

# Fats and oils — towards more specific quantitative and qualitative guidelines for South Africans?

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This paper briefly reviews the role of dietary fats (lipids) in human nutrition, summarises the contribution to the symposium on fats and oils during the 2000 South African Nutrition Congress, and examines the present recommendations and controversies regarding fat intake, in order to examine whether in a developing country such as South Africa, dietary fat recommendations to consumers should be more explicit, specific and detailed. It is concluded that to reach 'optimal' intakes, some groups in South Africa (e.g. rural blacks) should increase intakes, while those at risk for cardiovascular disease should lower intakes. It is suggested that all South Africans over the age of 2 years should aim for an intake in which fat provides approximately 30% of total energy. To meet the guidelines that more omega-3 ( $\omega$ -3), proportionally more monounsaturated fatty acids and less *trans* and saturated fatty acids should be eaten, alternative sources of and for these fatty acids in the South African diet should be promoted. Both consumers and the food industry should be targeted with information and advice to make healthier but affordable choices possible. Only then will dietary advice on the quality of fat (less saturated and *trans* fatty acids and more monounsaturated and ( $\omega$ 3 fatty acids) be practical and meaningful.

South Africa is a land of contrasts. It includes industrialised cities with an urban Western culture, and remote rural areas where many people still follow traditional African lifestyles. Of the total population — just over 43 million in 2000 — 77.6% were blacks, 2.5% Asians (Indian), 8.7% coloureds (mixed origin) and 19.4% whites (European).<sup>1</sup>

A working group, representative of nutrition professionals in South Africa, recently formulated a basic set of food-based dietary guidelines (FBDG) that can be used in nutrition education and recommendations to consumers of all ethnic groups.<sup>2</sup> One of these guidelines is to 'eat fats sparingly'. In their excellent technical support paper motivating this guideline, Wolmarans and Oosthuizen<sup>3</sup> show how the guideline embodies the principles that fat should be eaten because it provides essential, important and needed fatty acids and energy, but that it should be eaten 'sparingly' because overconsumption is associated with obesity and other non-communicable chronic diseases of lifestyle (NCDs). The emphasis in their paper is on 'moderate' intake.

During the 2000 biannual Nutrition Congress in Durban, the South African branch of the International Life Science Institute (ILSI-SA) initiated and sponsored a symposium entitled 'Fats and oils — forbidden foods but important nutrients'. The title of the symposium also reflects the essential nature of fats and oils (and their constituent fatty acids) as well as the perception that dietary intakes should be limited. The purpose of this symposium was to review the global situation regarding fat and oil availability and dietary recommendations, and to create a forum for South African researchers<sup>4-8</sup> to present and discuss their research on fats and oils. The need for such a symposium was partly motivated by the international debate regarding optimal fat and oil intakes. This debate is based on queries surrounding the quality of the scientific evidence, which originally led to the formulation of guidelines to limit fat intake.<sup>9</sup>

A 'typical' Western diet, associated with an increased risk of NCDs, may provide between 35% and 45% of energy as fat, while traditional diets associated with a low risk of NCDs may contain as little as 15% or less

energy as fat.<sup>10</sup> The South African recommendation<sup>3</sup> for consumers over 5 years of age is that less than 30% of total dietary energy should be provided by fat with not more than 10% of energy coming from saturated fatty acids (SATFAs). The Joint Expert Consultation of the Food and Agricultural Organisation/World Health Organisation (FAO/WHO)<sup>10</sup> recommends that until 2 years of age, a child's diet should contain 30 - 40% of energy as fat (breast-milk contains 50 - 60% energy as fat). The same source also mentions that most adults need at least 15% of their energy as fat, women of childbearing age at least 20%, active individuals not more than 35% and sedentary persons not more than 30%, provided that not more than 10% of total energy comes from SATFAs. Both these sources<sup>3,10</sup> emphasise the focus on beneficial effects of the omega-3 ( $\omega$ -3) and monounsaturated fatty acids (MUFAs) and warn against high intakes of trans fatty acids. The questions that arise from these recommendations are firstly, is there an optimal amount or range (quantity) of total fat (TF) intake (between 15% and 35%); secondly, what is the ideal fatty acid composition of the diet; and thirdly, how specific should the advice to consumers be regarding fat quantity and quality?

The objectives of this paper are: (i) to review very briefly the role of fats and oils in human nutrition, health and disease; (ii) to analyse the reasons for the present controversies surrounding dietary fat recommendations; (iii) to summarise the contributions during the ILSI-sponsored symposium in context of the South African situation and recommendations, and how this information should be applied in advice to consumers, health care professionals and the food industry; and (iv) based on the above, to answer the question whether we know what the optimal ranges of amounts and types of fats and oils in the diet should be. Can and should we be more specific in our fat recommendations to consumers?

## The role of fats and oils in human nutrition

Dietary fats and oils (lipids) consist of triglycerides (esters of glycerol with three fatty acids) and minor amounts of phospholipids and sterols (cholesterol in animal-derived fats and  $\beta$ -sitosterol, campesterol and stigmasterol in plant-derived fats). Table I shows the main fatty acids and their major sources. These are classified, according to degree of saturation, into SATFAs, MUFAs, and polyunsaturated fatty acids (PUFAs). PUFAs are further divided into omega-6 ( $\omega$ -6) or  $\omega$ -3 fatty acids based on the position of the first double bond nearest to the methyl end of the carbon chain. The PUFAs normally have *cis*-double bonds (hydrogen atoms at the same side of the carbon chain at the double bond). This configuration may change from the *cis* to the *trans* position (hydrogen atoms at

opposite sides of the carbon chain at the double bond) during hydrogenation modification of fats and oils. The shape of a *trans* fatty acid is similar to that of a SATFA and *trans*-isomers have higher melting points (are more solid) than *cis*-isomers.<sup>10</sup>

Fats and oils are concentrated energy sources and are stored in the body as adipose tissue for the provision of a continuous fuel supply. Adipose tissue also keeps the body warm and protects against mechanical shock. In addition, dietary lipids 'carry' the fat-soluble vitamins A, D, E and K and are necessary for their absorption. Specific fatty acids, phospholipids and cholesterol are necessary structural constituents of cellular membranes and are precursors of several hormones. As such they have important functions in regulation of transport of substances through membranes and in influencing metabolic processes, determining growth and development, brain development, and a variety of body functions.<sup>3,10</sup> The human body can synthesise cholesterol, phospholipids and all but two fatty acids. These fatty acids, linoleic (C18:2  $\omega$ -6) and  $\alpha$ -linolenic (C18:3  $\omega$ -3) are therefore named 'essential fatty acids' (EFAs). It is accepted that very low-fat diets, providing 10% and less of energy as fat, are related to an atherogenic lipoprotein phenotype, characterised by small dense low-density lipoprotein (LDL) particles, low high-density lipoprotein cholesterol (HDL) and high triglycerides, as well as hypertension and insulin resistance (reviewed by Wolmarans and Oosthuizen<sup>3</sup>). Furthermore, low intakes of EFAs and especially of the  $\omega$ -3 fatty acids, C18:3  $\omega$ -3 ( $\alpha$ -linolenic acid) or C20:5  $\omega$ -3 (eicosapentaenoic acid) and possibly C22:6  $\omega$ -3 (docosahexaenoic acid) are associated with impaired development, incompetent inflammatory and immunological responses and thrombotic tendencies.<sup>3,10</sup>

There is no doubt that 'adequate' amounts of fats and oils are necessary in the human diet. But because fats and oils are major determinants of flavour, tenderness, palatability and acceptability of foods, overconsumption is a constant temptation for those who can afford it. And overconsumption is generally accepted to be associated with obesity and a wide range of NCDs, particularly when combined with physical inactivity.

The known effects and putative consequences of overconsumption of TF and of specific fatty acids have been reviewed extensively.<sup>3,10-12</sup> Although these consequences are mechanistically inter-related, they can be categorised into: (i) increased risk of obesity (and associated disorders); (ii) dislipidaemia and atherosclerosis (leading to cardiovascular diseases such as coronary heart disease (CHD) and stroke); (iii) insulin resistance (associated with the metabolic syndrome, hypertension, and non-insulin-dependent diabetes mellitus); and (iv) certain forms of cancer (possibly breast, colon and prostate cancer).

Globally, there are large disparities among geographical regions in the amounts of total dietary fat available.

**Table I. A summary of fatty acids important in human nutrition**

Abbreviation	Common name	Major sources	Known effects
<b>Short-chain fatty acids</b>			
C2:0	Acetic	Vinegar	Suspected to influence several metabolic processes but more clarification is needed
C3:0	Propionic		
C4:0	Butyric		
<b>Saturated fatty acids</b>			
C10:0	Capric	Milk, butter	Formed in the colon as a result of anaerobic fermentation of carbohydrates
C12:0	Lauric		
C14:0	Myristic		
C16:0	Palmitic		
C18:0	Stearic	Animal products: butter, cream, beef tallow, mutton fat, lard, chicken	Hypercholesterolaemic but raises HDLC
C20:0	Arachidic		
C22:0	Behenic		
C24:0	Lignoceric		
<b>Monounsaturated fatty acids</b>			
C16:1 $\omega$ -7*	Palmitoleic	Vegetable oils, especially olives and olive oil, canola oils, avocados, nuts	Neutral effect on TC and LDLC when compared with carbohydrate, lowering effect compared with SATFAs
C18:1 $\omega$ -9	Oleic		
C20:1 $\omega$ -9	Gadoleic		Raise HDLC
C22:1 $\omega$ -11	Cetoleic		
C22:1 $\omega$ -9	Erucic		
C24:1 $\omega$ -9	Nervonic		
<b>Polyunsaturated fatty acids</b>			
C18:2 $\omega$ -6 <sup>†</sup>	Linoleic	Vegetable oils: sunflower, corn, safflower	Lower TC and LDLC but also HDLC
C18:3 $\omega$ -3 <sup>‡</sup>	$\alpha$ -linolenic		
C18:3 $\omega$ -6	$\gamma$ -linolenic	Green plants, algae, plankton, soya beans, walnuts, flaxseed oil	
C20:3 $\omega$ -6	Dihomo-g-linolenic		
C20:3 $\omega$ -9	Dihomo-g-linolenic	Nuts, seeds, vegetable and seed oils	
C20:4 $\omega$ -6	Arachidonic		
C20:5 $\omega$ -3	Eicosapentaenoic	Meat, eggs and other animal-derived foods	Decrease TG, inhibit platelet aggregation
C22:4 $\omega$ -6	Adrenic		
C22:5 $\omega$ -3	Docosapentaenoic		
C22:5 $\omega$ -6	Docosapentaenoic		
C22:6 $\omega$ -3	Docosahexaenoic		
<b>Trans fatty acid polymers</b>			
C18:1 $\omega$ -9	Elaidic	Most dairy products but especially hard margarines and products (cookies, biscuits and pastries) containing these margarines	Raise TC, TG, LDLC and decrease HDLC, may raise Lp(a)

\*Double bonds are designated by counting the carboxyl carbon as position 1.  $\omega$ -3, 6, 9, 11 indicate the position of last double bond.

<sup>†</sup>Linoleic acid is the first member of the  $\omega$ -6 series of fatty acids (essential fatty acid).

<sup>‡</sup> $\alpha$ -linolenic acid is the first member of the  $\omega$ -3 series of fatty acids (essential fatty acid).

C = number of carbon atoms in chain; TC = total serum cholesterol; LDLC = low-density lipoprotein cholesterol; TG = triglycerides; HDLC = high-density lipoprotein cholesterol; Lp(a) - lipoprotein a.

The FAO balance sheets<sup>10</sup> showed that in 1990 the daily caput availability of fat was 128 g in developed countries and not more than 49 g in developing countries. Tee concluded in his presentation at the symposium on fats and oils (unpublished data) that insufficient fat energy in developing countries is a widespread complication for protein utilisation, contributing to the high rates of

undernutrition in these countries. In his careful analysis of availability of dietary fats, he confirmed the observation of Drewnowski and Popkin<sup>13</sup> that the increased availability of cheap vegetable fats and oils in developing countries could, however, also lead to overconsumption and transition from traditional diets to high-fat diets in low-income subjects — a process accelerated by urbanisation.

## The controversy regarding total fat recommendations

Global recommendations and guidelines regarding fat intakes are summarised in Table II (as presented by Kumanyika during the 2000 symposium). The table shows the emphasis on lowering of fat intake (to between 15% and 35% of total energy), ways to lower intakes, and some advice on types of fat (less SATFAs and *trans* fat, more MUFAs and  $\omega$ -3 PUFAs). The USA and Thailand guidelines recommend a 'moderate' fat intake.

Some nutritionists have recently started to question low-fat guidelines. In his paper entitled 'The soft science of dietary fat', Taubes<sup>9</sup> traced the often controversial history of the USA fat recommendations and argued that there is little (or no) scientific evidence that following a lower fat diet will affect longevity of the USA population. He based his conclusion, that low-fat recommendations have not stood the test of time, on *inter alia* the following arguments and his interpretation of the available literature:

1. Despite decreased fat intake recommendations and the availability of more than 15 000 low-fat food products on the USA market, there is an alarming upward surge in obesity prevalence in the USA.
2. The relationships proved are between dietary fat intake and risk factors (or markers) of atherosclerosis and cardiovascular disease — and not between fat intake and the diseases *per se* or longevity. Computer modelling based on proven effects of lifelong low-fat diets on total serum cholesterol, and the latter's association with CHD, shows net increases in life expectancy counted in weeks and months rather than years. He also quotes sources indicating that the drop in CHD deaths in the USA started in the 1940s (before low-fat recommendations), that the drop between 1979 and 1996 was because CHD was better and more extensively treated, and that there is no evidence that this decline can be attributed to a decreased fat intake. He questions whether the incidence of CHD is in fact declining.
3. Data from more than 300 000 Americans who participated for more than 10 years in the Nurses Health Study and the Health Professionals Follow-up Study showed that total fat consumption had no relation to risk of heart disease, that MUFAs lower risk, that SATFAs are little worse than carbohydrates and that *trans* fatty acids are 'harmful'.
4. Low-fat diets are necessarily high-carbohydrate diets, and may lead to insulin resistance, the metabolic syndrome and an atherogenic lipoprotein profile (small dense LDL particles and low HDLC).
5. Low serum cholesterol levels in the Japanese population are thought to be associated with high

levels of haemorrhagic stroke (Japanese physicians actually advise patients to increase fat intake).

6. The World Cancer Research Foundation, together with the American Institute for Cancer Research, could find no 'convincing' or even 'probable' evidence to believe that dietary fat causes cancer.

7. Changing the fat quality of the diet (different fatty acids) also changes the lipid composition of cell membranes. There is, according to some scientists, insufficient information available on how this will change the transport functions of cell membranes to formulate dietary recommendations on types of fat.

There are many reasons for the dietary fat controversy. In addition to certain beliefs (developed into dogmas) and possible vested interests, the major reasons probably lie in the complexity of the issue, the different and often impossible study designs needed to prove certain hypotheses, and how nutrition scientists interpret and judge the available scientific evidence.

Dietary fat recommendations are a complex issue. In simple advice and core messages to consumers to lower or limit fat intake, fat is regarded as one nutrient, whereas it consists of a range of fatty acids, often with opposing effects on human physiology. For example, myristic acid (C14:0) raises serum cholesterol levels while linoleic acid (C18:2  $\omega$ -6) decreases it; arachidonic acid (C20:4  $\omega$ -6) gives rise to prostaglandins of the 2-series and leukotrienes of the 4-series, while eicosapentaenoic acid (C20:5  $\omega$ -3) inhibits the formation of these particular eicosanoids.<sup>10</sup> The final effect of a fat or oil will therefore depend on its total fatty acid composition, which should be reflected in recommendations on intake.

Designing any nutritional or dietary study to determine either acute (short-term) or long-term effects and associations of dietary fats with health outcomes is difficult. The design should enable scientists to judge if observed outcomes (results) are direct effects of the fat or the fatty acids, indirect effects because of substances in foods associated with the fat (e.g. fat-soluble vitamins), or replacement effects because increasing a particular fat or fatty acid in an experimental diet would lead to a decrease of other energy-providing nutrients if total energy is kept constant. There are also known individual variations in response to dietary intakes and manipulation because of genetic influences.<sup>14</sup> The choice of subjects (age, gender, body mass index, activity levels, smoking habits, background diet, presence of abnormal metabolic responses, etc.) as well as interactions between different dietary substances, are all factors that could further complicate the design of studies and interpretation of results. For example, it is conceivable that dietary fibre, known for its hypocholesterolaemic effects,<sup>15</sup> will impact on responses to changes in dietary fat intake. It is also possible that the presence of

Table II. Selected international guidelines for fat intake*		
Country	Year	Guidelines
South Africa	2001	Use fats sparingly; meat fish, chicken and eggs can be eaten every day.
India	1998	Eat less fat, sugar, salt; eat less eggs and flesh foods.
Chinese Nutrition Society	1997	Eat some fish, poultry, eggs and lean meat frequently; eat less fatty meat and animal fat.
Canada	1997	Choose lower fat dairy products, leaner meats and foods prepared with little or no fat.
Malaysia (proposed)	1996	Minimise fat in food preparation and choose prepared foods that are low in fat and cholesterol.
Denmark	1997	Choose milk products and cheese with a low content of fat; choose meat and meat products to be put on the bread with a low fat content; use only small quantities of butter, margarine and oil.
France	1992	Do not abuse fats.
Japan	1985	Be aware that the type of fat intake is as important as the quantity, avoid eating too much fat; use vegetable oils rather than animal fats.
Australia	1992	Eat a diet low in fat and in particular low in saturated fat; low-fat diets are not suitable for young children.
Korea	1986	Keep fat consumption at 21% of intake.
Singapore	1989	Restrict total fat intake to 20% and 30% of total energy; modify composition of fat in the diet to one-third polyunsaturated, one-third mono and one-third saturated.
Indonesia	1995	Obtain not more than 25% (a quarter) of total energy intake as fats and oils.
Norway	1997	Use soft vegetable margarine or oil instead of hard margarine or butter; replace full milk with light or skimmed milk, and choose dairy products with less fat on weekdays. Use white or brown sauce where it is suitable, instead of melted butter, mayonnaise or remodelled sauce. Eat more fish of all types; both fatty fish as mackerel and herring and lean fish as cod should be eaten more often.
Netherlands	1985	Ensure an average total fat intake of 30 - 35% of dietary energy; make sure that saturated fat consumption is around 10% of total energy and PUFA is 50 - 100% of saturated fat.
New Zealand	1991	Prepare foods with minimal added fat — especially saturated fat.
Hungary	1997	Avoid too much fat, use vegetable oil and margarine. Drink half a litre of low-fat milk per day.
Thailand	1991	Eat only a moderate amount of fats.
Nordic Nutrition Recommendations: Denmark, Finland, Norway, Sweden	1998	Fat should not provide more than 30% of energy (primarily reduce saturated fat); total fat should not however, be below 20 - 35% energy; essential fatty acids should contribute 3 - 10%; both $\omega$ -6 and $\omega$ -3 fatty acids are essential but the requirement of $\omega$ -6 is greater; linoleic and long-chain $\omega$ -3 should be 0.5% of energy or more.
United Kingdom	2000	Eat less total fat, 35% food energy recommended; eat less saturated fatty acids, 15% of energy recommended including trans fatty acids; PUFA may be increased from 5 to 7% of daily energy (P/S ratio up to 0.45).
United States	2000	Choose a diet that is low in saturated fat and cholesterol and moderate in total fat.

\* Summary of presentation by S Kumanyika at the symposium 'Fats and oils — forbidden foods but important nutrient', Durban, 15 - 18 August 2000.

dietary antioxidants will modulate oxidation of long-chain PUFAs and their metabolic effects.<sup>10</sup> In the evaluation of study results it is important to ask the question whether observed effects are because of the presence of a particular nutrient — or the absence of other nutrients. In layman's terms, in the context of high-fat diets, this question can be formulated as follows: are the detrimental effects 'because of too much of a bad thing (SATFAs and *trans* fatty acids) or

not enough of a good thing' (MUFAs or  $\omega$ -3 fatty acids)?

Another important component of any dietary study is the choice of outcomes that are measured. If dietary fats, oils, or their constituent fatty acids influence a metabolic marker or even risk factor of a disease, this is not necessarily evidence that the disease *per se* (morbidity) or mortality would be influenced. To measure the relationship between any dietary

substance and morbidity and/or mortality would need very large studies over long periods, which will be difficult to fund. This type of information is therefore scarce, if available at all.

## South African research in dietary fats and oils

The studies selected for oral presentations during the symposium on fats and oils<sup>4-8</sup> reflect an awareness of the newest developments in this field.

In the study by Tichelaar *et al.*,<sup>4</sup> presented by Marius Smuts, the effect of  $\omega$ -3 fatty acid supplementation on the nutritional status and development of 278 rural primary school children, aged 6 - 11 years, was examined in a placebo-controlled trial over 9 months. All children received antihelminthic therapy (400 mg albendazole every 4 months) and were fed biscuits fortified with  $\beta$ -carotene and iron (50% of the RDA). An oil supplement (11 ml per day) was provided during 162 school days. The experimental group received a lemon- and lime-flavoured mix of evening primrose, sunflower and fish oil, and the control children, flavoured sunflower oil. In addition to the significant reductions in the prevalence of anaemia and vitamin A deficiency in both groups, the experimental children showed a significant improvement in the total recall score on the Hopkins Verbal Learning Test from baseline to end compared with the control children. This study suggests that  $\omega$ -3 fatty acid supplementation improved verbal learning and memory among these rural children.

It is known that rural Africans in South Africa follow a low-fat diet, with a high polyunsaturated/saturated (P:S) ratio (exceeding 1.0)<sup>16</sup> because of liberal use of cooking oil, when available, in the cooking of food. A recent national study of South African children aged 1 - 9 years<sup>17</sup> showed that fat provided a mean of 22.9% ( $\pm$  8.1%) of the energy in the diet of the boys and 23.7% ( $\pm$  2.9%) of the girls. This study also showed that fish was not among the top 25 foods consumed the previous day (as measured using a 24-hour recall method). The quantitative food frequency questionnaire showed that of 2 883 children, 1 559 consumed fish (pilchards or sardines), but that the mean amount consumed daily was only 19 g.<sup>17</sup> These facts suggest that there may be an  $\omega$ -3 fatty acid deficiency in South African children, especially rural black children. This deficiency should be addressed in the South African dietary guidelines, accompanied by practical advice on how to increase  $\omega$ -3 fatty acid intakes in children often from food-insecure families and from areas where fish is not always available. The study by Tichelaar *et al.*<sup>4</sup> clearly shows that increased intake of  $\omega$ -3 fatty acids could increase learning potential of primary school children, suggesting that  $\omega$ -3 fatty acid supplementation of the diet of these children, for example in the Primary

School Nutrition Programme, should be considered.

The probable  $\omega$ -3 fatty acid deficiency in the diet of these South African children may also result in a skewed  $\omega$ -3/ $\omega$ -6 fatty acid ratio in the diet, because sunflower oil, as a major fatty acid source in the South African diet,<sup>3</sup> is a rich source of  $\omega$ -6 fatty acids.<sup>3</sup> Supplementation of the diet of these children as suggested with  $\omega$ -3 fatty acid-rich sources, will help to improve this ratio.

The study by Smuts and Carlson<sup>5</sup> examined the possibility that docosahexaenoic acid (DHA)-enriched eggs could improve pregnancy outcomes. They compared 19 women who ate regular eggs with 18 who ate DHA-enriched eggs (up to 12 per week) during the last trimester of pregnancy. Their study demonstrated that the mothers who ate the DHA-enriched eggs were less likely to deliver preterm or low-birth-weight infants.

Although this was a relatively small study, subsequently followed up with a larger one, it addresses the possibility that South Africans may take in too little  $\omega$ -3 fatty acids, and supports the suggestion that South African dietary guidelines should emphasise sources of these fatty acids. The importance of this paper is that it draws attention to the need for alternative sources of  $\omega$ -3 fatty acids in the diet of South Africans. The larger trial by Smuts and Carlson (unpublished) has been completed and demonstrated that after controlling for predefined factors, gestation was increased by 6.0 days ( $p = 0.009$ ) with intake of DHA-enriched eggs (C22:6  $\omega$ -3) compared with ordinary eggs ( $N = 142$  and  $N = 149$ ), respectively (C M Smuts — personal communication).

Blackhurst and Marais<sup>6</sup> focused on the lipid peroxidation status of edible oils in South Africa, measuring conjugated dienes (CD), lipid hydroperoxides (LOOH) and thiobarbituric acid reactive substances (TBARS) in a variety of 33 oils. They found that CD ranged from 9.7 to 36.1  $\mu$ M, LOOH from 9.5 to 14.7  $\mu$ M and TBARS from 66.4 to 3 175  $\mu$ M. They concluded that their results highlight the need to declare undesirable compounds in edible oils at manufacture. However, this was a small study and should be followed up with more research before any conclusions can be drawn and recommendations made. If there is a problem, it may be addressed by adding the antioxidant vitamin E to the oil. The FAO/WHO Consultation on Fats and Oils<sup>10</sup> recommended that oxidation of PUFAs may be prevented with a vitamin E-to-PUFA ratio greater than 0.6 mg tocopherol equivalents per gram of PUFA.

In the paper by Vorster *et al.*<sup>7</sup> the associations between nutrient intakes and plasma fibrinogen were examined in an epidemiological study in which the diet and health outcomes of rural, migrant and urban blacks were compared. This study was motivated by the high incidence of stroke in black South Africans, and their known high plasma fibrinogen levels, a major risk factor

for stroke. Of the many dietary factors associated with high fibrinogen (undernutrition in men and over-nutrition in women), intake of *trans* fatty acids emerged as a possible risk factor for hyperfibrinogenaemia.

There is little doubt that *trans* fatty acids have undesirable effects on serum lipids (increasing TC and LDLC and decreasing HDLC) and that intakes are associated with increased risk for CHD in epidemiological studies (reviewed by Ascherio *et al.*<sup>18</sup>). The relationship between *trans* fatty acid intake and haemostatic variables is, however, largely unknown. In a clinical intervention study, Mutanen and Aro<sup>19</sup> found no evidence that *trans* fatty acids, when replacing saturated fat and compared with stearic acid, had any effects on a number of haemostatic variables (fibrin degradation products, D-dimer, factor VIIc, tissue type plasminogen activity and plasminogen activator inhibitor activity). Therefore, the results of the study by Vorster *et al.*<sup>7</sup> should be followed up with more research, comparing *trans* fatty acid effects on haemostasis with MUFAs and PUFAs.

Because of their price compared with soft margarines, hard margarines, rich in *trans* fatty acids, are a favourite fat source for many South Africans.<sup>16,20</sup> At present, legislation to label for *trans* fatty acid content of fat and oil products is being finalised (A Booysen, National Department of Health — personal communication). However, it is doubtful if many South Africans will take notice or understand the meaning of such a label. Clearly, consumers must be educated, for example through the FBDG, to limit intake of *trans* fatty acids by choosing fats and oils with low levels of these fatty acids. In addition, the food industry should be targeted and motivated to reduce *trans* fatty acids in their products, also in the cheaper ranges. Legislation to label for *trans* content could accomplish this.

In the study by Bosman *et al.*,<sup>8</sup> the results of a detailed examination of the replacement of oil in high-fibre muffins with a protein-based fat substitute (Simplese) were presented. This study was motivated by a need for high-fibre, low-fat convenience products, especially for hypercholesterolaemic consumers. The results indicated that the sensory and physical characteristics as well as the microbiological safety of the refrigerated batter and the baked product were not adversely affected by the fat replacement.

This study demonstrates the awareness of the need to develop culturally sensitive, low-fat products for particular consumers, and the capacity to do this in a scientifically responsible way. The application of dietary guidelines can only be feasible and successful if foods and products that meet the recommended requirements are available.

## Discussion

The main question addressed in this paper is how explicit, detailed or specific South African guidelines should be regarding the quantity and quality (types) of fat intake. The FBDG directive to 'eat fat sparingly' is a good starting point, indicating that fats should be eaten, but that overconsumption should be avoided.<sup>2,3</sup> However, the guideline should be accompanied by practical advice on which foods and products to choose.<sup>3</sup>

The joint expert consultation of the FAO/WHO<sup>10</sup> on fats and oils in human nutrition pointed out that responsible, evidence-based recommendations are needed because of the wide-ranging implications for consumers, health care providers, nutrition educators, food producers, processors and distributors. There are those<sup>9</sup> who are of the opinion that available evidence is not sufficient to promote low-fat diets. Different interpretations of available evidence are possibly influencing fat recommendations. It seems that there is a movement towards more 'liberal' fat recommendations, with the word 'moderate' cropping up in some recommendations (Table II) and technical support papers.<sup>3,10,12</sup> The American Heart Association (AHA)<sup>12,21</sup> recently published a revision of their dietary guidelines, emphasising 'healthy eating patterns and behaviours rather than a singular focus on dietary fat intake'. These guidelines replace their former step 1 diet and contain advice for the general population for overall healthy eating patterns, how to maintain an appropriate body weight, desirable cholesterol levels and desirable blood pressure. The dietary fat recommendations are practical and positive with emphasis on a varied diet containing *inter alia* fruits, vegetables, whole grains, legumes and low-fat or non-fat dairy products, fish, lean meats and poultry. To reach desirable cholesterol levels, the advice is to limit SATFAs and *trans* fat intake. The AHA<sup>12,21</sup> also makes more specific recommendations for those individuals at increased risk of developing cardiovascular disease (their former step 2 diet). The latter recommendations are aimed at three groups of individuals (those with elevated LDLC or pre-existing cardiovascular disease; those with dislipidaemia characterised by increased triglycerides and small, dense LDL particles with low HDLC; and those suffering from diabetes mellitus and insulin resistance).

The guidelines for the general population are accompanied by more specific dietary recommendations (TF less than 30% of total energy, SATFAs plus *trans* fat less than 10%, replacing sources of these with whole grains, fish, vegetables, legumes, and nuts). An important development of these guidelines is that specific actions (e.g. decreasing total SATFA or *trans* fatty acid intakes, and increasing fish and therefore  $\omega$ -3 fatty acid intakes) are related to specific population goals (healthy eating patterns, appropriate body weight, desirable cholesterol levels and desirable blood pressure).

Wolmarans and Oosthuizen<sup>3</sup> pointed out that many South Africans still have low fat intakes (e.g. rural blacks). The National Food Consumption Survey<sup>17</sup> showed that rural children consumed approximately 21% of their energy as fat and children in the Free State only 20%, while many adult groups consume either below 20% (rural blacks),<sup>20</sup> or between 35% and 45% energy as fat (whites, Indians and coloureds).<sup>16</sup> There is also evidence that the groups consuming less than 20% of their energy as fat do not reach micronutrient needs.<sup>16,20</sup> Vitamin A deficiency for example, is a widespread problem among South African children,<sup>22</sup> especially in the rural areas, while many adult blacks in these areas are known to have deficient intakes of calcium, iron, zinc, riboflavin, vitamin B<sub>6</sub>, folate, ascorbic acid and vitamin A.<sup>16</sup> These data suggest that to meet micronutrient needs, diets with at least 20% of fat energy or more should be followed. The THUSA study, comparing nutritional status of rural and urban blacks<sup>20,23</sup> also indicated that urban blacks, following a diet in which 30% of energy was provided by fat, had a much better nutritional status than rural blacks on lower fat diets ( $\pm$  20% fat energy). It seems, therefore, that instead of advising that less than 30% of energy should come from fat (which suggests any amount between 15% and 30%), more specific advice, for example between 25% and 30%, or to aim for 30% should be given to South African populations. At a 30% level, blacks still have low total and LDLC and high HDLC levels.<sup>23,24</sup> The risk factors for chronic diseases present at this level of fat intake were obesity and hypertension, but these risk factors were not related to fat intake and were also present in rural black women on lower fat intakes.<sup>23,24</sup> A dietary message to aim for 30% energy as fat will also address those groups with high intakes to lower their fat consumption.

There is little information on *trans* and  $\omega$ -3 fatty acid intakes in South African populations. The studies reported at the symposium on fats and oils<sup>4,5,7</sup> reflect awareness that many South Africans may be consuming too much *trans* fatty acids and not enough  $\omega$ -3 fatty acids. To rectify this problem, specific advice should be given on eating and using less hard margarine in preparation of foods, and on eating more fish or other sources of  $\omega$ -3 fatty acids, such as soya products containing the parent  $\omega$ -3 fatty acid, alpha-linolenic acid. For many South Africans the advice to eat more soya products will probably be more culture-sensitive and affordable than advice to eat more fish. Another alternative is to develop more products containing  $\omega$ -3 fatty acids, such as the  $\omega$ -3-enriched eggs reported by Smuts and Carlson.<sup>5</sup>

The advantages of replacing cholesterol-raising SATFAs and *trans* fatty acids not only with PUFAs and carbohydrates, but also with MUFAs have been reviewed by Wolmarans and Oosthuizen.<sup>3</sup> MUFAs have cholesterol-lowering properties when compared with SATFAs and *trans* fatty acids. This effect is not as

strong or large as that of PUFAs, but MUFAs raise HDLC whereas PUFAs and *trans* fatty acids tend to lower HDLC. MUFAs also lower triglycerides, whereas high-carbohydrate diets tend to increase it. The major dietary sources of MUFAs are olives, and olive and canola oils, typical foods in the Mediterranean diet. These items are expensive and not traditionally part of the diet of most South Africans. However, other sources in the South African diet such as avocados and peanut, soybean, corn, and sunflower oils contain appreciable amounts of MUFAs.<sup>3</sup> However, sunflower oil is a rich source of  $\omega$ -6 fatty acids and increased intakes may skew the  $\omega$ -3/ $\omega$ -6 fatty acid ratio as already mentioned. Dietary recommendations should therefore not encourage higher intakes of sunflower oil in groups that already have this as a main fatty acid source. Sources of SATFAs such as butterfat, palm oil, beef tallow and lard all provide MUFAs.<sup>3</sup> The result is that MUFAs form about a third or more of the total fat intake of most South African groups.<sup>3,16</sup> At present it seems that adequate intake of MUFAs is not a problem in those who have adequate diets. The situation should, however, be closely monitored if dietary patterns change. The available data show that with an increase in fat intake, cheap sources of hard margarines and animal foods provide the additional fat.<sup>16</sup>

## Conclusion

In conclusion, it seems that to address nutritional deficiencies and excesses in South African population groups, more specific advice on both quantity and quality of fat intake is needed. Regarding quantity it seems that a message to aim for 30% dietary energy as fat could benefit those not eating sufficient fat, as well as those at high risk of NCDs because of overconsumption. Regarding the quality of fat, available research suggests that South Africans should increase intakes of  $\omega$ -3 fatty acids and possibly MUFAs at the expense of mostly SATFAs and should reduce their *trans* fatty acid intakes. However, this advice must be translated for consumers in terms of acceptable, available and affordable foods. The challenge to nutritionists is to educate and motivate consumers to follow 'optimal' fat diets. The challenge to the food

industry is to develop appropriate and affordable products so that more 'healthy' choices are available on the South African market.

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