Cardiac Remodeling Molecular Mechanisms

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Cardiac Remodeling - Part 1 - The Pathogenesis

Cardiac Remodeling - Part 2 - Pharmacological Management~~Pathology \u0026 Remodeling of Heart Failure~~ ~~Dr Zhao Wang - \"Hexosamine biosynthesis in pathological cardiac remodeling and heart failure\".~~ ~~Molecular Mechanisms of Cardiac Hypertrophy and Failure~~ ~~Biomechanics of Cardiac Remodelling in Heart Failure~~ Significance of Cardiac Remodeling in Heart Failure *Pathophysiology of Heart Failure Part II: Types and compensatory and remodeling mechanisms Bone Marrow Cells in Cardiac Remodeling* **What is VENTRICULAR REMODELING? What does VENTRICULAR REMODELING mean?** *Cardiac plasticity Pathological Cardiac Hypertrophy Part 1 Enlarged Heart Animation Layers Of The Heart // Cardiology* ~~Depression: Monoamine Hypothesis~~

One Minute #CardioEd: What's the difference between concentric and eccentric LVH?

Anatomy for Electrophysiologists. Author: Maxim Didenko MD PhD FEHRA Educational movie.Left ventricular hypertrophy Hypertrophy - Classification - Examples Left sided vs. Right sided heart failure **Heart Failure 6, Renin angiotensin aldosterone system**

Heart Failure 5, Pathophysiology**Right Ventricular Remodeling in Olympic Athletes Healing after a heart attack (myocardial infarction) | NCLEX-RN | Khan Academy** *E. Dejana - Molecular mechanisms of vascular remodelling and their alterations* **^MuniHealth - #143 What Is LV Remodeling?** 4 CIRCULATION: Local blood flow control |Angiogenesis |Collaterals |vascular remodelling | Guyton

Gene-Centric Mechanisms, Diagnosis, and Treatment for Inherited Cardiomyopathy

Dr. Filio Billia: \"Molecular Mechanisms in Cardiomyopathy - From Mice to Men\"**Exercise training in adverse cardiac remodeling - Dr. Dirk Duncker Cardiac Remodeling Molecular Mechanisms**
Molecular Mechanisms of Cardiac Remodeling and Regeneration in Physical Exercise Cells. 2019 Sep 23;8(10):1128. doi: 10.3390/cells8101128. Authors Dominik ...

Molecular Mechanisms of Cardiac Remodeling and ...
The main objective of Cardiac Remodeling: Molecular Mechanisms is to summarize the major research advances in molecular, biochemical and translational aspects of cardiac remodeling over the last 2 to 3 decades under one cover and touch on future directions. It provides a high profile and valuable publication resource on molecular mechanisms of cardiac remodeling for both the present and future generations of researchers, teachers, students and trainees.

Cardiac Remodeling - Molecular Mechanisms | Bodh I ...
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?Cardiac Remodeling on Apple Books
Regular physical activity with aerobic and muscle-strengthening training protects against the occurrence and progression of cardiovascular disease and can improve cardiac function in heart failure patients. In the past decade significant advances have been made in identifying mechanisms of cardiomyocyte re-programming and renewal including an enhanced exercise-induced proliferational capacity ...

Molecular Mechanisms of Cardiac Remodeling and ...
Cardiac remodeling : molecular mechanisms, treatment, and clinical implications / Published: (2016) Cardiac fibrillation-defibrillation clinical and engineering aspects / by: Valentinuzzi, Max E. Published: (2011) Ventricular fibrillation and acute ...

Cardiac remodeling molecular mechanisms
Molecular mechanisms of myocardial remodeling. Swynghedauw B (1). Author information: (1)Institut National de la Sante et de la Recherche Medicale U. 127, Hopital Lariboisiere, Paris, France. "Remodeling" implies changes that result in rearrangement of normally existing structures. This review focuses only on permanent modifications in relation to clinical dysfunction in cardiac remodeling (CR) secondary to myocardial infarction (MI) and/or arterial hypertension and includes a special ...

Molecular mechanisms of myocardial remodeling.
Molecular Mechanisms of Remodeling After Myocardial Injury and Infarction ; Subcellular Remodeling and Cardiac Dysfunction Due to Ischemia-Reperfusion Injury / Naranjan S. Dhalla, Vijayan Elimban, Larry Hryshko, Darren H. Freed ; Role of MicroRNAs in Cardiac Hypertrophy and Postinfarction Remodeling / Jian Ding, Da-Zhi Wang

Cardiac remodeling molecular mechanisms
Due to the reparative nature of many forms of cardiac fibrosis, targeting fibrotic remodeling following myocardial injury poses major challenges. Development of effective therapies will require careful dissection of the cell biological mechanisms, study of the functional consequences of fibrotic changes on the myocardium, and identification of ...

Cardiac fibrosis: Cell biological mechanisms, molecular ...
Cardiac remodeling may be defined as genome expression, molecular, cellular and interstitial changes that are manifested clinically as changes in size, shape and function of the heart after cardiac injury. The process of cardiac remodeling is influenced by hemodynamic load, neurohormonal activation and other factors still under investigation.

Cardiac remodeling—concepts and clinical implications: a ...
Cardiac Embryology and Molecular Mechanisms of Congenital Heart Disease: A Primer for Anesthesiologists ... (situated right superiorly and left inferiorly) that grow and connect in a spiral-like fashion. During this process, remodeling of the distal outflow tract cushion tissue (truncal cushions) results in the formation of the semilunar valves ...

Cardiac Embryology and Molecular Mechanisms of Congenital ...
Pathological molecular mechanisms involved in myocardial remodeling contribute to alter the existing structure of the heart, leading to cardiac dysfunction. Among the complex signaling network that characterizes myocardial remodeling, the distinct processes are myocyte loss, cardiac hypertrophy, alteration of extracellular matrix homeostasis, fibrosis, defective autophagy, metabolic abnormalities, and mitochondrial dysfunction.

A Review of the Molecular Mechanisms Underlying the ...
Several molecular pathways converge in cardiac remodeling. For example, it has been demonstrated that after a cardiac injury, inflammation is sustained through the upregulation of cytokine release, leading to fibroblast proliferation and metalloproteinases activation [3.

A Review of the Molecular Mechanisms Underlying the ...
The cardiac myocyte is the major cell involved in remodeling. Fibroblasts, collagen, the interstitium, and the coronary vessels to a lesser extent, also play a role. A common scenario for remodeling is after myocardial infarction. There is myocardial necrosis (cell death) and disproportionate thinning of the heart. This thin, weakened area is unable to withstand the pressure and volume load on ...

Ventricular remodeling - Wikipedia
At the molecular level, pathological cardiac remodeling is associated with aberrant up-regulation of a set of fetal genes in the myocardium, such as atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP), ?-skeletal actin and the ? isoform of myosin heavy chain (MHC), with concomitant down-regulation of genes associated with normal myocyte contractile functions, such as ?-MHC and sarcoplasmic reticulum Ca 2 + -ATPase 2a.

Heart Ventricle Remodeling - an overview | ScienceDirect ...
The main objective of Cardiac Remodeling: Molecular Mechanisms is to summarize the major research advances in molecular, biochemical and translational aspects of cardiac remodeling over the last 2 to 3 decades under one cover and touch on future directions. It provides a high profile and valuable publication resource on molecular mechanisms of cardiac remodeling for both the present and future generations of researchers, teachers, students and trainees.

Cardiac Remodeling | SpringerLink
Cardiac Remodeling : Molecular Mechanisms, Hardcover by Jugdutt, Bodh I. (EDT); Dhalla, Naranjan S. (EDT), ISBN 146145929X, ISBN-13 9781461459293, Brand New, Free shipping in the US This book examines the major research advances in molecular, biochemical and translational aspects of cardiac remodeling over the last decades.

Advances in Biochemistry in Health and Disease Ser ...
Rationale: Cardiac fibrosis is observed in nearly every form of myocardial disease. Long non-coding RNAs (lncRNAs) have been shown to play an important role in cardiac fibrosis, but the detailed molecular mechanism remains unknown.Object: We aimed at characterizing lncRNA 554 expression in murine cardiac fibroblasts (CFs) after myocardial infarction (MI) to identify CF-enriched lncRNA and ...

Frontiers | Long Non-Coding RNA 554 Promotes Cardiac ...
Nevertheless, the molecular mechanisms by which exercise improves cardiovascular health and prevents tissue injury remain unclear. The recurrent deviations in whole body homeostasis caused by exercise drive adaptations in several organs, including brain, liver, adipose tissue, skeletal muscle, and, the topic of this review—the heart (6, 19).

Metabolic Mechanisms of Exercise-Induced Cardiac Remodeling
Mechanisms of ischemia/reperfusion tissue injury and post injury responses: myocardial stunning, infarction, hibernation, early post-ischemic cardiac remodeling, cellular and molecular mechanisms that

govern the biology of stem cells in ischemic heart disease.

The main objective of Cardiac Remodeling: Molecular Mechanisms is to summarize the major research advances in molecular, biochemical and translational aspects of cardiac remodeling over the last 2 to 3 decades under one cover and touch on future directions. It provides a high profile and valuable publication resource on molecular mechanisms of cardiac remodeling for both the present and future generations of researchers, teachers, students and trainees. This book should stimulate future translational research targeted towards discovery and development for preventing, limiting and reversing bad remodeling over the next few decades, with the ultimate goal of preventing progression to systolic and/or diastolic heart failure. The chapters suggest potential novel strategies that should receive attention for translating basic research knowledge to application in patients at the bedside.

Cardiovascular diseases are the leading cause of death in almost 40% of patients suffering from end stage renal disease (ESRD). Cardiomyopathy and ischemic heart disease are the most frequent causes of cardiac death. The risk of cardiovascular mortality in dialysis patients is 10 to 20 times greater than the general population, particularly in younger patients, taking into account that the relative risk decreases with age. Left ventricular hypertrophy (LVH) is the most common cardiac abnormality in chronic kidney disease (CKD), and the survival risk ratio in such patients is independent. This book examines the molecular mechanisms, treatments and clinical implication of cardiac remodeling. The first chapter discusses risk factors for cardiovascular disease in patients on continuous ambulatory peritoneal dialysis. The following chapters examine the impacts tropomyosin, vitamin D, and coffee have on cardiac remodeling.

This book summarizes present knowledge of different mechanisms involved in the development of positive and negative consequences of cardiac adaptation. Particular attention is paid to the still underestimated adaptive cardiac responses during development, to adaptation to the frequently occurring pressure and volume overload as well as to cardiac changes, induced by enduring exercise and chronic hypoxia. Cardiac Adaptations will be of great value to cardiovascular investigators, who will find this book highly useful in their cardiovascular studies for finding solutions in diverse pathological conditions; it will also appeal to students, fellows, scientists, and clinicians interested in cardiovascular abnormalities.

Translational Cardiology: Molecular Basis of Cardiac Metabolism, Cardiac Remodeling, Translational Therapies and Imaging Techniques provides an up-to-date introduction to the role circadian rhythms, cardiac plasticity, and mechanotransduction play in the heart, while at the same time introducing new developments in cellular, viral, and non-biologic therapies that are in the process of being developed. Importantly, the focus of this book is on topics that, due to their novelty, are largely not covered in the other major textbooks. A special emphasis is placed on the molecular basis of cardiac metabolism, new concepts in cardiac remodeling, and translational therapies and imaging techniques currently under development for clinical use. The chapters are written by experts from diverse clinical and biomedical research backgrounds. Translational Cardiology: Molecular Basis of Cardiac Metabolism, Cardiac Remodeling, Translational Therapies and Imaging Techniques simplifies the complexity of the molecular basis of disease by focusing on patient-oriented disease mechanisms and therapies and is of great value to a broad audience including physicians (e.g. cardiologists, cardiovascular surgeons, pathologists) as well as translational biomedical researchers in a wide range of disciplines.

Cardiac remodeling is composed of molecular, cellular, and interstitial changes in the cardiac tissue that affect the size, shape, and function of the heart. There are two types of cardiac remodeling: physiological and pathological remodeling. Physiological remodeling of the heart is an adaptation of the organ based on the body's demand, such as changes due to physical exercises and during aging. Cardiac pathological remodeling can occur due to the evolution of a chronic disorder in the cardiovascular system or after an acute injury, such as myocardial infarction (MI). In this thesis, we investigated both physiological and pathological cardiac remodeling, with particular focus on the role of the extracellular matrix (ECM). Determining the mechanisms involved in cardiac remodeling by changes in ECM provides insight to distinguish the local and functional changes from external risk factors; and it provides identification of novel targets that could be used as therapeutic approaches to reduce cardiac dysfunction. In our first study, we hypothesized that changes in ECM composition during physiological remodeling with age occurs in a sex-specific manner, since cardiac function varies between sexes among cardiovascular disease patients. We assessed cardiac parameters using both conventional echocardiography and speckle tracking echocardiography (STE). Our results suggest that STE allows for early detection of changes in cardiac function between sexes during aging. ECM factors involved in collagen metabolism, such as decorin, osteopontin, Cthrc1, and Ddr1 expression were age-dependent but sex-independent; while periostin, lysyl oxidase, and Mrc2 displayed age-dependent and sex specific differences. These data highlight the importance of including sex-differences analysis when studying cardiac aging. In our second study, we investigated the role of a collagen-derived matricryptin in pathological remodeling. Matricryptins are biologically active peptides, generated from ECM proteolysis, able to regulate cell function and survival. We tested the potential of the matricryptin p1159 to reduce adverse cardiac remodeling using a rodent MI model. A previous study from our lab showed that p1159 plasma levels negatively correlate with left ventricle (LV) filling pressure, suggesting a beneficial role against adverse remodeling. In this thesis, we found that p1159 increases cardiac fibroblast migration by activating RhoA pathways via the membrane receptor integrin alpha 4. Fibroblast migration is an essential step during cardiac healing. In addition, p1159 significantly improved cardiac function post-MI by inducing the formation of a compliant and organized infarct scar, which promoted LV contractility and preserved the structural integrity of the heart. Our data strongly supports matricryptin p1159 as a therapeutic treatment to reduce adverse remodeling post-MI.

Molecular Defects in Cardiovascular Disease provides an in-depth discussion of the molecular mechanisms underlying the genesis of cardiovascular defects and the implications this has on current and emerging targeted therapeutics. Divided into three sections, this book covers the scientific foundations of our present understanding as well as the array of clinical manifestations and their treatment. The first section covers Molecular Mechanisms of Heart Disease, with discussion of the development of cardiovascular dysfunction. The remaining two sections provide a more clinical focus. The second, Cardiac Hypertrophy and Heart Failure deals with metabolic derangements, Ca2+ handling, and subcellular remodeling. It illustrates the wide variety of molecular defects which may serve as targets associated with the transition from cardiac hypertrophy to advanced heart failure. The third section, Hypertension and Diabetes, provides molecular rationale for the pathogenesis of hypertension and diabetic

cardiomyopathy, as well as highlighting the importance of hormones toward this end. A necessary resource for clinicians and researchers, this book elucidates the experimental basis of the practice of cardiology. It is the culmination of our advances in the understanding of cardiovascular molecular biology and a blueprint for the efficacious use of targeted therapies.

Exploring the causes, mechanisms, and pathophysiology of cardiac remodeling, this reference offers detailed descriptions of the various components of the remodeling process, as well as new therapeutic interventions and recent and future prospects for the treatment of cardiac remodeling.

Cellular and Molecular Pathobiology of Cardiovascular Disease focuses on the pathophysiology of common cardiovascular disease in the context of its underlying mechanisms and molecular biology. This book has been developed from the editors' experiences teaching an advanced cardiovascular pathology course for PhD trainees in the biomedical sciences, and trainees in cardiology, pathology, public health, and veterinary medicine. No other single text-reference combines clinical cardiology and cardiovascular pathology with enough molecular content for graduate students in both biomedical research and clinical departments. The text is complemented and supported by a rich variety of photomicrographs, diagrams of molecular relationships, and tables. It is uniquely useful to a wide audience of graduate students and post-doctoral fellows in areas from pathology to physiology, genetics, pharmacology, and more, as well as medical residents in pathology, laboratory medicine, internal medicine, cardiovascular surgery, and cardiology. Explains how to identify cardiovascular pathologies and compare with normal physiology to aid research Gives concise explanations of key issues and background reading suggestions Covers molecular bases of diseases for better understanding of molecular events that precede or accompany the development of pathology

Ischemic heart disease (IHD) is the major underlying cause of myocardial infarction (MI), scarring, and hypertrophy leading to heart failure which is one of the leading causes of death. Cardiac remodeling following induced pressure overload/myocardial infarction is a multiphase reparative process which involves replacement of damaged tissue with physiological (reparative) fibrosis to form scar that limit the expansion of the left ventricle/infarct of the heart. Although therapeutic approaches targeting soluble factor (ex: ACE inhibitors, ARBs, TGF-[beta] inhibitors: Pirfenidone, Halofuginone) signaling is available for the treatment of cardiac fibrosis and hypertrophy, they showed modest efficacy in clinics. Hence, it is indispensable to identify and develop an alternate and novel therapeutics to treat the heart failure. Mechanical cues are indeed necessary to integrate with soluble factor associated signaling to maintain cardiac physiological functions. Of late, TRPV4 has been shown to be mechanosensor and our lab has established that TRPV4 is a key mechanosensor in endothelial cells and cardiac fibroblasts (CF) and plays an important role in cardiovascular pathophysiology. We have recently demonstrated that TRPV4 mediates cardiac fibroblast differentiation into myofibroblasts in vitro. However, the physiological significance of TRPV4 in cardiac remodeling in vivo is not known. Based on our previous findings, we hypothesized that targeting TRPV4 may offer cardioprotection following pressure overload-induced hypertrophy and myocardial infarction. The first aim of the dissertation was to determine whether TRPV4 mediated mechanotransduction preserves the heart integrity and reduce fibrosis in vivo following pressure overload-induced hypertrophy. By inducing pressure overload hypertrophy (TAC), we found that TRPV4 knockout (KO) mice exhibited improved cardia function, decreased myocardial cross sectional area and left ventricular mass when compared with WT. Further, we have also revealed that TRPV4 KO mice hearts showed less cardiac fibrosis compared to WT. To unravel the unexplored integration of soluble and mechanical signaling behind the cardiac fibrosis, the second aim of this dissertation was to delineate the molecular mechanisms by which TRPV4 regulate cardiac fibroblasts differentiation into myofibroblasts. Our in vitro studies revealed that TGF-[beta]1 mediated fibroblasts differentiation was attenuated in TRPV4KO mCF compared WT mCF. Further, both TGF-[beta]1 and a specific activator of TRPV4, GSK1016790A, significantly enhanced pro-fibrotic a-SMA and Col1a1 promoter activities. Importantly, we have dissected the mechanism and found that both TGF-[beta]1 and GSK (TRPV4 agonist) induced TRPV4-dependent activation of the Rho/Rho Kinase pathway as well as a mechanosensitive transcription factor MRTF-A are involved in CF differentiation. To further corroborate the critical role of TRPV4 in cardiac remodeling, the third aim of this dissertation was to ascertain the functional role of TRPV4 in cardio protection following myocardial infarction. We found that after 8 weeks post- MI, significant improvement of cardiac function was observed in TRPV4KO mice compared to WT. Further, we found reduced cardiac fibrosis at infarct and remote zones in TRPV4KO-MI mice compared to WT-MI mice which display enhanced fibrosis at infarct/border zone as well as remote zones. Furthermore, TRPV4KO hearts exhibited decreased cardiomyocyte apoptosis (TUNEL assay) and increased capillary density (CD31 staining) post-MI compared to WT hearts. In conclusion, our results suggest that targeting a mechanosensor TRPV4, protects heart from induced pressure overload or myocardial infarction-induced damage by preserving cardiac structure, function and identifies TRPV4 as a novel therapeutic target for heart failure.