

Healthy lifestyle interventions in general practice

Part 8: Lifestyle and dyslipidaemia

^aSchwelInus MP, MBBCh, MSc(Med) Sports Science, MD(Sports and Exercise Medicine), FACSM, FFIMS ^bPatel DN, MBBCh, MMed(Paed) ^cNossel C, MBBCh, MBA
^dDreyer M, BSc(Hons Sports Science), BSc(Hons Dietetics) ^eWhitesman S, MBChB ^fDerman EW, MBChB, PhD(Sports and Exercise Medicine), FACSM, FFIMS
^gDepartment of Sports Medicine and Exercise Science, University of Cape Town ^hVitality Wellness ⁱInstitute for Mindfulness South Africa

Correspondence to: Prof Martin SchwelInus, e-mail: martin.schwelInus@uct.ac.za

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Abstract

Dyslipidaemia, in particular raised plasma concentrations of total cholesterol (TC), low density lipoprotein cholesterol (LDL-C), triglycerides as well as reduced plasma concentrations of high density lipoprotein (HDL-C) concentration is one of the major risk factors for coronary artery disease (CAD), cerebrovascular disease and peripheral vascular disease. The clinical assessment of a patient with suspected dyslipidaemia should include a medical history, clinical examination and special investigations to determine the presence and cause of dyslipidaemia, and to perform a risk assessment for cardiovascular disease using specific guidelines. Based on the risk scoring and the plasma lipid concentrations, an individualised management plan can be established for each patient. The focus of this article is on the cornerstone of management of dyslipidaemia which is lifestyle intervention. Lifestyle intervention for dyslipidaemia includes dietary intervention, increased physical activity, stress management and smoking cessation.

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Introduction and definition of dyslipidaemia

The dyslipidaemias represent a number of disorders of lipid metabolism that can be related to 1) increased production of atherogenic lipoprotein particles, 2) delayed degradation of atherogenic lipoprotein particles, 3) decreased synthesis of protective lipoprotein particles, 4) increased degradation of protective lipoprotein particles, or 5) combinations of the abovementioned (1 to 4).¹ The term dyslipidaemia is used to describe primary and secondary derangements of lipid metabolism, including some disorders that result in severe hypocholesterolaemia.² The primary and secondary causes of dyslipidaemia have recently been reviewed.²⁻⁵ The terms dyslipidaemia and hyperlipidaemia are used in many instances interchangeably to refer mainly to those disorders of lipid metabolism that are related to atherosclerosis. However, for the purposes of this article the term dyslipidaemia will be used as this is the correct term that refers to all disorders of lipid metabolism.

It is well established that dyslipidaemia is one of the major risk factors for atherosclerosis, in particular coronary artery disease (CAD), cerebrovascular disease and peripheral vascular disease.⁶ In particular, it is well established that there is a causal link between atherosclerosis and elevated plasma concentrations of total cholesterol (TC) and elevated plasma Low Density Lipoprotein (LDL) concentration.⁵⁻⁷ A 10% reduction in plasma TC is associated with a 25% reduction in 5-year risk of coronary heart disease events, while a 1 mmol/L reduction in plasma LDL concentration is associated with a 20% reduction in this 5-year risk.⁶ Furthermore, elevated plasma triglycerides (TG) and a reduced plasma concentration of High Density Lipoprotein (HDL) have also been positively linked to an increased risk of arteriosclerosis. The focus of this article, the 8th in this series, is on the role of lifestyle modification in the management of dyslipidaemia that is associated with increased risk of atherosclerosis.

Epidemiology of dyslipidaemia

The prevalence of dyslipidaemia varies, depending on the definition of dyslipidaemia and the population that is studied. In the adult population of the United States, the prevalence of dyslipidaemia increases with age and peaks at 40% of men aged 65 to 74.⁴ There is also a positive association between the prevalence of dyslipidaemia and body weight with the prevalence of dyslipidaemia being as high as 44% in obese patients.⁴ The prevalence of increased plasma total cholesterol (> 5 mmol/L) in the South African population groups is depicted in Table I.

Table I: The prevalence (%) of hypercholesterolaemia (total cholesterol > 5mmol/L) in the South African population groups (according to age)

	Age	African	Coloured	Indian	White
Males	20-44	13.9	80.8	84.9	83.9
	45-59	22.0	84.5	92.3	93.8
	60 +	33.3	79.6	78.4	96.0
	30 +	19.7	81.7	86.5	90.0
Females	20-44	10.4	66.9	65.1	75.0
	45-59	40.4	94.8	87.8	95.3
	60 +	53.9	97.9	90.3	99.4
	30 +	29.2	79.9	77.4	88.4
All persons *	20-44	12.1	73.6	74.9	79.5
	45-59	31.6	90.0	89.9	94.6
	60 +	44.7	90.4	85.2	97.9
	30 +	24.7	80.7	81.7	89.2

*: The total number of South Africans (> 30 years) with hypercholesterolaemia is estimated at 5 767 205

These data indicate that dyslipidaemia remains a major cardiovascular risk factor in the South African population, and as a larger part of the population becomes exposed to a westernised lifestyle, this prevalence will increase further.

General management of dyslipidaemia

There are a number of international organisations and groups that regularly release guidelines for the management of dyslipidaemia. The most widely applied guidelines are the most recent releases from the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults⁷ and the European Guidelines on Cardiovascular Disease Prevention in Clinical Practice.⁸ In South Africa, the Lipid and Atherosclerosis Society of Southern Africa (LASSA) adopted the European guidelines for the management of dyslipidaemia in 2006.⁹ Specific guidelines for the management of dyslipidaemia in children⁵ and adults have recently been reviewed in the international^{1,4} and South African medical literature.¹⁰⁻¹²

The general 6-step clinical approach to the patient with suspected dyslipidaemia is to 1) determine if the patient has a lipid disorder, 2) define the lipid disorder, 3) rule out secondary causes of dyslipidaemia, 4) set treatment goals based on risk, 5) initiate therapy based on the treatment goals, and 6) follow-up the patient.¹ A good medical history, clinical examination and special investigations are the cornerstones to assess for the presence of a lipid disorder, defining the disorder, ruling out secondary causes of dyslipidaemia and determining the risk of cardiovascular disease, stroke or other diseases associated with dyslipidaemia such as pancreatitis.¹⁰ Based on the clinical assessment and fasting lipogram results, the 10-year risk for cardiovascular disease (expressed as a %) can be calculated using the Framingham risk charts⁷ or the European SCORE system.⁶ These scoring systems are all based on risk factors for cardiovascular disease that include age, gender, systolic blood pressure, treatment for hypertension, smoking status, total cholesterol and/or HDL cholesterol.^{6,7} In the SCORE system different charts apply to a population that is at high risk compared with a population that is at low risk.⁶

Once the risk factor score has been determined, the guidelines provide specific target goals for plasma total cholesterol and LDL cholesterol concentrations that require management (Table II).⁶

Table II: Guideline cholesterol goals for management in low and high risk individuals^{6,9}

Risk category	Total cholesterol (mmol/L)	LDL-cholesterol (mmol/L)
Low risk	< 5.0	< 3.0
High risk*	< 4.5	< 2.5

*: patients with established atherosclerotic cardiovascular disease (CVD), asymptomatic individuals who are at increased risk of CVD (multiple risk factors resulting in > 5% risk over 10-years, diabetes mellitus, markedly increased single risk factor associated with end-organ damage)

The cornerstone of management of all patients with dyslipidaemia is lifestyle intervention.^{1,6,7} In low risk patients with normal plasma TC and LDL, the approach is to maintain a healthy lifestyle. In low risk patients with raised plasma TC and/or LDL the main treatment is lifestyle intervention with follow-up, usually every three months. Drug treatment must be considered in any high risk patient – those with established atherosclerotic CVD and asymptomatic individuals who are at increased

risk of CVD (multiple risk factors resulting in > 5% risk over 10-years, diabetes mellitus, markedly increased single risk factor associated with end-organ damage) (Table II).

A discussion of the appropriate choice and dose of medication for the treatment of dyslipidaemia is beyond the scope of this article and has been reviewed recently.^{1,4,10-12} The main focus of the remainder of this article is on the approach to lifestyle interventions for dyslipidaemia.

Lifestyle interventions in the management of dyslipidaemia

The principles of lifestyle intervention for dyslipidaemia are similar to those for other chronic diseases that have been reviewed in this series of articles. Specifically, lifestyle interventions for dyslipidaemia consist of dietary intervention, physical activity, stress management, and smoking cessation. These interventions will now be reviewed.

Dietary intervention for dyslipidaemia

In patients with dyslipidaemia, dietary and other lifestyle interventions are critical components for cardiovascular disease (CVD) risk reduction and the management of existing CVD.¹³ It should also be considered that dyslipidaemia may be familial and in these circumstances drug therapy will be essential in addition to recommended dietary and lifestyle interventions.¹⁴ The recommended dietary and lifestyle changes for preventing CVD are the same as for treating existing CVD, as well as the treatment and management of non-familial dyslipidaemia. Therefore, the goals and recommendations discussed below can be applied to both. Diet, exercise and weight reduction may result in patients reaching recommended serum lipid goals (Table II). Physicians are encouraged to refer patients to nutrition experts to assist in meeting these goals.

Definitions and types of fat and fibre in the diet

It is important to understand the different dietary fat components (Table III).¹⁵ The term “fats” represents a group of compounds (hard fats and liquid oils) that are made up of smaller components – fatty acids. These fatty acids are complex molecules and can be further classified as a saturated or unsaturated fatty acid, depending on its structure. Unsaturated fatty acids are further grouped as polyunsaturated fatty acids (PUFAs) and monounsaturated fatty acids (MUFAs). Saturated and unsaturated fats differ in structure and function in the body, with some types being highly detrimental – promoting disease, and others being beneficial – preventing disease.¹⁵

The therapeutic lifestyle change (TLC) diet is aimed at minimising the risk of, and managing dyslipidaemia. Essential components of the TLC dietary pattern are outlined in Table IV.¹⁸

In addition to this, the recommendations for CVD Risk Reduction by the American Heart Association are outlined in Table V. These guidelines should be followed by people with *existing* CVD, as well as those aiming to *reduce their risk* of developing CVD (adults and children over two years of age).^{13,14}

The role of antioxidants in dyslipidaemia

Although benefits from a diet rich in antioxidants (both dietary and supplementation) on CVD risk have been suggested by observational studies, very few clinical trials have demonstrated this benefit. In

Table III: Types of fats and fibre (description, effect on health and food sources)

Types of fat	Description	Health effects	Food sources
Saturated fats	Saturated – or full – of hydrogen atoms, these fats are solid at room temperature	Increases LDL cholesterol, as well as total cholesterol	Brick margarine, solid vegetable oils, butter, full cream milk, full fat hard cheese, fatty cuts of meat, sausage, poultry with the skin on, tropical oils, such as palm oil and cocoa butter
Trans fats	Packaged as “partially hydrogenated vegetable oil” or “vegetable shortening”	Increases LDL cholesterol; may lower HDL cholesterol, raising overall CVD risk	Fast food, snack products, packaged commercially baked goods, brick margarines
Mono-unsaturated fats (MUFA)	Liquid at room temperature	When replacing SFA in the diet these fats lower LDL cholesterol, may lower triglycerides and may elevate HDL cholesterol	Olive oil, canola oil, most nuts and avocado pear
Poly-unsaturated fats (PUFA)	Liquid at room temperature	Lower LDL cholesterol, lower CVD risk	Fish, as well as vegetable oils such as safflower, sunflower, corn, flaxseed, and most other seeds
Omega 3 fatty acids	A form of PUFA. Classified as an essential fatty acid because the body is incapable of producing this fatty acid itself, and relies purely on dietary sources for its intake. Alpha Linolenic Acid (ALA) – parent fatty acid from the family of Omega 3 fatty acids, is ineffectively metabolised by the body to form derivatives <i>eicosapentaenoic acid</i> (EPA) and <i>docosahexanoic acid</i> (DHA). Approximately 1–5% of ALA is converted to EPA and DHA, 14 DHA and EPA (from fatty fish) have been found to be essential for optimal functioning of the brain, central nervous system and all cell membranes in the body.	Tend to suppress triglyceride synthesis in the liver, may modify LDL particle size (increases seen in men), promote a small increase in HDL, are associated with a reduction in inflammation, suppress adhesion molecules and improve endothelial function. ^{16,17}	EPA and DHA are found primarily in fish oils, mainly from oily fish such as salmon, cod, pilchards, herring, mackerel and sardines. ALA is found primarily in certain vegetable oils such as linseed, rapeseed (canola), soya beans and green leafy vegetables
Soluble/ functional fibre	Has a high water holding capacity acting as a bulk former in the gastrointestinal tract and is thought to help lower blood lipid and cholesterol levels, through the production of short chain fatty acids (SCFA) produced by bacterial fermentation by gut microflora. SCFA reduce bile acid uptake by colonocytes thereby reducing cholesterol absorption.		Legumes, fruits, vegetables, wholegrains, and oat products
Insoluble fibre	Passes directly through the digestive system, helping the body to rid itself of waste products, regulating bowel movements and possibly preventing disease such as colon cancer.		Wholegrain products, nuts and skins and pips of some vegetables and fruits

Table IV: Essential components of the TLC dietary recommendations¹⁸

Dietary component	Recommendation	
LDL-raising nutrients	Saturated fats	Less than 7% of total calories
	Dietary cholesterol	Less than 200 mg per day
	Trans fatty acids	Kept to a minimum < 1% of total calories
Therapeutic options for LDL lowering	Plant sterols/stanols	2 grams per day
	Increased soluble (viscous) fibre	10–25 grams per day
Total calories (energy)	Adjust total caloric intake to maintain a desirable body weight/prevent weight gain	
Polyunsaturated fat	Up to 10% of total calories	
Monounsaturated fat	Up to 20% of total calories	
Total fat	25–35% of total calories	
Carbohydrate	50–60% of total calories (predominantly in the form of wholegrains, vegetables and fruit)	
Dietary fibre	20–30 grams per day. This recommendation can easily be reached through the intake of five or more servings of fruits and vegetables per day and six or more servings of grains per day. ¹⁴ At least half of grain intake should come from whole grains. ¹³	
Protein	Approximately 15% of total calories	
Physical activity	Include enough exercise to expend at least 200 kcal per day	

Table V: American Heart Association (AHA) guidelines for CVD risk reduction^{13,14}

Guideline	Recommendation
Balance caloric intake and physical activity to achieve and maintain a healthy body weight ^{13,14}	<ul style="list-style-type: none"> Energy intake must match energy expenditure in order to maintain weight and prevent weight gain. To control calorie intake, awareness around the calorie content of foods and beverages as well as portion control needs to increase.^{13,14} Energy expenditure and subsequent weight loss can be achieved through increasing physical activity.^{13,14} A physically active lifestyle is recommended to reduce the risk of CVD in all individuals, regardless of their weight.¹³
Consume a diet rich in fruits and vegetables ^{13,14}	<ul style="list-style-type: none"> Most fruits and vegetables are rich in nutrients, low in calories and high in fibre and can be included in substantial amounts, meeting micronutrient, macronutrient and fibre recommendations without adding significant extra calories to the diet, therefore lowering the overall energy density of the diet and assisting with weight control.¹³ They have also been shown to lower blood pressure and improve other CVD risk factors.¹⁴ A variety of fruits and vegetables should be included in the diet, particularly deeply coloured ones such as berries, beetroot, oranges, broccoli, red grapes, spinach, carrots and peaches, as these colours indicate rich micronutrient content.¹³
Choose wholegrain, high fibre foods ^{13,14}	<ul style="list-style-type: none"> Consuming a diet high in whole-grains and fibre increases diet quality and has been shown to decrease risk of developing CVD and demonstrate consistent beneficial effects on lipid metabolism.^{13,19} Soluble or viscous fibres (especially pectin and β glucan) have been found to lower blood cholesterol levels^{13,19} by lowering LDL cholesterol levels.¹⁴ Reduced cholesterol absorption due to an increase in short chain fatty acid synthesis as a result of soluble fibre intake may further result in reduction in serum cholesterol levels.¹³ The major soluble fibres are β glucan (found in oats, barley and yeast), psyllium (found in husks of blonde psyllium seeds), pectin (found in fruit) and gums, mucilages algal polysaccharides and some hemicelluloses.^{13,14,19} Stanols and sterols, found mainly in fruits and vegetables have been found to lower cholesterol levels significantly.¹⁴ Insoluble fibre is associated with a decreased risk of CVD and slower progression of CVD in high risk individuals.¹³
Consume fish, especially oily fish at least twice a week ^{13,14}	<ul style="list-style-type: none"> Eating two servings per week of oily fish, high in the long chain omega 3 polyunsaturated fatty acids; EPA and DHA has been found to reduce the risk of CVD¹⁴ and reduce risk of sudden death and death from CAD in adults.^{13,14} Recommendations for the general public are to consume fish high in omega 3 fatty acids (salmon, mackerel, pilchards and sardines) at least twice a week, and for people with CVD to take in a combined daily dose of 1 g of EPA and DHA, preferably from fish, otherwise from a fish oil based supplement.¹⁴ Patients with hypertriglyceridaemia require 2–4 g EPA and DHA daily in order to effectively lower triglyceride levels.¹⁴
Limit the intake of saturated fat to 7% of energy, trans fats to 1% of energy, and cholesterol to 300 mg/day ^{13,14} <i>(note 300 mg/day for prevention/ < 200 mg/day when CVD is already present)</i>	<ul style="list-style-type: none"> Diets low in saturated and trans fatty acids and cholesterol, are all directly related to reducing CVD risk, largely through their reduction in LDL cholesterol levels.^{13,14} The most atherogenic saturated fatty acids, in order of potency, are myristic acid (found in butterfat, coconut and palm kernel oil), palmitic acid (found mainly in animal products) and lauric acid (found in palm kernel and coconut oil). Palmitic acid is the most prevalent hypercholesterolaemic saturated fatty acid in the American diet while palm kernel oil, coconut, palm oils, lard and butter are the most hypercholesterolaemic promoting.¹⁴ The use of these saturated fats should be avoided. Very low fat diets may increase fasting triglyceride levels and reduce HDL cholesterol levels.¹⁹ Recommendations: choose lean meats and vegetable alternatives, fat free or low fat dairy products and minimise intakes of partially hydrogenated fats.^{13,14} Dietary cholesterol raises total and LDL cholesterol, but to a lesser extent than saturated fats does.¹⁴ Dietary cholesterol intake should be reduced to < 300 mg/day when trying to prevent raised cholesterol levels; when blood lipids are raised or heart disease is already present, dietary cholesterol intake should be < 200 mg/day, with meat limited to 50 g/day and eggs limited to four or less per week.¹⁴ Major sources of saturated fatty acids are found in animal fats such as meat and dairy; major sources of dietary cholesterol are found in eggs, shellfish, full fat dairy and meat.¹³ Choosing lower fat products reduces overall intake of saturated fats and eliminating saturated fats is twice as effective in lowering serum cholesterol levels as increasing PUFAs.¹⁴ Although low fat diets are recommended, very low fat diets are contraindicated as they tend to decrease HDL and increase triglyceride levels.^{13,14,19}
Minimise intake of partially hydrogenated fats ^{13,14}	<ul style="list-style-type: none"> Major sources of trans fatty acids are partially hydrogenated fats which are used to prepare commercially fried and baked goods.^{13,14} Substituting these with fats made from vegetable oils (except for tropical oils which are high in saturated fat), will assist in lowering trans fatty acid intake, however, it is important to remember that some trans fatty acids occur naturally in meat and dairy products and heating vegetable fats to high temperatures will produce some trans fatty acids.¹³ The AHA recommends no more than 1% of total calories (about 1–3 g fat/day) from trans fatty acids as they result in increased LDL cholesterol levels and may lower HDL cholesterol.^{13,14} A few clinical trials have documented that replacing saturated fats with PUFAs reduces the risk of developing CHD and prospective observational studies have documented that diets rich in MUFAs are associated with a reduced risk of CHD.¹³ Replacing saturated fats with MUFAs has been found to lower serum cholesterol levels, LDL cholesterol levels and triglyceride levels to the same extent as PUFAs.¹⁴ The effect of MUFAs on HDL cholesterol depends on the total fat content of the diet.¹⁴ Although they provide benefits in CVD, the use of large amounts of fats must be used with caution due to their high energy density.¹⁴ Dietary recommendations suggest that moderate amounts of both carbohydrates (45–65% kcal) and fat (20–35% kcal), focusing on PUFAs and MUFAs, should be consumed¹⁴ as these ranges have been found to promote weight loss when applied within an appropriate calorie restriction, lower TG levels, maintain HDL cholesterol levels and maintain ideal body weight once achieved.^{14,19}

<p>Minimise your intake of beverages and foods with added sugars^{13,14}</p>	<ul style="list-style-type: none"> • Intake of large amounts of added sugars (sucrose, corn syrup and high fructose corn syrup) may result in an increased calorie intake and a decrease in overall nutrient quality of the diet. Calories consumed as liquid, such as in sweetened drinks, may result in lower satiety levels than when solid food is consumed, which may negatively influence attempts to maintain a healthy weight.^{13,14} • Food products which have contributed the most to this increase in simple sugar consumption include cakes, cookies, sweetened cereals and sugar, sweets and beverages.¹⁹ • Carbohydrate enriched diets have been shown to induce atherogenic dyslipidaemia, characterised by small LDL particles, high TG levels and low HDL cholesterol levels.¹⁹ There is a strong linear relationship between the presence of small, dense LDL particles and the percentage of calories from dietary carbohydrates.¹⁹ • The intake of high fructose corn syrup (found in soft drinks and most processed foods) may also increase postprandial TG levels, while chronic use of fructose as an added “free” sugar has been found to increase lipogenesis, resulting in increased TG levels compared to consuming similar amounts of glucose.¹⁹ While it is advised to eat fruit, it is not recommended to use fructose as a sugar substitute.
<p>Choose and prepare foods with little or no salt^{13,14}</p>	<ul style="list-style-type: none"> • Choose low- or reduced-sodium, or no-salt-added versions of foods and condiments when available. • Choose fresh, frozen, or canned (low-sodium or no-salt-added) vegetables. • Use fresh poultry, fish and lean meat, rather than canned, smoked or processed types. • Choose ready-to-eat breakfast cereals that are lower in sodium. • Limit cured foods (such as bacon and ham); foods packed in brine (such as pickles, pickled vegetables, olives and sauerkraut); and condiments (such as mustard, horseradish and tomato sauce). Limit even lower sodium versions of soy sauce and teriyaki sauce. Treat these condiments sparingly as you do table salt. • Cook rice and pasta without salt. Cut back on instant or flavoured rice, pasta and cereal mixes, which usually have added salt. • Choose convenience foods that are lower in sodium. Cut back on frozen dinners, mixed dishes such as pizza, packaged mixes, canned soups and salad dressings, which often have a lot of sodium. • Rinse canned foods such as tuna and canned beans, to remove some of the sodium. • Use spices instead of salt. In cooking and at the table, flavour foods with herbs, spices, lemon, lime, vinegar or salt-free seasoning blends. Start by cutting salt in half. <p>When eating out:</p> <ul style="list-style-type: none"> • Ask how foods are prepared. Ask that they are prepared without added salt, MSG or salt-containing ingredients. Most restaurants are willing to accommodate requests. • Know the terms that indicate high sodium content: pickled, cured, smoked, soy sauce, broth. • Move the salt shaker away. • Limit condiments, such as mustard, tomato sauce, pickles and sauces with salt-containing ingredients. • Choose fruit or vegetables instead of salty snack foods.
<p>When consuming alcohol do so in moderation^{13,14}</p>	<ul style="list-style-type: none"> • In many populations alcohol has been associated with reduced CVD events.¹⁴ However, due to the addictive properties of alcohol and possible serious adverse health and social consequences of alcohol consumption, such as hypertriglyceridaemia, liver damage, physical abuse and the risk of breast cancer and increased blood pressure,¹⁴ it is recommended that if alcoholic beverages are consumed, they are limited to no more than two drinks per day for men and one drink per day for women, ideally taken with a meal.^{13,14} One alcohol unit is equivalent to a 340 ml beer, 1 medium glass (150 ml) red or white wine, and 1 tot (30 ml) spirits.¹³
<p>Make wise choices when eating foods prepared outside the home^{13,14}</p>	<ul style="list-style-type: none"> • Foods consumed outside of the home, including at restaurants and supermarkets, take-away establishments, schools, daycare centres and other locations away from home are generally higher in energy density, saturated fats, trans fatty acids, cholesterol, added sugar and sodium and lower in fibre and micronutrients than home-made options.¹³ • Portion sizes of these foods are generally much bigger than we would consume at home.¹³ • The consumption of these away foods has significantly increased over the past 30 years and unfavourable health consequences have emerged as a result.¹³ A positive association between frequency of meal consumption at quick-serve restaurants and total energy intake, weight gain and insulin resistance has been reported and it is thus recommended that individuals choose leaner protein sources such as grilled fish and chicken over fattier cuts of meat, avoid consumption of high fat cream and cheese based sauces, choosing vegetables and salads over the starchier options of potato and pasta and controlling portions by choosing half portions or taking leftovers home as a take away.

fact, potential harm has been documented, including increased risk of lung cancer (in smokers) and increased heart failure risk, with beta-carotene supplementation.¹³ Antioxidant supplementation is therefore not recommended, however, the intake of a wide variety of foods rich in naturally occurring antioxidants and phytonutrients (plant-based foods) is recommended, including vegetables, fruits, plant-based oils and wholegrains.^{13,14}

Physical activity and dyslipidaemia

There are many scientific investigations and reviews that indicate that an acute bout of physical exercise, as well as regular exercise training generally has favourable effects on the blood lipid profile.²⁰⁻²⁴ The effects of plasma lipids and lipoproteins on CVD risk and the effects of a single exercise session and regular physical training on these parameters are summarised in Table VI.

Table VI: The effects of plasma lipids and lipoproteins on CVD risk and the effects of a single exercise session and regular physical training on these parameters²⁰

	Relationship to CVD risk	Effect of a single exercise session	Effects of regular physical training
Total cholesterol	Strong positive	No change	No change
Triglycerides	Somewhat positive	Decrease (14–50%)	Decrease (4–37%)
LDL	Strong positive	No change	Decrease or no change
VLDL	Somewhat positive	–	Decrease
IDL	Somewhat positive	–	Decrease or no change
HDL	Strong inverse	Increase (4–18%)	Increase
Lp(a)	Strong positive	No effect	No effect

The greatest benefits on the lipid profile following a single exercise session are achieved when the bout of exercise is performed at a high intensity (> 70 % of VO_{2max}), is of the endurance exercise type (running, cycling, swimming, rowing) and is performed for at least 45 min or longer. The positive changes in the lipid profile following exercise can last up to a few days, and it is linked to the duration of the exercise session (longer exercise bouts gives rise to longer lasting changes). Most clinical trials that show the benefits of regular training are evident after six months or longer.²⁵ In a recent meta-analysis of 29 randomised clinical trials progressive resistance training has been shown to also significantly reduce TC, TG, LDL-C, and increase HDL-C.²³ Therefore, progressive resistance training can be included in the management of patients with dyslipidaemia.

Practical exercise prescription for patients with dyslipidaemia

The following are practical exercise prescription guidelines for patients with an abnormal atherogenic lipid profile:

- Before starting an exercise programme, all patients should undergo a comprehensive medical screening, including an exercise electrocardiogram
- Exercise training should be performed on a regular basis (on most days of the week)
- Exercise training should consist of mainly endurance type training (running, cycling, swimming, circuit weight training, rowing) but can include progressive resistance training
- The duration of each exercise bout should be at least 45 min
- The intensity of exercise should be at about 80% of the age predicted [220 - age (years)] heart rate

This exercise prescription will result in positive changes on the lipid profile very soon after starting the programme. In addition to the positive effects of exercise on the atherogenic lipid profile, regular exercise training also has additional beneficial effects in these patients. As discussed in previous article in this series, regular exercise training also reduces blood pressure, independently reduces the risk for coronary heart disease, stroke and peripheral vascular disease, improves psychological well-being, and has positive effects on the vascular endothelium.

Lipid lowering medication in exercising individuals

Lipid lowering medication, in particular the statin drugs, can be associated with general musculoskeletal symptoms (including muscle weakness, arthralgia and myalgia in 2–10%)⁴ as well as a very small risk of myopathy or rhabdomyolysis (< 0.5%).^{1,4,11} This is important as exercise training may unmask the early development of these musculoskeletal complaints or myopathies. Specific risk factors for statin related myopathy are: age > 80 years, small body frame and frailty, chronic renal insufficiency, diabetes mellitus, multiple medications, peri-operative period, concomitant use of other medications (fibrates, niacin, cyclosporine, azole antifungals, macrolide antibiotics, HIV protease inhibitors, nefazodone, verapamil, amiodarone, large doses of grapefruit juice, alcohol).

Stress, stress management and the relaxation response for patients with dyslipidaemia

The dysregulation of the hypothalamic-pituitary-adrenal axis as a result of chronic stress, forms one of the pathophysiological mechanisms whereby psychological processes may affect physiological functioning and predispose to disease states. The sustained elevation of cortisol is fundamental in this regard. Cortisol replenishes energy stores in the short term as a result of the 'adrenaline rush' initiated by the autonomic nervous system by converting a variety of food sources into storage forms such as glycogen or fat. Excessive cortisol – a consequence of chronic stress – blocks the action of insulin on muscles to take up glucose, promotes the loss of protein from muscle and converts it to fat, and also enhances the storage of abdominal fat.²⁶

While the direct relationship between psychological distress and dyslipidaemia is controversial,²⁷ evidence does exist that there is a correlation between high work-related stress and an increase in both plasma TC²⁸ and the TC/HDL-C ratio.²⁹ What is unequivocal however is that behavioural processes largely explain the association between psychological distress and cardiovascular risk factors such as dyslipidaemia.³⁰

Apart from the overwhelming evidence that a sedentary lifestyle, smoking and poor food choices are modifiable risk factors in cardiovascular and cerebrovascular disease, it is self-evident that patients themselves need to modify their behaviour/s: passivity is simply ineffective in this regard no matter how much coercion, support or motivation comes from health practitioners.

The paucity of emphasis in the literature considering human motivation and psychological process in this area is striking. Ultimately without behavioural compliance the required modifications cannot be made and/or sustained and behaviour is the action initiated by the mind of an individual. While a small minority of patients will be highly self-motivated, it remains a challenge for the primary care doctor (as well as the specialist) to successfully address the majority who will struggle in this regard.

Primary care physicians can consider the following approaches to support and facilitate patient behavioural compliance:

- Regular counselling focusing on active listening, positive reinforcements of gain made and directly addressing resistance to compliance.

- Directly asking: What is getting in the way of your making behavioural changes? (Note that obstacles should be positioned within the mind of the patient rather than external factors.)
- Describing the research linking stress to illness to co-opt the cognitive and intellectual aspects of the mind. This is especially useful with male patients who tend to be more emotionally defended.
- Encouraging techniques which increase body awareness such as Yoga, Tai chi and Mindfulness. Individuals who are more in touch with their bodies self-regulate more effectively.
- Refer patients to group programmes so that peer support is optimised.
- Embodying the health changes that you are suggesting and/or prescribing.

Smoking cessation and dyslipidaemia

It is well established that the smoking habit increases both cardiovascular and non-cardiovascular morbidity and mortality. Therefore, no lifestyle modification programme, including one for dyslipidaemia, can be seen as complete without a smoking cessation component. The general practitioner has a critical role to play in advising and assisting smokers to quit by integrating the various aspects of nicotine dependence. Counselling and pharmacotherapeutic interventions for smoking cessation are among the most cost-effective clinical interventions and have already been discussed before in this series of articles.

Conclusion

In this article, the strong association between dyslipidaemia and increased risk of cardiovascular disease was emphasised. Furthermore, guidelines for the general management of dyslipidaemia were provided, and specific lifestyle interventions were reviewed in more detail. Practical dietary and exercise guidelines for lifestyle intervention in patients with dyslipidaemia were provided.

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