Key Message 8

Limit intake of foods high in fats and minimise fats and oils in food preparation

Key Message 8

Limit intake of foods high in fats and minimise fats and oils in food preparation

1. Terminology

Cholesterol

Cholesterol is a waxy-like substance, with a molecular nucleus similar to that of other steroids like the male hormone, testesterone and vitamin D. It is found in significant amounts in animal food products but occur as only traces in foods of plant origin. As such, plant foods are usually considered as "cholesterol-free", that is, having a cholesterol content of <5mg/100g food. Cholesterol is needed by the body for steroid hormone synthesis and is broken down mainly as bile acid. The primary source of cholesterol for the body is produced by the liver itself, while the diet provides a much smaller amount by comparison (Whitney & Sharon, 2005).

Essential fatty acids

Essential fatty acids (EFAs) refer to the polyunsaturated omega-6 fatty acid, linolenic acid (C18:2) and the polyunsaturated omega-3 fatty acid, alpha-linolenic acid (C18:3) (Figure 8.1).

These fatty acids cannot be synthesised by the human body and must therefore be provided by the diet. "Omega" in fatty acid nomenclature refers to the position of the first carbon-carbon double bond counting from the methyl (CH_3) end of the molecule.

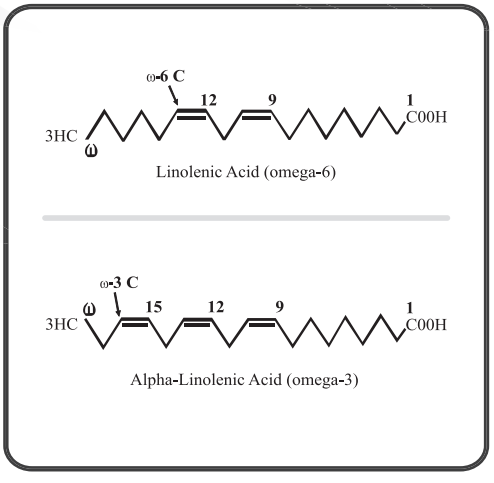


Figure 8.1. Essential fatty acids

Percentage kilocalories

Percentage kilocalories (% kcal) is the ratio of the calorie content of a dietary component (for instance, fat, protein, carbohydrate and fatty acid) to the total calorie intake, expressed as a percentage. For example, an intake of 60 grams of fat for a 2000 kcal diet is equivalent to:

$$\frac{60 \text{ g} \times 9.0 \text{ kcal/g} \times 100\%}{2000} = \frac{54}{2} = 27\% \text{ kcal}$$

Key Message 8

Saturated fatty acids

Saturated fatty acids (SFAs) from dietary fats and oils do not carry any carbon-carbon double bonds in the fatty acid chain. SFAs common to the human diet have 12 to 18 carbons, namely lauric acid (C12:0), myristic acid (C14:0), palmitic acid (C16:0) and stearic acid (C18:0). SFAs of 12 to 16 carbons are known to raise blood cholesterol levels, whilst stearic acid and SFAs less than 12 carbons are considered neutral.

Trans fatty acids

Trans fatty acids (TFAs) arising from the hydrogenation of unsaturated fatty acids carry a *trans-configuration* of at least one of the carbon-carbon double bonds (Figure 8.2). Commercial hydrogenation of edible oils and fats, produce ("hardened") fats that are more stable (longer shelf life) with semi-solid properties which render them suitable for the production of margarines, *vanaspati* (vegetable ghee), shortenings and bakery products. The most common TFA is elaidic acid arising from hydrogenation of vegetable oils whilst natural forms such as vacennic acid are found in ruminant meat and dairy products such as cheese and butter. The term "trans fatty acids" in this report does not include conjugated linoleic acid (CLA) formed from vaccenic acid in ruminants.

Unsaturated fatty acids

Unsaturated fatty acids from dietary oils carry one or more carbon-carbon double bonds in their molecular structure. There are two types of unsaturated fatty acids, namely monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA). The predominant MUFA in the diet is oleic acid whilst PUFAs include the omega-3 and omega-6 fatty acids.

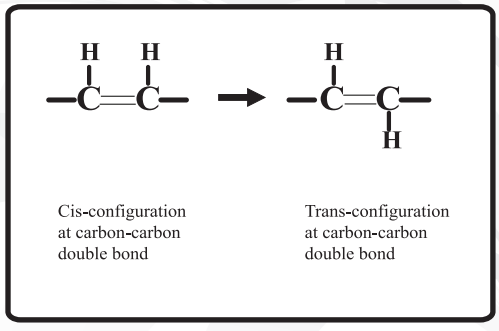


Figure 8.2. Conversion of cis-isomer to trans-isomer of fatty acids

2. Introduction

Major dietary fats are triacylglycerol molecules carrying a variety of fatty acids such as saturated (SFA), monounsaturated (MUFA) and polyunsaturated fatty acids (PUFA) with some minor components such as phospholipids, steroids (such as cholesterol), terpenes, waxes and the fat-soluble vitamins (A, D, E and K). Fats and oils are part of a healthful diet as they are an important source of energy (9.0 kcal/g), provide the essential fatty acids (EFAs) which the human body cannot synthesise as well as facilitate the absorption of the fat-soluble vitamins. Fats also impart taste and flavour to food thereby enhancing their palatibility. It is important to understand that in the Malaysian context, a large percentage of fatty acids in the diet is derived from vegetable oils and it is important to appreciate that these vegetable oils are all mixtures of PUFA, MUFA and SFA in varying proportions.

The daily diet should contain a minimum amount of fat to minimise the risk of inadequate intakes of EFA. In the Malaysian Dietary Guidelines 1999, the minimum amount of dietary fat recommended was 15% kcal or 33 g based on a 2000 kcal diet.

Key Message 8

There are indications that there is a need to raise this lower limit and this report shall address this issue among others, as too low a fat intake may contribute to unfavourable changes in plasma low-density lipoprotein cholesterol (LDL-C) and triglycerides (TGs).

Contrarily, an excess of daily consumption of dietary fat with regard to the type as well as the total amount of fat also adversely influences cardiac health and contributes to excess intake of calories. Cholesterol raising effects are associated with long-term high consumption of SFAs namely laurate, myristate and palmitate but a number of meta-analyses indicate that the effects are not similar with myristic being the most potent (Hegsted *et al*., 1965; Mensink & Katan, 1992; Clarke *et al*., 1997). Recent scientific evidence also indicated that the trans fatty acids formed in hydrogenated ("hardened") polyunsaturated vegetable oils used in semi-solid fat applications such as margarines, *vanaspati* (vegetable ghee), shortenings and bakery products are more detrimental to cardiac health than SFA (Ascherio *et al*., 1999; IOM, 2002).

3. Scientific basis

It is well-documented that genetic predisposition and environmental factors, which include an unhealthy diet, are major contributing factors to ischaemic heart disease (IHD) and cerebrovascular diseases - the top causes of mortality in Malaysia for many years now (WHO, 2006). Arterogenesis is a major contributory factor in cardiovascular disease and it is well established that dietary saturated fat and cholesterol contribute to the process through elevating total cholesterol and LDL-C (AHA, 2000). A prevalence study reported a 20% incidence of hypercholesterolaemia in the Malaysian population (Lim *et al*., 2000). Towards this end, Malaysians would benefit from a diet low in saturated fat and cholesterol.

3.1 Rationale to reduce SFA consumption

The Japan-Honolulu-San Francisco Study established strong correlations between SFA intake and coronary heart disease (CHD) incidence. Prospective population studies also indicated significant positive associations between SFA and CHD mortality. These include the Western Electric Study (Shekelle *et al*., 1981), Honolulu Heart Program (McGee *et al*., 1984) and Ireland-Boston Diet-Heart Study (Kushi *et al*., 1985).

Controlled human feeding trials predict that a major reduction in SFA would also lower blood total cholesterol by as much as 10% to 15% or about 0.8 mmol/l (Mensink & Katan, 1992; Clarke *et al*.,1997). It is thus advisable to restrict SFA consumption to less than 10% kcal in order to minimise the risk of raising blood total cholesterol.

3.2 Rationale to increase PUFA (linolenic acid) consumption

Cross-population studies such as the Eighteen Countries Study (Hegsted & Ausman, 1988) and Forty Countries Study (Artaud-Wild *et al*., 1993), found a negative correlation between PUFA intake and CHD mortality. Similarly, in the Multiple Risk Factor Intervention Trial (MRFT) involving 12,866 high-risk men aged 35 to 57 years, a beneficial relationship between PUFA intake and CHD mortality was reported (Dolecek, 1992).

Key Message 8

In the Nurses' Health Study conducted by the Harvard School of Public Health, 80,082 healthy women nurses, aged 34 to 59 years, were followed over 14 years (1980 to 1994). At baseline, none of the women had CHD. During the 14 years of follow-up, there were 939 cases of non-fatal heart attacks or death from CHD (Hu *et al*.,1997). Multivariate analyses of the food consumption data collected indicated polyunsaturated fat intake was found to be significantly inversely associated with CHD risk (Hu *et al*., 1997).

Blood cholesterol predictive equations established in the 1960s indicate that *n*-6 PUFA has the ability to decrease total blood cholesterol levels (TC) by approximately twice the cholesterol raising potential of SFAs (Keys, Anderson & Grande, 1965; Hegsted & Ausman, 1988). However, evidence from meta-analysis of the newer human feeding trials indicated that the cholesterol-lowering potential of *n*-6 PUFA is much lower than that previously reported (Mensink & Katan, 1992). Nevertheless, moderate replacement of SFA in the diet with *n*-6 PUFA (predominantly linoleic acid or C18:2) is desirable for achieving healthy population nutrient goals (WHO/FAO, 2003).

3.3 Effect of dietary cholesterol

Dietary cholesterol has been considered as second to SFA in its ability to raise blood concentrations of cholesterol. Epidemiological studies support a strong, independent correlation between dietary cholesterol and risk of CHD; these include the Western Electric Study (Shekelle *et al*., 1981), Honolulu Heart Program (McGee *et al*., 1984) and the Zutphen Study (Kromhout & Coulander, 1984).

The human liver maintains a daily turnover of about 1,000 mg of body cholesterol with an additional 100 to 500 mg absorbed from the diet. Both Keys *et al*., (1965) and Howell (1965) and Howell *et al*., (1997) estimated that an increased intake of 100 mg dietary cholesterol per day will produce only a small increase in blood cholesterol, namely 0.057 mmol/L (~3 mg/dl). Nevertheless, findings from epidemiological and dietary intervention trials support dietary modifications to limit cholesterol intake and as such, dietary cholesterol consumption should be below 300 mg per day (WHO/FAO, 2003). When making food choices, use Figure 8.3 as a guide to the cholesterol content of common foods.

3.4 Omega-3 fatty acids

These refer to the plant-derived *n*-3 fatty acids (α-linolenic acid or C18:3) and marine-derived *n*-3 fatty acids (eicosapentaenoic (EPA, C20:5) and docosahexaenoic acids (DHA, C22:6)). Alpha-linolenic acid is one of two essential fatty acids, while EPA and DHA are its long-chain PUFA derivatives.

EPA is the precursor of the series 3 eicosanoids ("local hormones") which tend to oppose the adverse effects (vasoconstriction, fever, pain and inflammation) of the series 2 eicosanoids derived from arachidonic acid. For this reason, EPA-derived eicosanoids are regarded as "good" while those produced from arachidonic acid as "bad" (Kris-Etherton *et al*., 2000).

Omega-3 fatty acids have a mixed effect on human lipoprotein metabolism. The marine-based fatty acids are highly potent in decreasing blood triglycerides by 25% in normal individuals and this beneficial effect is enhanced to 34% in hypertriglyceridaemic

Key Message 8

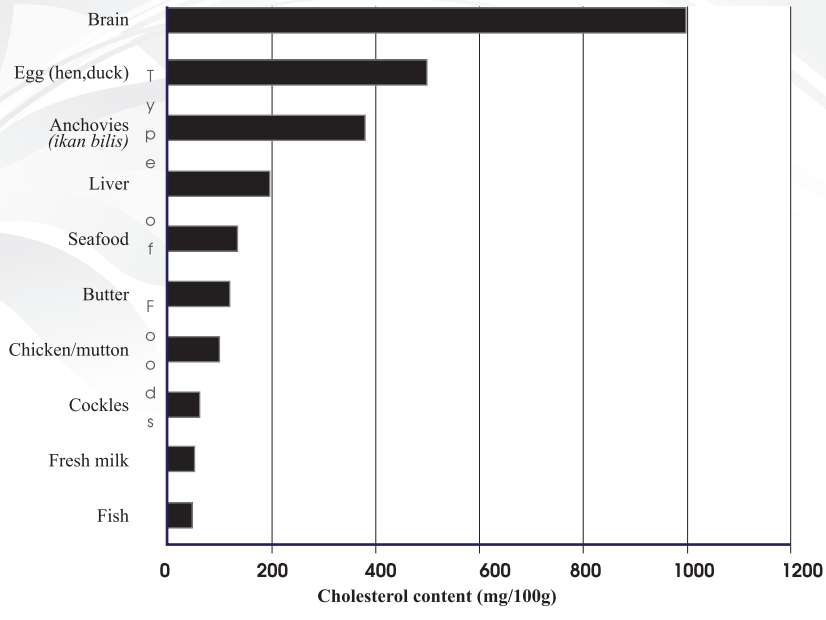


Figure 8.3. Cholesterol content of common foods
Source : Tee *et al.,* (1997).

individuals (Kris-Etherton, Harris & Appel, 2002; Lada & Rudel, 2003). On the other hand, the hypotriglyceridaemic effect of plant-derived *n*-3 fatty acids (alpha-linolenic acid) is only marginal by comparison.

Given the mixed role of *n*-3 fatty acids on cardiovascular risk, a regular consumption of natural sources such as fish is advisable at this point of time. A dietary approach (food-based) to increasing omega-3 fatty acid intake is preferable. This would be to include a minimum of two servings of fish per week (oily fish), using vegetable oil such as soya bean and canola oil and nuts as part of a healthy diet for the general population (Ng, 2006).

3.5 EFAs and minimum requirement

The biological importance of omega-6 fatty acids relates to growth, fertility and strengthening skin and red blood cell structure, whereas omega-3 fatty acids are important for cell structure and function of the retina and central nervous system. A deficiency causes growth failure in infants, dermatitis, poor wound healing, impaired vision and hearing, liver and kidney abnormalities and early death ensues. Skin problems are hallmarks of adult weight loss diets that are low fat or in young infants and children who are not consuming enough fat. These events connect to the human body's specific requirement for linoleic acid or alpha-linolenic acid and are therefore deemed as essential fatty acids (EFAs) (Kris-Etherton *et al*., 2000).

Humans depend primarily on plant sources for supplying EFAs. The "starter" EFAs generate even longer chain arachidonic, eicosapentaenoic (EPA) and docosahexaenoic

(DHA) fatty acids which form either biologically active compounds called eicosanoids that regulate blood clotting, blood pressure and other processes or contribute to formation of cell membranes (Hornstra & Lussenburg, 1975).

A minimum amount of long chain fatty acids which includes omega-6 fatty acids and omega-3 fatty acids is therefore essential to human health. In this connection, WHO/FAO (2003) has recommended a minimum EFA intake of 3% kcal (as linoleic acid) with daily needs increased by 2% to 4% kcal during pregnancy and lactation.

EFA deficiency is rare with diets providing a minimum total dietary fat of approximately 15% to 20% energy. A linoleic acid supply from vegetable oils by and large remains the most economical and practical source of EFAs in our diets.

In premature (<32 weeks) or low birth weight babies who have increased linoleic acid demand, supplementation with arachidonic acid and DHA is necessary to optimise normal brain development. There is much debate on the correct ratio of omega-3 and omega-6 PUFAs necessary to drive these processes, but the ratio one part omega-3:5 to 10 parts omega-6 has been recommended by WHO/FAO (2003).

3.6 Rationale to exclude TFA from the diet

The concern about TFA is that it has a strong causal link to CHD risk. In the Nurses' Health Study mentioned earlier, this 14-year prospective cohort study reported positive relationships with relative risk at 1.5 times in the highest quintile group of TFA intake (5.7 g/d), compared to those in the lowest quintile of intake (2.4 g/d)(Willet *et al*., 1993). The positive association was attributed to TFA intake patially hydrogenated vegetable fats rather than isomers from ruminant sources. After adjusting for total energy consumption, the relative risk for developing CHD was 50% greater in the highest quintile compared to those in the lowest quintile of TFA consumers.

Similarly, Ascherio *et al*., (1994) from the Harvard School of Public Health, USA found from a population retrospective study that the highest intake of TFA fat (6.5 g/day) more than doubled (2.4 times) the risk of myocardial infarction (MI) compared to the lowest consumption of TFA (1.7 g/day). Thus, the consumption of TFA is a public health concern, particularly in communities where commercially hydrogenated fats contribute to the bulk of dietary fat.

A meta-analysis comparing the impact of TFAs and SFAs on the LDL: HDL-C ratio, found the effect to be dose-dependent and that TFAs raise the LDL-C/HDL-C ratio (atherogenic effect) about two times higher than the C12-16 SFAs (Figure 8.4) (Ascherio *et al*., 1999). Subsequently, A WHO Expert Consultation (2003) and the United States Institute of Medicine (IOM, 2002) recommend that the TFA content in the diet should not exceed 1% kcal. This upper limit for TFA in the diet was adopted in the Recommended Nutrient Intakes for Malaysia (NCCFN, 2005).

The following commercially available foods are likely sources of trans fats, especially if they are imported:

1. Stick margarines imported from abroad.
2. *Vanaspati* (vegetable ghee), especially imported varieties.
3. Partially hydrogenated edible oils.
4. Bakery products containing partially hydrogenated fats.

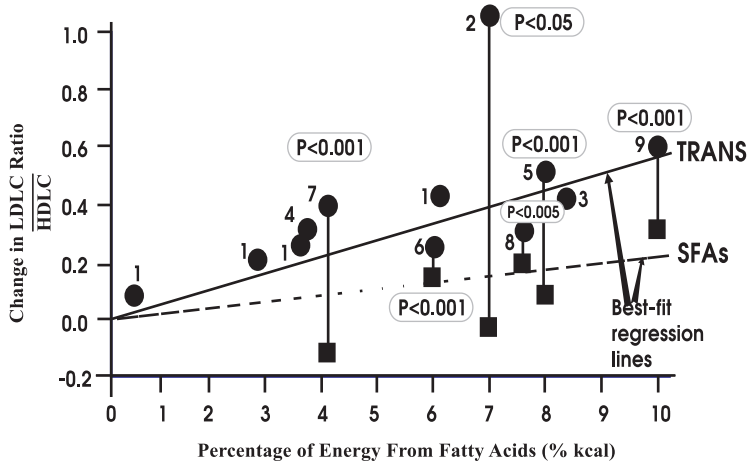
Key Message 8

3.7 Total daily fat intake should be moderate

The caloric density of 1g of fat is 9 kcal. This is almost twice the caloric capacity of 1g of either carbohydrate or protein (4 kcal/g). Excess consumption of fats through deep-fried foods, cream-filled confectionery, cheese and sauces will therefore be a source of calories. Obesity arises from excess energy consumption coupled with reduced energy expenditure. The regular consumption of a high fat diet will contribute to this problem. In the United States, excess fat consumption is indicated by dietary fat intakes greater than 35% kcal.

The digestion, absorption and metabolic disposal of dietary fat is dependent on the total fat content of the meal. A high fat-loaded meal is contributory to a rise in plasma triglycerides which is an independent risk factor for the arterogenesis (Patsch *et al*., 1992).

A moderate fat consumption of less than 30% kcal total fat is in the WHO diet model and this is recommended as a desirable level of fat intake for Malaysians. Compared to the Malaysian Dietary Guidelines (1999), the minimum level of dietary fat has been raised from 15% kcal to the present recommended minimum of 20% kcal, while the desirable range recommended for dietary fat remains unchanged at 20% to



[9]Mensink and Katan (1990) ; [8]Zock and Katan (1992); [7]Nestel *et al*., (1992); [6]Judd *et al*., (1994); [5]Judd *et al*., (1998); [4]Lichtenstein *et al*., (1993); [3]Aro *et al*., (1997); [2]Sundram *et al*., (1997); [1]Lichtenstein *et al*., (1999)

Figure 8.4. Impact of TFAs vs SFAs on plasma LDLC/HDLC ratio

Source : Ascherio *et al*., (1999)

30% kcal. Table 8.1 provides examples of total dietary fat in the diet for this range at various calorie levels.

4. Current status

An extensive food consumption survey (Malaysian Adult Nutrition Survey, MANS) was carried out in 2003 for the first time in Malaysia (MOH, 2007). Data from this survey indicates that mean fat intake is about 50 g for the Malaysian population with about a difference of 9 g between men (54 g) and women (45 g) (Mirnalini *et al*., 2008). Similar fat intakes were noted in both urban and rural residents. Given that mean calorie intake was 1776 kcal in men and 1447 kcal in women, the contribution of fat to these mean total daily energy intakes works out to be 27% and 28% fat respectively. This data indicates that adult Malaysians on average are deriving between 20% to 30% of their total energy intake from fat consumption.

Further information on habitual food consumption data from the MANS study indicates that about 40% of Malaysians consume about 100 g of marine fish daily with the practice more prevalent in rural (51%) compared to urban residents (34%) (Norimah *et al*., 2008). About 12% of the population were reported to consume an egg daily compared to 72% who consume about three eggs weekly. The practice of consuming more eggs was more prevalent in men compared to women and urban compared to rural residents. An excess consumption from the meat group was almost three times the recommended servings (nine vs two to three servings per day) in the Malaysian Food Pyramid. However, an excess consumption of the meat group would pose a risk in increasing saturated fatty acid if this intake consists red meat. Since the MANS data did not qualitatively and quantitatively report the intake of red meat, no conclusion can be made currently on the Malaysian intake of SFA, from this source.

Table 8.1. Desirable amount of dietary fat at selected calorie levels

	Lower and upper limits for desirable dietary fat intake							
	Lower Limit (20 % kcal)				Upper Limit (30 % kcal)			
Total calorie intake (kcal)	1500	2000	2500	2800	1500	2000	2500	2800
Dietary fat (g)	34	45	56	63	50	67	83	94
Maximum amount of SFA intake (g) to keep SFA below 10% kcal	17	22	28	31	17	22	28	31

Key Message 8

5. Key recommendations

Key recommendation 1

Limit the intake of saturated fats to less than 10% of total daily calorie intake.

How to achieve

1. Limit the use of oils and fats such as coconut oil, coconut milk (*santan*), palm kernel oil and animal fat (ghee or butter).

2. Trim the fat from meat or poultry before cooking.

3. Remove skin of poultry before cooking.

4. Use low fat or skimmed dairy products.

Key recommendation 2

Increase the intake of unsaturated fats (MUFA and PUFA).

How to achieve

1. Palm oil is a good source of MUFA, while corn oil, soya bean and sunflower oils are good sources of PUFA. Using these vegetable oils in cooking will improve the intake of MUFA and PUFA. For example, mix one part of palm olein with one part of soya bean oil as a cooking oil blend.

2. Increase consumption of foods such as nuts (groundnuts, cashew nuts, almonds and pistachios) and seeds (sesame and sunflower seeds) and legumes (chickpeas and soya bean).

3. Increase consumption of fresh fish such as *siakap, cencaru, selar kuning, bawal hitam, senangin, tongkol, kembung, tenggiri* or sardines. Canned sources of fish such as tuna and sardines can also be consumed.

Key Message 8

Key recommendation 3

Limit intake of foods high in cholesterol.

How to achieve

1. Limit organ meats (especially brain, heart, kidney and liver) and fish roe to less than twice a month.

2. Remove the head of *ikan bilis* and prawns before cooking.

3. Consume eggs in moderate amount, up to an average (whole or in dishes) of one egg a day. For individuals with hypercholesterolaemia, limit eggs to three per week.

Key recommendation 4

Limit foods containing trans fatty acids (TFAs).

How to achieve

1. Limit intake of margarines and shortenings made from hydrogenated or "hardened" fats.

2. Limit intake of foods prepared with partially hydrogenated or "hardened" fats such as french fries, doughnuts and bakery products.

3. Look for words such as "partially hydrogenated fats or oils or 'hardened' fats or oils" on the food label of processed foods as these contain TFAs.

Key Message 8

Key recommendation 5

Minimise the use of fat in food preparation in order to keep total daily fat intake between 20% to 30% energy.

How to achieve

1. Limit deep frying, shallow frying and batter frying when cooking.
2. Modify recipes which use excessive oils and fats such as *sambal tumis*, *goreng berlada* and *nasi minyak.*

Key recommendation 6

When eating out, choose low fat foods.

How to achieve

1. Choose dishes using minimal oil, fat or *santan* in the preparation.
2. Choose high fat foods less frequently.
3. Include vegetables and fruits in your meal choices.
4. Eat at places which provide a wider variety of healthy meal options.
5. Request for less fat and oil when ordering food.

Key Message 8

References

AHA Dietary Guidelines (2000). Revision: A statement for healthcare professionals from the Nutrition Committee of the American Heart Association. *Circulation* 102:2284-2299.

Ascherio A, Hennekens CH, Buring JE, Master C, Stampfer MJ & Willet WC (1994). Trans-fatty acids intake and risk of myocardial infarction. *Circulation* 89:94-101.

Ascherio A, Katan MB, Zock PL, Stampfer MJ & Willett WC (1999). Trans fatty acids and coronary heart disease. *N Engl J Med* 340(25):1994-1998.

Artaud-Wild SM, Connor SL, Sexton G & Connor WE (1993). Differences in coronary mortality can be explained by differences in cholesterol and saturated fat intakes in 40 countries but not in France and Finland. A paradox. *Circulation* 88:2771-2779.

Clarke R, Frost C, Collins R, Appleby P & Peto R (1997). Dietary lipids and blood cholesterol: quantitative meta-analysis of metabolic ward studies. *Br Med J* 314:112-117.

Dolecek TA (1992). Epidemiological evidence of relationships between dietary polyunsaturated fatty acids and mortality in the multiple risk factor intervention trial. *Proc Soc for Exp Biol and Med* 200:177–182.

Hegsted DM & Ausman LM (1988). Diet, alcohol and coronary heart disease in men. *J Nutr* 118:1184-1189.

Hegsted DM, McGandy RB, Myers ML & Stare FJ (1965). Quantitative effects of dietary fat on serum cholesterol in man. *Am J Clin Nutr* 17:281-295.

Hornstra G & Lussenburg RB (1975). Relationship between the type of dietary fatty acid and arterial thrombosis tendency in rats. *Atherosclerosis* 22:499-519.

Howell WH, McNamara DJ, Tosca MA, Smith BT & Gaines JA (1997). Plasma lipid and lipoprotein responses to dietary fat and cholesterol: a meta-analysis. *Am J Clin Nutr* 65:1747-1764.

Hu FB, Stampfer MJ, Manson JE, Rimm E, Colditz GA, Rosner BA, Hennekens CH & Willet WC (1997). Dietary fat intake and the risk of coronary heart disease in women. *N Eng J Med* 337:1491-1499.

IOM (Institute of Medicine) (2002). Dietary Reference Intakes for Energy, Carbohydrates, Fiber, Fat, Protein and Amino Acids (Macronutrients). National Academy Press, Washington DC.

Key Message 8

Keys A, Anderson JT & Grande F (1965). Serum cholesterol response to changes in the diet: IV. Particular saturated fatty acids in the diet. *Metabolism* 14:776-786.

Kris-Etherton PM, Harris WS & Appel LJ (2002). Fish consumption, fish oil, omega-3 fatty acids and cardiovascular disease. AHA Scientific Statement. *Circulation* 106:2747-2757.

Kris-Etherton PM, Taylor SD, Yu-Poth S, Huth P, Moriarty K, Fishell C, Hargrove RL, Zhao G & Etherton TD (2000). Polyunsaturated fatty acids in the food chain in the United States. *Am J Clin Nutr (Suppl.)*:179-188.

Kromhout D & Coulander CDL (1984). Diet prevalence and 10-year mortality from coronary Heart disease in 871 middle-age men. The Zutphen Study. *Am J Epidemiol* 119:733-741.

Kushi LH, Lew RA, Stare FJ, Ellison CR, el Lozy M, Bourke G, Daly L, Graham I, Hickey N & Mulcahy B & Kevancy J (1985). Diet and 20-year mortality from coronary heart disease: the Ireland-Boston diet-heart study. *N Engl J Med* 312(13):811-818.

Lada AT & Rudel LL (2003). Dietary monounsaturated versus polyunsaturated fatty acids: which is really better for protection from coronary heart disease? *Curr Opin Lipidol* 14:41–46.

Lim TO, Ding LM, Zaki M, Ismail M, Kew ST, Maimunah AH, Rozita HH & Rugayah B (2000). Clustering of hypertension, abnormal glucose tolerance, hypercholes terolaemia and obesity in Malaysian adult population. *Med J Malaysia* 55:196-208.

McGee DL, Reed DM, Yano KY, Kagan A & Tillotson (1984). Ten-year incidence of coronary heart disease in the Honolulu Heart Program. Relationship to nutrient intake. *Am J Epidemiol* 119:667-676.

Mensink RP & Katan MB (1992). Effect of dietary fatty acids on serum lipids and lipoproteins. A meta-analysis of 27 trials. *Arterioscler Thromb Vasc Biol* 12:911-919.

Mirnalini K, Zalilah MS, Safiah MY, Tahir A, Siti Haslinda MD, Siti Rohana D, Khairul Zarina MY, Mohd Hasyami S & Normah H (2008). Energy and nutrient intakes: Findings of Malaysia Adult Nutrition Survey (MANS). *Mal J Nutr* 14(1): 1-24.

MOH (2007). Seminar on findings of MANS 2003. Nutrition Section, Family Health Development Division, Ministry of Health Malaysia, Putrajaya.

NCCFN (National Coordinating Committee on Food and Nutrition) (2005). Recommended Nutrient Intakes for Malaysia (RNI). A Report of the Technical Working Group on Nutritional Guidelines.Ministry of Health Malaysia, Putrajaya.

Key Message 8

Ng TKW (2006). Omega-3 fatty acids: Potential sources in the Malaysian diet with the goal towards achieving recommended nutrient intakes. *Mal J Nutr* 12(2):181-188.

Norimah AK, Safiah MY, Jamal K, Siti Haslinda MD, Zuhaida H, Rohida S, Fatimah S, Siti Norazlin, MN Poh BK, Kandiah M, Zalilah MS, Wan Manan WM & Fatimah S (2008). Food consumption patterns: findings from the Malaysian Adult Nutrition Survey (MANS). *Mal J Nutr* 14:205-40.

Patsch JR, Miesenbock G, Hopferwieser T, Muhlberger V, Knapp E, Dunn JK, Gotto Jr AM & Patsch W (1992). Relation of triglyceride metabolism and coronary artery disease: studies in the postprandial state. *Arterioscler and Thromb* 12: 1336 –1345.

Shekelle RB, Shryock AM, Paul O, Lepper M, Stamler J, Liu S & Raynor WJ (1981). Diet, serum cholesterol, and death from coronary heart disease. The Western Electric Study. *N Engl J Med* 304:65-70.

Tee ES, Ismail MN, Mohd Nasir A & Khatijah I (1997). Nutrient composition of Malaysian foods (4th ed). Institute of Medical Research, Kuala Lumpur.

United States Department of Agriculture (2005). Dietary Guidelines for Americans 2005. *www.healthusgov/dietaryguidelines* (Accessed on 19 November 2008).

Whitney ER & Sharon R (2005). Understanding Nutrition. Thomson Wadsworth, United States.

Willett, WC, Stampfer MJ, Manson JE, Colditz GA, Speizer FE, Rosner BA, Sampson LA & Hennekens CH (1993). Intake of trans fatty acids and risk of coronary heart disease among women. *Lancet* 341: 581-585.

WHO (2006). Mortality Country Fact Sheet 2006. Regional Office for the Western Pacific, Manila, Philippines.

WHO/FAO (2003). Diet, nutrition and the prevention of chronic diseases, WHO Technical Report Series 916, World Health Organization, Geneva.