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Effect of different forms of coconut on the lipid profile in normal free-living healthy subjects: A randomized controlled trial (Phase II)

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ABSTRACT

Background: It has been postulated that the lipid effects of coconut could be mediated by its fatty acids, fiber and lysine/arginine ratio. Hence, the lipid effects of coconut oil could be different from the effects of the kernel flakes or milk extract because the constituents could be different in each coconut preparation. The present research investigated the lipid effects of different modes of coconut used in food preparation.

Methods: This study involved a total of 190 participants, randomized into four groups, which received coconut oil supplement (30 ml) ($n = 53$), kernel flakes (30 g) ($n = 52$) or coconut milk powder (30 g) ($n = 44$) for a period of 8 weeks. The control group ($n = 41$) received no supplement. Lipid assays were performed at baseline and at the end of the 4th and 8th weeks. The generalized estimating equations (GEE), ANOVA, and paired and independent *t*-tests were used in the analysis.

Result: The age range of the participants was 25–60 years, and 52.6% of them ($n = 100$) were men. Coconut milk supplementation induced beneficial changes in the lipid profile in that the LDL and non-HDL levels decreased while the HDL levels increased. The subgroup whose baseline LDL level was elevated appeared to benefit most from coconut milk supplementation. Coconut oil and kernel flakes failed to induce favorable lipid changes comparable to coconut milk supplementation.

Conclusion: Differing concentrations of protein, fat and fiber in coconut preparations could possibly explain the dissimilar effects on the lipid profile caused by the different coconut preparations. The benefits of coconut milk seen in the high basal LDL subgroup warrant a detailed study.

Introduction

The contribution of dietary fat intake to the atherosclerotic process is currently generating much debate [1–11]. The traditional view that saturated fat is atherogenic appears to be a contentious position, although it has been held since the initial studies of Ancel Keys. A recent meta-analysis by Puaschitz et al. [12] failed to establish an association between dietary saturated fat and ischemic events, including mortality. DiNicolantonio et al. [13] evaluated the evidence to link ischemic heart disease with dietary fat and concluded that sugars are probably more atherogenic than saturated fatty acids.

Harcombe et al. [14] critically examined the studies on which the 1977 and 1983 US and UK dietary guidelines were constructed. They reviewed six dietary trials that included a total of 2467 males. Five of these were secondary prevention trials. They found no beneficial effect of dietary intervention on coronary heart disease or all-cause mortality despite a higher reduction in serum cholesterol levels that occurred with intervention. The study by Mozaffarian and Ludwig [15] among post-menopausal women indicated that a higher saturated fat intake led to a reduced degree of coronary heart disease. The evidence, however, is not consistently negative regarding the benefits of reducing dietary saturated fat intake, as evidenced by a systematic review by Hooper et al. in

Abbrevations: HDL, high-density lipoprotein; LDL, low-density lipoprotein; CH-HDL ratio, cholesterol - high-density lipoprotein ratio.

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2015 [16]. This updated review found, in the author's words, 'a small but potentially important reduction in cardiovascular risk on reduction of saturated fat intake'.

It is relevant to emphasize that foods consumed by the general populace are complex and would not contain fatty acids or any other nutritive agent in isolation, and different saturated and unsaturated fatty acids could possibly give rise to different effects on lipid chemistry. Hence, the clinically relevant effects of saturated fats derived from different origins could well be different.

Coconut fats account for approximately 80% of the fat intake among Sri Lankans, of which 92% consists of saturated fats. Hence, some may postulate that coconut fat may increase the risk of ischemic heart disease among Sri Lankans. However, since saturated fats in coconut are medium-chain fatty acids and do not undergo degradation and re-esterification processes, the hypothesis that coconut fat is bad for health may be questionable [17].

Although coconut oil increases HDL levels, no clear evidence exists to date to claim that coconut oil reduces the risk of atherosclerotic heart disease [18]. While observational evidence suggests that consumption of coconut kernel or squeezed out coconut milk in the context of traditional dietary practices does not lead to adverse cardiovascular outcomes, intervention studies have found that lowering the intake of dietary saturated fat (into which category coconut oil is included) and replacing it with vegetable oils such as olive oil and soybean oil, which have polyunsaturated fats, reduces the risk of developing CVD by approximately 30% [19]. However, one must be cautious about these findings when applied to the general population because there are large differences in dietary and lifestyle patterns in free-living people. Our own work in 2013 showed that coconut milk significantly reduced LDL and raised HDL levels [20]. As coconut milk contains a significant arginine-rich protein content and soluble fiber, the lipid effects may be compounded by these dietary elements as well, and the effect observed on the lipid profile may not be entirely due to the fatty acids.

Coconut is consumed by the vast majority of people in many Asian countries [21]. Desiccated coconut (coconut flakes) is becoming popular in many European countries because of the changes in food preferences in Europe, where there is a rising interest in Asian cooking as a result of the large migration of Asians to Europe and North America. Thus, investigating the relationship between the lipid profile and coconut consumption would be of great value for public health nutrition and prevention of cardiovascular diseases in these populations.

Comparative investigations have not been conducted regarding the effects of different forms of coconut on lipid profiles. The present study was planned to investigate the lipid effects of different forms of coconut, i.e., oil, milk, and flakes, when consumed by free-living healthy subjects.

Materials and methods

Study design

A randomized, placebo-controlled, prospective clinical trial was conducted at the Institute of Medical Research (MRI), Colombo, Sri Lanka. The study protocol was approved by the Ethics Committee of the MRI, Colombo, Sri Lanka (No: 02/2016). Written informed consent was obtained from all subjects before initiation of the study. The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki.

Subjects

Participants were recruited from a local advertisement displayed in the Medical Research Institute, Colombo. Two hundred and five (205) healthy volunteers aged 25–60 years volunteered to participate in the research. A comprehensive medical history was obtained, and physical examination of the subjects was performed before recruitment. The exclusion criteria were as follows: total cholesterol level $\geq$ 250 mg/dl; triglyceride level $\geq$ 300 mg/dl and those who were on any form of medication or nutritional supplementation. After exclusion, a total of 200 subjects (104 men and 96 women) were deemed eligible for the study and all of them were enrolled in the study. In randomization process, first, a list of employees working in this study setting who had given informed consent was developed. Simple Randomization method was employed to select participants for each treatment arm. Thus, for each eligible participant, a random number, out of 1, 2, 3 and 4, was assigned. Those who got number 1 was assigned to coconut oil treatment, number 2 for kernel flakes treatment, 3 for coconut milk treatment and 4 for the control group. The kernel flakes used in the present study was desiccated coconut. By doing so, 54 for coconut oil, 53 for desiccated coconut, 46 for coconut milk and 47 for the control group were selected. Ten participants were excluded from the final analysis because they discontinued participation in the study due to personal reasons before the 4th week of the study. The final sample consisted of 190 participants. The compliance rate was 93%.

Study protocol

The participants were randomly allocated to three dietary supplementation regimens: coconut milk powder 30 g, desiccated coconut 30 g and coconut oil 30 ml and to the control group. The coconut supplements were not equi-caloric and had different fat, fiber, and protein contents. Average dietary intake at baseline was measured using 24-h dietary recall. Each subject was instructed to take the allocated supplement for 8 weeks and was asked to maintain a daily dietary diary to ensure that the diet did not vary significantly (see supplementary material 1). Measurements of body mass index (BMI), lipid profile (consisting of total cholesterol, HDL cholesterol, LDL cholesterol, and triglyceride concentrations), fasting blood sugar (FBS) and HbA1c were performed 3 times: baseline and after 4 weeks and 8 weeks. BMI was measured by a single research assistant, and all laboratory tests were performed by a single technician. Lipid profiles were performed after 10 h of mandatory fasting, and FBS was performed after an 8-h fast. Total cholesterol, HDL-cholesterol and triglyceride concentrations were measured in serum by a *Roch/Hitachi Modular P Chemistry Analyzer* (Mod P). The cholesterol assay used the cholesterol oxidase/peroxidase (CHOD/POD) method. HDL cholesterol was measured by a direct non-precipitating method using polyethylene glycol-coupled cholesteryl esterase (PEG-modified enzyme) and cholesterol oxidase. Triglycerides were analyzed by using the glycerokinase/glycerophosphate oxidase method after initial hydrolysis with lipoprotein lipase. LDL cholesterol was calculated using the *Friedewald formula*. FBS and HbA1c were measured by using *Abbott's Diabetes Care FreeStyle Libre.* In this paper, four main outcome variables were considered; HDL-cholesterol (HDL), LDL- Cholesterol (LDL), non-HDL cholesterol and Cholesterol: HDL ratio.

As this study was performed in free-living subjects, equalizing each nutrient component across all modes of coconut supplementation was deemed impractical [20], because a pre study preliminary trial indicated that such supplements would lead to noncompliance difficulties in daily consumption. In addition, equalizing one nutrient would lead to changes in other nutrient components. Hence, the described supplement method was employed to ensure maximal compliance.

Statistical analysis

The data were analyzed using SPSS 24 for Windows (SPSS, Inc., Chicago, IL, USA). The baseline characteristics of the sample subjects, as well as the data obtained at baseline, end of 4th week and end of 8th week, were analyzed using descriptive statistics. Group comparisons of continuous variables were carried out using *t*-tests, and categorical variables were compared using chi-square tests. The results are presented as numbers, percentages, means and standard deviations. Generalized estimating equations (GEEs) were used in repeated

measures analysis. In the analysis separate GEE models were fitted for the four outcome measures HDL, LDL, non-HDL and Cholesterol: HDL ratio by taking group (Coconut oil, Desiccated coconut, Coconut milk and the Control), time (Base, after 4 weeks and after 8 weeks) and group-time interaction as the predictors with independent working correlation structure. In addition, we were interested in investigating the effects of baseline LDL and HDL levels on the response to supplementation with the coconut. Thus, baseline and end of 8th week measures of the four response variables; HDL, LDL, non-HDL and Cholesterol: HDL ratio, were compared for baseline low and high HDL (i.e. ≤ 40 mg/dl and > 40 mg/dl), and baseline low and high LDL (i.e. ≤ 130 mg/dl and > 130 mg/dl) groups separately. Our previous study [20] demonstrated that coconut milk supplementation could suppress LDL levels in a subgroup of study subjects whose baseline LDL levels were high (i.e., >130 mg/dl). We compared baseline and 8th week measures because approximately 8 weeks would require to have somewhat stable lipid profile in the study participants who were on different coconut supplementary treatments.

A total of 200 subjects were randomized and allocated into 4 the treatment groups. An intention-to-treat analysis was performed; the missing values were replaced by mean values. The assumption used in the intent-to-treat model with regard to missing data and unmeasured endpoints for the dropouts was that they were missing at random. In the coconut oil group, 1 participant discontinued after 1 week; in the coconut milk group 2 participants discontinued after 1 week; in the desiccated coconut group 1 participant discontinued after 1 week and in the control 2 participants discontinued after 1 week and another 4 participants discontinued after the 2nd week. We did not consider them in intention-to-treat analysis as they had participated in the research only for the baseline measurement. In the final sample of participants ($n = 190$), the number of missing values were <5 for each and every outcome variable considered in the analysis. The participants in the study were well-educated adult group, thus we are confident that all the participants were adhered to the study protocol and none of them had consumed other products of oils/fats during the study period.

In this clinical trial between subject variable is the type of treatment (i.e. coconut oil supplement, desiccated coconut supplement, coconut milk supplement and the control). Within subject variable was the lipid measures at 3 time intervals; baseline, after the 4th week and after the 8th week. G*Power software was used to calculate the required sample size [22]. For this study with 4 groups and 3 repeated measures and with a moderate effect size of 0.1, with 80% power and significance level of 0.05, the required total sample size was 140. So a minimum of 35 subjects were needed for each treatment group.

Results

The demographic data of the study population are given in Table 1. The caloric intake of the study subjects at recruitment is given in Table 2.

Table 1
Demographics of the study sample ($N = 190$).

Variable	Treatment groups			
	Oil (n = 53)	Desiccated (n = 52)	Milk (n = 44)	Control (n = 41)
Age (years)	47 ± 11	46 ± 12	47 ± 10	47 ± 11
Current smoker	3	1	1	1
Systolic BP (mm/Hg)	118 ± 11.1	121 ± 12.3	120 ± 13	120 ± 11
Diastolic BP (mm/Hg)	76 ± 7.6	78 ± 8.1	80 ± 2.1	80 ± 11
Height (cm)	166 ± 5	166 ± 3	168 ± 3	168 ± 6
BMI (kg/m^2)	22 ± 1.1	21 ± 1.6	22 ± 1.6	22 ± 3.1
FBS (mg%)	99 ± 3.1	101 ± 5	97 ± 5	100 ± 6
e GFR < 60 ml/min/1.73^2	93 ± 07	92 ± 11	89 ± 11	93 ± 01
Physical activity (MET- h/w)	6.3 ± 2.3	6.6 ± 2.6	6.1 ± 1.9	6.1 ± 3.1

Table 2
Average dietary intakes at baseline (total cohort).

Dietary Intake	Mean value (± *SD*)
Total calories	1966 ± 498
% CHO	72.6 ± 6
% Protein	9.6 ± 5
% Fat	18.9 ± 4.4

Analysis of variance (ANOVA) was applied to examine whether the 4 groups are different with respect to baseline variables. It is observed that none of those mean values of the base variables were significantly differ in the 4 groups. So the groups do not differ in baseline characteristics due to randomization.

The macronutrient intake of the study population is in line with the values noted in other Sri Lankan dietary studies [23]. The nutritional content of each dietary preparation is given in Table 3.

Separate GEE models were fitted for the four outcome measures (HDL, LDL, CH-HDL ratio and Non-HDL) taking group, time and group-time interaction as predictors with independent working correlation structure. Normality and homoscedasticity of the outcome variables were assessed using p-p plots and standard residual plots (see supplementary material 2). It is observed that the outcome variables satisfy the normality and homoscedasticity assumptions. The changes in the lipid profile induced in the different study groups following supplementation, and model effects of HDL, LDL CH-HDL ratio and Non-HDL were estimated using generalized estimation equation (Table 4). The average values for the total cohort are given in Table 5.

It can be observed that there are significant interaction effects between time and group for all the response measure except for the LDL. This implies that the mean HDL, CH_HDL ratio and non HDL varies significantly among the 3 treatment groups and the control, varyingly at each time point that allowed us to interpret findings in a clinically relevant manner.

Pairwise comparisons of estimated marginal means based on the original scale of four dependent variables by GEE are graphically presented in Figs. 1–4, where the graphs depict the variations of the lipid mean values induced by different coconut supplements over the study period. The different lipid components are represented by dashed lines.

There was a slight decrease in the mean HDL value of the total cohort over the whole time period, from 44.58 ($SE = 0.82$) to 44.37 ($SE = 0.71$) (Fig. 1). In the oil group, the mean HDL level rose from 49.9 ± 1.8 at baseline to 52.6 ± 1.9 in week 4 and fell from there to 47.3 ± 1.6 in week 8. In the desiccated group, the mean HDL level fell from 45.0 ± 1.3 at baseline to 44.4 ± 1.5 in week 4, and further fell from there to 42.12 ± 1.2 in week 8. In the milk group, the mean HDL level rose from 41.6 ± 1.7 at baseline to 43.7 ± 1.5 in week 4 and further rose from there to 44.6 ± 1.5 in week 8. In the control group, the mean HDL level fell from 41.6 ± 1.3 at baseline to 41.2 ± 1.1 in week 4, and rose from there to 43.5 ± 1.1 in week 8. There was a significant interaction effect of the type of treatment and time (Table 4).

There was a slight increase in the LDL mean value of the total cohort over the entire time period, from 122.0 ($SE = 2.1$) to 122.8 ($SE = 1.7$) (Fig. 2). In the oil group, the mean LDL level rose from 112.2 ± 3.4 at baseline to 113.9 ± 3.2 in week 4, and rose from there to 114.58 ± 2.8 in week 12. In the desiccated group, the mean LDL level rose from 115.8 ±

Table 3
Nutritional composition of coconut preparations used as supplements.

Nutritional composition	Desiccated coconut 100 g	Coconut milk (powder) 100 g	Coconut oil 100 g
Energy (kcl)	1481.14	2974.82	3765.6
Protein (g)	3.3	6.8	0
Carbohydrate (g)	15.23	25.20	0
Fat (g)	33.4	64.80	100
Fiber (g)	9	5.4	0

Table 4
The GEE estimates of the marginal means and model effects of HDL, LDL CH-HDL ratio and Non-HDL in the 4 treatment groups.

Response variable	*Group*	*Baseline (Mean, 95% CI)*	*4th Week (Mean, 95% CI)*	*8th Week (Mean, 95% CI)*	Test of model effects			
					Sources	Wald χ^2	Degrees of freedom	*p* value
HDL	Oil	49.9 [46.4, 53.5]	52.6 [48.7, 56.5]	47.3 [44.2, 50.4]	Group	16.156	3	0.0010
	Desiccated	45.0 [42.4, 47.6]	44.4 [41.5, 47.3]	42.1 [39.7, 44.4]	Time	6.371	2	.0410
	Milk	41.6 [38.3, 44.9]	43.6 [40.7, 46.6]	44.6 [41,6, 47.6]	Group x Time	58.006	6	.000
	Control	41.6 [39.1, 44.1]	41.2 [38.9, 43.4]	43.5 [41.4, 45.7]				
LDL	Oil	112.2 [105.6, 118.9]	113.9 [107.7, 120.2]	114.5 [108.9, 120.2]	Group	18.101	3	0.0000
	Desiccated	115.8 [108.6, 123.0]	118.8 [111.3, 126.4]	122.9 [115.6, 130.2]	Time	1.565	2	.4570
	Milk	133.9 [123.8, 144.1]	125.9 [117.7, 134.2]	126.0 [118.9, 133.0]	Group x Time	10.638	6	.100
	Control	129.7 [122.6, 136.8]	128.2 [121.4, 134.9]	130.1 [123.0, 137.1]				
CH-HDL Ratio	Oil	4.3 [3.9, 4.6]	3.9 [3.5, 4.2]	4.1 [3.8, 4.3]	Group	16.246	3	0.0010
	Desiccated	4.7 [4.2, 5.1]	4.5 [4.1, 4.8]	4.9 [4.5, 5.5]	Time	5.958	2	.0510
	Milk	5.0 [4.5, 5.5]	4.8 [4.4, 5.2]	4.6 [4.3, 4.9]	Group x Time	30.792	6	.000
	Control	4.8 [4.4, 5.1]	4.9 [4.7, 5.2]	4.6 [4.3, 4.9]				
Non-HDL	Oil	147.9 [139.0, 156.8]	134.9 [126.5, 143.4]	133.9 [127.3, 140.5]	Group	8.184	3	0.0420
	Desiccated	152.9 [141.8, 164.1]	143.4 [134.0, 152.8]	145.3 [135.8, 154.6]	Time	11.700	2	.0030
	Milk	155.1 [142.9, 167.3]	154.5 [144.3, 164.6]	147.7 [139.2, 156.3]	Group x Time	28.000	6	.000
	Control	149.1 [140.8, 157.5]	156.4 [149.1, 163.6]	150.3 [142.2158.4]				

HDL – high-density lipoprotein cholesterol, LDL - low-density lipoprotein cholesterol, CH-HDL Ratio – Total cholesterol number divided by HDL cholesterol number, Non-HDL - subtracts HDL cholesterol number from the total cholesterol number, Groups – coconut oil, Desiccated coconut, Coconut milk and control (4 groups), Time – Base, after 4 weeks and after 8 weeks (3 time points).

Table 5
Baseline and end of 8th week lipid profile of the whole group ($N = 190$).

Parameters	Mean	95% CI of the Mean
HDL Baseline	44.54	43.03–46.05
End of 8th week	44.37	43.04–45.71
LDL Baseline	122.95	119.01–126.89
End of 8th week	123.38	119.99–126.77
Non-HDL Baseline	151.28	146.14–156.41
End of 8th week	144.30	140.18–148.41
Ch_HDL ratio Baseline	4.67	4.47–4.87
End of 8th week	4.56	4.38–4.74

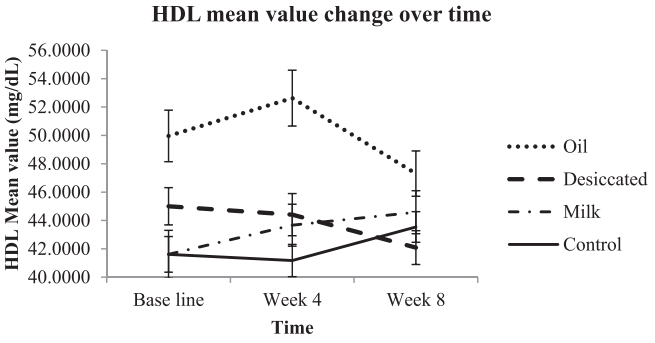

Fig. 1. Effect of different coconut preparations and a normal diet on HDL in healthy subjects. Data are presented as the means ± *SEs*.

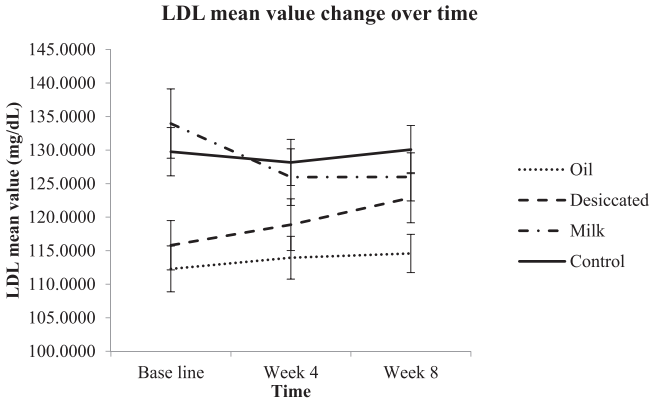

Fig. 2. Effect of different coconut preparations and a normal diet on LDL in healthy subjects. Data are presented as the means ± *SEs*.

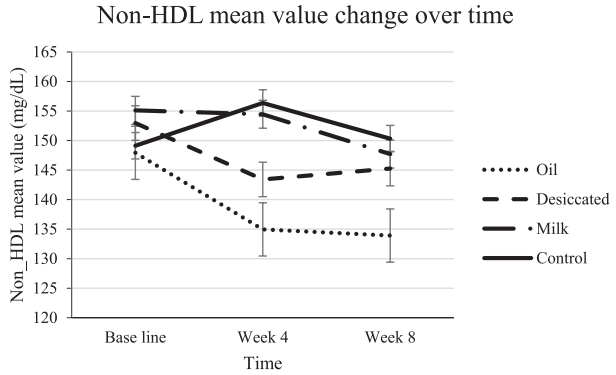

Fig. 3. Effect of different coconut preparations and a normal diet on non-HDL in healthy subjects. Data are presented as the means ± *SEs*.

3.7 at baseline to 118.8 ± 3.8 in week 4 and then rose from there to 122.89 ± 3.5 in week 8. In the milk group, the mean LDL level fell from 133.9 ± 5.2 at baseline to 125.9 ± 4.2 in week 4 and rose from there to 126.0 ± 3.5 in week 8. In the control group, the mean LDL level fell from 129.7 ± 3.6 at baseline to 128.2 ± 3.4 in week 4 and rose from there to 130.07 ± 3.5 in week 8. There was no significant interaction effect of the type of treatment and time (Table 4).

There was a decrease in the mean value of the non-HDL level of the total cohort over the entire time period, from 151.22 (*SE* = 2.6) to 143.75 (*SE* = 2.1) (Fig. 3). In the oil group, the mean non-HDL level fell from 147.9 ± 4.54 at baseline to 134.9 ± 4.31 in week 4, and fell from there to 133.91 ± 3.37 in week 8. In the desiccated group, the mean non-HDL level fell from 152.9 ± 5.69 at baseline to 143.41 ± 4.79 in week 4 and rose from there to 143.41 ± 4.79 in week 8. In the milk group, the mean non-HDL level fell from 155.11 ± 6.21 at baseline to 154.45 ± 5.17 in week 4 and fell from there to 147.70 ± 4.36 in week 12. In the control group, the mean non-HDL level rose from 149.12 ± 4.27 at baseline to 156.36 ± 3.69 in week 4 and fell from there to 150.32 ± 3.89 in week 12. There was a significant interaction effect of the type of treatment and time (Table 4).

There was a slight decrease in the mean value of the Ch HDL ratio of

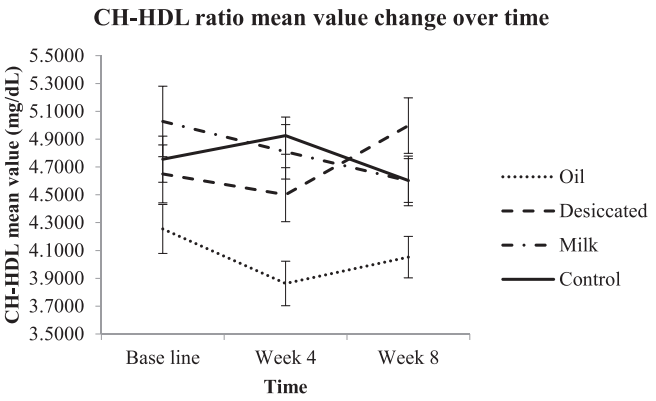

Fig. 4. Effect of different coconut preparations and a normal diet on the CH-HDL ratio in healthy subjects. Data are presented as the means ± SEs.

the total cohort over the entire time period, from 4.65 ($SE = 0.10$) to 4.55 ($SE = 0.09$) (Fig. 4). In the oil group, the mean CH-HDL level fell from 4.25 ± 0.17 at baseline to 3.86 ± 0.16 in week 4 and rose from 4.05 ± 0.14 in week 8. In the desiccated group, the mean CH-HDL level fell from 4.6 ± 0.21 at baseline to 4.5 ± 0.19 in week 4 and rose from there to 4.99 ± 0.26 in week 8. In the milk group, the mean CH-HDL level fell from 5.0 ± 0.25 at baseline to 4.8 ± 0.19 in week 4 and fell from there to 4.6 ± 0.17 in week 8. In the control group, the mean CH-HDL level rose from 4.7 ± 0.16 at baseline to 4.9 ± 0.13 in week 4 and fell from there to 4.60 ± 0.12 in week 8. There was a significant interaction for the type of treatment and time.

Tables 6–9 tabulate the changes in the mean values of the LDL, HDL, non-HDL and CH/HDL ratio as they occur in the study populations with high and low LDL and HDL values at baseline.

Discussion

The lipid dynamics seen in the whole cohort of study subjects revealed that there was no beneficial or adverse effect with regard to HDL, LDL and the CH:HDL ratio. However, the mean non-HDL value of the whole sample showed a reduction at the end of the study period (Table 5).

It is evident that the dietary supplements administered to the test subjects differed with regard to the nutrient composition and hence were not uniform with regard to the content of fat, protein, fiber and caloric value. The difference in the fiber content was inevitable due to the study design itself, which mandated that one mode of coconut should be the oil per se, which would obviously have a zero-fiber content.

The differences in the nutrient composition of the coconut supplement make it relevant to discuss each mode of supplementation separately to tease out possible associations between the changes in the lipid profile and nutrient components of the coconut supplements (Figs. 1–4).

Coconut oil supplementation

The lipid changes in this group could be summarized as follows: The LDL level increased slightly. The HDL level increased during the first 4 weeks, but subsequently, a decrease was observed. The non-HDL level showed a decrease. The CH/HDL ratio showed an overall decrease over the study period.

Saturated fats are well documented to raise LDL levels; hence, this finding is in accordance with generally accepted findings in lipid studies [24]. Likewise, saturated fats are accepted as raising HDL levels [25], and this was confirmed in the results of the first 4 weeks of the present study, although a reduction was observed thereafter.

The decrease in the non-HDL level is an interesting observation. The increase in the HDL level no doubt played an important part in reducing the non-HDL level, but the fact that the non-HDL levels continued to decrease even when the HDL levels dropped in the latter part of the study may indicate that other contributor(s) to the non-HDL component may also be reduced by coconut oil, as otherwise the elevated LDL should have mitigated the reduction in non-HDL.

The data regarding such effects are sparse. One study suggested that medium-chain fatty acids could affect VLDL lipolysis, which could contribute to the change in the non-HDL component [26].

Coconut kernel supplementation

In this group, the LDL level increased gradually over the study period. The HDL level decreased. The non-HDL level showed a decrease over the time period. The CH/HDL ratio remained unchanged.

The coconut kernel used in the present study was desiccated coconut. In increasing LDL, the coconut kernel preparation acted in line with coconut oil supplementation. It was postulated in our previous paper [20] that the soluble fiber and protein content in coconut milk contributed to the reduction in LDL. It appears that these effects are mitigated with respect to coconut kernels.

On analyzing the nutrient composition of the coconut preparation used in the present study, it is evident that the protein content was 50% less in the kernel supplement compared to the milk supplement. (i.e., 33.4 g compared to 64.8 g). The quality of the protein contained in desiccated coconut is also a factor that needs to be considered. The production of desiccated coconut involves the use of heat. Thermal exposure is known to cause denaturing of amino acids [27]. It is not possible to say whether this may have been a factor in the present study, as we have not performed amino acid assays of the preparation used by us.

Fiber content has also been postulated as a factor important in reducing LDL. At first glance, the fiber content of the kernel supplement appears to be greater than that of coconut milk (9 g vs 5.4 g). However, the greater part of the kernel fiber is the coarse variety, whereas lipid benefits probably accrue from the fine variety, which is more abundant in coconut milk preparations.

It is reasonable to assume that the quantity of fat that is consumed

Table 6
Mean value changes of LDL induced by different coconut preparations in the low and high baseline HDL and LDL subsets.

Base Level	Oil ($n = 53$)		Desiccated ($n = 52$)		Milk ($n = 44$)	
	Base Mean (95% CI)	End of 8 week Mean (95% CI)	Base Mean (95% CI)	End of 8 week Mean (95% CI)	Base Mean (95% CI)	End of 8 week Mean (95% CI)
Low HDL (< 40 mg/dl)	118.7 (103.1–134.3)	119.89 (106.8–132.8)	113.12 (100.6–125.6)	119.06 (104.8–133.3)	142.31 (125.7–158.9)	128.95 (117.5–140.4)
High HDL (> 40 mg/dl)	110.39 (102.7–118.0)	113.03 (106.6–119.4)	117.0 (107.8–126.2)	124.59 (115.8–133.4)	125.54 (113.7–137.3)	123.04 (114.0–132.0)
Low LDL (<130 mg/dl)	104.02 (98.4–109.6)	109.32 (104.1–114.5)	101.72 (96.9–106.5)	112.98 (105.9–119.9)	102.31 (94.2–110.4)	108.94 (100.3–117.5)
High LDL (>130 mg/d)	147.8 (137.8–157.8)	137.2 (123.6–150.8)	150.53 (142.7–158.3)	147.3 (134.9–159.7)	158.0 (149–167)	138.96 (131.4–146.6)

Table 7
Mean value changes of HDL induced by different coconut preparations in the low and high baseline HDL and LDL subsets.

Base Level	Oil (n = 53)		Desiccated (*n* = 52)		Milk (*n* = 44)	
	Base Mean (95% CI)	End of 8 week Mean (95% CI)	Base Mean (95% CI)	End of 8 week Mean (95% CI)	Base Mean (95% CI)	End of 8 week Mean (95% CI)
Low HDL (< 40 mg/dl)	33.83 (31.3–36.3)	35.82 (31.6–40.0)	34.68 (31.9–37.4)	33.25 (29.8–36.7)	32.90 (31.5–34.3)	38.80 (36.2–41.4)
High HDL (> 40 mg/d)	54.68 (51.2–58.2)	50.66 (47.4–53.9)	49.58 (47.2–51.9)	46.01 (43.9–48.1)	50.31 (46.2–54.4)	50.35 (45.9–54.8)
Low LDL (<130 mg/d)	51.09 (46.9–55.3)	48.18 (44.6–51.8)	46.13 (42.9–49.3)	43.55 (40.7–46.4)	45.26 (39.16–51.4)	48.91 (43.3–54.5)
High LDL (>130 mg/d)	45.1 (38.3–51.9)	43.5 (36.1–50.9)	42.2 (37.6–46.8)	38.45 (34.3–42.7)	38.84 (35.2–42.4)	41.28 (38.7–43.9)

Table 8
Mean value changes of non-HDL induced by different coconut preparations in the low and high baseline HDL and LDL subsets.

Base Level	Oil (n = 53)		Desiccated (*n* = 52)		Milk (n = 44)	
	Base Mean (95% CI)	8 weeks Mean (95% CI)	Base Mean (95% CI)	8 weeks Mean (95% CI)	Base Mean (95% CI)	8 weeks Mean (95% CI)
Low HDL (< 40 mg/dl)	163.91 (143.5–184.3)	140.72 (125.42–156.0)	156.43 (135.9–176.9)	147.0 (127–167)	164.95 (144.5–185.5)	152.03 (137.2–166.8)
High HDL (> 40 mg/dl)	143.24 (133.3–153.1)	131.91 (124.6–139.5)	151.41 (137.4–165.4)	144.48 (133.5–155.5)	145.27 (131.6–158.9)	143.38 (133.8–152.9)
Low LDL (<130 mg/dl)	137.27 (129.7–144.9)	128.00 (121.5–134.5)	132.5 (123.5–141.5)	131.47 (122.3–140.7)	117.63 (107.4–127.8)	127.20 (117.2–137.2)
High LDL (>130 mg/d)	193.7 (177.6–209.8)	159.3 (145–173.6)	203.4 (192–214.8)	179.26 (165.9–192.7)	183.6 (172.5–194.7)	163.29 (153.7–172.9)

Table 9
Mean value changes of CH/HDL ratio induced by different coconut preparations in the low and high baseline HDL and LDL subsets.

Base Level	Oil (n = 53)		Desiccated (*n* = 52)		Milk (n = 44)	
	Base Mean (95% CI)	12 weeks Mean (95% CI)	Base Mean (95% CI)	12 weeks Mean (95% CI)	Base Mean (95% CI)	12 weeks Mean (95% CI)
Low HDL (< 40 mg/dl)	5.86 (5.32–6.4)	5.09 (4.55–5.63)	5.73 (4.77–6.69)	5.66 (4.82–6.5)	6.07 (5.37–6.77)	5.15 (4.65–5.65)
High HDL (> 40 mg/dl)	3.78 (3.42–4.14)	3.74 (3.28–4.2)	4.16 (3.84–4.48)	4.7 (4.06–5.34)	3.98 (3.6–4.36)	4.04 (3.7–4.38)
Low LDL (<130 mg/dl)	3.94 (3.6–4.28)	3.85 (3.59–4.11)	4.05 (3.75–4.35)	4.33 (3.95–4.71)	3.80 (3.38–4.22)	3.81 (3.43–4.19)
High LDL (>130 mg/dl)	5.57 (4.97–6.17)	4.90 (4.2–5.6)	6.10 (5.3–6.9)	6.63 (5.43–7.83)	5.96 (5.34–6.58)	5.19 (4.79–5.59)

would have a bearing on the ensuing lipid changes. The potion of coconut kernel used in our study had a fat content of 33 g, whereas the potion of coconut milk was 64.8 g.

Furthermore, the fat in the kernel preparation would have been within the fibrous matrix of the coconut flakes and hence not fully accessible, whereas the fat in the milk would have readily lent itself to the metabolic processes of study subjects.

This could explain why the HDL effect was weak with coconut kernel supplementation.

That the quantum of fat being different could lead to disparate effects on the lipid profile is a concept we have utilized before as well in order to explain the differences in the effects of 78 g soya fat compared to 15 g soya fat [20].

Coconut milk supplementation

In this group, a reduction in the mean LDL level was observed over the study period. An increase in the mean HDL level was also observed in this group. A decrease in the mean non-HDL level was also observed. A reduction in the mean values of the CH/HDL ratio occurred in this group over the study period.

It appears that the lipid effects of coconut milk are largely beneficial, as the effects follow the currently accepted recommendations for lipid modification. In our previous study, we commented that the reduction in LDL that we observed was unexpected. The present study confirms the findings of our previous study in demonstrating a decrease in LDL in study subjects. In addition, a rise of the HDL was observed, and hence, the decrease in the non-HDL was predictable. The mechanisms that could be operational in reducing LDL and raising HDL are discussed in detail in our previous study paper [20].

Coconut contains fat consisting mainly of saturated fatty acids, which are well accepted as contributing to cholesterol synthesis. Hence, a reduction in saturated fatty acids in the diet is said to be beneficial [28–30]. In support of this view, some investigators have reported that coconut fat significantly raises S. cholesterol [31]. Comparing the S. cholesterol levels in different Polynesian populations with differing degrees of coconut intake, it is possible to postulate that energy derivation of approximately 34% via coconut fat does not raise cholesterol levels, but energy derivations of 63% would do so. Hence, the quantity of coconut consumed also plays a role in the end level of S. cholesterol.

Almost all trials that have been pivotal in concluding that coconut fat raises S. cholesterol have utilized extracted coconut oil. However, it is relevant to note that coconut is a complex food with constituents other than fat, i.e., amino acids and fiber.

When coconut flakes (i.e., coconut in toto) were studied, a reduction in the total cholesterol and LDL levels was demonstrated [32]. The soluble fiber content in coconut flakes, also present in coconut milk, could account in part for this beneficial effect.

Soluble dietary fiber is an inclusive term for many substances, such as beta glucan, guar gum, psyllium, and pectin. Bile salt micelles combine with various soluble fibers, which are thus prevented from reabsorption. This leads to more cholesterol being utilized for the synthesis of bile salts. [33].

Furthermore, soluble fiber reduces the absorption of sugar so that enhanced insulin secretion is mitigated. This reduces the insulin-mediated stimulation of hepatic cholesterol synthesis [33]. Dietary fiber appears to complement the effect of statins as well [34,35].

Furthermore, some investigations have commented on the effect coconut protein could have on LDL levels [36]. The arginine:lysine ratio has been invoked as important in this context. Arginine is a semi-essential amino acid involved in vital metabolic processes in which nitric oxide (NO) is produced. It functions as a substrate for the NO synthetase enzyme. This has many important beneficial effects, such as reducing the oxidation of LDL. However, our present concern is related to the role of arginine in cholesterol synthesis. Thripathi et al. [37] demonstrated that a significant reduction in total cholesterol and LDL triglycerides and an elevation in HDL levels could be achieved with L-arginine supplementation (3 g/day). Rajmohan [38] reported that a low lysine/arginine ratio supplementation fed to rats had a hypocholesterolemic effect, but some other researchers failed to confirm this finding. [38,39].

LDL receptor density is crucial for maintaining proper LDL levels [40–42], and arginine is known to be effective in reducing LDL levels in rats with low LDL receptor activity. Cooke et al. [43] reported in 1997 that arginine supplements administered to rats with disrupted LDL receptors showed a reduction in the formation of de novo lesions. The authors discuss various mechanisms by which arginine-mediated no synthesis could inhibit atherosclerosis.

Effect of baseline LDL and HDL levels on the response to supplementation with coconut

We have previously reported that a decline in LDL levels could be demonstrated only in the subgroup of study subjects who had an initial high baseline LDL level upon receiving coconut milk supplementation; this phenomenon has not been reported in the literature thus far.

MacNamara et al. [44] reported that baseline cholesterol levels and response to a cholesterol-rich diet showed no relationship. It may be argued that baseline lipid levels indicate the efficacy of negative feedback mechanisms operating to maintain lipids within an appropriate range. It is by no means suggested that the degree of efficiency of control mechanisms is the sole factor responsible for a given baseline lipid value. Many factors are responsible for a given cholesterol level. Cholesterol biosynthesis seems to be tightly regulated. The HMG coA reductase enzyme is subject to multiple feedback control mechanisms so that cholesterol production is tightly controlled. The feedback is subserved via sterol and nonsterol molecules, which facilitate the binding of HMG coA reductase enzyme to the endoplasmic reticulum (EPA) with subsequent ubiquitination and degradation.

These regulatory mechanisms appear to involve so many steps that the complete process could certainly be termed complex. Evolution has set this regulatory process in place, as cholesterol biosynthesis needs to be correctly controlled to preserve health. [45]

Ekanayaka et al. (2013) demonstrated that coconut milk supplementation could suppress LDL levels in a subgroup of study subjects whose baseline LDL levels were high (i.e., >130 mg/dl). It is postulated that some component(s) in coconut milk significantly reduced the LDL levels in these subjects whose physiological control mechanism was inadequate. The specific target on which any component of coconut milk acts is undefined at present.

We analyzed data of the present study to further evaluate this finding (Tables 6–9). The results confirm our previous findings with reference to coconut milk supplementation. The benefit with regard to LDL suppression is seen in the high LDL subgroup but not in the low LDL subgroup.

The benefit, however, seems to be universally manifest in both low and high HDL subgroups, with the cutoff being 40 mg/dL.

It is interesting that it is solely coconut milk supplementation that shows benefit in this sub-analysis. The results with coconut oil or kernel preparations show no such relationship.

When the non-HDL ratio parameter is considered, the high LDL subgroup demonstrates a clear beneficial alteration of the lipid profile, with all three coconut preparations used. With reference to the CH/HDL ratio, beneficial effects were observed in the high LDL group in the milk supplementation group and oil supplementation group. In the low HDL subgroups, the SC/HDL ratio showed beneficial effects in the milk-supplemented group. In the oil supplementation group, in the low HDL group showed beneficial effects. These results fortify the contention that individual responses to dietary oils are greatly influenced by many factors operating at the individual level, such as age, sex, genetics, etc., all of which could be responsible for modification of the metabolic milieu, which could be manifested in the LDL level being out of control of the inbuilt regulatory mechanisms of the body, resulting in a high baseline value.

There are some limitations in this study. The study was conducted using a sample of volunteers working in a single study setting. It was not possible to conceal the type of treatment that the participants had received. Six participants allocated to the control group, 2 allocated to the coconut milk group, one allocated to the desiccated coconut group and one allocated to the coconut oil group were excluded as they refused to participate in the study before end of the 2nd week of the trial. Exclusion of these participants, did not even slightly affect the results of this study. It is observed that after randomization the base characteristics of the 4 treatment groups did not differ significantly so that the estimates done on lipids were accurate. Intention-to-treat (ITT) analysis was performed using mean imputation for missing values. Although there was a slight deviation from using the standard protocol of ITT in this study, <5 missing values were observed in each measures. So, the results of the study could be considered as reliable and accurate.

Practical implications

Coconut in its varied forms is an important component in Asian and Polynesian diets. In view of the increasing incidence and prevalence of ischemic heart disease in these populations, sound recommendations that are evidence-based have become a priority in preventive programs. Diet and blood lipids are two important modifiable risk factors that are vital preventive targets. Dietary advice, if kept within the culinary-cultural framework of a given population, has a greater likelihood of generating better compliance within that society.

Our study results have shown that although coconut oil per se may adversely affect the lipid profile, coconut milk leads to a beneficial effect on lipid chemistry. Hence, at least coconut milk need not be excluded from a heart-healthy diet and could be allowed to remain on the menu as a good source of nutrition, as other researchers have pointed out following a structured literature review. [46]

Harcombe et al. conducted a review and meta-analysis of dietary fat guidelines issued in 1977 and 1983 by UK and USA government agencies and concluded that these guidelines were not supported by randomized trial data. [14] This type of analysis no doubt mandated the 2015 USA dietary guidelines omitting dietary cholesterol as a nutrient of concern. [15]

The confusing data regarding the benefit or harm of saturated fat on the pathogenesis of coronary heart disease is in part due to the wide differences in nutrient content seen in different sources of saturated fat. Animal-sourced saturated fats may have different lipid and health effects compared to plant-sourced saturated fats. Puaschitz et al. studied the dietary intake of saturated fats in relation to mortality and concluded that there was no association with SF in incident coronary events in patients with established coronary arterial disease [12]. The food items in their study did not include coconut, but entries included meat, poultry, dairy products, eggs, and cakes. It is evident that the nutrient components of these sources are somewhat different from those of coconut. Nevertheless, there was no demonstrable adverse effect on mortality. Despite such evidence, however, some regulatory agencies believe that dietary saturated fats should be reduced and that substitution with PUFAs in lieu of SF could lead to reduced morbidity and mortality due to coronary artery disease. A vast amount of research points to this conclusion [47–49]. However, the data regarding the health effects of coconut oil are inconclusive. [50]. The substitution of saturated fat with carbohydrates does not appear to generate a similar benefit [50,51]. A meta-analysis of trials where coconut oil consumption was compared to other nontropical vegetable oils showed a significantly higher elevation of LDL levels [24].

In conclusion, the present study reveals that different coconut preparations have different effects on the lipid profile. Coconut milk appears to give the greatest benefit in terms of reducing LDL and increasing HDL. An identical response was seen with coconut milk in our previous study [20], which compared coconut milk porridge with soya milk porridge. It has been postulated that differing concentrations of protein, fat and fiber in coconut preparations could explain the dissimilar effects on the lipid profile caused by the different coconut preparations. The present study data provide evidence in support of this hypothesis. The specific benefits seen in the high basal LDL subgroup are interesting and warrant a detailed study. This subgroup would be at high risk for atherosclerotic disease; hence, coconut, especially milk extract, may be of special benefit in this subset of individuals. It should be noted that the study does not provide data regarding the effects that different coconut preparations may have on clinical outcomes.

Ethics approval and consent to participate

The study protocol was approved by the Ethics Committee of the MRI, Colombo, Sri Lanka (No: 02/2016). Written informed consent was obtained from all subjects before initiation of the study. The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki.

Clinical trial registration

International Standard Randomized Controlled Trial Number of the study is ISRCTN10859733.

Consent for publication

Not applicable (The manuscript does not contain any individual person's data in any form).

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CRediT authorship contribution statement

Ruvan A.I. Ekanayaka: Conceptualization, Methodology, Investigation, Writing - original draft, Writing – review & editing. **P.G.S.M. de Silva**: Investigation. **Medhani K.I. Ekanayaka**: Writing – review & editing, Data curation. **W.M.M. Jayathilake**: Supervision, Investigation. **R.P.M.M.R. Pathirana**: Supervision, Investigation. **Y.N. Amaratunga**: Investigation. **Prasadhini J.D. De Silva**: Supervision, Investigation. **Bilesha Perera**: Writing – review & editing, Formal analysis, Data curation.

Declaration of competing interest

The authors declare that they have no competing interest. This research was self-funded.

Data availability

The raw data sets are available from the corresponding author on reasonable request.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.gloepi.2024.100138.

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