The Role of Nutrition and Physical Activity in Cholesterol and Aging

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KEYWORDS

• Aging • Cholesterol • Lipoproteins • Atherogenesis • Alzheimer disease • Diet • Physical activity • Physical exercise

KEY POINTS

- Cholesterol is fundamental to biologic processes, and at the same time, alterations in cholesterol are associated with several diseases.
- During aging, diseases associated with cholesterol alterations (eg, atherosclerosis and Alzheimer disease) can assume special importance.
- Because of the variability in the aging process among individuals, pharmacologic interventions to reduce cholesterol can present negative outcomes.
- Healthy lifestyle interventions represent the best option for optimizing cholesterol levels, and should include a diet with an appropriate balance of macronutrients, with inclusion of natural foods and reduction in processed foods, and daily practice of at least 30 minutes of physical activity.

INTRODUCTION

Lipids, including cholesterol, are fundamental to health because they have structural and metabolic functions. The different lipids that compose the human body are the phospholipids, triacylglycerol (TAG), and cholesterol. Because of their insolubility in water, most lipids need to be carried by lipoproteins (LPs), which contain specific carriers, the apolipoproteins (APOs).[1,2] A description of recent findings in LPs and APOs is found in **Table 1**.

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Table 1
Description of lipoprotein subclasses, functions, and composition

Name	CM	VLDL	LDL	HDL	IDL	LP(a)
Subclasses[a]	Remnant CM (CM_{rem})	VLDL1 to VLDL3	LDL1 to LDL5	HDL2 and HDL3	—	LP(a)F, LP(a)B, LP(a)S1, LP(a)S2, LP(a)S3, and LP(a)S4[b]
Main functions	Transport of lipids (mainly TAG) from intestine to the tissues (CM) and from the circulation to the liver (CM_{rem})	Transport of lipids from liver to the tissues	This LP is result of VLDL after the action of LPL; it transports mainly cholesterol to tissues	Responsible for the reverse transport of cholesterol	This LP is result of VLDL after the action of LPL, and it is rapidly removed from the blood Studies have shown the atherogenic property of this LP	There is a body of evidence associating this lipoprotein to the progression of atherosclerotic plaque
Composition in proteins and lipids (%), and order of the different lipids	Protein = 1.5%–2.5%; lipids = 97%–99% (1-TAG; 2-PL; 3-CE; 4-FC)	Protein = 5%–10%; lipids = 90%–95% (1-TAG; 2-PL; 3-CE; 4-FC)	Protein = 20%–25%; lipids = 75%–80% (1-CE; 2-PL; 4-TAG; 5-FC)	Protein = 40%–45% (HDL2); 50%–55% (HDL3); lipids = 55% (HDL2); 50% (HDL3) (1-PL; 2-CE; 4-TAG; 5-FC)	Protein = 15%–20%; lipids = 80%–85% (1-TAG; 2-PL + CE; 3-FC)	Protein = 27%–30.9%; lipids = 77.6% (1-CE; 2-TAG; 3-TC; 4-PL)
Description of the main APOs	APOA-IV, APOB-48, APOC-II, APOC-III, APOE	APOB-100, APOC-I, APOC-II, APOC-III, APOE	APOB-100	APOA-I, APOA-II, APOA-IV, APOC-I, APOC-II, APOD, APOE Recent studies have identified proteins in HDL particles, such as MPO, and PON1 that complexes with APOA-I	APOB-100	APOB-100, APOA

Abbreviations: CE, cholesteryl esters; CM, chylomicron; FC, free cholesterol; HDL, high-density lipoprotein; IDL, intermediate-density lipoproteins; LDL, low-density lipoprotein; MPO, myeloperoxidase; PL, phospholipids; PON1, paraoxonase-1; VLDL, very-low-density lipoprotein.

[a] Different methods and techniques have been proposed to identify subclasses of the lipoproteins.

[b] The letters in the subclasses were attributed to the speed of mobility, compared to APOB100. B, similar mobility to APOB100; F, faster than APOB100; S, slower than APOB100.

Data from Refs.[3–5]

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