The Effect of Virgin Coconut Oil on Lipid Profile and Fasting Blood Sugar: A Phase I Clinical Trial

Cecille dela Paz, M.D.[1], Cecilia Jimeno, M.D.[2], Rody Sy, M.D.[3], Felix Eduardo, Punzalan, M.D.[3], and Pepito dela Pena, M.D.[1]

Abstract

Background: Virgin coconut oil (VCO) is promoted as a dietary supplement for people with various ailments. However, there have been few formal scientific studies to validate its health benefits.

Objective: To determine the effect of VCO on lipid profile, FBS, weight and body mass index (BMI) of healthy humans and evaluate its safety profile by determining effect on creatinine, potassium, AST, ALT and CBC

Methods: Weight and height were obtained from 30 healthy males and BMI was computed on first visit. Baseline laboratories were taken: FBS, 2-hour glucose after 75 gram oral glucose, total cholesterol, triglyceride, LDL, HDL, creatinine, potassium, AST and ALT, and CBC. Each subject took VCO 15 mL t.i.d. before meals. After 6 weeks, repeat laboratory examinations were done. Weight was taken and BMI recomputed. Adverse events were reported.

Results: There was significant increase in mean FBS (76.03, 6.62 mg/dl to 80.11, 9.04 mg/dl, p=0.024), significant decrease in HDL (47.58, 7.54 to 44.09, 7.10, p=0.028), significant increase in creatinine (76.44, 9.88 umol/L to 82.99, 11.29 umol/L, p = 0.0002) and platelet count (281.03, 52.6 x 10^9/L to 295.1, 58.06 x 10^9/L, p = 0.040) and significant decrease in WBC count (7.63, 1.74 x 10^9/L to 6.85, 1.5 x 10^9/L, p = 0.012). There was no effect on the rest of the laboratory examinations done. Twenty participants reported mild gastrointestinal (GI) complaints, including diarrhea (8), abdominal pain (3), diarrhea and abdominal pain (5), and diarrhea and vomiting (4).

Conclusion: VCO significantly increases FBS and decreases HDL. VCO also increases creatinine and platelet count and decreases WBC count. Mild GI complaints were the most commonly reported adverse events. Further studies may be needed in order to validate the results of this exploratory study and elucidate the metabolic effects of VCO.

Keywords: virgin coconut oil, metabolic effect, lipid profile

Introduction

According to the universally accepted Lipid-Heart Theory, high saturated fats cause hypercholesterolemia and coronary heart disease[1]. Because coconut oil is rich in saturated medium chain fatty acids, it is believed to be cholesterogenic. Animal studies that showed these harmful effects were flawed because of their use of hydrogenated coconut oil. Hydrogenation of coconut oil is a process that prevents peroxidation or rancidity.[2] This process saturates the small amount of the essential fatty acid linoleic acid that makes hydrogenated coconut oil cholesterogenic. Without linoleic acid supplementation, the animals suffered from essential fatty acid deficiency.[1]

Coconut oil is a colorless to pale, brownish yellow oil with a melting point ranging from 23 to 26oC.[3] It is composed predominantly of medium chain fatty acids that do not participate in the biosynthesis and transport of cholesterol. Coconut oil, in fact, tends to raise the HDL and lower the LDL:HDL ratio.[2] Compared to long chain triglyceride fats, these medium chain triglycerides are easier to digest, absorb and oxidize. Short and medium chain fatty acids are solubilized in the aqueous phase of the intestinal contents, where they are vein. It is absorbed readily and is carried to the liver where it undergoes rapid oxidation to release energy.[2] Because of this property, they are deposited less into adipose tissue, and do not cause obesity. They also decrease protein catabolism in hypercatabolic states, raise thyroid function and do not form esters with cholesterol. When supplied with sufficient polyunsaturated fatty acids to avoid essential fatty acid deficiency, medium chain saturated fats fail to raise cholesterol levels. Animal fats, in contrast, have long chain fatty acids that do not mix easily with biologic fluids. They need pancreatic lipase for its digestion. Transport of long chain fatty acids are via lymphatic and systemic circulation. They bypass the liver and deposit cholesterol in tissues before going to the liver for final oxidation.

The medicinal effects of coconut oil are also due to its medium-chain fatty acids. Lauric acid, the major fatty

[1]*Former Fellow, Section of Endocrinology, Diabetes and Metabolism, Department of Medicine, UP-Philippine General Hospital*

[2]*Consultant, Section of Endocrinology, Diabetes and Metabolism, Department of Medicine, UP-Philippine General Hospital*

[3]*Consultant, Section of Cardiology, Department of Medicine, UP-Philippine General Hospital*

Reprint request to: Cecille R. dela Paz, MD, Department of Medicine, East Avenue Medical Center

acid from the fat of the coconut, has long been recognized for its unique properties that it lends to nonfood uses in the soaps and cosmetic industries.[5] Lauric acid and capric acid, another fatty acid found in coconut oil, possess antimicrobial properties. Lauric acid, converted into monolaurin in body is the antiviral, antibacterial, and anti-protozoal monoglyceride used by the human or animal to destroy lipid-coated viruses such as HIV, herpes, cytomegalovirus, influenza, various pathogenic bacteria, including Listeria monocytogenes and Helicobacter pylori, and protozoa such as Giardia lambdia.[4] In 1998, Dr. Conrado Dayrit performed the first clinical trial on HIV-treatment with coconut oil and monolaurin on 15 HIV-infected individuals. The initial trial confirmed the anecdotal reports that coconut oil does have anti-viral effect and can beneficially reduce viral loads of HIV patients.[5]

Virgin coconut oil is obtained from the fresh, mature kernel of the coconut by mechanical or natural means, with or without the use of heat, without undergoing chemical refining, bleaching or deodorizing, and which does not lead to the alteration of the nature of the oil.[6] It is suitable for consumption without the need for further processing. It was developed to meet the needs of the natural foods market that advocate minimal processing of products.

In recent years, virgin coconut oil has gained popularity as a nutraceutical. Promoted as a dietary supplement designed to optimize health through improved nutrition, it is said to be of benefit for patients with various ailments. Anecdotal reports and testimonials on the health benefits of virgin coconut oil have sprouted in various print and web publications. This has influenced a lot of people to join the bandwagon despite scarce scientific evidence of its efficacy in humans. In 2003, Indian biochemists set out to investigate the effect of virgin coconut oil on various lipid parameters in oil-fed rats. Virgin coconut oil, at 8 g/100 g weight, had a beneficial effect in lowering lipid component compared to copra oil and ground nut oil. It reduced total cholesterol, triglycerides, phospholipids, LDL and VLDL cholesterol levels and increased HDL cholesterol in serum and tissues.[7] The polyphenol component of the virgin coconut oil prevented in vitro LDL oxidation, implicated as a risk factor for atherosclerosis and coronary heart disease. Virgin coconut oil obtained by wet process may have more beneficial effects than coconut oil due to its higher unsaponifiable components like polyphenols and like α-tocopherols. Several studies have revealed the anti-oxidant activity of polyphenolic substances, especially from red wine and olive oils in oxidation of LDL.[7] Although these findings are very promising, it has yet to be demonstrated in humans.

While most of the published research on herbal or traditional medicine is pharmacological in nature, the World Health Organization 1993 Guidelines on the Evaluation of Herbal Medicines considers that clinical evaluation is ethical where drugs have long been in traditional use.[8] Safety is a primary concern in traditional and complementary therapies.[8] Thus, evaluation of safety should be the starting point in drug development strategies for herbal medications, like virgin coconut oil.

Objectives

To determine the effect of virgin coconut oil on lipid profile (serum cholesterol, low density lipoprotein (LDL), high density lipoprotein (HDL), and triglycerides) and fasting blood sugar levels of healthy human volunteers

To determine the effect of virgin coconut oil on weight and body mass index (BMI) of healthy human volunteers

To evaluate the safety profile of virgin coconut oil in healthy human volunteers by determining its effect on creatinine, potassium, aspartate aminotransferase (AST), alanine aminotransferase (ALT) levels and complete blood count (CBC)

Materials and Methods

This is a Phase I Clinical Trial that included 30 male healthy human volunteers to the study. A healthy volunteer was defined as a healthy person who agrees to participate in a health research project for reasons other than medical and receives no direct health benefit from participating.

Inclusion Criteria

1. Subjects between 18 and 40 years of age with no known medical conditions
2. Willing to give informed consent
3. Normal physical examination
4. Normal baseline laboratory examinations, including FBS, cholesterol, LDL, HDL, triglyceride, creatinine, potassium, ALT, AST, CBC and platelet count

Exclusion Criteria

1. Subjects with chronic illness like hypertension, diabetes, dyslipidemia
2. Subjects already on virgin coconut oil or other supplements
3. Known allergy to coconut oil
4. Subjects on any medications
5. Presence of conditions that would affect compliance (i.e. alcohol or drug dependence, psychiatric disorders)
6. Presence of significant and sufficient disability that prevents attendance at follow-up
7. Recipient of an investigational product within three months preceding the trial
8. Anthropometric data compatible with being overweight or obese as defined by the Asia-Pacific guidelines (BMI > 23 kg/m^2)
9. Dyslipidemia defined as serum cholesterol > 200 mg/dL, or HDL <40 mg/dL, or LDL > 130 mg/dL, or triglycerides > 150 mg/dL
10. Evidence of impaired fasting glucose (FBS 100 - 125 mg/d/L)

11. Evidence of hepatic dysfunction defined as AST or ALT greater than three times the upper limit of normal
12. Evidence of renal dysfunction defined as Creatinine clearance > 80

Study Procedure

Informed consent was obtained from the volunteers included in the study. Participants were asked to fast for 10 hours prior to the initial visit and screening interview. Blood pressure (BP) was measured once by the primary author using a Baumanometer desk mercurysphygmomanometer. Heart rate (HR), height and weight were also taken. Body Mass Index (BMI) was computed. Those who passed the screening had their baseline laboratory tests taken, which included fasting blood sugar (FBS), total cholesterol (TC), triglycerides (TG), low density lipoprotein (LDL), high density lipoprotein (HDL), creatinine, potassium, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels, and CBC with platelet count.

The participants with normal laboratory results were provided with a standard measuring cup and virgin coconut oil. The virgin coconut oil was sourced from the National Institute of Molecular Biology and Biotechnology (BIOTECH) at UP Los-Banos. The physico-chemical characteristics and fatty acid composition of BIOTECH VCO can be found in Appendix A.

The subjects were instructed to take 15 milliliters (mL) of virgin coconut oil three times a day before each main meal for six weeks. This is the dose recommended by manufacturers of virgin coconut oil. A diet rich in lauric acid would contain about 24 grams of lauric acid which could be found in about 3.5 tablespoons of coconut oil.[9]

They received standard dietary instruction from a clinical dietitian from the Dietary Department of the University of the Philippines – Philippine General Hospital (UP-PGH) based on the Recommended Energy Nutrition Intake (RENI) seen on Appendix B. Compliance to the dietary advice throughout the study period was emphasized by the primary author after consultation with the clinical dietitian. The subjects were instructed to report any adverse event to the principal investigator and record them in a weekly event diary.

All subjects were followed-up after six weeks. They were instructed to bring back the empty containers of the virgin coconut oil and the amount left were measured in order to monitor the compliance. Any adverse events were reported and described. Blood pressure, heart rate and weight were obtained again. BMI was recomputed. Blood were drawn again for total cholesterol, fasting blood sugar, triglycerides, LDL, HDL, creatinine, potassium, AST, ALT, CBC and platelet count.

Outcome Measurements

Primary Measures

Mean fasting blood glucose, serum total cholesterol, LDL, HDL, and triglycerides levels before and after virgin coconut oil intake. Mean weight and BMI before and after virgin coconut oil intake

Safety Measures

Mean serum creatinine, potassium, AST, ALT, and CBC before and after virgin coconut oil intake

Description of adverse events

Statistical Analysis

T-test for paired means was used to compare the pre- and post-treatment levels of the primary and safety measures.

Results and Discussions

Baseline Characteristics of Included Subjects

A total of 49 volunteers were screened and 30 were included in the study. The reasons for exclusion were the following: 13 subjects had low HDL, 2 had low HDL and high LDL, 1 had high triglyceride levels, 3 had high total cholesterol and LDL, and 1 had high total cholesterol, LDL and triglycerides. There was 1 drop-out. Table I shows the baseline characteristics of the study participants.

Table I. Baseline Characteristics of Study Participants, N = 29

	Mean, Standard Deviation (SD)
Age (years)	22.79, 4.46
Systolic BP (mmHg)	111.03, 10.80
Diastolic BP (mmHg)	71.03, 7.72
Heart Rate (bpm)	66.76, 7.75
Weight (kg)	55.18, 7.04
BMI (kg/m2)	19.84, 1.42

Primary Outcome Measures

Analysis of the results of 29 study participants showed a statistically significant increase in the fasting blood sugar from 76.03, 6.62 to 80.11, 9.04 mg/dl (p value 0.024) after 6 weeks on virgin coconut oil. One subject developed impaired fasting glucose. The elevated FBS may be due to added calories from intake of VCO. One tbsp coconut oil (14 g) contains 116 calories, so 1 tbsp three times a day would mean additional 348 calories daily.

Virgin coconut oil significantly decreased the mean HDL (47.58 ± 7.54 to 44.09 ± 7.10 mg/dl, p = 0.024). Six subjects had post-treatment HDL levels below 40 mg/dL. There was no significant effect on total cholesterol, triglyceride, and LDL. These results are in contrast to the study done by Nevin and Rajomohan on VCO-fed rats that showed VCO decreases TC, LDL and TG and increases HDL. It is also contradictory to reviews by Drs. Dayrit[1] and Enig[9] that coconut oil increases HDL. However, it must be noted that this conclusion was drawn from epidemiological and diet studies in the general population, rather than from clinical trials of VCO.

Eight subjects had weight loss of more than 1 kg but there was no significant effect on mean weight and BMI. This is in

Table II. Summary of the Results of the Primary Measures Comparing Pre-treatment and Post-treatment Values, N = 29

	Pre-treatment Mean, SD	Pre-treatment Mean, SD	t stat	p value (two-tail)
FBS (mg/dL)	76.03, 6.62	80.11, 9.04	- 2.374	0.024
Total Cholesterol (mg/dl)	144.67, 18.12	149.41, 20.88	- 1.508	0.143
LDL (mg/dl)	92.26, 17.18	91.48, 19.56	0.263	0.794
HDL (mg/dl)	47.58, 7.54	44.09, 7.10	2.325	0.028
Triglyceride (mg/dl)	74.99, 20.50	72.91, 22.74	0.443	0.661
Weight (kg)	55.18, 7.04	54.42, 5.41	0.904	0.374
BMI (kg/m2)	19.84, 1.42	19.86, 1.36	- 0.282	0.780

contrast to some data that medium chain triglycerides may have a potential role in treatment of obesity as it effects on certain hormones (CCK, Peptide YY, GIP, Neurotensin, Pancreatic Polypeptide).[10]

Table II summarizes the results of the primary measures.

Safety Measures

VCO significantly increases creatinine, from 76.44 + 9.88 to 82.99 ± 11.29 umol/L, with p value 0.0002. While this increase is statistically significant, it is important to note that this was not clinically significant as not one subject developed abnormal creatinine levels. There was no significant effect on K, AST and ALT. The pathophysiology of increased creatinine with VCO intake needs further elucidation as medium chain fatty acid metabolism occurs in the liver.

VCO also significantly increased platelet count (281.03, 52.6 x 10^9/L to 295.1, 58.06 x 10^9/L, p = 0.040) and significantly decreased WBC count (7.63,1.74 x 10^9/L to 6.85, 1.5 x 10^9/L, p = 0.012). There was no significant change in the rest of the hematological parameters.

This is the first study evaluating safety of VCO in humans. The only other study done on human subjects was in a clinical trial done by Dr. Dayrit on 15 HIV patients on Monolaurin/Coconut oil. There was no significant effect noted in renal and liver function in those patients with baseline normal function. In an unpublished paper by Pekson[11], an acute and subacute toxicity study on mice and rats, VCO induces significant increases in creatinine, ALT and AST levels. Histopathologic studies on the liver and kidneys showed hepatic and renal lesions in the sacrificed rats which included pyknosis of hepatocytes, moderate and severe cloudy swelling of hepatocytes, severe cloudy swelling of renal tubular cells, severe glomerular shrinking, and the presence of eosinophilic casts in the kidneys. Further animal studies are needed to confirm these findings.

Table III summarizes the results of the safety evaluation.

Gastrointestinal complaints were the most commonly reported adverse events, and they were generally mild and tolerated. Table IV shows the distribution of adverse events. Diarrhea was the most common adverse event noted. This is consistent with the acute toxidrome syndrome of VCO intake seen in rats which consisted of oily mucoid stools and blood-streaked feces[11]. No subject withdrew from the study due to an adverse event.

Table III. Summary of Safety Evaluation, N = 29

	Initial level Mean, SD	Repeat level Mean, SD	t stat	p value (two-tail)
Creatinine (umol/L)	76.44, 9.88	82.99, 11.29	- 4.146	0.0002
Potassium (mmol/L)	4.24, 0.31	4.35, 0.29	-0.096	0.924
AST (UI/L)	28.73, 15.46	27.36, 6.12	0.522	0.606
ALT (UI/L)	22.18, 13	23.58, 8.70	- 0.504	0.618
CBC				
Hemoglobin (g/L)	150.93, 10.56	149.52, 10.05	1.102	0.280
Hematocrit	0.46, 0.03	0.46, 0.03	1.126	0.270
Platelet (x 10^9/L)	281.03, 52.6	295.1, 58.06	- 2.147	0.040
WBC (x 10^9/L)	7.63, 1.74	6.85, 1.5	2.699	0.012
Neutrophils	0.65, 0.08	0.61, 0.1	1.527	0.138
Lymphocytes	0.32, 0.08	0.34, 0.09	- 1.263	0.217
Monocytes	0.02, 0.01	0.01, 0.01	1.301	0.204
Eosinophils	0.02, 0.02	0.03, 0.03	- 1.397	0.173

Table IV. Summary of Adverse Events, N = 99

Adverse Event	N
Diarrhea	8
Abdominal Pain	3
Diarrhea & Abdominal Pain	5
Diarrhea & Vomiting	4
None	9

Conclusion

This study shows that short-term use of virgin coconut oil appears to significantly increase FBS and decrease HDL but there is no significant effect on the rest of the lipid profile. VCO also appears to increase serum creatinine and platelet count and decreases WBC count. There were no significant effect on liver function and the rest of the hematologic parameters. Gastrointestinal complaints were the most commonly reported adverse events, but they were generally mild and tolerated.

Limitations and Recommendations

The authors were unable to adequately assess the diet of the subjects during the period of VCO intake, however they were given standard dietary advice at the beginning of the study. A diet that is high on carbohydrate or protein content may have had effects on the blood sugar or creatinine.

Because of the paucity of papers on VCO, this exploratory study was meant to investigate the safety of virgin coconut oil. A similar study with a larger sample size is warranted in order to validate the results of this exploratory study. Animal studies may also be needed in order to elucidate pathogenesis of the metabolic effects of the virgin coconut oil.

Acknowledgements

Funding for this study was provided by the Philippine Lipid and Atherosclerosis Society through the 2006 PLAS-Pfizer Research Award for Outstanding Research Proposal. The authors would like to thank the following people for their invaluable help in making this research possible: Mrs. Donna Rose P. Martinez of the Dietary Department of the UP-Philippine General Hospital, Dr. Teresita M. Espino of the National Institute of Molecular Biology and Biotechnology (BIOTECH) at UP Los Banos, Ms. Argeline V. Datu of the Lipid Research Unit, Medical Research Laboratory, UP-Philippine General Hospital, Mrs. Rhoda Montemayor of the Endocrine Laboratory, Medical Research Laboratory, UP-Philippine General Hospital, Ms. Gemma Abquina, and Mr. Jerald Ongsueco.

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Appendix A

Physico-chemical Characteristics of BIOTECH Coconut Oil[1]

PROPERTIES	BIOTECH Coconut Oil	AOAC Standard[2]
Color	Clear white	Colorless to brownish yellow
Specific gravity at 25°C	0.919	0.917-0.919
Free fatty acid value[3]	0.389	0.01-1.00
Saponification value	253	250-264
Iodine value	9.57	250-264
Refractive index at 20°C	1.455[4]	1.448-1.449[5]
Moisture	0.100	0.10-0.25
Viscosity	0.469	--

[1]The coconut oil was obtained through an enzyme catalyzed-centrifuge process developed by the group of Dr. T. M. Espino at BIOTECH-UPLB. The process did not utilize any form of heat treatment
[2] AOAC is The Association of Analytical Communities
[3]Expressed as % lauric acid
[4] Analyzed at the Industrial Technology Development Institute (ITDI), DOST, Taguig, Metro Manila.
[5]Determined at 40°C.

Fatty Acid Composition of BIOTECH Coconut Oil		
Fatty Acid[1]	Concentration (%)[2]	
	Control[3]	Enzyme-treated
C8:0 (caprylic)	5.45	5.59
C10:0 (capric)	3.04	3.09
C12:0 (lauric)	48.15	48.72
C14:0 (myristic)	20.05	21.01
C16:0 (palmitic)	9.80	10.44
C18:1 (oleic)	6.55	6.47
C18:2 (linoleic)	0.14	Trace

[1]Gas chromatographic analysis using flame ionization detector
[2] Percent (%) relative concentration is based on the percent (%) peak area
[3] Without enzyme treatment

Appendix B

Recommended Energy Nutrition Intakes (RENI)
Philippines, 2002 edition

Prepared by the RENI Committee, Task Forces and the Food and NutritionResearch Institute – Department of Science the Technology Secretariat

Population Group	Weight (kg)	Energy (kcal)	Protein (g)	Vit. A (ug RE)	Vit C (mg)	Vit B1 (mg)	Vit B2 (mg)	Vit B3 (mg)	Vit B9 (ug/DFE)	Ca (mg)	Fe (mg)	I (mg)
Infants, mos												
Birth to < 6	6	560	9	375	30	0.2	0.3	1.5	65	200	0.38	90
6 to < 12	9	720	14	400	30	0.4	0.4	4	80	400	10	90
Children, yrs												
1-3	13	1070	28	400	30	0.5	0.5	6	160	500	8	90
4-6	19	1410	38	400	30	0.6	0.6	7	200	550	9	90
7-9	24	1600	43	400	35	0.7	0.7	9	300	700	11	120
Males, yrs												
10-12	34	2140	54	400	45	0.9	1.0	12	400	1000	13	120
13-15	50	2800	71	550	65	1.2	1.3	16	400	1000	20	150
16-18	58	2840	73	600	75	1.4	1.5	16	400	1000	14	150
19-29	59	2490	67	550	75	1.2	1.3	16	400	750	12	150
30-49	59	2420	67	550	75	1.2	1.3	16	400	750	12	150
50-64	59	2170	67	550	75	1.2	1.3	16	400	750	12	150
65 +	59	1890	67	550	75	1.2	1.3	16	400	800	12	150
Female, yrs												
10-12	35	1920	49	400	45	0.9	0.9	12	400	1000	19	120
13-15	49	2250	63	450	60	1.0	1.0	14	400	1000	21	150
16-18	50	2050	59	450	70	1.1	1.1	14	400	1000	27	150
19-29	51	1860	58	500	70	1.1	1.1	14	400	750	27	150
30-49	51	1810	58	500	70	1.1	1.1	14	400	750	27	150
50-64	51	1620	58	500	70	1.1	1.1	14	400	750	27	150
65 +	51	1410	58	500	70	1.1	1.1	14	400	800	10	150
Pregnant women, trimester												
First			66	800	80	1.4	1.7	18	600	800	27	200
Second		+300	66	800	80	1.4	1.7	18	600	800	34	200
Third		+300	66	800	80	1.4	1.7	18	600	800	38	200
Lactating women												
1st 6 mos		+ 500	81	900	105	1.5	1.7	17	500	750	27	27
2nd 6 mos		+500	76	900	100	1.5	1.7	17	500	750	30	30