Tropical Agricultural Research Vol. 17: 20 - 30 (2005)

Association of Coconut Fat Intake and Cardiovascular Disease Risk Factors of Healthy Adults

P.W.M.L.H.K. Marambe, K.D.R.R. Silva[1] and R. Sivakanesan[2]

Department of Applied Nutrition Faculty of Livestock Fisheries and Nutrition
Wayamba University of Sri Lanka, Makandura
Gonawila, Sri Lanka

ABSTRACT. *Coconut fat (CF), which is rich in saturated fatty acids (SFA), is the major source of fat in the Sri Lankan diet. The association between CF and cardiovascular disease (CVD) risk factors is debatable. This cross-sectional study was conducted to determine the association between CF intake and CVD risk of healthy adults living in a coconut growing area. Randomly selected 303 healthy adults, aged 25-55 years were the participants. A range of biological and anthropometric risk markers of CVD and dietary intakes were determined. Mean CF intake of the subjects was 37.6 ± 18.9 g/d (58.9% of the total fat consumption). Subjects who consumed ≤ 16% energy from CF (low CF) and > 16% energy from CF (high CF) were compared for a range of risk factors. Subjects in the high CF group had significantly higher HDL-Cholesterol (HDL-C) levels, lower total cholesterol: HDL-C and waist: height ratio (WhtR) compared to subjects in low CF group. The subjects that consumed a high amount of CF were less likely to have a low HDL-C (Odds Ratio: OR=0.39, CI= 0.22-0.66) compared to the group that consumed a low amount of CF. In females, percentage SFA from CF and percentage energy from CF were significant predictors of HDL-C (β = 0.16, P = 0.03) and WhtR (β = -0.02, P = 0.001), respectively. CF did not show any unfavorable association with any of the risk markers investigated. In conclusion, this study indicates that CF intake has cardio-protective type of associations by decreasing the prevalence of low HDL-C.*

INTRODUCTION

The incidence of cardiovascular disease (CVD) is increasing in many developing countries and is the leading cause of hospital deaths among adults in Sri Lanka (Ministry of Health, 2000). The recent estimates for mortality from CVD for Sri Lanka (524 deaths per 100,000) is higher than that observed in many western industrial nations (Abeywardena, 2003). Among the different hypothesis put forward to explain the relatively high incidence of CVD in Sri Lanka is the high saturated fatty acid (SFA) content (70 - 80% of the total fat) of the habitual Sri Lankan diet (Mendis *et al.*, 2001).

The major source of fat in the Sri Lankan diet is coconut fat (CF), which contains about 92% saturated fats. Of the total fatty acids in CF, 63.3% are saturated medium chain

1 Department of Applied Nutrition, Faculty of Livestock Fisheries and Nutrition, Wayamba University of Sri Lanka, Makandura, Gonawila, Sri Lanka.

2 Department of Biochemistry, Faculty of Medicine, University of Peradeniya, Peradeniya, Sri Lanka.

fatty acids (MCFA) (C8:0 - C12:0) of which 48.3% is lauric acid (C12:0) (Chempakan, 1992). The saturated nature of CF and its deficiency in monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA) (Raheja and Bhoraskar, 1999), explains the reasons for intake of excess saturates and less unsaturates by Sri Lankans (Mendis *et al.*, 2001; Alahakoon and Silva, 2003). For many years there has been controversy whether CF contribute to the high prevalence of CVD in Sri Lankans. There are several experimental and epidemiological studies conducted to study the effect of CF on CVD risk of humans (Dayrit, 2003). However, findings of such studies are inconsistent as some of the studies showed beneficial effects whereas some others showed adverse effects of CF on CVD risk. Low serum cholesterol levels and low incidence of coronary heart disease (CHD) among high coconut consuming Polynesians (Prior *et al.*, 1981), Nicobar islanders (Chempakan, 1992) and Filipinos of the Bicol region (Dayrit, 2003) showed that CF intake does not have an unfavorable impact on CVD risk. Consumption of coconut is not a predictor for CHD in high coconut consumers in Indonesia (Lipoeto *et al.*, 2004).

Several animal and human studies suggest atherogenic effect of CF (Dayrit, 2003). Replacement of CF with a mixture of corn oil and soya oil in Sri Lankans reduced the total cholesterol (TC) level and low-density lipoprotein cholesterol (LDL-C) of the subjects (Mendis *et al.*, 1989; Mendis and Kumarasunderam, 1990). However, this has also shown a decrease in plasma high-density lipoprotein cholesterol (HDL-C) concentration. Partial replacement of CF with a mixture of soya bean oil and sesame oil had shown improved serum lipid profile of human volunteers (Mendis *et al.*, 2001). It has also been argued that CF has no significant effect on blood cholesterol because MCFA are metabolized rapidly. Since the effect of CF on CVD risk is controversial, further studies to investigate association between CF consumption and CVD risk will be of special relevance to Sri Lankans and other populations who consume coconut as the major source of dietary fat. Nutritional benefit of coconut, if any, will have indirect implications to boost the national economy of Sri Lanka, as coconut is an important plantation crop in the country. It will also aid in determining future food policy with regard to fat consumption in the general population. Therefore, the present cross-sectional study was performed to investigate the epidemiological association between CF intake and CVD risk factors in healthy adults.

MATERIALS AND METHODS

Study design: The study was carried out as a cross-sectional study.

Subjects: Subjects consisted of males and females aged 25-55 years, with no obvious health problems, living in two Grama Niladari (GN) divisions in Kuliyapitiya divisional secretariat, namely, Meegahakotuwa and Dandagamuwa. This area, which belongs to the coconut triangle, was considered as an ideal setting for conducting the present study due to high availability of coconuts.

A systematic random sample of subjects was obtained from the electoral lists. Those who had previously diagnosed CVD, diabetes, renal diseases and other health problems, using medications known to affect blood lipids and glucose were excluded. Written informed consent was obtained from all the participants. This study protocol was approved by the Ethical Review Committee of the Faculty of Medicine, University of Peradeniya.

Data collection

Socio demography and lifestyle

Information on socio-economic status, alcohol consumption, smoking and physical activity of subjects was obtained using a pre-tested interviewer administered questionnaire.

Anthropometry and blood pressure

Height (Stadiometer; Doherty, UK), body weight (Beam balance; Seca, Germany), waist circumference (WC) and hip circumference (HC) (measuring tapes) were measured using standard procedures. Blood pressure was measured using a digital sphygmomanometer (Omron, UK) on the right arm with the subject in the sitting position after 5 min of rest. All measurements were obtained at the Human Investigation unit of the Department of Applied Nutrition, Wayamba University of Sri Lanka. Body mass index (BMI; weight in kg divided by square of height in meters), waist-to-hip ratio (WhpR) and waist-to-height ratio (WhtR) were calculated.

Blood collection and serum measurements

Blood samples (3 ml each) were drawn by venepuncture after 12 hours fasting and were centrifuged at 3000 rpm for 10 minutes for serum separation. A total of 292 subjects gave fasting blood samples. The separation of HDL fractions in the serum was carried out within 3 hours of collection of blood using Polyethylene glycol as the reagent (Lunberg *et al.* 1984). The serum was stored at -20°C until assayed. TC, HDL-C and triacylglycerol (TAG) and glucose were determined by commercial enzymatic kits (Randox Laboratory Ltd., UK and Ecoline, India) using chemistry analyzer (Clin-Check Plus, Italy) at 546 nm. The LDL-C was calculated by the Friedwarld formula (Friedwarld, 1972).

Dietary intake assessment

The daily intake of energy, CF and other macronutrients by the subjects was assessed using two 24 hour dietary recalls conducted on two non consecutive days (including a week day and a week end). A recipe survey was conducted to determine the amount of CF added to home-made foods and to identify the daily intake of these ingredients by the study population and also to determine the compositions of the local mixed food dishes. Briefly, the individuals who cook meals for families to which the subjects belong to were interviewed on the same day that 24 hour recall was done to get information on recipes of different foods and beverages that the subjects had consumed. The amounts of ingredients used to prepare the dishes were determined by measuring the volume of ingredients using standard measuring devices (measuring cylinders and beakers). Diagrams of food models were used in determining portion sizes of foods and beverages. The weight of ingredients used to prepare the cooked products was calculated by weighing the volumes and portion sizes of the ingredients. The food intake data and the recipes were entered into the Foodbase 2000 computer software (Institute of Brain Chemistry, UK) to determine the nutrient intake. Mean daily intake of CF was calculated using the 24 hours dietary recall based on recipe survey data.

Data and statistical analysis

Subjects were divided into two groups based on the median percentage of daily energy intakes of CF/d for the comparison. Means of anthropometric indices, blood pressure, serum lipids and glucose concentrations of the two categories of CF intake were compared using independent two-sample t-test for normally distributed variables and Mann Whitney U-test for skewed variables. Association between the prevalence of CVD risk factors and CF intake was investigated using chi-square test. Logistic regression analysis was employed to calculate the odds ratios. Stepwise multiple linear regression analysis was performed to establish the independent association of CF and anthropometric indices, blood pressure, serum lipids and glucose concentrations. Statistical analysis was performed using SPSS computer program, version 10.00. Statistical significance was defined as $P< 0.05$.

RESULTS

General characteristics

A total of 303 subjects (males: 145 and females: 158; age: 40.5 ± 9.2 years) consented to the present study. Majority (96.1%) were physically active and only 3.9% were sedentary. Of the males 37% and 42% were smokers and alcohol consumers, respectively. All the participants were non-vegetarians. The average family size of the study population was 4.

The mean anthropometric indices, blood pressure values, serum lipids and glucose in the study population were below the cut off for CVD risk. Despite the favorable mean risk factor levels and physically active lifestyle, the study population had considerably high prevalence of CVD risk factors. The most prevalent risk factor in the study population was elevated TC: HDL-C (76.1%) (Table 1).

Coconut consumption

The average household consumption of coconut was 1.5 coconuts/day. Table 2 shows the intake of CF by the study population. The mean daily intake of CF was 37.6 ± 18.9 g/day. The major source of dietary fat consumed by the subjects was CF (59% of the total fat intake). Majority of the total SFA intake was also from CF (78.4 ± 12.3%). The majority of CF consumed was from coconut milk, followed by scraped coconut and coconut oil (results not shown in the Table).

Table 1. Mean (± SD) anthropometric indices, blood pressure values, serum lipids, glucose and prevalence of CVD risk factors in the study population

	Mean ± SD	CVD cutoff	Prevalence %
Body Mass Index (kg/m^2)	23.16 ± 3.80	>25 kgm^{-2}	31.3 (95)
Waist circumference (cm)	79.27 ± 9.68	Males>94 cm Females>88cm	17.4 (53)
Waist: hip ratio	0.86 ± 0.07	Males>0.9 Females>0.85	41.1 (125)
Waist: height ratio	0.49 ± 0.06	>0.6	2.3 (7)
SBP (mmHg)	120.5 ± 15.89	>140/90 mmHg	12.5 (38)
DBP (mmHg)	77.8 ± 9.37		
TC (mmol/L)	4.87 ± 1.02	>5.18 mmol/l	9.3 (27)
HDL-C (mmol/L)	0.98 ± 30	<0.9 mmol/l	35.8 (105)
LDL-C (mmol/L)	3.25 ± 0.99	>3.36 mmol/l	42.1 (128)
TAG (mmol/L)	1.44 ± 0.74	>2.3 mmol/l	46.4 (136)
Glucose (mmol/L)	5.33 ± 1.25	>6.1 mmol/l	11.3 (33)
TC: HDL –C	5.50 ± 2.09	>4	76.1 (223)

SBP= Systolic blood pressure, DBP= Diastolic blood pressure, numbers in parenthesis represent sample size.

Table 2. Mean (± SD) coconut fat intake of the study population

	Mean ± SD
Coconut fat (g/day)	37.6 ± 18.9
Coconut fat as a % of the total fat consumed/d	58.9 ± 14.6
SFA of coconut fat as a % of the total SFA consumed/d.	78.4 ± 12.3
% total energy from CF/day	15.9 ± 5.4

Comparison of high and low CF groups

The subjects grouped based on their daily CF intake as >16% energy from CF/d (High CF group) and ≤16% energy from CF/d (Low CF group) were compared for the mean energy and macro nutrient intake (Table 3). High CF group had significantly higher fat and SFA intake and significantly lower amount and % energy from carbohydrate, protein, P:S ratio and cholesterol intake compared to low CF group.

Subjects in high CF group had significantly lower ($P<0.05$) WhtR higher ($P<0.05$) HDL-C and lower ($P<0.05$) TC: HDL-C than those in low CF group (Table 4). There was a significantly higher ($P<0.05$) prevalence of low HDL-C among subjects in low CF group compared to those in high CF group (Table 5).

Table 3. Mean (± SD) daily energy and macronutrient intake

	Total group	Low CF group	High CF group	P*
Energy (kcal/d)	2130.0 ± 630.3	2126.7 ± 557.7	2133.9 ± 710.6	0.91[1]
Carbohydrate (g/d)	346.8 ± 100.8	358.9 ± 96.2	332.2 ± 104.7	0.02[1]
Protein (g/d)	58.0 ± 23.7	60.0 ± 24.3	55.6 ± 22.7	0.08[2]
Fat (g/d)	64.1 ± 25.9	57.6 ± 21.1	71.8 ± 28.9	<0.001[1]
Total SFA (g/d)	40.5 ± 17.5	33.3 ± 11.5	49.2 ± 19.5	<0.001[1]
Total PUFA (g/d)	4.8 ± 2.6	4.9 ± 2.8	4.6 ± 2.4	0.34[2]
Total MUFA (g/d)	7.6 ± 4.6	8.5 ± 5.2	6.9 ± 3.5	0.13[2]
P: S	0.1 ± 0.1	0.2 ± 0.1	0.1 ± 0.03	<0.001[1]
Cholesterol (mg/d)	91.5 ± 86.0	100.9 ± 83.8	80.0 ± 87.5	0.001[2]
% of total energy from				
Carbohydrate	61.3 ± 6.2	63.5 ± 6.4	58.7 ± 4.6	<0.001[1]
Protein	10.9 ± 2.5	11.3 ± 2.8	10.4 ± 1.9	0.004[2]
Fat	26.8 ± 5.7	24.1 ± 5.4	30.0 ± 4.1	<0.001[1]
SFA	16.9 ± 4.5	13.9 ± 3.1	20.5 ± 3.3	<0.001[1]
PUFA	2.0 ± 0.9	2.1 ± 0.9	1.9 ± 0.7	0.19[2]
MUFA	3.2 ± 1.6	3.4 ± 1.1	2.9 ± 1.1	0.15[2]

P: S= Polyunsaturated-to saturated ratio; *Comparison of CF groups using [1]independent samples t test and [2] Mann-Whitney U test

Table 4. Mean (± SD) anthropometric indices, blood pressure values, serum lipids and glucose in relation to coconut fat intake

Measurement	Low CF group	High CF group	P*
BMI (kg m^{-2})	23.4 ± 3.9	22.9 ± 3.8	0.20[1]
WC (cm)	80.2 ± 9.2	78.2 ± 10.1	0.07[1]
WhpR	0.9 ± 0.1	0.9 ± 0.1	0.16[1]
WhtR	0.5 ± 0.1	0.4 ± 0.1	0.04[1]
SBP (mmHg)	20.9 ± 12.4	120.2 ± 19.3	0.22[1]
DBP (mmHg)	78.5 ± 9.1	77.2 ± 9.6	0.72[1]
TC (mmol/L)	4.9 ± 1.1	4.9 ± 0.9	0.80[1]
LDL-C (mmol/L)	3.3 ± 1.0	3.3 ± 0.9	0.86[1]
HDL-C (mmol/L)	0.9 ± 0.3	1.0 ± 0.3	0.004[2]
TAG (mmol/L)	1.5 ± 0.8	1.4 ± 0.7	0.08[2]
TC:HDL-C	5.7 ± 2.2	5.2 ± 1.9	0.04[2]
Glucose (mmol/L)	5.2 ± 1.1	5.1 ± 1.3	0.07[2]

Number of subjects for anthropometry and blood pressure; Low CF group= 166; High CF group=137; Number of subjects for biological risk factors Low CF group= 160; High CF group =132; *Comparison of two CF groups using chi square test

Table 5. Prevalence of major anthropometric and biological CVD risk factors in relation to coconut fat intake

CVD risk factor	Low CF group % (n)	High CF group % (*n*)	Pearson χ^2	P*
BMI >25kg m^{-2}	34.3 (57)	27.7 (38)	1.77	0.62
WC: Males>94 cm Females>88 cm	12.7 (21)	8.0 (11)	1.69	0.19
WhpR: Males>0.9 Females>0.85	45.8 (76)	35.8 (49)	3.11	0.08
WhtR >0.6	3.0 (5)	2.2 (3)	0.19	0.66
Blood pressure >140/90 mmHg	12.7 (21)	12.4 (17)	0.004	0.95
TC >5.18 mmol/L	35.6 (57)	36.4 (48)	0.02	0.89
HDL-C <0.9 mmol/L	54.4 (87)	37.1 (49)	8.65	0.003
LDL-C >3.36 mmol/L	45.0 (72)	42.4 (56)	0.19	0.66
TC:HDL-C >4.00	78.8 (126)	73.5 (97)	1.11	0.29
TAG >2.3 mmol/L	12.5 (20)	9.8 (13)	0.51	0.48
Glucose >6.1 mmol/L	11.3 (18)	6.8 (9)	1.74	0.19

Number of subjects for anthropometry and blood pressure; Low CF group= 166; High CF group=137; Number of subjects for biological risk factors Low CF group= 160; High CF group =132; *Comparison of two CF groups using chi square test

Odds ratios for the prevalence of biochemical CVD risk factors for the high level of CF intake versus the low level of CF intake are presented in Table 6. Odds ratios were calculated after adjusting for variables which were significantly associated with the CVD risk factors. High CF consumption was negatively associated with prevalence of low HDL-C. There was no significant association between CF intake and prevalence of other estimated CVD risk factors.

Table 6. Adjusted odds ratios (OR) and confidence intervals (CI) for prevalence of biochemical CVD risk factors in high CF group

Biochemical risk factor	OR (95% CI)
TC>5.18 mmol/L[1]	0.93 (0.44-2.07)
HDL-C <0.9 mmol/L[2]	0.39 (0.23-0.66)
LDL-C>3.36 mmol/L[3]	0.76 (0.36-1.59)
TAG>2.3 mmol/L[4]	2.42 (0.61-9.64)
TC: HDL-C>4.0 mmol/L[5]	0.60 (0.28-1.29)
Glucose>6.1 mmol/L[6]	1.10 (0.22-5.63)

[1]Adjusted for age, waist, , BMI, energy and fat intake ; [2] Adjusted for age, BMI, fat intake; [3]Adjusted for age, waist circumference, BMI and fat intake ;[4]Adjusted for WhpR, family history of heart disease; [5] Adjusted for WhpR, age, fat intake, percentage energy from SFA; [6] Adjusted for smoking and alcohol intake

Table 7 summarizes the results of stepwise multiple linear regression analysis (only the models in which CF consumption was found to be a significant independent predictor are given in the Table). In females, % fat intake from CF was found to be an independent negative predictor (β=-0.02, P=0.01) of WhtR. The % SFA from CF/d was a significant positive predictor (β=0.16, P=0.03) of HDL-C in females. CF was not a predictor of other anthropometric indices and serum parameters in males, females or the total group.

Table 7. Relationship of CF intake with anthropometric indices and serum lipids by sex as determined by multiple linear regression analysis

		Males			Females	
Dependent variable	**Model R^2**	**Predictor variables**	**β**	**Model R^2**	**Predictor variables**	**β**
WhtR	0.13	Age	0.30	0.06	% energy from CF	-0.02
		Energy	0.19		Age	0.17
		% energy from PUFA	0.17			
		Smoking	0.18			
HDL-C	0.16	Waist	-0.36	0.17	Waist	-0.35
		Smoking	0.18		% SFA from CF	0.16
					Age	0.16

DISCUSSION

Overall, the cross-sectional design of this study does not allow causal conclusions to be drawn. However, when adjusted for potential confounding factors an inverse association was found between CF intake and CVD risk. High CF group had a high HDL-C. The results further showed that CF intake is a significant independent predictor of HDL-C and WhtR in females.

CF consumption by the subjects reported in the present study is much greater than the reported CF consumption of Sri Lankans (Wikramanayake, 1996; Gunewardana, 1997). The present study was carried out in an area that belongs to the coconut triangle, where coconut is highly available and this might be a reason for high CF consumption by the subjects. Despite a diet high in SFA (16.9% energy/day) and low in P:S ratio (0.1) the mean anthropometric indices, blood pressure and concentrations of serum lipids of the study population were below the cut off levels for CVD risk. The variation in the study group, especially the physically active lifestyle may be a contributory factor towards this. Serum cholesterol has no major effect on the occurrence of CHD if the population mean is 4.5 mmol/l or less (Schaefer, 2002). The mean TC level of the present study is slightly above that level. Although the mean values are generally acceptable, the prevalence of CVD risk factors in the study population was considerably high.

The causative factors or associated factors are important to be investigated. It has been suggested that the dietary SFA intake should be <7% energy per day to reduce CHD risk (Schaefer, 2002). However, only 1% of the subjects in the study population obtained <7% energy from SFA (results not shown). If the high prevalence of CVD risk factors is attributed to high SFA intake of the subjects then it is arguable that CF, which is the major source of SFA, has a contribution to increase CVD risk in the study group. In contrast, high CF group who consumed significantly higher fat, SFA and lower carbohydrate, protein and P: S than the low CF group, had a favorable serum lipid profile with significantly higher HDL-C and lower TC:HDL-C than the latter. This finding is in agreement with Katan (1998) who reported that diets low in fats and high in carbohydrates decrease HDL-C. The mean TC and LDL-C levels of subjects in high CF group were not significantly different from that of subjects in low CF group. This finding strengthens the already suggested beneficial effects of SFA in CF on serum lipids, specifically raising HDL-C, without raising TC and LDL-C. Logistic regression analysis confirmed that those who consume high level of CF are less likely to have low HDL-C (OR=0.39, CI= 0.23-0.66).

Multiple linear regression analysis further confirmed that percentage SFA from CF is a significant positive (β = -0.16, P = 0.03) determinant of HDL-C, specifically in females. None of the other CVD risk factors investigated showed any adverse associations with CF consumption. These findings indicate CF has a cardio- protective effect at least through increasing HDL-C. Data from metabolic studies have shown that SFA 12:0-16:0, in particular 12:0 and 14:0 (Lauric and Myristic) are far more HDL-C increasing than MUFA or PUFA (Muller *et al.*, 2003). The beneficial effects of CF consumption on HDL-C may be therefore due to lauric and myristic acids in CF. The inverse relationship observed between WhtR in females and CF intake may be due to the presence of MCFA in CF. Both human and animal studies have indicated that MCFA of 8-12 C are preferentially oxidized compared to long chain SFA composed of 14 or more C atoms and thereby reduces adipose tissue deposition and increase adipose tissue mobilization (Papamandjaris *et al.* 2000).

Other epidemiological studies have also found that CF intake does not increasing CVD risk. It was found that vascular diseases were uncommon in Polynesians who consume higher level of CF (Prior *et al.*, 1981). Case control studies conducted in Kerala and Indonesia using CHD patients and healthy adults found no specific role of coconut in the causation of CHD (Kumar, 1999; Lipoeto *et al.*, 2004). These findings together with the findings of the present study support the cardio protective effect of CF.

CONCLUSIONS

The present study has demonstrated that CF does not raise CVD risk whereas high CF intake has cardio protective associations with certain risk factors, specifically with HDL-C. The findings of the present study will be useful in planning further experimental studies on CF and CVD risk.

REFERENCES

Abeywardena, M.Y. (2003). Dietary fats, carbohydrates and vascular disease: Sri Lankan perspectives. Atherosclerosis. 171 (2): 157 - 161.

Alahakoon, C.P. and Silva, K.D.R.R. (2003). High saturated and low unsaturated dietary fat intakes and high prevalence of risk factors of coronary heart disease among urban middle aged office workers in Sri Lanka. Proc. Nutr. Soc. 62 (OCA/B). 91A.

Chempakan, B. (1992). Health hazards of coconut oil-A myth or reality? CORD. VIII (2): 18 - 23.

Dayrit, C.S. (2003). Coconut oil: Atherogenic or not? Phili. J. Cardiol. 31 (3): 97 -104.

Friedwald, W.T., Levy, R.I. and Fredrickson, D.S. (1972). Estimation of the concentration of low density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. Clin. Chem. 18: 499 - 502.

Gunawardena, D.G.R. (1997). The nutritional role of coconut oil in the pathogenesis of ischaemic heart disease in Sri Lanka. Coconut Development Authority. Sri Lanka. pp. 17 - 24.

Katan, M.B. (1998). Effect of low fat diets on plasma high density lipoprotein concentrations. Asia Pac. J. Clin. Nutr. 67 (3): 573S - 576S.

Kumar, P.D. (1999). The role of coconut and coconut oil in coronary heart disease in Kerala, South India,. Trop. Doct. 27 (4): 215 - 217.

Lipoeto, N.I., Agus, Z., Oenzil, F., Whalqvist, M. and Wattanapenpaiboon, N. (2004). Dietary intake and the risk of coronary heart disease among the coconut consuming Minangkabu in west Sumatra, Indonesia. Asia Pac. J. Clin. Nutr. 13 (4): 377 - 384.

Lunberg, B., Hogstorm, S., Pietilainen, P., Suominer, M. and Vilkary, J. (1984). Separation of plasma high density lipoprotein by precipitation method using polyethyleneglycol 6000. Scand. J. Clin. and Lab. Investig. 44: 305 -309.

Mendis, S. and Kumarasunderam, R. (1990). The effect of daily consumption of coconut fat and soya bean fat on plasma lipids and lipoproteins of young normolipidaemic men. Br. J. Nutr. 63: 547 - 552.

Mendis, S., Samarajeewa, U. and Thatil, R.O. (2001). Coconut fat and serum lipoproteins: effects of partial replacement with unsaturated fats. Br. J. Nutr. 85: 583 - 589.

Mendis, S., Weissler, R.W. and Bridenstein, R.T. (1989). The effects of replacing coconut oil with corn oil on human serum lipid profiles and platelet derived factors active in atherogenesis. Nutrition Reports International. 40: 773 - 782

Ministry of Health (2000). Ann. Health Bull.2000. Department of Health Services, Sri Lanka.

Muller, H., Lindman, A.S., Blomfeldt, A., Seljeflot, I. and Pedersen, J. I. (2003). A diet rich in coconut oil reduces diurnal postprandial variations in circulating tissue plasminogen activator antigen and fasting lipoprotein (a) compared with a diet rich in unsaturated fat in women. J. Nutr. 133 (11): 3422 - 3427.

Papamandjaris, A.A., White, M.D., Raeini-Sarjaz, M. and Jones, P.J.H. (2000). Endogenous fat oxidation during medium chain versus long chain triglyceride feeding in healthy women. Int. J. Obes. 24: 1158 - 1166.

Prior, I.A., Davidson, F., Salmond, C.E. and Czochanska, Z. (1981). Cholesterol, coconuts and diet on Polynesian atrolls: a natural experiment: the Pukapuka and Tokelau island studies. Am. J. Clin. Nutr. 34 (8): 1552 -1561.

Raheja, B.S. and Bhoraskar, A.S. (1999). Health benefits of coconut oil. Ind. Coc. J. 11: 7 - 11.

Schaefer, E. (2002). Lipoproteins nutrition and heart disease. Am. J. Clin. Nutr. 75: 191 - 212.

Wikramanayake, T.W. (1996). Food and nutrition, Hector Kobbekaduwa Agrarian Research Institute, Colombo.