



## A comparison between the effects of coconut oil and other vegetable oils on human health, with emphasis on heart disease

Shamina Azeez\*, Rajagopal Vellamoor, T. Jayarama Pai<sup>1</sup>  
and Arunachalam Vadivel<sup>2</sup>

Central Plantation Crops Research Institute, Kasaragod 671124, Kerala

<sup>1</sup>KG Heart Surgery Centre, KG Hospital, Coimbatore - 641018, Tamil Nadu

<sup>2</sup>ICAR Research Complex for Goa, Ela, Old Goa - 403402, Goa

(Manuscript Received: 12-10-11, Revised: 11-12-11, Accepted: 08-02-12)

### Abstract

Medical records of 550 subjects (394 men and 156 women), from an age group of 18-83 years were collated using a standard proforma exploring personal details, their habits, dietary pattern including the dietary oils used such as coconut oil, gingelly oil, sunflower oil and groundnut oil, physiological and clinical status, blood biochemical profile, medications, socio-psychological status, *etc.* Biochemical data of blood samples were related to the type and quantity of oil consumed. Gingelly oil correlated well with low serum cholesterol levels, followed by sunflower oil, coconut oil and groundnut oil. Irrespective of the type of oil used, quantity of oil consumed played a major role in the serum cholesterol, HDL and triglyceride levels directly. However, all the four vegetable oils studied, appeared safe for human health. This study also challenges the belief that coconut oil is hypercholesteremic in nature.

**Keywords:** Cardiovascular disease, coconut oil, cholesterol, gingelly oil, groundnut oil, lipid profile, sunflower oil

### Introduction

Coconut (*Cocos nucifera*) is a perennial palm, grown in the humid tropics for the dietary use of its fresh kernel, desiccated copra and oil. It is one of the most important and richest sources of vegetable oil. Coconut oil is very heat-stable, and suitable for cooking at high temperatures for frying. It is slow to oxidize and resistant to rancidity for up to two years due to its high saturated fat content.

The traditional use of coconut oil as a dietary oil in the tropics has been challenged since the 1950s, due to its alleged hypercholesteremia, (Keys *et al.*, 1957; Hegsted *et al.*, 1965). Several organizations like United States Food and Drug Administration, World Health Organization, International College of Nutrition, United States Department of Health and Human Services, American Dietetic Association,

American Heart Association, British National Health Service, Dietitians of Canada *etc.* recommend against the consumption of significant amounts of coconut oil due to its high levels of saturated fat content. But, coconut oil contains 31-47 per cent of lauric acid, a saturated fat that can increase the high density lipoprotein (HDL) cholesterol in the blood though it is unclear if coconut oil may promote atherosclerosis through other pathways (Mensink *et al.*, 2003). Advocacy against coconut and palm oils in the 1970s and 80s due to their perceived danger as a saturated fat, the companies started to substitute trans fats, unaware of their health-damaging effects (McNamara, 2010). But a fact largely overlooked is the quality of the saturated fatty acids in the oil, *viz.* that the saturated fatty acids in coconut oil are predominantly medium and small chain fatty acids and glycerides (Child, 1974; Azeez, 2007). In

\*Corresponding Author: shamina@spices.res.in

addition, virgin coconut oil is composed mainly of medium-chain triglycerides, which may not carry the same risks as other saturated fats (Tarrago-Trani *et al.*, 2006; Marina *et al.*, 2009). Early studies on the health effects of coconut oil, was conducted on partially hydrogenated coconut oil, which creates trans fats, and not virgin coconut oil, which has a different health risk profile (Kintanar, 1988; Enig, 1990). Epidemiological studies do not support the claim that coconut oil is contraindicated in coronary heart disease patients (Kaunitz and Dayrit, 1992).

Gingelly or sesame oil (also known as til oil) is an edible vegetable oil derived from sesame seeds, used in South India. It is also widely used as a flavor enhancer in Chinese, Japanese, Korean and to a lesser extent in southeast Asian cuisine. Sesame oil is predominantly made up of equal amounts of oleic and linoleic acids (35-50%). Despite sesame oil's high proportion (41%) of polyunsaturated (Omega-6) fatty acids, among cooking oils with high smoke points, it is least prone to turn rancid due to the natural antioxidants present in the oil.

Sunflower oil is a non-volatile oil light in taste and appearance, commonly used as a frying oil. It is mainly a triglyceride, and consists of 4-9% palmitic acid, 1-7% stearic acid, 14-40% oleic acid, 48-74% linoleic acid (Thomas, 2002). Sunflower oil also has high vitamin E content, and contains lecithin, tocopherols, carotenoids and waxes. It is a combination of mono- and polyunsaturated fats with low saturated fat levels.

Groundnut oil is derived from groundnut, with its unique aroma and taste and high smoke point. It is often used in Chinese and southeast Asian cuisine. Its major component fatty acids are oleic acid (46.8%), linoleic acid (33.4%), and palmitic acid (10.0%). The oil also contains some stearic acid, arachidic acid, arachidonic acid, behenic acid, lignoceric acid and other fatty acids (USDA National Nutrient Database).

Coronary artery disease (CAD) accounts for 25-30% deaths globally, every year. The predominant risk factors associated with CAD are modifiable factors like hypertension, elevated serum cholesterol, diabetes mellitus, obesity, sedentary habits and stress while others are non-modifiable

such as age, sex, family history and genetic factors (<http://www.webmd.com/>).

According to World Health Report, it was predicted that cardiovascular disease (CVDs) will be the major cause for death and disability in India by 2020. The major risk factors for developing CVD are mainly unhealthy dietary habits. In this context, a study was carried out with the objective to correlate the consumption of coconut oil *vis-a-vis* sunflower, gingelly and groundnut oils, and its effect on human health, with emphasis on heart diseases.

### Materials and methods

The medical records of 550 subjects (394 men and 156 women) who appeared for normal check up and for treatment of illness were used for in this study by collecting both personal and clinical information using a standard proforma. The proforma contained information such as personal details of individuals, their habits, dietary pattern, physiological and clinical status, blood biochemical profile, medications, socio-psychological status, *etc.* The data was collected from the Cardiology Division of the KG Hospital, K. Govindaswamy Naidu Medical Trust, Coimbatore, Tamil Nadu, India.

Since the objective of this study is to correlate the effect of coconut oil *vis-a-vis* other edible vegetable oils on coronary heart disease (CHD), emphasis was given to collect details on the quality and quantity of the oils used by the individuals. Apart from coconut oil, coconut kernel is also used directly in the dietary preparations for consumption. Hence, to correlate the calories derived from coconut kernel and oil with CHD, the number of coconuts used per week by the subjects were quantified. It was estimated that an average fresh, mature coconut kernel weighs approximately 200–250 g, and contains 444 kcal energy, 4.5 g protein, 13 g carbohydrates, 41.6 g fat and 3.6 g fiber per 100 g of coconut (Assa *et al.*, 2010). Coconut kernel is the richest source of oil, yielding 65–70%. These standard values were used for calculating the calories and fat content in the daily diet of individuals and to correlate with parameters like lipid profile, blood sugar, BMI and waist:hip ratio. The data collected were analyzed statistically using, spearman correlation coefficient, regression and multivariate

means and compared with the normal, prescribed physiological and biochemical values for the healthy adult (Murray, 1990) using SPSS 10.0.

Cholesterol, HDL, LDL, triglyceride contents and HDL/LDL ratio in the serum of the individuals were analyzed for assessing the association with number of coconuts consumed per week. Using the calorific value and quantity of the foods consumed by the individuals, total calories consumed per day per individual was worked out. Percentage of calories from coconut kernel and oil were also worked out as described above and correlated with the biochemical parameters of the subjects. Mean values of serum cholesterol (mg %) and triglycerides (mg %) in the subjects consuming coconut, gingelly, groundnut, sunflower were worked out and compared statistically. Pair wise comparison of each of the four vegetable oils was performed with other three oils for total serum cholesterol (mg %) of the subjects using SPSS 10.0.

Correlation coefficients were worked out for height, weight, BMI, waist:hip ratio, oil consumed (liters/month) number of coconuts consumed per week, total calories consumed per day, total fat consumed per day, prandial sugar level, serum cholesterol, HDL, LDL and triglyceride contents and HDL/LDL ratio.

### Results and Discussion

The data on the dietary habits of the subjects revealed that a vast majority (70%) of the subjects

used sunflower oil and an equal number - 11% - used either branded or home-made gingelly or groundnut oil; and only 5% used coconut oil. A small number of subjects used other oils like palm oil, rice bran oil or olive oil and only 10% used more than one type of oil for cooking.

**BMI and lipid profile:** Analysis of the data revealed that regression equations were not significant for most combinations except in a few important quantitative parameters (Table 1). The subjects conformed to the pattern seen in the general population, *viz.*, high positive correlation between weight and body mass index (BMI) (0.8), LDL and total serum cholesterol (0.84), high negative correlation between total serum cholesterol and HDL/LDL ratio (-0.76), and high negative correlation between LDL and HDL/LDL ratio (-0.912). The total calories consumed had significant correlation with serum total fat, LDL and cholesterol.

**Coconuts, calories and lipid profile:** The number of coconuts consumed per week was negatively correlated with calories intake (0.2) (Table 1). Calories from coconut kernel or oil did not affect the lipid profile of the subjects (Table 3). This observation corroborates with the earlier observation made by Blackburn *et al.* (1992) that coconut oil is a neutral dietary fat in terms of atherogenicity, when adequately supplemented with the essential fatty acid linoleic acid, which is deficient in coconut oil. Comparison of the parameters relating to coconut consumption revealed that no significant

**Table 1. Spearman's correlation table of clinical data**

Clinical data	Height	Weight	BMI	Waist/Hip ratio	Oil/ Month	Coconut/ Week	Total Calories	Total Fat	Sugar Prandial	Cholesterol	HDL	LDL	Triglyceride	HDL/LDL ratio
Height		0.467**												
Weight			0.800**											
BMI														
Waist/Hip	0.245**	0.347**	0.245**											
Oil/Month				0.142**										
Coconut/ Week														
Total Calories	0.179**	0.120**				0.101*								
Total Fat					0.094*	-0.203**	0.485**							
Sugar Prandial		0.146**	0.188**											
Cholesterol			0.095*				0.114**	0.158**						
HDL	-0.093*							-0.094*						
LDL							0.110**			0.840**				
HDL/LDL							-0.097*	-0.119**	-0.760**	0.274**	-0.912**	-0.179**		
Triglyceride		0.230**	0.207**	0.165**				0.205**	0.394**	-0.310**				

association was noticed between number of coconuts consumed per week and cholesterol, HDL, LDL, triglyceride contents and HDL/LDL ratio (Table 2), showing that the number of coconuts consumed per week has almost no effect on the blood lipid profile. Regression analysis showed significant correlation in the prandial blood sugar and many of the physiological and biochemical parameters as shown in Table 4. BMI, height, weight, oil consumed/month, coconuts

**Table 2. Measures of association number of coconuts consumed per week and serum lipid profile**

Association	R	R Squared
Cholesterol*Coconut consumed/week	0.001	0.000
HDL*Coconut consumed/week	-0.041	0.002
LDL*Coconut consumed/week	0.010	0.000
HDL:LDL*Coconut consumed/week	-0.036	0.001
Triglycerides*Coconut consumed/week	-0.021	0.000

**Table 3. Correlation of calories from coconut oil and coconut kernel to serum biochemical parameters**

Biochemical parameters	Calories from coconut oil (%)	Calories from coconut (%)	Calories from oil and coconut (%)
Cholesterol	-0.029	-0.057	-0.058
HDL	-0.020	-0.012	-0.029
LDL	-0.024	-0.011	-0.029
Triglyceride	-0.026	-0.011	-0.029
Prandial sugar	0.004	-0.013	-0.029
BMI	-0.010	-0.012	0.021
WHR	0.013	-0.012	0.021

**Table 4. Coefficient of prandial blood sugar content and physiological/biochemical parameters**

Model	Unstandardized Coefficients		Standardized Coefficient	t	Sig.
	B	SE			
(Constant)	63.999	30.935		2.069	0.039
BMI	3.019	0.892	0.282	3.383	0.001
Weight	-0.611	0.316	-0.170	-1.935	0.053
Height	0.145	0.162	0.046	0.896	0.371
Total calories	-4.47E-03	0.009	-0.030	-0.483	0.629
Total fat	0.214	0.152	0.102	1.405	0.161
Coconuts/week	2.027	0.759	0.115	2.672	0.008
Oil/month	0.759	2.544	0.013	0.298	0.766
Breakfast calories	8.210E-03	0.026	0.021	0.321	0.748
Breakfast fat	-0.372	0.581	-0.048	-0.641	0.522
Early morning calories	1.087E-02	0.078	0.008	0.138	0.890
Early morning fat	-3.098	1.658	-0.103	-1.868	0.062

<sup>a</sup>Dependent variable: Prandial sugar

consumed/week, total calories and total fat were significantly related to HDL:LDL ratio ( $p=0.052$ ).

Dietary fat yields 9 kcal/g energy, compared to 4 kcal/g for carbohydrate and protein (FAO/WHO, 1978). But metabolism of the predominantly small to medium chain fatty acid (MCT) containing triglycerides present in coconut oil, produce ~10% less energy (8.3 kcal/g) compared to the long chain triglycerides (LCT) (Enig, 1990). MCT are thus easily digestible, oxidized faster and absorbed faster than LCT. It can enter into the liver mitochondria without the carrier carnitine. MCT are deposited less in adipose tissues, decrease protein catabolism in hyper-catabolic states, increase thyroid function and do not form cholesterol esters.

**Fatty acid profiles in other vegetable oils:** In this study, the use of gingelly oil was correlated with the lowest serum cholesterol levels, followed by sunflower oil, coconut oil and groundnut oil. Gingelly oil is mainly composed of the polyunsaturated fatty acids (PUFA) - equivalent amounts of oleic and linoleic acids. Sunflower oil also contained polyunsaturated, omega-6, high linoleic acid, with lesser amounts of oleic acid. Coconut oil consisted predominantly small to medium-chain fatty acids, and the triglyceride, monolaurin in particular. Groundnut oil is mainly composed of oleic acid. The differences in the fatty acid profile of the oils are expected to influence the mode of its metabolism and assimilation influencing its effects on human health.

**Fatty acid and hypercholesterolemia:** There is considerable disagreement about the relative metabolic activity of saturated fatty acids such as lauric, myristic and palmitic acids. Hegsted *et al.* (1965) suggested that myristic acid was the most hypercholesterolemic in butterfat and coconut oil. However, both myristic and palmitic acids raised LDL cholesterol compared to oleic acid, but myristic acid was slightly more powerful (Zock *et al.*, 1994). Khosla and Hayes (1993) reported that palmitic acid is hypercholesterolemic when the total cholesterol intake is high. Palmitic acid is the major saturated fatty acid in most diets and lauric, myristic and palmitic acid are considered to be the principal hypercholesterolemic fatty acids although they may

differ in their metabolic potency (Denke and Grundy, 1992; Zock *et al.*, 1994).

Sindhurani and Rajamohan (1999) have reported that rats fed with coconut fibre showed a decrease in serum total cholesterol and LDL+VLDL cholesterol and an increase in HDL cholesterol, along with a hypocholesterolemic effect in the aorta and other tissues. Despite increased cholesterogenesis, increased hepatic bile acids, faecal excretion of neutral sterols, bile acids and lower release of lipoproteins into the circulation were also noticed.

**CVD and dietary fats:** Traditionally, the risk of CVD from dietary fats has been estimated from their effects on total serum cholesterol (Keys *et al.*, 1957; Hegsted *et al.*, 1965.) The multiple Risk factor intervention trial (Stamler *et al.*, 1993) indicated that higher serum cholesterol levels were associated with increased risk of CVD. Studies revealed that the amount and composition of dietary fat are primary determinants of serum cholesterol levels and LDL (Keys *et al.*, 1957). The shape of the dose-response curve in the serum cholesterol was highly variable and depend partly upon the nature of the dietary fat, cholesterol intake and also may be due to other dietary constituents (Hopkins, 1992).

Saturated fats such as dairy fats and tropical oils that are high in lauric, myristic and palmitic acids, were considered as the largest risk factors for CVD. This overlooks the fact that the ratio of total

to HDL cholesterol is considered more precisely as a specific marker of CVD than total cholesterol and lipoprotein content (Kinosian *et al.*, 1995; Assman *et al.*, 1996). Several studies have shown that elevated levels of serum cholesterol and LDL, the primary carrier of cholesterol in the serum constitutes a major risk factor for atherosclerosis and coronary heart disease (Mensink and Katan, 1992; Hegsted *et al.*, 1993).

However, Mensink *et al.* (2003) reported that lauric acid increased total cholesterol, though much of its effect was in the production of HDL cholesterol. As a result, oils rich in lauric acid decreases the ratio of total cholesterol to HDL cholesterol. Myristic and palmitic acids had little effects and stearic acid reduced the ratio slightly. Hence, it is not appropriate to assume that coconut oil, with a lauric acid content of 31–47% (Azeez, 2007), promotes CVD based only on its high saturated fatty acid content. High levels of high density lipoproteins (HDL) are strongly associated with reduced risk of CVD (Knuiman *et al.*, 1987; Wilson *et al.*, 1988; Gordon and Rifkind, 1989; Gordon *et al.*, 1989;). Several clinical and epidemiological studies revealed that low HDL-cholesterol is an independent risk factor for developing CVD and there is often an inverse relationship between HDL-cholesterol content and triglyceride concentration (Twomey and Pledger, 2007).

HDL level is determined by both genetic and environmental factors. Serum HDL level can be lowered by smoking, obesity and male hormones, and can be raised by physical activity, consumption of alcohol, saturated fats and cholesterol. However,

**Table 5. Measures of association between serum lipid profile and oil intake**

Association	R	R Squared
Cholesterol*Oil (litres/month)	-0.065	0.004
HDL*Oil (litres/month)	-0.002	0
LDL*Oil (litres/month)	-0.005	0
HDL:LDL*Oil (litres/month)	-0.021	0
Triglycerides*Oil (litres/month)	-0.038	0.001

**Table 6. Type of oil consumed and serum total cholesterol and triglycerides content (mg%)**

Oil	Cholesterol content (mg %)	Triglycerides content (mg %)
Coconut	192.50 + 6.01	154.22 + 10.25
Gingelly	178.40 + 5.53	162.99 + 9.44
Groundnut	215.02 + 10.91	154.41 + 18.63
Sunflower	177.34 + 7.10	167.76 + 12.12

Mean + SE; P = 0.05

**Table 7. Coefficients of physiological/ dietetic variables, against the dependent variable HDL:LDL**

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.
	B	SE			
(Constant)	8.864E-02	0.214		0.415	0.679
Total calories	1.555E-04	0	0.153	3.169	0.002
Total fat	-1.21E-03	0.001	-0.083	-1.754	0.080
Coconuts/week	-7.33E-03	0.005	-0.060	-1.392	0.165
Oil/month	-1.10E-02	0.018	-0.026	-0.622	0.534
BMI	2.961E-03	0.006	0.040	0.476	0.634
Weight	4.640E-04	0.002	0.019	0.212	0.832
Height	5.661E-04	0.001	0.026	0.502	0.616

increases in HDL attributed to saturated fats and cholesterol is outweighed by greater increases in LDL. Studies by Mensink and Katan (1992) and Hegsted *et al.* (1993) indicated that all the three classes of fatty acids tend to elevate HDL levels, with saturated fatty acids being the most potent and linoleic acid is the least influential fatty acid. But, low-fat diets which protect against CVD also lower HDL levels (Denke and Breslow, 1988). The major lipid alterations associated with the progression of CVD include not only increases in total and LDL cholesterol, but also in serum triglycerides, a decrease in HDL cholesterol, as well as compositional changes in HDL and LDL cholesterol. Triglyceride-rich LDL, intermediate density lipoprotein (IDL), and chylomicron remnants are considered atherogenic due to their relative ease of oxidative modification. A triglyceride elevation above 1.7 mmol/l is associated with a compositional change in LDL. These LDL particles are oxidized more rapidly than normal LDL, and cleared less efficiently by normal receptor mediated clearance allowing more retention time in the blood plasma and exposure to the arterial wall (Feher, 2003).

The data correlating the physiological parameters to the quantity of oil consumed revealed the following observations:

- Quantity of oil consumed (litres/month) was positively correlated with waist to hip ratio (0.14).
- Height was positively correlated with weight (0.47) and daily calories intake (0.18), and negatively to waist to hip ratio (-0.25).
- Quantity of oil consumed (litres/month) was positively correlated with fat content in blood (0.09).
- BMI is positively correlated with prandial blood sugar (0.19), serum triglycerides (0.21) and cholesterol content (0.1).

This study found a positive correlation between oil consumption and waist to hip ratio (W:H) ratio and the fat content in blood. Similarly, Doucet *et al.* (1998) found significant positive correlations between dietary energy as total fat and body fatness. However, higher intake of PUFA had no effects on adiposity. A review by Moussavi *et al.*

(2008) has shown that dietary short- and medium-chain fatty acids compared to long-chain fatty acids appear to promote weight loss. Coconut oil is principally composed of monolaurin (over 45%), 15% of small chain fatty acids, and 8-9% of mono unsaturated fatty acid (MUFA) and PUFA (Blackburn *et al.*, 1992; Azeez, 2007). Similarly, MUFA appear to favor weight loss compared to saturated fatty acids.

The study concludes that of the four predominant oils consumed by the subjects, gingelly oil was correlated with the lowest serum cholesterol levels, followed by sunflower oil, coconut oil and groundnut oil. The analysis on the association of oil consumption (litres/month) with lipid profile revealed that except cholesterol content, association was not significant in any other parameters studied. The cholesterol, HDL, LDL, triglycerides contents and the HDL/LDL ratio varied according to type of oil used for consumption. However, the HDL/LDL ratio was negatively correlated with cholesterol content (-0.76) and total calories intake (-0.1). Hence, it is advisable to gradually establish a healthy lifestyle based on a low-fat, varied and balanced diet, combined with regular physical activity to reduce the risk for CVD.

### References

- Assa, A.R., Jean-Louis, K.K., Alexia, P., Jean, N. and Ernest, K. 2010. Physicochemical characteristics of kernel during fruit maturation of four coconut cultivars (*Cocos nucifera* L.). *African J. Biotech.* **9**(14): 2136-2144.
- Assman, G., Schulte, H., von Eckardstein, A. and Huang, Y 1996. High-density lipoprotein cholesterol as a predictor of coronary heart disease risk: The PROCAM experience and pathophysiological implications for reverse cholesterol transport. *Atherosclerosis.* **124**: Suppl, S11-S 20.
- Azeez, S. 2007. Fatty acid profile of coconut oil in relation to nut maturity and season in selected cultivars/hybrids. *British Fd J.* **109**(4): 272-279.
- Blackburn, G.L., Kater, G., Mascioli, E.A., Kowalchuk, M., Babayan, V.K. and Bistrrian, B.R. 1992. A reevaluation of coconut oil's effect on serum cholesterol and atherosclerosis. *Philippine J. Coconut Studies.* **17**(2): 21-28.
- Child, R. 1974. Coconuts. Longman Group, London, pp. 169.
- Denke, M.A. and Breslow, J.L. 1988. Effects of a low fat diet with and without intermittent saturated fat and cholesterol ingestion on plasma lipid, lipoprotein and

- apolipoprotein levels in normal volunteers. *J. Lipid Res.* **29**: 963-970.
- Denke, M.A. and Grundy, S.M. 1992. Comparison of effects of lauric acid and palmitic acid on plasma lipids and lipoproteins. *Am. J. Clin. Nutr.* **56**: 895-898.
- Doucet, E., Almeras, N., White, M.D., Despre's, J-P., Bouchard, C. and Tremblay, A. 1998. Dietary fat composition and human adiposity. *Eur. J. Clin. Nutr.* **52**(1): 2-6.
- Enig, M.G. 1990. Fats and oils: understanding the functions and properties of partially hydrogenated fats and oils and their relationship to und hydrogenated fats and oils. *Philippine J. Coconut Studies.* **15**(1): 27-31.
- Feher, M.D. 2003. Lipid lowering to delay the progression of coronary artery disease. *Heart.* **89**(4): 451-458.
- Gordon, D.J. and Rifkind, B.M. 1989. High density lipoprotein - the clinical implications of recent studies. *New England J. Med.* **321**: 1311-1316.
- Gordon, D.J., Probstfield, J.L., Garrison, R.J., Neaton, J.D., Castelli, W.P., Knoke, J.D., Jacobs, D.R. Jr., Bangdiwala, S. and Tyroler, H.A. 1989. High-density lipoprotein cholesterol and cardiovascular disease. Four prospective American studies. *Circulation.* **79**(1): 8-15.
- Hegsted, D.M., Ausman, L.M., Johnson, J.A. and Dallal, G.E. 1993. Dietary fat and serum lipids: an evaluation of the experimental data. *Am. J. Clin. Nutr.* **57**: 875-883.
- Hegsted, D.M., McGandy, R.B., Myers, M.L. and Stare, F.J. 1965. Quantitative effects of dietary fat on serum cholesterol in man. *Am. J. Clin. Nutr.* **17**: 281-295.
- Hopkins, P.N. 1992. Effects of dietary cholesterol on serum cholesterol: a meta-analysis and review. *Am. J. Clin. Nutr.* **55**: 1060-1070. <http://www.fao.org/>, <http://www.webmd.com>
- Kaunitz, H. and Dayrit, C.S. 1992. Coconut oil consumption and coronary heart disease. *Philippine J. Coconut Studies.* **17**(2): 18-20.
- Keys, A., Anderson, J.T. and Grande, F. 1957. Prediction of serum-cholesterol response of man to changes in fats in the diet. *Lancet.* **2**: 959-966.
- Khosla, P. and Hayes, K.C. 1993. Dietary palmitic acid raises plasma LDL and cholesterol relative to oleic acid only at a high intake of cholesterol. *Biochim. et Biophys. Acta.* **1210**: 13-22.
- Kinosian, B., Glick, H., Preiss, L. and Puder, K.L. 1995. Cholesterol and coronary heart disease predicting risks in men by changes in levels and ratios. *J. Invest. Med.* **43**: 443-450.
- Kintanar, Q.L. 1988. Is coconut oil hypercholesterolemic and atherogenic? A focused review of the literature. *Transactions of the National Academy of Science and Technology (Philippines).* **10**: 371-414.
- Knuiman, I.T., West, C.E., Katan, M.B. and Hautvast, J.G.A.J. 1987 Total cholesterol and high density lipoprotein cholesterol levels in populations differing in fat and carbohydrate intake. *Arteriosclerosis.* **7**: 612-619.
- Marina, A.M., Che Man, Y.B. and Amin, I. 2009. Virgin coconut oil: emerging functional food oil. *Trends Fd. Sci. Tech.* **20**(10): 481-487.
- McNamara, D.J. 2010. Palm oil and health: A case of manipulated perception and misuse of science. *J. Am. College Nut.* **29** (3 Suppl): 240S-244S.
- Mensink, R.P. and Katan, M.B. 1992. Effects of dietary fatty acids on serum lipids and lipoproteins: A meta-analysis of 27 trials. *Arteriosclerosis Thrombosis.* **12**: 911-919.
- Mensink, R.P., Zock, P.L., Kester, A.D.M. and Katan, M.B. 2003. Effects of dietary fatty acids and carbohydrate on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. *Am. J. Clin. Nutr.* **77**: 1146-1155.
- Moussavi, N., Gavino, V. and Receveu, O. 2008. Could the quality of dietary fat, and not just its quantity, be related to risk of obesity? *Obesity.* **16**: 7-15.
- Murray, R.K. 1990. Biochemistry of disease. In Harper's Biochemistry, XII edition, RK Murray, PA Mayes, DK Granner and VW Rodwell (eds), Appleton and Lange, Connecticut, USA, p 679-690.
- Sindhurani, J.A. and Rajamohan, T. 1999. Effect of dietary fiber from coconut kernel on cholesterol metabolism. *Indian Coconut J.* 12-16.
- Stamler, J., Stamler, R., Brown, W.V., Gotto, A.M., Greenland, P., Grundy, S., Hegsted, M., Luepker, R.V., Neaton, J.D., Steinberg, D., Stone, N., Van Horn, L. and Wissler, R.W. 1993. Serum cholesterol: doing the right thing. *Circulation.* **88**: 1954-1960.
- Tarrago-Trani, M.T., Phillips, K.M., Lemar, L.E. and Holden, J.M. 2006. New and existing oils and fats used in products with reduced trans-fatty acid content. *J. Am. Dietetic Assoc.* **106**(6): 867-880.
- Thomas, A. 2002. Fats and Fatty Oils. *Ullmann's Encyclopedia of Industrial Chemistry.* Weinheim: Wiley-VCH.
- Twomey, P.J. and Pledger, D.R. 2007. HDL-cholesterol and triglycerides: an overlooked issue? *J. Clin. Path.* **60**: 1065-1066.
- USDA National Nutrient Database for Standard Reference. Nutrient Data Laboratory, Agricultural Research Service, United States Department of Agriculture. <http://www.nal.usda.gov/fnic/foodcomp/search/>.
- Wilson, P.W.F., Abbott, B.M. and Castelli, W.P. 1988. High density lipoprotein cholesterol and mortality. *Arteriosclerosis.* **8**: 737-741.
- Zock, P.L., de Vries, J.H. and Katan, M.B. 1994. Impact of myristic acid versus palmitic acid on serum lipid and lipoprotein levels in healthy women and men. *Arteriosclerosis, Thrombosis Vascular Biol.* **14**: 567-575.