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Mitochondria in Liver Disease (Oxidative Stress and ...

Abstract. Mitochondria are critical for respiration in all tissues; however, in liver, these organelles also accommodate high-capacity anaplerotic/cataplerotic pathways that are essential to gluconeogenesis and other biosynthetic activities. During nonalcoholic fatty liver disease (NAFLD), mitochondria also produce ROS that damage hepatocytes, trigger inflammation, and contribute to insulin resistance.

Mitochondrial metabolism mediates oxidative stress and ...

During nonalcoholic fatty liver disease (NAFLD), mitochondria also produce ROS that damage hepatocytes, trigger inflammation, and contribute to insulin resistance....

Mitochondrial metabolism mediates oxidative stress and ...

Abstract . Oxidative stress and hepatic mitochondria play a role in the pathogenesis of nonalcoholic fatty liver disease. The aim of the present study was to evaluate the role of hepatic mitochondrial dysfunction and oxidative stress in the pathogenesis of the disease.

Liver mitochondrial dysfunction and oxidative stress in ...

Assessment of mitochondrial function in metabolic dysfunction-associated fatty liver disease using obese mouse models Metabolic dysfunction-associated fatty

liver disease (MAFLD) is characterized by deregulated hepatic lipid metabolism; however, the association between MAFLD development and mitochondrial dysfunction has yet to be confirmed.

Assessment of mitochondrial function in metabolic ...

These roles assign mitochondria a gateway function in protecting hepatocyte from injury since unbalanced mitochondrial function unequivocally affects cell survival by actively causing the onset and perpetuation of liver diseases. Abnormal mitochondrial function is reported to be involved in a variety of liver diseases including drug-induced liver injury, alcoholic liver disease, non-alcoholic fatty liver disease, viral hepatitis, primary and secondary cholestasis, hemochromatosis, and Wilson ...

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Indexes of mitochondrial integrity, calcium loading capacity, and oxidative phosphorylation efficiency were unchanged between liver mitochondria from treated and control animals. In the animals tested, no evidence of degraded mitochondrial function due to nimesulide administration could be found.

Unaltered hepatic oxidative phosphorylation and ...

Conclusion: As mitochondria are a major source of ROS, the present review summarizes the role of mitochondrial oxidative stress in ASH and NASH and presents emerging data indicating the need to preserve mitochondrial antioxidant balance as a potential approach for the treatment of human fatty liver disease, which may pave the way for the design of

future trials to test the therapeutic role of antioxidants in fatty liver disease.

Mitochondrial Oxidative Stress and Antioxidants Balance in ...

Abnormal mitochondrial function is reported to be involved in a variety of liver diseases including drug-induced liver injury, alcoholic liver disease, non-alcoholic fatty liver disease, viral...

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mitochondrial liver disease. Although liver congestion caused by cardiomyopathy was present, it was specu-lated that the mitochondria mutation in the liver caused oxidative stress and cellular damage, and induced the hepatic failure in addition to the damage from liver congestion. In terms of hearing loss, it has been generally specu-

Hepatic Failure and Enhanced Oxidative Stress in ...

Mitochondrial respiratory chain is the main subcellular source of reactive oxygen species (ROS), which may damage mitochondrial proteins, lipids and mitochondrial DNA. Cardiolipin, a phospholipid located at the level of the inner mitochondrial membrane, plays an important role in several reactions and processes involved in mitochondrial bioenergetics as well as in mitochondrial dependent steps of apoptosis.

Oxidative stress, cardiolipin and mitochondrial ...

mitochondria) may be especially sensitive to oxidative stress, resulting in diminished energy production. Medications that reduce oxidative stress in mitochon-dria may ameliorate liver disease. KEY

WORDS: ethanol metabolism; physiological stress; oxidation-reduction; pathogenesis; alcoholic liver disorder; glutathione;

OXIDATIVE STRESS AND ALCOHOLIC LIVER DISEASE

Excess amounts of oxidant agents may be released in liver mitochondria in various cases of liver diseases, including ALD. Clinical research has revealed that mitochondria that are chronically exposed to ethanol display increased production of ROS and undergo several irreversible changes.

Oxidative stress as a crucial factor in liver diseases

One suggested mechanism involves the oxidation of biomolecules by mitochondrial ROS which initiates a vicious cycle of exacerbated mitochondrial

dysfunction and increased hepatocellular oxidative damage. This may ultimately pave the way for hepatic inflammation and liver failure.

Mitochondria in non-alcoholic fatty liver disease ...

Abstract. Aging induces liver morphological, structural, and functional changes that are consistent with increased levels of reactive oxygen species and oxidative damage. Aging not only increases oxidative-induced mitochondrial damage but also induces mitochondrial dysfunction in the liver. Therefore increased damage to the mitochondria may set the aged liver at increased risk of liver disease and poor prognosis.

Aging, oxidative stress, mitochondrial dysfunction, and ...

Redox state constitutes an important

background of numerous liver disorders. The redox state participates in the course of inflammatory, metabolic and proliferative liver diseases. Reactive oxygen species (ROS) are primarily produced in the mitochondria and in the endoplasmic reticulum of hepatocyte …

Oxidative stress as a crucial factor in liver diseases

Mitochondria are the main energy source in hepatocytes and play a major role in extensive oxidative metabolism and normal function of the liver. This key role also assigns mitochondria a gateway function in the center of signaling pathways that mediate hepatocyte injury, because impaired mitochondrial functions affect cell survival and contribute to the onset and perpetuation of liver diseases.

"...excellent, well-organized, and timely." —Lester Packer and Enrique Cardenas, University of Southern California, Los Angeles, from the Series Preface The liver is a vital organ that is responsible for a wide range of functions, most of which are essential for survival. The multitude of functions the liver performs makes it vulnerable to a wide range of diseases. Mitochondrial dysfunction plays an important role in many liver diseases including drug-induced liver injury, alcoholic liver disease, and nonalcoholic fatty liver disease. Mitochondria in Liver Disease gathers the most current research regarding the role of mitochondria in the liver and various diseases to which it is susceptible. The book is separated into two sections, the first of which highlights the latest developments in mitochondrial research. It includes cutting-edge topics such as the regulation of mitochondrial

respiration using hydrogen sulfide and the regulation of mitochondrial fusion–fission via the endoplasmic reticulum. The second section reviews the most current research on the role of mitochondria in a wide range of liver diseases. It also addresses novel topics such as the importance of liver mitochondrial constituents as biomarkers of liver injury in plasma and as regulators of the immune system. Mitochondria in Liver Disease represents the current state of knowledge and research on mitochondrial roles in liver diseases. Written by a group of global experts, it provides an authoritative and comprehensive overview of the latest advances and methods that mark key starting points for future research.

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.

This volume is the first text to concisely yet comprehensively cover developments for both alcoholic and nonalcoholic fatty liver disease in an organized fashion. Aspects of these two diseases covered in the book include global epidemiology and risk factors, pathogenesis, animal models, hepatic and extra-hepatic malignancies, treatment models, and current and emerging therapies. Written by experts in the field, Alcoholic and Non-Alcoholic Fatty Liver Disease: Bench to Bedside is a valuable resource for gastroenterologists, pathologists, and hepatologists who treat

patients with alcoholic and nonalcoholic fatty liver disease.

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

The topic of alcohol and the liver is long overdue for re-evaluation. The authors of this book have clearly taken up this challenge. Human interest in alcohol dates back to the Neolithic period (circa 10,000 BC). Only a small amount of alcohol is found naturally in food. Therefore, humans have yet to adapt to the high quantities of alcohol consumed daily by alcohol abusers. Alcohol is a simple carbohydrate and is metabolized as a fuel. However, unlike more naturally occurring carbohydrates such as glucose, the pathways for its metabolism are not subject to the same checks and balances. Ethanol oxidation reduces NAD+, which starves reactions that depend on this substrate. This accounts for the accumulation of fat in alcoholic steatosis.

Alcohol metabolism generates significant amounts of reactive oxygen species (ROS), which damages mtDNA and subsequently mitochondrial integrity. The authors provide several lines of evidence that link the clinical picture of alcoholic liver disease with mitochondrial damage. Other important topics highlighted in this book include histo-pathological aspects of the disease. Clinical assessment of nutrition and vitamin supplementation is addressed as well. The book concludes with the highly topical and controversial aspect of liver transplantation in patients with alcoholic liver disease.

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.

This new edition of Bioenergetics presents a clear and up-to-date explanation of the chemiosmotic theory and covers mitochondria, bacteria, and chloroplasts. It takes account of the many newly determined structures, such as ATP synthase and the two photosystems of

photosynthesis, that provide molecular insight into chemiosmotic energy transduction. This edition includes additional color figures of protein structures and many newly drawn illustrations designed to enable the reader to grasp the fundamental insights that are derived from knowing the structure. Every chapter has been extensively revised and updated and a new chapter on the study of the bioenergetics of mitochondria in the intact cell is included to satisfy the enormous interest in this topic. Written for students and researchers alike, this book is the most current text on the chemiosmotic theory and membrane bioenergetics available. Key Features * Chapter on the study of bioenergetics of mitochondria in the intact cell * Appendix listing protein structure resources * Additional colour plates of protein structures * Many newly drawn illustrations * Website

This issue contains cutting edge information on oxidative stress in small animals. Topics will include oxidative stress and liver disease; mitochondrial pathophysiology, reactive oxygen species, and cardiovascular diseases; oxidative stress, aging, and CNS disease; free radicals and antioxidants in inflammatory processes. Marketing of novel therapies will be also addressed.

Since the publication of the first edition, there have been advances in both the diagnosis and the management of many of the cholestatic liver diseases. Cholestatic Liver Disease, Second Edition thoroughly updates the topics previously addressed, such as primary biliary cirrhosis, primary sclerosing cholangitis and cholestatic variants of drug hepatotoxicity and viral disease. New treatments, such as the

development of the farnesoid X receptor agonists for the treatment of PBC, are highlighted. Current guidelines and areas of uncertainty are also covered. Additionally, new chapters have been added to reflect the changing landscape of cholestatic liver disease. Cholestatic Liver Disease, Second Edition is a concise yet comprehensive summary of the current status of the field and is of value to clinicians and researchers interested in patients with cholestatic liver disease provide that will help to guide patient management and stimulate investigative efforts.

Liver Pathophysiology: Therapies and Antioxidants is a complete volume on morphology, physiology, biochemistry, molecular biology and treatment of liver diseases. It uses an integral approach towards the role of free radicals in the

pathogenesis of hepatic injury, and how their deleterious effects may be abrogated by the use of antioxidants. Written by the most prominent authors in the field, this book will be of use to basic and clinical scientists and clinicians working in the biological sciences, especially those dedicated to the study and treatment of liver pathologies. Presents the most recent advances in hepatology, with a special focus on the role of oxidative stress in liver injury. Provides in vivo and in vitro models to study human liver pathology. Explains the beneficial effects of antioxidants on liver diseases. Contains the most recent and modern treatments of hepatic pathologies, including, but not limited to, stem cells repopulation, gene therapy and liver transplantation.

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