Review article

The Fallacy of Cholesterol-free Vegetable-oil and the Effects of Fatty Acid Composition of Vegetable Versus Fish Oils on Health and Diseases

Md. Shahdat Hossain[1], Ishtiaq Mahmud[2], Hossain Uddin Shekhar [3], Yearul Kabir[3] and Michio Hashimoto[4]*

Department of Biochemistry and Molecular Biology, Jahangirnagar University, Savar, Dhaka[1], Department of Food Science and Nutrition, CAMS, Sultan Qaboos University, Oman[2], Department of Biochemistry and Molecular Biology, University of Dhaka[3] and Department of First Physiology, Shimane Medical University, Izumo 693-8501, Japan[4].

Abstract

This report will first discuss the presence of sterol in vegetable oils and then review basic features about the fatty acid composition of different edible oils of plant and fish origin. Vegetable oils namely palm, olive, soy, corn and a wide variety of other seeds are of plant origin. On the other hand, cholesterol is synthesized predominantly in animal tissues, while it is nearly absent and/or, if present only in trace amounts in plant tissues. Thus 'cholesterol-free vegetable oil' does not have scientific basis and it could be considered as a misleading information for the consumers. An important component of this review is to disseminate information on cholesterol and other sterols to help formulate guideline(s) on edible oils, at least, for the population that desires a good maintenance of health on the basis of available scientific evidence. This article also describes the comparative effects of vegetable vs. fish oils on health and diseases with a special reference to the saturated and (poly)unsaturated fatty acid contents of these edible oils. In addition, the emergence of structural modification of vegetable oils would be discussed. Finally, some suggestions for the ingestion of fats in terms of edible oils are proposed on the basis of epidemiological and experimental evidence.

Key wards: Vegetable oil, fish oil, hypercholesterolemia, saturated and polyunsaturated fatty acids

Introduction

Research on cholesterol has been in continuous progress for last two centuries, thus indicating its crucial impact on human life. Cholesterol is referred to as 'double-

Bangladesh Journal of Nutrition. Vol 16 December 2003. Institute of Nutrition and Food Science, University of Dhaka-1000, Bangladesh

* Author for correspondence

edged blade' due to its roles as both a friend and a foe[1]. Without it cell could not be thought of being constructed, while increased concentrations of cholesterol in the body can be lethal for the organism as a whole and produce severe alterations in the blood vessels leading to atherosclerosis, a life-threatening problem for human population. Though mammalian cell is capable of producing its own cholesterol, but it does also obtain dietary cholesterol through blood. Thus the amount of cholesterol in the diet plays crucial role in determining the severe disease states of the body. Accordingly, intake of cholesterol-free food products is advocated to decrease mortality from cardiovascular diseases (CVD). This document will primarily deal with the issue of 'cholesterol-free vegetable oil,' as advertised by different oil-producers in Bangladesh and will then address the background for the interest in designing of an edible vegetable oil that would exert minimal lipidemic-load on plasma and cardiovascular health as well.

On the other hand, voluntary choices of edible oil can give rise to disproportionate intake of saturated versus polyunsaturated fatty acids and n-6 versus n-3 polyunsaturated fatty acids. It is anticipated that the saturated, n-6 and n-3 polyunsaturated fatty acid (PUFA) contents of these oils have an influence on human health and disease processes, if so, then what should be the ideal composition of edible oils to minimize their effects on disease processes. The n-6 and n-3 PUFA ratio in the Paleolithic diet was believed to be less than 1.0[2], however, this ratio has dramatically been increased in the present day human diets in different countries including United Kingdom, United States of America and Japan[3]. In the light of the altered n-6/n-3 ratios, the association of CVDs with the n-6/n-3 ratio would be discussed. Also we will stress on the intake of fish oil rich in n-3 PUFAs as a way to reduce cardiovascular diseases. Attentions have been paid to explore the probable correlation between intake of edible vegetable oils and increased prevalence of diabetes and diabetes-related complications in Bangladesh.

Obesity and its interaction with consumers'-producers' interest

The present era of automation is a major contributor to obesity. Moreover, an increase in energy intake owing to a palatable food supply and a decrease in energy expenditure have further weighted this problem of over-weights. Obesity is associated with numerous diseases including diabetes, atherosclerosis, coronary heart disease and many others[4]. These problems have increased the consumers' interest in the nutritional aspects of health, and in accordance, various recommendations have been put forward to overcome the obesity-related complications in both the developed and developing countries[5]. Consequently consumer's interest has led food

producers to develop foods that would help to minimize these complications. The introduction of 'cholesterol-free vegetable oil' is one of such food products and its campaign has drawn a huge attention of the consumers. The campaign is, however, non-scientific and sometimes might be unphysiologic to health, and it deserves further scientific discussion.

The compounds found in nature with the parent nucleus 'perhydrocyclopentanophenenthrene' (consisting of fused phenanthrene and cyclopentene ring) are called steroids. The steroids containing alcoholic hydroxyl functional groups (-OH) are referred to as 'sterol' compounds. In vertibrate animals, the most abundant steroidal compound is 'cholesterol' and it has not been found in plant tissue [6,7] and/or, if present only in trace amounts[8]. Reeves and Weihrauch, (1979) reported, however, that flaxseed, safflower, sunflower, corn, olive, soybean, margarine, peanut, palm and coconut oils do not contain cholesterol[9]. Cholesterol was first discovered as component of gallstone by French chemist M.E. Chevreul[10] Several sterol compounds (called as phytosterol) are abundantly present in plant kingdom.

Stigmasterol is a member of such phytosterol compounds and its structure is similar but not identical to that of the cholesterol. Stigmasterol has an ethyl group at C_{24}-position and a double bond between C_{22} and C_{23}. This sterol occurs in soybean oil, while sitosterol, another phytosterol occurs in wheat germ oil[11]. It is notable that cholesterol and the phytosterols are not bio-convertible, otherwise they could be substituted for each other in case of dietary supplementation. Therefore, the labeling of 100% cholesterol-free vegetable oil does not make any sense. The labeling is, thus, being irrationally used. It is plain cheating.

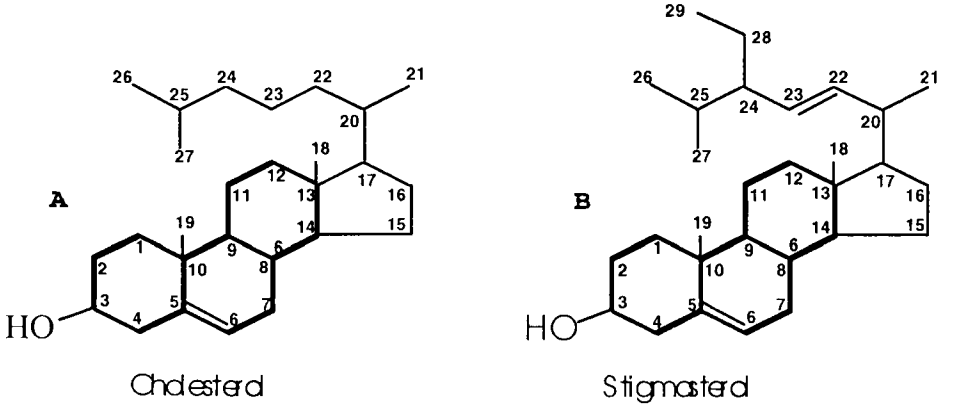

Figure 1. Structures of cholesterol (A) and stigmasterol (B)

Vegetable oil with phytosterol is rather beneficial for adult health

Edible vegetable oils that contain sterol components could rather be considered as food with substantial health benefits as because plant sterol-containing foods decrease plasma cholesterol[12-15]. Plant sterols compete with cholesterol for absorption[16,17], hence decreasing the intestinal absorption and accumulation of cholesterol in the plasma. Intakes of 2~3 g plant sterol per day have been reported to decrease total cholesterol and LDL-cholesterol levels by 9% ~ 20%[15]. Therefore, cholesterol and phytosterol could be referred to as antagonistic attributes to each other. The commercial campaign label should, thus, be that a given edible vegetable oil is 'cholesterol reducing' rather than 'cholesterol free'.

However, recent concern has been raised regarding the tendency of plant sterol-containing foods to decrease plasma -plus -carotene, -tocopherol levels[18]. These are very important physiological antioxidants and crucially required at the developmental stages. This suggests that the use of the foods with plant sterol should be discouraged in early stages of life. Therefore, the food products rich in plant sterols should be reserved for adults requiring lowering of cholesterol levels because of hypercholesterolemia and related cardiovascular complications.

Does edible vegetable oil has any effect on blood cholesterol?

Even if the vegetable oil does not contain cholesterol at all, and/or even if the vegetable oil is 100% refined from cholesterol, still the ingestion of oil is problematic for the health of the individuals with increased plasma cholesterol levels. Vegetable oils namely soybean, olive and palm oil, respectively, contained 17, 13 and 50% saturated fatty acids (see Table 3). Dietary saturated fat is the principal dietary determinant of LDL-C (low density lipoprotein-cholesterol) even than that of the dietary cholesterol[19].

Emergency of architectural modification of oil molecule

Vegetable oils cannot customarily be discarded from the human dietary culture. Thus, molecular modification of vegetable oil, with minimum load on body fat and no major changes in taste, is indeed a revolutionary step for the oil-craving consumers. Conventional vegetable oil is neutral fat and present as mostly (>90%) as 1, 2, 3-triacylglycerol (TG) of different fatty acids, as depicted in Figure 2. Only, 1-10% of the conventional oil is 1, 3-diacylglycerol (1, 3-DAG). The digestion of TG proceeds by the action of pancreatic lipase in the small intestine.

Pancreatic lipase is specific for the hydrolysis of 'primary ester linkages' *i.e.* at positions 1 and 3 of TG. Thus pancreatic lipase can only hydrolyze the terminal fatty acids and produces 2-monoacylglycerol (2-MAG), the major end products of TAG digestion. Seventy percent of total TG (which is the major component of the conventional oil) is absorbed as 2-MAG, six percent as 1 (or 3)-MAG and the rest of the 22% as free glycerol[20].

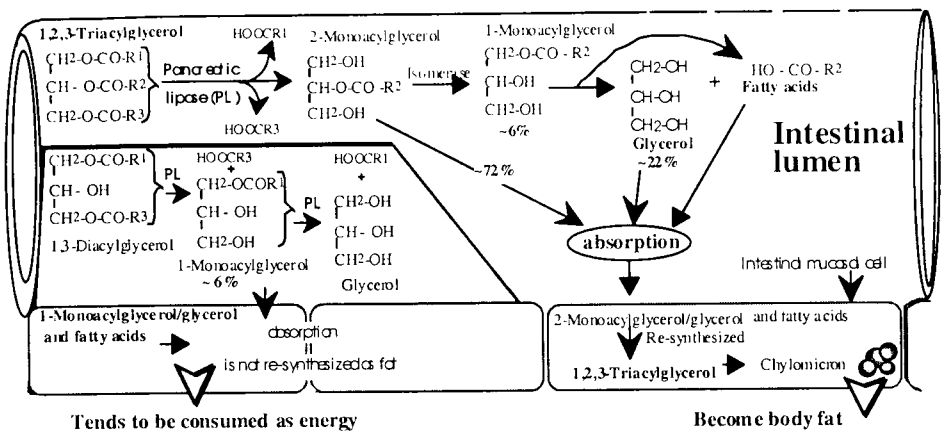

Figure 2. Schematic diagram of the digestion of the major component of conventional vegetable oil (1,2,3-triacylglycerol, TG) and that of the modified vegetable oil (1,3-Diacylglycerol, DAG) (structures in the shaded-area) by pancreatic lipase (PL) specific enzyme capable of hydrolyzing the 1° ester linkage, i.e. at carbon-positions 1 and 3 of the TG, and their absorption.

The human intestine does not have enzyme capable of hydrolyzing the secondary ester bond of 2-MAG or TG. Recently, some food producers in developed countries have manufactured 'vegetable edible oil' which contains primarily 1, 3-DAG. This strategy has circumvented the increasing effects of dietary oil on plasma cholesterol and triacylglycerol associated with obesity. Compared with TG, the main digestive product of DAG is 1 (or 3) monoacylglycerol,which is poorly backed(re-esterified) into TG in the small intestine[21,22]. Experiments with obese mice[21] and clinical trials in human[22,23] have shown that the oils containing 1,3-DAG decrease the body weight, fat mass, serum postprandial 1,2,3-TG and cholesterol significantly at a greater extent than that of the conventional vegetable oils.

Edible oil, its saturated, n-6 and n-3 (poly)unsaturated fatty acids

Fats are simply divided into two groups: one group is saturated fatty acids which are contained mainly in meat and eggs; the other is unsaturated fatty acids which are found abundantly in fish oils and vegetables. The unsaturated fatty acids are again

divided into n-3, which is mainly present in fish and n-6 group, which is contained in vegetable oils and other various kind of foods (figure 3). Unlike plants, animals are unable to insert double bonds at the n-6 and n-3 positions, therefore, are unable to synthesize n-6 linoleic acid (C18:2, n-6) and n-3 alpha-linolenic acid (C18:3, n-3) *de novo* (hence they are essential fatty acids).

Animals require these pre-formed PUFAs and exploit them as precursors to synthesize long chain fatty acids. The meat of the domestic animals is a rich source of arachidonic acid (C20:4, n-6)[24]. Dietetically, it is vital for us to maintain nutritious balance between n-3 and n-6.

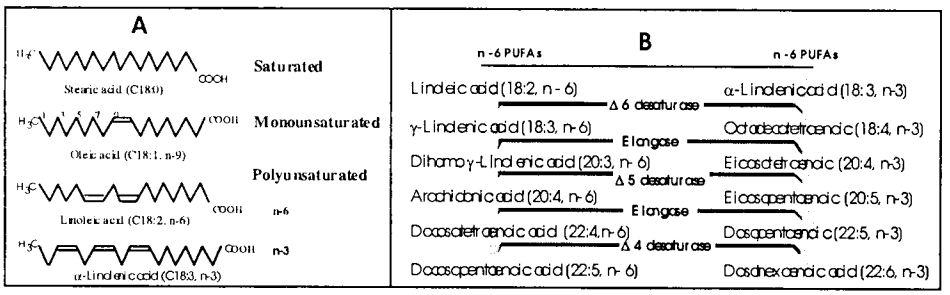

Fig 3. Structures of the physiological important fatty acids. Fatty acids are aliphatic chains of ten or more carbons with a methyl group at one end of the molecule and a carboxy group at the other. Fatty acids without double bonds (i.e. containing only the single bonds) are saturated, with one double monounsaturated, with 2 or more are referred to as polyunsaturated fatty acids (**A**). The first number denotes the length of carbon chain, the second number, following the colon, refers to the number of double bonds, and the third number, after n-, represents the number of carbons from the methyl end of the molecule to the first double bond (**B**).

The major food components that raise LDL-cholesterol are saturated fatty acids[19]. One of the dietary factors that lower LDL-cholesterol includes n-3 polyunsaturated fatty acids like eicosapentenoic acid (EPA), docosahexaenoic acid (DHA).[25,26] Saturated fatty acids promote thrombogenesis.[27] Dietary trans-unsaturated fatty acids, which are found in hydrogenated vegetable oils and used in cookies, crackers, baked goods, fried foods, margarines and fast food chains, also increase LDL-cholesterol and reduce HDL (high density lipoprotein)-cholesterol[28,29]. Limiting the intake of saturated and trans-fatty acids requires the substitution of other nutrients. The n-3 PUFAs of fish oil particularly EPA and DHA[25,30] and certain soluble fibers (e.g. oat products, pectin and guar gum)[31] can be used as substitutes of saturated fatty acids to reduce cholesterol, particulalrly in hypercholesterolemic individuals.

A number of organizations have already made dietary recommendations regarding fats[32-34]. An international group of scientists organized a Workshop on the Essentiality of and Recommended Dietary Intakes for n-6 and n-3 Fatty Acids in April 1999. The group's recommendations are shown in Table 1[35]. In addition to these recommendations, food-related industries in different countries have already begun to return n-3 fatty acids to food supply in order to maintain a nutritious balance between n-6 and n-3 PUFAs[36].

Table 1. Recommended adequate amount of individual fatty acids per day*

Fatty acids	Adults (% energy)	Infants (% energy)
LLA	2.0-3.0	10
LLN	1.0	1.5
[a]AA	—	0.5
[b]$EPA_{(upper\ limit)}$	—	< 0.1
DHA	0.1	0.35
EPA+DHA	0.3	—
[c]SFA	< 8.0	—
[d]MUFA	—	—
[e]$TFA_{(upper\ limit)}$	1.0	—

LLA = Linoleic acid $_{(C18:2,\ n-6)}$; LLN = -Linolenic acid ($C_{18:3,\ n-3}$); AA = Arachidonic acid ($C_{20:4,\ n-6}$); EPA = Eicosapentaenoic acid ($C_{20:5,\ n-3}$); DHA = Docosahexaenoic acid ($C_{22:6,\ n-3}$). SFA = Saturated fatty acid; MUFA = Monounsaturated fatty acid; TFA = Trans fatty acid.

[a]Arachidonic acid, unlike in the infants, is not essential for the adults because adults can easily elongate and desaturate linoleic acid to arachidoinc acid.

[b]EPA is contained in the breast milk, thus excess intake may interfere with AA and infant growth.

[c]Saturated fatty acid should not exceed 8%.

[d]The majority of the fatty acids be obtained as monounsaturated fatty acid.

[e]Other than dairy products, natural foods do not contain trans fatty acids. The trans fatty acid, as a result of hydrogenation of unsaturated fatty acids (if unavoidable, it must not exceed >1%), is not recommended to be in the food supply.

* reference 35

Alterations of fatty acid pattern from the Paleolithic diet to present-day diets

The Paleolithic diet (diet from the Paleolithic period ~ 40,000 – 15,000 y ago) was characterized by a lower saturated fat intake and a balanced intake of n-6 and n-3 essential fatty acid[2,37,38]. The Paleolithic-diet is the diet on which we evolved and our genetic profile was programmed. Over the past 10,000 yr, no significant changes have occurred in our genes (perhaps 0.005%), but major changes have occurred in our food supply, particularly during the last 150 yr. Today the ratio of n-6/n-3 has dramatically changed (See the Table 2), whereas during our evolution it was 1:1 or even less (Table 2).

Table 2. n-6/n-3 ratios in various populations

Population	n-6/n-3
[a]Paleolithic	0.79
[a]USA	16.75
[b]Greece before 1966	1.0-2.0
[c]UK and Northern Europe	15.0
[d]Japan	4.0
[e]Mongol	7.33

Source: [a]Eaton et al. (1998) (2); [b]GISSI-Prevenzione Investigators (1999)[39]; [c]Sanders (2000)[40]; [d]Sugano and Hirahara (2000)[41]; [e]Hashimoto et al. (2003)[42].

The change in the unsaturated fatty acid balance came about because of the recommendation in the 1960s to substitute vegetable oils for saturated fats. These vegetable oils are very high in n-6 fatty acids and very low in n-3 fatty acids. Though, the ratio of n-6/n-3 in various countries changed (Table 2)[2,39-42] only the traditional diet of Greece has a ratio similar to that of the Paleolithic diet. The Greek diet was associated with the longest life expectancy and lowest rate of cardiovascular disease[43,44]. Several studies using Greek-diet showed a 70% decrease in mortality in 2 yr[45] and decreased death rate from cardiovascular disease[39,46].

Historical perspectives of fish oil and its effects on Norwegians health during German invasion

During German occupation in Norway in 1940 there were rumors that war-affected Norwegians had little CVDs. The low rate of CVD in the Norwegians was ascribed

to obligatory consumption of large amounts of fish as the principal source of fat and protein[47]. Further interests in this field stemmed in 1970s when Bang (1972, 1976)[48, 49] reported that the incidences of CVD are very low in the Greenlandic Eskimoos. Contemporarily, Hirai *et al.*, (1984)[50], Kagawa *et al.*, (1982)[51] reported a low rate of heart diseases in the Japanese islanders as compared to the main-landers. Newman *et al.* (1993)[52], reported a similar findings in the Alaskan Eskimoos. A negative correlation between ischemic heart disease and fish intake was reported during 20-yr follow up study[53]. Very recently, Hashimoto *et al.*, (2003)[42] reported that the risk of CVDs, including hypertension and atherosclerosis in Mongolian population is higher than that in the Japanese workers because of lower intake of marine fish in the former populations. These epidemiological studies, followed by a great deal of experimental studies in rats, rabbits, mouse, monkeys, swines, and clinical trials in human have now confirmed the notion that the intake of n-3 PUFAs of fish oil is associated with a low incidence of cardiovascular diseases[54].

Bangladesh perspectives

Once, the main challenges were to provide food for all of the people. However, at present the dietary problems have changed and appeared a problematic dimension. The economy has relatively improved, and due to expansion of the free market a great variety of foods within markets are available and is being expanded in day and day out. With energy-rich foods in abundance, a considerable number of people could now eat according to the longstanding recommendation to avoid undernutrition. No one understood that this could create health problems. Overnutrition and a diet unbalanced in fatty acids have been reported to increase in mortality from cardiovascular disease in abroad and interestingly, this increase in mortality was associated with changes in food habits and life style[3].

The prevalence of diabetes particularly of type 2 diabetes and diabetes-related complications is increasing year to year in Bangladesh and the majority of the patients of this form of diabetes are obese[55], seen usually associated with intake of excess fat[4]. The factor(s) responsible for an increased prevalence of diabetes in Bangladesh is not known clearly. The causes of diabetes are, however, multi-factorial which involve genetic, environmental and dietary patterns too. The dietary patterns of the recent Bangladesh are being altered with fast pace. Thus changed dietary pattern could be one of the most important factors for the increased prevalence of type 2 diabetes in Bangladesh. In the present report, dietary lipids are emphasized as underlying reasons of diabetes-associated high levels of blood total cholesterol, LDL-

cholesterol and triacylglycerol as well. Decreased incidence of CVDs in the fish Eskimoos,[48-49] Japanese islanders,[50-51] Alaskans[52] and other western countries are attributed to a lower intake of n-6 PUFAs and a higher intake of n-3 PUFAs.

In addition, the risk factors of CVDs including hypertension, total cholesterol and triglyceride and an increased plasma level of n-6 PUFA linoleic acid were higher in the vegetarian Tanzania villagers when compared with their counterparts based on fish diet of Lake-shore areas[56]. These CVD risk factors remain associated with type 2 diabetes[57]. Therefore, comparatively, the Tanganian vegetarians may suffer from increased incidence of hypertension, CVDs risks etc. that is because Lake-shore peoples were ingesting more n-3 polyunsaturated fatty acids through fish diet. These data thus suggest that the n-3 PUFA have better beneficial effects on plasma atherogenic lipid parameters over that of the n-6 PUFA. Whatever may be the mechanism(s), the increasing prevalence of diabetes and diabetes related-cardiovascular complications in Bangladesh thus warns that a strategy is urgently needed to prevent or delay diabetes and related problems.

Table 3. Major fatty acid composition (%) of some important fish and vegetable oils consumed in Bangladesh

Fatty acid	[1]Hilsa	[2]Pangus	[3]Rupchada	[4]Soybean	[5]Olive	[6]Palm
PLA (C16:0)	35.4 ± 2.5	43.0 ± 4.5	40.0 ± 2.3	13.5 ± 1.1	10.5 ± 2.7	45.0±3.2
STA (C18:0)	27.0 ±1.6	13.0 ± 2.2	30.0 ± 6.0	3.80 ± 0.2	2.00 ± 0.5	5.0 ± 1.1
OLA (C18:1, n-9)	23.5 ± 1.2	28.0 ± 1.5	13.5 ± 1.5	5.50 ± 0.5	77.5 ± 4.0	40.0 ±1.2
LLA (C18:2, n-6)	1.10 ± 0.1	2.80 ± 0.40	1.25 ± 0.05	69.0 ± 0.7	9.30 ± 1.8	9.7 ± 0.9
LLN (C18:3, n-3)	0.30 ± 0.01	0.30 ± 0.01	0.60 ± 0.1	8.0 ± 1.1	0.50 ± 0.25	n.d.
AA (C20:4, n-6)	1.58 ± 0.07	0.80 ± 0.30	1.40 ± 0.3	n.d.	n.d.	n.d.
EPA (C20:5, n-3)	6.60 ± 0.60	2.10 ± 0.20	3.25 ± 0.60	n.d.	n.d.	n.d.
DPA (C22:5, n-3)	1.80 ± 0.14	0.40 ± 0.10	2.80 ± 0.40	n.d.	n.d.	n.d.
DHA (C22:6, n-3)	2.70 ± 0.10	1.30 ± 0.25	7.6 ± 1.0	n.d.	n.d.	n.d.

Results are expressed mean ± SE (standard error of mean) of three independent determinations.

[1-6] The fatty acid profile was determined by one-step analysis as previously described (Hashimoto *et al.*, 1998)[69]. Five mg of fish or vegetable oil was weighed and diluted with octane. 100 □l volume of this oil solution was mixed with 400 □l of methanol containing 100 g tricosanoic acid as internal standard and 50 □l trichloroacetic acid for transmethylation.

Tubes were tightly closed with teflon-lined caps and heated at 100°C for 1 hr. After tubes had been cooled in water, 5.0 ml 0.1 N NaOH was added to stop the reaction and neutralize the mixture. The tubes were then shaken and centrifuged, and aliquot of the octane upper phase was injected into the gas chromatography with a Hewlett Packard model 5890 (Avondale, PA, USA) equipped with dual flame ionization detectors and an autosampler (Model HP 7673). The conditions for measurement were as follows: injection temperature of 200 °C, detector temperature of 250 °C, and carrier (He) flow of 2.0 ml per min. The initial temperature of 180 °C was held for 4 min after injection, then the temperature was raised at 2 °C per min to 240 °C and maintained for 30 min, then again it was raised at 5 °C every 3 min to 255 °C and maintained for 5 min. To identify the peaks, gas chromatography-mass spectrometry (GC-MS) was done on a JEOL JMS mass spectrometer (Nippon Denshi Ltd., Osaka, Japan) model D300 equipped with a chemical ionizer. The chromatograms were identified and quantified by a JEOL JMA- 2000S mass data analysis system (Nippon Denshi Ltd., Osaka, Japan).
PLA = Palmitic acid; STA = Stearic acid; OLA = Oleic acid; LLA = Linoleic acid; LLN = ••Limolenic acid; AA = Arachidonic acid; EPA = Eicosapentaenoic acid; DPA = Docosapentaenoic acid; DHA = Docosahexaenoic acid.
n.d = not detected.

The edible vegetable oil of local market contains mostly (~70%) the n-6 linoleic acid (LLA, $C_{18:2,n-6}$) (Table 3). In humans LLA exists in the tissue in part as a moiety of acylceramide and involved in epidermal barrier system[59]. Deficiency of this PUFA produces atopic eczema[60]. Its other known functions in the human body are to be oxidized for fuel and to act as one of the structural constituent of the membrane phospholipid at SN-2 position (like any PUFA) or to serve as the substrate to produce gama linolenic acid (GLA) by delta 6 desaturase.

Gama linolenic acid acts as precursor to synthesize dihomo gama linolenic acid, which in turn is converted to arachidonic acid (AA, $C_{20:4,\ n-6}$) by delta 5 desaturase enzyme (see figure 3B). The n-6 eicosanoids (for example, prostaglandins) are formed from AA-cascade. The n-6 eicosanoid production, which accompanies many health disorders including platelet hyper-aggregation and atherosclerosis, can be diminished by dietary n-3 fats[61]. Therapeutic treatment to inhibit excessive n-6 eicosanoid signalling-associated diseases would cost a lot in a developing country like Bangladesh, whereas a preventive nutrition to decrease an excessive n-6 eicosanoid signalling by cutting the intake of AA-precursor remains an achievable alternative.

The individual fatty acid compositions of the vegetable oils of local market are not mentioned on the container except total average values of three kinds of fatty acids

(contents/10 gm: saturated fatty acids, 1.5 gm; monounsaturated fatty acids, 2.0 gm and polyunsaturated fatty acid 6.5 gm). If the oils are of soy origin, then it must be admitted that the major PUFA of these local vegetable oils is linoleic acid (C18; 2, n-6). The fatty acid composition of local vegetable oil thus is not encouraging with respect to the effects of linoleic acid on cardiovascular healths. Hydrogenation occurs at the time of refining and deodorization treatments of soybean oil. This decreases •-linolenic acid content with a concurrent increase of deleterious trans-fatty acids of soybean oil[62]. Decrease in PUFA content of local soybean oil during storage[63] is probably due to auto-oxidation (air oxidation) and/or transformation cis-unsaturated to trans-unsaturated fatty acids.

Hilsa (*Hilsa Ilsha*), Pangaus (*Pangasius pangus*) and Rupchada (*Leiagnathus eguulus*) are popular fishes in Bangladesh, abundant in her Bay/river area. The fatty acid composition analyses of these fish oil showed that n-3 EPA plus DHA are present at a significant amount (Table 3). The content of n-3 PUFAs may vary among fish species[64,65], this relates to the location, time of capture and food sources including phyto-and zooplankton of the fish.

The effects of these local fish oils on atherogenic lipid profiles have been investigated (Table 3). Pangus fish oil[66] as well as Hilsa fish oil was fed to experimentally-induced hypercholesterolemic and diabetic model rats[67]. These oils significantly decreased the atherogenic lipid profile, platelet aggregation and hypercholesterolemia in diabetic model rats, though no significant effects were observed on glycemic control. Fish oil and soybean oil exhibit differential effects on the lipid profile of both normal (nondiabetic)[66] and diabetic[67] conditions. The fish oils provided greater anti-lipidemic effects than do equivalent dietary intakes of soybean oil containing in excess of PUFAs as linoleic acid[66]. The purified (>90%) fish oil components EPA and DHA significantly decreases high blood pressure, platelet hyperaggregation and atherogenic lipid profile[67-70]. Thus the results obtained after feeding of local fish oil were in line with those reported for purified EPA and DHA.

Table 4. Effects of local fish oil, purified fish oil components EPA and DHA, and *mushroom (Pleuretus ostreatus)* fiber on atherogenic lipid profile, platelet aggregation and high blood pressure.

Parameters	Fish oil and/or purified EPA and DHA				Mushroom fiber
	[1]Pangus	[2]Hilsa	[3]EPA	[4]DH	[5]*Plerotus ostreatus*
Total Cholesterol	↓	↓	↓	↓	↓
Triacyglycerol	↓	↓	↓	↓	↓
Platelet aggregation	**ND**	↓	↓	↓	**ND**
High blood pressure	**ND**	**ND**	↓	↓	**ND**

[1, 2] Fish oil was extracted from *locally purchased* Hilsa and Pangas fish by using Soxhlet apparatus and orally administered for three weeks (Quazi et al., 1993[66]; Mahmud et al., 2003[67]).

[3, 4] 95-99% purified fish oil component EPA and DHA were orally administered at a dose of 300 mg/KgBW/d for twelve weeks (Hossain *et al.*, 1995[68]; Hashimoto *et al.*, 1998[69], 1999[30]).

[5] Fresh *Pleaurotus ostreatus* mushrooms were locally purchased from the Japan-collaborated Mushroom Cultivation Center, Savar, Dhaka, Bangladesh. The fruiting bodies were dried in sunlight and crushed into powder and supplemented as 5% with basal diet for 5 weeks (Hossain *et al.*, 2003)[70].

Plasma total cholesterol and triglyceride were measured enzymatically using commercially available reagent kits (Total Cholesterol E and TG-test; Wako Pure Chemical Industries Ltd.).

Platelet aggregation was measured using ADP as agonist (Hossain et al., 1995[70]; Mahmud et al., 2003[67]).

Blood pressure was measured by the tail-cuff plethysmographic method (Ueda, UR-1000, Tokyo, Japan) (Hashimoto et al., 1998[69], Hashimoto et al., 1999[30]). ↓ = Decrease; **N.D.** = Not determined

A better health benefit is obtained if dietary saturated fatty acid is limited in exchange of fiber. The edible mushroom *Pleuretus ostreatus*, collected from local cultivation center (Mushroom Cultivation Center, Savar, Dhaka-1342) significantly ameliorated the atherogenic lipid profile[70] consistently with those of the *Lentinus edodes* and *Grifola frondosa* mushrooms[71]. These findings suggest that hypercholesterolemia and hypertriglyceridemia, seen associated with diabetes, can be prevented by feeding of Hilsa, Pangus fish oil and mushroom fiber as well.

Conclusion

The development of a guideline for the dietary intake of fatty acids is urgently needed. Nutrition and genetics have a strong influence on health and disease. For the prevention and/or treatment of chronic disease, universal recommendations are, however, inappropriate because of inter-individual physiological needs, genetic variation and the differences in frequency of polymhorphisms as well. This issue emphasizes that one cannot adopt another ones' dietary recommendations, as nations cannot adopt each other's dietary recommendations. Undernourished populations, for example, cannot be discouraged to intake fats, as those could be prohibited for the overnourished individuals. All these indicate that continuous education of professionals and the public is essential to bring about change and improvement of the health and well being of people.

Finally, this report has focused on appropriate choice of edible oil that should be included in an overall dietary program. The choice has potential benefit on cardiovascular health. Because the reformulation of products is expensive, one could first begin to change the cooking and salad oils. The beneficial effects depend on the fatty acid compositions of the oil ingested. Thus avoiding hydrogenated oil/margarine-based "food" products, eating only low-fat meat may be helpful. The strategy is that the type of fats consumed should be the right kind and requires a higher ratio of n-3 to n-6 fatty. The avoidance of increased use of vegetable oils rich in n-6 (e.g. soybean oil, safflower oil) and the use of oils rich in n-3 fatty acid (e.g. flaxseed oil, perilla oil and fish oil) may help to bring about an improvement in the n-3/n-6 ratio. Lowering excessive levels of n-6 ecosanoid production involves eliminating as much arachidonic acid from our diet as possible[61], which could be achievable by eating as little red meat fat as possible[72]. Beef, pork, etc. is high in AA[24], whereas low-fat beef is lower, and free roaming poultry, is still lower. Ocean and cold water fatty fish (not farm raised), are better still, since they contain little AA, but lots of n-3 fatty acids[48-50]. Fatty acid profile of the individual's blood plasma and/or erythrocytes or platelet membranes, as indicator of tissue fatty acid profile, might be required before making any dietary recommendations for fatty acids. In this context, physicians engaged in both obese centers and hospital clinics must take an active part because they have been in the profession responsible for the diagnosis and treatment of chronically ill peoples. The guidelines may thus reduce the risk for other chronic health problems including diabetes mellitus, obesity seen associated with cardiovascular diseases.

In conclusion, this effort based on the current state of knowledge could be considered as of great significance regarding the roles of cholesterol, n-6 and n-3 PUFAs in cardiovascular health and disease processes.

References

1. Smith LL. Another cholesterol hypothesis: Cholesterol as antioxidant. Free Rad Biol Med 1991; 11: 47-61.
2. Eaton SB, Eaton SB, III Sinclair AJ, Cordain L and Mann NJ. Dietary intake of long chain polyunsaturated fatty acids during the paleolithic. In: Simopoulos, AP. ed. The Return of -3 Fatty Acids into the Food Supply, I. Land-Based Animal Food Products and Their Health Effects, World Review of Nutrition and Dietetics. S. Karger, Basel, 1998; Vol. 83: 12-23.
3. Simopoulos AP. n-3 fatty acids and human health: defining strategies for public policy. Lipids 2001; 36: S-83-S89.
4. Truswell AS. Dietary guidelines: Theory and Practice. Proc Nutr Soc Aust 1995; 19: 1-10.
5. Pan American Health Organization. Scientific Publication No. 576. IN: Pena M and Bacallao J (eds.) Obesity and Poverty, A New Public Health Challenge, Washinton DC.
6. Cook RP. Cholesterol: Chemistry, Biochemistry and Pathology. Academic Press. New York, 1958.
7. Gibbons GF, Mitropoulos KA and Myant NB. *In*: Biochemistry of cholesterol. Amsterdam, Elsevier, 1982. p 369.
8. Goodwin TW, and Mercer E I. In : Introduction to Plant Biochemistry, Pergamon Press, Oxford, 1983, p-411.
9. Revees JB and Weihrauch JL. Composition of Foods, Agriculture Handbook No. 8-4, 1979, US, Department of Agriculture, Washington, DC.
10. Vance DE and Van den Bosch H. Cholesterol in the year 2000. Biochim Biophys Acta 2000; 1529: 1-8.
11. West ES, Todd WR. and Bruggen JTV eds. Text Bock of Biochemistry, Oxford and IBH Publishing Co. Ltd., 1974; New Delhi.
12. Vahanen HT, Blomqvist S, Ehnholm C. et al., Serum cholesterol, cholesterol, Precursors, and Plant sterols in hypercholesterolemic subjects with different apo E phenotypes during dietary sitostanol ester treatment. J Lipid Res 1993; 34:1535-1544.
13. Miettinen TA, Puska P, Gylling H. et al. Reduction of serum cholesterol with sistanol-ester margarine in a mildly hypercholesterolemic population. N Eng J Med 1995; 333: 1308-1312.
14. Hendriks HFJ, Weststrate JA, van Vliet T, et. al.. Spreads enriched with three different levels of vegetable oil sterols and the degree of cholesterol lowering in normocholesterolemic and mildly hypercholesterolemic subjects. Eur J Clin Nutr 1999; 53: 319-327.
15. Weststrate JA, Meijer GW. Plant sterol-enriched margarines and reduction of plasma total- and LDL-cholesterol concentrations in normocholesterolemic and mildly hypercholesterolemic subjects. Eur J Clin Nutr 1998; 52: 334-343.

16. Gylling H and Miettinen TA. Cholesterol reduction by different plant stanol mixures and with variable fat intake. Metabolism 1999; 48: 574 -580.

17. Vahouny GV and Kritchevsky D. Plant and marine sterols and cholesterol metabolism. In: Spiller GA ed. Nutritional Pharmacology. New York, NY: Alan R. Liss Inc, 1981; 31-72.

18. Gylling H, Siimes MB and Miettinen TA. Sitostanol ester margarinr in dietary treatment of children with familial hypercholesterolemia. J Lipid Res 1995; 36, 1807-1812.

19. Hegsted DM, Ausman LM, Johnson JA et al., Dietary fat and serum lipids: an evaluation of the experimental data [published erratum appears in Am. J Clin Nutr 1993; 58: 245] Am J Clin Nutr 1993; 57: 875-883.

20. Mayes PA. Digestion and absorption. *In:* Murray RK, Granner DK, Mayes PA, Rodwell VW (eds). Harper's Biochemistry (International edn), MacGraw Hill Education, 2002, p 662.

21. Watanabe H, Onizawa K, Tagichi H, Kobori M, Chiba H, Naito S, Matsuo N, Yasukawa T, Hattori M, and Shimasaki H. Nutritional characterization diacylglycerols in rats. Jpn Oil Chem Soc 1997; 46: 301-308.

22. Maki KC, Davidson MH, Tsushima R, Matsuo N, Tokimitsu I, et al. Consumption of diacylglycerol oil as part of a reduced-energy diet enhances loss of body weight and fat in comparison with consumption of a triacylglycerol control oil. Am J Clin Nutr 2002; 76: 1230-1236.

23. Nagao T, Watanabe H, Goto N, Onizawa K, Taguchi H, Matsuo N, Yasukawa T, Tsushima R, Shimasaki H and Itakura H. Dietary Diacylglycerol Suppresses Accumulation of Body Fat Compared to Triacylglycerol in Men in a Double-Blind Controlled Trial. J Nutr 2000; 130: 792-797.

24. Wainwright PE. Do essential fatty acids plat a role in brain and behavioral development? Neurosci Behav Rev 1992; 16: 193-205.

25. Harris WS. n-3 fatty acids and serum lipoproteins: human studies. Am. J Clin Nutr 1997; 91: 9886-9890.

26. Grundy SM. Hypertriglyceridaemia, insulin resistance, and the metabolic syndrome. Am J Cardiol 1999, 83: 25F- 29F.

27. Hoak JC. Fatty acids in animal: thrombosis and haemostasis. Am J Clin Nutr 1997; 59: 861-868.

28. Judd JT, Vlevidence BA, Muesing RA et al., Dietary trans fatty acids. Effects on plasma lipids and lipoproteins of healthy men and women. Am J Clin Nutr 1994; 59:861-868.

29. Lichtenstein AH, Jalbert SM et al. Effetc of different forms of dietary hydrogenated fats on serum lipoprotein cholesterol levels: N Eng J Med 1999; 340: 1933-1940.

30. Hashimoto M, Shinozuka K, Gamoh S, Tanabe Y, Hossain MS, Kwon YM, Hata N, Misawa Y, Kunitomo M and Masumura S. The hypotensive effect of docosahexaenoic

acid is associated with the enhanced release of ATP from caudal artery of aged rats. J Nutr 1999; 25: 676-681.

31. Brown L, Rosner B, Willett W, et al. Cholesterol-lowering effects of dietary fiber: a meta analysis. Am J Clin Nutr 1999; 69: 3-42.

32. Scientific Review Committe, Canada. Nutrition Recommendations, Ministry of National Health and Welfare Canada, Ottawa H49-42/1990E, 1990.

33. British Nutrition Foundation. Unsaturation fatty acids: Nutritional and Physiological Significance. The report of the British Nutrition Foundation's Task Force, Chapnav and Hall, New York, 1992.

34. World Health Organization. Fats and Oils in Human Nutrition: Report of a Joint Expert Consultation. Food and Agriculture Organization of the United Nations and the World Health Organizations, FAO Food Nutr. Paper 57, pp. 1-47, Rome, 1995.

35. Simopoulos AP, Leaf A and Salem N. Jr. Essentiality of and recommended dietary intakes for omega-6 and omega-3 fatty acids. Ann Nutr Metab 1999; 43: 127-130.

36. Simopoulos AP. The return of omega-3 fatty acids into the food supply, I. Land-baesd animal food products and their health effects, World review of nutrition and dietetics. 1998. Vol. 83, S. Karger, Basel.

37. Eaton SB, Konner M and Shostak M. Stone ages in the fast lane: Chronic Degenrative Diseases in Evolutionary perspective. 1988; 84: 739-749.

38. Eaton SB and Konner M. Paleolithic nutrition. A consideration of its nature and current implications. N Eng J Med 1985; 312: 283-289.

39. GISSI-Prevenzione Investigators. Dietary supplementation with n-3 polyunsaturated fatty acids and vitamin E after myocardial infarction. Results of the GISSI-Prevenzione Trial. Lancet1999; 354: 447-455.

40. Sanders TAB. Polyunsaturated fatty acids in the food chainin Europe. Am J Clin Nutr 2000; 71(suppl.): 176S-178S.

41. Sugano M and Hirahara F. Polyunsaturated fatty acids in the Food chain in Japan. Am J Clin Nutr 2000; 71(Suppl) 189S-196S.

42. Hashimoto M, Shiwaku K, Nogi A, Fuji Y, Anuuard E, Byambaa E, Kitajima, Shahdat HM, Yamane Y, Oyunsuren T and Shido O. Effect of dietary intake of body composition, blood pressure and serum lipid profiles – comparision between Mongolian and Japanese workers. Jpn J Food Chem 2003;10: 46-50.

43. Simopoulos AP and Robinson J. The omega diet. The lifesaving nutritional program based on the diet of the island of Crete, 1999; Harper Collins, New York.

44. Simopoulos AP and Visioli F. Mediterranean Diets. World Review of Nutrition and Dietetics, S. Karger, Basel Vol. 87; 2000.

45. DeLorgeril M, Renaud S, Mamelle N, Salen P, Martin JL, Monjaud I, Huidollet J, Touboul P and Delaye J. Mediterranean Alpha-linolenic Acid-rich Diet in the Secondary Prevention of coronary Heart Disease, Lancet 1994; 343: 1454-1459.

46. Pientinen P, Vatiainen E, Seppanen R, Aro A and Puska P. Changes in Diet in Finland from 1972-1992: Impact on coronary Heart Disease Risk. Prev Med 1996; 25: 243-250.

47. Bang HO, Dyerberg J. Personal reflections on the incidence of ischaemic heart disease in Oslo during the Second World War. Act Med Scand 1981;210:245–248.

48. Bang HO and Dyerberg J. Plasma lipids and lipoproteins in Greenlandic West Coast Eskimos. *Acta Scand. Med.* 1972; 192: 85-94.

49. Bang HO, Dyerberg J and Hjorne N. The composition of Food Consumed by the Greenland Eskimoos. Acta Med. Scand. 1976; 200: 69-73.

50. Hirai A, Terano T, Saito H, Tamura Y, Yoshida S, Sajiki J and Kurinagai A. In: Lovenberg W. and Y. Yamoori. eds. Nutritional Prevention of Cardiovascular Diseases Academic Press, London., 1984; 231-239.

51. Kagawa Y, Nishizawa M, Suzuki M, Miyatake T, Hamamoto T, Goto K, Motonaga E, Izumikawa H, Hirata H and Edihara H. polyenoic acids of serum lipids of Japanese Islanders with low incidence of cardiovascular diseases. J Nutr Sci Vitaminol 28: 441-453.

52. Newman WP, III Propst MT. And Middaugh JP. Atherosclerosis in Alaska Natives and Non-natives. Lancet 1993; 341: 1056-1057.

53. Kromhout D, Bosschier EB and Coulder CDL. The inverse relation between fish oil consumption and 20-year mortality from coronary heart disease. N Eng J Med 1985; 312: 1205-1209.

54. Caterin RR and Zampolli A. n-3 fatty acids: Antiatherscleritic effects. Lipids 2001; 36:369-378.

55. Rahim MA. *In*: Diabetes in Bangladesh: Prevalence and Determinants: A thesis submitted as partial fulfillment of the requirements for the degree of Master of Philosophy in International Community Health. Institute of General Practice and Community Medicine Faculty of Medicine, University of Oslo, Norway, 2002, p-104.

56. Pauletto P, Puato M, Caroli MG, Casiglia E, Munhambo AE, Cazzolato G, Bon GB, Angeli MT, Galli C and Pessina A. Blood pressure and atherogenic lipoprotein profiles of fish-diet and vegeterian villagers in Tanzania: the Lugalawa study. Lancet 1996; 348: 784-788.

57. Howards BV. Lipoprotein metabolism in diabetes mellitus. J Lipid Res 1978; 28: 613-628.

58. Winocour PD. Platelets, vascular disease, and diabetes mellitus. Can J Physiol Pharmacol 1994; 72: 295-303.

59. Ziboh VA. Metaboilism of polyunsaturated fatty acids by skin epidermal enzymes: generation of anti-inflammatory/anti-proliferative metabolites. In: Abstract and Programs: International Conferrence on Highly Unsaturated Fatty acid in Nutrition and Disease Prevention. Barcelona, Spain, 4-6 Novem, 1996. P-7.

60. Horribon DF. Essential fatty acids and atpic eczema. In: Abstract and Programs: International Conferrence on Highly Unsaturated Fatty acid in Nutrition and Disease Prevention. Barcelona, Spain, 4-6 Novem, 1996. P-7.

61. Lands WEM. The impact of daily food choices on health promotion and disease prevention. In: 4th Congress of the International Society for the Study of Fatty acids and Lipids (ISSFAL 2000). June 4-9, 2000. Tsukuba International Congress Center, Tskuba, Japan. 2001; PL1-2, 37.

62. Tang TS. Fatty acid composition of edible oils in the Malaysian market, with special reference to trans-fatty acids. J Oil Palm Res 2002; 14: 1-8.

63. Shekhar HU, Alam MN, Howlader ZH, Huda MN, Hossain MA. Effect of storage on the physiochemical characteristics of soybean oil. Bangladesh J Biochem 1996; 2: 41-48.

64. Yusuf HKM, Alim SR., Rahman R, Salamatullah Q and Hossain, A. Fatty acids of 12 marine fish species of the Bay of Bengal. J Food Comp Anal 1993; 6: 346-353.

65. Chowdhury AHMS, Shekhar HU, Quddus MMAM and Mahmud I. Bichemical analysis of Tenualosa Ilisha (Hamilton). J Asiat Soc Bangladesh Sci 1995; 21: 83-88.

66. Salamatullah Q, Shahdat H, Mahmud I and Bashar K. Dose effects of Pangas (*Pangsius pangasius*) fish oil and soybean oil on serum and liver lipids in experimental hypercholesterolemic rats. Dhaka Univ J Biol Sci 1993; 2: 69-76.

67. Mahmud I, Hossain A, Hossain S, Hannan A, Ali L. and Hashimoto M. Effects of *Hilsa Ilsa* Fish Oil on Atherogenic Lipid Profile and Glycemic Status of Streptozotocin-treated Type I Diabetic Model Rats. Clin Exp Pharmacol. Physiol. (*In press*).

68. Hossain MS, Hashimoto M, Hara T, Masumura S. Age-related change in anti-platelet aggregation in feeding of a high fat diet and w-3 fatty acid in rats. Jpn J Pharmacol 1995; 67: p165.

69. Hashimoto M, Shinozuka K, Shahdat H, Kwon YM, Tanabe Y, Kunitomo M and Masumura S. Antihypertensive effects of all-cis-5,8,11,14,17-icosapentaenoate of aged rats is associated with an increase in the release of ATP from caudal artery. J Vasc Res 1998; 35: 55-62.

70. Hossain S, Hashimoto M, Chowdhury EK, Alam N, Shahjalal HM, Hasan M, Chowdhury SK and Mahmud I. Dietary mushroom (*Pleurotus ostreatus*) ameliorates atherogenic lipid in hypercholesterolaemic rats. Clin Exp Pharmacol Physiol 2003; 130:470-475.

71. Kabir Y, Yamaguchi M, Kimura S. Effect of Shitake (*Lentinus edodes*) and Mitake (Grifola frondosa) mushrooms on blood pressure and plasma lipids of spontaneously hypertensive rats. J Nutr Sci Vitaminol 1987; 33:341-346

72. Li D, Ng A, Mann NJ, Sinclair AJ. Contribution of meat fat to dietary arachidonic acid. Lipids 1998; 33:437-440.