

Health Effects of Palm Oil

Sutapa Mukherjee and Analava Mitra

*School of Medical Science and Technology, Indian Institute of Technology,
Kharagpur 721 302, West Bengal, India
E-mail: amitra@smst.iitkgp.ernet.in*

KEYWORDS Saturated Fats. Palm Oil. Cholesterol. Coronary Heart Disease

ABSTRACT Increasingly, over the past 40 years, the conception of diet has undergone major changes. Many of these changes involve changes in dietary intake of fats and oils. There has been an increasing consumption of partially hydrogenated *trans*- vegetable oils and a decreasing intake of lauric acid-containing oils. Although popular literature usually attribute an increased risk of coronary heart disease (CHD) to elevated levels of serum cholesterol, which in turn are thought to derive from an increased dietary intake of saturated fats and cholesterol. The palm oil and palm kernel oil are high in saturated fatty acids, about 50% and 80% respectively and are esterified with glycerol. In developing countries, vegetable oils are replacing animal fats because of the cost and health concerns. It is reassuring to know that the consumption of palm oil as a source of dietary fat does not pose any additional risks for coronary artery disease when consumed in realistic amounts as part of a healthy diet.

INTRODUCTION

Palm oil has been used in food preparation for over 5,000 years. Palm oil, obtained from the fruit of the oil palm tree, is the most widely produced edible vegetable oil in the world and its nutritional and health attributes have been well documented (Chandrasekharan et al. 2000). According to the Malaysian Oil Palm Statistics in 2005 (<http://www.tocotrienol.org/en/index/news/58.html>), it surpassed soybean oil as the most widely produced vegetable oil in the world. Palm oil is currently enjoying strong appeal worldwide as a cooking aid because it is free of artery-clogging *trans*-fats. Besides being cleaner and more stable, cooking with palm oil leaves the kitchen becomes less greasy and easy to clean. It is consumed worldwide as cooking oil, in making of margarine and shortening, apart from being used as an ingredient in fat blends and a vast array of food products. In the United States, palm oil's principal edible use is as an ingredient in prepared foods (primarily baked goods). Food manufacturers choose palm oil because it has a distinct quality, requires little or no hydrogenation, and prolongs the shelf life of different products (Anonymous 2003).

The palm fruit (*Elaeis Guineensis*) is the source of both palm oil (extracted from palm fruit) and palm kernel oil (extracted from the fruit seeds). Babassu oil is extracted from the kernels of the Babassu palm. Malaysia and Indonesia account for 83 percent of production and 89 percent of

global exports. Oil Palm is grown as an industrial plantation crop, often (especially in Indonesia) on newly cleared rainforest or peat-swamp forests rather than on already degraded land or disused agricultural land. In Malaysia, the area devoted to Oil Palm has increased 12-fold to 13,500 square miles. The vast plantations that grow Oil Palm trees have contributed to the destruction of the rainforest and wildlife of Southeast Asia (http://en.wikipedia.org/wiki/Palm_oil).

PALM OIL

Composition of Palm Oil

The palm oil and palm kernel oil are high in saturated fatty acids, about 50% and 80% respectively and esterified with glycerol. The Oil palm gives its name to the 16 carbon saturated fatty acid palmitic acid; monounsaturated oleic acid is also a constituent of palm oil while palm kernel oil contains mainly lauric acid. Palm oil is the largest natural source of tocotrienol. Palm oil is also high in vitamin K and dietary magnesium. Napalm derives its name from naphthenic acid, palmitic acid and pyrotechnics or simply from a recipe using naphtha and palm oil. Table 1 shows fatty acids composition of palm oil and palm kernel oil. Palm oil contained about 10% linoleic acid, which is an unsaturated omega-6 fatty acid. Linoleic acid is one of the two essential fatty acids that humans require. Palm oil also contains small amounts of squalene (possible cholesterol lower-

Table 1: Fatty acids composition of palm oil and palm kernel oil (Chow 1992)

<i>Palm Oil</i>	<i>Palm Kernel Oil</i>
Saturated acids- Palmitic (C ₁₆) 44.3%, Stearic (C ₁₈) 4.6%, Myristic (C ₁₄) 1.0%	Saturated acids- Lauric (C ₁₂) 48.2%, Myristic (C ₁₄) 16.2%, Palmitic (C ₁₆) 8.4%, Capric (C ₁₀) 3.4%, Caprylic (C ₈) 3.3%, Stearic (C ₁₈) 2.5%
Mono Unsaturated acids- Oleic (C ₁₈) 38.7%	Mono Unsaturated acids-Oleic (C ₁₈) 15.3%
Poly Unsaturated acids- Linoleic (C ₁₈) 10.5%	Poly Unsaturated acids-Linoleic (C ₁₈) 2.3%
Other/Unknown - 0.9%	Other/Unknown 0.4%

ing and anti-cancer properties) and ubiquinone (energy booster). Besides Red palm oil is also rich in co-enzyme Q10.

Antioxidants in Palm Oil

Effects of Carotenoids: Crude palm oil is considered to be the richest natural source of carotenoids (about 15 times more than in carrots). The human body uses carotenoids as Vitamin A. Carotenoids also enhance immune function by a variety of mechanisms, and can improve cardiovascular health. Carotenoids also play an important potential role by acting as biological antioxidants, protecting cells and tissues from the damaging effect of free radicals. When on being exposed to pollutants in cigarette smoke, industrial pollution, stress, unbalanced diets, pesticide and insecticide residues in food and water, and many other negative environmental influences, one is also exposed to free radicals. A build-up of free radicals in the body is associated with degenerative diseases such as heart disease and cancer, as well as general ageing. It is, therefore, in one's own best interest to ensure that to eat a diet rich in antioxidants that will prevent the damage that is done to bodies by free radicals. Red palm oil is a form of processed palm oil (deacidified and deodorised) which retains 80% of the original carotenoids, making it a remarkable source of Vitamin A. These natural antioxidants act as buffers against free radicals and are believed to play a protective role in cellular ageing, atherosclerosis, cancer, arthritis, and Alzheimer's disease.

Effects of Tocopherols and Tocotrienols: In fact, no other vegetable oil has as much Vitamin E as compared to palm oil (Chow 1992). Natural vitamin E exists in eight different forms or isomers, four tocopherols and four tocotrienols. Natural palm oil contains alpha, beta, gamma, and delta-tocopherols and alpha, beta, gamma, and delta-tocotrienols. Tocotrienols in Vitamin E have been found to have antioxidant and anti-cancer activities. Tocotrienols by its action on liver

enzymes lowers blood cholesterol levels without reduction in good cholesterol (High Density Lipoprotein or HDL). Its antioxidant properties bring many benefits to the human body, such as preventing skin aging, preventing fat oxidation, reducing blood pressure etc. Human studies have shown that palm tocotrienols have the ability to reverse blockage of the carotid artery and platelet aggregation (the clumping together of cells) thereby reducing the risk of stroke, arteriosclerosis, and ischaemic heart diseases. Palm tocotrienols have been shown to be protective after a strenuous bout of exercise by preventing protein oxidation and lipid peroxidation. Tocotrienol-rich fraction of palm oil is capable of protecting brain against oxidative damage and thereby from the ensuing adverse alterations that accompany aging. Lipid peroxides in blood vessels and plasma show a positive correlation with blood pressure. The antioxidant ability of gamma-tocotrienol may prevent development of increased blood pressure by reducing lipid peroxides and enhancing the total antioxidant status, including superoxide dismutase activity. Melanoma, also on the increase, can be inhibited with the delta fraction of tocotrienols. When applied topically, vitamin E/tocotrienols is quickly absorbed into the deep layers of the skin. Gamma- and delta-tocotrienols derived from palm oil exhibit a strong activity against tumor promotion by inhibiting Epstein- Barr virus. The delta and gamma fractions of tocotrienols can inhibit certain types of cancer, including both the estrogen- positive and estrogen-negative breast cancer cells. The inhibition of the growth of breast cancer cells by palm tocotrienols could have extraordinarily important clinical implications on world health. Not only can the palm tocotrienols prevent the growth of these unwanted cells, but they can also do this in the presence as well as in the absence of estradiol, thereby protecting against both hormone-related and other kinds of breast cancer. It is interesting to note that tocotrienols can inhibit or even kill normal cells, but only in extremely high amounts — just as most any

beneficial substance can be detrimental in excessive quantity. Malignant cells, on the other hand, are very sensitive to tocotrienols. In fact, the more cancerous the cell, the more susceptible it is to the destructive effects of tocotrienol, so very little is required to accomplish its favorable role of cancer-cell annihilation (Ebong et al. 1999).

Benefits of Palm Oil

Palm oil is consumed in the fresh state and/or at various levels of oxidation. Feeding experiments in various animal species and humans have highlighted the beneficial role of fresh palm oil to health. These benefits include reduction in the risk of arterial thrombosis and atherosclerosis, inhibition of cholesterol biosynthesis and platelet aggregation, and reduction in blood pressure. However, on being used in the oxidized state possesses potential dangers to the physiological and biochemical functions of the body. Oxidized palm oil induces an adverse effect on plasma lipid profile, free fatty acids, phospholipids and cerebrosides. Additionally, oxidized palm oil induces reproductive toxicity and organ toxicity particularly of the kidneys, lungs, liver and heart. Available evidence suggests that at least part of the oxidized oil impact on health is due to generation of toxicants due to oxidation. The reduction of the dietary level of oxidized oil and/or the level of oxidation may reduce the health risk (Ebong et al. 1999).

A study by a group of researchers in China comparing palm, soybean, peanut oils and lard showed that palm oil actually increased the levels of good cholesterol and reduced the levels of bad cholesterol in the blood (Zhang et al. 1997 cited by Koh 2007). A study by Hornstra in 1990 also showed similar results. Zhang et al. (1997) found that in normo and hypercholesterolemic subjects, the use of palm oil in the diet should be safe and will not increase the risk of Cerebro Vascular Disease (CVD). Toxicological and pharmacological studies show that supplementation with palm tocotrienols up to 2,500 milligrams per day per kilogram of body weight does not produce any significant side effects. Although higher levels can be used for therapeutic purposes, those who want to enhance their antioxidant intake can use 30 to 50 milligrams of tocotrienols daily. Additional phyto-nutrients, designed by some of our best formulators,

provide synergy for the palm tocotrienols (<http://www.bettykamen.com/newsletters/palmoil.htm>).

Controversies of Palm Oil

For many years now, it has been established that the primary cholesterol-elevating fatty acids are the saturated fatty acids with 12 (lauric acid), 14 (myristic acid) and 16 (palmitic acid) carbon atoms with a concomitant increase in the risk of coronary heart disease. The World Health Organization in its report (2003) states that there is convincing evidence that palmitic acid consumption contributes to an increased risk of developing of cardiovascular diseases.

In the past, palm oil was attacked as “saturated” since it contains 44% palmitic acid and 5% stearic acid, and thereby allegedly raises blood cholesterol and increases the risk of cardiovascular disease. However, a sizeable and growing body of scientific evidence indicates that palm oil’s effect on blood cholesterol is relatively neutral when compared to other fats and oils. Palm oil raises plasma cholesterol only when an excess of dietary cholesterol is presented in the diet. Two meta-analysis have examined the effect of palmitic acid (found in palm oil) on serum cholesterol. In a 1997 study based on 134 clinical studies, British researchers concluded that, compared to carbohydrates, palmitic acid raises blood cholesterol levels (Clarke et al. 1997). In 2003, Dutch scientists conducted a meta-analysis of 35 clinical studies (Mensink et al. 2003) and examined what many experts consider the best indicator of heart-disease risk: the ratio of total cholesterol to HDL cholesterol (Institute of Medicine, National Academies 2002). Palmitic acid increased the total: HDL cholesterol ratio more than other saturated fatty acids, including lauric acid and myristic acid, which are abundant in palm kernel oil and coconut oil; the other highly saturated tropical oil (Enig 1993). Palm oil increases the total: HDL cholesterol ratio more than the average U.S. or British dietary fat (Jensen et al. 1999; Keys et al. 1957). That finding indicates that, in terms of blood cholesterol, palm oil is somewhat more harmful than the average U.S. dietary fat and much more harmful than such liquid oils as olive, soy, and canola. The World Health Organization has stated that there is “convincing evidence” that palmitic acid increases the risk of cardiovascular disease (World Health Organization 2003).

A number of pre-1990 human feeding studies

reported that palm oil diets resulted in lower serum cholesterol levels than pre-study values. Indeed, scientists concluded that these studies, although not specifically designed to study palm oil, have revealed that a palm oil diet lowered plasma cholesterol compared with the starting periods during which the subjects were eating their habitual Western diets. These conclusions were questioned because the studies were not designed to measure the effects of palm oil. But subsequent studies, specifically designed to evaluate palm oil, confirmed that palm oil's impact on serum lipid and lipoprotein profiles compares favorably to corn oil, lightly hydrogenated soybean oil, and olive oil. Thus, palm oil's impact on serum lipids is more like a monounsaturated than saturated oil. Palm oil contains a high percentage of monounsaturates (40%). Palm oil's saturated fatty acids are palmitic (44%) and stearic (5%), which do not appear to elevate blood cholesterol in people with cholesterol levels within normal ranges. Palm oil stimulates the synthesis of protective HDL cholesterol and removal of harmful Low Density Lipoprotein (LDL) cholesterol. Palm oil is rich in vitamin E, (particularly tocotrienols), which appear to reduce serum cholesterol concentrations (Ebong et al. 1999).

Ansel Keys is largely responsible for starting the anti-saturated fat agenda in the United States. From 1953 to 1957 Keys made a series of statements regarding the atherogenicity of fats. These pronouncements were: "All fats raise serum cholesterol; Nearly half of total fat comes from vegetable fats and oils; No difference between animal and vegetable fats in effect on Coronary Heart Disease (CHD) (1953); Type of fat makes no difference; Need to reduce margarine and shortening (1956); All fats are comparable; Saturated fats raise and polyunsaturated fats lower serum cholesterol; Hydrogenated vegetable fats are the problem; Animal fats are the problem." Recently, an editorial by Harvard's Walter Willett, in the *American Journal of Public Health* (1990) acknowledged that even though "the focus of dietary recommendations is usually a reduction of saturated fat intake, no relation between saturated fat intake and risk of CHD was observed in the most informative prospective study to date." Another editorial, this time by Framingham's William P. Castelli in the *Archives of Internal Medicine* (1992), declared for the record that "...in Framingham, Mass, the more saturated fat one ate, the more cholesterol one ate, the more calories one

ate, the lower the person's serum cholesterol... the opposite of what the equations provided by Hegsted et al. (1965) and Keys et al. (1957) would predict..." Castelli (1992) further admitted that "...in Framingham, for example, we found that the people who ate the most cholesterol, ate the most saturated fat, ate the most calories, weighed the least, and were the most physically active".

Dietary fat and CHD

Dietary fat is principally composed of triacylglycerol (TAG). Therefore, following digestion of a meal, there is a significant increase in the plasma TAG concentration. Prospective epidemiological studies show that plasma TAG, especially the non-fasting level is an important factor in the pathogenesis of coronary heart disease (CHD) (Roche 2000). A positive correlation between plasma TAG levels and CHD risk has been found in women, diabetics, the Japanese and those with elevated LDL or decreased HDL levels (Betterbridge 1999; Mann 1993). An Israeli study suggests that elevated plasma TAG levels are an independent risk factor for mortality among CHD patients (Vogel et al. 1997). Elevation of post-prandial TAG rich plasma lipoproteins and suppression of HDL-C concentrations are considered potentially atherogenic (Patsch 1994). Several clinical studies have shown that elevated levels of TAG rich lipoprotein and its remnants during the post-prandial phase of lipid metabolism are related to the presence and progress of coronary atherosclerosis (Cohn 1994; Roche and Gibney 1995). Atherosclerosis and thrombosis are the two key patho-physiological processes, which lead to the development of CHD. An excessive postprandial TAG response to a meal high in fat can be due to over production of TAG rich lipoproteins to inadequate lipolysis or to abnormalities in the metabolism of remnant lipoproteins. In the latter instance, the remnant lipoproteins will accumulate in the circulation. In this situation, chylomicrons remain in the circulation longer and interact with both LDL and HDL. Chylomicrons give their TAGs to the LDLs, which become smaller and denser, and more atherogenic (Patsch 1994). The HDLs also become over enriched with TAGs. The resultant HDLs are more susceptible to catabolism, whereby the liver removes the cardio protective HDL fraction from the circulation. These chylomicron remnants are a component of the atherosclerotic plaque; therefore

excessive chylomicron remnant concentrations promote the process of atherogenesis (Slyper 1992). It is known that the quantity of dietary fat increases postprandial lipemia in a dose dependent manner and that the production and clearance of lipoproteins and lipoprotein derived remnants are affected by the composition of the diets. A prothrombotic state is produced by elevated post-prandial lipemia because a high concentration of TAG rich lipoprotein in the circulation activates coagulation factor VII (FVII). There are indications that dietary fat intake is a major determinant of factor VII activity (FVIIc). Postprandial TAG rich lipoprotein enhances postprandial FVIIc (Larsen et al. 1997). A positive relationship between FVIIc and CHD mortality has been demonstrated. Patients with CHD have high levels of FVIIc. The possible mechanisms involved could be that the plasma TAG concentration affects the concentration and catabolism of FVII and converts the inactive FVII (zymogen) to FVIIa. Altering the saturated: monounsaturated fatty acid (SFA: MUFA) ratio of an acute test meal does not influence the magnitude of post-prandial FVIIc (Marckman et al. 1990; Roche and Gibney 1997). Several studies have demonstrated that all the traditional coronary risk factors are associated with endothelial dysfunction, independent of the presence of CHD (Vogel et al. 1997). It has become increasingly clear that endothelial cells play important roles in the maintenance of the homeostatic balance in vivo and in the modulation of vascular function in health and disease. Ong et al. (1999) have reported that the consumption of a meal high in monounsaturated fat was associated with acute impairment of endothelial function (as measured by flow mediated reactivity of the brachial artery) when compared with a carbohydrate rich meal. It is pertinent in this context to note that a study in the US found that men with the highest level of saturated fatty acids had the lowest incidence of ischaemic stroke (Gillman et al. 1997). These findings once again reaffirm that fats and oils with different fatty acid compositions do not differ in their acute effects on plasma TAG and FVII levels. The present observations lend further testimony to the merits of Palm Oil in that it is comparable to the other oils in terms of its postprandial lipid response and effects on prothrombotic activity (Thomsen et al. 1999). In developing countries, vegetable oils are replacing animal fats because of the cost and health concerns and Palm Oil has

become one of the major edible oils in the world (Chandrasekharan 1999). It is reassuring to know that the consumption of Palm Oil as a source of dietary fat does not pose any additional risks for coronary artery disease when consumed in realistic amounts as part of a healthy diet (Pedersen et al. 1999).

Increasingly, over the past 40 years, the conception of diet has undergone major changes. Many of these changes involve changes of fats and oils. There has been an increasing supply of the partially hydrogenated *trans*-containing vegetable oils and a decreasing amount of the lauric acid-containing oils (Enig 1996). As a result, there has been an increased consumption of *Trans*' fatty acids and linoleic acid and a decrease in the consumption of lauric acid. This type of change in diet has an effect on the fatty acids the body has available for metabolic activities.

Although popular literature of epidemiological studies usually attribute an increased risk of coronary heart disease (CHD) to elevated levels of serum cholesterol, which in turn are thought to derive from a dietary intake of saturated fats and cholesterol (Mann 1993). But, saturated fats may be considered a major culprit for CHD only if the links between serum cholesterol and CHD and between saturated fat and serum cholesterol are each firmly established. Decades of large-scale tests and conclusions there from have supported to establish the first link. In fact, this relationship has reached the level of dogma. But the scientific basis for these relationships has now been challenged as resulting from large-scale misinterpretation and misrepresentation of the data (Enig 1993; Mann 1993; Smith 1991; Ravnskov 1995; Roche 2000).

CONCLUSION

Epidemiological studies usually attribute an increased risk of coronary heart disease (CHD) to elevated levels of serum cholesterol, which in turn are thought to derive from a dietary intake of saturated fats and cholesterol. Dietary fat is principally composed of triacylglycerol (TAG). Therefore, following digestion of a meal, there is a significant increase in the plasma TAG concentration. Prospective epidemiological studies show that plasma TAG, especially the non-fasting level is an important factor in the pathogenesis of coronary heart disease. In the past, palm oil was attacked as "saturated" since

it contains 44% palmitic acid and 5% stearic acid, and thereby allegedly raises blood cholesterol and increases the risk of cardiovascular disease. However, a sizeable and growing body of scientific evidence indicates that palm oil's effect on blood cholesterol is relatively neutral when compared to other fats and oils. Palm oil raises plasma cholesterol only when an excess of dietary cholesterol is presented in the diet. Palm oil stimulates the synthesis of protective HDL cholesterol and removal of harmful LDL cholesterol. Palm oil is rich in vitamin E, (particularly tocotrienols), which appear to reduce serum cholesterol concentrations and has potent anti-oxidant effects.

REFERENCES

- Anonymous 2007. The Truth About Palm Oil. Available in Official site of American Palm Oil Council <<http://www.americanpalmoil.com/palmtruth.html>> (2003-2004). Retrieved March 28, 2007.
- Anonymous 2007. NST-Life & Times. Good oil in our palm. Official site of tocotrienol.org. Available in News at <<http://www.tocotrienol.org/en/index/news/58.html>> dated 27.2.2007. Retrieved March 28, 2007.
- Anonymous 2007. Palm Oil. Available From Wikipedia, the free encyclopedia at <http://en.wikipedia.org/wiki/Palm_oil> May 2006. Retrieved March 28, 2007.
- Betterbridge DJ 1999. Nutrition and therapeutics: current opinions. *Lipidology*, 10: 467-469.
- Castelli WP 1992. Editorial: Concerning the possibility of a nut. *Archives of Internal Medicine*, 152: 1371-72.
- Chandrasekharan N 1999. Changing concepts in lipid nutrition in health and disease. *Medical Journal of Malaysia*, 54: 408-428.
- Chandrasekharan N, Sundram K, Basiron Y 2000. Changing nutritional and health perspectives on palm oil. *Brunei International Medical Journal*, 2: 417-427.
- Chow CK 1992. *Fatty Acids in Foods and their Health Implications*. New York: Marcel Dekker Inc., pp. 237-262.
- Clarke R, Frost C, Collins R, Appleby P, Peto R 1997. Dietary lipids and blood cholesterol: quantitative meta-analysis of metabolic ward studies. *British Medical Journal*, 314: 112-117.
- Cohn JS 1994. Postprandial lipid metabolism: current opinions. *Lipidology*, 5: 185-190.
- Ebong PE, Owu DU, Isong EU 1999. Influence of palm oil (*Elaeis guineensis*) on health. *Plant Foods for Human Nutrition (Formerly Qualitas Plantarum)*, 53(3): 209-222.
- Enig MG April 25, 1996. Health and Nutritional Benefits from Coconut Oil: An Important Functional Food for the 21st Century. Paper presented in the seminar *AVOC Lauric Oils Symposium* in Ho Chi Min City, Vietnam.
- Enig MG 1993. Diet, serum cholesterol and coronary heart disease. In: GV Mann (Ed.): *Coronary Heart Disease: The Dietary Sense and Nonsense*. London: Janus Publishing, pp. 36-60.
- Gillman MW, Cupples LA, Millen B, Ellison RE, Wolf PA 1997. Inverse association of dietary fat with development of ischaemic stroke in men. *Journal of the American Medical Association*, 278: 2145-2150.
- Hegsted DM, McGandy RB, Myers ML, Stare FJ 1965. Quantitative Effects of Dietary Fat on Serum Cholesterol in Man. *American Journal of Clinical Nutrition*, 17: 281-295.
- Hornstra 1990. Effects of dietary lipids on some aspects of the cardiovascular risk profile. In G Ziant (Ed.): *Lipids and Health: Proceedings of the 2nd European Symposium on Lipids and Health: Lipids and Cancer, Lipids and Cardiovascular Diseases*, Brussels, (International Congress Series). Amazon.com.
- Institute of Medicine, National Academies 2002. *Dietary Reference Intakes: Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients)* pp. 11-17. National Academies Press, Washington DC.
- Jensen J, Bysted A, Dawids S, Hermansen K, Holmer G 1999. The effect of palm oil, lard, and puff-pastry margarine on postprandial lipid and hormone responses in normal-weight and obese young women. *British Journal of Nutrition*, 82: 469-479.
- Kamen B 2007. Palm Oil Tocotrienols Vitamin E for the New Millennium available at <http://www.bettykamen.com/newsletters/palmoil.htm> dated 19.3.2006. Retrieved March 28, 2007.
- Keys A, Anderson JT, Grande F 1957. Prediction of serum-cholesterol responses of man to changes in the diet. *Lancet*, 273(7003): 959-966.
- Koh CS 2007. Comments On Draft Document: Diet, Nutrition, and the Prevention of Chronic Diseases. Available in <http://www.who.int/dietphysicalactivity/media/en/gsfao_cmo_068.pdf> (2006). Retrieved March 28, 2007.
- Larsen LF, Bladbjerg EM, Jespersen J, Marckman P 1997. Effects of fat quality and quantity on postprandial activation of blood coagulation factor VII. *Arteriosclerosis, Thrombosis and Vascular Biology*, 17: 2904-2909.
- Mann GV 1993. A short history of the diet/heart hypothesis. In: GV Mann (Ed.): *Coronary Heart Disease: The Dietary Sense and Nonsense*. London: Janus Publishing, pp. 1-17
- Marckman P, Sandstrom B, Jespersen J 1990. Effect of total fat content and fatty acid composition in diet on factor VII coagulant activity and blood lipids. *Atherosclerosis*, 80: 227-233.
- Mensink RP, Zock PL, Kester ADM, Katan MB 2003. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled studies. *American Journal of Clinical Nutrition*, 77: 1146-55.
- Ong PJJ, Dean TS, Hayward CS, Monica PLD, Sanders TAB, Collins P 1999. Effect of fat and carbohydrate consumption on endothelial function. *Lancet*, 354(9196): 2134.
- Patsch JR 1994. Triglyceride rich lipoproteins and atherosclerosis. *Atherosclerosis*, 110: S 23-S 26.
- Pedersen A, Marckman P, Sandstrom B 1999. Post-

- prandial lipoprotein, glucose and insulin response after two consecutive meals containing rapeseed oil, sunflower oil or palm oil with or without glucose at the first meal. *British Journal of Nutrition*, 82: 97-104.
- Ravnskov U 1995. Quotation bias in reviews of the diet-heart idea. *Journal of Clinical Epidemiology*, 48: 713-719.
- Roche HM, Gibney MJ 1995. Postprandial triacylglycerolemia-nutritional implications. *Progress in Lipid Research*, 34 (3): 249-266.
- Roche HM, Gibney MJ 1997. Postprandial coagulation factor VII activity: the effects of monounsaturated fatty acids. *British Journal of Nutrition*, 77: 537-549.
- Roche HM 2000. Low-fat diets, triglycerides and coronary heart disease risk. *Nutrition Bulletin*, 25: 49-53.
- Slyper AH 1992. A fresh look at the atherogenic remnant hypothesis. *Lancet*, 340: 289-291.
- Smith RL 1991. *The Cholesterol Conspiracy*. St. Louis, Missouri: Warren H Green Inc.
- Thomsen C, Ramussen O, Lousen T, Holst JJ, Fenselau S, Schrezenmeir J, Hermansen K 1999. Differential effects of saturated and monounsaturated fatty acids on postprandial lipemia and incretin responses in healthy subjects. *American Journal of Clinical Nutrition*, 69: 1135-43.
- Vogel RA, Coretti MC, Plotnick GD 1997. Effect of a single high fat meal on endothelial function in healthy subjects. *American Journal of Cardiology*, 79: 350-354.
- WHO 2003. Diet, Nutrition and the Prevention of Chronic Diseases. *WHO Technical Report Series 916*. Geneva: WHO, P. 82, 88 & 88c.
- Willett W 1990. (Editorial) Challenges for public health nutrition in the 1990s. *American Journal of Public Health*, 80: 1295-1298.
- Zhang J, Ping W, Chunrong W, Shou XC, Keyou G 1997. Nonhypercholesterolemic Effects of a Palm Oil Diet in Chinese Adults. *The Journal of Nutrition*, 127: 509S-513S.