Interim Summary of Conclusions and Dietary Recommendations on Total Fat & Fatty Acids

From the Joint FAO/WHO Expert Consultation on Fats and Fatty Acids in Human Nutrition, November 10-14, 2008, WHO HQ, Geneva

Introduction and Definitions

There are the inherent limitations with the convention of grouping fatty acids based only on the number of double bonds, i.e. saturated fatty acids (SFA), monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA) insofar as describing the effects of fatty acids on human health and in developing dietary recommendations. The large body of epidemiological evidence about total fats, fatty acids, and human health apply these groupings and show that the major groups of fatty acids are associated with different health effects. However, the expert consultation recognised that individual fatty acids within each broad classification of fatty acids may have unique biological properties and health effects. This has relevance in making global recommendations because intakes of the individual fatty acids that make up the broad groupings will differ across regions of the world depending on the predominant food sources of total fats and oils. The expert consultation also recognized that in spite of these limitations, the scientific community in general and an increasing proportion of the general population continue to use the groupings based on chemical structure and thus, there would be disadvantages in abandoning them. Moreover, few countries have food composition databases that enable dietary assessment of individual fatty acid intake.

For the sake of clarity and in recognition that often we use generalized terms to refer to specific fatty acids, the expert consultation thought it appropriate to provide details as to the use in this document. In particular:

- The expert consultation recognises that grouping of fatty acids into these three broad groups (SFA, MUFA and PUFA) is based on chemical classifications, but it is clear that individual fatty acids within these groups have distinct biological properties. However, most of the epidemiological evidence reviewed by the experts uses broad groupings, which make it difficult to distinguish and disentangle the effects of individual fatty acids.

- SFA refers to the major SFA in our diet, namely C14, C16, C18, except in the case of milk and coconut oil where SFA range from C4 to C18.

- MUFA refers to the major monounsaturated fatty acid in Western diets, which is oleic acid (C18:1n-9). It should be recognised that in some populations, a major monounsaturated fatty acid is erucic acid (C22:1n-9), as for example, found in culinary oils derived from some Brassica spp. such as rapeseed and mustard seed.

- PUFA refers to the major PUFA in our diet, which includes mainly linoleic acid (C18:2n-6), a lower proportion of alpha-linolenic acid (C18:3n-3), and depending on seafood intake a variable but relatively low proportion of long chain PUFA such as AA, EPA, DPA and DHA. For the purposes of food labelling, the terms EFA, PUFA, long chain PUFA, n-6 and n-3 lack precision and should not be used without fully specifying the actual fatty acids and their amounts. Many different fatty acids with quite different properties fall under these umbrella terms.

- TFA refers to the major *trans* fatty acids in our diet which are typically isomers of 18:1 trans derived from partially hydrogenated vegetable oils.

- Some fatty acids (e.g. *trans* monoenes, conjugated linoleic acid [CLA], etc.) are members of more than one chemical classification but by convention are interpreted as in only one category (*trans* monoenes in MUFA, CLA in PUFA, etc.).

- There are many fatty acids that are usually minor components of most foods but are major components of some specialty foods and/or of supplements. FAO/WHO recommendations must be carefully interpreted with respect to unusual fatty acids ["usual" = straight chain, all-cis, methylene-interrupted (homoallylic); "unusual" = *trans*, branched chain, non-methylene interrupted double bond structure].

Levels and Strength of Evidence

During the preparatory process for the expert consultation the experts agreed on the criteria that would be used to judge the levels and strength of evidence required to conclude that total fat and fatty acids affect major health and disease outcomes. It was decided to follow the same criteria employed in the report *Diet, Nutrition, and the Prevention of Chronic Diseases; Report of a Joint WHO/FAO Expert Consultation (*WHO TRS 916, Geneva, 2003), which had based its criteria on a modified version of that used by the World Cancer Research Fund. In doing so the Experts acknowledged other equally valid criteria that exist.

Four levels of judgment were identified:

- Convincing
- Probable
- Possible
- Insufficient

Given the limited number of randomized controlled trials of dietary fat and chronic disease or death it was agreed that only evidence of sufficient strength to be "convincing" or "probable" would allow a dietary recommendation to be formulated.

Summary of Total Fat and Fatty Acid Requirements for Adults, Infants (0-24 months) and Children (2-18 years)

There was convincing evidence that energy balance is critical to maintaining healthy body weight and ensuring optimal nutrient intakes, regardless of macronutrient distribution expressed in energy percentage (%E). The requirements on total fat and different fatty acid groups are summarized in the following tables: Table 1 for adults and Table 2 for infants and children. It was emphasized that requirements should be tailored to individuals and that the general requirements for certain groups, e.g. children and elderly subjects, have not yet been adequately established.

Table 1: Recommended dietary intakes for total fat and fatty acid intake: Adults

Fat/FA (Explanations	Measure of the abbreviations are listed after Table 2 on page 5)	Numeric amount	Level of Evidence			
			Convincing	Probable	Possible	Insufficient
Total fat	AMDR: U-AMDR: L-AMDR:	20 – 35%E 35%E 15%E		No relation with CHD events, fatal CHD, total cancer, or cancer subtypes.		Risk of diabetes, metabolic syndrome components, body weight/adiposity.
SFA	U-AMDR:	10%E	C12:0–16:0 ↑ LDL and total/HDL ratio in comparison to *cis* MUFA or PUFA; ↑ LDL but no effect on total/HDL in comparison to carbohydrate.		↑ risk of diabetes	Risk of hypertension, body weight/adiposity.
MUFA	AMDR:	By difference ˜, $	↓ LDL and total/HDL ratio when substituting SFA (C12:0–16:0)		↓ risk of metabolic syndrome components.	Risk of diabetes, body weight/adiposity, CHD events, total cancer or cancer subtypes.
Total PUFA	AMDR (LA + ALA + EPA + DHA): U-AMDR: L-AMDR: AI:	6 – 11%E 11%E 6%E 2.5 – 3.5%E	See above, for exchange of SFA for PUFA. Essential (LA, ALA). ↓ risk of CHD events when PUFA replace SFA		↓ risk of metabolic syndrome components, diabetes. ↑ lipid peroxidation with high consumption, especially when tocopherol intake is low. Specific minimum to prevent deficiency unclear.	Risk of body weight/adiposity, total cancer or cancer subtypes.
n-6 PUFA	AMDR (LA): EAR: AI:	2.5 – 9%E 2%E (SD of 0.5%) 2 – 3%E	See above, for exchange of SFA for PUFA. Essential (LA).	↓ risk of metabolic syndrome components, diabetes.	Specific minimum to prevent deficiency unclear.	Risk of body weight/adiposity, total cancer or cancer subtypes.
n-3 PUFA	AMDR (n-3§): L-AMDR (ALA): AMDR (EPA + DHA):	0.5 – 2%E ≥ 0.5%E 0. 250 – 2* g/day	↓ risk of fatal CHD events (EPA+DHA). Essential (ALA).		↓ risk of total CHD events, stroke. Specific minimum to prevent deficiency unclear.	
TFA	UL: (total TFA from ruminant & industrially-produced sources)	<1%E	↓ HDL and ↑ total/HDL ratio in comparison to SFA (C12:0 – C16:0), *cis* MUFA or PUFA. ↑ risk of CHD events	↑ risk of fatal CHD and sudden cardiac death. ↑ risk of metabolic syndrome components, diabetes.		Risk of body weight/adiposity, diabetes, total cancer or cancer subtypes.

˜ Total fat [%E] – SFA [%E] – PUFA [%E] – TFA [%E]; $ can be up to 15 – 20 %E, according to total fat intake;   § (ALA + n-3 long-chain PUFA);   * for secondary prevention of CHD

Table 2: Recommended dietary intakes for total fat and fatty acid intake: Infants (0-24 months) & Children (2-18 years)

Fat/FA (*Explanations*	Age Group *of abbreviati*	Measure *ons are listed after*	Numeric Amount *this Table on page 5)*	Level of Evidence
Total fat	0-6 mo	AMDR:	40 – 60%E	Convincing
		AI:	based on composition % of total fat in HM	Convincing
	6-24 mo	AMDR:	gradual reduction, depending on physical activity, to 35%E [a]	Convincing
	2-18 yr	AMDR:	25 – 35%E	Probable
SFA	2-18 yr	U-AMDR:	8%E	Probable
			Children from families with evidence of familiar dyslipidemia (high LDL cholesterol) should receive lower SFA but not reduced total fat intake	
MUFA	2-18 yr	AMDR:	Total fat [%E] – SFA [%E] – PUFA [%E] – TFA [%E]	Probable
Total PUFA	6-24 mo	U-AMDR:	<15%E	Probable
	2-18 yr	U-AMDR:	11%E	Probable
LA & ALA	0-24 mo	Comment:	essential and indispensable	Convincing
n-6 PUFA				
AA	0-6 mo	AI:	0.2 – 0.3%E [b]	Convincing
LA	0-6 mo	AI:	HM composition as %E of total fat	Convincing
	6-12 mo	AI:	3.0 – 4.5%E	Convincing
	6-12 mo	U-AMDR:	<10%E	Probable
	12-24 mo	AI:	3.0 – 4.5%E	Convincing
	12-24 mo	U-AMDR:	<10%E	Probable
n-3 PUFA				
ALA	0-6 mo	AI:	0.2 – 0.3%E [b]	Convincing
	6-12 mo	AI:	0.4 – 0.6%E	Probable
	6-12 mo	U-AMDR:	<3%E	Probable
	12-24 mo	AI:	0.4 – 0.6%E	Probable
	12-24 mo	U-AMDR:	<3%E	Probable
DHA	0-6 mo	AI:	0.1 – 0.18%E [b]	Convincing
	0-6 mo	U-AMDR:	No upper value within the HM range up to 1.5%E	Convincing
	0-6 mo	Comment:	conditionally essential due to limited synthesis from ALA	Probable
	6-12 mo	AI:	10 – 12 mg/kg	Probable
	12-24 mo	AI:	10 – 12 mg/kg	Probable
	0-24 mo	Comment:	critical role in retinal and brain development	Convincing
EPA+DHA	2-4 yr	AI:	100 – 150 mg (age adjusted for chronic disease prevention) [c]	Probable
	4-6 yr	AI:	150 – 200 mg (bridged from an infant value of 10 mg/kg	Probable
	6-10 yr	AI:	200 – 250 mg to the adult value assigned at age 10 years)	Probable
TFA	2-18 yr	UL:	<1%E (total TFA – from ruminants and industrially-produced sources)	Convincing

[a] For infants 6-12 mo, the proposed fat intake as a %E is lower than those recommended in the 1994 report. The primary reasons are the concern over increased obesity rates and the redefined growth standards based on human milk-fed infants, associated with leaner growth in later infancy (WHO MGRS 2006).

[b] The amounts are expressed as %E in order to be consistent with the other entries in the table. However based on human milk composition as is often the case when referring to infants of breast feeding age, the amounts for AA and ALA would be expressed as 0.4-0.6%FA and for DHA as 0.20-0.36%FA. This conversion assumes that half of the energy in human milk comes from fat. For children 6-24 months of age the estimation is based on provision of breast milk to meet half of the daily energy needs, the rest of the energy would come from non breast milk diet.

[c] Although there is no specific data from long term studies on the relationship between fatty acid intake and chronic disease prevention from children the assumption is that children also benefit from a lower saturated fat, higher PUFA intakes.

Abbreviations in tables:

%E	percent of energy ;
AI	adequate intake (expressed as a range);
EAR	estimated average requirement ;
AMDR	acceptable macronutrient distribution range;
L-AMDR°	lower level of acceptable macronutrient distribution range°;
U-AMDR°	upper level of acceptable macronutrient distribution range°;
UL	upper level; this term was developed for instances where biochemical indicators are needed to confirm any adverse effects, measurable with a probability of occurrence. In the case of FA this only applies to TFA.

° These two terms refer to the upper and lower range of the AMDR, very much similar to the use of UCI and LCI for the upper and lower bounds of confidence intervals.

HM	Human milk;
E	energy;
SFA	saturated fatty acids;
MUFA	monounsaturated fatty acids;
PUFA	polyunsaturated fatty acids;
TFA	*trans*-fatty acids;
LA	linoleic acid;
ALA	alpha-linolenic acid;
EPA	eicosapentaenoic acid;
DHA	docosahexaenoic acid;
AA	arachidonic acid.

Conclusions and Recommendations for total fat

The consultation examined the background papers, scientific reports and various studies assessing the relationship between total dietary fats as well as selected fatty acids and various physiological conditions and illnesses. The experts agreed with the evidence summarized in two recent reports (*Diet, Nutrition, and the Prevention of Chronic Diseases; Report of a Joint WHO/FAO Expert Consultation.* WHO TRS 916, Geneva, 2003; *Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective.* World Cancer Research Fund/American Institute for Cancer Research, Washington, DC, 2007) that there is no probable or convincing evidence for significant effects of total dietary fats on coronary heart disease or cancers. Therefore, of primary concern and importance was the potential relationship between total dietary fats and body weight (overweight and obesity).

There was convincing evidence that energy balance is critical to maintaining healthy body weight and ensuring optimal nutrient intakes, regardless of macronutrient distribution of energy as % total fat and % total carbohydrates.

Although the specific evidence was not reviewed in-depth at the consultation it is felt sensible that maintaining appropriate dietary patterns and energy levels, and adequate physical activity levels are critical in preventing unhealthy weight gain (e.g. overweight and obesity) and to ensure optimal health for those predisposed to insulin resistance.

Some older intervention studies from industrialized countries suggest that diets with lower % of energy from fat (i.e. %E fat) tend to be hypocaloric and are therefore associated with short term weight loss. Conversely, more recent randomized controlled trials in predominantly overweight populations from industrialized countries, which compared isocaloric diets with different levels of total fat, have shown that a higher %E fat can lead to greater weight loss than observed with low fat diets. However, the differences in the intake of other macronutrients such as amount and type of carbohydrates and the relatively high drop-out rate in some studies limit the strength of the evidence and the generalization of these results.

Various ecological data from observational studies in developing and transitional countries suggest that shifting from a lower to a higher %E fat has been associated with both lower and higher total energy intake and to unhealthy weight gain; thus, potentially contributing to the increasing problem of overweight and obesity. The opposite is observed in industrialized countries where %E fat has decreased while obesity has increased.

The insufficient evidence and conflicting interpretation of results on the nature of the relationship between the %E fat and adult body weight convinced the consultation that at this time it was not possible to determine at a **probabl**e or **convincing** level the causal relationship of excess energy intake and unhealthy weight gain.

Full agreement among the experts regarding the upper value of acceptable macronutrient distribution range (AMDR) for %E fat was not achieved; thus maintaining the current recommendation for a maximum intake level of 30 - 35% E fat was considered prudent. Further studies and a systematic review of all available evidence are needed to provide better evidence on which to base a recommendation on AMDR for %E fat that are applicable globally.

There was agreement among the experts that in populations with inadequate total energy intake, such as seen in many developing regions, dietary fats are an important macronutrient that contribute to increase energy intake to more appropriate levels.

Conclusions:

Based on the considerations provided in the preceding section, the expert consultation proposed the following AMDR which are consistent with the existing WHO/FAO recommendations (i.e. TRS 916):

Minimum total fat intakes for adults[a]
- 15%E to ensure adequate consumption of total energy, essential fatty acids, and fat soluble vitamins for most individuals.
- 20%E for women of reproductive age and adults with BMI < 18.5, especially in developing countries in which dietary fat may be important to achieve adequate energy intake in malnourished populations.

Maximum total fat intakes for adults[a]
- 30-35%E for most individuals.

[a] To optimize health, special attention should be given to both the overall dietary pattern, in terms of types of food consumed, and total energy intakes, in relation also to anthropometric (age group, BMI) and lifestyles characteristics.

Conclusions and Recommendations for saturated fatty acids (SFA)

Individual saturated fatty acids (SFA) have different effects on the concentration of plasma lipoprotein cholesterol fractions. For example, lauric (C12:0), myristic (C14:0) and palmitic (C16:0) acids increase LDL cholesterol whereas stearic (C18:0) has no effect.

There is **convincing** evidence that:
- Replacing SFA (C12:0 – C16:0) with polyunsaturated fatty acids (PUFA) decreases LDL cholesterol concentration and the total/HDL cholesterol ratio. A similar but lesser effect is achieved by replacing these SFA with monounsaturated fatty acids (MUFA).
- Replacing dietary sources of SFA (C12:0 – C16:0) with carbohydrates decreases both LDL and HDL cholesterol concentration but does not change the total/HDL cholesterol ratio.
- Replacing SFA (C12:0 – C16:0) with *trans*-fatty acids (TFA) decreases HDL cholesterol and increases the total /HDL cholesterol ratio.

Based on coronary heart disease (CHD) morbidity and mortality data from epidemiological studies and controlled clinical trials (using CHD events and death), it was also agreed that:

- There is **convincing** evidence that replacing SFA with PUFA decreases the risk of CHD.
- There is **probable** evidence that replacing SFA with largely refined carbohydrates has no benefit on CHD, and may even increase the risk of CHD and favour metabolic syndrome development. (Re: recent pooled individual data from multiple studies, Jakobsen *et al.*, AJCN:89, 2009). Reducing SFA by itself (reducing the amount of SFA or the % energy from SFA) has no effect on CHD and stroke (Re: recent pooled study data, Siri-Tarino et al., AJCN:91, 2010). However, the methodology used by Siri Tarino *et al* in the pooling of these studies has been questioned by Stamler in an Editorial (AJCN:91, 2010) who highlights the important limitations of the pooled analysis.
- There is a possible positive relationship between SFA intake and increased risk of diabetes.
- There is insufficient evidence relating to the effect on the risk of CHD in replacing SFA with either MUFA or largely whole grain carbohydrates; however, based on indirect lines of evidence this could result in a reduced risk of CHD.
- There is insufficient evidence that SFA affects the risk for alterations in indices related to the components of the metabolic syndrome.

Based on cancer morbidity and mortality data, it was also agreed that:
- There is insufficient evidence for establishing any relationship of SFA consumption with cancer.

Therefore, it is recommended that SFA should be replaced with PUFA (n-3 and n-6) in the diet and the total intake of SFA not exceed 10% E.

Conclusions and Recommendations for monounsaturated fatty acids (MUFA)

- There is **convincing** evidence that replacing carbohydrates with MUFA increases HDL cholesterol concentrations.
- There is **convincing** evidence that replacing SFA (C12:0 – C16:0) with MUFA reduces LDL cholesterol concentration and total/HDL cholesterol ratio.
- There is possible evidence that replacing carbohydrates with MUFA improves insulin sensitivity.
- There is insufficient evidence for relationships of MUFA consumption with chronic disease end points such as CHD or cancer.
- There is insufficient evidence for relationships of MUFA consumption and body weight and percent adiposity.
- There is insufficient evidence of a relationship between MUFA intake and risk of diabetes.

The determination of intake of MUFA is unique in that it is calculated by difference, i.e. Total fat intake (%E) – SFA (E%) – PUFA (E%) – TFA (%E). Therefore, the MUFA intake resulting may cover a wide range depending on the total fat intake and dietary fatty acid pattern.

Conclusions and Recommendations for polyunsaturated fatty acids (PUFA)

- There is **convincing** evidence that linoleic acid (LA) and alpha-linolenic acid (ALA) are indispensable since they cannot be synthesized by humans.
- There is **convincing** evidence that replacing SFA with PUFA decreases the risk of CHD.
- There is **convincing** and sufficient evidence from experimental studies to set an acceptable intake to meet essential FA needs for linoleic acid (LA) and alpha-linolenic acid (ALA) consumption.
- There is possible evidence that PUFA affect the risk of alterations in indices related to the metabolic syndrome.
- There is possible evidence of a relationship between PUFA intake and reduced risk of diabetes.
- There is insufficient evidence for establishing any relationship of PUFA consumption with cancer.
- There is insufficient evidence for relationships of PUFA consumption and body weight and percent adiposity.

The minimum intake levels for essential fatty acids to prevent deficiency symptoms are estimated at a convincing level to be 2.5%E LA plus 0.5%E ALA. Based on epidemiologic studies and randomized controlled trials of CHD events, the minimum recommended level of total PUFA consumption for lowering LDL and total cholesterol concentrations, increasing HDL cholesterol concentrations and decreasing the risk of CHD events is 6%E. Based on experimental studies, risk of lipid peroxidation may increase with high (>11%E) PUFA consumption, particularly when tocopherol intake is low. Therefore, the resulting acceptable range for total PUFA (n-6 and n-3 fatty acids) can range between 6 and 11%E. The adequate intake to prevent deficiency is 2.5 – 3.5%E.

Thus, the recommended range (ADMR) for PUFA is 6 – 11%E.

Conclusions and Recommendations for n-3 polyunsaturated fatty acid intake

The available evidence indicates that 0.5 to 0.6%E alpha-linolenic acid (ALA) per day corresponds to the prevention of deficiency symptoms. The total n-3 fatty acid intake can range between 0.5 - 2%E whereas the minimum dietary requirement of ALA ($\geq$ 0.5%E) for adults prevents deficiency symptoms. The higher value 2%E (ALA) plus n-3 long-chain polyunsaturated fatty acids (LCPUFA) eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) (AMDR 0.250 g – 2.0 g) can be part of a healthy diet. Whilst ALA may have individual properties in its own right, there is evidence that the n-3 LCPUFA may contribute to the prevention of CHD and possibly other degenerative diseases of aging. For adult males and non-pregnant/non-lactating adult females 0.250 g/day of EPA plus DHA is recommended, with insufficient evidence to set a specific minimum intake of either EPA or DHA alone; both should be consumed. For adult pregnant and lactating females, the minimum intake for optimal adult health and fetal and infant development is 0.3 g/d EPA+DHA, of which at least 0.2 g/d should be DHA.

The U-AMDR for EPA + DHA consumption is set at 2 g/d due to experimental evidence indicating that high supplement intakes of n-3 LCPUFA may increase lipid peroxidation and reduce cytokine production. However, this consultation also acknowledged that higher consumption levels, as high as 3 g/d reduce other cardiovascular risk factors and have not had adverse effects in short- and intermediate-term randomized trials, and that some individuals in populations with high seafood consumption consume higher levels with no apparent evidence of harm. In this regard, the experts noted that the Australian and New Zealand reference value for the upper level of intake of EPA + DPA + DHA has been set at 3 g/d and The US Food and Drug Administration (DHHS 1997) having set a 'Generally Regarded as Safe' level of 3000 mg/day for n-3 LCPUFA. Following careful consideration and extensive debate and considering the issue of sustainability of the supply of fish, the experts agreed on the value of 2 g/d as the U-AMDR for EPA plus DHA with the acknowledgement that future randomised controlled trials (RCT) and other research may justify raising this figure in the future. It was decided not to include DPA in the recommendations due to the fact that DPA is currently a research issue with limited evidence from RCT studies.

Conclusions and Recommendations for n-6 polyunsaturated fatty acids

It is recognized that only a sparse amount of human data is available for establishing a precise quantitative estimate of the linoleic acid (LA) requirement to prevent deficiency; thus a range rather than an average LA requirement is recommended. Animal and human studies demonstrate that the prevention of deficiency signs (e.g. in rats reduced growth, scaliness of skin, necrotic tail) occurs when 1 to 2% of total energy is provided by LA. Therefore, an estimated average requirement (EAR) for LA of 2%E and an adequate intake (AI) for LA of 2 – 3% E are proposed. In accepting that the U-AMDR levels of total PUFA and total n-3 fatty acids are 11% E and 2% E respectively, the resulting acceptable range (AMDR) for n-6 fatty acids (LA) intake is 2.5 to 9%E. The lower value or AI (2.5–3.5%E) corresponds to the prevention of deficiency symptoms, whereas the higher value as part of a healthy diet contributing to long term health by lowering LDL and total cholesterol levels and

therefore the risk for CHD. For infants 6 to 12 months of age as well as children 12 to 24 months of age, an AI range of 3.0-4.5%E is recommended with a U-AMDR of <10%E. There is insufficient evidence for establishing any relationship of n-6 PUFA consumption with cancer.

Arachidonic acid (AA) is not essential for a healthy adult whose habitual diet provides LA > 2.5%E. For infants 0-6 months AA should be supplied in the diet within the range of 0.2-0.3%E[1] based on human milk composition as a criteria.

Conclusions and Recommendations for n-6 to n-3 ratio

Based on both the scientific evidence and conceptual limitations, there is no compelling scientific rationale for the recommendation of a specific ratio of n-6 to n-3 fatty acids or LA to ALA, especially if intakes of n-6 and n-3 fats lie within the recommendations established in this report.

Conclusions and Recommendations for *trans*-fatty acid intake (TFA)

The Consultation devoted substantial time and discussion to the issue of *trans*-fatty acid (TFA) but in doing so drew heavily from the conclusions of the recently concluded and published reports of the *WHO Scientific Update on trans fatty acids* (Nishida & Uauy, EJCN, Vol 63, Suppl 2, 2009). There is convincing evidence that TFA from commercial partially hydrogenated vegetable oils (PHVO) increase CHD risk factors and CHD events – more so than had been thought in the past. There also is probable evidence of an increased risk of fatal CHD and sudden cardiac death in addition to an increased risk of metabolic syndrome components and diabetes. In promoting the removal of TFA, which are predominantly a by-product of industrial processing (partial hydrogenation) usually in the form of PHVO, particular attention must be given to what would be their replacement; this is a challenge for the food industry. It was noted that among adults, the estimated average daily ruminant TFA intake in most societies is low. The experts acknowledged the current recommendation of a mean population intake of TFA of less than 1%E may need to be revised in light of the fact that it does not fully take into account the distribution of intakes and thus the need to protect substantial subgroups from having dangerously high intakes. This could well lead to the need to remove partially hydrogenated fats and oils from the human food supply.

Considerations for food-based dietary guidelines

The experts agreed that in addition to dietary requirements for total fat and fatty acids, food-based dietary guidelines are essential for promoting health and preventing disease. However, the consultation did not conduct a review of this subject. A general recommendation is to follow a dietary pattern predominantly based on whole foods (i.e., fruits and vegetables, whole grains, nuts, seeds, legumes, other dietary fibre sources, LCPUFA-rich seafood) with a relatively lower intake of energy dense processed and fried foods, and sugar sweetened beverages; and to avoid consumption of large portion sizes. Moderate consumption of dairy products and lean meats and poultry can also be an important part of recommended food-based dietary guidelines. Maintaining recommended dietary patterns, appropriate energy intake and adequate physical activity levels are critical to prevent unhealthy weight levels (e.g. overweight and obesity) and to ensure optimal health for those predisposed to insulin resistance.

Recommendations for further research

Further research and investigation are needed on:

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[1] If based on human milk composition as is often the case when referring to infants of breast feeding age, the amount would be expressed as 0.4-0.6%FA. This conversion assumes that half of the energy in human milk comes from fat.

- The effects of total fat consumption as a percentage of energy on weight gain, weight, maintenance, and weight loss in developing countries;
- The effects of different saturated fatty acids of varying chain lengths on CHD, diabetes, and metabolic syndrome risk and endpoints;
- The influence of different saturated fatty acids of varying chain lengths on *de novo* synthesis of fatty acids, and the implications for health outcomes;
- The effects of monounsaturated fatty acids on CHD, diabetes, and metabolic syndrome risk and endpoints;
- The effects of n-3 and n-6 polyunsaturated fatty acids on diabetes and metabolic syndrome risk and endpoints;
- Human studies to determine the dose-dependent effects of LA and ALA on formation of long-chain PUFA as well as the assessment of conversion rates of LA to AA in relation to the intakes;
- The effects of ALA on cardiovascular outcomes;
- Establishing the adult brain daily requirement of AA and DHA and translating these into daily dietary intakes of AA and DHA;
- The effects of long chain n-3 PUFA on depression and other mood disorders; and on aggression, hostility and antisocial behaviour; These studies should include:
    - ✓ both prospective observational studies and randomized clinical trials;
    - ✓ in trials, purified preparations of long chain n-3 PUFA (alone and in combination);
    - ✓ dose response studies;
    - ✓ studies on the duration of dietary consumption required for greatest benefit;
    - ✓ larger numbers of subjects in each treatment group;
    - ✓ delineating the importance of n-3 PUFA as monotherapy or adjunct therapy and identifying the mechanism(s) of action of these PUFA in mood disorders;
    - ✓ sufficiently sensitive tests designed to measure effects in mood & cognition.
- The effects of long-chain n-3 PUFA on the prevention and treatment of cognitive decline and Alzheimer's disease, including larger and longer duration randomized clinical trials;
- The relationship of *trans* fatty acid and saturated fatty acids with prostate cancers;
- The relationship of n-3 PUFA and fish with colorectal, prostate and breast cancers, including both incidence and progression;
- Simplified, low-cost rapid methods for analyzing fatty acid profiles of biological and food samples.

Recommendations on dietary information and program needs

- To provide sufficient and adequate information on dietary fatty acid intakes, it is strongly recommended that countries monitor food consumption patterns of their population groups; data on country-specific fatty acid composition of foods, on bioavailability of fatty acids from food sources and supplements, and on biomarker levels in specific populations are also required for designing and monitoring the impacts of national dietary guidelines and programmes that are aiming to make changes in dietary patterns over time to improve nutrition, including the promotion of appropriate intakes of different dietary fats and oils.

- Fatty acid analysis of whole blood is a representative biological specimen for the assessment of the fatty acid status in tissues in relation to physiopathological conditions; Analysis of whole blood or other samples (e.g., adipose, erythrocytes, phospholipids) should be conducted to monitor the fatty acid status in populations; This information is useful in relating to dietary fat intakes to health outcomes; whole blood analyses can be performed on drops of blood collected from fingertips.

Recommendations for Nomenclature

The following definitions for the sub-classes of saturated fatty acids are recommended:

- *Short-chain fatty acids*: These are fatty acids with carbon atoms from three to seven.
- *Medium chain fatty acids*: These are fatty acids with carbon atoms from eight to thirteen.
- *Long-chain fatty acids*: These are fatty acids with carbon atoms from fourteen to twenty.
- *Very-long chain fatty acids*: These are fatty acids with twenty one or more carbon atoms.

The following designations for the sub-classes of polyunsaturated fatty acids are recommended:

- *Long-chain polyunsaturated fatty acids*: These are polyunsaturated fatty acids with twenty to twenty four carbon atoms.
- *Very-long chain polyunsaturated fatty acids*: These are polyunsaturated fatty acids with twenty five or more carbon atoms.

ABBREVIATIONS

Measure

AI	adequate intake (expressed as a range)
EAR	estimated average requirement
AMDR	acceptable macronutrient distribution range;
L-AMDR°	lower level of acceptable macronutrient distribution range°;
U-AMDR°	upper level of acceptable macronutrient distribution range°;
UL	upper level; this term was developed for instances where biochemical indicators are needed to confirm any adverse effects, measurable with a probability of occurrence. In the case of fatty acids this only applies to *trans* fatty acids.

° These two terms refer to the upper and lower range of the AMDR, very much similar to the use in statistics of the upper and lower bounds of confidence intervals (UCI and LCI).

Other abbreviations used in tables and in the text

%E	percent of energy
%FA	percentage fatty acid composition ("wt:wt")
AA	Arachidonic acid
ALA	Alpha-linolenic acid
CHD	Coronary heart disease
CLA	Conjugated Linoleic Acid
DHA	Docosahexaenoic acid
DPA	Docosapentaenoic acid
EPA	Eicosapentaenoic acid
HDL	High density lipoprotein
HM	Human milk
LA	Linoleic acid
LCPUFA	Long Chain Polyunsaturated Fatty Acid ($\geq$ 2 double bonds; $\geq$ 20 C atoms)
LDL	Low density lipoprotein
MUFA	Monounsaturated Fatty Acid(s)
PUFA	Polyunsaturated Fatty Acid(s) (2 or more double bonds)
PHVO	Partially Hydrogenated Vegetable Oil

RCT   Randomised controlled trials
SFA   Saturated Fatty Acid(s)
TAG   Triacylglycerol(s)
TFA   Trans Fatty Acids(s)

Specific Fatty Acids IUPAC notation; trivial name (if common); systematic name

LA   18:2n-6 ; linoleic acid ; 9z,12z-octadecadienoic acid
ALA   18:3n-3 ; alpha linolenic acid ; 9z,12z,15z-octadecatrienoic acid;
EPA   20:5n-3 ; timnodonic acid ; 5z,8z,11z,14z,17z-eicosapentaenoic acid
DHA   22:6n-3 ; cervonic acid ; 4z,7z,10z,13z,16z,19z-docosahexaenoic acid
AA   20:4n-6 ; arachidonic acid ; 5z,8z,11z,14z-eicosatetraenoic acid

Note: C:Dn-#, where C=number of C atoms: D=number of double bonds and # = number of C atoms the first double bond is separated from the Methyl group; n-6 (IUPAC notation) = ω6 (Holman notation);