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Diabetes Sex Differences in Type-2 Diabetes Potential Therapeutic Targets in the CAMP Signaling Pathway for the Prevention and Treatment of Diabetic Cardiomyopathy Metabolic Cardiomyopathy The Diabetic Heart Glucose Intake and Utilization in Pre-Diabetes and Diabetes Inflammatory Heart Diseases Diabetic Cardiomyopathy Myocardial Metabolism Cardiomyopathies Diabetes and Cardiovascular Disease Regulation of Sarcoplasmic Reticulum in Diabetic Cardiomyopathy Understanding Molecular Mechanisms in Diabetic Cardiomyopathy (DCM) Regulation of Sarcoplasmic Reticulum in Diabetic Cardiomyopathy Pathophysiology of Cardiovascular Disease The Solution to a Better Healthy Life Molecular and Functional Interactions Between Apolipoprotein O and Caveolin 3 in the Heart Ukpds The Heart in Diabetes Acute Heart Failure Diabetic Cardiomyopathy ABC of Obesity The Diabetes Textbook Non-coding RNAs in Cardiovascular Diseases Diabetes and Cardiovascular Disease Antioxidant-Antidiabetic Agents and Human Health Essential Radiology Review Diabetes Transplantation of IPS Cells Reduces Apoptosis and Fibrosis and Improves Cardiac Function in Streptozotocin-induced Diabetic Rats Mitochondrial Dysfunction Medical Management of Diabetes and Heart Disease Global Report on Diabetes Heart Disease and Diabetes Oxidative Stress in Heart Diseases Diabetes and Heart Failure: Pathogenesis and Novel Therapeutic Approaches Cardiac Energy Metabolism in Health and Disease Diabetes and Diabetic Complications Molecular Cardiology The ESC Textbook of Cardiovascular Medicine Mitochondrial Biology and Experimental Therapeutics

Diabetes mellitus (DM) is the most common metabolic disorder associated with high mortality, which is mostly due to its cardiovascular complications. Diabetic cardiomyopathy (CM) is characterized by abnormal ventricular function in the absence of DM-associated risk factors such as obesity, hypertension, hypercholesterolemia, or coronary artery disease. Oxidative stress plays a pivotal role in the development of diabetic CM, in which chronic hyperglycemia plays a major role. As this develops, the endogenous antioxidant system becomes suppressed and so cannot counter-balance the increased oxidative stress. The metabolic abnormalities of DM cause mitochondrial superoxide overproduction, which further enhances the production of other reactive species, including nitric oxide, hydroxyl radical, hydrogen peroxide and peroxy nitrite, causing aggravation of the myocardial damage. In addition, free-radical-mediated platelet activation in the narrowed arteries culminates in acute myocardial infarction and stroke, indirectly affecting cardiac function. This chapter focuses on various aspects of the oxidative stress induced by reactive species during the pathogenesis of diabetic CM. "Diabetes mellitus is one of the major causes of morbidity and mortality in the modern world. Persistent hyperglycaemia is responsible for several complications of diabetes, such as diabetic

retinopathy, diabetic nephropathy, diabetic neuropathy, diabetic cardiomyopathy, diabetic autonomic neuropathy, diabetic ketoacidosis, diabetic foot ulcer, gestational diabetes, and diabetic mastopathy. With the advancement of technology, several new drugs have been developed for treatment of diabetes and diabetic complications. Moreover, alternative and complementary medicines have also been employed to manage diabetes and its associated complications. Despite the availability of a plethora of therapeutic agents, the management of diabetes and diabetic complications is an uphill battle associated with several limitations. Various leading research groups across the world are investing billions of dollars to develop impeccable solutions for the treatment of diabetes and diabetic complications. This book focuses on the understanding of recent advancements in the pathogenesis of diabetes and diabetic complications, the molecular basis of the disease and recent advancements in diabetic treatment. The chapters are specifically dedicated to different complications associated with diabetes. Moreover, recent advances in treatments and medications in clinical trials for these complications are explained. Some chapters are dedicated to the use of herbal medicines, alternative and complementary therapy and personalized medicines. Furthermore, the role of epigenetics in diabetic complications is described, as well as the role of antidiabetic drugs and their interactions. Due to its widespread prevalence, diabetes is presently considered a pandemic. This book provides contemporary information to researchers and health care practitioners about diabetes and diabetic complications, which may pave the way for designing new strategies to manage diabetes and diabetic complications. Moreover, the insights on alternative and complementary medicines will help in providing a background for inclusion of these medicines as an important therapeutic option for treatment of diabetes"-- 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. Diabetes: Oxidative Stress and Dietary Antioxidants, Second Edition, builds on the success of the first edition, covering updated research on the science of oxidative stress in diabetes and the potentially therapeutic usage of natural antioxidants in

the diet and food matrix. The processes within the science of oxidative stress are not described in isolation, but rather in concert with other processes, such as apoptosis, cell signaling and receptor mediated responses. This approach recognizes that diseases are often multifactorial and oxidative stress is a single component of this. Since the publication of the first edition, the science of oxidative stress and free radical biology continues to rapidly advance with thousands of the research articles on the topic. New sections in this update cover the role of dietary advanced glycation end products (AGEs) in causing OS in diabetes, oxidative stress and diabetes-induced bone metabolism, and oxidative stress and diabetic foot ulcer. Saves clinicians and researchers time in quickly accessing the very latest details on a broad range of diabetes and oxidation issues Combines the science of oxidative stress and the putative therapeutic usage of natural antioxidants in the diet, its food matrix or plant Includes preclinical, clinical and population studies to help endocrinologists, diabetologists, nutritionists, dieticians and clinicians map out key areas for research and further clinical recommendations This book is intended to help the reader realize that the solution to better health, does not depend on your physician, drugs, or health care insurance. In many cases, it may be the choices of each individual, triggered by lifestyle. Except for congenital and epidemic diseases, which we may not have control over, the choice to be healthy or unhealthy is ultimately yours. Cardiovascular disease is a common cause of morbidity and mortality in people with diabetes, and it is an issue that is becoming increasingly important to both cardiologists and specialists in diabetes. The routine management of patients with diabetes now includes close attention to the methods to control hyperglycaemia that give maximum reduction in cardiovascular events. This pocketbook details the complications that diabetes presents in relation to heart disease, introduces the reader to various risk factors and discusses the possible treatments that can be explored. This second edition has also been updated throughout to include new trial data, new drugs and updates on treatment of diabetes in heart failure patients. The information in this book is presented in an easy to use format, and supplemented with key figures, tables and summarized research findings. This state-of-the-art reference details current and effective symptom-specific strategies for the diagnosis and management of diabetic patients-emphasizing the exploration of therapeutic options available for the treatment of accelerated coronary complications associated with diabetes. Addresses the pathophysiology underlying advanced heart Pathophysiology of Cardiovascular Disease has been divided into four sections that focus on heart dysfunction and its associated characteristics (hypertrophy, cardiomyopathy and failure); vascular dysfunction and disease; ischemic heart disease; and novel therapeutic interventions. This volume is a compendium of different approaches to understanding cardiovascular disease and identifying the proteins, pathways and processes that impact it. The book is an on-the-spot reference for residents and medical students seeking diagnostic radiology fast facts. Its question-and-answer format makes it a perfect quick-reference for personal review and studying for board examinations and re-certification. Readers can read the text from cover to cover to gain a general foundation of knowledge that can be built upon through practice or can use choice chapters to review a specific subspecialty

before starting a new rotation or joining a new service. With hundreds of high-yield questions and answer items, this resource addresses both general and subspecialty topics and provides accurate, on-the-spot answers. Sections are organized by subspecialty and body area, including chest, abdomen, and trauma, and chapters cover the anatomy, pathophysiology, differential diagnosis, hallmark signs, and image features of major diseases and conditions. Key example images and illustrations enhance the text throughout and provide an ideal, pocket-sized resource for residents and medical students. Diabetic cardiomyopathy is defined as heart failure independent of the coronary artery, valve disease and hypertension. It has multifactorial aetiology but the pathogenesis is incompletely understood. Hyperglycemia, hyperlipidemia and inflammation with high oxidative stress lead to structural and functional alterations of the left ventricle (LV) and promote diabetic cardiomyopathy. Diastolic dysfunction is an early sign of diabetic cardiomyopathy. It has a long asymptomatic period, but with time leads to loss of contractile function. Hence, the identification of subclinical diabetic cardiomyopathy and correction of potentially modified risk factors are very important to delay the onset of heart failure. The aim: We aimed to assess the LV function in asymptomatic diabetic patients and its correlation with clinical and biochemical parameters. Study design: Cross-sectional study that includes a total number of 137 subjects. The target group consists of 72 asymptomatic normotensive patients with diabetes mellitus type 2, without coronary artery and valve disease. The control group is composed of 65 healthy subjects. Methods: All patients were subject to echocardiography (conventional 2D, M-mode, PW Doppler analysis and contemporary techniques-TDI and 2D-Speckle-Tracking Echocardiography). We evaluate LV diastolic and systolic function and its correlation with basic clinical characteristics (age, gender, BMI, BSA, waist to hip ratio, duration of diabetes) and biochemical analyses (glucose profile, lipid profile, CRP). The correlation between clinical, biochemical and echocardiographic parameters was assessed by the Pearson Product Moment of Correlation. A p-value of During the last years the understanding for the aetiology of cardiomyopathies could be greatly improved. A great deal of information has accumulated in the field of inherited metabolic diseases, which provides a new basis for our understanding of many heart muscle problems and their corresponding clinical disease entities. This book is meant to give the reader a comprehensive overview of the cardiological manifestations of inborn errors of metabolism. Latest information, such as cardiomyopathy in Fabry disease or in patients with CDG-syndrome is included. It should be helpful, not only to cardiologists, paediatricians, internists and general practicioners, but also to all those interested in a better understanding of the metabolic basis of clinical disease entities. With the dramatic modification of the lifestyle in the last century, new pandemic public health issues have emerged. The increase in the prevalence of obesity and its strong association with cardiovascular diseases has aroused interest in the understanding of mechanisms linking metabolic disorders and cardiac dysfunctions. It has recently emerged that cardiac altered energy metabolism, lipotoxicity, insulin resistance and mitochondrial alterations are leading causes in the development of metabolic or diabetic cardiomyopathy. Through a functional genomics study aimed at identifying genes

differentially regulated in the heart by obesity, we discovered a new apolipoprotein (ApoO) which is also overexpressed in the myocardium from diabetic patients. In attempt to uncover how changes in the expression of this protein relate to modifications of cardiac function, we used cardiac myoblasts, human heart samples a well as cardiac specific transgenic mouse lines constitutively expressing ApoO at physiological levels. We show that ApoO localizes within mitochondria and induces mitochondrial dysfunction in mouse and human heart. ApoO interacts with adenine nucleotide translocase (ANT) which is known as mitochondrial permeability transition pore (mtPTP) regulator. This interaction enhances mtPTP opening, thereby inducing "mild uncoupling". Consequently, mitochondrial respiration, oxidative phosphorylation and fatty acid metabolism are enhanced. This cascade of events generates a mitochondrial metabolic sink whereby cells accumulate lipids and lipotoxic byproducts leading to apoptosis, loss of cardiac cells and cardiomyopathy, mimicking the metabolic phenotype of the diabetic heart. As a spin-off of these observations, we proposed a model for the original molecular mechanisms accounting for ApoO induced mitochondrial dysfunction and lipotoxicity. Besides, we observed that ApoO expressing cardiomyocytes develop adaptive mechanisms to protect cells from the ApoO-induced excessive oxidative metabolism. As revealed in human auricular heart samples and expression database from human heart ventricles, Caveolin-3 (Cav-3) expression is positively correlated to ApoO levels. Cav-3, the main caveolin isoform in cardiac myocytes, is known to have scaffolding domains that anchor and regulate the function of proteins, thereby modulating a variety of cellular processes. These properties make Cav-3 as an actor for cardiac protection. Interestingly, ApoO-induced metabolic stress, both in mouse heart and in vitro in cardiac cells, leads to a rise in Cav-3 levels and its translocation to mitochondria where it interacts with ApoO, through a direct association between the C-terminal scaffolding domain of Cav-3 and the specific aromatic caveolin binding motif (CBM) of ApoO. Blue native polyacrylamide gel electrophoresis of mouse heart mitochondrial protein complexes reveals that ApoO and Cav-3 are present in the same macromolecular complex with known mtPTP regulators. We show that ApoO and Cav-3 interaction results in a protective effect through reduction of ApoO-induced mild uncoupling and consequently restoration of coupled respiration and reduction in apoptosis. Site-directed mutagenesis in ApoO CBM domain prevents its interaction with Cav-3 and led to a loss of Cav-3-mediated protection, as reflected by strongly enhanced uncoupling which is considered as one hallmark of mitochondrial dysfunctions. Therefore, the involvement of ApoO and Cav-3 in mitochondrial homeostasis may reveal novel strategies to control pathophysiological situations involving mitochondrial dysfunctions, such as metabolic disorders and cardiomyopathies. Although Ca2+-transport activities in the cardiac sarcoplasmic reticulum (SR) have been shown to be depressed in chronic diabetes, the status of its regulatory mechanisms is not fully understood. Since Ca2+-calmodulin and cAMP-dependent protein kinases (CAMK and PKA) are known to stimulate SR function, it is possible that these enzymes may be altered in the diabetic heart. For this purpose, rats were made diabetic by an injection with streptozotocin; vehicle injected animals served as control. Some of the 4 week diabetic animals were

treated with insulin (3 U/day) for 2 weeks. Hearts were removed at 6 weeks after the induction of diabetes and the ventricular tissue was used for either SR preparation or other biochemical determination. The decreased level of glucose, increased level of insulin and depressed ventricular function in diabetic animals were prevented by insulin treatment. Both Ca 2+-uptake and Ca2+-release activities in SR preparations from diabetic hearts were decreased. The SR protein content as estimated by Western blot analysis for Ca2+-pump ATPase, Ca2+-release channels and phospholamban proteins were also decreased in the diabetic heart . Both CAMK- and PKA-mediated protein phosphorylations were increased in the diabetic SR. These changes in the diabetic heart were associated with increased SR CAMK, PKA and phosphatase activities. Although insulin treatment of diabetic animals provided partial recovery of SR function, it had no effect on changes in CAMK and PKA activities. These results suggest marked changes in the regulatory mechanisms for SR function in the diabetic hearts. This book bridges the gap between fundamental and translational research in the area of heart disease. It describes a multidisciplinary approach, and demonstrates biochemical mechanisms associated with dysregulation of redox signaling, which leads heart disease. Presenting recent studies on improved forms of ROS scavenging enzymes; specific inhibitors for different ROS generating enzymes; and oxidant induced signaling pathways and their antagonists that allow subtle modulation of redox signaling, it also discusses the spatial and temporal aspects of oxidative stress in the cardiovascular system, which are of vital importance in developing better strategies for treating heart disease. Each chapter offers researchers valuable insights into identifying targets for drug development for different types of heart disease. Diabetes has long been recognized as a disease of high blood sugar, and there has been a continuous search of the exact reason for its development and effective treatment. In 2005, the World Health Organization had estimated that more than 180 million people worldwide suffer from diabetes mellitus and indicated that this figure is likely to double within the next 20 years. Among the 3.8 million deaths each year associated with diabetes, about two thirds are attributable to cardiovascular complications, and diabetes is now considered to be a major metabolic risk factor for the occurrence of heart disease. Diabetic Cardiomyopathy: Biochemical and Molecular Mechanisms is a compilation of review articles devoted to the study on the topic with respect to biochemical and molecular mechanisms of hyperglycaemia. The wide range of areas covered here is of interest to basic research scientists, clinicians and graduate students, who are devoted to study the pathogenesis of diabetes-induced cardiovascular dysfunction. Furthermore, some chapters are directed towards increasing our understanding of novel ways for the prevention/treatment of cardiomyopathy. Twenty five articles in this book are organized in three sections. The first section discusses general aspects of the metabolic derangements in diabetic cardiomyopathy including metabolic alterations and substrate utilization as well as cardiac remodelling in the heart; role of diet in the development of metabolic syndrome in the heart; effect of hyperglycaemia in terms of biochemical and structural alterations in heart. In the second section, several cellular and molecular mechanisms are discussed indicating that diabetic cardiomyopathy is a

multifactorial and complex problem. The third section discusses the prevention and treatment of diabetes using appropriate diet, proper supplements including antioxidants, angiotensin inhibitors and some other drugs. All in all, this book discusses the diverse mechanisms of diabetic cardiomyopathy with some information on new therapeutic approaches for finding solutions to prevent or reverse the development of cardiac dysfunction. The rise of type 2 diabetes (T2DM) increases the risk of diabetic cardiomyopathy. Worsened cardiovascular outcomes have been clinically observed in female diabetic patients; however, there is a lack of sex specific treatments for diabetes associated heart complications. Caveolins are integral membrane proteins expressed in caveolae and mitochondria that act as cellular signaling platforms to regulate stress responses. The cardiac-specific caveolin-3 overexpression (Cav3 OE) has been shown to protect the heart from morphological changes induced by stressors and preserve mitochondrial function, but this has not yet been shown in a diabetic heart. We tested the rationale that Cav3 will protect the heart from diabetes-induced metabolic injury and be especially advantageous for females. Mid-aged male and female Cav3 OE mice and transgene-negative (Tg-Neg) littermates were either injected with streptozotocin (STZ) and fed a 60% kcal high-fat diet (HFD) to induce diabetes or injected with citrate buffer and fed a 4% kcal low-fat diet for controls. STZ/HFD significantly increased weight and impaired glucose tolerance of male and female mice, with worsened glucose tolerance in males. Tg-Neg T2DM mice presented cardiac hypertrophy in males and females, mitochondrial dysfunction in females, diastolic dysfunction in males, and cardiac mitochondrial disarray in males and females. Meanwhile, Cav3 OE prevented these incidences in T2DM mice. Therefore, Cav3 may be a novel target to protect diabetic mice, especially females, from cardiac complications. Cardiomyopathies are the most featured cardiac pathologies in the twenty-first century, that threaten public health and burden healthcare budgets. This book is composed of the main topics on pathophysiology, general forms and specific types of cardiomyopathies and it also introduces new research in the field. Specific forms with or without genetic inheritance are discussed separately to attract the readers' attention on these topics. Well-known medical follow-up strategies occur ineffective at the end-stage heart failure, however, new surgical approaches can be an alternative for these patients to get a chance at the last crossroad and to improve their life quality and survival and also to gain or prolong time until possible heart transplantation. Diabetes mellitus is a disease that affects a growing number of Americans, with the Type 2 form being increasingly observed in younger patients. Cardiovascular disease is a major complication of diabetes and includes diabetic cardiomyopathy, which occurs in the absence of coronary artery disease or hypertension. The mechanisms by which exposure to high glucose can induce the development of heart failure are unknown. One key contributor to diabetic cardiomyopathy is cardiac fibrosis, which results from increased accumulation of collagen and other extracellular matrix (ECM) components. Exposure of isolated rat cardiac fibroblasts to high glucose leads to their conversion to myofibroblasts (profibrogenic fibroblasts) that have increased ECM protein synthesis. Interestingly, fibroblast-to-myofibroblast transformation and fibrosis can be blunted by increases in the

second messenger adenosine 3'5' cyclicmonophosphate (cAMP). It is unclear whether expression or activity of components of the cyclic AMP (cAMP) pathway (G-protein-coupled receptors [GPCR, Gs and Gi linked], adenylyl cyclases [AC, which catalyze the formation of cAMP], phosphodiesterase [PDE, which catalyze cAMP degradation], and cAMP effectors [protein kinase A and Exchange protein activated by cAMP, Epac]) are altered in cardiac fibroblasts in response to high glucose. The specific aims of this project were to : (1) identify which components of the cAMP pathway are expressed in cardiac fibroblasts and determine if their expression changes in response to high glucose treatment, and (2) investigate if targeting cAMP components via GPCR agonists or antagonists can blunt myofibroblast actions in response to high glucose by increasing intracellular cAMP and its downstream signaling mediators. The results showed that TGF[Beta] induces a 2.5-fold increase in [alpha]SMA suggesting cardiac fibroblast to myofibroblast transformation. I compared respose to TGF[Beta] in media that contained a physiologically normal concentration (5 mM) of glucose (NG), a higher level (25 mM) of glucose (HG) or media that included mannitol as an osmotic control (OC) for the HG media. TGF[Beta] increased cardiac fibroblast [alpha]SMA mRNA expression 2.5-fold in NG and 1.5-fold in OC but not in HG media. TGF[Beta] and (angiotensin II) ATII increased cardiac fibroblast PAI-1 mRNA expression about 2.5-fold in OC media and 3.5-fold in HG media. Myofibroblasts had decreased mRNA expression of adenylyl cyclases 5 and 6 (AC5 and AC6) but increased mRNA expression of cyclic nucleotide phosphodiesterase (PDE) isoforms 4A, 4D, 8A, and 10A. ATII increased cardiac fibroblast collagen 1[alpha] 1 mRNA expression about 1.5 fold in OC and HG media. Forskolin attenuated ATII-induced collagen synthesis by about 50% in all 3 media. Collagen production was greater in OC and HG than in NG cardiac fibroblasts. I conclude that tonicity may play a greater role than glucose levels in mRNA expression of certain fibrotic genes and collagen synthesis in cardiac fibroblasts. Increasing cAMP levels can blunt myofibroblast fibrotic effects and might prove useful in the treatment or prevention of diabetes-induced cardiac fibrosis. This important reference, edited by Ronald Ross Watson and Betsy Dokken, collects the research needed to make the distinct connection between pre-diabetes, diabetes, and cardiovascular disease. Glucose Intake and Utilization in Pre-Diabetes and Diabetes: Implications for Cardiovascular Disease explains the mechanisms of progression from pre-diabetes to diabetes to cardiovascular disease. Since pre-diabetes and diabetes are important cardiovascular disease risk factors, and impaired glucose metabolism among cardiac patients is extremely prevalent, the importance of reviewing pre-diabetes and its involvement in CVD complications is vital as one applies food and glycemic control to slow progress to diabetes and heart disease. The book further focuses on glucose intake and utilization in diabetes, including coverage of diabetes in the development and pathology of cardiovascular disease, risks and epidemiology of cardiovascular problems promoted by diabetes, macrovascular effects and their safety in therapy of diabetics, beta cell biology and therapy of diabetes, and nutrition to modulate diabetes. Offers a complete review of cardiac health problems occurring with significant frequency in patients relative to their ability to regulate glucose Presents coverage of the

role of glucose utilization, development of pre-diabetes and the ultimate development of various cardiovascular diseases Provides thorough dietary, nutrition, complementary and alternative botanical therapies for pre-diabetes and diabetes to halt the progression to cardiovascular disease 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. Diabetes has become a worldwide health problem, the global estimated prevalence approaches ten percent and the burden of this disease in terms of morbidity and mortality is unprecedented. The advances acquired through the knowledge of the mechanisms of the disease and the variety of therapeutic approaches contrast with the inability of private and public health systems in underdeveloped and even developed countries to achieve the goals of treatment. This paradox has been described in many sources: the surge of scientific advances contrast with an unprecedented amount of human suffering. Thus, a patient centered and an evidence based approach with the capacity to produce measurable clinical and economic outcomes is required. The purpose of this textbook is multiple: to offer a comprehensive resource covering all aspects of outpatient management; to address diabetes as a health problem from an epidemiological, economic and clinical perspective; to discuss the role of social determinants of health on the worldwide increase in diabetes; to highlight the challenges and obstacles in providing adequate care; and to outline a multidisciplinary approach to management in which medical visits retain their importance as part of a team comprising the patient, his or her family and a multidisciplinary group of health professionals who are able to move beyond the traditional approach of diabetes as a disease and greatly improve outcomes. Background: Streptozotocin (STZ) induced diabetes leads to various complications including cardiomyopathy. Recent data suggests transplanted bone marrow stem cells improve cardiac function in diabetic cardiomyopathy. However, whether modified ES, iPS cells, or factors released from these cells can inhibit apoptosis and fibrosis remains completely unknown. The present study was designed to determine the effects of transplanted ES cells overexpressing pancreatic transcription factor 1 a (Ptf1a), a pro-pancreatic endodermal transcription factor, iPS cells, or their respective conditioned media (CM) on diabetic cardiomyopathy. Methods:

Experimental diabetes was induced in male Sprague Dawley rats (8-10 weeks old) by intraperitoneal STZ injections (65 mg/kg body weight for 2 consecutive days). Animals were divided into six experimental groups including control, treated with sodium citrate buffer IP, STZ, STZ + ES-Ptf1a cells, STZ + iPS cells, STZ + ES-Ptf1a CM and STZ + iPS CM. Following STZ injections, appropriate cells (1 X 106/mL/injection/day) or CM (2 mL injection/day) were given intravenously for 3 consecutive days. Animals were sacrificed and hearts were harvested at day 28. Histology, TUNEL staining, and Caspase-3 activity were used to assess apoptosis and fibrosis. ERK1/2 phosphorylation was quantified using ELISAs. M-mode echocardiography fractional shortening was used to assess cardiac function. Results: Animals transplanted with ES cells, iPS cells, or both CMs showed a significant (p[less than]0.05) reduction in interstitial fibrosis, and apoptosis compared with STZ group. ERK expression was not significantly different compared with STZ. Echocardiography showed a significant (p[less than]0.05) improvement in fractional shortening in cell and media transplanted groups compared with STZ. Conclusions: Our data suggest that ES cells, iPS cells, and/or CMs inhibit apoptosis, reduce fibrosis, and improve cardiac function in STZ-treated diabetic rats. This book presents the latest research on non-coding RNAs in cardiovascular disease, a major cause of death worldwide. Non-coding RNAs play a significant role in development, proliferation, differentiation and apoptosis. Since altered non-coding RNA expression is often associated with various diseases, their potential use in diagnostics, prognostics and therapeutics is an important current area of study. The book consists of six parts: 1) An overview of non-coding RNAs and cardiovascular system, 2) Bioinformatics and interactions, 3) Non-coding RNA regulation in cardiovascular system, 4) Non-coding RNAs and cardiovascular diseases, 5) Potential biomarkers and therapeutic implications, 6) Future prospects. It is particularly useful for researchers and students in the field of non-coding RNA and cardiovascular biology, as well as for cardiologists, pharmacologists and physiologists. Obesity is a hugely expensive and increasing problem worldwide,leading to disability, reproductive problems, depression andaccelerated metabolic and vascular diseases in a large proportionof men, women and children. The ABC of Obesity is a newguide which will aid its effective management, addressing issuessuch as dieting, exercise, self esteem, drug treatment and surgery.Recent evidence is used to highlight frequent problems, successfultreatment options, and the most common causes. Written by leading experts, this is a widely accessible text andan indispensable guide for all general practitioners, juniordoctors, nurses, and other healthcare professionals who areinvolved in the treatment and research of this commoncondition. The human system employs the use of endogenous enzymatic as well as non-enzymatic antioxidant defence systems against the onslaught of free radicals and oxidative stress. Enzymatic antioxidants and non-enzymatic antioxidants work synergistically with each other, using different mechanisms against different free radicals and stages of oxidative stress. Dietary and lifestyle modifications are seen as the mainstay of treatment and management of chronic diseases such as diabetes mellitus. The major aims of dietary and lifestyle changes are to reduce weight, improve glycaemic control and reduce the risk of coronary heart disease, which accounts for 70- 80% of

deaths among those with diabetes. It is also important to note that medicinal plants have been used as medicines since ancient time, and continue to play significant role even in modern medicine in management and treatment of chronic diseases. Impressive numbers of modern therapeutic agents have been developed from plants. Phytochemicals have been isolated and characterised from fruits such as grapes and apples, vegetables such as broccoli and onion, spices such as turmeric, beverages such as green tea and red wine, as well as many other sources. The WHO estimates that approximately 80% of the worlds inhabitants rely on traditional medicine for their primary health care and many medicinal plants have ethno-medical claims of usefulness in the treatment of diabetes and other chronic diseases globally, and have been employed empirically in antidiabetic, antihyperlipidemic, antihypertensive, antinflammatory and antiparasitic remedies. This book examines the role of antioxidant-rich natural products in management and treatment of diabetes and other chronic diseases. For many years, there has been a great deal of work done on chronic congestive heart failure while acute heart failure has been considered a difficult to handle and hopeless syndrome. However, in recent years acute heart failure has become a growing area of study and this is the first book to cover extensively the diagnosis and management of this complex condition. The book reflects the considerable amounts of new data reported and many new concepts which have been proposed in the last 3-4 years looking at the epidemiology, diagnostic and treatment of acute heart failure. The aim of Molecular Cardiology: Methods and Protocols is to document state-of-the-art molecular and genetic techniques in the area of cardiology. These modern approaches enable researchers to readily study heart diseases at the molecular level and will promote the development of new therapeutic str- egies. Methods for genetic dissection, signal transduction, and microarray analysis are excellent tools for the study of the molecular mechanisms of cardiovascular diseases. Protocols for transgenesis take advantage of recent advances in many areas of molecular and cell biology. Transgenic models of heart diseases (cardiac hypertrophy, cardiac dysfunction, and so on.) are powerful tools for the study of heart disease pathogenesis. Methods for gene transfer to heart tissue using viral and nonviral vectors form the basis of gene therapy for heart diseases. Heart-specific promoters containing a hypox- inducible cardioprotective gene switch are key for protection of the heart from ischemia. Gene and stem cell therapies open novel and exciting avenues for the prevention and treatment of heart diseases. Molecular Cardiology: Methods and Protocols consists of 26 chapters de- ing with various aspects of molecular cardiology, including gene transfer and gene therapy for cardiovascular disease, stem cell therapy for cardiovascular disease, gene analysis in the injured and hypertrophied heart, and transgenesis in cardiovascular research. This book provides step-by-step methods for the successful completion of experimental procedures, and would be useful for both experienced and new investigators in the field of molecular cardiology. Inflammatory Heart Diseases presents comprehensive information on pericardial diseases, cardiomyopathies, and atherosclerotic cardiovascular diseases. Chapters are written by experts in the field and cover such topics as advanced concepts in pericardial disease, pericardial disease in the elderly, inflammation and diabetic

cardiomyopathy, medical imaging in myocarditis, and the role of lifestyle in development of coronary heart disease, among others. "On the occasion of World Health Day 2016, WHO issues a call for action on diabetes, drawing attention to the need to step up prevention and treatment of the disease. The first WHO Global report on diabetes demonstrates that the number of adults living with diabetes has almost quadrupled since 1980 to 422 million adults. This dramatic rise is largely due to the rise in type 2 diabetes and factors driving it include overweight and obesity. In 2012 alone diabetes caused 1.5 million deaths. Its complications can lead to heart attack, stroke, blindness, kidney failure and lower limb amputation. The new report calls upon governments to ensure that people are able to make healthy choices and that health systems are able to diagnose, treat and care for people with diabetes. It encourages us all as individuals to eat healthily, be physically active, and avoid excessive weight gain."--Publisher's description. The heart has a very high energy demand but very little energy reserves. In order to sustain contractile function, the heart has to continually produce a large amount of ATP. The heart utilizes free fatty acids mainly and carbohydrates to some extent as substrates for making energy and any change in this energy supply can seriously compromise cardiac function. It has emerged that alterations in cardiac energy metabolism are a major contributor to the development of a number of different forms of heart disease. It is also now known that optimizing energy metabolism in the heart is a viable and important approach to treating various forms of heart disease. Cardiac Energy Metabolism in Health and Disease describes the research advances that have been made in understanding what controls cardiac energy metabolism at molecular, transcriptional and physiological levels. It also describes how alterations in energy metabolism contribute to the development of heart dysfunction and how optimization of energy metabolism can be used to treat heart disease. The topics covered include a discussion of the effects of myocardial ischemia, diabetes, obesity, hypertrophy, heart failure, and genetic disorders of mitochondrial oxidative metabolism on cardiac energetics. The treatment of heart disease by optimizing energy metabolism is also discussed, which includes increasing overall energy production as well as increasing the efficiency of energy production and switching energy substrate preference of the heart. This book will be a valuable source of information to graduate students, postdoctoral fellows, and investigators in the field of experimental cardiology as well as biochemists, physiologists, pharmacologists, cardiologists, cardiovascular surgeons and other health professionals. Diabetes is a major public health problem which is expected to affect 160 million people worldwide by the year 2000. Clearly an understanding of the effects of diabetes on the heart is an important step in the development of strategies to reduce the incidence of heart disease for diabetic patients, thus increasing their overall life-expectancy and quality of life. In this book, the editors bring together the different lines of evidence supportive of the idea of a diabetic cardiomyopathy. The first chapter provides an overview of the impact of cardiac dysfunction on the mortality and morbidity of the diabetic population in general, as well as a presentation of clinical aspects of heart disease in diabetes. This is followed by chapters concerned with the pathological and functional changes that occur in the heart as

a result of diabetes and a description of the various therapeutic interventions that are available to reverse the effects of diabetes on the heart. Subsequent chapters focus on changes in protein synthesis, membrane function and intermediary metabolism that take place following the onset of diabetes. Since these alterations precede many of the functional and pathological changes, it may be that the processes responsible for the functional decline and tissue injury are initiated by diabetes-induced changes at the cellular and/or biochemical level. Diabetes and cardiovascular disease together account for the largest portion of health care spending compared to all other diseases in Western society. This work seeks to provide an understanding of the causes of diabetes and its cardiovascular complications. As this understanding becomes more widely appreciated, it will serve as a foundation for evidence-based care and wider acceptance of sound science. The International Conference on Diabetes and Cardiovascular Disease, held in Winnipeg, in June 1999, was organized to bring together a multi-disciplinary group of researchers dedicated to further knowledge amongst researchers, care givers, and the managers of the health system. The invited speakers submitted their works for publication, which serves as the basis for this book. Major themes include: epidemiology of diabetes mellitus, metabolic risk factors in diabetes and cardiovascular disease, hypertension in diabetes mellitus, cardiac function in diabetes, glycemic control and improved cardiovascular function, diabetes management, and endothelial function in diabetes.

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