Modulation of human lipids and lipoproteins by dietary palm oil and palm olein: a review

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Several human clinical trials have now evaluated palm oil's effects on blood lipids and lipoproteins. These studies suggest that palm oil and palm olein diets do not raise plasma TC and LDL-cholesterol levels to the extent expected from its fatty acid composition. With maximum substitution of palm oil in a Western type diet some coronary heart disease risk factors were beneficially modulated: HDL2-cholesterol was significantly increased while the apolipoprotein B/A1 ratio was beneficially lowered by palm oil. Comparison of palm olein with a variety of monounsaturated edible oils including rapeseed, canola, and olive oils has shown that plasma and LDL-cholesterol were not elevated by palm olein. To focus these findings, specific fatty acid effects have been evaluated. Myristic acid may be the most potent cholesterol raising saturated fatty acid. Palmitic acid effects were largely comparable to the monounsaturated oleic acid in normolipidaemic subjects while *trans* fatty acids detrimentally increased plasma cholesterol, LDL-cholesterol, lipoprotein Lp(a) and lowered the beneficial HDL-cholesterol. Apart from these fatty acids there is evidence that the tocotrienols in palm oil products may have a hypocholesterolaemic effect. This is mediated by the ability of the tocotrienols to suppress HMG-CoA reductase. These new findings on palm oil merit a scientific reexamination of the classical saturated fat-lipid hypothesis and its role in lipoprotein regulation.

Key words: human lipids, lipoproteins, fatty acids, palm oil, coronary heart disease

Introduction

Dietary fats (and fatty acids) are known to modulate plasma lipids and lipoproteins. This concept has been extensively researched upon since the early 1950s and evidence has steadily accumulated hypothesising a positive correlation between saturated fat intake and increased levels of plasma total cholesterol (TC) in humans. The classical Keys and Hegsted equations[1,2] indicated that the three saturated fatty acids lauric, myristic and palmitic were equally cholesterol raising. Hegsted[3] originally showed that myristic acid was more cholesterolaemic than palmitic acid in humans. Nevertheless, this conclusion was subsequently revised after a series of experiments with modified triglycerides. Thereafter, both investigators developed their own regression equations that predicted plasma cholesterol response on the basis of energy contributed by the sum of saturated and polyunsaturated fatty acids in one's diet. These equations assumed that the monounsaturates were neutral but that dietary cholesterol affected plasma cholesterol besides the fatty acids.

Resulting from these and other findings, there has been a tremendous effort to educate the consumer to choose fats containing fatty acids that could help maintain normal cholesterol levels. Such recommendations are embodied in almost every major national health report focused at reducing the incidence and mortality from coronary heart disease (CHD). Awareness of these recommendations by the consumers has been shown by a switch from animal saturated fats to polyunsaturated oils. Such changes are however related to the functionality of the oils and fats concerned. The replacement of butter with margarine and the trend towards the increased consumption of polyunsaturated margarines and other low saturates containing fat-rich products was seen as a positive stride in reducing CHD incidence. New data has now shown that hydrogenation of liquid polyunsaturated and monounsaturated oils used in such product formulations results in *trans* fatty acids that increase the lipid associated risk factors. Since palm oil contains 44% of its composition as saturated palmitic acid, it is generally assumed that TC elevation following its long term consumption would be imminent. Indeed several human studies[4-8] have reported that palmitic acid enriched diets derived from palm oil resulted in higher TC and low density lipoprotein cholesterol (LDL-C) than did diets enriched either in oleic or linoleic acids. However, newer studies to be examined below have since produced results that are contradictory to the above. At least one population (epidemiology) study has reported that normal TC values are possible in a dietary environment in which palm oil was the predominant fat source[9]. The issue is further confounded by reported effects of triglyceride species[10] and the minor components[11] on cholesterol modulation.

Historical studies evaluating palm oil effects

One of earliest clinical trials evaluating palm oil was pioneered by Arhens *et al*[12] who fed two of their subjects a liquid formula diet containing 40% energy as palm oil under metabolic conditions. The TC levels of both these subjects

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fed palm oil was significantly higher than during a corn oil period. Nevertheless, the TC values after the palm oil period was lower than the baseline values. Grande *et al*[13] showed that a palm oil enriched diet resulted in higher TC than a diet predominated by stearic acid derived from cocoa butter. This study was also noteworthy in that it confirmed Key's earlier observation that stearic acid lacked a cholesterol raising effect.

Anderson *et al*[4] fed 12 volunteers diets containing 35% saturated fat contributed by two parts of palm oil and one part coconut oil and compared its cholesterolaemic effects with a polyunsaturated safflower oil diet. The safflower oil diet resulted in lower serum TC than the saturated fat diet. However, the saturated fat diets actually resulted in approximately 10% lower serum TC levels than the subjects' habitual diets. In 1984, Baudet *et al*[5] undertook a dietary trial using Benedictine nuns to evaluate the effect of 30% fat calories contributed predominantly (two thirds) by palm oil, sunflower seed oil, peanut oil or milk fat on serum lipid and lipoprotein levels. The sunflower seed oil diet reduced serum TC and LDL-C significantly compared with all other diets. Serum TC and LDL-C were essentially similar after the palm oil and peanut oil diets whereas milk fat resulted in significantly higher TC and LDL-C levels than all the other test diets.

Mattson and Grundy[6] fed 20 male volunteers a liquid formula diet containing 40% calories contributed either by palm oil, high oleic safflower oil or high linoleic safflower oil. After four weeks, the high oleic and high linoleic safflower oil diets produced significantly lower TC and LDL-C than the palm oil diet. HDL-C on the palm oil and high oleic safflower oil diets were similar but HDL-C on the high linoleic safflower oil diet was significantly lower.

In a follow-up study, Grundy and Vega[7] fed 11 patients liquid formula diets containing 40% fat calories (high fat) and compared their effects with a 20% fat calories (low fat) diet. The high fat diets were formulated with either coconut oil, palm oil or high oleate safflower oil. The 11 patients were then subdivided into two groups in which seven were fed the coconut oil diet while the remaining four the palm oil diet. The patients were al so rotated through the high-oleate safflower oil and low-fat diets. TC and LDL-C were significantly lower on the high oleate safflower oil diet compared with all other test diets. The four patients on the palm oil diet had TC, LDL-C and HDL-C values than were lower than the coconut oil diets and the habitual diets of these patients.

Bonanome and Grundy[8] evaluated the impact of palm oil, high oleic safflower oil and an interesterified fat blend (43% 18:0 and 40% 18:1) using liquid formula diets in 11 elderly patients. The diets contributed 40% fat calories and were consumed by the subjects for three weeks in a random order. Mean TC and LDL-C after the palm oil diet was significantly higher than the values of either the high oleic safflower oil or the high stearate interesterified fat. Cholesterol levels after the palm oil period were 11% lower than the entry (habitual) levels but this was discounted by the authors who suggested that lowering of the cholesterol levels of subjects on admission to a metabolic ward was a commonly observed phenomenon. The study was also important in that it concluded that stearic acid had a neutral impact on cholesterol and lipoprotein levels in humans.

Laine *et al*[14] compared the effect of palm oil, corn oil, soybean oil and lightly hydrogenated soybean oil added to cholesterol rich diets containing 35% fat energy in 24 normocholesterolaemic students. Cholesterol levels after the corn oil, soybean oil and lightly hydrogenated soybean oil were lower by 14, 13 and 9% respectively compared with the palm oil diet. The analysis of this data was however complicated by the higher levels of dietary cholesterol consumed during the palm oil period.

These studies are often cited as examples of the cholesterol raising properties of palm oil containing 50% of its fatty acid composition as saturates. On closer examination of these studies, several fallacies have been pointed out. For example, these studies were characterised by:

1) the use of liquid formula diets in which fats contributed about 40% energy,
2) the use of relatively older subjects with moderate to severe hypercholesterolemia,
3) the feeding of atypical diets in which the target fatty acid often represented an excessive intake of the total fatty acids.

These characteristics led to plasma lipid changes that seemingly established the cholesterol raising effects of palm oil. However, most latter date studies in which solid-food diets were used with more realistic fatty acid exchanges and mildly hypercholesterolaemic to normocholesterolaemic younger subjects, the cholesterol raising attribute of palm oil was either muted or disappeared. In contrast to the older studies, recent trials have used palm olein, the liquid fraction of palm oil rather than palm oil itself. Whether the switch to palm olein having a higher unsaturated fatty acid composition (reduced palmitic, increased oleic and linoleic acids) resulted in the muted cholesterol response in the subjects is not clearly defined. Some of these recent studies are discussed below.

Palm olein versus polyunsaturated oils

Marzuki *et al*[15] using young volunteers evaluated the effect of consuming foods containing either palm olein or soybean oil. In normal healthy volunteers the level of serum TC and LDL-C was not affected by the palm olein or soybean oil diets. In hypocholesterolaemic (HYPO or HYPER?) subjects however the soybean oil diet induced higher serum TC and LDL-C levels than the palm olein diet. In a similar study[16] when volunteers were switched from a coconut oil diet to a palm olein or a corn oil diet, serum TC dropped by 36 mg/dL and 51 mg/dL respectively. Hence a reduction in serum TC was observed on administering a palm olein or corn oil diet relative to a coconut oil diet. However the decrease in TC due to corn oil was significantly better than that on palm olein. Ghafoorunissa *et al*[17] substituted palm olein for groundnut oil in the typical Indian diet contributing 27% energy as fat. This effectively doubled the availability of the saturated fatty acids and decreased by half the linoleic acid content of the diet. In spite of these major shifts in the fatty acid composition due to the use of palm olein, plasma levels of cholesterol and the lipoproteins were not altered in this population.

Palm olein versus the monounsaturated oils

Ng *et al*[18] evaluated the effects of palm olein and olive oil on serum lipids and lipoproteins in comparison to a coconut

oil diet. Each test oil was served as the sole cooking oil and contributed two thirds of the total fat intake. The coconut oil diet significantly raised all the serum lipid and lipoprotein parameters, ie TC, LDL-C and HDL-C. However, the one-to-one exchange between palm olein (rich in 16:0) and olive oil (rich in 18:1) resulted in identical TC, LDL-C and HDL-C values. This showed that in healthy normocholesterolaemic humans, palm olein can be exchanged for olive oil (high oleic) without adversely affecting the serum lipids and lipoprotein levels. Choudhury *et al*[19] managed a 5% en exchange between palm oil (16:0-rich) and olive oil (18:l-rich) in 21 healthy normocholesterolaemic Australian men and women consuming a low fat (30% en) and low dietary cholesterol (<200 mg/day) diet. Under these conditions, TC and LDL-C were almost identical between the two oils, so that when 16:0 in palm oil was replaced with 18:1 in olive oil, the expected increase in TC and LDL-C were not evident. A similar effect between palm olein and canola oil was also reported by the same authors in a previous human study[20].

Sundram *et al*[21] fed 23 healthy normocholesterolaemic male volunteers carefully designed whole food diets containing canola oil (18:1-rich), palm olein (16:0-rich) or an American Heart Association Step 1 diet (AHA), all contributing approximately 3 l % en fat and <200mg dietary cholesterol/day. These diets represented the direct exchange of 7% en 18:1+18:2 between canola oil and palm olein whereas the main difference between palm olein and AHA was < 4% en exchanged between 16:0 and 18:2. Serum TC, VLDLC and LDL-C were not significantly affected by these three diets despite manipulations of the key fatty acids. The effects between the high 18:1 canola and the high 16:0 palm olein were essentially identical. Only HDL-C after the AHA diet attained significance compared with the other two diets.

In contrast to the above studies, Zock *et al*[22] reported that replacing 10% en from 16:0 with 18:1 in normocholesterolaemic subjects significantly lowered TC and LDL-C. This Dutch study did not use natural fat sources. The 18:1-rich diet was prepared by blending high 18: 1 sunflower oil, fully hydrogenated sunflower oil and high 18:2 sunflower oil and interesterified palm oil mixed with other edible oils. The 16:0-rich diet was formulated by blending fractionated palm oil, cottonseed oil, and fully hydrogenated sunflower oil. The feeding of fat blends containing atypical triglyceride moieties may have been partially responsible for the observed increase in TC and LDL-C. By contrast, when Sundram *et al*[22] maximally replaced the habitual Dutch diet with palm oil TC and LDL-C was unaffected. The palm oil diet however resulted in significant improvements in the HDL2-C and the apolipoprotein A1/B ratio signalling some cardiovascular benefits rather that the reverse to be true for palm oil.

Palm oil (16:0-rich) versus other saturates

The human diet contains a mixture of different fats, and therefore mixtures of different fatty acids. The net effect of such a mixture on TC and/or the individual lipoproteins will be the sum of many fatty acids, some acting in opposite directions to each other. It is therefore important to decipher the key cholesterol modulating fatty acids to determine the cholesterolaemic index of the fat or oil consumed. Fortunately, several recent human studies have focussed on these issues and have provided additional observations that tend to support the Hegsted[3] observation that saturated fatty acids differ in their cholesterol regulating ability. Some of these studies that used palm oil as a source of 16:0 in their test diets are described below.

Sundram *et al*[24] fed 17 normocholesterolaemic subjects whole food diets that exchanged 5% en between 16:0 and 12:0+14:0 (LM). Compared with the LM diet, the 16:0 rich diet produced a 9% lower TC concentration reflected primarily by a lower (11%) LDL-C concentration. Heber *et al*[25] evaluated diets enriched in palm oil, coconut oil or hydrogenated soybean oil for 3-week test periods in healthy American males. Significant increases in TC, LDL-C and apolipoprotein B was apparent following consumption of the coconut oil diet but not the palm oil and the hydrogenated soybean oil diet. In the Ng studies[16,18], coconut oil enriched diets were compared to palm olein. In both populations, the coconut oil feeding resulted in significant increases in TC and LDL-C compared with the palm olein feeding.

These studies compared the effects of 12:0+14:0 occurring naturally in coconut oil and palm kernel oil. They suggest that the cholesterolaemic effect due to 16:0 (palmitic acid) is significantly lower than that of a LM combination. Coconut oil is almost 85% saturated and it has been suggested that the higher cholesterol values after a coconut oil diet may be simply due to the lower availability of linoleic acid. This suggestion has been discounted in the recent study of Sundram *et al*[26] wherein, despite the incorporation of a high level of 18:2 (5.6% en) in the LM diet, it induced significantly higher concentrations of TC and LDL-C in healthy volunteers compared to a 16:0-rich palm olein diet (3.3% en as 18:2).

The higher TC and LDL-C induced by the LM diets are inconsistent with the values expected based on the Keys-Hegsted equations[1-3] which predict that identical TC concentrations would result from both fatty acids. However it is arguable that the simplified combination of the different dietary saturates effects in the Keys-Hegsted regressions tend to overestimate the importance of 16:0 and underestimate the impact of 12:0+14:0. The question that remains is of the two fatty acids namely 12:0 and 14:0 which is more cholesterolaemic? The separation of 12:0 and 14:0 from natural fat sources is difficult since they tend to co-occur. However, by manipulating coconut oil (higher 12:0) and butter fat (higher 14:0) intake separation of the 12:0 versus 14:0 cholesterolaemic effects has been achieved[27]. The data suggests that 14:0 is the most potent cholesterol raising saturate and this potency has been calculated to be four times that of 16:0. The lower cholesterol raising ability of 12:0 in relation to 16:0 is however, less clearly defined.

Using 15 normocholesterolaemic women fed solid-food diets, Schwaab *et al*[28] failed to find any difference on plasma lipid levels following a 4% en exchange between 12:0 and 16:0. Temme *et al*[29] reported the effects of feeding diets enriched in lauric and palmitic acids on plasma lipids. The subjects consumed solid food diets that exchanged 8% en between lauric and palmitic acids. The lauric acid diet induced higher TC and LDL-C than the palmitic acid diet but this could not be explained by the somewhat higher myristic acid content in the diet. Accordingly, the plasma

lipid changes would appear to suggest that lauric acid per se was more cholesterol raising than the palmitic acid. In the Denke and Grundy study[30], the 12:0-rich diet (contributing 17.6% en) raised TC by 9 mg/dL compared with a diet with 17.4% en 16:0. The increase occurred exclusively in LDL-C. These data therefore suggest that the cholesterolaemic effects of 16:0 derived from palm oil/palm olein are lower than that of 12:0 and 14:0 derived from natural fats including coconut oil, palm kernel oil and butter fat.

Palm oil versus hydrogenated fats (*trans* fatty acids)

Controversy continues over the significance of *trans* fatty acids in human nutrition, particularly concerning their negative impact on the plasma lipoprotein profile and its untoward implications for atherogenesis. *Trans* fatty acids can deleteriously affect lipoproteins by increasing TC, LDL-C, lipoprotein Lp(a) and decreasing HDL-C relative to their cis isomers. This has raised the need to replace hydrogenated fats with natural solid fats in a large number of food formulations. The nutritional efficacy of the solid fats replacing hydrogenated fats should be such that they do not adversely affect plasma lipids and other CHD risk factors. In this context palm oil is perceived as a suitable alternative.

Nestel *et al*[31] compared a *trans* elaidic rich fat with a 16:0-rich blend (16:0 contributed mainly by palm oil). Both test blends resulted in higher TC and LDL-C than an oleic-rich control diet. There was essentially no difference in TC and LDL-C between the elaidic-rich and palm oil-rich test diets. HDL-C was however significantly raised on the 16:0-rich diet and the resulting LDL/HDL-C ratio was more favourable than the *trans* diet. This led the authors to conclude that there is little benefit from avoiding the use of palm oil by substituting *trans* fatty acids in food formulations. Sundram *et al*[26] undertook a direct comparison between *trans* elaidic fat designed to replace the saturates (16:0, 12:0+14:0) in foods and food processing. Feeding of elaidic acid at 5.5% en significantly elevated TC and LDL-C relative to the 16:0-rich (palm olein) and 18: l-rich fats and uniquely depressed HDL-C and increased lipoprotein Lp(a) relative to all the fats tested (including 12:0+14:0). Identical effects on lipoproteins were elicited by the 16:0 and cis 18:1-rich diets. The authors concluded that the impact of *trans* elaidic acid on the lipoprotein profile of humans appears to be worse than the saturates occurring in natural oils and fats.

Conclusion

These studies suggest that the cholesterolaemic properties of palm oil and palm olein are dependent upon several set points. Palm oil and palm olein have been shown to be hypocholesterolaemic in comparison with diets contributing variable amounts of lauric and myristic fatty acids. This augers well for the hypothesis that the cholesterolaemic effects of the saturated fatty acids are not equal. Indeed the neutrality of stearic acid has long been advocated. In comparison to diets enriched by canola, rapeseed and olive oils, palm olein appears to be comparable in its ability to modulate the lipids and lipoproteins. The studies that lend credence to this fact were conducted with normal healthy volunteers consuming moderate fat energy loads (30% en) and moderate dietary cholesterol (<300 mg/day). When hypercholesterolaemic subjects and high fat liquid formula diets were used palm oil appeared to raise TC and LDL-C. There is a lack of data for palm oil's effects on hypercholesterolaemics and this issue will need to be addressed soon. Palm oil and palm olein could also continue as important ingredients in food applications requiring solid fats without hydrogenation. It certainly seems nutritionally superior to hydrogenated fats by not increasing TC and LDL-C while sometimes even aiding in the increase of the beneficial HDL-cholesterol. Apart from its fatty acids, the minor components present in palm oil, especially the tocotrienols have been reported to reduce TC and LDL-C[32] through their ability to suppress HMG-CoA reductase activity. These findings merit a reevaluation of the nutritional properties of palm oil and palm olein especially since it is poised to continue its importance as a major edible oil for human consumption worldwide.

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棕櫚油和棕櫚油酸甘油脂對人體脂類和脂蛋白的調節: 一個評論

摘要

目前有若干人體臨床試驗去評估棕櫚油對血脂及脂蛋白的影響. 這些研究指出棕櫚油及棕櫚油酸甘油酯膳食不會相應地升高血漿甘油三酯和低密度脂蛋白膽固醇水平. 應用大量棕櫚油于西方膳食中對某些冠心病危險因子起到有益的調節. 可引起脱輔基脂蛋白 (apolipoprotein) B/A 比例下降, 而高密度脂蛋白2-膽固醇明顯升高. 比較棕櫚油酸甘油酯和各種單不飽和脂肪酸食用油如油菜子油 (canola) 和橄欖油等, 顯示棕櫚油酸甘油酯并不增加血漿低密度脂蛋白膽固醇水平. 豆蔻酸也許是最有力的升高膽固醇的飽和脂肪酸. 在血脂正常的對象中, 棕櫚油酸的作用類似單不飽和脂肪酸, 可使血漿膽固醇, 低密度脂蛋白膽固醇, 脂蛋白Lp (a) 增高; 高密度脂蛋白膽固醇降低. 除了這些脂肪酸外, 棕櫚油中的生育三烯酚也許有降低血液膽固醇的作用. 這是由于生育三烯酚有抑制HMG-CoA 還原酶的作用. 這個在棕櫚油的新發現, 值得對飽和脂肪酸假說及其在脂蛋白調節中的作用進行一個重新的科學評估.