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ISSN: 0974-8369
Biology and Medicine

Opinion Article

Molecular Mechanism of Atherosclerosis Disease

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DESCRIPTION

Atherosclerosis is a chronic disease of large arteries due to accumulation of oxidized lipid on the vascular wall, fibrous cap formation and migration of inflammatory cells onto the injured endothelium. The molecular mechanism of atherosclerosis begins when injured endothelial cells start to increase the expression of adhesion molecules such as E-selectin, vascular cell adhesion molecule-1, and intercellular adhesion molecule-1. Which assist the progress of leukocyte adhesion to the endothelium and migration to the sub-endothelial area, ultimately producing atherosclerosis in the advanced stage. Atherosclerosis is a leading cause of vascular disease worldwide. During the past several decades, landmark discoveries in the field of vascular biology have evolved our understanding of the biology of blood vessels and the pathobiology of local and systemic vascular disease states and have led to novel disease-modifying therapies for patients. The molecular mechanism of atherosclerosis for these future therapies advances in molecular biology and technologies have facilitated *in vitro* and *in vivo* studies which revealed that blood vessels regulate their own metabolism, mechanical environment, and phenotype, through complex interactions between cellular components of the blood vessel wall and circulating factors. Dysregulation of these homeostatic interactions has also been implicated as the mechanism by which risk factors for cardiopulmonary vascular disease lead to vascular dysfunction, structural re modeling and, ultimately, adverse clinical events. Atherosclerosis is a chronic inflammatory disease of the inner wall of the aorta and middle arteries. It is a multifactorial disease believed to be the leading cause of heart disease and cerebrovascular disease.

Pathologically, atherosclerosis is a chronic arterial inflammation secondary to prolonged exposure to oxidative stressors and involves multiple cell types and cellular mediators. Oxidized lipids derived from low-density lipoprotein contribute to multiple stages of atherosclerotic plaque development and progression through production of inflammatory cytokines. Diet and dietary habits are the major driving forces for development and modification of atherosclerotic diseases. Genetics and epigenetics have a significant influence on development and progression of atherosclerosis. Future therapeutic options may target the pathogenic mediators of atherosclerosis at multiple molecular levels. Atherosclerosis is a complex disease caused by multiple genetic and environmental factors and complex gene-environment interactions. Coronary artery disease is the most common cause of death in the Western hemisphere and by the year 2020 is expected to become the leading cause of morbidity and mortality in the world. The molecular mechanisms of atherosclerosis are a complex web of cellular events that is only gradually becoming explained. These mechanisms involve lipid metabolism, inflammatory signaling, and interaction with the complex vascular system involved in thrombosis. Risk factors involved in these areas (e.g: dyslipidemia and diabetes, procoagulant, and anticoagulant factors) have provided information about important genes that seem to play an important role in establishing the risk of atherosclerosis. Although common, atherosclerosis is clearly polygenic; critical molecular information concerning the process has been gleaned from rare monogenic forms of atherosclerosis and thrombosis.

Biochemical analysis of High Density Lipoprotein (HDL) or apolipoprotein A-I indicates the major protein component of HDL particles, is readily oxidized *in vivo*. Oxidation is mediated by various radicals or enzymes. Multiple oxidation pathways have been demonstrated and target amino acids have been identified. The high susceptibility of high density lipoprotein to oxidation and its association with loss of anti atherogenic capacity has attracted several groups as a mechanism of atherosclerosis. Oxidized high density lipoprotein has been developed and applied to the measurement of clinical samples. Unexpectedly, some of the results are paradoxical. Patients with suspected increased oxidative stress had decreased oxidized HDL. Oxidized HDL is cleared faster than native HDL, so oxidized HDL is rapidly cleared from circulation. Furthermore, the reduction in oxidized HDL levels in patients suggests that pathways to clearance or elimination of oxidized HDL are accelerated in some disease states. Therefore, quantitative measurement of oxidized HDL may not be useful as a clinical marker.

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Received: 27-Jul-2022, Manuscript No. BLM-22-18115; **Editor assigned:** 29-Jul-2022, Pre QC No. BLM-22-18115 (PQ); **Reviewed:** 15-Aug-2022, QC No. BLM-22-18115; **Revised:** 22-Aug-2022, Manuscript No. BLM-22-18115 (R); **Published:** 29-Aug-2022, DOI: 10.35248/0974-8369.22.14.504.

Citation: Sabry S (2022) Molecular Mechanism of Atherosclerosis Disease. Bio Med. 14:504.

Inflammation is part of the mechanism of atherosclerosis, low density lipoproteins are taken up by monocytes. The monocytes bind to endothelial cells of the coronary artery and then monocytes differentiate into macrophages, which accumulate in the artery, resulting in the "fatty streak," which later becomes an atherosclerotic lesion.