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Virgin coconut oil improves hepatic lipid metabolism in rats–compared with copra oil, olive oil and sunflower oil

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Effect of virgin coconut oil (VCO) on lipid levels and regulation of lipid metabolism compared with copra oil (CO), olive oil (OO), and sunflower oil (SFO) has been reported. Male Sprague-Dawley rats were fed different oils at 8% level for 45 days along with synthetic diet. Results showed that VCO feeding significantly lowered (P<0.05) levels of total cholesterol, LDL+ VLDL cholesterol, Apo B and triglycerides in serum and tissues compared to rats fed CO, OO and SFO, while HDL-cholesterol and Apo A1 were significantly (P<0.05) higher in serum of rats fed VCO than other groups. Hepatic lipogenesis was also down regulated in VCO fed rats, which was evident from the decreased activities of enzymes viz., HMG CoA reductase, glucose-6-phosphate dehydrogenase, isocitrate dehydrogenase and malic enzyme. In addition, VCO significantly (P<0.05) increased the activities of lipoprotein lipase, lecithin cholesterol acyl transferase and enhanced formation of bile acids. Results demonstrated hypolipidemic effect of VCO by regulating the synthesis and degradation of lipids.

Keywords: Apolipoproteins, Bile acids, Lipid levels, Lipogenic enzymes, Lipoprotein lipase, Olive oil, Virgin coconut oil

Coronary heart disease (CHD) resulting from atherosclerosis continues to be the most prevalent cause of death and disability in most developed countries. Several epidemiological studies showed that the risk of CHD rises progressively with high concentration of low density lipoprotein (LDL) cholesterol but there is an inverse correlation with high density lipoprotein (HDL) cholesterol[1,2]. Dietary fatty acids are one of the most important factors determining plasma lipid concentrations and consequently the CHD risk[3].

Poly unsaturated fatty acids (PUFA) are reported to be the strongest down regulators of hepatic lipogenesis[4]; replacing saturated fatty acids with PUFA have been recommended to lower the LDL cholesterol levels, but it may promote a modest lowering in HDL concentration[5]. Although Mediterranean diet, which is rich in olive oil (OO), was associated with a low prevalence of CHD[6], but its effect on plasma lipids has been controversial and it is positively correlated with higher activities of hepatic lipogenic enzymes[7] and induces triacylglycerol in the liver[8].

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Coconut oil has been an important component of the diet of the Kerala population for decades. But being saturated fatty acid rich oil; it is unfortunately maligned as hypercholesterolemic[9] compared with PUFA rich oils like sunflower oil (SFO). In fact, the habitual consumption of coconut oil has no specific role in causation of CHD, because the nature of the fatty acid present in the dietary oil have a role in modulating hepatic lipid metabolism[10] and the fatty acids in coconut oil are preferentially utilized for energy production[11] and are less implicated in the accumulation of body fat[12].

Recently virgin coconut oil (VCO) extracted by wet processing has gain a lot of attention among scientific population due to its therapeutic values. VCO by wet processing is quite different from the traditional processing of coconut oil from dried copra, that has been exposed to very high temperature or sunlight to remove moisture, which may inactivates most of the biologically active minor components. But VCO is directly extracted from coconut milk under controlled temperature; this type of extraction retains most of the biologically active components like vitamins, phytosterols and polyphenols[13]. These unsaponifiable fraction rich in antioxidants have an influence on the occurrence of CHD. Previous studies have reported that VCO is more beneficial than copra

oil (CO) in reducing oxidation of LDL and plasma lipid levels[14] and in enhancing antioxidant status[15]. In this context, the objective of the present study was to examine the effects of consumption of VCO with CO and other unsaturated oils like OO and SFO on various lipid parameters, lipoproteins, apolipoproteins and the enzyme activities related to hepatic lipid metabolism. In addition, the synthesis and excretion of bile acids were also determined.

Materials and Methods

Extraction of virgin coconut oil and copra oil—Coconut palm (*Cocos nucifera* L.) grown at the Kerala University campus were used for the extraction of VCO and CO. Extraction of oils as follows: For extracting VCO, solid endosperm of mature coconut (West coast tall variety) was crushed, made in to viscous slurry and squeezed through cheese cloth to obtain coconut milk which was refrigerated for 48 h, then subjected to mild heating (50°C) in a thermostat oven. The obtained VCO filtered through cheesecloth was used for the present study[14]. CO was extracted from coconut meat, which was dried in sunlight continuously for 4 days to remove moisture and the resulting copra was pressed in a mill to obtain CO[14].

Olive oil and sunflower oil—Olive oil (Pietro Coricelli brand) and sunflower oil (Sundrop brand) were purchased from local market.

Chemicals—Nicotinamide adenine dinucleotide phosphate, Glucose-6-phosphate disodium salt, malic acid and isocitrate were purchased from Sigma Chemical Co., St. Louis, MO. All the other chemicals used were of analytical grade. Turbidimetric immuno assay kit for apolipoprotein A1 (Apo A1) and apolipoprotein B (Apo B) were purchased from Agappe Diagnostics Ltd. Kochin, India.

Animals and diets—Male Sprague- Dawley rats (100-120 g) bred in our department animal house was used for the study. Animals were individually housed under hygienic conditions in polypropylene cages in a room maintained at an ambient temperature of 25±1°C with12:12-h light-dark cycle. Each rat was given 12 g synthetic diet containing 8% dietary oils daily for 45 days (Table 1). Experimental groups were as follows; Group I rats given 8% CO, Group II rats given 8% VCO, Group III rats given 8% OO and Group IV rats given 8% SFO. Entire protocol was approved by Animal Ethics Committee, University of Kerala. Food intakes of rats were noted daily and the body weight was determined weekly. After 45 days, animals were fasted overnight and sacrificed by sodium pentathone injection, blood and tissues were collected for various estimations.

Analytic determinations—Total lipids from liver, heart and aorta were extracted using chloroform/ methanol as described by Folch *et al*[16]. From this, aliquots were used for the estimation of total cholesterol and triglycerides (TG)[17,18]. HDL cholesterol was measured in serum[19] and LDL + very low density lipoprotein (VLDL) cholesterol levels were calculated using the standard Friedwald equation[20]. Apo A1 and Apo B in Serum was determined by PEG enhanced turbidimetric immuno assay[21]

For assaying the enzyme activities related to lipid metabolism, rat liver was washed, minced with scissors and homogenized in glycyl glycine buffer in ice. Homogenates were centrifuged at 9000 *g* at 4°C for 20 min and the supernatant fraction was used for various enzyme activities. Activity of lipogenic enzymes like Glucose-6- phosphate dehydrogenase (G6PDH)[22], malic enzyme (ME)[23] and isocitrate dehydrogenase (ICDH) by spectrophotometric assays based on the absorbance change at 340 nm[24]. Enzyme activity was expressed as units per milligram of protein, where a unit is the activity of enzyme which converted one nanomole of substrate per min. HMG CoA reductase activity[25] in the liver was determinined by the ratio of HMG CoA to mevalonic acid. For assaying the catabolism of lipids, the activity of lipoprotein lipase (LPL)[26] in heart and adipose tissue was determined and plasma lecithin cholesterol acyl transferase (LCAT) was assayed by the method described by Schoenbeimer and Sperry[27], which represents the percentage increase in the ratio of ester cholesterol to free cholesterol. Procedure of Okishio *et al*.[28] was used for extraction of hepatic bile acids. Total bile acids were estimated as total vanillin reactive substance[29]. For estimating the fecal bile acids and neutral sterols, fecal samples from rats of each group was homogenized with an equal volume of water

Table 1—Formulation of synthetic diet used for the study

Ingredients (%)	Group I	Group II	Group III	Group IV
Corn Starch	71	71	71	71
Casein	16	16	16	16
Copra oil	8	--	--	--
Virgin coconut oil	--	8	--	--
Olive oil	--	--	8	--
Sunflower oil	--	--	--	8
Salt mixture	4	4	4	4
Vitamin Mixture	1	1	1	1

and lyophilized to a fine powder. From this powder fecal bile acids and neutral sterols were extracted[30] and estimated as described earlier[29,31]. Protein was determined using Folin-Ciocalteau reagent[32].

Statistical analysis—Statistical differences were determined using one way ANOVA followed by Duncan's, post hoc test to identify the differences using SPSS 11.5 (SPSS Inc., Chicago IL, USA). Differences of $P<0.05$ were considered to be significant. Data are reported as mean ± SD unless otherwise stated.

Results

Effect on lipids, lipoprotein and apolipoproteins — In the present study no significant change in body weight of animals from 4 groups were observed. Serum total cholesterol, HDL cholesterol, LDL+ VLDL cholesterol concentrations in serum of rats fed different dietary fat are presented in Fig. 1. Cholesterol levels in serum and tissues (liver, heart and aorta) were significantly ($P<0.05$) decreased in rats fed VCO (75.12 mg/100 mL serum, 128.57 mg/100 g liver, 125.03 mg/100 g heart and 160.07 mg/100 g aorta) compared to CO, OO and SFO fed rats. Highest cholesterol levels were observed in OO fed rats compared to other oils and which were decreased in SFO fed rats. HDL cholesterol in VCO fed rats (61.50 mg/100 mL) was increased significantly than CO (55.53 mg/100 mL), OO (51.49 mg/100 mL) and SFO (47.96 mg/100 mL) fed rats and the least HDL levels were observed in SFO fed rats. LDL+VLDL cholesterol levels were decreased significantly in VCO fed rats (12.33 mg/100 mL) compared to CO (35.87 mg/100 mL), OO (45.08 mg/100 mL) and SFO (34.56 mg/100 mL) fed rats. In addition, there was a significant increase in serum Apo A1 levels in VCO fed rats (38.91 mg/100 mL) compared to rats fed with CO (33.84 mg/100 mL),OO (31.26 mg/100 mL) and SFO (28.47 mg/100 mL); while Apo B levels in serum were significantly decreased in rats fed VCO (8.64 mg/100 mL) compared to rats fed other oil and the highest levels were observed in OO fed rats (12.73 mg/100 mL) (Fig. 2). VCO feeding also significantly decreased the levels of triglycerides in tissues (liver, heart and aorta) and serum (9.93 mg/ 100 mL) as compared to rats fed CO (11.28 mg/100 mL),OO (13.47 mg/ 100 mL) and SFO (12.06 mg/100 mL); the highest triglyceride level is seen in OO fed rats (Table 2).

Effect of VCO on hepatic lipogenesis —Activity of HMG CoA reductase, a key enzyme in the cholesterol biosynthesis was significantly decreased in VCO fed rats compared to rats fed other oils. Since the result is expressed as ratio of HMG CoA to mevalonate, lower ratios indicate higher enzyme activity (Table 3). In addition, the activities of lipogenic enzymes namely G6PDH, ME and ICDH were significantly decreased in liver of rats fed VCO, which resulted decreased lipogenesis in VCO fed rats compared to rats fed CO,OO and SFO (Table 3).

Effect on catabolism of lipids—Activity of LPL, which hydrolyzes triglycerides in lipoproteins was increased significantly in heart and adipose tissue of rats fed VCO diet as compared to CO,OO and SFO diet and the activity of plasma LCAT (expressed as the ratio of ester cholesterol to free cholesterol) was also increased significantly in VCO fed rats compared to rats fed CO,OO and SFO (Table 4). For estimating the degradation of cholesterol, we analyzed the

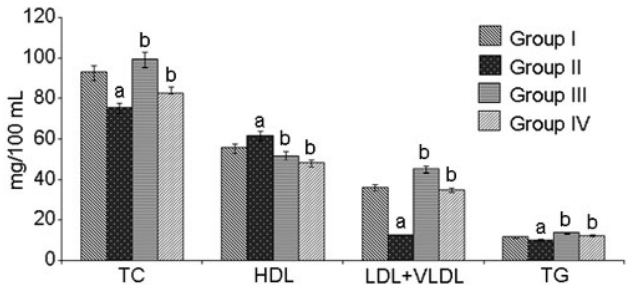

Fig. 1—Concentration of serum lipids (mg/ 100 mL) in CO, VCO, OO and SFO fed rats [Values are mean ± SD from 6 rats in each group]. Significance at $P<0.05$. [a]Values are significantly different from Group I ($P<0.05$); [b]values are significantly different from Group II ($P<0.05$).

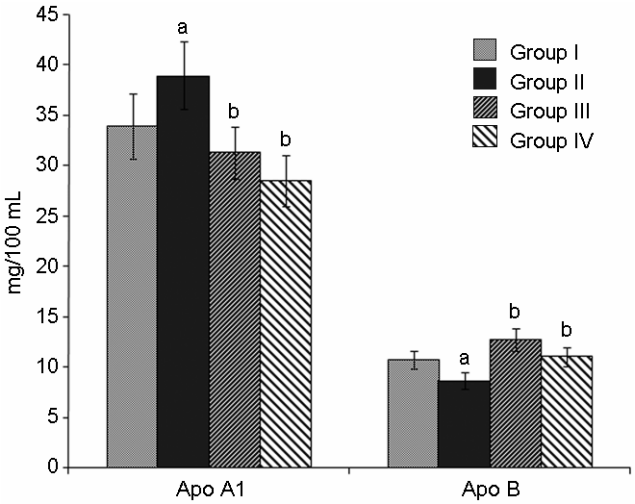

Fig. 2—Concentration of apolipoproteins (mg/ 100 mL) in serum of rats fed CO, VCO, OO and SFO [Values are mean ± SD from 6 rats in each group]. Significance at $P<0.05$. [a]Values are significantly different from Group I ($P<0.05$); [b]values are significantly different from Group II ($P<0.05$).

Table 2—Concentration of cholesterol and triglyceride in tissues
[Values are mean ± SD from 6 rats in each group]

Parameters	Liver	Heart	Aorta
Cholesterol (mg/100 g)			
Group I	263.75 ± 4.00	224.40 ± 7.68	205.35 ± 7.60
Group II	128.57 ± 4.78[a]	125.03 ± 4.90[a]	160.07 ± 5.70[a]
Group III	279.79 ± 9.97[b]	252.69 ± 9.41[b]	349.73 ± 12.94[b]
Group IV	265.36 ± 9.85[b]	211.36 ± 7.64[b]	206.41 ± 8.00[b]
Triglycerides (mg/100 g)			
Group I	182.38 ± 7.14	51.33 ± 1.90	708.52 ± 26.23
Group II	154.78 ± 3.33[a]	39.20 ± 1.46[a]	523.01 ± 19.09[a]
Group III	286.33 ± 10.06[b]	54.88 ± 5.00[b]	1029.15 ± 38.22[b]
Group IV	215.84 ± 8.93[b]	47.16 ± 6.10[b]	994.34 ± 87.39[b]

[a]Values are significantly different from Group I (P <0.05); [b]values are significantly different from Group II (P <0.05)

Table 3—Activity of HMG CoA reductase and lipogenic enzymes
[Values are mean ± SD from 6 rats in each group]

Groups	HMG CoA reductase*	G6PDH (U^{Ψ}/ mg protein)	ME (U^{Ψ}/ mg protein)	ICDH (U^{Ψ}/ mg protein)
I	2.75 ± 0.10	122.02 ± 4.97	870.69 ± 32.43	151.77 ± 5.65
II	4.58 ± 0.42[a]	94.33 ± 3.46[a]	715.33 ± 27.89[a]	135.99 ± 5.06[a]
III	2.16 ± 0.19[b]	210.28 ± 7.63[b]	1650.66 ± 61.16[b]	235.97 ± 8.85[b]
IV	3.01 ± 0.27[b]	212.47 ± 7.63[b]	1054.77 ± 101.52[b]	265.08 ± 9.43[b]

[a] Values are significantly different from Group I (P <0.05); [b]values are significantly different from Group II (P <0.05). *Ratio of HMG CoA to mevalonate, lower the ratio higher the activity; $^{\Psi}$One unit is defined as the activity of enzyme which converted 1 nanomole of substrate/ min.

Table 4— Activity of LCAT and LPL
[Values are mean ± SD from 6 rats in each group]

Groups	LCAT in Plasma*	LPL (U^{Ψ}/ mg protein)	
		Heart	Adipose tissue
I	31.84 ± 1.29	24.08 ± 0.80	148.99 ± 5.42
II	38.50 ± 1.43[a]	29.02 ± 1.07[a]	214.49 ± 7.65[a]
III	21.96 ± 0.82[b]	18.95 ± 0.71[b]	115.24 ± 9.96[b]
IV	33.85 ±1.26[b]	24.47 ± 0.97[b]	148.53 ± 12.38[b]

[a]Values are significantly different from Group I (P <0.05); [b]values are significantly different from Group II (P <0.05). *LCAT (Lecithin cholesterol acyl transferase) measured as ratio of ester cholesterol to free cholesterol; $^{\Psi}$One unit is defined as μ moles of glycerol liberated/hour.

concentration of total hepatic bile acids, which was found to be significantly increased in rats fed with VCO (47.18 mg /100 g liver) compared to CO (36.74 mg /100 g liver), OO (30.31 mg /100 g liver) and SFO (40.65 mg /100 g liver) (Fig. 3). In addition, there was a significant increase in the concentration of fecal bile acids (27.38 mg /100 g feces) and neutral sterols (6.75 mg /100 g feces) were observed in VCO fed rats, which indicating higher rate of its excretion in VCO fed rats compared to other oil fed groups (Table 5).

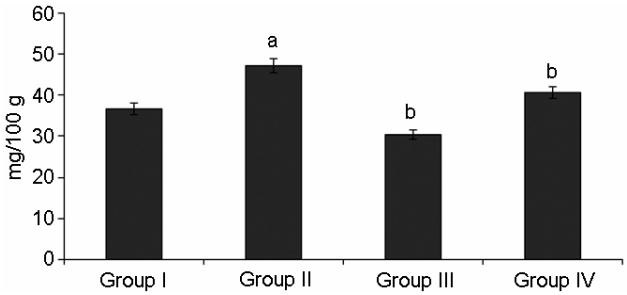

Fig. 3—Concentration of hepatic bile acids (mg/100 g liver) in CO, VCO, OO and SFO fed rats [Values are mean ± SD from 6 rats in each group]. Significance at P <0. 05. [a]Values are significantly different from Group I (P <0.05); [b]values are significantly different from Group II (P <0.05).

Table 5— Concentration of fecal bile acids and neutral sterols [Values are mean ± SD from 6 rats in each group]

Groups	Fecal bile acids (mg/100 g feces)	Neutral sterols (mg/100 g feces)
I	18.94 ± 1.72	4.85 ± 0.37
II	27.38 ± 2.48[a]	6.75 ± 0.55[a]
III	18.52 ± 1.61[b]	4.48 ± 0.38[b]
IV	20.84 ±1.81[b]	5.05 ± 0.48[b]

[a]Values are significantly different from Group I (P <0.05); [b]values are significantly different from Group II (P <0.05).

Discussion

The present study illustrates the effect of VCO on lipid parameters and hepatic lipid metabolism as compared to CO, OO and SFO in rats. Results indicate that VCO feeding leads to a much lower concentrations of cholesterol and triglycerides in serum and tissues (liver, heart and aorta) and an increase in HDL cholesterol levels when compared to feeding equienergic amounts of CO, OO and SFO. Previous studies also revealed decreased levels of lipids in rats fed VCO than CO[14]. Since the fatty acid compositions of VCO and CO are similar, the major difference exists especially in the presence of more amounts of unsaponifiable components in VCO compared to CO. In fact, wet processing under controlled temperature retains higher amounts of unsaponifiable components viz., polyphenols, tocopherols, tocotrienols, β-carotenes and phytosterols. Reports from other studies are consistent with the above findings[13]. These observations indicate that greater quantity of these unsaponifiable components may compensate for its higher saturated fatty acids. The results suggest that these unsaponifiable components may play an important role in hypolipidemic action of VCO.

Total polyphenol content of VCO (84 mg/100 g oil) and CO (64.4 mg /100 g oil) were estimated and reported previously[15]. Furthermore, another study revealed that phenolic fraction of VCO contains higher levels of caffeic acid, P-coumaric acid, ferulic acid and catechin than CO[33]. In addition, we reported previously that VCO contains significantly higher amounts of vitamin E (30.87 μg /100 g oil) than CO (12.76 μg /100 g oil)[34]. Recently we have estimated the total tocopherols including tocotrienols[35], significantly higher levels of total tocols including tocotrienols were found in VCO (33.12 μg /100 g oil) as compared to CO (15.89 μg / 100 g oil). Tocotrienols are effective in lowering serum total and LDL-cholesterol levels by inhibiting the hepatic enzymic activity of HMG-CoA reductase through the post-transcriptional mechanism[36]. Moreover, the amount of β-carotene, a precursor form of vitamin A in VCO (196 μg /100 g oil) was estimated[37]and which also found to be significantly greater than that of CO (109.95 μg / 100 g oil). There are reports that increased concentration of β-carotene decreases the concentration of lipids and increases the fecal secretion of bile acids[38]. In addition, we reported previously that VCO contains increased levels of phytosterols (Stigma sterol – 63.13 ng/ dL and β-Sitosterol-73.03 ng/dL) as compared to CO (Stigma sterol –57.07 ng/ dL and β-Sitosterol-57 ng/dL)[34]. It is clear that the phytosterols competitively blocks the absorption of cholesterol and increases fecal excretion of bile acids and neutral sterols for improving circulating lipid profiles to reduce the risk for CHD[39]. All these biologically active micronutrients` present in VCO act synergistically and results in beneficial alterations in lipid levels.

Animals fed VCO showed a dramatic increase in HDL cholesterol as compared to other groups and the HDL cholesterol was lower in OO and SFO diet than CO. Moreover, increased Apo A1 levels in serum of VCO fed rats directly correlated with the increased HDL levels in rats fed VCO compared to other oil fed rats. There are reports that HDL cholesterol and Apo A1 levels were increased in coconut oil diet compared to MUFA or PUFA diet[40]. HDL cholesterol in combination with Apo A1 and LCAT plays a key role in mediating reverse cholesterol transport[41]. The increased HDL cholesterol in VCO fed rats showed a beneficiary effect compared to other oils.

LDL+VLDL cholesterol were decreased in VCO fed rats compared to other groups, which can correlate with the decreased levels of Apo B in serum of VCO fed rats as compared to rats fed CO, OO and SFO. But highest levels of LDL+VLDL and Apo B in serum were found in rats fed OO, which is due to the fact that oleate in OO increases the synthesis and secretion of Apo B and which protects nascent Apo B from degradation by ER-proteases and leads to Apo B secretion[42]. Thus increased synthesis of VLDL enhances lipid accumulation in heart and aorta and excess LDL associated Apo B formed by oleate rich diet invade the arterial wall, get oxidized there and taken up by the scavenger receptors and further leads to atheroma.

Triglycerides in serum and tissues were significantly decreased in VCO fed rats than OO and

SFO, which may be due to difference in transport and catabolism of constituent fatty acids. Coconut oil contains mostly short and medium chain fatty acids viz., capric acid (7.46%), caprylic acid (7.69%) and lauric acid (46.4%), which are mostly absorbed through the hepatic portal vein and rapidly oxidized by both mitochondrial and peroxismal pathways[43]. But increased TG level in PUFA fed rats may be due to the increased synthesis and release of VLDL from the liver into the circulation[44]. It is reported that diet rich in OO resulted higher TG levels, which may positively correlated with higher activities of hepatic lipogenic enymes[7]. In addition, there are reports that OO enriched diet decreases activity of mitochondrial carnitine palmitoyl transferase-1(CPT-1), which results in impairment of fatty acid oxidation[8] and therefore have a role in lipid accumulation in OO fed rats.

HMG CoA reductase catalyzes the major rate limiting step in cholesterol biosynthesis. Decreased hepatic cholesterogenesis is evident from the decreased activity of HMG CoA reductase in VCO fed rats compared to CO, OO and SFO fed rats. Coconut oil contains appreciable amounts of tocotrienols[45], it is reported that tocotrieniols and polyphenols inhibits the HMG CoA reductase activity[36,46.] This may be one of the reasons for the decreased cholesterogenesis in VCO fed rats compared to other oil fed groups. Decreased activity of hepatic lipogenic enzymes (G6PDH, ME and ICDH) in VCO fed rats indicate decreased lipogenesis as compared to other groups. These lipogenic enzymes provide NADPH for fatty acid synthesis and the increased hepatic lipids in OO fed rats are positively correlated with higher activities of hepatic lipogenic enzymes[7,8].

LPL is a triacylglycerol hydrolase, whose activity was increased significantly in VCO fed rats compared to CO, OO and SFO fed rats. The increase in the activity of this enzyme indicates increased clearance of TG from the circulation and directly correlated with the lower levels TG in VCO fed rats compared to others. It is previously reported that dietary coconut oil has higher LPL activity[47]. Moreover, the activity of LCAT also increased in VCO fed rats compared to all other groups. LCAT is critical for the maintenance of plasma lipoproteins and is involved in the trans-esterification of cholesterol, the maturation of HDL and the flux of cholesterol from cell membranes into HDL[48]. The increased LCAT activity agrees with the decreased concentration of cholesterol in VCO fed rats. In addition, increased LCAT activity has also been reported in rats fed coconut oil[49]. The results suggested that decreased activity of both LPL and LCAT in OO fed rats may be one possible mechanism for accumulation of lipids in OO fed rats compared to rats fed VCO.

Major pathway for disposal of cholesterol from body is via the synthesis and excretion of bile acids and fecal neutral sterols. The results showed that rats fed with VCO resulted in increased synthesis of hepatic bile acids and also enhanced excretion of fecal bile acids and neutral sterols as compared to rats fed CO, OO and SFO. There are reports that excretion of bile acids was appreciably lower in PUFA fed rats compared to coconut oil fed rats[50].

In conclusion, findings of this study indicated that dietary VCO beneficially modulates the hepatic lipid metabolism, by regulating the synthesis and degradation of lipids as compared to other dietary oils like copra oil, olive oil and sunflower oil. This benefial effect of VCO over OO and SFO may be due to the difference in absorption, transport and catabolism of its constituent fatty acids as well as the higher amounts of biologically active unsaponifiable minor components present in VCO.

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