

EAT FATS SPARINGLY — IMPLICATIONS FOR HEALTH AND DISEASE

P Wolmarans, W Oosthuizen

Objective. To assess the scientific merit and feasibility of the food-based dietary guideline to 'eat fats sparingly'.

Scientific background. Recent evidence from the literature has shown that increased fat intake, especially intake of saturated fatty acids (SFAs), is positively associated with cardiovascular disease, obesity and certain types of cancer such as breast, colon and prostate cancer. The prevalence of these chronic diseases is high in South Africans. Many South Africans follow diets high in fat and SFAs, mainly provided by meat and meat products. The high consumption of hydrogenated fats, high in *trans* fatty acids, by a majority of South Africans is also a major concern. A very low-fat diet may have adverse effects on health. A moderate-fat diet, providing less than 30% of total energy, is therefore recommended. Foods contributing to fat intake and recommendations on how to control fat intake in diets of South Africans are discussed.

Conclusions. The guideline, 'eat fats sparingly' embodies the recommendation that fat should be eaten (addressing undernutrition), but that it should be used sparingly (addressing overnutrition).

For the prevention of chronic diseases of lifestyle South Africans should be encouraged to lower their fat intake from animal sources and non-dairy creamers, and to consume unsaturated tub margarine and oils instead of hydrogenated fats and animal fats. Fat in food preparation and as bread spread should be used sparingly.

In conclusion, there is convincing scientific evidence to support the guideline for fat intake. It is practical, realistic, culturally sensitive and sustainable, and in combination with the other food-based guidelines will contribute to better nutrition among South Africans.

The guideline, 'eat fats sparingly', aims to lower fat intakes, especially intake of saturated fatty acids (SFAs), among those who follow a typical Western diet high in fat, and to control fat intake in those following a diet low in fat. To meet the objective of the first part it is necessary to decrease the total intake of

foods from animal sources and to make lower fat choices when eating foods of animal origin.

Dietary fat plays an important role in the health and functioning of the human body but overconsumption is linked with coronary heart disease (CHD), obesity and cancers such as breast, colon and prostate cancer.^{1,3} The guideline to 'eat fats sparingly' is therefore primarily aimed at lowering the prevalence of these chronic diseases of lifestyle among South Africans. Cross-sectional studies^{4,6} have shown a high prevalence of overweight and obesity among South Africans and the majority of these studies have indicated a fat intake of more than 30% of energy. South Africans with a high prevalence of CHD⁷ also follow high-fat diets.⁸ CHD prevalence among black South Africans is still low;⁹ this may be partly ascribed to their diet which is still low in fat.⁸ However, Bourne and Steyn¹⁰ have shown that increased urban exposure has resulted in an increased intake of fat, from 15% energy in 1940 to about 30% energy in 1990. Continued urbanisation may therefore lead to an increase in the development of chronic diseases of lifestyle in those presently following low-fat diets.

In this paper the scientific evidence to support the guideline to 'eat fats sparingly' will be reviewed, the possible nutritional consequences and health implications of implementing the guideline will be discussed, and practical recommendations based on current food patterns will be given. Because this subject has been reviewed extensively by several authors, information from review articles is used in many instances.

GENERAL FUNCTIONS OF DIETARY FAT AND RECOMMENDED INTAKES

Dietary fats are classically defined as triglycerides (fats and oils), phospholipids and sterols (cholesterol). According to the degree of saturation, fatty acids can be classified as saturated (SFAs) from animal origin and β -sitosterol, campesterol and stigmasterol from plants, and monounsaturated (MUFA) and polyunsaturated (PUFA) fatty acids. PUFAs are further classified into omega 6 (n-6) and omega 3 (n-3) fatty acids.¹¹

Dietary fats provide the body with a continuous fuel supply, keep it warm, and protect it from mechanical shock. The human body can synthesise all but two fatty acids — the PUFAs, linoleic and alpha-linolenic acids, termed essential fatty acids (EFAs). These fatty acids are precursors for eicosanoids (hormone-like substances) that help regulate blood pressure, heart rate, blood clot formation, blood lipids and the immune response. They are also essential in the growth and development of infants. Docosahexaenoic acid (DHA), a derivative of linolenic acid, plays a major role in retinal function and brain development. In food, dietary fat is also a carrier of the fat-soluble vitamins A, D, E and K and many other compounds that give foods their flavour, tenderness and palatability.¹¹

The phospholipids and cholesterol contribute to the structure of cell membranes. Cholesterol is also used as a substrate for sex hormones (oestrogens, testosterone and progesterone), bile acids, and vitamin D, and it is a major component of brain and nerve cells.¹⁶

Dietary fat therefore plays a critical role in the health and functioning of the human body. Overconsumption of dietary fat has, however, been implicated in the aetiology of cardiovascular disease (CVD), certain types of cancer and obesity.¹⁶ These detrimental effects of dietary fat led to the formulation of the dietary guideline proposed by many national and international scientific bodies, namely that intake of dietary fat should be less than 30% of total energy (%E) for individuals over the age of 2 years.¹⁶ Further dietary recommendations for fat intake in adults, specifically to reduce the risk of chronic diseases, suggest SFAs of < 10%E, MUFAs of > 10%E, PUFAs of < 10%E and dietary cholesterol of < 300 mg/day.¹⁶ At least 15%E should come from fat, while women of reproductive age need at least 20%E from fat.¹⁶ Children up to the age of 2 years need 30 - 40%E from fat.¹⁶ At least 1 - 3%E should be contributed by linoleic acid,¹⁶ but desirable levels are between 4 and 10% of energy.¹⁶ In adults the requirement for linoleic acid will be met by the inclusion of 15 g of sunflower oil or 20 g of margarine high in PUFAs. It is recommended that the ratio of linoleic acid to alpha-linolenic acid should be between 5:1 and 10:1.¹⁶

FAT INTAKE OF SOUTH AFRICANS

A review of the nutritional status of South Africans from 1975 to 1996¹⁷ showed that total fat intake (Fig. 1) in white, coloured and Indian South Africans exceeded the recommended 30%E. While rural blacks had low fat intakes, urban blacks were found to be following diets much higher in fat than the rural diet, although less than 30%E. Eleven to 16-year-old boys and girls of all ethnic groups were noted to be following diets in which fat contributed 30%E or more. Intake of SFAs by white,

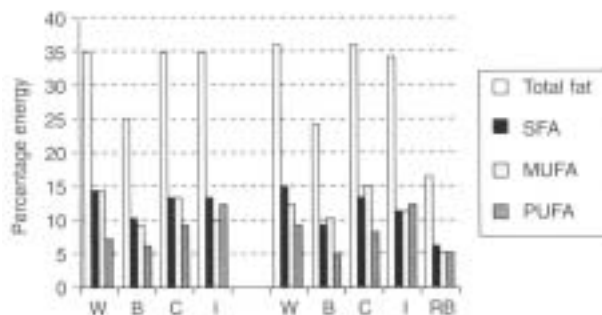


Fig. 1. Percentage of total energy provided by fat intake in the diets of adult South Africans aged 25 - 65 years* (W = whites; B = blacks; C = coloureds; I = Indians; RB = rural blacks; SFA = saturated fatty acids; MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids).

coloured and Indian South Africans was high and PUFA and MUFA intakes of white and coloured South Africans were in accordance with the recommendations. The Indian diet provided too much PUFA at the expense of MUFA intake, indicating the liberal use of PUFA-rich plant oils in cooking of food.

A meta-analysis of cholesterol intakes by South Africans, measured using the 24-hour recall method, showed that except for white and coloured adult men, all groups had cholesterol intakes that fell within the dietary guidelines.¹⁸ Studies that used other dietary intake methods found high intakes among Indian and coloured men and white and coloured women.¹⁸

FAT INTAKE AND CHRONIC DISEASE

Coronary heart disease (CHD)

CHD is one of the leading causes of mortality and morbidity in South Africa.¹⁹ CHD mortality rates are highest among Asians, whites and coloureds.¹⁹

Total fat intake

A meta-analysis by Hooper *et al.*²⁰ of randomised controlled intervention trials of at least 6 months' duration showed that a reduction or modification (where a proportion of saturated fat is replaced with unsaturated fats) of dietary fat intake reduced the incidence of cardiovascular events. The protective effect was seen almost exclusively in those who continued to modify their diet over a period of at least 2 years.

Most of the studies²⁰ that examined the effect of dietary fat intake and prevention of CHD focused primarily on fat intake and blood lipid levels. Elevated serum total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG) and decreased high-density lipoprotein cholesterol (HDL-C) levels are well-recognised risk factors for CHD.²⁰

In South Africa four large epidemiological studies were undertaken to study the relationship between dietary intake and risk factors for CHD. These studies were the Risk Factors for Coronary Heart Disease in the Black Population of the Cape Peninsula (BRISK) study,²¹ the study of Indian South Africans,²² the Coronary Risk Factors in the Coloured population of the Cape Peninsula (CRISIC) study of South Africans of African-European-Malay descent,²³ and the Coronary Risk Factor Intervention Study (CORIS) of white South Africans.²⁴ These studies showed that increased fat intake was associated with increased serum cholesterol levels (Fig. 2). Only black South Africans were found to be consuming diets with a fat content of less than 30%E and they were also found to have low serum cholesterol levels.²¹

Different levels of total fat intake in the diet may have different effects on lipid levels. A very low-fat diet, where fat is replaced with carbohydrates, reduces LDL-C but in addition

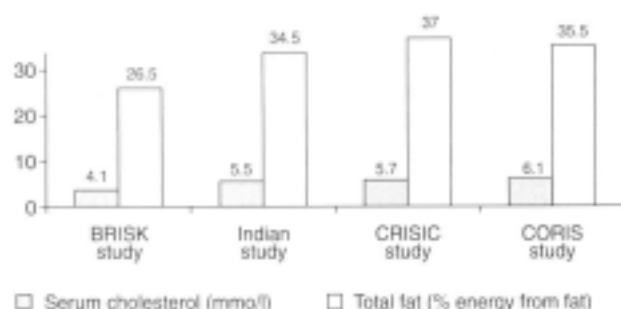


Fig. 2. Mean fat intake and mean serum cholesterol levels of participants in four large epidemiological studies conducted in South Africa (BRISK study,¹ Indian study,² CRISIC study,³ CORIS study⁴).

also reduces HDL-C, increases TG levels and small density LDL particles. This pattern is called the atherogenic lipoprotein phenotype and raises the risk for CHD.¹⁰ When very low-density lipoprotein (VLDL) concentrations are elevated TGs are transferred from VLDL to LDL and HDL particles in exchange for cholesteryl esters. TGs transferred to LDL and HDL are susceptible to hydrolysis by hepatic lipase which reduces the size of these lipoproteins resulting in more atherogenic LDL particles and reduced HDL particles.¹⁰ Table I summarises the physiological and biochemical effects of different levels of fat intake on health outcomes.¹¹

Compared with a low-fat diet, a moderate-fat diet (30%E), low in SFAs (< 10%E) and high in either MUFA or PUFA only lowered LDL-C levels and in some instances also increased

HDL-C levels.¹¹ A moderate-fat diet may not only have biochemical advantages over a very low-fat diet, but may be more palatable and may thereby increase compliance.

High-fat diets increase LDL-C and TG levels as well as HDL-C levels.¹¹

The effects on lipid levels differ with the different classes of fatty acids in the diet. The balance between these fatty acids is therefore important.

Type of fat in the diet

Table II summarises the effects of the different classes of fatty acids in food on the lipid profiles of humans. The fatty acid content of some fats and oils are summarised in Fig. 3.

SFAs: The SFAs with lauric (C12:0), myristic (C14:0) and palmitic (C16:0) acids are hypercholesterolaemic, whereas stearic acid (C18:0) has little effect on cholesterol levels.¹² The conversion of stearic acid to oleic acid (C18:1) could explain why stearic acid does not increase plasma cholesterol levels.¹² Dietary sources of these cholesterol-raising fatty acids include animal products (such as butter, beef tallow, mutton, lard and chicken), and vegetable fats (such as coconut oil, palm kernel oil and palm oil) (Fig. 3), coffee creamers and dairy blends. Palm oil has, however, been shown in some studies to have a neutral and even cholesterol-lowering effect, probably because of its high tocotrienol and oleic acid content.¹³

Trans fatty acids: Several epidemiological investigations indicated that trans fatty acids, found in hydrogenated brick

Table I. Different levels of total fat intake and the effects on physiological and biochemical variables and possible health outcomes/risk¹¹

Level of fat intake	Physiological/biochemical effects	Health outcomes/risk
Low-fat diet (10%E)	↓LDL-C; ↓HDL-C; ↑TG Insulin resistance (glucose intolerance)	Delays development of atherosclerosis ↓CHD Atherogenic lipoprotein phenotype* Hypertension Diabetes mellitus ↓Cancer risk
Moderate-fat diet (30%E, low in SFA < 10%, MUFA/PUFA instead of SFA)	↓LDL-C, ↑HDL-C, ↓TG Lower body mass	Delays development of atherosclerosis ↓CHD ↓Cancer ↓Diabetes mellitus ↓Hypertension
High-fat diet (> 30%E)	↑LDL-C, ↑HDL-C, ↑TG Overweight and obesity	Atherosclerosis CHD Hypertension Some types of cancer (e.g. colon, breast and prostate cancer) Diabetes mellitus Insulin resistance syndrome (Syndrome X)

* Atherogenic lipoprotein phenotype: ↓HDL-C, ↑TG, small LDL particles.

LDL-C - low-density lipoprotein cholesterol; HDL-C - high-density lipoprotein cholesterol; TG - triglycerides; CHD - coronary heart disease; SFA - saturated fatty acid; MUFA - monounsaturated fatty acid; PUFA - polyunsaturated fatty acid.

Table II. Effects of the different classes of fatty acids on lipid profiles^a

Fatty acids	TC	LDL-C	HDL-C	TG
Saturated fatty acids	↑↑↑	↑↑	↑	↑
Trans fatty acids	↑↑	↑	↓	↑
Monounsaturated fatty acids	↓	↓	↑	↓
Polyunsaturated fatty acids	↓↓	↓	↓	↓

TC = total cholesterol; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; TG = triglycerides.

margarine and hydrogenated plant and fish oils used in commercial food processing, may increase the risk for CHD.¹⁰ Although to a lesser extent than SFAs, *trans* fatty acids raise TC, LDL-C and TG levels and decrease HDL-C levels.¹¹ *Trans* fatty acids are also one of the rare dietary factors that may increase lipoprotein (a) (Lp(a)) levels, although this is not a consistent finding.¹² It may be useful to consider the sum of SFAs and *trans* fatty acids in evaluating the health effect of fats and oils.¹³

MUFA and PUFA: MUFA has a neutral effect on TC and LDL-C concentrations when compared with carbohydrate, and a cholesterol-lowering effect when compared with SFAs. In some studies MUFA and PUFA seem to be equally effective in lowering TC and LDL-C levels, whereas in other studies MUFA was found to be less effective than PUFA.¹⁴ MUFAs, like SFAs, raise HDL-C and are not as easily oxidised as PUFAs.¹⁵ Calculations from a meta-analysis of 27 studies showed that isocaloric replacement of SFAs in the diet with PUFAs would result in a statistically, but perhaps not biologically, significant decrease in HDL-C levels.¹⁶ There are, however, indications that PUFA does not lower HDL-C if the intakes are less than 10 - 13%E.¹⁷ Lower TG levels were observed on a high-fat high MUFA diet compared with a low-fat high carbohydrate diet.¹⁸ Oleic acid (C18:1n-9) is the best known MUFA. Although all foods contain MUFA, rich sources of oleic acid are olive and canola oils (Fig. 3). Avocados and nuts also have high MUFA contents.

PUFAs can be divided into n-6 and n-3 PUFAs. Linoleic acid (C18:2n-6) is the parent fatty acid of the n-6 PUFAs and alpha-linolenic acid (C18:3n-3) of the n-3 PUFAs. Vegetable oils such as sunflower, corn and safflower oils are rich sources of the n-6 PUFAs (Fig. 3). In the Western diet the n-3 PUFAs mainly come from fatty fish (such as mackerel, pilchards, salmon, herring and sardines) and plant oils such as canola and soybean oil (Fig. 3). Flaxseed oil has a high n-3 PUFA content,¹⁹ but it is not commonly used in South Africa (Fig. 3). The role of the n-6 and n-3 PUFAs in the prevention of CHD has been investigated for several decades.²⁰ While the main effects of n-6 PUFAs are anti-atherogenic, those of the n-3 PUFAs are antithrombotic because they inhibit platelet aggregation.²¹ A salient but consistent characteristic of n-3 PUFAs is also to lower TG levels in normo- as well as hyperlipidaemic subjects.²² Although high intakes of

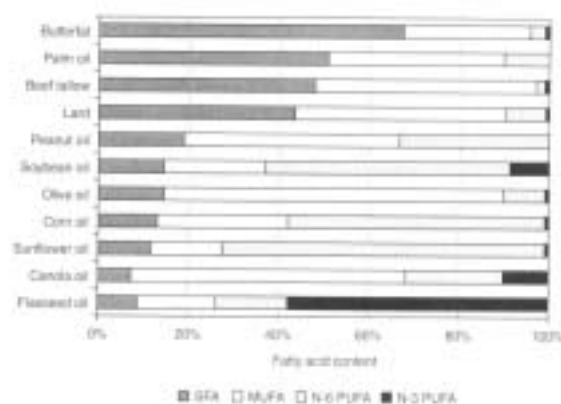


Fig. 3. Fatty acid content of some fats and oils (adapted from Vaisey-Genser and Morris²³) (SFA = saturated fatty acids; MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids).

n-3 PUFAs also lower LDL-C, a tendency of n-3 PUFAs to increase LDL-C in hyperlipidaemic patients has been observed.²⁴

Since there is at present no country with traditionally very high PUFA intakes, only time will tell whether a high intake of PUFA has health implications.²⁵ There are concerns that a high intake of PUFAs may promote cancer or lead to the oxidative modification of lipoproteins that probably play a role in atherosclerosis.²⁶ Oxidation of PUFAs may be prevented with a vitamin E-to-PUFA ratio of > 0.6 mg tocopherol equivalents per gram of PUFA.²⁷

Cholesterol intake can also increase TC and LDL-C levels in the serum of susceptible individuals, but the response is less than that induced by changes in the fatty acid composition of the diet.²⁸

Obesity

Obesity (body mass index > 30 kg/m²) is common among South African women. The highest prevalence of 59.4% was observed among black women aged 45 - 54 years.²⁹ Coloured women of the same age have a prevalence of 42.6%, Indian women of 40% and white women of 23.8%.³⁰ The prevalence of obesity may rise even further with continued urbanisation. Other chronic diseases associated with obesity, namely hypertension and diabetes mellitus, are also major public health problems in South Africa.³¹

Although the evidence from ecological studies is less convincing, most cross-sectional studies, and experimental studies on animals and humans have shown a positive association between the percentage of energy consumed from fat, and obesity.³² Obesity is, however, a multifactorial condition and although dietary fat plays an important role in its aetiology, it is probably not the primary cause.³³ Decreased physical activity also plays a very important role in the causation of obesity.³⁴

Dietary fat may contribute to obesity through several mechanisms. Firstly, obesity is ultimately an issue of positive energy imbalance.¹² Dietary fat may contribute to this imbalance because of its high energy density.¹² Secondly, fat ingestion is subjectively less satiating than carbohydrate and therefore could promote overeating. Obese persons also prefer high-fat foods and therefore overeat. Thirdly, excess dietary fat does not acutely increase fat oxidation as does carbohydrate and protein. The capacity for fat storage in adipose tissue in the body is virtually unlimited and very efficient.³⁸ Obese individuals may also be more susceptible to dietary fat because of defects in the ability to oxidise dietary fat.^{12,39}

A very low-fat diet, high in carbohydrate, will, however, not necessarily prevent the development of obesity^{36,37} and may even have other adverse effects (Table I). A study of Finnish children suggested that fat restriction may suppress height velocity.⁴⁰ The question of at what age to introduce the guideline to 'eat fats sparingly' is therefore very important in South Africa where the prevalence of stunting is high in certain communities, especially those known to follow a diet low in fat.⁸

A moderate-fat diet in combination with the other guidelines, especially to 'be active', is therefore recommended for the treatment and prevention of obesity.

Cancer

Ecological data suggest that cancer risk is lower in populations with a fat intake of less than 30%E.³

Reports published by authoritative sources in the USA and by the World Health Organisation have concluded that dietary fat may influence the risk of certain types of cancer such as breast, colon and prostate cancer.³ The findings from these reports were consistent and were supported by findings of other studies not included in the abovementioned reports.³ To use controlled trials for the investigation of a relationship between fat intake and cancer is problematic. Therefore, the findings of a relationship between fat intake and cancer is primarily based on observational studies and supported by studies done on animals.³ It has therefore been recommended that health claims about fat and cancer should not be site-

specific, and that fat intake should thus not be linked with specific cancers.³

Very few studies have examined the effect of different types of fat on cancer risk and the results have been controversial. Meta-analyses of case-controlled and cohort studies have shown a positive association between SFA intake or intake of foods high in SFAs (meat, milk, cheese) and breast cancer.¹² Ecological studies have shown protective effects against colon cancer in populations consuming high-fat diets containing olive or fish oils compared with the promoting effect of animal fat.¹² Results from the EURAMIC study⁴¹ showed an inverse association between increased n-3 to n-6 fatty acid ratio and breast cancer.

Questions arise regarding whether the relationship between fat intake and cancer is a direct relationship or whether cancer is caused by a higher intake of energy, and obesity, which is usually associated with a higher fat intake. Cancer development is probably influenced by both.⁴² Results from the prospective Nurses' Health Survey, which studied women between 34 and 59 years of age, showed a positive association between fat intake and the risk of colon cancer after adjustment for energy intake.⁴³

FOODS THAT CONTRIBUTE TO FAT INTAKE IN SOUTH AFRICAN DIETS AND PRACTICAL RECOMMENDATIONS

The large epidemiological studies undertaken in South Africa found that the meat group (red meat, chicken, fish and meat products) was the main contributor of fat in the diet, followed by the fat group (butter, margarine, animal fat and oil).⁴⁴⁻⁴⁶ Unfortunately the South African Food Composition Database does not allow for separate analysis of fat used in food preparation. Therefore the possibility exists that the contribution of fats and oils to total fat intake could have been higher than reported in these studies.

The percentage of South Africans consuming food items contributing to fat intake, which fell within the top 10 foods consumed by participants in the CORIS (whites), CRISIC (coloureds)²¹ and BRISK (blacks)¹⁹ studies, is summarised in Table III. The foods were ranked in order of the percentage of

Table III. Foods, contributing to fat intake, which fell in the top ten foods eaten by most South Africans and the percentage of adults who consumed the foods (P Wolmarans — unpublished data)

CORIS (1983)* (N = 1 784)			CRISIC (1982)* (N = 976)			BRISK (1990)* (N = 983)		
Rank order	Food item	Consumers (%)	Rank order	Food item	Consumers (%)	Rank order	Food item	Consumers (%)
3	Milk — full-cream	78	3	Margarine — brick	64	3	Milk — full-cream	45
6	Margarine — brick	61	6	Milk — full-cream	57	8	Margarine-brick	40
7	Mutton	50	10	Beef/chicken	28			

*Dietary data collected by 24-hour recall.

subjects who consumed a specific food item on the day the 24-hour recall was done (P Wolmarans — unpublished data). Full-cream milk and brick margarine were among the top 10 foods in all the studies. There is, however, a marked difference between the studies with regard to the percentage of respondents who consumed these foods, for instance only 45% of blacks (BRISK study) consumed full-cream milk. Only in the white (CORIS study) and coloured (CRISIC study) groups did meat fall within the top 10 foods. Correspondingly, the total fat intake in these subjects^{21,22} was high compared with the intake of participants in the BRISK study.¹⁹

Milk intake

South Africans prefer full-cream milk (Table III). A national cross-sectional questionnaire survey undertaken in 1991 and involving 2 000 South Africans over 18 years old, showed that white and coloured South Africans consume more milk than their black and Indian South African counterparts.⁴⁷ This difference in dietary pattern needs to be taken into account when South Africans are advised to make lower fat dairy choices. There is a tendency to exclude milk from the diet when people are advised to make lower fat dairy choices and this should be avoided because there are already indications that South Africans do not consume enough milk.⁴⁷ Non-dairy creamers which are high in fat are often consumed instead of milk. A study of 11-year-old children showed that 6 - 18% of urban and 4 - 25% of rural children used non-dairy creamers.⁴⁸ Additional information supporting this guideline for fat intake should therefore advise against the use of non-dairy creamers as a substitute for milk intake, not only because this practice contributes to total fat intake, but also because these products contain plant oils high in cholesterol-raising SFAs.

Intake of fat spreads and oils

Table III shows that brick margarine, which has a high *trans* fatty acid content, is preferred by most South Africans. The national cross-sectional survey also showed that the majority of participants consumed brick margarine, with as many as 71% of coloured South Africans indicating that they used brick margarine.⁴⁷ Tub margarine was used by between 16 and 37% of South Africans who participated in this survey. In the Indian diet vegetables and other foods are often fried in fats and oils, especially vegetable oils, resulting in a high-fat diet with a PUFA/SFA ratio of 0.9 to 1.0.⁸ The guideline to 'eat fats sparingly' should therefore not only encourage those who consume a lot of fat and oils to eat less fat, it should also encourage the choice and consumption of unsaturated tub margarine and oils instead of hydrogenated fats and animal fats.

Meat intake

The meat group, which included red meat, chicken, fish and meat products, was found to be the main contributor of energy

and fat in the diets of the CORIS and CRISIC study populations.^{45,46} Although meat did not fall within the top 10 foods eaten by the BRISK population, it was the main contributor of fat to the diet.⁴⁴ Mutton, chicken, beef, fish and sausage appear in the top 20 list of foods consumed by the CORIS population, while fish, beef, chicken and mutton appeared in the list of the CRISIC population. In the BRISK study only chicken appeared in the top 20 list of foods eaten by this study population. This could explain, in part, the Western-type diet followed by white and coloured South Africans^{21,22} since the meat group is an important source of fat and SFAs in the diet. As shown earlier, white, coloured and Indian South Africans also had the highest intakes of SFAs (Fig. 1). The guideline to 'eat fats sparingly' will be especially applicable to South Africans with a preference for meat.^{44,46} Cutting down on the intake of visible fat from this component of the diet will make a major contribution to lowering total fat intake.

IMPACT ON OTHER NUTRIENTS OR FOOD PATTERNS AND HEALTH OUTCOMES

The current review shows that a balance between fat intake and other nutrients, especially carbohydrate intake, is important to ensure optimal nutrition and health. The lowering of fat in the diet may result in a shift towards a higher intake of carbohydrates and also result in a lower intake of fat-soluble vitamins. The higher intake of carbohydrates may affect insulin metabolism, raise TG and lower HDL-C levels (Table I).^{12,24} The lowering of fat intake may also not be sufficient to prevent obesity.^{36,37} These concerns may be addressed by eating a moderate-fat diet instead of a low-fat diet and by implementing the other dietary guidelines such as: (i) be active; (ii) make starchy foods the basis of most meals; (iii) eat plenty of vegetables and fruits; and (iv) eat dry beans, peas, lentils and soya regularly. These guidelines will assist in the prevention of obesity and will result in higher intakes of dietary fibre that will counteract the negative effects on insulin resistance, TG and HDL-C²⁵ and prevent decreased intakes of some vitamins.

SUMMARY AND CONCLUSION

There is enough evidence from the literature to conclude that dietary fat plays a role in the development of CHD, obesity and cancer. The guideline to 'eat fats sparingly' is therefore based on sound scientific knowledge. The literature also clearly indicates that, especially in terms of the development of CHD, the type of fat eaten plays an important role. Not only should those South Africans who consume a high-fat diet be encouraged to lower their fat intake, but they should also be encouraged to make the right choices in terms of the type of fat they consume. A moderate-fat diet with a low SFA and high MUFA content was found to have the most beneficial effect on lipid profiles. South Africans who followed a Western diet had a high intake of SFAs, probably related to their high intake of

What does it mean in practice to 'eat fats sparingly'?

Only small changes in food intake are required to meet the 'eat fats sparingly' guideline. The following can serve as an example. Say the energy intake of a person is 8 000 kJ/day and 37% of energy comes from fat, this means a fat intake of 80 g/day. To lower fat intake to 30% of energy, this person needs to lower fat intake to 65 g/day, thus fat intake needs to be lowered by 15 g. About 3 teaspoons (15 ml) of fat or oil contain 15 g fat.

Note: 1 gram of fat = 37 kJ.

The following foods serve as examples of how fat intake can be decreased by following the guideline 'eat fats sparingly':

Choose	Fat (g) ⁴⁹	Instead of	Save	
			Fat (g) ⁴⁹	Fat (g)
250 ml low-fat milk*	4.8	250 ml full-cream milk	8.3	3.5
100 g lean beef [†]	7.5	100 g beef with fat	15.3	7.8
1 medium boiled potato [‡]	0.1	1 medium potato, chips, fried	16.1	16.0
Margarine, thinly spread (5 g) [§]	4.1	Margarine, thickly spread (10 g)	8.2	4.1
1 medium apple (150 g) [¶]	0.5	2 commercial cookies (20 g)	2.7	2.2

*Lower fat food choices.

[†]Remove visible fat.

[‡]Use less fat in food preparation.

[§]Use less fat on bread.

[¶]Eat fruit instead of snack foods with fat.

meat. They should be encouraged to reduce the intake of meat and visible fat from meat. MUFA intakes of > 10%E have been reported for those following a Western diet, but Indian South Africans had higher PUFA intakes at the expense of MUFA intake. Oils with a high MUFA content are expensive in South Africa and are at present not a practical option when it comes to the recommendation to increase MUFA intake. The fat intake of the black population is still prudent, but continued urbanisation might cause increased fat intake in future. The high intake of hydrogenated fats and oils, high in *trans* fatty acids, by the majority of South Africans in the form of bread spread and in food preparation is also of concern. South Africans should be advised to use unsaturated tub margarines and oils instead. The guideline to 'eat fats sparingly' does not address the question of the type of fat in the diet properly, but by lowering the total fat intake, SFAs and *trans* fatty acids intake will probably also be lowered.

Eating fats sparingly does not mean a no-fat diet. From the current review it was clear that a very low-fat diet has other adverse effects. In addition to contributing to undernutrition and stunted growth in infants and children it could also result in the atherogenic lipoprotein phenotype, increasing the risk for CHD in adults. The very low-fat diet consumed by South Africans living in rural areas had probably contributed to the high prevalence of stunting in children. Terms such as 'limit' and 'avoid' were not used in the guideline. The recommendation is that fat should be eaten, but that it should be eaten sparingly.

In conclusion, there is convincing evidence to support this guideline. It is practical, realistic, and its application can be potentially culturally sensitive and sustainable because of the variety of food choices that can be made to either increase or decrease fat intake. In combination with the other guidelines, it will contribute to improved nutrition among South Africans.

References

- Keys A. *Coronary Heart Disease in Seven Countries*. American Heart Association Monograph No. 29. New York:the American Heart Association, 1970: 1-162 - 1-183.
- Bray GA, Popkin BM. Dietary fat intake does affect obesity. *Am J Clin Nutr* 1998; **68**: 1157-1173.
- Lewis CJ, Yetley EA. Health claims and observational human data: relation between dietary fat and cancer. *Am J Clin Nutr* 1999; **69**: suppl. 1357S-1364S.
- Rossouw JE, Du Plessis JP, Benadé AIS, et al. Coronary risk factor screening in three rural communities. The CORIS baseline study. *S Afr Med J* 1983; **64**: 430-436.
- Steyn K, Jooste PL, Langenhoven ML, et al. Coronary risk factors in the coloured population of the Cape Peninsula. *S Afr Med J* 1985; **67**: 619-625.
- Seedat YK, Mayet FGH, Khan S, Somers SR, Joubert G. Risk factors for coronary heart disease in the Indians of Durban. *S Afr Med J* 1990; **78**: 447-454.
- Bradshaw D, Bourne D, Schneider M, Sayed R. Mortality patterns of chronic diseases of lifestyle in South Africa. In: Fourie J, Steyn K, eds. *Chronic Diseases of Lifestyle in South Africa*. Cape Town:Medical Research Council, 1995: 5-36.
- Vorster HH, Oosthuizen W, Jerling JC, Veldman FJ, Burger HM. *The Nutritional Status of South Africans. A Review of the Literature, Narrative and Tables*. Durban:Health Systems Trust, 1997: 1-48, 1-122.
- Walker ARP, Sareli P. Coronary heart disease: outlook for Africa. *J R Soc Med* 1997; **90**: 23-27.
- Bourne LT, Steyn K. Rural/urban nutrition-related differentials among adult population groups in South Africa, with special emphasis on the black population. *S Afr J Clin Nutr* 2000; **13**: suppl 1, 523-528.
- Whitney EN, Cataldo CB, Rolfe SR. *Understanding Normal and Clinical Nutrition*. 5th ed. Belmont: West/Wadsworth, 1998: 1-963.
- Lichtenstein AH, Kennedy E, Barrier P, et al. Dietary fat consumption and health. *Nutr Rev* 1998; **56**: S3-S28.
- Mahan LK, Escott-Stump S. *Food, Nutrition and Diet Therapy*. 9th ed. Philadelphia: WB Saunders, 1996: 1-1194.
- WHO and FAO Joint Consultation. Fats and oils in human nutrition. *Nutr Rev* 1995; **53**: 202-205.
- FAO and WHO. *Fats and Oils in Human Nutrition*. Report of a Joint Expert Consultation. Rome: Food and Agriculture Organisation of the United Nations, 1994.

16. Hooper L, Summerbell CD, Higgins JPT, *et al*. Reduced or modified dietary fat for prevention of cardiovascular disease. *The Cochrane Library* 2000; 2: 1-16.
17. Shrapnel WS, Clavert GD, Nestel PJ, Truswell AS. Diet and coronary heart disease. *Med J Aust* 1992; 156: suppl., S9-S16.
18. Steyn K, Jooste PL, Bourne L, *et al*. Risk factors for coronary heart disease in the black population of the Cape Peninsula. The BRISK study. *S Afr Med J* 1991; 79: 480-485.
19. Bourne LT, Langenhoven ML, Steyn K, Jooste PL, Laubscher JA, van der Vyver E. Nutrient intake in the urban African population of the Cape Peninsula, South Africa. The Brisk study. *Cent Afr JMed* 1993; 39: 238-247.
20. Wolmarans P, Seedat YK, Mayet FGH, Joubert G, Wentzel E. Dietary intake of Indians living in the metropolitan area of Durban. *Public Health Nutrition* 1999; 2: 55-60.
21. Langenhoven ML, Wolmarans P, Groenewald G, Richter MIC, van Eck M. Nutrient intakes and food and meal patterns in three South African population groups. *Frontiers of Gastrointestinal Research* 1988; 14: 41-48.
22. Wolmarans P, Langenhoven ML, Benadé AIS, Swanepoel ASP, Kotze TJvW, Rossouw JE. Intake of macronutrients and their relationship with total cholesterol and high-density lipoprotein cholesterol. The Coronary Risk Factor Study 1979. *S Afr Med J* 1988; 73: 12-15.
23. Lechleitner M, Hoppichler F, Föger B, Patsch JR. Low-density lipoprotein of the postprandial state induce cellular cholesteryl ester accumulation in macrophages. *Arterioscler Thromb* 1994; 14: 1799-1807.
24. Grimm JJ. Interaction of physical activity and diet: implications for insulin-glucose dynamics. *Public Health Nutrition* 1999; 2(3A): 363-368.
25. Wolmarans P. The effect of lean red meat or chicken and fish, in a prudent diet, on the lipid metabolism of hypercholesterolaemic subjects. PhD thesis, Potchefstroom University, 1997: 1-230.
26. Narasinga Rao BS. Palm oil as an edible oil in India and its role in meeting the nutritional needs of its population. *Nutrition Research* 1992; 12: suppl 1, S3-S21.
27. Ascherio A, Willett WC. Health effects of trans fatty acids. *Am J Clin Nutr* 1997; 66: suppl. 1006S-1010S.
28. Katan MB. Commentary on the supplement trans fatty acids and coronary heart disease risk. *Am J Clin Nutr* 1995; 62: 518-519.
29. Denke MA. Serum lipid concentrations in humans. *Am J Clin Nutr* 1995; 62: suppl 1, 693S-700S.
30. FAO and WHO Consultation. *Preparation and Use of Food-based Dietary Guidelines*. Nutrition Programme WHO/NUT 96.6. Geneva:WHO, 1996: 1-99.
31. Mensink RP, Katan MB. Effect of dietary fatty acids on serum lipids and lipoproteins. A meta-analysis of 27 trials. *Arterioscler Thromb* 1992; 12: 911-919.
32. Vaisey-Genser M, Morris DH. *Flaxseed: Health, Nutrition and Functionality*. Winnipeg:Flax Council of Canada, 1997: 1-96.
33. Ulbricht TLV, Southgate DAT. Coronary heart disease: seven dietary factors. *Lancet* 1991; 338: 985-992.
34. American Heart Association. Rationale of the diet-heart statement of the American Heart Association. Report of the Nutrition Committee. *Circulation* 1993; 88: 3008-3029.
35. Lissner L, Heitmann BL. Dietary fat and obesity: evidence from epidemiology. *Eur J Clin Nutr* 1995; 49: 79-90.
36. Willett WC. Is dietary fat a major determinant of body fat? *Am J Clin Nutr* 1998; 67: suppl. 556S-562S.
37. Grundy SM. Multifactorial causation of obesity: implications for prevention. *Am J Clin Nutr* 1998; 67: suppl. 563S-572S.
38. WHO Consultation on Obesity. *Obesity: Preventing and Managing the Global Epidemic*. Geneva: WHO, 1998: WHO/NUT/NCD:98.1: 1-276.
39. Raben A, Astrup A. Manipulating carbohydrate content and sources in obesity prone subjects: effect on energy expenditure and macronutrient balance. *Int J Obes Relat Metab Disord* 1996; 20: suppl 2, S24-S30.
40. Vartiainen E, Puska P, Pietinen P, Nissinen A, Leino U, Uusitalo U. Effects of dietary fat modifications on serum lipids and blood pressure in children. *Acta Paediatr Scand* 1986; 75: 396-401.
41. Simonsen N, Van't Veer P, Strain JJ, *et al*. Adipose tissue omega-3 and omega-6 fatty acid content and breast cancer in the EURAMIC study. *Am J Epidemiol* 1998; 147: 342-352.
42. Kuller LH. Dietary fat and chronic diseases: Epidemiologic overview. *J Am Diet Assoc* 1997; 97: S9-S15.
43. Willett WC, Stampfer MJ, Colditz GA, Rosner BA, Speizer FE. Relation of meat, fat, and fibre intake to the risk of colon cancer in a prospective study among women. *N Engl JMed* 1990; 323: 1664-1672.
44. Bourne LT, Langenhoven ML, Steyn K, Jooste PL, Nesamvuni AE, Laubscher JA. The food and meal pattern in the urban African population of the Cape Peninsula, South Africa: the BRISK Study. *Cent Afr JMed* 1994; 40: 140-148.
45. Wolmarans P, Langenhoven ML, van Eck M, Swanepoel ASP. The contribution of different food groups to the energy, fat and fibre intake of the Coronary Risk Factor Study (CORIS) population. *S Afr Med J* 1989; 75: 167-171.
46. Langenhoven ML, Steyn K, van Eck M. The food and meal pattern in the Cape Peninsula coloured population. *Ecology of Food and Nutrition* 1988; 22: 107-116.
47. Langenhoven ML, Wolmarans P, Jooste PL, Dhansay MA, Benadé AIS. Food consumption profile of the South African adult population. *S Afr JScience* 1995; 91: 523-528.
48. Steyn NP, Wicht CL, Rossouw JE, Kotze TJvW, Laubscher R. The eating pattern of adolescents in the Western Cape. *SAJ Food Science Nutr* 1990; 2: 23-27.
49. Langenhoven ML, Kruger M, Gouws E, Faber M. *MRC Food Composition Tables*. 3rd ed. Parow: South African Medical Research Council, 1991.

Nutrition and Dietetics Unit, Department of Medicine, University of Cape Town
K E Charlton, MPhil, MSc, PG Dip Diet
 Research Programme for Nutritional Intervention, Medical Research Council,
 Tygerberg
P LJooste, PhD