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Dyslipidemia: evidence of efficacy of the pharmacological and non-pharmacological treatment in the elderly

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Abstract

The clinical decision to control risk factors for cardiovascular disease (CVD) in the elderly takes the followings into consideration: (1) the elderly life expectancy; (2) the elderly biological age and functional capacity; (3) the role of cardiovascular disease in the elderly group; (4) the prevalence of risk factors in the elderly; and (5) The effectiveness of treatment of risk factors in the elderly. A large number of studies showed the efficacy of secondary and primary prevention of dyslipidemia in the elderly. However, the only trial that included patients over 80 years was the Heart Protection Study (HPS). Statins are considered the first line therapy for lowering low-density lipoprotein cholesterol (LDL-C). Because lifestyle changes are very difficult to achieve, doctors in general tend to prescribe many drugs to control cardiovascular risk factors. However, healthy food consumption remains a cornerstone in primary and secondary cardiovascular prevention and should be implemented by everyone.

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1 Introduction

Until recently, a discussion of risk factors and prevention of atherosclerosis would be considered unprecedented, since atherosclerosis was considered a natural aging process. The control of risk factors was supposed to have less or little importance in the elderly and it was questioned if it were really needed in the octogenarians and beyond.

Nevertheless, this concept was gradually changed by the inclusion of the elderly in clinical studies, a small number in the beginning and progressively a larger number. Besides verifying the important distinction between relative risk and absolute risk which had a greater value when applied to the elderly, it was observed that although the relative risk of cardiovascular disease associated with any risk factor decreased with aging, the absolute risk was markedly increased in the elderly.

The term “elderly” accounts for three distinct aging groups: the young elderly (from 65–74 years-old) the old-old

(from 75–79 years old) and octogenarians and beyond. The heterogeneity, usual within any age group, is more prevalent among the octogenarians and beyond group composed of very old, lucid, and independent patients with an active lifestyle, but also stroke patients depending on caregivers, or with Alzheimer’s, and experiencing poor quality of life.

The clinical decision to control risk factors for cardiovascular disease (CVD) in the elderly takes into consideration five topics.

1.1 The elderly life expectancy

The elderly life expectancy in Brazil according to the 2005 national census, and a theoretical projection evaluating the life expectancy of the oldest made in 2008 are listed in Table 1.^(1,2)

According to US NHANES III,⁽³⁾ the treatment of a risk factor, for instance, the dyslipidemia, is not indicated if the life expectancy is inferior to 2 years. This was not the case in any of the age group above.

1.2 The elderly biological age and functional capacity

The elderly biological age and functional capacity will guide the treatment more effectively than only the chronological age. The biological age is affected by the presence of co-morbidities, such as severe pulmonary disease, vascular

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Table 1. Life expectancy of the elderly in Brazil.^[1]

Age (year)	Life Expectancy (year)
65	17.40
66	16.70
67	16.10
68	15.50
69	14.90
70	14.30
71	13.70
72	13.10
73	12.60
74	12.10
75	11.60
76	11.10
77	10.60
78	10.20
79	9.70
80	9.30
Theoretical projection ^[2]	
85	7.64
90	6.16
95	5.02
100	4.14

disease, anemia, renal insufficiency, thyroid disease, diabetes, cognitive dysfunction, pulmonary hypertension, depression, and so on. These co-morbidities will affect the dosage and the choice of treatment. The functional capacity can be affected by stroke, dementia, fragility, or severe osteoporosis and will have a profound impact on the decision of treatment.

1.3 The role of cardiovascular disease in the elderly group

Cardiovascular disease is a major threat to the health of older people. Elderly population accounting for more than 75% of total coronary heart disease (CHD) mortality and well over 50% of all acute myocardial infarctions (MI) in the United States.^[4] Risk reduction in people older than 65 years is essential because two-thirds to three-quarters of them have either clinical CHD or subclinical atherosclerosis.^[5] According to van Oostrom *et al.*,^[6] the morphology of atherosclerotic plaques in patients with carotid artery stenosis changes predisposes them, with increasing age, to more instability with an increased chance of clinical events.

1.4 The prevalence of risk factors in the elderly

According to the “Estudo Multicêntrico em Idosos” (Multicenter study in the elderly or EMI study),^[7] the prevalence of sedentary lifestyle in the Brazilian elderly is

74%, hypertension 53%, dyslipidemia 33%, obesity 30%, diabetes 13% and smoking 6%. Dyslipidemia is prevalent even in those at an advanced age. Despite this high prevalence and the frequent occurrence of CHD, there is a strong bias in the prevention and treatment of cardiovascular disease due to the poor assessment and treatment of lipid disorders through primary and secondary prevention in the elderly.^[4]

1.5 The effectiveness of management of risk factors in the elderly

There is currently overwhelming evidence that the treatment of elderly dyslipidemia is effective in the reduction of CVD in the elderly. For instance, meta-analysis of 26 published interventional studies of the last Cholesterol Treatment Trialists Collaboration,^[8] showed the reduction of about 25% in the occurrence of a first major vascular event due to a 1 mmol/L (38.6 mg/dL) decrease in low-density lipoprotein cholesterol (LDL-C), with benefits experienced in every age range.^[8] In a previous review of the treatment of dyslipidemia in the elderly, Shao *et al.*^[9] pointed out the benefit of treatment of dyslipidemia in the elderly might be evident as early as the first two years of treatment.

2 Clinical evidence of the efficacy of the pharmacological treatment of dyslipidemia in the elderly

2.1 Secondary prevention

The 2010 “II Brazilian Guidelines of Geriatric Cardiology from the Brazilian Society of Cardiology” (Table 2) pointed out studies with expressive numbers of elderly.^[10] They reported the efficacy of secondary and primary prevention of elderly dyslipidemia treatment with statins, considered as first line therapy for lowering LDL-C. There are also studies with fibrates as well, with their major effect on triglycerides and high-density lipoprotein cholesterol (HDL-C). These studies showed the following:

(1) Reductions of relative risk for mortality from all causes and cardiovascular mortality with statins in the elderly were equal or higher than those observed in the younger patients (Scandinavian Simvastatin Survival Study^[11] (4S): 4,444 patients with coronary artery disease with 1021 within the age range 65 to 70 years old).

(2) Higher reductions of coronary death, non-fatal MI angioplasty or surgery in the elderly (with the use of statins) than in the non-elderly: Cholesterol and Recurrent Events (CARE) Study^[12] in older patients (1283 elderly from 65 to 75 years of a total of 4159 post-infarction patients with discrete elevated levels of total cholesterol (TC) and LDL-C).

Table 2. II Brazilian guidelines of geriatric cardiology recommendations.^[10]

LDL-C goal in elderly patient with 1 risk factor: < 130 mg/dL
Level of LDL for change of life style with optional pharmacological treatment: 130–159 mg/dL.
Level of LDL for change of life style and pharmacological treatment \geq 160 mg/dL.
LDL-C goal in high risk elderly: < 100 mg/dL (High risk elderly are identified by the presence of several risk factors)
Level of LDL-C in order to prescribe change of life style and pharmacological treatment: \geq 100 mg/dL.
LDL-C goal in the very high risk elderly < 70 mg/dL
Very high risk factor elderly are identified by the presence of CAD in the presence of one or more risk factors of difficult correction as diabetes or smoking, or with ACS.
Goal of LDL-C requiring change of life style with optional pharmacological treatment 70–99 mg/dL
Goal of LDL-C requiring change of life style and pharmacological treatment: \geq 100 mg/dL.
Recommendations
I: level of evidence A
Diet and physical activity for the treatment of dyslipidemia.
Statin in high LDL for secondary and primary prevention.
Ia: level of evidence C
Physical activity and niacin in low HDL-C isolated.

CAD: coronary artery disease; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol;

(3) Reductions of 22% of deaths for coronary artery disease (CAD) or non-fatal MI and 59% of transitory stroke with gemfibrozil-(Veterans Affairs HDL-C Intervention Trial (VA-HIT)^[13]: 50% of the participants were 65–74 years of age). There is no clinical trial with fibrate in patients over 75 years old.

(4) Reductions of mortality, incidence of first MI and stroke in all age groups, including octogenarians and beyond, with statins in primary and secondary prevention (Heart Protection Study (HPS)^[14]: 20,536 patients from 40–80 years of age with 5806 patients \geq 70 years. It was the first study to show that patients prescribed statins with relatively low levels of cholesterol had similar benefits to cohorts with higher cholesterol levels).

(5) Reductions of mortality and composed end-point (mortality, MI, and stroke) in secondary and primary prevention study in the Prospective Study of Pravastatin in the Elderly at Risk (PROSPER)^[15]: It was performed exclusively in elderly patients: 5804 participants from 70–82 years, with vascular disease (coronary, cerebral, peripheral) or risk factors (hypertension, smoking, diabetes) without established CVD.

(6) Higher reductions of clinical events with higher reductions of LDL-C (Pravastatin or atorvastatin evaluation

and infection therapy-thrombolysin myocardial infarction 22 (PROVE-IT-TIMI 22)^[16]: a study with 4,162 patients with acute coronary syndrome, 30% with age \geq 65 years, was performed in order to evaluate if a reduction of LDL-C levels to 70 mg/dL with atorvastatin 80 mg was most effective in reducing events than the standard level of LDL-C (100 mg/dL) obtained with pravastatin 40 mg.

(7) The reduction of LDL-C to 70 mg/dL in acute coronary syndrome (ACS) had similar beneficial effects in patients \geq 70 years as well as in younger patients, according to the analysis of elderly sub-group (Pravastatin or atorvastatin evaluation and infection therapy-thrombolysin myocardial infarction 22 (PROVE-IT-TIMI 22)^[17]: 3150 patients \leq 70 years vs. 634 patients with \geq 70 years).

(8) Reductions of 22% of major cardiovascular events with the use of statins at 80 mg/d compared to 10 mg/d. The study included 10,001 patients with stable CAD, 37% with age \geq 65 years, to evaluate cardiovascular risk effects on reduction of LDL-C to levels below the recommended by guidelines. No differences were observed in total mortality (Treating To New Targets (TNT)^[18]: Atorvastatin 10 mg vs. Atorvastatin 80 mg).

(9) Significant reductions of mortality and trends to less acute events in a study that included only elderly: 893 elderly patients from 65 to 85 years, stable CAD and ischemia in a 48 hours Holter with a higher doses of statin (Atorvastatin 80 mg vs. Pravastatin 40 mg): Study Assessing Goals in the Elderly (SAGE)^[19].

(10) Reductions of 44% of composed end-points, 55% of non-fatal MI, 48% of non-fatal stroke and 20% of mortality with statins. (JUPITER trial: 17,802 male > 55 years and women > 65 years, without established cardiovascular disease, with LDL-C < 130 mg/dL and C-reactive protein \geq 2 mg/L).^[20]

The National Cholesterol Education Program III (NCEP III)^[21] presented an optional, more aggressive, desirable level of LDL-C at < 70 mg/dL in very high risk patients, with the goal of 100 mg/dL for the ones with high risk.

It is worthy to note that these clinical trials included patients both from the young elderly group (65–74 years old) and from the old-old group (75–79 years old). The only trial that included patients over 80 years was the HPS. Unfortunately, there is no information about the number of octogenarians included in the HPS. Also, there is no data regarding their ages at the end of the study and whether any reached the mean five year follow-up end-point that will provide us with information about 85 year old patients.

Based on this, for the moment, it is possible to acknowledge the benefits of statin therapy in the elderly from 65 to 80, and perhaps 85 year old elderly with established CVD.

The absolute risk reduction was as effective as in other age groups with the drug well tolerated by them.

For the oldest old group, the biological age and functional capacity will guide the choice of treatment.

2.2 Primary prevention

In the elderly without established CAD, but with risk factors for CAD, drug treatment should be introduced when indicated by the high prevalence of subclinical disease. This recommendation is supported by the studies: Cardiovascular Health Study (CHS),^[22] PROSPER,^[14] and HPS.^[13] The National Cholesterol Education Program III (NCEP III)^[23] also recommends dyslipidemia treatment in elderly patients without CAD, since the studies above mentioned certify the efficacy of statin therapy in CAD high risk elderly, even without diagnosed disease.

Since 1989, CHS followed 5,888 healthy elderly ≥ 65 years. In order to evaluate statin use in primary prevention, 2,914 elderly without CVD were followed for seven years. At the end of the study, a 56% reduction of the incidence of cardiovascular events and a 44% reduction in mortality from all causes in the 65–73 year old participants and in the ≥ 74 years was observed. Pharmacological therapy in primary prevention should be considered in high risk elderly with diabetes, several risk factors, or subclinical disease.

Again, the evidence for primary prevention is consistent for the young old and old-old group. As mentioned previously, the only trial that included the oldest-old group of patients over 80 years in secondary and primary prevention was the HPS.

Based on this, for the moment, it is possible to recognize the benefits obtained with the statins among the elderly from 65 to 80 years of age, and possibly elderly 85 years old with established CVD. The absolute risk reduction was as effective as in the other age group and the drug was well tolerated by them. There are no large trials on the use of fibrates on lipid reduction or cardiovascular risk reduction in patients over 75 years old.^[9]

For the oldest old group regarding primary prevention the biological age and functional capacity will guide the choice of treatment.

2.2.1 HDL-C

The HDL-C is a very important lipid fraction in the elderly.^[9] Epidemiologic studies show low levels of HDL-C have a strong correlation with global risk of atherosclerosis and CAD. The results of the Framingham Heart Study showed that the cardiovascular risk was more important in patients with high HDL-C and low HDL-C, but it remained present in patients with relatively low LDL-C and low

levels of HDL-C.^[9,24] A study from the Veterans Administration demonstrated a strong inverse risk relationship between HDL-C and CAD, even in patients with very low levels of LDL-C.^[9,25]

Exercises, weight-loss, moderate use of alcohol and use of nicotinic acid, fibrate and statins can raise HDL-C levels. Niacin or nicotinic acid is considered the best drug to raise low HDL-C. Nevertheless, the side effects of flushing of the skin make it intolerable for some elderly. This can be ameliorated by an initial low dose and gradual increase of the drug together with ingestion after meals and no hot liquid. Recently, there is the option of niacin linked to laropirant that reduces the chance of flushing occurrence.

2.2.2 Cholesteryl ester transfer protein (CETP) inhibitor

A novel class of therapeutic agent in development is the CETP inhibitors. They promote the transfer of cholesteryl esters from HDL-C to other lipoproteins; the inhibition of this protein raises HDL-C levels and decrease LDL-C levels.^[26]

Torcetrapib was the first one of this class. Torcetrapib^[27] was tested initially in 162 individuals in the age range 18 to 65 years old with below-average HDL-C levels (men < 44 mg/dL; women < 54 mg/dL). It showed a substantial dose-dependent elevation in HDL-C, followed by a moderate decrease in LDL-C at the higher doses. It was generally well tolerated.

It was then tested on a first phase 3 CETP inhibitor trial as part of a larger clinical trial at the Investigation of Lipid Level Management to Understand its Impact in Atherosclerotic Events (ILLUMINATE trial^[26]). This trial involved 15,067 patients from 45 to 75 years old (mean age 61 years, 78% male, 93% white) with CHD or CHD risk equivalent (type 2 diabetes). Subjects were randomized to torcetrapib plus atorvastatin or to atorvastatin alone. After an average 12 months of follow-up, the torcetrapib patients showed an increase of 72.1% in HDL-C and a decrease of 24.9% in LDL-C, compared with baseline. But the drug was also associated with an increase of 5.4 mmHg in systolic blood pressure, an increase of 25% in cardiovascular events, and an increase of almost 60% in all-cause deaths. Torcetrapib was also associated with a decrease in serum potassium and increases in serum sodium, bicarbonate, and aldosterone. *Post hoc* analyses showed an increased risk of death in torcetrapib-treated patients whose reduction in potassium or increase in bicarbonate was greater than the median change. Further analyses on the causes of death showed more fatal stroke with torcetrapib (6 vs. 0), more deaths due to cancer (24 vs. 14) and infection (9 vs. 0). The drug trial was suspended by the pharmaceutical company.

Further investigations suggested that the adverse effects of torcetrapib were not necessarily related to the inhibition

of CETP^[28-31] and are not necessarily shared by the others members of the class of CETP inhibitors.^[28]

Other new CETP drugs were then tested, including anacetrapib and dalcetrapib.

Anacetrapib was tested under the DEFINE trial (The Determining the efficacy and tolerability of CETP inhibition with Anacetrapib study^[28]). It was a randomized, double-blind, placebo-controlled trial. The aim was to assess the efficacy and safety profile of anacetrapib in patients with CHD or at high risk for CHD. It was tested on 1623 patients between 18 and 80 years of age with LDL-C levels between 50 mg/dL and 100 mg/dL (1.3 mmol/L and 2.6 mmol/L) while taking statin, an HDL-C level of less than 60 mg/dL (1.6 mmol/L) and a triglyceride level of 400 mg/dL (4.5 mmol/L) or less. The mean age was 62.5 ± 8.7 years of the anacetrapib group and 62.9 ± 9.0 years of the placebo group. They were assigned to receive anacetrapib or placebo daily for 18 months. The primary end-points were the change in LDL-C (HDL-C level was a secondary end point) and safety of the drug. The authors concluded that treatment with anacetrapib had robust effects on LDL-C and HDL-C levels and, within the limits of the power of the study and did not result in the adverse effects observed with torcetrapib, and more recently evacetrapib.

Dalcetrapib was tested under two phase 2b trials, dal-PLAQUE and dal-VESSEL. The purpose of these two studies was to assess the safety and efficacy of dalcetrapib with different imaging modalities.^[32] In the dal-PLAQUE trial, dalcetrapib showed no evidence of plaque progression or pro-inflammatory properties at 24 months, suggesting a favorable safety profile. The dal-VESSEL trial revealed that administration of dalcetrapib does not cause endothelial dysfunction, or has an effect on ABPM. Nevertheless, the confirmation of the long term safety and clinical efficacy of dalcetrapib on cardiovascular outcomes must be still established. This will be provided by the results of the dal-OUTCOMES trial, which is still ongoing triglyceridemia. High levels of triglycerides have been related to the risk of CAD, but they lose significance when adjusted to other variables like glycemia and HDL.^[9] Nevertheless, even after adjusting for HDL-C, the composed hyperlipidemia (i.e., elevated levels of LDL-C and triglycerides) is related to the risk of CAD in a higher proportion than higher individual levels of LDL-C or triglycerides.^[9]

Hypertriglyceridemia treatment should include diet, exercise, and nutritional re-education programs.^[9] The recommended triglycerides level is ≤ 150 mg/dL. When drug use is necessary, nicotinic acid is the first line drug due to its beneficial effect on LDL-C, HDL-C and triglycerides, despite the higher incidence of adverse effects than the other

dyslipidemia's drugs. The inhibitors (statins) of hydroxyl-methylglutaryl CoA reductase are the next choice for their efficacy in the reduction of LDL-C, although they have modest effects on triglycerides and HDL-C. A higher dose of statin seems to significantly decrease triglycerides. Statins can be the first treatment choice in those patients with very high levels of LDL-C. The fibrates have variable effects on LDL-C levels and probably should be used only if the fasting levels of triglycerides are ≥ 400 mg/dL to 500 mg/dL. Finally, when the goal of LDL-C levels is attained, but the triglyceride levels remain high with low HDL, the use of fibrates in combination with statin can be effective. Gemfibrozil should not be used in association with statin due to its adverse effects.

3 Non-pharmacological treatment of dyslipidemia

Because lifestyle changes are very difficult to achieve, doctors in general, are prone to prescribe many drugs to control cardiovascular risk factors. However, it is very important to remember that healthy food consumption remains a cornerstone in primary and secondary cardiovascular prevention and should be implemented by everyone.

3.1 Diet

One of the most important aspects of human life related to atherogenesis is the dietary pattern. Over the years, an exhaustive pool of evidence have been accumulating that many food items are related positively or negatively with the prevalence of atherosclerosis and its complications. Nutrients are linked to many atherosclerosis risk factors: hypertension, alteration of the lipid profile, obesity, diabetes, altered coagulation and probably others still not totally understood.

Diet is a multi-component mixture of many nutrients which may interact with one another. We still do not have a definitive study of the impact of nutrients on cardiovascular disease. Many problems exist in the design of the studies, the population being studied is quite often heterogeneous as is the diet, and it is the diet that is used as the baseline for interventions. Many approaches have been used to examine the influence of nutrition on atherosclerosis including cross population comparisons, nutritional questionnaires administered to large population groups, and interventional studies. Some of these approaches can be very informative about individual nutrients. However, metabolic ward studies where the nutrients are varied in a specific fashion without changing total calories or nutrient balance, are most likely to yield relatively definitive answers, aside from the variable being studied. However, they are not closely related to the real lives of free living peoples.

The primary dietary determinants of hypercholesterolemia are fats, particularly saturated fats, and dietary cholesterol. Unlike dietary fat which is almost completely absorbed in the intestinal tract, the absorption of cholesterol is incomplete and is regulated at the intestinal epithelium. The evolution of our knowledge about the link between hypercholesterolemia and atherosclerosis has been recently revised by Steinberg in his recent thematic reviews.^[33–35]

3.2 Unsaturated fatty acids

The action of fatty acids on the lipid profile depends mainly of two distinct characteristics: (1) If they have double bonds in their carbon chain, and (2) If their isomeric formula is *cis* or *trans*.

Saturated fatty acids do not present any double bonds in their carbon chains, only single bonds. Their main representatives in human nutrition are lauric, myristic and palmitic acids. They are present in milk and meat, their derivatives are in typical Brazilian food, such as palm oil (dende oil) and coco-nut fat. The regular consumption of foods containing high amounts of saturated fatty acids leads to an increase in plasma LDL-C levels accompanied by an increase in HDL-C levels. However, the first effect prevails, leading to an atherogenic lipid profile. Milk fatty acids, present in whole milk and its derivatives, are the worst ones for the lipid profile.

The first step when orientating a prudent diet is to reduce the consumption of foods that contain saturated fats. There is one exception among the saturated fatty acids. This is stearic acid (18 carbons with no double bonds) that does not increase LDL-C levels because upon entering the blood stream, it is rapidly transformed in oleic acid (18 carbons with one double bond) that has a beneficial action on the lipid profile.^[36] Stearic acid is an important component of cocoa butter. With this in mind, dark chocolate (with no other kind of fat) can be an option for those people who like this delicacy.

Unsaturated fatty acids fall into several different categories: (1) monounsaturated fatty acids, which the main representative is oleic acid. It has 18 carbons in its molecular chain and only one double bond (C18:1) and is present mainly in olive and canola oils, dry fruits (walnuts, almonds, hazelnuts, cashew-nuts, Brazilian-nuts) and avocado. Its main action on the lipid profile is to decrease LDL-C levels with no effect on HDL-C levels. When it is used as a substitute for saturated fats in the diet, it can compensate the HDL-C decrease observed with the reduction of those.^[37] It is noteworthy to remember that olive oil is one of the important components of the Mediterranean Diet which has a protecting effect against CHD.^[38] (2) Polyunsaturated fatty acids—they

can be omega 3 or omega 6 according to the first double position on the carbon chain.

The main representative of omega 6 is linoleic acid, which is found mainly in plant oils as safflower, corn and soy oils. Their action on the lipid profile is to decrease LDL-C levels, but they do not counteract the decrease of HDL-C observed with the reduction of saturated fatty acids consumption. Linoleic acid is an essential fatty acid because it is the precursor of arachidonic acid, which in turn is the source of leukotrienes and prostanoids. These molecules have a variety of effects on the cardiovascular system, some beneficial (e.g., prostacyclin) and others not (e.g., thromboxane). Since the unsaturated fatty acids (e.g., linoleic acid) present in the LDL-C particles are prone to oxidation it can be discussed that such moieties enriched in these fatty acids would be proatherogenic.^[28] However, there is no evidence that diets enriched in omega 6 fatty acids promote lipid oxidation.

The Omega 3 fatty acids can be derived from fish oil (eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) or from some plants and oils (alpha-linolenic acid). Data showing a role in cardiovascular prevention are more robust for fatty acids derived from fish oil. A high consumption of fish oil can lower plasma triglycerides levels, blood pressure, platelet aggregation, and inflammation, and increase vascular relaxation.^[39] However, the main benefit derived from fish oil consumption appears to be in the decrease of sudden cardiac deaths due to fatal arrhythmias rather than on the underlying atherosclerosis.^[40] There is a beneficial effect with the use of fish oil supplements after MI as shown by the GISSI-Prevenzione group.^[41]

Trans fatty acids, even as unsaturated fatty acids, can propitiate bad changes of the lipid profile, worse than the saturated ones, leading to an increase in plasma LDL-C levels accompanied by a decrease in HDL-C levels and an increase of triglycerides. The main representative of this class in human nutrition is elaidic acid, which is found in vegetable hydrogenated fats present in many margarines, biscuits and cookies, sauces and ice creams. Their consumption must be kept as low as possible.

Only foods coming from animal sources have cholesterol with higher concentrations than in sea food, animal viscera,

6 Conclusion

Treatment with statins is recommended for elderly patients with established cardiovascular disease. Since elderly people often have comorbidities and have altered pharmacokinetics, it is recommended to begin lipid lowering medication at a low dose and then titrate with caution to achieve target lipid

levels. Statin therapy may be considered in elderly subjects free of cardiovascular disease, particularly in the presence of at least another cardiovascular risk factor, besides age.

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