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Dietary fat and cardiovascular disease

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Cardiovascular disease remains a significant cause of death in industrialized society. Although some controversy persists, scientific evidence largely suggests that it is possible to reduce blood cholesterol by dietary measures and that reduction of hypercholesterolaemia is associated with a reduced risk of coronary artery disease. There is, furthermore, general consensus amongst health authorities with regard to dietary guidelines for modifying hypercholesterolaemia.

This paper examines how dietary intervention, with particular emphasis on dietary fat, may influence the pathogenesis of coronary artery disease. (JCCA 1989; 33(1): 15–21)

KEY WORDS: coronary artery disease, fat, cholesterol, dietary prevention, chiropractic.

La maladie cardio-vasculaire demeure une importante cause de décès dans la société industrielle. Malgré l'existence de certaines controverses, les preuves scientifiques indiquent en grande partie qu'il est possible de réduire le cholestérol sérique grâce à des mesures diétitiques et que cette réduction de l'hypercholestérolémie est associée à un risque réduit de maladie coronarienne. De plus, les experts dans le domaine de la santé sont en général d'accord en ce qui concerne les directives diétitiques permettant de modifier l'hypercholestérolémie.

Cette communication permet d'examiner comment l'intervention diétitique, attachant une importance toute particulière aux lipides diétitiques, peut influer sur la pathogénèse de la maladie coronarienne. (JCCA 1989; 32(1): 15–21)

MOTS CLEFS: maladie coronarienne, lipides, cholestérol, prevention par des moyens diétitiques, chiropraxie.

Cardiovascular disease – a persistent problem

Despite a continuing reduction in deaths attributable to cardiovascular disease, coronary heart disease remains the major contributor to mortality statistics in Australia[1]. Further reductions in mortality are desirable from both a personal cost and community expenditure perspective. The annual cost to Australia of cardiovascular disease is estimated to be $1,126 million; 36% of this is a direct health cost, the remainder is loss of the potential income and contribution to society by sufferers of this condition[2]. The 1985 Australian cost estimate by the federal Minister for Sport Recreation and Tourism of cardiovascular disease exceeded $5.46 million every day[3]. These statistics reflect trends in the industrialized western world.

Intervention to reduce the prevalence of cardiovascular disease may be achieved by primary prevention and by risk factor reduction in otherwise healthy people. Between 1966 and 1983 the mortality attributable to ischaemic heart disease in Australia decreased by 40%; during this time serum cholesterol levels fell by between 0.04 and 0.07 mmoles/1/year, cigarette smoking decreased and diastolic blood pressure fell by 0.05 – 0.6 mmHg/annum[4]. Risk factors believed to contribute to the pathogenesis of this condition include hyperlipidaemia, hypertension and smoking; but to a lesser extent by obesity and an abnormal glucose tolerance. It has been estimated that the reduction of the three major cardiovascular risk factors accounts for 40-60% of the decline in ischaemic heart disease in Australia and an estimated 60% reduction in the New Zealand cardiovascular mortality[5]. Despite this enthusiasm for the effectiveness of risk factor intervention, doubts remain. In two prospective randomized multifactorial intervention clinical trials, it was found that while risk factors were successfully reduced, no reduction in overall coronary artery disease mortality was achieved[6,7]. "Prospective studies to date do not offer conclusive results for the prevention of ischaemic heart disease .... Furthermore, collective evidence fails to corroborate findings from the observational studies about risk factors except for cholesterol"[8]. So convinced are some by the relationship between cholesterol and cardiovascular disease, that the influence of additional cardiovascular risk factors have been quantified in terms of cholesterol increases. For example, the effect of smoking has been equated to an estimated risk increase of 1.29–2.59 mmol/l (50–100 mg/dl) of blood cholesterol[9].

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Secondary prevention, ie. intervention in persons with evidence of disease, is also disappointing. Burr analysed the outcome of secondary prevention trials involving alteration of dietary fat and found that only two of the seven studies quoted were successful in reducing the mortality rate of their study population[10]. The Ireland-Boston heart study did show that a high intake of saturated fat and cholesterol with a relatively low intake of polyunsaturated fats and fibre, did correlate with increased coronary artery disease mortality. These workers concluded that diet is related, "albeit weakly", to the development of coronary artery disease[11].

Fat is a major target for dietary intervention in prevention and control of cardiovascular disease. The Cardiovascular Disease Taskforce of the Better Health Commission recommends, that by the year 2000, fat should contribute less than 33% of the total energy in the Australian diet[12]. Burr in an analysis of six prospective studies of diet and ischaemic heart disease found a positive association between ischaemic heart disease and fat in two thirds of the investigations studied[10]. Of the dietary variables studied vis-à-vis energy, alcohol, sugar and fibre, only fat showed a positive correlation. As cardiovascular disease remains a major health problem in Australia and dietary fat constitutes a target for clinical intervention, it would appear opportune to analyse the relationship between the pathogenesis of this condition and the dietary ingestion of diverse fats.

Fat and atherosclerosis

The Anatomy

The earliest lesion of atherosclerosis is the fatty streak. It is a flat vascular lesion rich in lipid containing smooth muscle cells and macrophages. Fatty streaks have been detected in children (10 years of age) and young adults. With increasing age, a fibrous cap develops overlying an accumulation of cells, connective tissue, cholesterol crystals and calcified necrotic material. This fibrous, and later calcific plaque, protrudes into the lumen of the vessel. Superimposed thrombosis with or without ulceration of the underlying plaque may result in complete vascular occlusion. The principal cells involved in the pathogenesis of an atheroma are monocytes/macrophages, platelets, endothelial and smooth muscle cells. All these cells contain or can synthesize chemotoxins and growth factors, similar to platelet derived growth factor; all these cells with the exception of platelets, can bind low density lipoprotein[13].

The Physiology

Low density lipoproteins are the main carriers of cholesterol to the peripheral tissues. Their composition is: 46% cholesterol, 22% phospholipid, 11% triglycerides and 21% protein. The major surface protein component of low density lipoprotein (LDL) is apoprotein B-100. Low density lipoprotein is removed from the circulation of LDL receptors recognizing apoprotein B-100. Receptors bind most efficiently when blood LDL levels are below 50 mg/dl. The LDL-receptor hypothesis suggests that hyperlipidaemia results in increased delivery and accumulation of cholesterol in cells. Cellular accumulation of cholesterol promotes cellular storage of cholesterol, inhibits further endogenous cholesterol synthesis and reduces cellular synthesis of LDL receptors, thereby restricting removal of circulating lipids[14]. Logical extension of this postulate would suggest that hyperlipidaemia may result from excessive fat ingestion, inhibiting or impairing genetic coding, limiting production of LDL receptors. This hypothesis has been further extended to explain the increased LDL concentration associated with aging in the industrialized society[15]. In any event, it is generally acknowledged that sustained hyperlipidaemia / hypercholesterolaemia is associated with severe and premature atherosclerosis. While many authorities are persuaded that hypercholesterolaemia is a cause of coronary heart disease, the mechanism whereby hyperlipidaemia potentiates atherosclerosis remains obscure. Postulates which are proposed include[13,16,17]:

1 peroxidation of lipids by macrophages with production of metabolites toxic to endothelial cells,
2 alterations to cell permeability with consequent fatty acid accumulation,
3 modification of endothelial cells resulting in monocyte adhesion and chemotaxis, and
4 increased viscosity and reduced cellular malleability as a result of increased cholesterol:phospholipid ratios in cell membranes.

The overall thrust of the postulated mechanisms is increased permeability of the vascular endothelial lining with penetration of lipids and cells into the subendothelial space. The basic pathogenesis postulated is that of chronic inflammation with lipid moieties acting as irritants or stimulants to the generation of pro-inflammatory metabolites.

Risk Identification

Analysis of blood lipids has shown that LDL is highly atherogenic[18]. In contrast, chylomicrons and very low density lipoprotein (VLDL), respectively representing predominant plasma carriage of exogenous and endogenous triglycerides, do not appear to be of major importance in the pathogenesis of this condition. High density lipoproteins (HDL), involved in the transport of cholesterol from tissue to the liver, are believed to be protective. In fact, an inverse relationship has been identified between HDL-cholesterol and coronary artery disease[19]. LDL cholesterol correlates positively with an increased risk of coronary artery disease; on the other hand, HDL cholesterol has an inverse correlation with coronary heart disease[16]. The strongest predictors of coronary heart disease appear to be summary estimates of cholesterol indices[20]. If hypercholesterolaemia is an aetiological factor in the pathogenesis of atherosclerosis, it is important to identify the level at which serum cholesterol does not constitute a health hazard.

The risk of atheroma increases in a continuously graded fashion as blood cholesterol increases; no threshold serum cholesterol level has been identified[21]. A threshold atheroma-

tous plaque level has been identified; ie. when 60% of the surface area is covered with raised plaque a critical zone associated with clinical manifestations of the ischaemia is entered[9]. It has been suggested that the ideal cholesterol range for the whole population would be between 3.36–4.19 mmol/l (130–190 mg/dl). The Cardiovascular Disease Taskforce of the Better Health Commission recommended that the 1983 mean fasting serum cholesterol levels in Australian adults be reduced from 5.61 mmol/l in men and 5.65 in women to 4.8 mmoles per litre[22]. This would mean a reduction of blood cholesterol levels of about 1.7 mmols/l over a 25 year period. A reduction of 0.05–0.07 mmols/l of blood cholesterol has been predicted to cause a 2% reduction in coronary artery disease[23]. The best lipid predictor of coronary heart disease currently available to clinicians is the total cholesterol:HDL-cholesterol ratio. This ratio in vegetarians is 2.9, in Boston marathon runners it is 3.5, in females without coronary heart disease it is 4.4, and in males without coronary heart disease it has been found to be 5.1. Females with coronary heart disease have a total cholesterol: HDL-cholesterol ratio of 5.3, while in males with coronary heart disease the ratio has been found to be 5.8. It has been suggested, that a desirable total cholesterol: HDL-cholesterol ratio is less than 4.0. However, the current average risk of coronary artery disease could be halved by reducing this ratio to 3.43 in men and to 3.27 in women.

Dietary recommendations for cardiovascular health

A number of risk factor intervention studies, while not uniformly successful, do suggest that deaths attributable to coronary heart disease may be reduced by risk factor modification or elimination[24]. If dietary alteration is to be used as an interventive measure, it is desirable that the links between diet and coronary artery disease be clearly established, both in terms of the quantity and quality of dietary fats involved. The whole lipoprotein spectrum is influenced favourably when the dietary recommendations of "moderate reduction in total fat, marked reduction in saturated fat and cholesterol, moderate increase of polyunsaturated fat, moderate increase in complex carbohydrates and fiber, foods of low caloric density to assist in avoiding or correcting obesity while ensuring a high intake of all essential macro- and micronutrients, and avoidance of high intake of sodium and alcohol" are followed[25]. Dietary alteration may mediate a beneficial effect through mechanisms other than blood lipid modification, eg. epidemiological studies show that moderate alcohol consumption has a cardioprotective effect[26,27] while any benefits attributable to sodium restriction are mediated through modification of hypertension[28], another major risk factor. Diverse dietary modifications can also alter hyperlipidaemia.

Complex carbohydrates eg. oats, have been shown to favourably reduce blood cholesterol levels[29], while a non-significant but interesting inverse correlation has been found between simple sugars and HDL-cholesterol[30]. In the Leiden trial, it was found that a vegetarian diet with a polyunsaturated: saturated fatty acid ratio of at least 2 and a cholesterol intake of less than 100 mg/day, could result in a lowering of total blood cholesterol with a decrease in the total cholesterol:HDL-cholesterol ratio; it was also found that lower blood lipid indices were associated with stable rather than progressive coronary lesions[31]. Different dietary constituents may induce specific alterations in blood lipid patterns. Mensink and Katan conducted a controlled clinical trial in which complex carbohydrate was found to reduce total cholesterol levels while olive oil, a monounsaturated fatty acid, reduced blood cholesterol without simultaneously lowering HDL cholesterol and raising serum triglycerides[32].

The type and source of dietary fat also appears an important variable in controlling hyperlipidaemia. In a controlled clinical trial it was found that vegetable oil achieved a reduction in serum cholesterol while salmon oil, in addition to reducing blood cholesterol, also reduced serum triglycerides[33]. Both these unsaturated fatty acid sources achieved an improvement in blood lipids, despite the underlying diet containing 500mg cholesterol with 40% of its calories derived from fat.

Fish oils have been shown to alter serum lipid parameters in both human and animal feeding trials[34]. A longitudinal study performed in Zutphen demonstrated an inverse relationship between fish consumption and the 20 year mortality rate from coronary artery disease[35]. In fact, coronary heart disease mortality was 50% lower in persons who had a daily fish consumption of at least 30 grams.

Dietary intervention to modify blood lipids includes: a reduction in total fat consumption with a relative increase in polyunsaturated and a relative decrease in saturated fats, a reduction in dietary cholesterol, and an increase in complex carbohydrates[36]. As restricting saturated fat and cholesterol has been found to only have a moderate effect on lowering blood cholesterol, it is important to examine fat intake in the context of the total diet. Consideration of dietary fat modification to control hyperlipidaemia, therefore includes consideration of cholesterol, saturated and unsaturated fatty acids.

Dietary Cholesterol

While dietary fatty acids are recognized for their impact on cardiovascular health, the impact of dietary cholesterol remains somewhat disputed. Dietary cholesterol does not appear to contribute more than 10% of the total serum cholesterol and some suggest that the correlation between dietary cholesterol and coronary heart disease is weak[24]. Others, however, maintain that a reduction of cholesterol intake correlates with a significant reduction in coronary heart disease[37].

Reduction of dietary cholesterol can be achieved by avoiding cholesterol rich foods such as butter, brains, kidney, liver, caviar, whole eggs and cheese; in other words foods of animal rather than vegetable origin. Furthermore, it has been suggested that the form in which cholesterol is eaten determines the cardiovascular effect of this compound. The possibility that oxidation of cholesterol during cooking may produce a metabo-

lite toxic to arterial walls has been proposed. This may to some extent explain the ongoing egg-cholesterol controversy[38]. It has been found that raw egg has a negligible effect on serum cholesterol compared to fried and hard boiled eggs. Appropriate dietary information about cholesterol intakes may also imply information about food preparation methods.

In view of the association of cholesterol with saturated fat, it is difficult to clearly identify the particular role of each of these dietary compounds in hypercholesterolaemia[39]. It is also worth noting, that dietary ingestion of unsaturated omega-3 fatty acids in the form of fish oil is also associated with increased cholesterol ingestion; 100 grams of cod liver oil delivers 19.2 grams of omega-3 fatty acids and 570 mg of-cholesterol[40]. Certainly all of these dietary elements are believed to contribute to serum cholesterol levels and should therefore be discussed.

Dietary Fatty Acids

Alteration of dietary fatty acids implies careful dietary selection. In practice, a reduction of saturated fat from 20% to 10% of the daily kilojoule intake with a relative increase of polyunsaturated fats to 10–15% of daily kilojoule intake with an overall reduction of fat intake, can be achieved by a vegetarian diet or a diet which emphasizes complex carbohydrates and fish or wild game rather than meat[41]. In terms of food selection a similar cardioprotective effect from essential fatty acids and their products can be achieved by selecting from:

- safflower, soyabean, corn and cottonseed vegetable oils, barley and wheat flour, apples and beans (all sources of linoleic acid; n-6)
- linseed oil, spinach and beans (sources of linolenic acid; n-3).
- deep and cold water fish eg. cod, mackerel; oysters, shrimps and crabs as sources of eicosapentaenoic acid, an intermediate product of the n-3 linolenic acid.

Both classes of essential fatty acids, linoleic (n-6) and linolenic (n-3), are required for the transport and oxidation of cholesterol, as components of phospholipids in cell membranes and for the formation of prostaglandins.

As with cholesterol, cooking or processing of unsaturated fats can alter their metabolic influence. Hydrogenation of vegetable or fish oils rich in cis-unsaturated fatty acids results in increased saturation and trans-configuration of fatty acids[42]. Transunsaturated fatty acids behave metabolically like saturated fatty acids. In addition to a dietary deficiency of essential fatty acids the normal prostaglandin synthetic pathways may be imbalanced by inhibition of delta-6-desaturase, a rate limiting enzyme in the synthesis of cardiac protective and other prostaglandins. This enzyme's activity has been shown to be impaired by saturated fatty acids, trans-fatty acids, alcohol and the diabetic state[43,44]. Increased unstaurated fatty acid consumption increases the individual's requirement for vitamin E. Decreased dietary cholesterol reduces an exogenous source of cholesterol and hence serum cholesterol. The mechanisms whereby an increased polyunsaturated:saturated fatty acids ratio may promote cardiovascular health is postulated to include[44,45a]:

1 a reduction of endogenous cholesterol. The availability of unsaturated fatty acids in cell membranes may enhance cholesterol mobilization and hence potentially facilitate cholesterol excretion. It has been postulated that mobilization of free cholesterol from cell membranes is facilitated by esterification of cholesterol to an unsaturated fatty acid (cholesteryl linoleate) rather than a saturated one (cholesteryl oleate)[46]. Omega-3 fatty acids have been shown to lower total serum cholesterol even when challenged by a 'high'(750mg/ day) dietary cholesterol intake[47]. A reduction in coronary heart disease correlates with a reduction in total and LDL-cholesterol[48].

2 altered platelet function. The critical occlusion in a heart attack may be clot formation in a vessel narrowed by an atheromatous plaque. The type of dietary polyunsaturated fatty acid ingested has been shown to modulate prostaglandin synthesis. Linolenic acid and its product eicosapentaenoic acid (a fatty acid rich in eskimo diets) produce prostaglandins of the III series. These prostaglandins reduce blood lipids and prolong clotting time. Linoleic acid and its products may produce prostaglandins of the I or II series. Prostaglandin I series products, reduce platelet aggregation. Prostaglandins of the II series, derived from arachidonic acid (a product of linoleic acid (vegetable oil) or structural protein of animal cells) cause vasospasm and enhance platelet aggregation. Fish is therefore, the essential fatty acid source of choice in patients in whom platelet stickiness is of paramount importance. A case study, found reduced platelet aggregation in response to collagen in a subject receiving eicosapentaenoic and docosahexaenoic acids[49]. Follow up studies confirmed these findings. Dietary supplementation with fish oils leading to inclusion of omega-3 fatty acids into plasma and platelet phospholipids, alters prostanoid metabolism shifting the prostaglandin I/thromboxane A balance, in favour of a more antiaggregatory and vasodilatory state[50].

   In clinical studies, dietary supplementation of 10 grams per day eicosapentaenoic acid achieved a change in the pattern of thromboxane and prostacyclin synthesis, supplementation with 1 gram per day failed to maintain the changes in thromboxane synthesis[51].

3 modification of atheroma pathogenesis by suppression of inflammatory mediators. It has been shown that coronary artery disease mortality is inversely proportional to the linoleic acid content of adipose tissue[52]. Levels of linoleic acid in adipose tissue reflect the concentration of this unsaturated fatty acid in other tissues. Dietary essential fatty acids are incorporated into tissues; activation of inflammatory cells rich in fish oil derived fatty acids demonstrate an antiinflammatory effect by inhibition of the 5-lipoxygenase pathway and leukotriene production[53].

4 reduction of blood pressure levels[54] and consequent minimization of a second major risk factor for coronary artery

disease. Achievement of such postulated changes is influenced by the chemical structure of the essential fatty acid series involved, the quantity of essential fatty acid consumed, the duration of dietary consumption, and the interaction of other ischaemic heart disease risk factors. The impact of unsaturated fatty acids on coronary artery disease extends beyond their effect on blood cholesterol levels; certain of the essential unsaturated fatty acids and their products may have particular effects in the pathogenesis of atherosclerosis and the precipitation of vascular occlusion. In this context, it is interesting to note Stamler's comment, that the decisive lipid nutritional recommendations for the general population for coronary heart disease prevention "are low saturated fat and low cholesterol intake. High intake of polyunsaturated fat is not one of the recommendations ..."[27]

Dietary advice is often best implemented when information is provided in terms of foods rather than saturated fat or cholesterol. Reduction of saturated fat means limiting foods rich in saturated fats. Foods with a saturated fatty acid content of more than 30% include red meat, butter, butterfat, coconut and palm oils; those with less than 20% saturated fatty acids include fish, nuts, olive/safflower/peanut and corn oils[55]. Eating foods with a polyunsaturated:saturated fat ratio which exceeds 1, lowers blood cholesterol. Foods which have a polyunsaturated:saturated fat ratio of greater than 1 include: cereal grains, legumes, nuts, seeds and most fish, exceptions include eel, Greenland halibut, chinook salmon, and some mackerel[40]. While adequate polyunsaturated fats may exert a protective effect against dietary cholesterol, cholesterol should not be ignored. Food indices have been developed which can be used as a measure of the atherogenicity of food, for example the Keys and Hegsted scores, and more recently, the cholesterol/saturated fat index (CSD)[56]. A low CSI indicates a low saturated fat and cholesterol content and identifies food with a low atherogenicity. Applying this index to 100 gram quantities, shows that the comparative 'atherogenicity' of red meat is between 9–18; poultry (without skin) is 6; whitefish is 4; while vegetable oils is 8, soft margarine 10, bacon grease 23, butter 37 and cocoa butter(chocolate) 47; bread and legumes 1 and cereals, potatoes, vegetables and fruit 0(zero) (except for avocado).

An overview

In 1964, a pragmatic approach was adopted in America and recommendations were made that there should be a reduction in cholesterol and saturated fat consumption; over the next seventeen years a decline in the consumption of animal fats, butter, eggs, milk and an increased consumption of fish and vegetable fat paralleled a decline in age-specific coronary mortality[57]. This decline also coincided with Americans being warned about the hazards of smoking. In 1985 an editorial in the Journal of the American Medical Association, remarks that while "it is appropriate to want scientific proof before making recommendations about therapy and changes in the American lifestyle" ... the reality of problems associated with large multi-centre randomized clinical trials makes the "usual pragmatic clinical approach" very attractive in the middle of the 1980's[58]. In 1984 the National Heart, Lung, and Blood Institute and the National Institutes of Health Office of Medical Applications of Research convened a Consensus Development Conference on Lowering Blood Cholesterol to Prevent Heart Disease. A consensus panel of lipoprotein experts, cardiologists, primary care physicians, experts in preventive medicine, epidemiologists, biomedical scientists, biostatisticians and laypersons reviewed available evidence and concluded, "it is beyond reasonable doubt that lowering definitely elevated blood cholesterol (specifically, blood levels of low density lipoprotein cholesterol) will reduce the risk of heart attacks caused by coronary disease."[59] The dietary approach should be to lower total fat, saturated fat, and cholesterol consumption specifically, "We recommend a diet composed of approximately 30% of the caloric intake from fats and no more than 250–300 mg of cholesterol per day."[59] Pyorala, after reviewing the literature, concluded that reducing dietary cholesterol to less than 300 mg daily as part of a prudent diet has a good scientific basis; he did however emphasize the marked individual differences to dietary cholesterol[60]. Another consideration is the reduction of the total saturated fat intake to 10% or less of total calories. It is recommended that "polyunsaturated fat intake be increased but to no more than 10% of calories."[56] The recommendations listed with regard to total, saturated, polyunsaturated fatty acids and cholesterol are precisely mirrored in the 1988 position statement of the American Heart Association[61]. As Truswell remarks after examining the advice resulting from twenty-one expert committees representing eight different countries or regions, the main recurring points to prevent coronary artery disease are less total fat, less saturated fat and control of obesity; most sources also advised less dietary cholesterol and partial replacement of saturated by polyunsaturated fat[62]. While some still maintain "there is no certainty about the optimal nutritional approach to lowering of LDL cholesterol"[63], similar health care trends are occurring in Australia. In 1985, the Commonwealth Minister for Health in Australia set up the Better Health Commission with the aim of changing the basic direction of health policy so as to better emphasize illness prevention. The Cardiovascular Taskforce set community targets for reduced levels of plasma cholesterol, diastolic blood pressure and smoking by the year 2000.[12] They also supported the dietary recommendations of the Nutrition Taskforce. The recommendations of the Nutrition Taskforce of the Better Health Commission include a reduction of over weight and obesity, a modification of the Australian diet emphasizing a reduction of the energy contribution derived from fat, refined sugar, alcohol and sodium, with an increase of dietary fibre[64].

Concluding remarks

Scientific information used in clinical practice is the outcome of correlating and integrating data from numerous epidemiological surveys, animal experiments and clinical trials. The outcome

of such studies is not always congruent. Despite pockets of dissent, there is today general consensus among health professionals regarding the importance of reducing blood cholesterol levels as a strategy for minimizing the cardiovascular risk of atherogenesis and coronary occlusion. Dietary intervention appears to offer a feasible approach to disease prevention and health promotion of this condition. Adoption of recommended dietary guidelines may be anticipated to favourably modify cardiovascular prognosis; this generalization is even believed to be applicable to those who reform their dietary habits in midlife.

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