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 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. HDL-cholesterol was significantly increased while the apolipoprotein B:A1 ratio was beneficially lowered by palm oil. Comparison of palm oil with a variety of monoensaturated 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 normocholesterolemic subjects while trans fatty acids disproportionately raised total 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 hypcholesterolemic 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-cholesterol 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 demonstrating a positive correlation between saturated fat intake and increased levels of plasma total cholesterol (TC) in humans. The classical Keys and Hegsted equations indicated that the three saturated fatty acids lauric, myristic and palmitic acid increased cholesterol raising. Hegsted originally claimed that myristic acid was more cholesterolegenic 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 best.

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. One such strategy is the substitution of polyunsaturated fats for saturated fats in the diet. Such changes are however related to the functionality of the oils and fats consumed. 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 hydrolysis of liquid polyunsaturated and monounsatuated 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 increases following its low cholesterol risk factors. Indeed several human studies have reported that palmitic acid enriched diets derived from palm oil resulted in higher TC and low density lipoprotein cholesterol (LDL-C) than diet 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. The issue is further confounded by reported effects of triglyceride species and the minor components 11 on cholesterol modulation.

Historical studies evaluating palm oil effects

Several clinical trials evaluating palm oil was pioneered by Arbene et al 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 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 showed that a palm oil enriched diet resulted higher in TC than a diet predominated by stea acid light from canned corn oil and butter. This study was also noteworthy in that it confirmed Key’s earlier observation that stearic acid lacked a cholesterol raising effect.

Andero et al fed 12 volunteers diets containing 35% saturated fat contributed by two parts of palm oil and one part coconut oil and compared its cholesterol regulatory 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 consuming the safflower oil diets. Baut et al 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 lipids and lipoproteins. 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 as the result of the palm oil and peanut oil diets whereas milk fat contributed significantly to an increase in the high density lipoprotein cholesterol compared to higher TC and LDL-C levels than all the other test diets.

Marston and Grundy fed 20 male volunteers a liquid formula diet containing 40% calories of either 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, but HDL-C for the palm oil diet and high oleic safflower oil diets were similar but LDL-C on the high linoleic safflower oil diet was significantly lower.

In a follow-up study, Grundy and Vega fed 11 patients liquid formula diets containing 40% fat calories (high oleic 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 oleic 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 also rotated through the high-oleic safflower oil diet and the low oleic soybean oil diet. LDL-C were significantly lower on the high oleic safflower oil diet compared with all other test diets. The four patients on the palm oil diet had TC, LDL-C and HDL-C levels than were lower than the coconut oil diets and the habitual diets of these patients.

Banoose and Grundy evaluated the impact of palm oil, high oleic safflower oil and an intersaturated 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. The TC, TG, LDL-C and HDL-C after the palm oil diet was significantly higher than the values of either the high oleic safflower oil or the high stearic interesterified fat. Cholesterol levels after the palm oil period were 11% lower than the corn oil diets but this was discounted by the authors who suggested that lowering of the cholesterol levels of subjects on admission to a metababolic ward was a commonly observed phenomenon. The study was also important because it indicated that stearic acid had a neutral impact on cholesterol and lipoprotein levels in humans.

Laine et al 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 normocholesterolemic students. Cholesterol levels after the corn oil diet were significantly higher than for the other three oils. Serum TC levels 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 diet.

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 are consumed at 40% of daily energy intake, which is an unphysiological level.
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, more recent studies in patients with moderate to severe hypercholesterolemia showed no cholesterol raising effects of palm oil. Although diet studies in hypercholesterolemic patients did not show cholesterol raising effects of palm oil, the diets were used with more realistic fatty acid exchanges and mildly hypercholesterolemic normocholesterolemic young volunteers fed a diet containing 40% energy from 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 has a effect on cholesterol raising or reduction is currently being followed in a number of studies. These studies have suggested that the liquid olein itself does not raise cholesterol despite the fact that the olein contains a high proportion of saturated fatty acids. This may be due to the fact that the olein contains only 44% saturated fatty acids compared to 50% in palm oil.

Palm olein versus polyunsaturated oils

Marniri et al 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. Dietary linoleic acid subjects however the soybean oil diet induced higher serum TC and LDL-C levels than the palm olein diet. In a similar study when volunteers were switched from a coconut oil diet to a diet containing 40% palm olein 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. Ghafourinia et al substituted palm olein for groundnut oil in the typical Indian diet and found that a diet containing 40% of palm olein reduced the fat content and 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 cholesterol and the lipoproteins were not altered in this population.

Palm olein versus the monounsaturated oils

Ng et al evaluated the effects of palm olein and olive oil on serum lipids and lipoproteins in comparison to a coconut
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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 the positive correlation between saturated fat intake and increased levels of plasma total cholesterol (TC) in humans. The classical Keys and Hegsted equations indicated that the three saturated fatty acids lauric, myristic and palmitic acid were elevated when cholesterol raising. Hegsted originally suggested that the linoleic acid was more cholesterololemic 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 levels.

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. One of these is cod liver oil which is rich in polyunsaturated fatty acids, but cod liver oil is not a practical dietary choice for humans.

The replacement of butter with margarine and the trend towards the increased consumption of palm oil was significantly higher than during a corn oil period. Nevertheless, the TC values after the palm oil period were lower than the baseline values. Grandje et al. showed that a palm oil enriched diet resulted in higher TC than a diet predominated by stearic acid light fraction from hydrogenated corn oil butter. This study was also noteworthy in that it confirmed Key’s earlier observation that stearic acid lack a cholesterol raising effect.

Andersen et al. fed 12 volunteers diets containing 35% saturated fat contributed by two parts of palm oil and one part coconut oil and compared its cholesterololemic effects with a unsaturated sunflower oil diet. The same diet was also used as diet therapy. A study using healthy volunteers showed that this modified diet resulted in lower serum TC and the saturated fat diet. However, the saturated fat diets actually resulted in approximately 10% lower serum TC levels than the subjects had been on a low fat diet. Baudet et al. undertook a dietary trial using Beneclin guidelines 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 actually reduced serum lipid and lipoprotein levels. The high saturated fat diets were used with more realistic fatty acid exchanges and markedly hypocholesterolemic to normolipidemic young male subjects were used whereas the initial palm oil was either muted or disappeared. In contrast to the older studies, recent studies have used palm olein, the liquid fraction of palm oil rather than palm oil itself. Whether the switch to palm olein has a beneficial influence on serum lipids or composition (reduced palmitic, increased oleic and linoleic acids) resulted in the reduced cholesterol response in the subjects is not clearly defined. Some of these recent studies are discussed below.

Palm olein versus polyunsaturated oils

Marnsiki et al. assessing the effect of palm olein and olive oil on serum lipids and lipoproteins showed that palm olein was more hypolipidemic than olive oil. In normal healthy volunteers the level of serum TC and LDL-C was affected by the palm olein or soybean oil diets, while no significant differences were observed between the subjects however the soybean oil diet induced lower serum TC and LDL-C levels than the palm olein diet. In a similar study 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. Ghaffouriania et al. substituted palm olein for groundnut oil in the typical Indian diet composition which would contribute 27% energy contribution. The fat composition of the diet was not altered and 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 LDL-cholesterol and the lipoproteins were not altered in this population.

Palm olein vs. monounsaturated oils

Ng et al. evaluated the effect of palm olein and olive oil on serum lipids and lipoproteins in comparison to a coconut oil diet.
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, i.e. TC, LDL-C and HDL-C. However, the one containing 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 normocholesterolemic humans, palm olein can be exchanged for olive oil, without adversely affecting the serum lipids and lipoprotein levels. Choudhury et al. (26) managed a 5% en exchange between palm oil (16:0-rich) and olive oil (18:1-rich) in 21 healthy normocholesterolemic Asians, replacing daily about 100 g of these oils, 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 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.28

Sunram et al. (29) fed 23 healthy normocholesterolemic 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 31% en fat and 200mg/dietary cholesterol/day. These diets represented the direct exchange of 18:1 in canola oil for 16:0-rich palm olein whereas the main difference between palm olein and AHA was 4% en exchanged between 16:0 and 18:2. Serum TC, VLDL-C and LDL-C were not significantly affected by these three diets despite significantly lower dietary fatty acids. The effects of these high 18:1 canola and the high 16:0 palm olein were essentially identical. Only HDL-C by the AHA diet attained significance compared with the other two diets. In contrast, the results of de Jong et al. (30) reported that replacing 10% en from 16:0 with 18:1 in normocholesterolemic 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 ethyl alcohol, and it was formulated by blending fractionated palm oil, cottonseed oil, and fully hydrogenated sunflower oil. The feeding of fat blends containing stearoyl triglycerides may have been partially responsible for the no responses in TC and LDL-C. By contrast, when Sunram et al. (29) 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 HDL-C and the apolipoprotein A/I ratio suggesting 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 the metabolism of 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 cholesterol lowering effects of the diet consumed. Fortunately, several recent human studies have focussed on these issues and have provided additional observations that tend to support the Hagedorn 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.

Sunram et al. (29) fed 17 normocholesterolemic subjects whole food diets that exchanged 5% en between 16:0 and 12:0+14:0 (LME). Compared with the LME diet, the 16:0 rich diet produced significant increases in TC, LDL-C and apolipoprotein B was apparent following consumption of the coconut oil diet but not the palm oil diet and the hydrogenated soybean oil diet. In the Ng study assessed coconut oil enriched diets without trans fatty acids. 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 cholesterol lowering 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 supported in the recent study of Sunram et al. (29) wherein, despite the incorporation of a high level of 18:2 (5.6% en) in the LM diet, it has not been shown to produce similar 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 in agreement with the Hagedorn equations' which predict that identical TC concentrations would result from both fatty acids. However, it is argued that this simplified combination of the different dietary saturates affects the results when the Hagedorn equations tend to overestimate the importance of 16:0 and underestimate the impact of 12:0+14:0. The question that remains is what are the effects of the other fatty acids, i.e. 16:1 which is more cholesterologenic? 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 hydrogenated soybean oil (lower 14:0) it is possible to determine the 12:0 versus 14:0 cholesterol lowering effects has been achieved.2 The data suggests that 14:0 is the most potent cholesterol lowering saturate and this potency has been calculated to be four times higher in the serum cholesterol in saturated diet.\n


References
iod oil. 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-exchange coconut oil diet (rich in 16:0) and olive oil diet (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 coconut oil without adversely affecting the serum lipids and lipoprotein levels. Choudhury et al[29] managed a 5% en exchange between palm oil diet (16:0-rich) and olive oil diet (18:1-rich) in 21 healthy normocholesterolaemic American males. Significant differences were found between men consuming a low fat (30% en) and low dietary cholesterol (<200 mg/d/d) diet. Under these conditions, TC and LDL-C were almost identical between the diets 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.[29]

Sundaram et al[27] 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 31% en fat and <200mg dietary cholesterol/day. These diets represented the direct exchange of 18:1 in canola oil for 16:0 palm olein whereas the main difference between palm olein and AHA was 4% en exchanged between 16:0 and 18:2. Serum TC, VLDL-C and LDL-C were not significantly affected by these three diets despite significant differences in fatty acids. The effects between the high 18:1 canola and the high 16:0 palm olein diets were essentially identical. Only HDL-C after the AHA diet attained significance compared with the other two diets. In contrast, the study by Albers et al[20] reported that replacing 10% en from 16:0 with 18:1 in normocholesterolaemic subjects significantly lowered TC and LDL-C. The 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 with other edible oils. This was formulated by the blending fractionated palm oil, cottonseed oil, and fully hydrogenated sunflower oil. The feeding of fat blends containing stearoyl triglyceride moieties may have been partially responsible for the changes observed in TC and LDL-C. By contrast, when Sundaram et al[27] 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 HDL-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 mixtures on blood lipids remains unclear. Palm fatty acids 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 cholesterol lowering effect of palm oil for consumption. Fortunately, several recent human studies have focussed on these issues and have provided additional observations that tend to support the Hedegred 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 tests are described below.

Hedegrad et al[30] 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 significant increase in TC which was primarily by a lower (11%) LDL-C concentration. Heber et al[31] evaluated diets enriched in palm oil, coconut oil or hydrogenated soybean oil for 3-week test periods in healthy middle-aged 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 study, coconut oil enriched diets did not affect cholesterol. 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 12:0+14:0 diet may be simply due to the lower availability of linoleic acid. This suggestion has been supported in the recent study of Sundaram et al[27] wherein, despite the incorporation of a high level of 18:2 (5.6% en) in the LM diet, it failed to cause significant increases in concentrations of TC and LDL-C in healthy volunteers compared to a 16:0-rich palm olein diet (3.5% en as 18:2).

The higher TC and LDL-C induced by the LM diets are in accordance with the data from the Keys-Lipid study where the Keys-Hedegrad equations[1] predict that identical TC concentrations would result from both fatty acids. However, it is argued that a simplification of the different dietary saturates effects in the Keys-Hedegrad equations tend to overestimate the importance of 16:0 and underestimate the impact of 12:0+14:0. The question that remains is how does the simplified combination of the different dietary saturates effects in the Keys-Hedegrad equations tend to overestimate the importance of 16:0 and underestimate the impact of 12:0+14:0. The question that remains is how does the simplified combination of the different dietary saturates effects in the Keys-Hedegrad equations tend to overestimate the importance of 16:0 and underestimate the impact of 12:0+14:0. The question that remains is...
Vervet monkeys and whole-food diets for studying the effects of dietary lipids on plasma lipoprotein metabolism and atherosclerosis

AJS Benadé DSc, JE Fincham, CM Smuts MSc, MJ Weight, PJ van Jaarsveld PhD, M Kruger

It is well established that some species of nonhuman primates are models of choice for polygenic hyperlipoproteinemia and atherosclerosis induced and promoted by diets as occur in man. The vervet monkey (Cercopithecus aethiops) has proved to be an excellent model for studying the effects of dietary lipids. The vervet monkey is highly motivated to consume food and may be used to study the effects of dietary fat intake on plasma lipoprotein metabolism and atherosclerosis.

Key words: African green monkey, dietary lipids, plasma lipoprotein metabolism, atherosclerosis

Introduction

It is well established that some species of non-human primates are models of choice for polygenic hyperlipoproteinemia and atherosclerosis induced and promoted by diets as occur in man. The vervet monkey (Cercopithecus aethiops) has proved to be an excellent model for studying the effects of dietary lipids. The vervet monkey is highly motivated to consume food and may be used to study the effects of dietary fat intake on plasma lipoprotein metabolism and atherosclerosis.

Materials and methods

Vervet monkeys were all healthy and conditioned to the laboratory environment for six months or more. Diets fed were either an average Western diet (WD), a pradein diet (PD) or a high carbohydrate diet (HCD), which have been described in detail elsewhere. The period of time diets were fed ranged from four to 47 months. Diets were composed entirely of normal foods without any added cholesterol and provided a realistic nutritional range.

Comparison of the effect of the amount and degree of unsaturation of dietary fat on plasma low density lipoprotein

Krugcr et al. studied the effects of the degree of unsaturation and of the amount of dietary fat on low density lipoprotein (LDL) concentration and composition in the African green vervet monkey (12 females, aged 15–4.5 years). Animals received diets with fat contents of 41, 31 and 18% energy each with a low and high polysaturated to saturated fatty acid ratio (P/S; 0.27–0.38 and 1.13–1.47; major fatty acids were palmitic and linoleic acids) for a period of two months. Cholesterol content of the diet was low (6.0–9.3 mg/100g). LDL cholesterol concentrations showed significant decreases when the dietary fat content was decreased from 31 to 18% of dietary energy. Dietary fat P/S ratio only affected LDL cholesterol concentrations during moderate (31% of energy) fat intake. Low density lipoprotein cholesterol increased with a decrease in dietary P/S. The changes in LDL cholesterol concentrations were the result of changes in the number of circulating LDL particles as the molecular composition of LDL did not significantly affect between dietary periods. Dietary fat changes had no influence on the high density lipoprotein cholesterol and plasma triglyceride concentrations. During the high P/S diets, the percentage of linoleic acid (18:2 w6) in LDL, esterified cholesterol (CE) and apoprotein triglycerided (TAG) increased as compared to the low P/S diets.

Results of this study provides evidence that the amount of dietary fat had a greater influence on plasma cholesterol concentration than a moderate change in dietary P/S in Vervet. The effects of dietary fat on plasma cholesterol were mainly through changes in LDL cholesterol concentrations. The animals showed marked individual differences in LDL cholesterol concentration response to both the amount and the degree of unsaturation of fat in the diet.

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