, and discussion of environmental toxicants affecting mitochondria

Uncommon Causes of Stroke Elsevier

Methods in Toxicology, Volume 2: Mitochondrial Dysfunction provides a source of methods, techniques, and experimental approaches for studying the role of abnormal mitochondrial function in cell injury. The book discusses the methods for the preparation and basic functional assessment of mitochondria from liver, kidney, muscle, and brain; the methods for assessing mitochondrial dysfunction in vivo and in intact organs; and the structural aspects of mitochondrial dysfunction are addressed. The text also describes chemical detoxification and metabolism as well as specific metabolic reactions that are especially important targets or indicators of damage. The methods for measurement of alterations in fatty acid and phospholipid metabolism and for the analysis and manipulation of oxidative injury and antioxidant systems are also considered. The book further tackles additional methods on mitochondrial energetics and transport processes; approaches for assessing impaired function of mitochondria; and genetic and developmental aspects of mitochondrial disease and toxicology. The text also looks into mitochondrial DNA synthesis, covalent binding to mitochondrial DNA, DNA repair, and mitochondrial dysfunction in the context of developing individuals and cellular differentiation. Microbiologists, toxicologists, biochemists, and molecular pharmacologists will find the book invaluable.

Clinical Mitochondrial Medicine BoD – Books on Demand

Historically, regulations governing chemical use have often focused on widely used chemicals and acute human health effects of exposure to them, as well as their potential to cause cancer and other adverse health effects. As scientific knowledge has expanded there has been an increased awareness of the mechanisms through which chemicals may exert harmful effects on human health, as well as their effects on other species and ecosystems. Identification of high-priority chemicals and other chemicals of concern has prompted a growing number of state and local governments, as well as major companies, to take steps beyond existing hazardous chemical federal legislation. Interest in approaches and policies that ensure that any new substances substituted for chemicals of concern are assessed as carefully and thoroughly as possible has also burgeoned. The overarching goal of these approaches is to avoid regrettable substitutions,

which occur when a toxic chemical is replaced by another chemical that later proved unsuitable because of persistence, bioaccumulation, toxicity, or other concerns. Chemical alternative assessments are tools designed to facilitate consideration of these factors to assist stakeholders in identifying chemicals that may have the greatest likelihood of harm to human and ecological health, and to provide guidance on how the industry may develop and adopt safer alternatives. A Framework to Guide Selection of Chemical Alternatives develops and demonstrates a decision framework for evaluating potentially safer substitute chemicals as primarily determined by human health and ecological risks. This new framework is informed by previous efforts by regulatory agencies, academic institutions, and others to develop alternative assessment frameworks that could be operationalized. In addition to hazard assessments, the framework incorporates steps for life-cycle thinking - which considers possible impacts of a chemical at all stages including production, use, and disposal - as well as steps for performance and economic assessments. The report also highlights how modern information sources such as computational modeling can supplement traditional toxicology data in the assessment process. This new framework allows the evaluation of the full range of benefits and shortcomings of substitutes, and examination of tradeoffs between these risks and factors such as product functionality, product efficacy, process safety, and resource use. Through case studies, this report demonstrates how different users in contrasting decision contexts with diverse priorities can apply the framework. This report will be an essential resource to the chemical industry, environmentalists, ecologists, and state and local governments.

Case Studies in Genes and Disease John Wiley & Sons

Mitochondria are crucial organelles for any cell type. Mitochondria take responsibility for not only energy production but also regulation of cell death, also called apoptosis; calcium storage; and heat production. Therefore, mitochondrial disease is implicated in the mode of action of many harmful factors for cells such as drugs and environmental contaminants, dysfunction of the oxygen transport system, malnutrition, intense exercise, and genetic variations. This book presents up-to-date knowledge about mitochondrial disease and its complex relation to some diseases such as cardiac failure, cancer, and Alzheimer's and Parkinson's diseases. This book will, therefore, be essential for readers who are interested in life sciences, especially in medicine.

Drug Discovery Toxicology Springer

A large number of newly-synthesized polypeptides must cross one or several intracellular membranes to reach their functional locations in the eukaryotic cell. The mechanisms of protein trafficking, in particular the post-translational targeting and membrane translocation of proteins, are of fundamental biological importance and are the focus of intensive research world-wide. For more than 15 years, mitochondria have served as the paradigm organelle system to study these processes. Although key questions, such as how precisely proteins cross a membrane, still remain to be answered, exciting progress has been made in understanding the basic pathways of protein import into mitochondria and the components involved. In addition to a fascinating richness and complexity in detail, the

analysis of mitochondrial protein import has revealed mechanistic principles of general significance: Major discoveries include the demonstration of the requirement of an unfolded state for translocation and of the essential role of molecular chaperones on both sides of the membranes in maintaining a translocation-competent conformation and in protein folding after import. It is becoming clear how a polypeptide chain is "reeled" across the membrane in an ATP-dependent process by the functional cooperation of membrane proteins, presumably constituting part of a transmembrane channel, with peripheral components at the trans-side of the membrane. In this volume, eminent experts in the field take the time to review the central aspects of mitochondrial biogenesis. The logical order of the 16 chapters is determined by the sequence of steps during protein import, starting with the events taking place in the cytosol, followed by the recognition of targeting signals, the translocation of precursor proteins across the outer and inner membranes, their proteolytic processing and intramitochondrial sorting, and finally their folding and oligomeric assembly. In addition, the mechanisms involved in the export of mitochondrially encoded proteins as well as recent advances in understanding the division and inheritance of mitochondria will be discussed.

Mitochondria in Health and Diseases Cambridge University Press

This is the definitive, one-stop resource on preclinical drug evaluation for potential mitochondrial toxicity, addressing the issue upfront in the drug development process. It discusses mitochondrial impairment to organs, skeletal muscle, and nervous systems and details methodologies used to assess mitochondria function. It covers both in vitro and in vivo methods for analysis and includes the latest models. This is the authoritative reference on drug-induced mitochondrial dysfunction for safety assessment professionals in the pharmaceutical industry and for pharmacologists and toxicologists in both drug and environmental health sciences.

Mitochondrial Case Studies John Wiley & Sons

Developed as a one-stop reference source for drug safety and toxicology professionals, this book explains why mitochondrial failure is a crucial step in drug toxicity and how it can be avoided. • Covers both basic science and applied technology / methods • Allows readers to understand the basis of mitochondrial function, the preclinical assessments used, and what they reveal about drug effects • Contains both in vitro and in vivo methods for analysis, including practical screening approaches for drug discovery and development • Adds coverage about mitochondrial toxicity underlying organ injury, clinical reports on drug classes, and discussion of environmental toxicants affecting mitochondria

Protein Targeting to Mitochondria Elsevier

Mitochondria are far more than the “powerhouse” of the cell as they have classically been described. In fact, mitochondria biological activities have progressively expanded to include not only various bioenergetic processes but also important biosynthetic pathways, calcium homeostasis and thermogenesis, cell death by apoptosis, several different signal transduction pathways mainly related to redox control of gene expression and so on. This functional and structural complexity may undergo important derangements so to justify the definition of ‘mitochondrial medicine’, which should include all the clinical

consequences of congenital or acquired mitochondrial dysfunctions. There are actually a growing number of studies which assign a significant pathogenic role to damaged mitochondria in different diseases: ischemia/reperfusion injury, neurodegenerative diseases, cancer with its dramatic sequelae (i.e, metastasis), metabolic syndrome, hyperlipidemias, just to mention a few of the most important pathologies. In this context, a further aspect that should not be disregarded is the interaction of pharmacological agents with mitochondria, not only in regard of the toxicological aspects but, above all, of the potential therapeutic applications. In fact, it is interesting to note that, while the properties of different so-called “mitoxicants” are well-known, the subtle linkages between drugs and mitochondria is still in need of a real pharmacological and therapeutic control at the clinical level. This lack of consideration can often lead to an underestimation of unwanted toxic effects but also of desirable therapeutic activities. A reevaluation of the potential clinical role of mitochondria could give a new light on some yet obscure aspects of human pathophysiology.