



Editorial

Atherosclerosis: From Molecular Biology to Therapeutic Perspective 3.0

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Atherosclerosis is a multifactorial chronic disease triggered and sustained by different risk factors such as dyslipidemia, hypertension, diabetes mellitus (DM), smoke, elevated homocysteine, and hormones [1]. Atherosclerosis develops over many years with a long asymptomatic phase and progresses through a complex interplay between vascular cells, infiltrating inflammatory cells, growth factors/cytokines, oxidative stress, and lipids. In the early stage of lesion formation, low-density lipoprotein (LDL) particles accumulate in the intimal layer of the artery where they undergo oxidative modifications, thus acquiring proinflammatory and immune-stimulatory properties. With time, histologic changes have become increasingly complex, and these initial lesions (called fatty streaks) are evolving toward fibroatheromas which are composed of a lipid-rich atheromatous core covered by a fibrous cap with a loosely arranged matrix dominated by collagen fibers, smooth muscle cells (SMCs), and inflammatory cells. Progression to major acute cardiovascular events (i.e., acute myocardial infarction, sudden cardiac death, and stroke) is often triggered by an atherosclerotic plaque complicated by calcification, endothelial erosion, intraplaque hemorrhage, and, finally, rupture. In recent years, the rapid development of a vast array of techniques in the field of cellular and molecular biology has led to a more in-depth understanding of the molecular history of atherosclerosis and the cellular phenotypes that occur during vascular remodeling. These include biological molecules, biochemical pathways, and processes that, together with the migration, proliferation, and apoptosis of vascular cells, are vital to the pathogenesis of atherosclerosis [2–4].

This Special Issue entitled “Atherosclerosis: From Molecular Biology to Therapeutic Perspective 3.0” contains a combination of original articles and reviews written by an international panel of researchers and reviewed by several other experts in the field of cardiovascular sciences, focused on the molecular mechanisms related to the pathogenesis of the disease, the methods that can detect and characterize the lesions, and the potential novel therapeutic strategies for targeting atherosclerotic plaques.

SMCs, as the major cell component of the vessel wall, play an integral role in vasoconstriction and blood pressure regulation and maintain vascular structural integrity. SMCs can also participate in vascular remodeling in both physiological or pathological states by virtue of their capacity to change their phenotype in response to changes in local environmental cues. Under healthy conditions, they exist in a contractile, well-differentiated phenotype. In cases of disease or injury, SMCs undergo a profound transformation to a less differentiated state, acquiring a proliferative potential and migratory properties. A wide range of regulatory factors and signaling pathways can promote this phenotypic transition [5,6]. Miyabe and colleagues demonstrated that treatment of human aortic SMCs with porphyromonas-gingivalis-derived LPS leads to the phosphorylation of p38 MAPK and SAPK/JNK via TLR4/MyD88 and produces an increase in cell proliferation, while the TLR4-mediated activation of ERK in a MyD88-independent pathway culminates in the enhancement of both proliferation and migration. These results provide new insights to explain the potential role for periodontal pathogens in the induction of atherosclerosis.



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Atherosclerosis A Pediatric Perspective

David Baud



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