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Cigarette Smoking and Serum HDL Levels for the Eye Patients.

Yazar Hayrullah[1]*, Yarbağ Abdülhekim[2], Akdoğan Mehmet[1], and Kaleli Süleyman[3].

[1]Sakarya University Faculty of Medicine, Department of Medical Biochemistry, Sakarya Üniversitesi Tıp, Sakarya, Turkey.
[2]Special Marmara Eye Clinic Center, Sakarya, Turkey.
[3]Sakarya University Faculty of Medicine, Department of Medical Biology, Sakarya, Turkey.

ABSTRACT

HDL particles increase in size as they circulate through the bloodstream and incorporate more cholesterol and phospholipid molecules from cells and other lipoproteins, for example by the interaction with the ABCG1 transporter and the phospholipid transport protein (PLTP). Higher native HDL levels are correlated with better cardiovascular health however, it does not appear that further increasing one's HDL improves cardiovascular outcomes. The effect of cigarette smoking on HDL-C level was analysed in many studies, with aim to determine whether smoking causes lowering its level. WHO (The world health organization) acording to; tobacco products are products made entirely or partly of leaf tobacco as raw material, which are intended to be smoked, sucked, chewed or snuffed. All contain the highly addictive psychoactive ingredient, nicotine. Despite this, it is common throughout the world. A number of countries have legislation restricting tobacco advertising, and regulating who can buy and use tobacco products, and where people can smoke. The literature review confirmed a strong association between current smoking and AMD (Age-related macular degeneration), which fulfilled established causality criteria. Cigarette smoking is likely to have toxic effects on the retina. In spite of the strength of this evidence, there appears to be a lack of awareness about the risks of developing eye disease from smoking. Studies of exposure to smoking lowers HDL revealed that the cholesterol level. Another fact about smoking, however, is the increase in the incidence of macular degeneration. The aim of this review article; HDL cholesterol level and macular degeneration, and additionally draw attention to a possible relationship between the researcher to contribute to science by offering a new perspective to provide.

Keywords: HDL cholesterol, macular degeneration

Corresponding author

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INTRODUCTİON

High-density lipoprotein (HDL) is one of the five major groups of lipoproteins, which, in order of molecular size, largest to smallest, are chylomicrons, very low-density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), low-density lipoprotein (LDL), and HDL. Lipoprotein molecules enable the transportation of lipids (fats), such as cholesterol, phospholipids, and triglycerides, within the water around cells (extracellular fluid), including the bloodstream. Because of the high cost of directly measuring HDL and LDL protein particles, blood tests are commonly performed for the surrogate value, HDL-C, i.e. the cholesterol associated with ApoA-1/HDL particles. In healthy individuals, about 30% of blood cholesterol, along with other fats, is carried by HDL [1]. This is often contrasted with the amount of cholesterol estimated to be carried within low-density lipoprotein particles, LDL, and called LDL-C. HDL particles remove fats and cholesterol, from cells, including within artery wall atheroma and transport it back to the liver for excretion or re-utilization, the reason why the cholesterol carried within HDL particles (HDL-C) is sometimes called "good cholesterol" (despite the fact that it is exactly the same as the cholesterol in LDL particles). Those with higher levels of HDL-C tend to have fewer problems with cardiovascular diseases, while those with low HDL-C cholesterol levels (especially less than 40 mg/dL or about 1 mmol/L) have increased rates for heart disease [2]. Higher native HDL levels are correlated with better cardiovascular health [3], However, it does not appear that further increasing one's HDL improves cardiovascular outcomes [4-7].

HDL is the smallest of the lipoprotein particles. It is the densest because it contains the highest proportion of protein to lipids. Its most abundant apolipoproteins are apo A-I and apo A-II [5]. The liver synthesizes these lipoproteins as complexes of apolipoproteins and phospholipid, which resemble cholesterol-free flattened spherical lipoprotein particles; the complexes are capable of picking up cholesterol, carried internally, from cells by interaction with the ATP-binding cassette transporter A1 (ABCA1). A plasma enzyme called lecithin-cholesterol acyltransferase (LCAT) converts the free cholesterol into cholesteryl ester (a more hydrophobic form of cholesterol), which is then sequestered into the core of the lipoprotein particle, eventually causing the newly synthesized HDL to assume a spherical shape. HDL particles increase in size as they circulate through the bloodstream and incorporate more cholesterol and phospholipid molecules from cells and other lipoproteins, for example by the interaction with the ABCG1 transporter and the phospholipid transport protein (PLTP). HDL transports cholesterol mostly to the liver or steroidogenic organs such as adrenals, ovary, and testes by both direct and indirect pathways. HDL is removed by HDL receptors such as scavenger receptor BI (SR-BI), which mediate the selective uptake of cholesterol from HDL. In humans, probably the most relevant pathway is the indirect one, which is mediated by cholesteryl ester transfer protein (CETP). This protein exchanges triglycerides of VLDL against cholesteryl esters of HDL. As the result, VLDLs are processed to LDL, which are removed from the circulation by the LDL receptor pathway. The triglycerides are not stable in HDL, but are degraded by hepatic lipase so that, finally, small HDL particles are left, which restart the uptake of cholesterol from cells. The cholesterol delivered to the liver is excreted into the bile and, hence, intestine either directly or indirectly after conversion into bile acids. Delivery of HDL cholesterol to adrenals, ovaries, and testes is important for the synthesis of steroid hormones.

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Level mg/dL	Level mmol/L	Interpretation
<40 for men, <50 for women	<1.03	Low HDL cholesterol, heightened risk for heart disease
40–59	1.03–1.55	Medium HDL level
>60	>1.55	High HDL level, optimal condition considered protective against heart disease

Table 1: HDL Cholesterol

Age-related macular degeneration (AMD) is the leading cause of severe and irreversible vision loss. As there is no effective treatment for all types of AMD, identifying modifiable risk factors is of great importance. The most of studies found a statistically significant association between smoking and AMD with increased risk of AMD of two- to three-fold in current-smokers compared with never-smokers. Macular degeneration gene: The genes for the complement system proteins factor H (CFH), factor B (CFB) and factor 3 (C3) are strongly associated with a person's risk for developing AMD. CFH is involved in inhibiting the inflammatory response mediated via C3b (and the alternative pathway of complement) both by acting as a cofactor for cleavage of C3b to its inactive form, C3bi, and by weakening the active complex that forms between C3b and factor B. C-reactive protein and polyanionic surface markers such as glycosaminoglycans normally enhance the ability of factor H to inhibit complement. But the mutation in CFH (Tyr402His) reduces the affinity of CFH for CRP and probably also alters the ability of factor H to recognise specific glycosaminoglycans. This change results in reduced ability of CFH to regulate complement on critical surfaces such as the specialised membrane at the back of the eye and leads to increased inflammatory response within the macula. In two 2006 studies, another gene that has implications for the disease, called HTRA1 (encoding a secreted serine protease), was identified [8,9].

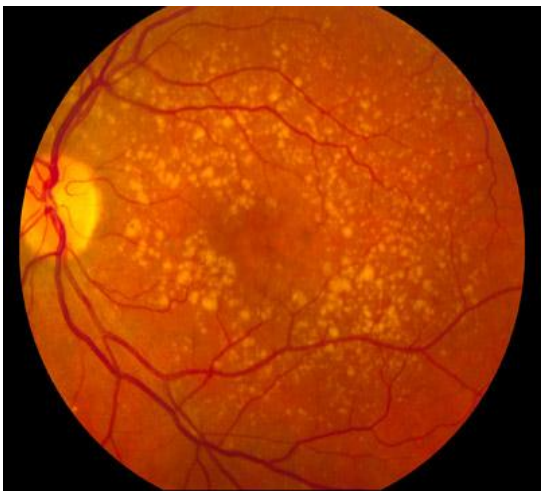

Figure 1: Macular degeneration

The mitochondrial genome (mtDNA) in humans is contained on a single circular chromosome, 16,569 basepairs around, and each mitochondrion contains five to 10 copies of the mitochondrial chromosome. Several essential genes in mtDNA are involved in replication and translation, along with some genes that are crucial for the machinery that converts metabolic energy into ATP. These include NADH dehydrogenase, cytochrome C oxidase, ubiquinol/cytochrome C oxidoreductase, and ATP synthase, as well as the genes for unique ribosomal RNA and transfer RNA particles that are required for translating these genes into proteins. Specific diseases are associated with mutations in some of these genes.
Fat intake Consuming high amounts of certain fats likely contributes to AMD, while monounsaturated fats are potentially protective [10]. In particular, ω-3 fatty acids may decrease the risk of AMD.

CONCLUSİON

The literature review confirmed a strong association between current smoking and AMD, which fulfilled established causality criteria. The other conclusion, cigarette smoking is likely to have reduced effects on HDL cholesterol (yazar H at al). In our opinion; combining these two pieces of data from a scientific perspective and the new work would have been done.

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