



The Role of Lipid and Glucose Metabolism on Atherosclerosis

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Abstract

Diabetes mellitus comprises a group of carbohydrate metabolism disorders that share a common main feature of chronic hyperglycemia that results from defects of insulin secretion, insulin action, or both. Insulin is an important anabolic hormone, and its deficiency leads to various metabolic abnormalities in proteins, lipids, and carbohydrates. Atherosclerosis develops as a result of a multistep process ultimately leading to cardiovascular disease associated with high morbidity and mortality. Alteration of lipid metabolism is a risk factor and characteristic feature of atherosclerosis. Possible links between the two chronic disorders depending on altered metabolic pathways have been investigated in numerous studies. It was shown that both types of diabetes mellitus can actually induce atherosclerosis development or further accelerate its progression.

Keywords: Diabetes mellitus; Insulin; Lipid metabolism

Description

Atherosclerosis is a widespread chronic inflammatory disorder of the arterial wall that often leads to disability and even death. At its final stages, atherosclerosis manifests itself as a lesion of the intimal layer of the arterial wall and accumulation of plaques. Subsequent erosion or rupture of atherosclerotic plaques triggers thrombotic events that can potentially be fatal. Decades of intensive research made it clear that atherosclerosis has complex pathogenesis, the main components of which are lipid accumulation and chronic inflammation in the arterial wall [1]. Atherosclerosis is classically associated with altered lipid metabolism and hypercholesterolemia [2]. An elevated level of circulating modified low-density lipoprotein (LDL) is a known risk factor of cardiovascular diseases [3]. However, the disease pathogenesis appears to be more complex than lipid metabolism changes and involves multiple factors, the most prominent of which is inflammation [4]. The chain of pathological events that leads to atherosclerosis development is believed to be initiated by local endothelial dysfunction, which may be caused by blood flow turbulence near the sites of artery bends or bifurcations [5].

Detailed study of atherosclerotic lesion development is complicated by the fact that the process may differ considerably in humans and available model animals [6,7]. However, the main outlines of the process could be established. The early stage of atherosclerotic lesion development is known as “fatty streak”, an area in the vascular wall that is characterized by intracellular lipid accumulation by foam cells, which also contains vascular smooth muscle cells (VSMCs) and T lymphocytes. Fatty streaks can further progress to atherosclerotic lesions if chronic injury of the endothelium persists.

Diabetes mellitus and atherosclerosis appear to be connected through several pathological pathways. Increased risk and accelerated development of atherosclerosis have been shown in studies on diabetic patients.

Conclusion

Both types of diabetes mellitus have been shown to be independent risk factors for accelerated atherosclerosis development. It is now clear that the pathogenesis of diabetes mellitus and atherosclerosis are closely linked, but the mechanisms and molecular interactions of this linkage are still under discussion.

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Received December 03, 2020; Accepted December 24, 2020; Published December 31, 2020

Citation: Siraj A (2020) The Role of Lipid and Glucose Metabolism on Atherosclerosis. *Atheroscler Open Access* 5: e145.

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