Trans. Nat. Acad. Science & Tech. (Phils.)
1988: 10: 371-414

IS COCONUT OIL HYPERCHOLESTEROLEMIC AND ANTHEROGENIC? A FOCUSED REVIEW OF THE LITERATURE

Quintin L. Kintanar
Executive Director
Philippine Council for Health Research & Development
Department of Science and Technology
Bicutan, Taguig, Metro Manila

and

Jose S. Castro
Senior Science Research Specialist
Philippine Council for Health Research & Development
Department of Science and Technology
Bicutan, Taguig, Metro Manila

ABSTRACT

One hundred nineteen articles, consisting of original scientific papers, review papers and citations, were reviewed. The papers were either "for" (Prosecution) or "against" (defense) the statement that coconut oil is hypercholesterolemic and atherogenic. Seventy-three percent of the papers is against the statement, while 27% is in agreement. Based on this, it is hoped that the scientific and medical communities both here and abroad will continue the scientific debate until a clear consensus will have emerged which can serve as a firm basis for future policies and actions on the use of coconut oil.

Introduction

The question raised in the title of this paper has become an urgent and extremely important issue, in the light of recent developments impugning the value and safety of coconut oil as an item of the diet. Coconut oil is a major contributor of calories to the Filipino diet (Florentino & Aguinaldo, 1987) ranging from a low of 3.91% in Central Visayas to a high of 10.2% in the Bicol Region (Kintanar, 1988). Any possible connection of dietary coconut oil to elevation of blood cholesterol level (hypercholesterolemic effect) and the consequent early or accelerated development of atherosclerosis or hardening and thickening of the arterial wall (atherogenic effect) which leads to premature cardio-vascular disease and death are, therefore, of major public health concern.

Atherosclerosis is a degenerative disease that is characterized by accumulation of lipids, primarily cholesterol, but also phospholipids and triglycerides in the walls of medium and large arteries. Clinically, it is associated with myocardial infarction or heart attacks and cerebral thrombosis and hemorrhage or strokes. In the Philippines, in 1984, diseases of the heart already ranked as the No. 2 leading cause of death at 61.0/100,000. An additional category: diseases of the vascular system – outside that of the heart – causes another 39.6/100,000 or a combined cardivascular mortality of 100.6/100,000. This ranks only second to respiratory disease mortality with pneumonia accounting for 89.3/100,000 and tuberculosis for 52.9/100,000 or a combined respiratory disease death of 142/100,000 (Philippine Health Statistics, 1984).

The situation in the U.S. as regards atherosclerosis-based death is even more alarming. Cardiovascular disease accounts for more deaths each year than all other causes combined. Of more than 1 million deaths in 1967, over 50% were due to atherosclerotic heart disease. The total national economic impact of premature atherosclerotic cardiovascular disease disease affecting those under 65 years was estimated at a cost of $17.8 billion for the year 1967 (Levy & Ernst, 1973).

This health concern is separate and apart from the potential adverse impact of this recent development questioning the safety of coconut as food on the Philippine coconut industry which supplies 50% of the world's production and earns about $460 million in exports annually. Of this export value, $347 million is accounted for by coconut oil, about half of which, or $174 million, comprises the edible products component (UCAP Coconut Statistics Annual, 1985).

Therefore, there is an urgent need to answer the question in an objective and scientific manner as possible.

Methodology

Since the issue is both complex and complicated and presents many grey areas where opinions and points of view differ, it is proposed that an approach and process analogous to that utilized by the judicial system through the courts of law in the settlement of controversies be followed. This paper attempts to contribute to this process by presenting both sides of the issue as published in the world literature, focusing particularly on coconut oil as distinguished from other saturated fats. It is hoped that the scientific and the medical communities both here and abroad will continue the scientific debate until a clear consensus will have emerged which can serve as a firm basis for future policies and actions.

The scientific evidence for the two sides will be presented in this paper in summation form, analogous to the final summation for the prosecution and for the defense in a court of law. The details of the evidence will be attached in tabulated form as annexes to substantiate the conclusions and summation for both sides under the following headings and formats:

Annex A. *Scientific Evidence for the "Proseuction," i.e., Coconut oil is hypercholesterolemic and atherogenic.*

A.1 Original Scientific Papers

A.1.1 Animal Experiments

Author(s)	Year	Animal Species	No.	Treatment	Results	Remarks

A.1.2 Human Clinical Studies

Author(s)	Year	No.	Treatment	Results	Remarks

A.1.3 Human Epidemiological Studies

Author(s)	Year	Population	Parameter	Findings	Remarks

A.2 Review Papers and Policy Guidelines

Author(s)	Year	Conclusions	Remarks

A.3 Papers Cited by Authors of Original or Review Papers

Author(s)	Year	Finding/Conclusion Cited	Remarks

Annex B. *Scientific Evidence for the "Defense," i.e., Coconut oil is neither hypercholesterolemic nor atherogenic. The information included and format for Annex B will be the same as in Annex A under the headings below.*

B.1 Original Scientific Papers

B.1.1 Animal Experiments
B.1.2 Human Clinical Studies
B.1.3 Human Epidemiological Studies

B.2 Review Papers and Policy Guidelines

B.3 Papers Cited by Authors of Original or Review Papers

Summations

A. For the "Prosecution" i.e., Coconut oil is hypercholesterolemic and atherogenic.

Animal Experiments

1. In several species of animals, including rabbits (Wigand 1959, Kritchevsky *et al.*, 1976 and 1982, Vles *et al.*, 1964, Paletta *et al.*, 1980, Stange and Papenberg 1974) rats (Hostmark *et al.*, 1980) dogs (Grande and Prigge 1974, Malmros *et al.*, 1968, McCullogh *et al.*, 1976), swine (Hill *et al.*, 1971) gerbils (Nicolasi *et al.*, 1981) monkeys (Kritchevsky 1969, Wissler *et al.*, 1962, Nicolosi *et al.*, 1977), feeding diets high in coconut oil, with or without cholesterol, produced elevated cholesterol levels and/or atherosclerosis.

Human Clinical Studies

2. In The Proceedings of the 1968 Deuel Conference on Lipids (Cowill *et al.*, 1969), it was reported that during coconut oil diet, plasma cholesterol increased from 160 to 280 mg./100 mL., but cholesterol level returned to normal when trilinoleic was given.

3. In two patients, one with xanthelasma and diabetes and another with myocardial infarction, supplemental feeding with coconut oil formula, which was rather unphysiologic, induced an increase in serum cholesterol. (Kinsell, 1958)

4. Grande *et al,* in 1961, studying 27 schizophrenic but physically healthy subjects in a cross-over experiment showed an increase in cholesterol level in the group that received 56% of its fat as coconut oil which was minimal, 196.4 +– 27.7 versus 187.4 +– 27.9, although considered significant by the authors.

5. Compared to corn oil, coconut oil diet was associated with significantly higher serum cholesterol, Apo E and triglyceride level (Fisher *et al.*, 1983).

6. In 3 out of 4 patients, feeding of synthetic or commercial coconut oil at 40% of total calories increased plasma cholesterol. (Spritz and Ahrens, 1965).

Human Epidemiologic Studies

There are no reports or studies which show that populations with a high intake of coconut oil in their diet have elevated blood cholesterol levels or an increased incidence and prevalence of cardiovascular disease or death.

B. For the "defense" – i.e., Coconut oil is not hypercholesterolemic and atherogenic.

Animal Experiments

1. In rats (Hostmark *et al.*, 1980) total serum cholesterol with coconut oil or sunflower did not differ significantly although coconut increased serum triglyceride level.

2. In rats, the pathology, including predisposition to atherosclerosis, was been shown to be due to essential fatty acid deficiency (Deuel *et al.*, 1955, Morin *et al.*, 1964) reversible by supplementation with adequate linoleic acid. Therefore, in many of early adequate linoleic acid. Therefore, in many of early experiments using semipurified or purified diets of coconut oil or hydrogenated coconut oil where hypercholesterolemia and atherosclerosis were demonstrated, the pathology was complicated or caused by essential fatty acid deficiency and not coconut oil *per se.*

3. In rabbits fed lard or coconut oil at 26% of total calories, serum cholesterol was higher in lard although it was also elevated in the coconut group. Only one showed gross atheromatous plaques in the lard group plaques and none in the coconut group (Santos *et al.*, 1983).

4. In dogs, feeding with Hydrogenated coconut oil (HCNO) comprising 16% of diet and five percent supplemental cholesterol caused severe atherosclerosis. In another fed the same diet except for the substitution of four percent of 16% of the diet with safflower oil for HNCO, the atherogenic effect was not demonstrted (McCullogh *et al.*, 1976). The authors concluded that the atherogenic effect was due to essential fatty acid deficiency and not to coconut oil.

5. In pigs fed tallow or coconut oil with hog bile, the tallow group had twice as high serum cholesterol and more severe atheroclerosis than the coconut group. (Hill *et al.*, 1971).

Coconut oil did not block the beneficial effects of marine menhaden oil which was not the case with the tallow group.

Human Clinical Studies

6. Using physiologic diets with different fat sources namely coconut oil, corn oil and safflower oil in salad dressing, mayonnaise or cooking/frying oil, corn and safflower depressed serum cholesterol. Coconut oil was not hypercholesterolemic nor did it block the hypocholesterolemic effect nor did it block the hypocholesterolemic effect of corn or safflower oils fed as a third of fat calories. (Malmros and wigand, 1957).

7. In clinical studies using physiologic diets containing 40% fat calories from saturated and polyunsaturated fatty acids, the steady state cholesterol level in the coconut oil group was found to be 224 +— 8.4 mg./100 mL. while cholesterol levels on house diet were 232 and 225 mg/100 mL. The butterfat group had mean levels of 241, 235 and 232 mg./100 mL. which appears not significantly different from the coconut oil mean. Using these results, Keys *et al.*, (1957) developed a regression equation predicting changes in the serum cholesterol from changes in the percentage of calories as polyunsaturated and saturated fats. The authors remarked that their equation was quite accurate in predicting the cholesterol level except in the case of corn oil or hydrogenated coconut oil.

8. In twelve healthy male subjects, half was given corn oil supplement and the other, HCNO on top of an American type meal. After one month, the serum

and liver cholesterol did not change significantly with the coconut oil supplementation but decreased with the corn oil supplementations (Frantz and Carey, 1961).

9. Ten hypercholesterolemic patients received either safflower alone or an equicaloric equivalent of a 50-50 mixture of safflower and coconut oil. Both safflower and coconut oil mixture reduced the hypercholesterolemic level to 268 mg./100 mL. and 256 mg./100 mL., respectively (Hashim *et al.*, 1959).

10. In twelve hypercholesterolemic subjects fed a diet composed of 80 g. coconut oil, five percent fat as sunflower and five percent as olive oil (used as part of a physiologic diet), it was reported that from a baseline of a range of 170 to 370 mg/100 mL., the supplemented coconut oil diet caused the range to fall afer 10 weeks to 140-240 mg/100 mL. Removal from the diet back to usual resulted in an increase to 170-320 mg./100 mL. Resumption of the coconut supplemented with sunflower and olive oil again decreased cholesterol levels to 150-260 mg./100 mL. A switch to pure 100% coconut oil resulted in a level of 170-270 mg./100 mL.; but all the time that the subjects were on coconut oil supplemented or pure diet, the cholesterol levels were always lower than the baseline range. (Halden and Lieb, 1961)

11. In a 5-year study of 100 young men with coronary heart disease showing ECG evidence of myocardial infarction, it was shown that reducing fat calories to 28% of total calories either by using Diet 1: 50-50 mixture of corn and safflower oil or Diet 2: 50-50 mixture of coconut oil and peanut oil resulted in significant reduction in serum cholesterol after 1 year and 5 years on either diet. The polyunsaturated to saturated fat ratio was 3:1 with Diet 1 and the reverse 1:3 with Diet 2. The Keys equation did not predict the levels of cholesterol. Coconut oil did not cause increase in atherosclerotic heart disease or death. (Bierenbaum *et al.*, 1967).

12. In a human clinical study involving three subjects, coconut oil was found to have less cholesterol lowering effect than corn oil but the data do not show that coconut oil is hypercholesterolemic compared to baseline. In fact, in the only case where baseline cholesterol on regular diet was given, this level (450 mg/100 mL.) was higher by 80 mg./100 mL. over that during the coconut oil feeding (Ahrens *et al.*, 1957).

13. In Filipino medical students, adding supplemental coconut oil in the diet to raise fat calories to 30% and 40% of total calories did not increase serum cholesterol levels significantly over controls given 20% of calories as fat (Icayan *et al.*, 1964).

Human Epidemiological Studies

14. In human epidemiologic studies, two populations in Polynesian atolls consuming 63% and 34% of total energy from coconut had satisfactory levels of serum cholesterol 68-82 mg./100 mL. lower than predicted levels using Keys equation, i.e., the predicted values were 40% over the actual values obtained. (Prior *et. al.*, 1981).

15. Studing another two population of the same broad ethnic type (Pukapukans and Rarotogans) from Cook Islands, Prior, in 1974, found that Pukapukans

who derived 75% of fat in the diet from coconut had cholesterol level of 155-195 mg./100 mL. which was 20 mg./100 mL. lower than Rarotongans who derived 25% from coconut.

16. Serum cholesterol levels of population in the Bicol region consuming the highest amounts of dietary coconut oil among Filipinos – as much as 10.2% of total calories (Kintanar, 1988) – have been reported to be in the normal range (less than 200 mg./100 mL. mean) (Castillo, 1982, Camara-Besa *et. al.*, 1974). The Region ranks seventh out of 10 regions surveyed in the prevalence of cardiovascular mortality (Florentino and Aquinaldo, 1987).

17. The human epidemiologic studies which served as bases for the recommendation that saturated fat intake of hyperlipidemic patients should not exceed 10% of total calories were on populations consuming no or insignificant coconut oil calories in the diet (Keys, 1957 and 1980).

Annex A. Scientific Evidence for the "Prosecution," i.e. Coconut oil is hypercholesterolemic and atherogenic.

A.1. Original Scientific Papers

A.1.1 Animal Experiments

Author(s)	*Year*	*Animal Species*	*Number*	*Treatment*	*Results*	*Remarks*
Malmros, et al.	1968	Dogs	22	Fed with semi-synthetic diet containing 5% cholesterol and hydrogenated coconut oil (17%).	Cholesterol went up to 500 mg% from control level of 200.	Source of essential fatty acids not indicated.
Vesselinovitch, et al.	1980	Rhesus Monkeys	18	Fed with 3 types of diet, containing 25% of fat: (a) butterfat, coconut oil + cholesterol (2%) (b) cholesterol-free with corn oil, (c) cholesterol (2%) + peanut oil.	All diets produced hyperlipemia and hyperlipoproteinemia. Highest level of serum cholesterol was found in diet (a).	Severity of atherosclerosis was greatest with peanut oil and cholesterol.
Kritchevsky, D.	1969	Rats (hypophysectomized or with deficiency in essential fatty acids)	NNM	Fed with cholesterol or hydrogenated coconut oil.	Development of coronary atherosclerosis.	Coconut oil is more cholesterolemic than corn oil.
Wissler, R.W.	1962	Monkeys (Cebus)	12	Fed with synthetic diets containing 25% food fats of	Microscopically, all of the coconut-oil fed monkeys and 3 of	The results indicate that there are defi-

Annex A (Continued)

Author(s)	*Year*	*Animal Species*	*Number*	*Treatment*	*Results*	*Remarks*
				varying fatty acid composition (butter-fat, coconut oil and corn oil).	the 4 butter-fat-fed animals revealed aortic deposition – none in corn-oil-fed monkeys.	nite diet-related trends in the lipid fractions of the serum, liver and aorta.
Kritchevsky, D.	1969	Monkeys (New World)	297 (Three species)	Feeding with coconut oil, corn oil, corn + cholesterol, and coconut + cholesterol.	Coconut oil is more cholesterolemic than corn oil.	
Young, S.J. and Renner, R.	1977	Chicks	160	Coconut oil was compared to soybean oil in terms of their ketogenicity.	Coconut oil produced relative reduction in appetite and growth.	MCT in coconut oil have been show to resemble carbohydrates in some respects in both rats and humans.
Ehrhart, et al.	1971	Dogs, adult male mongrel	34	Fed with 5% cholesterol and 16% hydrogenated coconut oil.	Radioactivity in lipids increased by 50% compared to control.	Leukocyte fatty acid synthesis from acetate occurs predominantly by the chain elongation pathway.
Hill, et al.	1971	Swine	33	Four groups of animals were fed with either tallow or co-	All groups had elevated serum cholesterol. The coconut oil group deve-	

Annex A (Continued)

Author(s)	*Year*	*Animal Species*	*Number*	*Treatment*	*Results*	*Remarks*
				conut oil diet with or without menhaden oil.	loped less atherosclerosis than the tallow group. Menhaden oil did not appear to make any difference.	
Divakaran, P. et al.	1977	Rats, wealing male albino	20	Two groups were fed with either safflower seed oil or hydrogenated coconut oil, at 20% levels.	Oxidation phosphorylation for glutamate and malate was higher in the safflower oil group compared to coocnut oil, but not for succinate.	Fatty acid composition of mitochondrial lipids varied with dietary fat source.
Nicolosi, R.J. et al.	1981	Mongolian Gerbils	48	Fed with either coconut oil or safflower oil.	Coconut oil-induced hypercholesterolemia was associated with increases in VLDL, LDL and HDL.	Lipoprotein composition and size were not influenced by the type of dietary fat.
Kritchevsky, et al.	1976	Rabbits, dutch belted	56	Four groups of the animals were fed a diet containing 40% sucrose, 25% casein, and 14% fat for 10 months. The fats were in the form of peanut oil, corn oil, butter oil and coconut oil.	Cholesterol levels that resulted were the following: mgm/dl Peanut = 205 Corn = 152 Butter = 292 H. Coconut oil = 330	Semi-purified diet without essential fatty acids was used. (without added cholesterol)

Annex A (Continued)

Author(s)	*Year*	*Animal Species*	*Number*	*Treatment*	*Results*	*Remarks*
					Percentages of surface area affected with aortic lesions were the following: Peanut = 10% Corn = 2% Butter = 14% Coconut oil = 25%	
Labadan, et al.	1962	Chicks broiler	630	Coconut oil was added to the basal ration.	Concentration of serum cholesterol increased with the addition of both kinds of coconut oil. Cholesterol tended to increase as the levels of coconut oil increased from 5 to 15% for both kinds of oil (crude and Philcoa-Hiller coconut oil).	(1) Refer to basal mixture to determine the presence of essential fatty acids.
Wissler, et al.	1962	Monkeys	12	Four monkeys in a group were fed 25% corn oil, coconut oil or butter fat	The animals developed elevated serum-cholesterol with coconut group having the highest and corn oil the lowest levels. Gross or mi-	Basal diet did not contain essential fatty acid.

Annex A (Continued)

Author(s)	*Year*	*Animal Species*	*Number*	*Treatment*	*Results*	*Remarks*
					croscopic fatty lesions in the aorta were found in 3 out of four monkeys in butterfat group, all 4 animals in the coconut oil group and none in the corn oil group.	
Paletta, et al.	1980	Rabbits, New Zealand	12	Cholesterol free, semisynthetic diet containing hydrogenated coconut oil (13.21 g.) and soybean oil (1.48 g.), among others, was given.	Elevation of serum cholesterol associated with increased hepatic cholesterol synthesis was observed.	
Stange and Papenberg	1974	Rabbits	NNM	Four groups of rabbits were fed the following diets: a. I = control diet b. II = diet with 1% cholesterol c. III = diet with !% cholesterol +% coconut oil	Structural anomalies in LDL and VLDL (ellipsoidal or square branching tendencies, maximum d=200-360 A) observed in both groups II & III can probably be related to severe atheromatosis, confirmed by finding of similar alterations in the LDL of patients with type-II a hyperlipopro-	

Annex A (Continued)

Author(s)	*Year*	*Animal Species*	*Number*	*Treatment*	*Results*	*Remarks*
				d. IV = diet with 1% cholesterol + 5% corn oil	teinemia, which is accompanied by accelerated atherosclerosis. Group IV had less extensive atheromatosis.	
Hostmark, A., et al.	1980	Rats, Male Wistar	18	Fed purified diet containing either 10% coconut oil or sunflower oil.	Cholesterol increased from 100 mg.% to 170 mg.% while triglyceride increased from 100 to 360 with coconut oil. With sunflower oil, it increased to 140 only for plasma triglyceride.	Output of VLDL, cholesterol and triglyceride from perfused livers were normal and similar in both groups.
Wigand, G.	1959	Rabbits, male	88	Hydrogenated coconut oil was fed to rabbits at 26% of the calories.	It produced the highest hyperlipidemia.	1. Basal diet was deficient in essential fatty acids (.006% only) 2. Groups not observed in the same year. 3. Evidence for essential fatty acids deficiency was the return of cholesterol

Annex A (Continued)

Author(s)	*Year*	*Animal Species*	*Number*	*Treatment*	*Results*	*Remarks*
						to normal when coconut oil was changed with corn oil.
Grande, et. al.	1974	Dogs	12	Animals were fed with coconut oil instead of sucrose or protein.	Compared with low fat diet, coconut oil diets caused significant elevation of cholesterol and phospholipid levels.	Essential fatty acid content of basal diet was 3.6%.
Kritchevsky, et al.	1982	Rabbits, New Zealand White	48	Corn oil, palm kernel oil, cocoa butter and coconut oil at about 32.6% of calories were fed to rabbits.	They all increased cholesterol from 50 to 400 except for corn oil. HDL cholesterol was redued.	The animals were fed a semi-purified, cholesterol-free atherogenic diet.

A.1.2 Human Clinical Studies

Author(s)	*Year*	*Number*	*Treatment*	*Results*	*Remarks*
Ahrens, et al.	1957	40	In three succeeding periods, the subjects derived their dietary fat from corn oil, coconut oil, and corn oil, respectively.	Cholesterol level, starting at 263 with corn oil, went up to 367 under coconut oil. It returned to 286 on corn oil.	Coconut oil was not supplemented with any essential fatty acids during the coconut oil period.

Annex A (Continued)

Author(s)	*Year*	*Number*	*Treatment*	*Results*	*Remarks*
			+1 gm. day cholesterol, (3) coconut oil, (4) coconut oil +1 gm. day cholesterol.	with significant elevations in cholesterol, apo E and triglycerides.	specific metabolic pathways for cholesterol saturated fats, or plasma lipoproteins.

A.1.3 Human Expidemiological Studies

NO REPORT OR STUDIES

A.2 Review Papers and Policy Guidelines

A.2.1 Animal Experiments

Author(s)	*Year*	*Conclusions*	*Remarks*
Ehrhart, et al.	1973	Dog leukocyte fed atherogenic diet, 5% cholesterol and 16% hydrogenated coconut oil, was noted to have 50% more radioactivity from AC 1-14C in all lipids.	The increase was not specific for any particular lipid.
McGovern, R.F.	1973	Neither absorption of cholesterol from gut nor secretion of B hydroxy sterol into gut accounts for hypercholesterolemic effect of polyunsaturated fatty acids.	Hypercholesterolemic rats were used.

Annex A (Continued)

A.3. Papers Cited by Authors of Original or Original or Review Papers

A.3.1 Animal Experiments

Author(s)	*Year*	*Finding/Conclusion Cited*	*Remarks*
Lambert, G.F. et al.	1958	Atherosclerosis can be induced in rabbits fed with cholesterol-free, semi-synthetic diet containing saturated fat.	
Stormby, N.G. and N. Wigand	1963	Feeding rats with coconut oil without adequate linoleic acid supplementation induced linoleic acid deficiency lesions.	The production of the lesions was interpreted by the authors as caused by saturated fats.

A.3.2 Human Clinical Studies

Grundy, S.M.	1987	Substituting monounsaturated fatty acid for saturated fatty acid lowered LDL cholesterol as effectively as low fat diet.	

A.3.3 Human Epidemiological Studies

NO CITATIONS AVAILABLE

Annex B. Scientific Evidence for the "Defense," i.e. Coconut oil is neither hypercholesterolemic nor atherogenic.

B.1 Original Scientific Papers

B.1.1 Animal Experiments

Author(s)	*Year*	*Animal*	*Number*	*Treatment*	*Result(s)*	*Remarks*
Deuel, et al.	1955	Rats, weanling	206	Saturated coconut oil (30%) was added to the fat-free diets of rats.	There was a depletion of essential fatty acid in a shorter time than when the rats were fed with fat-free diet alone. The continued feeding of hydrogenated coconut oil did not inhibit the response of the animals to linoleic. After an initial decrease in plasma cholesterol from 70 to 50 mg./100, the cholesterol returned to normal even with continued coconut oil feeding.	
Babayan, V. et. al.	1958	Rats, mother	NNM	Feeding with saturated medium chain triglycerides (MCT) 20%, without essential fatty acids.	Essential fatty acid deficiency was minimized by MCT.	
Kaunitz, H. et al.	1960	Rats, linoleic acid-depleted	16	Fed with purified diets containing no fat or containing 20% saturated LCT or MCT.	Neutral fat deposition was higher with LCT than with MCT or FF.	

Annex B (Continued)

Author(s)	*Year*	*Animal*	*Number*	*Treatment*	*Result(s)*	*Remarks*
Castillo, L.S. et al.	1962	Rabbits, New Zealand	60	Rabbits were fed with either crude coconut oil or Philcoa-Hiller coconut oil.	Increase in digestibility of crude protein was noted; there was decrease in the digestibility of crude fiber.	
Morin, et al.	1964	Rats, EFA-deficient	42	Rats were fed initially with either; (1) fat free diet, or (2) stock diet. After 16 weeks, the diet was added with 20% HCNO or none at all.	Coronary atheromas were produced in essential fatty acid deficient rats fed 20% hydrogenated coconut oil for 10 weeks. The rats given stock diet containing adequate amounts of essential fatty acids and 20% HCO did not develop atheromas.	EFA-deficiency may lead to defective cholesterol transport machanism.
Vles, et al.	1964	Rabbits, Viennese, atherosclerotic	72	Three types of diet were used; (a) low-fat, (b) soyabean oil, (c) coconut oil, all without cholesterol.	Diet (a) and (b) produced rapid fall in cholesterol levels within 5 weeks – 80% reduction. In the group with diet (c), a fall in cholesterol level was noted, but only 50% of original level.	At the end of the preliminary period, when cholesterol-containing food was administered, the mean serum cholesterol rose to 1600 mg./100 mL.

Annex B (Continued)

Author(s)	*Year*	*Animal*	*Number*	*Treatment*	*Result(s)*	*Remarks*
Kaunitz, H. et al.	1970	Rats	727	Different groups of the animals were fed with: 1. coconut oil; 2. beef fat; 3. MCT, 4. lard; 5. corn oil 6. butter; 7. soybean oil; 8. chicken fat, 9. olive oil; 10. cottonseed oil. seed oil.	Mortality in the group fed with coconut oil was 8%, compared to 10% for soybean oil and 19% for cottonseed oil. The mortality rate in the coconut oil group was reduced when HCNO was supplemented with linoleate at 1.5% of the diet.	
Wignjosoesastro, N. et al.	1972	Hens, leghorn	240	Fed wth coconut oil.	Increase in the rate of production and efficiency of egg-laying hens was noted. Feeding with coconut oil also resulted in weight gain.	
McGregor, Lilian	1974	Rats, Sprague-Dawley	86	Feeding with either HCNO or maize oil	Coconut oil produced significantly slower clotting than corn oil in essential fatty acid deficient rats.	
Miranda, et al.	1974	Rats, white	60	Coconut oil and corn oil, on various mixtures of the two, incorporated in nutritionally adequate diet, were fed to rats.	The diet produced normal growth, serum cholesterol and liver morphology. However, the group given coconut oil only had a higher liver fat.	

Annex B (Continued)

Author(s)	*Year*	*Animal*	*Number*	*Treatment*	*Result(s)*	*Remarks*
McCullagh, et al.	1976	Dogs	16	Semisynthetic diet containing 5% cholesterol and 16 hydrogenated coconut oil was fed to the animals for 12 to 14 months.	Occlusive atheromatous plaques were seen in 7 out of 8 dogs. Addition of 4% safflower oil completely protected the dogs from the atherogenic process.	The addition of linoleic acid (8%) to the diet prevented hypercholesterolemia and atherosclerosis.
Nicolosi, et al.	1977	Squirrel and cebus monkeys	12	Fed semipurified diets with either coconut oil or corn oil.	They developed comparable hypercholesterolemia, but the squirrel primarily expands the CE and FC of its LDL2 cholesterol fraction, whereas the cebus monkeys primarily increased its HDL fraction of cholesterol.	These results, coupled with the greater accumulation of aortic lipid, particularly cholesteryl ester, in the atherosclerotic-susceptible squirrel monkeys, support the concept of the protective nature of HDL and the atheroganic potential of LDL. They also suggest that a species' genetic control of the lipoprotein response to diet is variable.
Privett, O.S. et al.	1977	Rats,	42	Diets containing mix-	Addition of trans fatty acids	

Annex B (Continued)

Author(s)	*Year*	*Animal*	*Number*	*Treatment*	*Result(s)*	*Remarks*
		weanling male		tures of safflower and HCNO with elaidate or linolelaidate were fed on rats.	lowers the growth response to linoleic acid. Both elaidate and linolelaidate accumulate in the serum and liver, impair the conversion of oleic acid to eicosantrienoic acid and linoleic acid to arachidonic acid, and the incorporation of eicosantrienoic acid into cholesteryl esters. Serum lecithin: cholesterol acyl transferase activity was elevated by an essential fatty acid deficiency, was unaffected by dietary elaidate, but was significantly decreased by linolelaidate. These effects were nullified by the addition of safflower oil to the diet. Postherparin plasma extrahepatic and hepatic lipase activities were also affected by an essential fatty acid deficiency, and by addition of elaidate or linolelaidate alone or in combination with safflower oil to the diets of essential fatty acid deficient rats. It is suggested that trans fatty acids exhibit particular	

Annex B (Continued)

Author(s)	*Year*	*Animal*	*Number*	*Treatment*	*Result(s)*	*Remarks*
					effects on the metabolism of lipids in addition to aggravation of an essential fatty acid deficiency.	
Aguilar and Florencio	1980	Rats, albino	60	Either coconut oil or corn oil was fed to rats.	Coconut oil and corn oil did not produce any differential effects on protein metabolism in rats.	
Paletta, S. et al.	1980	Rabbits, New Zealand	12	Cholesterol-free, semisynthetic diet was given.	Hypercholesterolemia was not due to a defect in removal of cholesterol from the plasma compartment, but to an increased biosynthesis.	
Chu, Shu-heh W. et al	1980	Rats	16	Infusion of saturated fats of different chain lengths was done.	Fatty acid with chain length less than 14 were not significantly incorporated into lymph phospholipid and cholesterol ester.	Feeding HCNO produced essential fatty acid deficiency.
Santos, Kintanar, Q.L. et al.	1983	Rabbits, male	30	Coconut oil, or lard, at 24% of total calories was given to the rabbits.	Neither diet produced atherosclerosis in rabbits.	One rabbit in the lard group developed 20% atheromatous plaque.
Mok, King Tong, et al.	1984	Rats, Spraque-	32	Structured medium chain and long chain	The emulsion of MCT and LCT was superior to	

Annex B (Continued)

Author(s)	*Year*	*Animal*	*Number*	*Treatment*	*Result(s)*	*Remarks*
		Dawley		triglycerides were fed to burned rats.	physical mixture of same substances in sparing body protein.	
Maiz, A. et al.	1984	Rats, injured	35	Dextrose, soybean oil lipid emulsion (LCT), MCT and structured lipid in the amount of 100 calories/kg. body wt./day were given and compared.	Compared to dextrose or soybean oil emulsion, supplementation of I.V. diet with structured lipid emulsion, containing safflower oil and medium chain triglyceride, decreases leucine oxidation and improves protein utilization.	Structural lipid emulsion contains sufficient amounts of EFA.
Wissler, Robert W. et al.	1985	Monkeys, Rhesus, adult male	48	Fed semisynthetic rations containing cholesterol with varying amounts of coconut oil, peanut oil and soybean oil.	Coconut oil elevated LDL cholesterol but less than peanut and soybean oil.	Coconut oil was the only one which elevated HDL and Apo A-I.
Hamawy, K.J. et al.	1985	Rats, Sprague-Dawley, males	56	Parenteral nutrition formula, where long chain triglyceride was partially replaced with MCT, was used, among others.	The formula supported host bactericidal capacity better.	The findings demonstrate that the response by a septic organism to various nutritional regimens depends on the presence

Annex B (Continued)

Author(s)	*Year*	*Animal*	*Number*	*Treatment*	*Result(s)*	*Remarks*
						and type of lipid emulsion administered.
Pomposelli, J.J. et al.	1986	Rats, Sprague-Dawley, males	14	Mixed fuel regimen containing MCT and LCT was given.	The regimen prevented abnomal morphologic changes in the liver.	This suggests that MCT may provide an additional benefit in patients with liver dysfunction.
Jandacek, R.J. et al.	1987	Rats, Sprague-Dawley, males	NNM	Two mL. of an emulsion containing 2-[1 14 C] linoleoyl-1,3, dioctanoyl glycerol was injected to an isolated loop of small intestine.	In vitro, the pancreatic lipase of rat hydrolyzed 1,3 positions preferentially to that of the 2 positions, using the structured lipid.	It provided the advantages of rapid absorption and oxidation of MCT without producing essential fatty acid deficiency.
Wisller, R.W. et al.	1985	Monkeys, Rhesus, adult male	48	The animals were fed with either cholesterol + peanuts, or cholesterol + corn oil or cholesterol + coconut oil.	Cholesterol + peanuts produced severe atheromata than the other diets. Monkeys fed peanut oil had the lowest serum cholesterol levels, which is consistent with report (Emerson) that saturated fat is more cholesterolemic than unsaturated fat.	

Annex B (Continued)

Author(s)	*Year*	*Number*	*Treatment*	*Result(s)*	*Remarks*
B.1.2 Human Clinical Studies					
Keys, Ancel	1957	84	Controlled experiments were done covering butter-fat, lard, olive oil, cotton-seed oil, corn oil, sunflower-seed oil, safflower oil, sardine oil, and HCNO.	The experiments formed the basic of Keys' equation: Cholesterol = 2.74 (ΔS) − 1.31 (ΔP)	The results do not support the suggestion that deficiency of EFA produces high serum-cholesterol levels.
Malmros, et al.	1957	Eight healthy volunteers	The volunteers were given hydrogenated coconut oil from free diet for 4 weeks at 40% of calories, followed by corn oil.	Serum-cholesterol persisted at the its original levels during the weeks that HCON was given. However, when given corn oil, levels decreased from 220 to 180 mg./100 ml', as well as when a combination of HCNO and corn oil was given.	
Keys', A. et al.	1957	NNM	Diet adequate and constant in proteins, vitamins, and minerals in which total fats provide 9.5-41.5% of total calories was given.	Keys' equation: cholesterol = 2.74 (ΔS) − 1.31 (ΔP)	The equation overestimates cholesterol values on sardine-oil comparison by 0.2 mg/100 mL which may be due to the short chain fatty acid in coconut oil.

Annex B (Continued)

Author(s)	*Year*	*Number*	*Treatment*	*Result(s)*	*Remarks*
Kinsell, et al.	1958	4	Vegetable oil was used.	Linoleic acid in various vegetables oils was responsible for lowering plasma cholesterol.	Linoleic acid probably regulates the production of choles terol in the body.
Hashim, et al.	1959	10	An equal mixture of safflower and CO (representing 40% of total calories) was fed to hypercholesterolemic humans, compared with safflower oil alone.	The mixture of oils reduced the serum cholesterol more than safflower oil alone.	This suggests an investigation of the potency of mixtures of oils in man.
Kaunitz, H. et al.	1959	10	Two thousand (2000) calorie diet with MCT as the only fat was given.	Serum cholesterol declined 20-80 mg. % from the original of over 250.	
Hashim, et al.	1960	8	Corn oil, MCT and butter were used as source of fat.	Compared with corn oil, MCT induced a slight rise in serum-total-cholesterol; but compared with butter, the serum-lipids increased appreciably when butter followed MCT, and decreased appreciably when MCT followed butter.	The cholesterol-lowering effect of MCT cannot be explained in terms of the content of linoleic acid or other polyunsaturated fatty acids.

Annex B (Continued)

Author(s)	*Year*	*Number*	*Treatment*	*Result(s)*	*Remarks*
Halden, V.W. et al.	1961	10	Coconut oil with sunflower (5%) and olive oil (5%) was used in place of other fats.	The human volunteers showed an initial high levels of cholesterol (250 mg.%) with their normal diet. When other fats were substituted with CO with sunflower and olive oil, cholesterol dropped to 200 mg. %.	Diet with pure coconut oil led to increase in cholesterol levels, but still lower than original values before the beginning of the experiments.
Frantz, Ivan V. and J.B. Carey	1961	12 healthy males	Three (3) ounces of either corn oil or HCNO was daily added to the American diet.	The diet produced insignificant changes in serum cholesterol and liver cholesterol after one month of feeding.	Fall in serum cholesterol produced by corn oil feeding in man is probably not due to a shift of cholesterol from the blood to the liver.
Icayan, E.E. et al.	1964	10	Different levels of coconut oil were fed to normal medical students.	Although there was an observed increase in serum cholesterol in the experimental groups by 20 mg% & 25 mg% versus 2 mg% and 9 mg% for the control, difference was not significant.	Results are consistent with the hypothesis that serum cholesterol is related to the saturated fat intake.
Erickson, B.A. et al.	1964	42	Forty two healthy men were given fat diets	Addition of cholesterol increased serum choles-	The results disprove the concept that

Annex B (Continued)

Author(s)	*Year*	*Number*	*Treatment*	*Result(s)*	*Remarks*
			of various P/S ratios.	terol. With cholesterol free formula, plasma cholesterol level is unaffected by variation in the P/S ratio from 1.6 to 0.1.	blood cholesterol is a function of the P/S ratio of the dietary fat.
Haberland, et al.	1987	NNM			Altered LDL may induce the secretion of neutral proteases, plasminogen-activating factor and tumor cytolytic factors by macrophages, initiating the atherosclerotic reaction.
Mascioli, et al.	1987	NNM	Intravenous MCT as a 75% MCT/25% LCT physical mixture was tested on hospitalized patients.	MCT are hydrolyzed and cleared rapidly, more readily oxidizable fuel, with less interference of the reticuloendothelial component of the immune system.	
Holman, R.T. et al.	1957	13	Dietary fat was given.	Feeding of hydrogenated coconut oil was accompanied by decrease in plasma cholesterol.	Essential fatty acids may be necessary for normal transport of saturated fatty acids and/or cholesterol.

Annex B (Continued)

Author(s)	*Year*	*Number*	*Treatment*	*Result(s)*	*Remarks*
Krauss, et al.	1987	NNM			IDL and denser LDL-III A may be associated with coronary disease.
Miller, N	1987	104	HDL2 and HDL3 cholesterol were measured in patients with chest pain.	It appears that low HDL cholesterol levels in many men with clinically significant coronary disease reflect reduction of both HDL2 and HDL3.	Low HDL in IHD is accompanied by a shift in the frequency distribution of HDL particles to a higher density.

B.1.3 Human Epidemiological Studies

Author(s)	*Year*	*Population*	*Parameter*	*Finding(s)*	*Remark(s)*
Keys, et al.	1970	Seven (7) countries 12,770	Weighing all items consumed during 7 days of the survey and were repeated in different seasons.	(1) Deitary intake of satuated fat and cholaterol play an important role in determining the average level of lipids and cholesterol in 7 countries. (2) Saturated fatty acids with 12 or more but less than 18 carbon atoms in the chain raise the cholesterol level while the major	In none of seven countries was coconut oil a major component of the diet.

Annex B (Continued)

Author(s)	*Year*	*Population*	*Parameter*	*Findings*	*Remark(s)*
				polyunsaturated fatty acids have a weaker opposite effect.	
Prior, I.A.M.	1974	Rarotongans versus Pukapukans	Rarotongas consume 27% calories from fat and 7% from sucrose; Pukapukans consume 35% of calories from fat and 2% from sucrose	Rarotongans had a 20 mg./100 mL. higher level of mean cholesterol levels despite the very low coconut intake.	Seventy five percent of fat in the Pukapukan diet was from the highly saturated coconut fat, while Rarotongans had less than 25%.
Prior, I.A.M. et al.	1981	Tokelauans		Major differences in serum cholesterol levels were attributed to higher saturated fat intake of Tokelauans. Harmful effects of highly saturated fat intake in these populations were not proven.	
Prior, et al.	1981	Two (2) Polynesian populations, living in two different islands.	Tokelauans: – consume over 50% of total calories in the form of fat, and – 63% of energy was derived from coconut. Pukapukans: – consume 35% of	The serum cholesterol level of Tokelauans are 35-40 mg./100 mL. higher than Pukapukans.	However, vascular disease is uncommon in both populations.

Annex B (Continued)

Author(s)	*Year*	*Population*	*Parameter*	*Findings*	*Remark(s)*
			total calories in the form of fat, and 34% of energy was derived from coconut.		
Castillo, Emma del	1982	Rural Bicolano population	Effect of coconut milk/oil consumption on blood cholesterol levels was determined.	Population frequently consuming vegetables with coconut milk showed normal cholesterol levels. Compared with meat, fish and eggs consumed at the same frequency per week, coconut milk consumption gave the lowest means of cholesterol values.	

B.2 Review Papers and Policy Guidelines

B.2.1 Animal Experiments

Author(s)	*Year*	*Conclusion(s)*	*Remark(s)*
Gerpacio, A.L	1963	Swines given 10 and 15% coconut oil in diet had better feed conversion efficiencies and thicker backfat than control.	High sturated oils like coconut oil when fed to animals yield fat with higher degrees of saturation.

Annex B (Continued)

Author(s)	*Year*	*Conclusion(s)*	*Remark(s)*
		via the portal system, are not carnetine-dependent, and do not require chylomicron formation. MCT undergo portal system absorption, liver first before heart, unlike the LCT. MCT are absorbed and metabolized as rapidly as glucose, while having twice as much caloric density. They are easily oxidized and have little tendency to deposit as body fat. For this reason, MCT have been utilized for many years as oral supplementations and for enteral feeding formulas for infants, epileptic children, cystic fibrosis patients and patients who underwent intestinal resection.	
B.2.3 Other Reviews			
Ernst and Levy	1984	Hypercholesterolemia and hyperlipoproteinemia are considered a third major risk factor in addition to hypertension and dietary sodium which are modifiable by diet Hypercholesterolemia is also a sign of genetic and environmental disorders.	Hyperlipoproteinemia can now be managed effectively through diet.

NO REVIEWS NOTED

B.3 Papers Cited by Authors of Orignal or Review Papers

B.3.1 Animal Experiments

Author(s)	*Year*	*Finding/Conclusion Cited*	*Remark(s)*
Glavin, J., et al.	1952	Lipid peroxides were found in atherosclerotic blocks.	

Annex B (Continued)

Author(s)	*Year*	*Finding/Conclusion Cited*	*Remark(s)*
Hegsted, D.M. et al.	1959	Saturated fatty acid and linoleic acid lowered levels of serum cholesterol in rats synergistically.	The results do not support the view that the saturated fats counteracts the effect of polyunsaturated fats.
Malmros, et al.	1957	Feeding of coconut oil as sole source of fat did not affect serum cholesterol.	
Kaunitz, H. et al.	1958	MCT in the diet of rats reduced the requirements for essential fatty acid compared with the long chain triglyceride diet without added linoleic.	These results are important because of the relation of linoleic to thrombo treatment phenomenon via prostalandins and to the formation of free radicals which are implicated in carcinogenesis and ageing.
Kaunitz, H. et al.	1959	MCT intake is associated with lower serum cholesterol in rats, man and many other mammalian species.	MCT with linoleic acid is associated with reduction of liver cholesterol.
Grande, F. et al.	1961	Short and medium chain triglycerides are digested, absorbed and metabolized differently from long chain fatty acid.	
Harcraft, W.S. and Porta, G.A.	1965	Linoleic acid forms unstable peroxide which leads to free radicals which have been associated with accelerated ageing.	
Senion, et al.	1968	Stomach and intestine contain enzymes which hydrolyze MCT more rapidly and completely than LCT. Fatty acid and monoglycerides of MCT are much more water soluble and can be absorbed in the absence of bile salts.	
Moore, et al.	1968	Rats fed hydrogenated coconut oil requires higher level of linoleate.	
Yogi, K.	1976	Linoleic acid peroxide is related to formation of atherosclerotic lesions.	

Annex B (Continued)

Author(s)	*Year*	*Finding/Conclusion Cited*	*Remark(s)*
Kaunitz, H.	1986	Polyunsaturated vegetable oils may have toxic effects.	
B.3.2 Human Clinical Studies			
Bloom, et al.	1951	Short and medium chain triglycerides are digested, absorbed and metabolized differently from long chain fatty acid.	
Kaunitz	1958	Humans fed with reducing diets with medium chain triglycerides lost weight more rapidly than with equal amounts of natural fats.	
Hashim, et al.	1960	C10 and below are not hypercholesterolemic. Saturated fatty acid of C10 and below do not raise serum cholesterol levels.	
Halden, W. and Lieb, H	1961	Coconut oil containing 5% safflower oil added to standard diet of patients reduced cholesterol level.	
Sheig, A.	1968	MCT easy oxidation provides a quick source of available energy for after major surgery or for premature infants. Seemingly, it promotes solubilization of gallstones.	
Kaunitz, et al.	1971	MCT corrects abnormal serum triglyceride levels and is associated with reduced fat deposition and increased caloric requirements for weight.	This suggests that MCT can be used for human obesity. It can be used also to ameliorate stress condition.
Thistle, J.L. et al.	1977	MCT leads to solubilization of gallstones.	
Ehrhart, et al.	1984	When total fat calories is decreased from 40% to 20%, HDL cholesterol are decreased independently from ratio of P/S maintenance.	

Annex B (Continued)

Author(s)	*Year*	*Finding/Conclusion Cited*	*Remark(s)*
Babayan, V.K. and G.L. Blackburn	1987	Lauric acid is the bridge getween MCT and LCT, both in its intestinal transport pattern, entry into the circulation system and digestion.	While lauric acid is not a pure MCT, it is important in metabolism.
B.3.3 Human Epidemiologic Studies			
Withrow, C.D.	1980	Because of MCT's ketogenic effects, it may help in controlling convulsions of epileptics.	
Bach, A.C. and Babayan, V.K.	1982	MCT offers advantages in the treatment of malabsorption syndrome, hyperlipidemia or obesity.	
Caroll, K.K.	1984	Fats in general, particularly unsaturated vegetable oils, lead to more frequent occurrence mammary cancer.	
Stemmermann, G.N. et al.	1984	Saturated animal fat favors development of colon tumors.	
B.3.4 Other Citations			
Mc Gandy	1966	The relative position of individuals in the study population remain in the same tertiles as far as cholesterol and lipoprotein levels are concerned, when given different diets in controlled metabolic experiments.	This suggests a large non-dietary intrinsic control on lipid and lipoprotein levels. The effect of diet is only superimposed on this intrinsic difference.

Glossary

CE = esterified cholesterol

CO = coconut oil

EFA = essential fatty acid

FC = unesterified cholesterol

FF = fat-free

HCNO or HCO = hydrogenated coconut oil

HDL = high density lipoprotein

IDL = intermediate density lipoprotein

IDH = ischemic heart disease

LCT = long-chain triglyceride

LDL = low density lipoprotein

MCT = medium-chain triglyceride

NNM = number not mentioned

P/S = ratio of polyunsaturated to saturated fatty acids

VLDL = very low density lipoprotein

References

Aguilar, C. and C. Florencio. 1980. Effects of Type and Amount of Vegetable Oil on Protein Utilization in Rats. *Phil. J. of Nutr.* April-June 1986, pp. 83-95.

Ahrens, E.H., J. Hirsch, W. Insull, T.T. Tsaltas, R. Blomstrand, and M.L. Peterson. 1957. The Influence of Dietary Fats on Serum Lipid Levels in Man. *Lancet.* 1: 943-953.

Ahrens, E.H. 1979. Dietary Fats and Coronary Heart Disease: Unfinished Business. *The Lancet,* December 22-29, 1979, pp. 1345-1348.

American Heart Association. 1986 *Dietary Guidelines for Healthy American Adults.* A Statement for Physicians and Health Professionals by the Nutritional Committee. American Heart Association.

Babayan, V., H. Kaunitz, C.A. Slanetz, and R.E. Johnson. 1958. Essential Fatty Acid Sparing Action of Saturated Medium Chain Length Triglycerides. Federation Proceedings. Vol. 17, No. 1.

Babayan, V.K. 1987. Medium Chain Triglycerides and Structured Lipids. *LIPIDS.* Vol. 22, No. 6: 417-420.

Bach, A.C., and V.K. Babayan. 1982. Medium Chain Triglycerides: An update. *Amer. J. Clin. Nutr.* 36: 950-962.

Bierenbaum, M.L., D.P. Green, A. Florin, A.T. Fleischman, and A.B. Caldwell. 1967. Modified-fat Dietary Management of the Young Male With Coronary Disease: A Five-Year Report. *J. Amer. Med. Assoc.* Vo. 202, No. 13, pp. 1119-23.

Blackburn, G.L. and V.K. Babayan. 1987. Letter to the Editor. *Amer. J. Clin. Nutr.* 45: 399-405.

Caselli, W.P. 1986. The Triglyceride Issue: A View from Framinoham. *Amer. Heart. J.* 112(2): 432-437.

Camara-Besa, S.F., M. Bataclan, and J.N. Eleazar. 1974. The Serum Cholesterol and Phospholipid Levels of the Population of Nine Regions of 0he Philippines and their Relation to the Regional Dietary Patterns. *J. Phil. Med. Asso.* 50: 65-100.

Castillo, L.S., A.L. Gerpacio, T.R. Javier, I.B. Cano, L.A. Gloria, and C.P. Gerpacio. 1962. The Utilization of Coconut Oil in Livestock and Poultry Feeding: II. In Rabbit Rations. *The Philippine Agriculturist.* V. 46, pp. 266-287.

Chu, S.W. and D.M. Hegsted. 1980. Lymph Fatty Acid Composition During Constant Infusion of Saturated Fats of Different Chain Lenths in the Rat. *J. Nutr.* 110: 2198-2206.

Connor, S.L., S.M. Artaudwild, C.J. Classick-Kohn, W.E. Connor, J.R. Gustaeson, D.P. Flavell, and L.F. Hatcher. 1986. The Cholesterol/Saturated Fat Index: An Indication of the Hypercholesterolaemic and Atherogenic Potential of Food. *Lancet.* p. 1229-132.

Cornwell, D.G. and R.V. Panganamala. Atheroscerosis: An Intracellular Deficiency in Essential Fatty Acids. *J.P.L.T.* 20: 365-376.

Cowgill, G., D.L. Estrich and P.D. Wood. 1969. Proceedings of the 1968 Deuel Conference on Lipids on *The Turnover of Lipids and Lipoproteins.* Feb. 21-24, 1968. Carmel, California. USDHEW. Maryland. 188 pages.

del Castillo, E. Ma. M. 1982. Effect of Coconut Milk/Oil Consumption on Blood Cholesterol Levels of a Rural Bicolano Population. B.S. in Agricultural Chemistry Thesis, UPLB.

Department of Health. 1984. Philippine Health Statistics. 1984.

Deuel, H.J., Jr., R.B. Alfin-Slater, A.F. Wells, G.D. Kryder, and L. Aftergood. 1955. The Effect of Fat Level of the Diet on General Nutrition. J. XIV. Further Studies of the Effect of Hydrogenated Coconut Oil on Essential Fatty Acid Deficiency in the *Rat. J. of Nutr.* 55: 337-346.

Divakaran, P. and A. Venkataraman. 1977. Effect of Deitary Fats on Oxidative Phosphorylation and Fatty Acid Profile of Rat Liver Mitochondria. *J. Nutr.* 107: 1621-1631.

Dyerberg, J. 1986. Linolenate-derived Polyunsaturated Fatty Acids and Prevention of Atherosclerosis. *Nutrition Reviews.* Vol. 44, No. 4, pp. 125-134.

Ehrhart, L.A., R. Balachandran, A. Butkus, L.A. Lewis, and A. L. Robertson. 1973. Lipid Metabolism of Acetatate-1-14C by Leukocytes From Dogs Fed on Arteriosclerosis – Inducing Diet. *Lipids.* Vol. 6, No. 12, pp. 895-900.

Erickson, B.A., R.H. Coots, F.H. Mattson and A.M. Kligman. 1964. The Effect of Partial Hydrogenation of Dietary Fats, of the Ratio of Polyunsaturated to Saturated Fatty Acids, and of Dietary Cholesterol Upon Plasma Lipids in Man. *J. Clin. Investigation.* 43(11): 2017-2025.

Ernst, N.D. and R.I. Levy. 1984. *Present Knowledge and Nutrition.* 5th ed., p. 124-139. Nutrition Foundation, Inc., Washington.

Fisher, E.A., C.B. Blum, V.I. Zannis, and J.L. Breslow. 1983. Independent Effects of Dietary Saturated Fat and Cholesterol on Plasma Lipids, Lipoproteins, and Apolipoprotein E.J. *Lipid* Res. 24:1039-1046.

Florentino, R.F. and A.R. Aguinaldo. 1987. Diet and Cardiovascular Disease in the Philippines. *Phil. J. Coconut Studies.* Vol. XIII, No. 2: 56-70.

Frantz, I.V. and J.B. Carey, Jr. 1961. Cholesterol Content of Human Liver After Feeding of Corn Oil and Hydrogenated Coconut Oil. P.S.B.M. *106:* 800-801.

Fries, M.W. 1983. Coconuts and Allergy. *Coconut Today.* Sept. 23, 1983: 31-34.

Gerpacio, A.L., R.B. Puyaoan, T.R. Javier, and L.S. Castillo. 1963. Utilization of Coconut Oil in Livestock and Poultry Feeding. III. Growing-Finishing Swine Rations. *The Philippine Agriculturist.* V. 47, pp. 383-392.

Goldbourt, U. 1987. High Risk Versus Public Health Strategies in Primary Prevention of Coronary Heart Disease. *Amer. J. Clin. Nutr.* 45: 1185-1192.

Grande, F., J.T. Anderson and A. Keys. 1961. The Influence of Chain Length of the Saturated Fatty Acids on Their Effect on Serum Cholesterol Concentration in Man. *J. Nutrition.* 74: 420-428.

Grande, F. and W.F. Prigge. 1974. Serum Lipid Changes Produced in Dogs by Substituting Coconut Oil for Either Sucrose or Protein in the Diet. *J. Nutr.* 104: 613-618.

Grundy, S.M., D. Bilheimer. H. Blackburn, W.V. Brown, P.O. Kwiterovich. 1982. Rationale

of the Diet-Heart Statement of the American Heart Association: report of nutrition committee. Circulation 65, No. 4: 839A-854A.

Grundy, S.M. 1987. Monounsaturated Fatty Acids, Plasma Cholesterol, and Coronary Heart Disease. *Amer. J. Clin. Nutr.* 45: 1168-1175.

Guerrero, R. Ma., C.G. Lopez, I. Y. Zalamea, R.N. Lopez, and C. Z. Zaballero. 1957. Clinical Trial With a Coconut-Oil-Evaporated Milk Preparation. Philipp. *J. Pediat.* 6(1): 19-23.

Haberland, M.E. and A.M. Fogelman. 1987 The Role of Altered Lipoproteins in the Pathogenesis of Atherosclerosis. *American Heart Journal.* 113(2): Part 2 573-557.

Halden, V.W. and H. Lieb. 1961. Uber den EinfluB Veredelter Kokosfett-Produkte auf die Serum-Cholesterinwerte von Versuchspersonen. Nutr. Dieta. *3(2):* 75-88.

Hamawy, V.W., L.L. Maldawer, M. Georgieff, A.J. Valicente, V. K. Babayan, B.R. Bistrian, and G.L. Blackburn. 1985. The Effect of Lipid Emulsions on Reticuleondothelial System Function in the Injured Animal. *Journal of Parenteral and Enteral Nutrition.* 9: 559-565.

Harper, A.E. 1983. Coronary Heart Disease – An Epidemic Related to Diet? *Amer. J. Clin. Nutr.* 37: 669-681.

Hashim, S.A., R.E. Claney, D.M. Hegsted, and F.J. Stare. 1959. Effect of Mixed Fat Formula Feeding on Serum Cholesterol Level in Man. *Amer. J. Clin. Nutr.* 7: 30-34.

Hashim, S.A., A. Arteaga, T.B. Van Itallie. 1960. Effect of a Saturated Medium-Chain Triglyceride on Serum-Lipids in Man. *Lancet.* 1: 1108-1108.

Hautvast, J.G.A.J., J.T. Knuiman, and C.E. West. 1984. International Studies on Nutrition and Serum Lipoproteins in Children. *Journal of Food and Nutrition.* 41(1): 9-13.

Hill, E., V. Lundberg, and J.L. Titus. 1971. Experimental Atherosclerosis in Swine. 1. A Comparison of Menhaden-Oil Supplements in Tallow and Coconut Oil Diets. Mayo Clin. Proc. 46: 613-620.

Holman, R.T., H. Hayes, H. Malmros, and G. Wigand. 1957. Effects of Dietary Fat Upon Plasma Polyunsaturated Acids. *Proc. Soc. Exp. Biol. Med.* 96: 705-709.

Hostmark, A.T., O. Spydevod, and E. Eilertson. 1980. Plasma Lipid Concentration and Liver Output of Lipoproteins in Rats Fed Coconut Fat or Sunflower Oil. *Artery.* 7(5): 367-383.

Icayan, E.E., R.T. Lagua, F. Martires, R. Batungbacal, and C. I. Pamintuan. 1964. Effects of Feeding Different Level of Coconut Oil on the Serum Lipids of Normal Medical Students. *U.E.J. Med.* 1: 3-13.

Jandacek, R.J., J.A. Whiteside, B.N. Holcombe, R.A. Volpenhein, and J. D. Taulbee. 1987. The Rapid Hydrolysis and Efficient Absorption of Triglycerides with Octanoic Acid in the 1 and 3 Positions and Long-chain Fatty Acid in the 2 Position. *Amer. J. Clin. Nutr.* 45: 940-945.

Katan, M. B. and A.C. Beyten. 1981. Linoleic Acid Consumption and Coronary Heart-Diease in U.S.A. and U.K. *The Lancet.* 2: 371.

Kater, G., E.A. Mascioli, V.K. Babayan, B.R. Bistrian, and G. L. Blackburn. 1987. "A Reevaluation of Coconut Oil's Effect on Serum Cholesterol and Atherogenesis" A Technical Report Presented to the U.S. Congress, September 1987, p. 22.

Kaunitz, H., C.A. Slanetz, R.E. Johnson and V.K. Babayan. 1959. Relation of Cholesterol Levels to Dietary Medium and Long Chain Saturated Triglycerides and Linoleic Acid. Federation Proceedings. 18(1).

Kaunitz, H., C.A. Slanetz, R.E. Johnson and V.K. Babayan. 1960. Relation of Linoleic Acid and Chain Length of Triglycerides to Natural Fat Deposition. Federation Proceedings. 19(1) Part 1.

Kaunitz, H. 1986. Chemically Manipulated Fats – Human Health. Paper Presented at the Foundation of Appropriate Nutrition. Unpublished.

Kaunitz, H. 1970. Nutritional Properties of Coconut Oil. *J. Am. Oil Chemists' Soc.* Vol. 47: 462A-482A.

Kaunitz, H. 1972. Nutritional Properties of Coconut Oil. Ceylon Cocon. Q. 23: 85-91.

Kaunitz, H. 1973. Nutritional Properties of Coconut Oil: Its Use in Filled Milk. *Chemist's Quarterly.* 11: (& 2): 4-15.

Kaunitz, H. 1973. Biological Effects of Coconut Oil and Its Constituent Fatty Acids. *Phil. J. of Internal Med.* 11: 43-52.

Kaunitz, H. 1986. Medium Chain Triglycerides (MCT) In Aging and Arteriosclerosis. *Journal of Environmental Pathology, Toxicology and Oncology.* 6: 115-125.

Keys, A., J.T. Anderson, and F. Grande. 1957. Letters to the Editor, Serum-Cholesterol Response to Dietary Fat. *Lancet,* April 13, 1957, p. 787.

Keys, A., J.T. Anderson, and F. Grande. 1957. Prediction of Serum Cholesterol Responses of Man to Changes in Fats in the Diet. *Lancet.* 2: 959-966.

Keys, A., R. Buzina, E. Ferber, A. Brodarec, B. Agneletto, A. Horvat, P. Roine, M. Pekkavinen, M.J. Karvonen, C. Aravanis and H. Sdrin. 1970. XVIII. The Diet, Coronary Heart Disease in Seven Countries. Circulation, Supplement 1, XLI and XLII: pp. I-162 – I-198.

Kinsell, L.W., R.W. Friskey, G.D. Michaels, and F.R. Brown, Jr., after 1955. Effect of a Synthetic Triglyceride on Lipid Metabolism. Letters to the Editor. *Amer. J. Clin. Nutr.* Vol. 4, No. 3, pp. 285-286.

Kinsell, L.W., G.D. Michaels, R.W. Friskey, and S. Splitter. 1958. Essential Fatty Acids, Lipid Metabolism, and Atherosclerosis. *Lancet.* 1: 334-339.

Kintanar, Q.L. 1987. Absorption of Fats and Oils and Lipid Metabolism. *Phil. J. of Coconut Studies,* Vol. XII, No. 2, pp. 18-29.

Kintanar, Q.L. 1988. Coconut Oil as Metabolic Energy Source. *Coconut Today.* (In Press)

Knuiman, J.T. S, Westenbrink, E. Heyden, C.L. West, J. Burema, J. De Boer, J.G.A.J. Hautvast, L. Rasanen, L. Virkkunen, J. Vhkari, P. Lokko, J.O.M. Pobre, A. Ferro-Luzzi, A.M. Ferrini, C. Scaccini, S. Sette, G.M. Villavieja, and J. Bulatao-Jayme. 1983. Determinants of Total and High-Density Lipoprotein Cholesterol in Boys from Finland, The Netherlands, Italy, the Philippines and Ghana with Special Reference to Diet. *Human Clin. Nutr.* 37C: 237-254.

Krauss, R.M. 1987. Relationship of Intermediate and Low-Density Lipoprotein Subspecies to Risk of Coronary Artery Disease. *American Heart Journal.* 113(2): Part 2: 578-582.

Kritchevsky, D., S.A. Tepper, H.K.Kim, J.A. Story, O. Vesselinovitch, and R.W. Wissler. 1976. Experimental Atherosclerosis in Rabbits Fed Cholesterol-free Diets. 5 Comparison of Peanut, Corn, Butter and Coconut Oils. *Exp. Mol. Pathol.* 24: 375-391.

Kritchevsky, D. 1969. Experimental Atherosclerosis in Primates and Other Species. *Ann. N.Y. Acad. Sci.* 162: 80-88.

Kritchevsky, D., S.S. Tepper, G. Bises, and D.M. Klurfeld. 1982. Experimental Atherosclerosis in Rabbits Fed Cholesterol-Free Diets. Part 10. Cocoa, Butter, and Palm Oil. *Atherosclerosis.* 41: 279-284.

Labadan, M.M., L.S. Castillo, A.A. Ovejera, A.L. Gerpacio, and T.R. Javier. 1962. The Utilization of Coconut Oil in Livestock and Poultry Feeding: I. In Broiler Rations. *The Philippine Agriculturist.* V. 56: 143-155.

Levy, R.I. and N. Ernst. 1973. Diet, Hyperlipidemia and Atherosclerosis. In: *Modern Nutrition in Health and Disease.* (Diet Therpay) (R.S. Goodhart & M.E. Shils, eds.) Lea & Febiger, pp. 895-901.

Maiz, A., K. Yamasaki, J. Sobrado, V.K. Babayan, L.L. Moldawer, B.R. Bistrian, and G.L. Blackburn. 1984. Protein Metabolism During Total Parenteral Nutrition (TPN) in Injured Rats Using Medium-Chain Triglycerides. Metabolism. Vol. 33, No. 10, pp. 901-909.

Malmros, H. and G. Wigand. 1957. The Effect on Serum Cholesterol of Diets Containing Different Fats. *Lancet.* 2: 1-7.

Malmros, H. and N.H. Sternby. 1968. Induction of Atherosclerosis in Dogs by a Thiouracil-Free Semisynthetic Diet Containing Cholesterol and Hydrogenated Coconut Oil. *Progr. Biochem. Pharmacol.* 4: 482-487.

Mascioli, E.A., B.R. Bistrian, V.K. Babayan, and G.L. Blackburn. 1987. Medium Chain Triglycerides and Structure Lipids as Unique Nonglucose Energy Sources in Hyperalimentation. *Lipids.* Vol. 22, No. 6, p. 421-423.

McCullagh, K.G., K.A. Ehrhart, and A. Butkus. 1976. The Dietary Induction of Severe Coronary, Cerebral, Aortic, and Iliac Atherosclerosis and Its Prevention by Safflower Oil. Laboratory Investigation . Vol. 34, No. 4, pp. 394-405.

McGill, H.C. and G.E. Mott. 1976. Diet and Coronary Heart Disease in *Present Knowledge in Nutrition.* 4th ed., Nutrition Foundation, Inc., Washington, D.C., pp. 376-391.

McGovern, R.F. and F.W. Quackenbush. 1973. Effect of Dietary Fat Saturation on Absorption and Intestinal Secretion of Cholesterol by the Hypercholesterolemic Rat. *Lipids.* Vol. 8, No. 8, pp. 470-472.

McGregor, L. 1974. Effect of Feeding with Hydrogenated Coconut Oil on Platelet Function in Rats. (Abst.) Proc. Nutr. Soc. 33: 1A-2A.

Miettinen, T.A., Y.A. Kesaniemi, M. Farkkila, P. Koivisto and M. Vuoristo 1987. Enhanced Cholesterol Elimination and Synthesis Related to Low-density Lipoprotein Metabolism. *American Heart Journal.* 113(2): Part 2: 487-492.

Miller, N.E. 1987. Associations of High-Density Lipoprotein Subclasses and Apolipoproteins with Ischmeic Heart Disease and Coronary Atheroclerosis. *American Heart Journal.* 113(2): Part 2: 589-597.

Miranda, C.L., M. Caedo, and J.M. Gonzales. 1974. Effects of Coconut-Corn Oil Mixtures on White Rats. *Phil. J. Nutr.* 27: 13-17.

Mok, K.T., A. Maiz, K. Yamazaki, J. Sobrado, V.K. Babayan, L.L. Moldawer, B.R. Bistrian, and G.L. Blackburn. 1984. Structured Medium-Chain and Long-Chain Triglyceride Emulsions are Superior to Physical Mixtures in Sparing Body Protein in the Burned Fat. *Metabolism.* Vol. 33, No. 10, pp. 910-915.

Monmaney, T., M. Hager, G. Raine, and L. Drew. "One for the Heart." *Newsweek,* June 29, 1987.

Morin, R.J., S. Bernick, and R.B. Alfin-Slater. 1964. Effects of Essential Fatty Acids Deficiency and Supplementation on Atheroma Formation and Regression. *J. Atheroscler.* Res. 4: 387-396.

Multiple Risk Factor Intervention Trial Research Group, 1982. *J.A.M.A.* Vol. 248, No. 12: 1465-1477.

Navarro, M.D. 1958. Poor Cholesterol and Low Fat Diet In Cancer. *J. Philipp. Med. Assn.* 34(5): 311-315.

Nestel, Paul J. 1987. Polyunsaturated Fatty Acids (n-3, n-6) 1-3. *Amer. J. Clin. Nutr.* 45: 1161-67.

Nichaman, M.Z., and P. Mann. 1987. Low-fat, High-carbohydate Diets and Plasma Cholesterol. *Amer. J. Clin. Nutr.* 45: 1155-1160.

Nicolosi, R.J., J.L. Hojnacki, N. Llansa, and K.C. Hayes. 1977. Diet and Lipoprotein Influence on Primate Atherosclerosis. *Proc. Soc. Exp. Biol. Med.* 156: 1-7.

Nicolosi, R.J., J.A. Marlett, A.M. Morello, S.A. Flanagan, and D.M. Hegsted. 1981. Influence of Deitary Unsaturated and Saturated Fat on the Plasma Lipoproteins of Mongolian Berbils. Atherosclerosis. *38:* 359-371.

NIH, 1971. *Arteriosclerosis,* A Report by the National Heart and Lung Institute Task Force on Arteriosclerosis (NIH), DHEW Publ. No. (NIH) 72-219., Vol. 2: 57-59.

NIH, 1981. *Arteriosclerosis,* Report of the Working Group on Arteriosclerosis of the National Heart, Lung and Blood Institute, NIH, (NIH Publ. No. 82-2035), Vol. 2: 52-59.

Paletta, B., W. Mlekusch, and K. Vrecko. 1980. The Influence of an Atherogenic, Semisynthetic Diet on Cholesterol Metabolism in Rabbits. *Atherosclerosis.* 37: 371-375.

Podbielski, F.J., R.T. Bridenstine, D. Vesselinovitch, and R. W. Wissler. 1987. Plasma Platelet Factor 4 Response in Rhesus Monkeys Fed Coconut Oil. Department of Pathology and the Specialized Center of Research on Atherosclerosis (SCOR); The Program of Arts and