EMBO Molecular Medicine
Review Series
Cardiovascular Diseases

Cardiovascular disease review series

Cardiovascular diseases are still the leading cause of death worldwide. Impaired endothelial function followed by inflammation of the vessel wall leads to atherosclerotic lesion formation that causes myocardial infarction and stroke. The increase of major risk factors such as obesity and diabetes in industrialized but also developing countries further increases the burden of vascular disease. Heart failure can occur as consequence of large myocardial infarctions or can be caused by genetic predisposition or infectious disease. In its more severe stages, heart failure patients have a life expectancy that is similar to aggressive cancers. Due to the rising age of the population, the incidence of heart failure is further increasing. Thus, the increase in risk factor load by metabolic diseases and age augments the incidence for vascular and cardiac diseases and provides a challenge for developing efficient treatments.

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Targeting Chemokines: Despite improved pharmacological and interventional therapies, a causative treatment of atherosclerosis is missing. It is well known that vascular inflammation plays a key role in the pathogenesis of atherosclerosis and modulating this inflammatory reaction may interfere with this chronic disease. Chemokines comprise one group of the regulators of inflammation by directing the movement and activity of leukocytes. The review article by Christian Weber and Rory Koenen (University Munich) will summarize the pathophysiological role and the challenges in targeting chemokines for therapy of atherosclerosis.

Targeting HDL: The pharmacological lowering of low-density lipoproteins, the 'bad' cholesterol, for example by HMG-CoA reductase inhibitors ('statins'), enabled the successful treatment of hypercholesterolemia and significantly reduced coronary artery disease. However, despite ample evidence for a protective function of high-density lipoprotein (HDL), attempts to increase this protective lipoprotein failed to protect against atherosclerotic disease in patients. Currently, it is unclear why strategies to elevate HDL failed. One hypothesis is that HDL is modified in patients with coronary artery disease, thereby, losing its protective function. Ulf Landmesser, Christian Besler and Thomas Lüscher (University Zürich) will discuss the current state and future of using HDL as a therapeutic protective target for the treatment of coronary artery disease.

Targeting MicroRNA: MicroRNAs are small non-coding RNAs that post-transcriptionally control gene expression by inducing RNA degradation or inhibiting protein translation. MicroRNAs play a crucial role in tissue homeostasis and disease and gained increasing interest as biomarkers for various diseases. Several studies meanwhile also provide evidence that microRNAs are therapeutic targets and that inhibition of microRNAs can ameliorate cardiovascular diseases in experimental models. Thomas Thum, Hannover, will discuss the role of microRNAs in cardiovascular diseases.

Targeting Regeneration: One of the most promising options for treating heart failure is to generate cardiomyocytes. Stem cell-based approaches may help to maintain organ function by replacing damaged or lost cells. Some tissues including the heart harbour stem/progenitor cells that to some extent may contribute to regeneration. However, in disease conditions, the endogenous repair capacity of the heart is not sufficient and either transplantation of cells or further activation of endogenous cells might provide a therapeutic option. The possibility to stimulate the replacement of cardiomyocytes by activating endogenous repair (including endogenous stem cells) and by reprogramming cells within the heart will be discussed by Richard Lee and Matthew Steinhauser (Harvard).

The author declares that she has no conflict of interest.

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DOI 10.1002/emmm.201100182

