Effects of palm oil on cardiovascular risk

Y.H. Chong, PhD
Palm Oil Research Institute of Malaysia,
P.O. Box 10620, 50720 Kuala Lumpur

T.K.W. Ng, PhD
Division of Human Nutrition
Institute for Medical Research, 50588 Kuala Lumpur

Introduction

For nearly a generation now, health agencies in Western countries have been warning about the health hazards of excessive intake of dietary fats, especially of those rich in saturated fats and derived from animal sources such as tallow, lard, butter and cream. Since 1970, world production and consumption of palm oil, particularly of Malaysian origin have increased rapidly. As a result, all saturated fats whether animal or vegetable have been discredited and Malaysian palm oil has been a major target and victim of such avoidance of saturated fats campaigns.

The principal allegation against palm oil is that it is a highly saturated fat and its consumption supposedly raises the levels of blood cholesterol, thereby increasing the risk of coronary heart disease. Unfortunately, the allegation is based more on myths rather than on facts as it takes little consideration of basic lipid nutrition or the advent of emerging new data.

It is also increasingly recognised that the recent anti-palm oil campaigns in the United States were conducted more for economic gains than for genuine concerns of the health of the Americans. This is reflected by the recent estimate of intake of tropical oils in the United States amounting to less than 4% of the daily fat intake and no more than 2.6g of palm oil per capita daily.[1]

The following attempts to put the health effects of palm oil in proper perspective and should also serve to dispel myths and allay the concerns of those who have been misinformed.

Key words: Palm oil, saturated fat, blood cholesterol, blood clotting, experimental atherosclerosis, palm oil vitamin E tocotrienols, polyunsaturated oils, hydrogenated products.

Should palm oil be called a saturated fat?

In recent years, palm oil has been discredited by its competitors as a saturated fat and for its adverse effect on blood cholesterol levels.[2] This undeserved publicity partly stems from confusing palm oil with palm kernel oil as there is a tendency to group palm oil together with palm kernel oil and coconut oil as tropical oils. Palm oil should be distinguished from the latter two oils by its lower level of saturation and its lack of lauric (C12:0) and myristic (C14:0) acids, the latter being the principal cholesterol-raising fatty acid in saturated fats.[3] In fact no lesser an authority than the American Heart Association erred by stating that "palm oil, like coconut and palm kernel oils contains large amounts of lauric (C12:0) and myristic (C14:0) acids both of which are known to raise serum cholesterol".[4] It is comforting to note that the 1988 US Surgeon General's Report on Nutrition and Health listed only coconut oil and palm kernel oil as examples of

vegetable oils rich in saturated fatty acids.[5] Thus whether palm oil or its liquid fractions should continue to be labelled as a saturated fat and consequently stigmatised could be a point of contention.

Palm oil is derived from the mesocarp of the palm fruit whereas palm kernel oil is a minor oil originating from the seed of the palm fruit. Thus while palm kernel oil has a saturated fatty acid content of about 80%, in comparison, palm oil is 50% saturated. Palm olein, the liquid fraction of palm oil and the doubly fractionated palm olein (super olein) now major edible oils in Malaysia, in fact are even more unsaturated than saturated.[6,7]

Table 1 provides the fatty acid composition of refined, bleached and deodorised (RBD) palm oil and its liquid fractions, olein and super olein with those of other common edible oils and fats.

It is to be noted that fractionation of palm oil brings about an enrichment of the monounsaturated oleic acid (18:1, omega-9) and the polyunsaturated linoleic acid (18:2, omega-6) and a concomitant reduction of palmitic acid (16:0), the major saturated fatty acid in palm oil.

In the tropics, olein and super olein have now replaced coconut and groundnut oils as the preferred cooking oils. The oils have moderately good cold stability and remain clear at ambient temperatures of 22°–25°C in air-conditioned supermarkets.

Super olein, when blended in the proportion of 7:3 with seed oils such as soybean and rapeseed oils, permits the use of palm oil as a salad oil in temperate climates. Such blends also have increased oxidative stability.[7,8]

TABLE 1. FATTY ACID COMPOSITION OF PALM OIL AND ITS LIQUID FRACTIONS AND OTHER EDIBLE OILS

Fatty Acid		Coconut Oil	RBD Palm Oil	Palm Olein	Super Olein	Olive Oil	Groundnut Oil	Soyabean Oil	Corn Oil
		- per cent of total fatty acids -							
Caproic	6:0	0.5	-	-	-	-	-	-	-
Caprylic	8:0	8.0	-	-	-	-	-	-	-
Capric	10:0	6.4	-	-	-	-	-	-	-
Lauric	12:0	48.5	0.2	0.2	0.4	-	-	-	-
Myristic	14:0	17.6	1.1	1.0	1.1	-	0.1	0.1	-
Palmitic	16:0	8.4	44.0	39.8	31.5	13.7	11.6	11.0	12.2
Stearic	18:0	2.5	4.5	4.4	3.2	2.5	3.1	4.0	2.2
Oleic	18:1	6.5	39.2	42.5	49.2	71.1	48.5	23.4	27.5
Linoleic	18:2	1.5	10.1	11.2	13.7	10.0	31.4	53.2	57.0
Linolenic	18:3	-	0.4	0.4	0.3	0.6	-	7.8	0.9
Arachidic	20:0	-	0.4	0.4	0.4	0.9	1.5	-	0.1
Saturates		91.9	50.2	45.8	36.6	16.2	*16.3	15.1	14.4
Mononusaturates		6.5	39.2	42.5	49.2	71.1	48.5	23.4	27.5
Polyunsaturates		1.5	10.5	11.6	14.0	10.6	31.4	61.0	57.9

Source : PORIM ; F.D. Gunstone et al (The Lipid Handbook, 1986)

** contains 3% behenic acid (C22:0)*

Effect of palm oil on blood cholesterol levels

The basis for the cholesterol-raising effect of saturated fats dates back to the early study of Keys and Anderson conducted over 30 years ago. Unfortunately in this much quoted study carried out

with various oils (corn, soybean, sunflowerseed, rapeseed, safflowerseed, cottonseed, coconut, olive, sesame, peanut, mustardseed, sardine and menhaden oils and butterfat), palm oil was never used.[9]

Since palm oil was grouped as a source of saturated fat, the assumption was made that palm oil raises blood cholesterol in accordance with the data of Keys-Anderson.

Subsequently a few studies reported that palm oil feeding did result in blood cholesterol values that were higher than those found after feeding the highly unsaturated oils. However, seldom highlighted, was the fact that in all these experiments, the blood cholesterol values after palm oil feeding, were invariably lowered (between 7 and 38%) compared to the periods when the subjects were eating their habitual Western diet.[10–15]

Recent human and animal feeding experiments show that not only palm oil does not raise the levels of blood cholesterol and LDL-cholesterol, it lowered these values compared to other sources of saturated fats of animal and vegetable origin. The cholesterolaemic effect of palm oil is intermediate between the more unsaturated oils and the traditional sources of saturated fats.

The highlights of four recent human palm oil feeding studies are as follows:

- Hornstra & Sundram[16] demonstrated that the maximal replacement of the habitual fats in the Dutch diet with palm oil in a group of 40 male volunteers (in a double blind crossover design consisting of two periods of six weeks' feeding) had no significant effect on blood cholesterol. The levels were 190 mg/dl for Dutch fat blend and 191 mg/dl for palm oil diet. In contrast, the palm oil diet caused a significant increase in the beneficial HDL_2-cholesterol and a significant reduction in the LDL-triglycerides.

- Ng et al[17] compared the effects of diets containing palm olein, corn oil and coconut oil in three groups of adult volunteers in Malaysia in the following dietary sequence:

  Coconut oil – palm olein – coconut oil (Group I, n=27)
  Coconut oil – corn oil – coconut oil (Group II, n=26)
  Coconut oil – coconut oil – coconut oil (Group III, n=27)

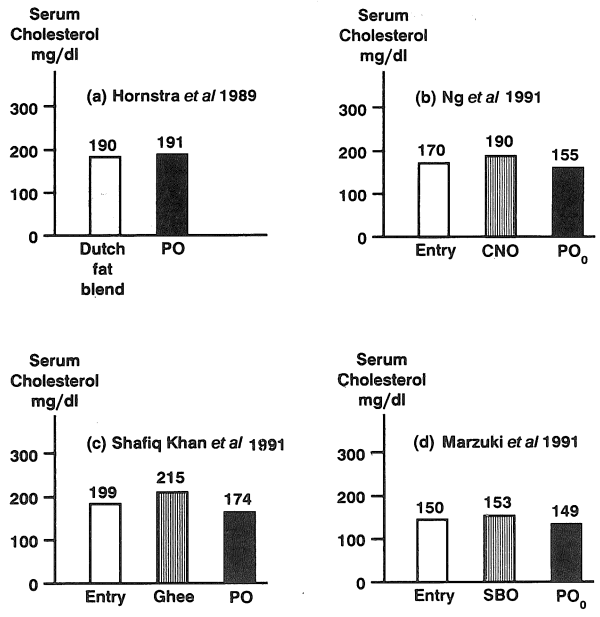
  Each dietary fat was consumed for five weeks at 30 energy percent of which the test fats comprised 75 percent of the total fat. In Group I, palm olein consumption following coconut oil feeding caused a mean serum cholesterol reduction of 36 mg/dl (191 ± 50 mg/dl during coconut oil period and 155 ± 34 mg/dl during palm olein period). For Group II, corn oil feeding following coconut oil reduced serum cholesterol by a mean of 68 mg/dl (190 ± 38 mg/dl during coconut oil period and 122 ± 23 mg/dl during corn oil period) whereas serum cholesterol levels for Group III subjects who were fed a coconut oil diet throughout, remained significantly higher at around 190 mg/dl. The levels of serum cholesterol at entry for all the three groups whose mean age was 24 years were around 170 mg/dl.

- Shafiq Ahmad Khan et al[18] fed four groups of human volunteers in Pakistan, diets enriched with one of the following fats: refined palm oil, butter ghee, vanaspati or hydrogenated cottonseed oil. Each diet was consumed for 60 days. After completion of the first 60 days' feeding, the groups underwent a 10-day washout period, after which the groups interchanged dietary fats, each of which was consumed for another 60 days. Thus the same dietary fat

was consumed over two separate 60-day feeding periods by two different groups of volunteers. On both the 60-day feeding periods during which the palm oil diet was fed, the levels of serum cholesterol remained at 174 and 202 mg/dl, a reduction of 13 and 15 percent respectively compared to the period of entry. A similar effect was, however, not observed during the periods when butter ghee, vanaspati or hydrogenated cottonseed oil was consumed.

- Marzuki and associates[19] provided 110 student volunteers between the ages of 11–17 years with a palm olein diet followed by a soyabean oil diet for five weeks each, interspersed by a six-week washout period. Plasma cholesterol levels during the palm olein period (149 mg/dl) and soyabean oil period (153 mg/dl) were comparable.

EFFECT OF PALM OIL AND OTHER EDIBLE OILS ON SERUM CHOLESTEROL LEVELS



PO : Processed Palm Oil
PO_0 : Palm Olein
CNO : Coconut Oil
SBO : Soyabean Oil

In support of the above observations involving humans, are several animal experiments that have also demonstrated that a palm oil diet lowered blood cholesterol levels as opposed to sheep tallow, lard, the lauric oils and olive oil.[20–22] A recent experiment on monkeys, a species closest to man, showed that increasing the amounts of palm oil by five-fold (palmitic acid) in the diets of three species (cebus, squirrel & rhesus) not only did not raise blood cholesterol levels, but total cholesterol actually declined by 22 mg/dl to 183 ± 9 mg/dl compared to the entry value of 205 ± 11 mg/dl.[23] The palm oil diet lowered the LDL-cholesterol and favourably shifted the ratio of LDL and HDL.

The latter observation was corroborated by enhanced production of HDL-cholesterol and LDL receptors in hamsters fed a palm oil enriched diet.[24]

Effect of palm oil on blood clotting

It is recognised that arterial thrombotic tendency or the potential for a thrombus (clot) to be formed in the blood vessel wall is another important risk factor for cardiovascular disease. Arterial thrombosis can be induced by injury to the blood vessel wall and by alterations to the reactivity of blood platelets which are associated with the process of blood clotting.

In general, studies have shown that the polyunsaturated oils and fish oils decreased platelet aggregation, thereby reducing blood clotting tendency while saturated fats such as beef fat and coconut oil have the opposite effects.[25] Interestingly, a palm oil diet was found to reduce platelet aggregation and decreased blood clotting. Palm oil's behaviour in this respect was similar to the polyunsaturated oils.[26,27]

Arterial thrombotic tendency is closely associated with the balance of local hormones, thromboxane (TxA_2) and prostacyclin (PGI_2). TxA_2 is a very powerful platelet aggregating and vaso-constrictive substance that promotes clotting, while the effects of PGI_2 are opposite to that of TxA_2. Platelet aggregation is inhibited by PGI_2 which also relaxes vessel tone.

The balance of TxA_2 and PGI_2 is thus very important in the maintenance of fluidity of the blood and it is known that people who suffer from coronary heart disease or diabetes have unfavourable TxA_2 to PGI_2 ratios that favour clotting.

There are now a number of reports which show that a palm oil diet in animals either promotes the production of the anti-clotting prostacyclin or decreases the formation of the prothrombotic thromboxane.[22,26–29]

Effect of palm oil on experimental atherosclerosis

Coronary heart disease, the end point of which is a heart attack, is usually preceded by atherosclerosis, a progressive disease of thickening of the arteries with the laying down of fatty deposits.

By feeding diets high in cholesterol and saturated fats, such as found in milk fats, tallow and coconut oil, atherosclerosis can be produced in a variety of animals such as rabbits, quails, pigs and monkeys. Obviously such studies cannot be done on humans.

There are now two reports showing that a palm oil diet does not promote atherosclerosis. The first experiment was conducted by Hornstra[25] in the Netherlands who showed that in the rabbit model, a palm oil enriched diet fed for one and a half years induced the least atherosclerosis compared to fish oil, linseed oil, olive oil and sunflowerseed oil.

More recently Klurfeld[30] from the United States also using the rabbit model compared the effects of palm oil with coconut oil, cottonseed oil and an American fat blend containing a mixture of butterfat, tallow, lard, shortening, salad oils, peanut oil and corn oil. While the coconut oil fed rabbits appeared to have the highest aortic lesions at the end of 14 months feeding, the effects of palm oil were considerably less and no different from the other edible oils including the American fat blend, providing confirmation that consumption of large amounts of palm oil at 32% of fat energy did not result in increased atherogenesis.

Beneficial effects of palm oil Vitamin E tocotrienols

Palm oil is a rich source of Vitamin E and its Vitamin E level is comparable to that found in corn and soyabean oil (Table 2). The predominant palm oil Vitamin E are tocotrienols which are the unsaturated analogues of tocopherol.[31] Most commercial oils including soybean oil and corn oil are devoid of tocotrienols, although tocotrienols are found also in rice-bran oil, wheat germ oil and the oil of barley and oats.[32,33]

Table 2
Vitamin E in refined palm oil

	Mean/Range in ppm
Vitamin E	716 (559–902)
α – tocopherol	158
α – tocotrienol	143
γ – tocotrienol	329
δ – tocotrienol	86

Tocopherols and tocotrienols: They act as potent antioxidants[34–36] serving to protect cellular membrane from destruction by free radicals catalysed lipid peroxidation. Recent evidence indicates that α-tocotrienol has a much higher antioxidant potency than α-tocopherol.[37]

Both tocopherols and tocotrienols promote an anti-thrombotic state by reducing platelet aggregation and modulating prostanoids synthesis.[38–41] In addition tocopherols and tocotrienols reduced the risk of certain types of experimental cancers.[42–44] However only the tocotrienols have been reported to suppress cholesterol production in the liver, thereby lowering blood cholesterol and the atherogenic LDL-cholesterol in animals and human subjects.[32,40,45,46]

Reservations on excessive intake of polyunsaturated oils and their hydrogenated products

Polyunsaturated fatty acids (PUFA) such as linoleic acid (18:2, omega-6) and alpha-linolenic acid (18:3, omega-3) are considered "essential" as the body cannot make these fatty acids. They are necessary for the membrane structure of cells and in the production of an important class of local hormones known as eicosanoids that include the prostaglandins.

Numerous experimental studies have now shown that a diet rich in PUFA, such as found in corn, soybean, safflower and sunflowerseed oils, lowered serum cholesterol. These led to dietary intervention trials being carried out to try to reduce the levels of serum cholesterol by increasing the intake of PUFA. Unfortunately these dietary trials did not significantly alter the mortality due to coronary heart disease and fatalities due to non-cardiovascular causes actually increased in the experimental groups.

Excessive intake of polyunsaturates especially linoleic acid is now associated with gallstone formation, reduction of the beneficial HDL-cholesterol levels, suppression of immune response,

cancer promotion and possibly even atherosclerosis itself through free-radical mediated lipid peroxidation and damage.[47–50] Indeed recent evidence suggests that oxidative modification of low density lipoprotein (LDL) converts it to a more atherogenic form and that LDL samples isolated from subjects fed a diet enriched with linoleic acid were more susceptible to peroxidation and therefore more atherogenic.[51] The recent American Heart Disease Association recommendation[4] that total fat intake should not exceed 30% of calories and that polyunsaturated oils should not exceed 10% of calories is testimony to the restraint and caution now exercised with regard to PUFA, in direct contrast to "the more the better" attitude of previous years.

All polyunsaturated oils are prone to oxidative rancidity. When used for the manufacture of margarines and shortenings they usually need to be hydrogenated producing trans fatty acids isomers. Trans fatty acids should no longer be regarded as harmless. A recent study by Mensink & Katan[52] provided evidence that trans monounsaturated fatty acids raised the levels of the harmful LDL-cholesterol and lowered the levels of the beneficial HDL-cholesterol. Trans fatty acids inhibit the activities of certain membrane-bound enzymes involved in prostaglandin metabolism, promote platelet aggregation[53] and also adversely affect the reproductive performance of animals by way of smaller litter size, irregular oestrous cycles and abnormal sperm morphology.[54]

Palm oil and its fractions can be used directly in a variety of food applications and seldom need to undergo the process of hydrogenation. It contains none of the potentially harmful trans fatty acid isomers.

Summary and Conclusion

A major public health concern of affluent nations is the excessive consumption of dietary fats which are now closely linked to coronary heart disease.

Against this scenario, the tropical oils and palm oil in particular, have been cast as major villains in the U.S.A., despite the fact that palm oil consumption there is negligible. The unsuspecting public may not realise that the call to avoid palm oil is nothing more than a trade ploy since in recent years palm oil has been very competitive and has gained a major share of the world's edible oils and fats market. Many also lose sight of the fact that, palm oil, like other edible oils and fats, is an important component of the diet.

The allegation that palm oil consumption leads to raised blood cholesterol levels and is therefore atherogenic is without scientific foundation. Examination of the chemical and fatty acid composition of palm oil or its liquid fraction should convince most nutritionists that the oil has little cholesterol-raising potential. The rationale for these are:

- it is considered cholesterol free.
- its major saturated fatty acid, palmitic acid (16:0) has recently been shown to be neutral in its cholesterolaemic effect, particularly in situations where the LDL receptors have not been down-regulated by dietary means or through a genetic effect.[55]
- palm oil contains negligible amounts (< 1.5%) of the hypercholesterolemic saturated fatty acids, namely lauric acid (12:0) and myristic acid (14:0).

- it has moderately rich amounts of the hypocholesterolaemic, monounsaturated oleic acid (18:1, omega-9) and adequate amounts of linoleic acid. (18:2, omega-6).

- It contains minor components such as the vitamin E tocotrienols which are not only powerful antioxidants but are also natural inhibitors of cholesterol synthesis.

Feeding experiments in various animal species and humans also do not support the allegation that palm oil is atherogenic. On the contrary, palm oil consumption reduces blood cholesterol in comparison with the traditional sources of saturated fats such as coconut oil, dairy and animal fats. In addition, palm oil consumption may raise HDL levels and reduce platelet aggregability.

As with all nutrients, there is a need to obtain a balance of different fatty acids found in fats in edible oils and other food sources. There is no single ideal source of fat that answers to the recent American Heart Association's call to reflect a 1:1:1 ratio of saturated, monounsaturated and polyunsaturated fats in relation to the recommended dietary fat intake of 30% of calories or less.

Drastic dietary fat changes such as increasing the consumption of polyunsaturates, may upset such a balance so as to alter the composition of cell membranes, prostanoids balance, activity of membrane bound enzymes and receptors, with unknown health implications in the long-run.

On the basis of palm oil's chemistry and the evidence for its favourable cholesterolaemic effect, the incorporation of palm oil in the traditional Asian diet of cereals, legumes, vegetables and meat or fish is certainly nutritionally sound and possibly protective from the viewpoint of cardiovascular health.

References

1. Park YK, Yetty EA. Trend changes in use and current intakes of tropical oils in the United States. Am J Clin Nutr 1990; 51: 738–738.

2. Jones JM. Tropical Oils: Truth and consequences. Cereal Foods World 1989; 34(10): 866–871.

3. Keys A, Anderson JT, Grande F. Serum cholesterol response to changes in the diet. IV. Particular saturated fatty acids in the diet. Metabolism 1965; 14(7): 766–787.

4. Dietary guidelines for healthy American adults. Circulation 77(3) March 1988.

5. The Surgeon General's Report on Nutrition and Health 1988. US Dept of Health and Human Services, Public Health Service, DHHS (PHS) Publication No. 88–50210. p 57.

6. Tan BK, Oh FCH. Malaysian palm oil: chemical and physical characteristics. PORIM Technology No 3 & 4, May 1981.

7. Tan BK. Novel fractions & fats from Palm & Palm Kernel Oils. Palm Oil Development, 11. September 1989, PORIM.

8. Timms RE. Crystallisation behaviour of palm oil. Symposium Proceedings New Developments in Palm Oil. Ed by KB Berger, PORIM, 1990.

9. Keys A, Anderson JT, Grande F. Prediction of Serum cholesterol responses of man to changes in fats in the diet. Lancet 1957; ii: 959–966.

10. Ahrens Jr. EH, Insull W, Blomstrand R, Hirsch J, Tsaltas T, Peterson ML. The influence of dietary fats on serum lipid levels in Man. Lancet 1957; i: 943–953.

11. Anderson JT, Grande F, Keys A. Independence of the effects of cholesterol and degree of saturation of the fat in the diet on serum cholesterol in man. Am J Clin Nutr 1976; 29: 1184–1189.

12. Baudet MF, Dachet C, Lassere M, Estera D, Jacotot B. Modification in the composition and metabolic properties of human low-density lipoproteins by different fats. J Lipid Res 1984; 25: 456–465.

13. Mattson FH, Grundy SM. Comparison of effects of dietary saturated, monounsaturated and polyunsaturated fatty acids on plasma lipid and lipoproteins in man. J Lipid Res 1985; 26: 194–202.

14. Grundy SM. Comparison of monounsaturated fatty acids and carbohydrate for lowering plasma cholesterol. N Engl J Med 1986; 314: 745–748.

15. Bonanome A, Grundy SM. Effect of dietary stearic acid on plasma cholesterol and lipoprotein levels. N Engl J Med 1988; 318: 1244–1248.

16. Hornstra G, Sundram K. The effect of dietary palm oil on cardiovascular risk in man. Abstracts 1989 PORIM International Development Conference, 5–9 September 1989, Kuala Lumpur. N3.

17. Ng TKW, Hassan K, Lim JH, Lye MS, Ishak R. Non-hypercholesterolemic effects of a palm oil-diet in Malaysian volunteers. Am J Clin Nutr 1991; 53: 1015S–1020S.

18. Khan SA, Chugtai AB, Khalid L, Jaffrey SA. Comparative physiological evaluation of palm oil and hydrogenated vegetable oils in Pakistan. Proceedings 1989 PORIM Palm Oil International Development Conference, 5–9 September 1989, Kuala Lumpur. Module 1: Nutrition & Health Aspects of Palm Oil 1991; 16–20.

19. Marzuki A, Arshad F, Tariq AR & Jaarin K. Influence of dietary fat on plasma lipid profiles of Malaysian Adolescents. Am J Clin Nutr 1991; 53: 1010S – 4S.

20. Ong ASH, Qureshi N, Qureshi AA et al. Effects of palm oil and other dietary fats on cholesterol regulation in chickens. The FASEB Journal Vol 2: No 5, March 20, A1541, 1988.

21. Sugano M. One counter argument to the theory that tropical oils are harmful. Yukagaku (J Jap Oil Chem Soc) 1987; 40: 48–51.

22. Sundram K, Khor HT, Ong ASH. Effect of dietary palm oil and its fractions on rat plasma and high density lipoprotein lipids. Lipids 1990; 25(4): 187–193.

23. Hayes KC, Pronczuk A, Lindsey S. Dietary plamitic acid lowers cholesterol by comparison to lauric and myristic acids in monkeys. Am J Clin Nutr 1991; 53: 491–498.

24. Lindsey S, Benattar J, Pronczuk A & Hayes KC. Dietary palmitic acid enhances HDL-cholesterol and LDL receptor MRNA abundance in hamsters. Proc Exp Biol & Med 1990; 195: 261–269.

25. Renaud S. Nutrients, platelet functions and coronary heart disease. In emerging problem in human nutrition, bibliotheca nutritio et dieta, Vol 40. Somogyi JC, Renaud S, Asttier-Dumas M (ed), pp 1–17. Basel: Karger, 1987.

26. Hornstra G. Dietary lipids and cardiovascular disease: effects of palm oil. Oleagineux 1988; 43: 75–81.

27. Rand ML, Hennissen AHM, Hornstra G. Effects of dietary palm oil on arterial thrombosis, platelet response and platelet membrane fluidity in rats. Lipids 1988; 23: 1019–1023.

28. Abeywardena MY, McLennan PL, Charnock JS. Increase in myocardial PGI/TXA balance following long-term palm oil feeding in the rat. J Molec Cell Cardiol (Supp II) 1989: 21:599.

29. Charnock JS Abeywardena MY, McLennan PL. Effects of palm oil-enriched diet on cardiac arrhythmia and thrombogenesis in a rat model of sudden death. International Conference on fats for the Future II. 12–17 February 1989, Auckland, New Zealand. Elaeis (The International Journal of Oil Palm Research and Development) Abstract 1989; 1(1): 84.

30. Klurfeld D, Davidson LM, Lopez-Guisa, JM et al. Palm and other edible oils: atherosclerosis study in rabbits. The FASEB Journal. 4: February, 1990.

31. Gapor AB, Berger KG, Hashimoto T, Kato A, Tanabe K, Mamoro H, Yamaoka M. Effects of processing on the content and composition of tocopherols and tocotrienols in palm oil. Proceedings of the International Conference on Palm Oil Product Technology in the Eighties, 22–24 June 1981, Kuala Lumpur, 145–156.

32. Qureshi AA, Burger WC, Peterson DM, Elson CE. The structure of an inhibitor of cholesterol biosynthesis isolated from barley. J Biol Chem 1986; 261: 10544–10550.

33. Tan B. Palm carotenoids, tocopherols and tocotrienols. JAOCS 1989; 66: 770–76.

34. Walton JR and Packer L. Free radical damage and protection: Relationship to cellular aging and cancer. In: Vitamin E, A Comprehensive Treatise, ed by LJ Machlin, Marcel Dekker, Inc, New York 1980, 495–512.

35. Hirai S, Okamoto K and Morimatsu M. Lipids peroxide in the aging process. In: Lipid Peroxides in Biology and Medicine ed by K Yagi, Academic Press, New York 1982, 305–314.

36. Gapor AB, Ong ASH, Kato A, Watanabe H, Kawada T. Antioxidant activities of palm vitamin E with special reference to tocotrienols. Elaies (The International Journal of Oil Palm Research and Development) 1989; 1(1): 63–67.

37. Serbinova E, Kagan V, Han D and Packer L. Free radical recycling and intra membrane mobility in antioxidant properties of α-tocotrienol. Free Rad Biol Med (in press)

38. Steiner M and Anatasi J. Vitamin E and platelet aggregation. J Clin Invest 1975; 57: 732–737.

39. Chan AC and Leith MK. Decreased prostacylin synthesis in vitamin E deficient rabbit aorta. Am J Clin Nutr 1981; 34: 2341–2347.

40. Qureshi AA, Qureshi N. Lowering of serum cholesterol in hypercholesterolemic humans by tocotrienols (Palmvitee). Am J Clin Nutr 1991; 53: 1021S–6S.

41. Holub BJ, Sicilia F, Mahadevappa VG. Effect of tocotrienol derivatives on collagen – and ADP-induced human platelet aggregation. Abstracts 1989 PORIM International Palm Oil Development Conference, 5–9 September, Kuala Lumpur, N9.

42. Tengerdy RP. Effect of vitamin E on immune response. In Vitamin E, A Comprehensive Treatise, ed by LJ Machlin, Marcel Dekker, Inc, New York 1980, 495–512.

43. Kato A, Yamaoka K, Tanaka A, Komiyama K and Umezauk L. Physiological effect of tocotrienol. Yukagaku (J Jap Oil Chem Soc) 1985; 34: 375–376.

44. Komiyama K, Iizuka K, Yamaoka M, Watanabe H, Tsuchiya N, Umezawa I. Studies on the biological activity of tocotrienols. Chem Pharm Bull 1989; 37(5): 1369–1371.

45. Wentworth BC, N Qureshi, Kim Wright, et al. Suppression of apoliprotein B, thromboxane B2 and platelet factor four by palm oil and its tocotrienols in genetically hypercholesterolemic quail. INFORM 1990; 1: 331.

46. Weber FE, Chaudhary V, N Qureshi et al. Dietary tocotrienols reduce levels of plasma cholesterol, apolipo-protein B, thromboxane B2 and platelet factor four in pigs with inherited hyperlipedemias. INFORM 1990; 1:331.

47. Ahrens EH Jr. In: Diet and Prevention of Coronary Heart Disease and Cancer. Hallgren B (ed), Raven Press, NY 1986 p 81–111. 47. Connor WE and Connor SH. In: Diet and Prevention of Coronary Heart Disease and Cancer. Hoaagren B (ed), Raven Press, NY, 1986: 113–143.

48. Connor WE, Connor SH. In: Diet & Prevention of Coronary Heart Disease & Cancer. Hoaagren B (ed.) Raven Press, N.Y. 1986: 113–143.

49. Smith AD. Can diets rich in polyunsaturated fatty acids be harmful? In: Role of Fats in Human Nutrition, Padley FB (ed), Ellis Horwood Ltd, England, 1985.

50. Grundy SM. Effects of fatty acids in lipoproteins in man. In: Health Effects of Polyunsaturated Fatty Acids in Sea Foods, Simopoulis P (ed), Academic Press, 1986.

51. Parthasarathy S, Khoo JC, Miller E et al. Low density lipoprotein rich in oleic acid is protected against oxidative modification: Implications for dietary prevention of atherosclerosis. Proc Natl Acad Sci, 1990; 87: 3894–3898.

52. Mensink R, Katan MB. Effect of dietary trans fatty acids on high-density and low-density lipoprotein cholesterol levels in healthy subjects. New Engl J Med 1990; 323 (7): 439–445.

53. Alam SQ, Ren YF, Alam BS. Effect of dietary fatty acids on some membrane-associated enzymes and receptors in rat heart. Lipids 1989; 24: 39–43.

54. Hanis T. Effects of dietary trans-fatty acids on reproductive performance of Wistar Rats. Br J Nutr 1989; 61: 519–529.

55. Pramod Khosla and Hayes KC. Comparison between dietary palmitate, oleate and linoleate on plasma lipoprotein metabolism in Cebus and Rhesus monkeys. Am J Clin Nutr (in press).