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.\textsuperscript{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.\textsuperscript{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°–28°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.\textsuperscript{7,8}

\begin{table}
\centering
\begin{tabular}{|l|c|c|c|c|c|c|c|}
\hline
Fatty Acid & Coconut Oil & RBD Palm Oil & Palm Olein & Super Olein & Olive Oil & Groundnut Oil & Soyabean Oil & Corn Oil \\
\hline
Caproic 6:0 & 0.5 & - & - & - & - & - & - & - \\
Caprylic 8:0 & 8.0 & - & - & - & - & - & - & - \\
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 & 45.6 & 36.6 & 16.2 & *16.3 & 15.1 & 14.4 \\
Monounsaturates & 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 \\
\hline
\end{tabular}
\caption{Fatty Acid Composition of Palm Oil and Its Liquid Fractions and Other Edible Oils}
\end{table}


\textit{* 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 & Sundram16 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 HDL2-cholesterol and a significant reduction in the LDL-triglycerides.

- Ng et al17 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 al18 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\(^1\)\(^9\) 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**

![Chart showing serum cholesterol levels](chart.png)

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.\(^2\)\(^0\)—\(^2\)\(^2\) 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.\(^2\)\(^3\) 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.\(^2\)\(^4\)
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. 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.

Arterial thrombotic tendency is closely associated with the balance of local hormones, thromboxane (TxA2) and prostacyclin (PGI2). TxA2 is a very powerful platelet aggregating and vaso-constrictive substance that promotes clotting, while the effects of PGI2 are opposite to that of TxA2. Platelet aggregation is inhibited by PGI2 which also relaxes vessel tone.

The balance of TxA2 and PGI2 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 TxA2 to PGI2 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.

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 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 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. 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.

Table 2
Vitamin E in refined palm oil

<table>
<thead>
<tr>
<th>Vitamin E</th>
<th>Mean/Range in ppm</th>
</tr>
</thead>
<tbody>
<tr>
<td>α - tocopherol</td>
<td>158</td>
</tr>
<tr>
<td>α - tocotrienol</td>
<td>143</td>
</tr>
<tr>
<td>δ - tocotrienol</td>
<td>329</td>
</tr>
<tr>
<td>δ - tocotrienol</td>
<td>86</td>
</tr>
</tbody>
</table>

Tocopherols and tocotrienols: They act as potent antioxidants 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.

Both tocopherols and tocotrienols promote an anti-thrombotic state by reducing platelet aggregation and modulating prostanoids synthesis. In addition tocopherols and tocotrienols reduced the risk of certain types of experimental cancers. 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.

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.\textsuperscript{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.\textsuperscript{51} The recent American Heart Disease Association recommendation\textsuperscript{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\textsuperscript{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\textsuperscript{53} and also adversely affect the reproductive performance of animals by way of smaller litter size, irregular oestrous cycles and abnormal sperm morphology.\textsuperscript{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.\textsuperscript{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


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