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Chemical composition and health benefits of coconut oil: an overview

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Abstract

Coconut oil is an integral part of Sri Lankan and many South Asian diets. Initially, coconut oil was classified along with saturated fatty acid food items and criticized for its negative impact on health. However, research studies have shown that coconut oil is a rich source of medium-chain fatty acids. Thus, this has opened new prospects for its use in many fields. Beyond its usage in cooking, coconut oil has attracted attention due to its hypocholesterolemic, anticancer, antihepatosteatotic, antidiabetic, antioxidant, anti-inflammatory, antimicrobial and skin moisturizing properties. Despite all the health benefits, consumption of coconut oil is still underrated due to a lack of supportive scientific evidence. Even though studies done in Asian countries claim a favorable impact on cardiac health and serum lipid profile, the limitations in the number of studies conducted among Western countries impede the endorsement of the real value of coconut oil. Hence, long-term extensive studies with proper methodologies are suggested to clear all the controversies and misconceptions of coconut oil consumption. This review discusses the composition and functional properties of coconut oils extracted using various processing methods.

Keywords: coconut oil; composition; medium-chain fatty acids; functional properties

ABBREVIATIONS

C-VCO	Cold-extracted virgin coconut oil
CNO	Coconut oil
CVD	Cardiovascular disease
F-VCO	Fermented virgin coconut oil
FA	Fatty acid
H-VCO	Hot-extracted virgin coconut oil
HDL	High-density lipoprotein
LCFA	Long-chain fatty acid
LDL	Low-density lipoprotein
MCFA	Medium-chain fatty acid
MCT	Medium-chain triglyceride
ML	Monolaurin
MUFA	Monounsaturated fatty acid
PUFA	Polyunsaturated fatty acid
RBD	Refined, bleached, deodorized
SFA	Saturated fatty acid
TAG	Triacylglycerol
TC	Total cholesterol
TMP-SMX	Trimethoprim-sulfamethoxazole
VCO	Virgin coconut oil

INTRODUCTION

The coconut tree (*Cocos nucifera* L.) is considered as a precious gift from nature to mankind. It is also known as the 'tree of life' since each part of the tree has its own value.[1,2] Coconut is the major source of fat in the diets of South Asians and South East Asians. The Coconut Development Authority of Sri Lanka reports that 60% of total harvest was locally consumed in 2015–2016. Despite the large consumption of coconut oil (CNO), Sri Lankans have a higher life expectancy than countries where coconut consumption is comparatively low.[3] Athauda *et al.*[4] examined the health effects of CNO consumption in Sri Lanka from 1992 to 2006 and revealed that there was a consistency of CNO consumption during the period along with a negative correlation with the increased cardiovascular disease (CVD) death rates in the country. Thus, the study showed that a rise in cardiac mortality was unlikely due to CNO consumption in Sri Lanka. Recently, the demand for CNO has rapidly increased among Western countries due to its

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notable health benefits. Further, the Asian Pacific coconut community records show that CNO export from Asia has grown 3.3% annually over a five-year period.[4]

The basic chemistry of CNO was figured out back in the 1920s and 1930s. Back then it was found to contain approximately 90% saturated fatty acids (SFAs) of the total fat content and condemned for its adverse effects on health.[5–9] However, it was found that some of the nutritional and therapeutic benefits of CNO were due to SFA in the oil leading to properties such as resistance to oxidation, long shelf life and notable taste and flavor in cooking.[10] Later on it was found that CNO was composed of medium-chain fatty acids (MCFAs) which correspond to 64% of total fat and make it unique and remarkable.[1] In addition to MCFAs, CNO contains phospholipids, tocopherol and other minor constituents. CNO is also referred to as lauric oil since it contains approximately 40–50% of lauric acid of the overall fatty acid (FA) composition.[11–13] The significant presence of lauric acid plays an important role in making CNO chemically unique among other oils.[14]

Numerous research studies have demonstrated the beneficial effects of CNO allaying all the misconceptions that it held due to being a source of SFAs. Health advisors claim that this sensational food has notable functional properties such as hypocholesterolemic, antiobesity, antihepatosteatotic, antioxidant, anti-inflammatory, antimicrobial and HIV preventive activities and cardioprotective effect (Table 1).[15-17] Moreover, CNO exerts antidiabetic property by balancing blood sugar levels. Marten *et al.*[18] showed that medium-chain triglycerides (MCTs) present in CNO improve insulin secretion and insulin sensitivity. Further, CNO is gaining attention as a potential cancer controlling and chemotherapy protective agent.[6] Apart from major health claims, the abundance of vitamin E in CNO aids in moisturizing hair and skin.[19]

Despite the substantial number of studies conducted reinforcing the beneficial health properties of CNO, the hypocholesterolemic and cardioprotective effects of oil remain a matter of controversy.[11,12,20] A recent review, including 21 research studies, stated that CNO is unhealthy in terms of cardioprotection.[12] However, studies by Nevin and Rajamohan,[21] Cox *et al.*[22] and Feranil *et al.*[23] showed CNO consumption has a positive effect on serum lipid profile and hypocholesterolemic effect. Thus, there are limitations in predicting the fact that CNO is completely healthy in terms of cardioprotection, and well-planned studies are needed to clear the disagreements. The main aim of this review is to focus on the composition along with the beneficial functional properties of CNO.

COMPOSITION OF COCONUT OIL

CNO has become one of the most desired oils in the world due to its high degree of saturation and good stability. There are different types of CNO derived from different parts of the coconut. Copra oil is extracted from the dried kernel by mechanical milling and virgin coconut oil (VCO) is extracted from the fresh kernel without high heat or chemical treatment. The oil extracted with isopropyl alcohol from coconut testa is known as coconut testa oil.[24] CNO is predominately composed of SFAs which account for 90% of its composition.[6] In addition to triacylglycerols (TAGs) esterified with component FAs, CNO contains other minor components such as phospholipids, sterols, tocopherols and volatile substances. The presence of these substances plays an important role in modulating the chemical and physical characteristics of CNO.[25] For instance, the melting pattern of CNO, which passes abruptly from solid to a liquid within a short range, is mainly due to the nature of the composition. Also, VCO is found to be healthier than commercial copra oil due to its medium-chain SFA content and higher amounts of polyphenols.[26] Hence, it is vital to understand the relationship of the composition to the exact health benefits exerted by the oil.

Fatty acids

Fats and oils are concentrated forms of energy and the energy is obtained from the complete oxidation of FAs in food. Generally, FAs are classified according to chain length and the degree of saturation. Concerning the degree of saturation, FAs can be classified as SFAs, monounsaturated FAs (MUFAs) and polyunsaturated FAs (PUFAs). Further, based upon chain length they can be subgrouped into short- (C2–C6), medium- (C8–C12) and long-chain (C14–C24) FAs.[27] SFAs are generally considered to be hypercholesterolemic, whereas MUFAs are thought to be mildly hypercholesterolemic and PUFAs are hypocholesterolemic.

CNO contains 92% of SFAs, which is significantly greater than in other commonly consumed vegetable oils. A portion size of 100 g of CNO is found to contain 890 kcal and 82.5 g of saturated fat.[28] Thus, CNO is always classified along with butter, palm oil and animal fats due to its high content of SFAs. Multiple reports confirmed that the major FAs of CNO are lauric (12:0), myristic (14:0) and palmitic (16:0) acids, which represent about 32–51%, 17–21% and 6.9–14%, respectively (Table 2). A comprehensive study by Zambiazi *et al.*[29] also revealed that lauric acid was the major FA present in CNO comprising 45% of total FAs.

Interest in MCFAs in plant oils has grown rapidly over the last few years due to the increasing awareness of their health benefits.[9] In contrast to long-chain fatty acids (LCFAs), MCFAs have smaller molecular sizes and lower melting points. Further, MCFAs are liquid at room temperature and less energy dense (8.4 *versus* 9.2 kcal g^{-1}). These distinct physiochemical characteristics make MCFAs unique in terms of absorption and metabolism compared to LCFAs.[18] As such, they are directly absorbed by the intestine and sent to the liver to be used as energy.[6] Among various plant oils, coconut, palm kernel and babassu oils are the only commercially important sources of MCFAs.[6] According to multiple reports, the health benefits exerted by CNO are attributed to its high content of MCFAs which correspond to 64% of the total FAs.[1,30] A comparative study done on five edible oils, namely sunflower oil (five samples), soybean oil (three samples), palm oil (three samples), mustard oil (five samples), and CNO (six samples), showed that CNO had the highest MCFA content.[27] Further, a comprehensive study involving 14 vegetable oils revealed that CNO had the highest percentage of MCFAs and the lowest percentage of LCFAs.[31]

Phenolic compounds

Phenolic compounds are important phytochemicals that exhibit several bioactive properties including antioxidant activity.[32] Several plant oils are known to be excellent sources of phenolics that can scavenge free radicals produced in our bodies.[33] Unlike other plant oils, studies focused on quantitative and qualitative analysis of phenolic compounds of CNO are less common. According to previous studies, phenolic acids present in CNO are attributed to health benefits such as anti-inflammatory, antihepatosteatotic, antioxidant and chemoprotective activities.[26] However, the method of processing such as wet processing and dry processing plays a significant role in modulating phenolic compounds

Table 1. Significant human and animal intervention studies on beneficial health effects of CNO

Property	Ref.	Study (country)	Intervention	Outcome
Cardioprotective effect/ hypocholesterolemic	53	Randomized crossover trial. Plasma lipoproteins levels were measured after 8 weeks (Thailand)	Group 01: 15 mL VCO Control group: 15 mL 2% carboxymethylcellulose (CMC) solution	VCO-consumed group showed an increase in HDL-C levels compared to control group
Cardioprotective effect/ hypocholesterolemic	51	Randomized trial. Plasma lipoproteins levels were measured after 4 weeks (UK)	Group 01: extra-VCO Group 02: extra-virgin olive oil Group 03: unsalted butter consumed 50 g per day for 4 weeks	Short-term consumption of extra-VCO and extra-virgin olive oil did not show any significant change in serum LDL-C levels
Antiobesity	76	Open-label pilot study. Weight, associated anthropometric parameters and lipid profile were measured one week before and one week after VCO intake (Malaysia)	30 mL of VCO per day was given half an hour before each meal	Waist circumference of men significantly reduced with a mean reduction of 2.86 cm
Antiobesity	77	Randomized, crossover, controlled study. Energy metabolism factors and cardiometabolic risk markers were measured (Brazil)	Group 01: breakfasts containing 25 mL of VCO Control: 25 mL of extra-virgin olive oil	VCO consumption did not acutely change energy metabolism and cardiometabolic risk markers observed
Anticancer	85	Two human lung cancer cell lines (NCI-H1299 and A549) were used. Morphological changes of the cancer cells were observed after treatment (Malaysia)	Two lung cancer cell lines were exposed to series of concentrations of VCO for 72 h	VCO inhibited the growth of cancer cells and induced cell death via the apoptosis pathway at concentrations as low as 8.64% (v/v) and 12.04% (v/v)
Antidiabetic	95	Rat study, 3 groups ($n = 6$). Blood glucose level, serum lipid levels, liver enzymes were observed (India)	Group 1: control Group 2: semi-synthetic diet composed of 60% fructose and CNO Group 3: semi-synthetic diet composed of 60% fructose and VCO	Animals fed VCO diet had only 17% increase in blood glucose level compared to CNO-fed animals which was 46%
Antidiabetic	92	Rat study, 4 groups ($n = 8$). Blood glucose and lipid levels were observed (India)	Group 1: control Group 2: commercial CNO) Group 3: C-VCO Group 4: H-VCO	Overall effect of H-VCO was better than C-VCO in enhancing the antioxidant status, reducing the blood glucose and lipid levels
Anti-inflammatory	100	Rat study, 5 groups ($n = 7$). Carrageenan-induced rat paw edema test was performed to analyze acute inflammation (Malaysia)	1% Tween 80, 100 mg kg^{-1} ASA, VCO_A and VCO_B in 10, 50 or 100% concentrations	Both VCOs exhibited significant ($p < 0.05$) anti-inflammatory activity
Hepatoprotective activity	102	Rat study, 4 groups ($n = 5$). Biochemical parameters, organ weights and coefficients were evaluated (Nigeria)	Group A: control Group B: TMP-SMX at dose of 8/40 mg kg^{-1} body weight twice daily Group C: VCO at dose of 600 mg kg^{-1} body weight per day Group D: TMP-SMX (8/40 mg kg^{-1}) and CNO (600 mg kg^{-1})	VCO was found to ameliorate TMP-SMX-induced effects by restoring the levels of total bilirubin, alkaline phospahatase and lactate dehydrogenase
Antiviral	105	Clinical study of 15 HIV-infected patients, randomly assigned to 3 treatment	1st group: 7.2 g of ML 2nd group: 2.4 g of ML	Thirrd month, 50% showed reduced viral load and by sixth month 8 patients (2 receiving

Table 1. Continued

Property	Ref.	Study (country)	Intervention	Outcome
		groups. Viral, CD4 and CD8 counts and important other health parameters were observed after 3 and 6 months of treatment (Philippines)	3rd group: 50 mL of CNO daily for 6 months	7.2 g ML, 4 receiving 2.4 g ML and 3 receiving, CNO) had a lowered viral count
Antiviral	111	Clinical study of 40 HIV-infected patients, randomly assigned to 2 groups ($n = 20$). $CD4^+$ T lymphocyte counts were taken after 6 weeks of the experiment (Indonesia)	Group 01: VCO 3 × 15 mL/day for 6 weeks Group 02: non-VCO group	VCO supplementation 3 × 15 mL/day for 6 weeks significantly increased $CD4^+$ T lymphocyte concentration in HIV patients compared to non-VCO

Table 2. Fatty acid composition of various CNOs

Fatty acid	CO[96]	RBD[114]	CTO[115]	HEVCO[36]	CEVCO[36]
Caproic acid (C6:0)	—	0.02	—	0.35	0.45
Caprylic acid (C8:0)	9.6	7.24	1.6–3.9	7.87	7.10
Capric acid (C10:0)	6.4	5.25	2.2–3.8	6.07	5.55
Lauric acid (C12:0)	51.5	50.9	32.4–42.9	49.55	50.0
Myristic acid	19.1	21.3	20.2–20.9	17.03	18.01
Palmitic acid (C16:0)	6.9	9.22	11.3–14.1	8.02	7.05
Stearic acid (C18:0)	1.1	0.38	1.2–1.6	2.71	2.42
Linoleic acid (18:1)	4.3	4.81	12.2–17.8	7.01	7.26
Linolenic acid (C18:2)	1.1	0.81	5.3–10.6	1.39	1.66

CO, copra oil; RBD, refined, bleached, deodorized; CTO, coconut testa oil; HEVCO, hot-extracted virgin coconut oil; CEVCO, cold-extracted virgin coconut oil.

present in CNO.[34] Likewise, phenolic content of VCO is perceived to be higher than that of ordinary CNO. Hence, phenolic content in VCO has grabbed the attention of many. Several studies have reported quantitative and qualitative analyses of phenolic compounds present in VCO.[26,34–36] A previous study by Seneviratne and Dissanayake[37] indicated that phenolic compounds such as ferulic acid, *p*-coumaric acid and catechin present in CNO aid in exerting antioxidant activity.[33] Table 3 summarizes the phenolic acid composition of different types of CNOs.

Triacylglycerol

The major TAG species of CNO are CCLa, CLaLa, LaLaLa and LaLaM (where C represents capric, La lauric, M myristic) and the rest represent TAG molecular types that occur at less than 10% level. Owing to their esterification with MCFAs, the dominant TAG molecules of CNO are popularly known as medium-chain TAGs, which contribute to the nutritional significance and functional properties of CNO.[38] The major TAGs of CNO are about 19% of trilaurin (C36) followed by 16% each of diaurylcaprylglycerol (C34) and diaurylmyristylglycerol (C38) and 10% each of lauryldicaprylglycerol (C32) and lauryldimyristyglycerol (C40).[25] Confirming the above, a study by Bezard *et al.*[39] revealed that trilaurin represented10.6% of CNO TAGs. Despite the high content of SFAs, CNO has still become an elite choice for treating certain alarming health conditions and promoting weight loss due to the presence of MCTs.[1]

Phospholipids

Phospholipids are generally found in most natural oils and fats yet the amount and the composition differ depending on the source of origin.[40] As an important functional attribute, they are known to have a stabilizing effect on fatty foods. Moreover, crude CNO has a relatively low amount of phospholipids (0.2%) in comparison to other vegetable oils (1–3%).[25] The major components of the phospholipids present in CNO are phosphatidylcholine (34.6% total phospholipids), phosphatidylethanolamine (24.6%) and phosphatidylinositol (19.0%).[25]

Tocopherols (as vitamin E)

Tocopherols (as vitamin E) are the lipid-soluble natural antioxidants found in most vegetable oils. Among various vegetable oils, CNO is relatively low in tocopherols since it is less prone to autooxidation due to the low degree of unsaturation.[25] Researchers across the globe agreed that occurrence of the greater amount of tocopherols in oils is a self-defense mechanism to protect oils. For instance, Schwartz *et al.*[41] reported that CNO contained the lowest amount of tocopherols (0.32 mg $(100\ g)^{-1}$) when compared to olive oil (18 mg $(100\ g)^{-1}$), extra virgin olive oil (26 mg $(100\ g)^{-1}$), sunflower oil (63 mg $(100\ g)^{-1}$) and corn oil (66 mg $(100\ g)^{-1}$). Yet another study also confirmed that CNO had the lowest amount of tocopherol (1.7 mg $(100\ g)^{-1}$) among various vegetable oils.[42] Despite the low amount of tocopherols

Table 3. Amount of phenolic acids present in various CNOs (mg kg^{-1})

	CO[116]	RBD[37]	VCO[37]	CTO[116]	HEVCO[36]	CEVCO[36]
Total polyphenols	131.2	618	322	313.9	—	—
Protocatechuic acid	—	0.16	—	—	—	—
Phenolic acid						
Gallic acid	24.7	—	—	32.1	25.29	18.01
Hydroxybenzoic acid	7.6	—	—	126.4	—	—
Vanillic acid	63.8	—	2.08	—	1.80	1.03
Syringic acid	17.9	—	0.45	—	—	—
p-Coumaric acid	10.0	0.34	2.0	42.1	0.53	—
Caffeic acid	3.1	0.13	3.0	12.8	1.59	—
Ferulic acid	1.7	0.31	3.3	47.5	12.83	2.36
Cinnamic acid	2.4	—	—	4.1	—	—
Catechin	—	—	—	—	18.15	12.35
Sinapic acid	—	—	—	—	3.35	1.89

CO, copra oil; RBD, refined, bleached, deodorized; VCO, virgin coconut oil; CTO, coconut testa oil; HEVCO, hot-extracted virgin coconut oil; CEVCO, cold-extracted virgin coconut oil.

present in CNO, they prevent air oxidation, and aid in exerting anticancer properties and moisturizing effect on hair and skin.[8,43]

Sterols

In recent years, sterols have received much attention among researchers due to their hypocholesterolemic capacity and potential contribution to a decreased risk of CVD.[44] In edible oils, sterols are primarily in the free and esterified forms.[45] The sterol content of crude CNO was reported to be 100 mg (100 g)$^{-1}$.[35] According to Sabir *etal.*,[46] coconut contains the lowest amount of sterols (0.8 mg g^{-1}) when compared to other edible vegetable oils such as corn oil (23 mg g^{-1}) and soybean oil (9 mg g^{-1}). A comparative study by Schwartz *et al.*[41] also confirmed the lowest sterol content (114 mg (100 g)$^{-1}$) compared to olive oil (283 mg (100 g)$^{-1}$), extra virgin olive oil (256 mg (100 g)$^{-1}$), sunflower oil (451 mg (100 g)$^{-1}$) and corn oil (871 mg (100 g)$^{-1}$). Several studies report the ability of β-sitosterol and stigmasterol to control cancer by inhibiting cancer cells in esophageal tissues, ovaries, breast, colon and prostate. Despite a lower content of sterols, β-sitosterol was found to be the major sterol present in CNO, which corresponds to 70.4% of the total sterol fraction.[25] Supporting the fact, a study by Schwartz *et al.*[41] showed that the sitosterol content of CNO (45 mg (100 g)$^{-1}$) was greater than that of other sterols estimated in the study. Thus, the high content of β-sitosterol helps in understanding the anticancer activity of CNO.

Volatile compounds

The total volatile compounds of crude CNO were reported to be about 900 mg L^{-1}. The two volatile compounds identified were methyl ketones of odd carbon number (CN) from CN-7 to CN-15 and γ-lactones of even carbon number from CN-6 to CN-14.[25] Generally, ketones present in crude oil are a result of microbiological decomposition of FAs in the oil before it was pressed or extracted. This was further confirmed by the analysis of fresh CNO, which indicated the absence of ketones.[12] Also the significant amount of lactones present in crude CNO is believed to be responsible for the flavor and aroma of coconut.[12] Confirming the above fact, a study of Santos *et al.*[47] on volatile organic compounds in VCO and their sensory attributes revealed that lactones impart the coconut-like aroma and the presence of octanoic acid was responsible for rancidity and acid-like aroma in the oil.

FUNCTIONAL PROPERTIES OF COCONUT OIL

Hypocholesterolemic effect and cardioprotective effect

Owing to the perceived health benefits associated with CNO, the use of CNO and related products has increased over the past decade, particularly in the Western market.[20] The main concern limiting the widespread use of CNO is its reported link to the development of CVD. Despite the various health benefits reported for CNO, reports on the relationship between CNO consumption and cardiovascular health are scarce and controversial.

Blood cholesterol is explained in terms of total cholesterol (TC), low-density lipoprotein (LDL-C), high-density lipoprotein (HDL-C) and very-low-density lipoprotein (VLDL-C) cholesterol.[48] Derangements in the blood lipid profile are considered as an important factor indicating increased risk for cardiovascular complications. Human feeding studies have proven the beneficial effect of coconut fats on HDL-C without a doubt. Several studies report coconut fat to increase HDL-C, the so-called 'good cholesterol' in the blood with no significant effect on the TC and LDL-C levels.[6,49–52] Increase in TC and LDL-C is associated with the pathogenesis of cardiovascular complications. In a randomized, controlled crossover trial, involving healthy volunteers aged between 18 and 25, VCO supplementation (30 mL per day for 8 weeks) improved HDL-C, with no change in TC and LDL-C compared to the control (2% carboxylmethylcellulose).[53] Similar type of outcome was reported by Damayanti*et al.*[50] where CNO intervention (35 g per day) for 8 weeks significantly increased HDL-C and reduced the plasma inflammatory markers associated with CVD, the soluble vascular cell adhesion molecule 1 (sVCAM1) and matrix metalloproteinase levels compared to peanut oil intervention. Voon *et al.*[54] conducted a randomized crossover study in 45 healthy Malaysian adults to investigate the effect of virgin olive oil, palm olein and CNO consumption for 5 weeks on thrombogenicity indices and cell adhesion molecules. The three test fat diets did not affect thromboxane B2 (TXB2), TXB2/PGF1α ratios and soluble intracellular cell adhesion molecule and sVCAM levels significantly. However, the olive oil diet effectively reduced the plasma

proinflammatory LTB4 levels compared to the other two test diets.

A recent randomized trial conducted with 94 men and women (healthy) in the UK to study the effect of daily consumption of 50 g of extra-VCO or extra-virgin olive oil or unsalted butter for 4 weeks showed no significant difference in the serum LDL-C levels between short-term consumption of extra-VCO and extra-virgin olive oil. LDL-C level was found to be significantly higher in the butter treated group compared to those treated with the extra-VCO and extra-virgin olive oil.[51] Also, CNO treatment significantly increased the HDL-C level compared to the other two test diets.[51] Extra-VCO is reported to retain the phenolic compounds better than standard CNO. Polyphenols present in VCO are identified as a great source, which can help in attenuating CVD risk factors.[55–57] A study to evaluate the beneficial effects of VCO over copra oil revealed that VCO was more effective in improving the serum lipid profile (TC, triglycerides, phospholipids, LDL-C and VLDL-C levels and increased HDL-C in serum and tissues) than copra oil.[58] The positive outcome was attributed to the occurrence of high polyphenolic content in VCO.

Contrary to studies reporting a cardioprotective effect of coconut consumption, several studies also report a negative impact of CNO on the biomarkers of CVD. According to a review presented by Eyres *et al.*,[12] covering 8 clinical trials and 13 observational studies, CNO was found to raise TC and LDL-C in general, compared to other plant oils with unsaturated FAs, but not as much as butter. Two recent systematic reviews indicated CNO consumption to significantly increase LDL-C and HDL-C levels.[59,60] For instance, Neelakantan*et al.*[59] indicated that CNO consumption would increase LDL-C and HDL-C levels by 10.47 and 4.00 mg dL^{-1}, respectively, compared to non-tropical vegetable oils. According to Teng *et al.*,[60] CNO significantly raised LDL-C by 0.26 mg dL^{-1} and reduced HDL-C by 0.37 mg dL^{-1} in comparison to animal fats. Feranil *et al.*[23] examined the association between CNO consumption and lipid profiles in a cross-sectional, community-based study involving 1839 pre-menopausal Filipino women (aged 35–69 years). According to that study, the levels of TC, LDL-C, HDL-C and triglycerides were found to increase with increasing CNO consumption. In another study, Cox *et al.*[61] compared the effect of CNO, safflower, and butter consumption on the blood lipid levels of healthy and moderately hypercholesterolemic individuals.[62] The test was designed in a way to derive at least 50% of the total fat energy from the test fat. Both studies demonstrated the consumption of CNO to significantly increase serum LDL-C levels compared with safflower oil. However, the LDL-C levels of these groups were significantly lower than that of the butter group. Another study comparing effects of palm olein, corn oil and CNO on serum lipids of healthy subjects (75% of the fat calories from test fats) reported CNO to increase the TC levels by over 10% compared to the baseline levels, whereas palm olein and corn oil significantly reduced TC, LDL-C and HDL-C serum levels.

Mounting evidence suggests the type of fat to have a major effect on health outcomes.[63,64] SFAs are generally considered to be hypercholesterolemic, whereas MUFAs and PUFAs are considered hypocholesterolemic. According to studies published so far, the replacement of saturated fats with unsaturated fats (especially polyunsaturated fats) is reported to improve blood lipid profile (reduce TC and LDL-C and increase HDL-C). The American Heart Association Presidential Advisory Report suggests the replacement of 5% of energy intake derived from saturated fats with polyunsaturated fats or monounsaturated fats to lower the incidence or risk of CHD by 25% and 15%, respectively.[63] The 2015–2020 Dietary Guidelines for Americans suggests reducing saturated fat consumption to less than 10% of total energy intake while replacing them with unsaturated fats, especially PUFAs.[63,64] In support of this statement, Mendis *et al.*[65,66] reported the replacement of saturated fats with unsaturated fats to favorably modulate the lipid profile. In a feeding trial carried out on 25 healthy Sri Lankan male prison inmates, CNO intake for 8 weeks was associated with increased LDL-C levels (non-significant) compared to soybean oil.[66] Another study reported lowering of dietary saturated fat (derived from coconut) or partly replacing it with unsaturated fats to have a favorable effect on serum lipoprotein levels.[65] However, some studies reported contrasting results to the above observation. For example, Damayanti *et al.*[50] reported CNO to improve the CVD markers better than peanut oil, which was comprised of 39% MUFA (oleic acid) and 31.8% PUFA (linoleic acid), whereas the unsaturation level in CNO were only around 6.8%. Similar outcomes have been reported with regards to soybean oil, a major source of PUFAs, linoleic acid (omega-6) and α-linolenic acid (omega-3).[13] Assunção *et al.*[13] reported soybean oil supplementation (30 mL) for 12 weeks to increase TC and LDL-C and to reduce HDL-C significantly in women aged 20–40, whilst CNO increased HDL-C, with no change in TC or LDL-C. In addition, in a study done by Voon *et al.*[67] aimed at determining the effect of palm olein, CNO and olive diet on plasma homocysteine and inflammatory markers, no difference was found in the total homocysteine level and the inflammatory markers TNF-α, IL-1β, IL-6 and IL-8, high-sensitivity C-reactive protein and interferon-γ.

The effect of dietary fats on CVD risk factors does not seem to depend solely on the saturation level of the FAs, but it may also depend on the FA composition, processing methods and dietary patterns. Owing to the high content of SFAs (92%), CNO has always been classified along with butter, palm oil and animal fats, and is not advocated as a healthy dietary choice. A well-established misconception concerning CNO is that since coconut fats are saturated, they are believed to elevate plasma lipids in a manner similar to that of animal fats, such as butter. It should be noted that CNO is different from palm oil and animal fats in terms of its FA composition and TAG profile. Several studies have reported the favorable effect of coconut fats on cardiovascular markers over animal fats.[51,60–62] The SFA profiles of CNO vastly differ from those of animal fats, where CNO is predominantly made of lauric acid (*ca* 45–53%), while the major FAs of butter are palmitic and stearic acids.[14,51] Studies done on lauric acid metabolism indicate that unlike other saturated fats, lauric acid is rapidly absorbed, directly transported to the liver and oxidized to produce energy rather than being stored as fat.[14] This phenomenon probably explains the differential effects exhibited by coconut fats compared to other saturated fats. However, it should be noted that there are controversial results published in this regard.[14,68,69] Although lauric acid is classified as a MCT, some report it to behave as a long-chain triglyceride in terms of solubility and intestinal absorption.[12,20]

Overall, the studies done so far on the association between CNO consumption and cardiometabolic health have reported conflicting outcomes. Majority of the studies are found to lack a proper study design and the evidence supporting the beneficial effect of CNO consumption on cardiometabolic health is vague and not informative enough to support the claim. For instance, a recent study to evaluate the effect of cooking oil such as CNO and sunflower oil on the blood lipid profile of patients with coronary artery disease over a period of 2 years found no significant

difference in the blood lipid profile of the two study groups. However, the point to consider here is that the study participants were continuously on statin therapy, which makes it hard to interpret the study outcome.[70] Although CNO increases the HDL-C level, unfortunately, there is no proven evidence stating the positive effect of high HDL-C on CVD outcomes as does the decrease in LDL-C. The causal relationship between LDL-C and CVD risk is well established, where 1 mmol L^{-1} increase in LDL-C concentration is reported to increase the risk by 15%.[51] This was clearly mentioned in a report published by the American Heart Association.[63] However, from the evidence available so far, there is no strong reason to link CNO consumption directly with CVD incidence or otherwise. For decades, some nations have been consuming CNO and related products with low CVD incidence. For example, despite being a nation that derives its dietary fat mainly from CNO, Sri Lanka had the lowest death rate from ischemic heart diseases as quoted in the Demographic Yearbook of the United Nations in 1978.[49] Although, the *per capita* consumption of coconut in Sri Lanka was around 130 nuts per person per year in the 1960s, it has dropped to around 100–110 nuts per person per year in the recent times.[71] On the other hand, the CVD incidence in Sri Lanka was found to increase at an alarming rate compared to developed nations.[71] This shows that coconut consumption alone is not the reason for the increasing trend of CVD incidence. According to health authorities, changes in dietary habits and lifestyle factors over the years have played a major role in the increase of CVD incidence. In general, consumption of any dietary FA in excess may not be desirable, and moderately balanced inclusion of dietary FAs in day-to-day diet is encouraged.

Antiobesity effect

Obesity has become a serious health issue around the world due to changing lifestyles and dietary habits. It has reached epidemic levels in many countries representing a severe public health problem.[13] The most interesting argument for weight control of CNO over other oils is that the energy expenditure of MCFAs is greater than LCFAs. CNO contains a larger proportion of water-soluble MCTs which are easily hydrolyzed by lipase and absorbed through the intestine and directly sent to the liver to be rapidly metabolized into energy without storing in adipose tissue.[72] Hence, this is thought to decrease the basal metabolic rate.[13,72,73] Although CNO has recently attracted attention as a source of weight loss, still, it is a controversial topic due to the effect of SFAs and their association with CVD.

Studies have indicated some promising outcomes on the use of CNO as a MCT oil to promote weight loss, despite its high content of saturated fat.[1] However, the FA profile of CNO is different from those of the MCT oils used in studies. The majority of MCTs in CNO comes from lauric acid. Classification of lauric acid as a MCFA is still controversial due to clinical studies pointing out the lower percentage of lauric acid directly transported to the liver.[73] However, Kinsella *et al.*[74] revealed that CNO cannot be promoted as a MCT oil since it has a different effect on food intake and satiety. In support, Maher*et al.*[75] also reported that MCT oils are better in fullness perception compared to CNO. Nevertheless, few clinical studies of the impact of CNO consumption on body weight control showed positive outcomes. In a study of 40 women with abdominal obesity, supplementing with 30 mL (2 tablespoons) of CNO per day led to a significant reduction in both body mass index and waist circumference within 12 weeks.[13]

A pilot study by Liau *et al.*[76] reported a dosage of 30 mL of VCO for a period of four weeks reduced the weight circumference significantly among male subjects. But according to the results, there was no change in lipid profile among the subjects. Hence the study failed to conclude an impact of VCO consumption on weight control. Further, Xavier *et al.*[77] evaluated the effect of consumption of VCO and extra-virgin olive oil on energy metabolism, fat oxidation rates and cardiometabolic risk markers in 17 women aged between 19 and 42. The authors reported that there was no difference in the above parameters studied between the two groups yet lower hunger suppression, satiety and total fullness were observed for the VCO-ingested group. Still, the experimental duration in the study brought a limitation in concluding the beneficial effect of CNO. According to a review by Clegg[9] on CNO consumption and weight loss, there is not enough evidence to support the beneficial effect of CNO consumption and weight loss. Therefore, consumption of CNO as a weight-controlling method remains unsupported by enough scientific evidence.[73]

Antidiabetic property

There is a general belief that saturated fats might induce insulin resistance in humans, which leads to the development of metabolic disorders such as diabetes. On the contrary, VCO is found to exert an antidiabetic effect by balancing blood sugar levels.[90] A comparative study by Siddalingaswamy *et al.*[91] on the protective potential in a streptozotocin-induced diabetic model using hot-extracted VCO (H-VCO), cold-pressed VCO and commercial CNO showed H-VCO to be a better hypoglycemic and insulin-sensitizing agent comparing to the other CNOs used. Iranloye *et al.*[92] and Madin and Ahamed[93] showed that fermented VCO (F-VCO) effectively reduces hyperglycemia in alloxan-induced diabetic rats. Similarly, Narayanankutty *et al.*[94] also found F-VCO to prevent the development of insulin resistance and dyslipidemia in high-fructose-fed rats. However, detailed scientific investigations on the mechanism of the antidiabetic effect of different types of VCOs are scarce. However, Lekshmi *et al.*[95] have stated that the presence of phenolic acids in VCOs aids in the inhibition of dipeptidyl peptidase-4 or insulin sensitization. In addition, the presence of phenolic compounds in VCOs is believed to offer protection against secondary diabetic complications such as diabetic nephropathy by inhibiting the polyol pathway. A study by Akinnuga *et al.*[96] showed that F-VCO could exert a protective effect on diabetic nephropathy in animals.

Anticancer property and chemotherapy protective effect

Some studies reported the anticancer activity of CNO against mammary, colon, liver, lung and oral cancer cells.[24] supporting this, a study done by Salerno and Smith[79] showed that CNO rich in lauric and palmitic acids had a greater inhibition effect on HT-29 malignant human colon cells than linoleic acid. Further, in a separate study, Enos *et al.*[80] observed that a diet rich in CNO efficiently reduced ulcerative colitis and associated colon cancer incidence in an azoxymethane/dextran sodium sulfate-induced colon cancer model. It has been found that treatment with CNO increases the levels of intestinal protein mucin 2, which is involved in the proper maintenance of intestinal barrier integrity. In the *in vitro* system, lauric acid, which is the major form of FA in VCO, has recently been shown to induce apoptotic changes in various colorectal cancer cells and breast and endometrial cancer cells mediated by reactive oxygen species.[81,82] Lauric acid, the predominant FA in CNO, showed cytotoxicity towards HCT-15 (human colon cancer), HepG2 (human hepatocellular carcinoma) and Raw 264.7 (murine macrophages) cells *in silico* and *in vitro*.[83] Moreover, CNOs have shown an inhibitory effect of mammary

tumorigenesis in experimental animal models. In a separate study, VCO, processed CNO and fractionated CNO have shown anticancer activity towards liver and oral cancer cells. Further, Kamalaldin *et al.*[84] reported that VCO induced apoptosis in NCI-H1299 and A549 lung cancer cell lines, and was safe to be consumed. Distinctive morphological changes, such as the appearance of massive cytoplasmic vacuolization and blebbing of the cell membrane, were observed in both cell lines after treatment with VCO.

Apart from anticancer properties, VCO is emerging as a functional oil to ameliorate the undesirable side effects associated with chemotherapy. In an animal study, F-VCO effectively ameliorated the myelosuppression and disturbed antioxidant status induced by the chemotherapeutic drug cyclophosphamide.[85] Further, methotrexate-induced hepatotoxicity and oxidative damage have been reduced by F-VCO via improving antioxidant status in rats.[86] In another study, oxidative nephrotoxicity induced by methotrexate was reduced by antioxidative and anti-inflammatory effects of VCO.[87] Further the chemotherapy-induced side effects of breast cancer patients are shown to improve with the consumption of VCO.[88] Similarly VCO-incorporated mouthwash has been shown to reduce mucositis caused by radiation in nasopharyngeal carcinoma patients.[89] Bioactive polyphenols in VCO may be responsible for these observations and more comprehensive studies are needed to investigate the anticancer properties and chemoprotective effect of CNO.[86]

Antioxidant and anti-inflammatory activities

Free radicals adversely affect biomolecules such as proteins, lipids and DNA and trigger oxidative stress. Antioxidants are compounds capable of either delaying or inhibiting the oxidation process by scavenging free radicals.[97] Several studies have been performed to analyze the antioxidant property of highly consumed vegetable oils including CNO. When compared to other oils, the number of studies of the antioxidant activity of CNO is limited. Nevertheless, VCO is attracting attention as oil with high antioxidant activity. A comparative study by Janu *et al.*[33] to investigate the total phenolic content and antioxidant potential of commonly consumed vegetable oils, namely CNO, sunflower oil (SFO), rice bran oil (RBO), groundnut oil (GNO), sesame oil (SESO) and mustard oil (MO), showed the order GNO > CNO > RBO > MO > SFO > SESO, and the study revealed that GNO, CNO and RBO had a higher potency towards free radicals.[33]

Recent studies have indicated that unrefined VCO be given priority due to its notable health benefits. Among the various VCO preparations, F-VCO and H-VCO showed higher radical scavenging and inhibition activity.[24] Unlike ordinary CNO, VCO also has been shown to have the capability of increasing antioxidant enzymes and reducing lipid peroxidation content. Nevin and Rajamohan[98] investigated the effect of VCO in comparison to CNO and ground oil on both *in vitro* and *in vivo* lipid peroxidation. Results showed that consumption of VCO extracted from fresh coconut meat, with its high content of biologically active components, was superior in antioxidant property than ordinary CNO extracted by dry process.

In addition to antioxidant activity, the anti-inflammatory activity of VCO was proven several years ago. Inflammation involves many processes of the immune system; for example, during both acute and chronic inflammatory responses, the immunological component cells are activated in response to foreign organisms or antigenic substances.[2] Certain research studies have shown that VCO tends to increase antioxidant enzymes and decrease the expression of inflammatory genes such as COX-2, iNOS and IL-6. Intahphuak *et al.*[2] reported that a dosage of 4 mg (20 μL)$^{-1}$ of VCO showed moderate anti-inflammatory effects on ethyl phenylpropionate-induced ear edema in rats. However, the efficacy of VCO was not higher than that of the standard drug indomethacin. Further, Zakaria *et al.*[99] using *in vivo* models observed that F-VCO efficiently reduces acute inflammation, whereas, in chronic models, it was found to be less effective. There should be more studies focused on the anti-inflammatory activity of different types of CNOs for better understanding of the property with scientific support.

Inhibition and reversal of hepatosteatosis

The liver is an important organ dealing with detoxification and elimination of wastes and toxic products of metabolism. During this process, hepatocytes are known to become damaged. Ingestion of drugs and chemicals through diets are known to harm hepatocytes adversely leading to hepatotoxicity. Hepatosteatosis, a form of non-alcoholic fatty liver disease, is recognized as a major global issue. Hence, the trend towards discovery of dietary nutraceuticals to prevent this condition is increasing. VCO was found to reduce the effect of paracetamol-induced toxicity by restoring liver function markers and hepatic morphology. Zakaria *et al.*[100] found that elevated levels of hepatic damage serum markers (AST, ALT, ALP) due to paracetamol-induced toxicity were reduced at the highest concentration (10 mL kg^{-1}) of VCO used. Similarly, Otuechere*et al.*[101] found that common antibiotic trimethoprim-sulfamethoxazole (TMP-SMX)-induced toxicity is also reduced by cold-pressed VCO intake. Supplementation of VCO ameliorated TMP-SMX-induced toxicity by restoring the levels of total bilirubin, alkaline phosphatase and lactate dehydrogenase. According to a study by Narayanankutty*et al.*,[78] reversal of hepatosteatosis in male Wistar rats fed a high-fructose diet was observed in 4 weeks. In that study, administration of VCO caused the natural reversion of established hepatosteatosis condition by improving HDL-C level and reducing hepatic and serum triglycerides. Based on studies done so far, it can be concluded that VCOs, regardless of the differences in their methods of preparation, exhibit a promising hepatoprotective effect. This hepatoprotective effect of VCO may be partly attributed to its antioxidant activity. Furthermore, studies are needed before exact conclusions can be drawn on the actual hepatoprotective activity of CNO.

Antimicrobial activity

A series of studies reported in the 1970s that MCFAs with 6–12 carbons are responsible for potent activity towards Gram-positive bacteria, lipid-coated viruses as well as fungi and protozoa.[16,102–103] The presence of 12-carbon lauric acid makes the oil potent towards microbes.[13] According to multiple reports, particularly lauric acid (C12:0) in its monoglyceride form (monolaurin or ML) was found to be responsible for antimicrobial properties.[14,104–105]According to studies reported so far, CNO was identified as an effective source against lipid-coated microorganisms such as visna virus, cytomegalovirus, influenza virus, leukemia virus, pneumono virus and hepatitis C virus.[102] Moreover, the presence of ML has broadened the antimicrobial spectrum to some fungal species such as *Aspergillus* sp., *Penicillium* sp., *Cladosporium* sp., *Fusarium* sp. and *Candida albicans*. A study by Ríháková *et al.*[106] using CNO as a monoglycerol source for antifungal activity showed that CNO could be used as a preservative with antifungal activity. Further, Kannan and Mohammed[107] revealed that there is a strong potential therapeutic value of VCO against *Candida* sp. which is one of the common

causes of oral candidiasis in the world. Shino *et al.*[108] performed a study using two types of CNO, namely activated VCO and crude extract of VCO, and showed that exposure of *C. albicans* to activated VCO inhibited its growth.

CNO was found to exhibit antibacterial activity against *Pseudomonas aeruginosa*, *Escherichia coli*, *Proteus vulgaris* and *Bacillus subtilis*.[72] Manohar *et al.*[16] conducted a comparative rodent study using two types of coconut oil, namely refined, bleached, deodorized (RBD) CNO and VCO, and found that these two types of CNO were less effective than pure ML towards *Staphylococcus* organism. However, Oyi *et al.*[17] formulated a cream using CNO which was found to exhibit both antibacterial and antifungal properties. Hence, the study demonstrated the importance of formulating CNO into a cream. Emulsifying agents present in the cream aid the penetration of active compound ML present in CNO. Bacterial resistance to antibiotics is considered an important public health issue because of the potential effect on ecosystems and human health. A novel study done infusing nanotechnology with CNO has addressed this issue. Low-cost eco-friendly silver nanoparticles made using CNO were found to display antibacterial activity. The silver nanoparticles reduced the growth rate of multi-antibiotic-resistant bacteria such as *Citrobacter* sp., *Aeromonas* sp. and *Acinetobacter* sp.[109]

In addition to antifungal and antibacterial activities, CNO is attracting attention due to its potency towards HIV. According to reports, 42 million people in the world have been affected by HIV/AIDS for 22 years.[11] The first ever clinical trial of ML on 15 HIV-infected patients was conducted at the San Lazaro Hospital, Manila; these patients never received any anti-HIV medication. The study was conducted by assigning three random groups based on the amount of oil given (7.2 g of ML, 2.4 g of ML and 50 mL of CNO daily for 6 months). Viral, CD4 and CDS counts, complete blood counts, blood lipids and liver and kidney function were determined before and after 3 and 6 months of treatment. The results showed a reduction in the viral load of 50% of the patient by 3 months. When the study was continued to 6 months, eight patients (two receiving 7.2 g of ML, four receiving 2.4 g of ML and three receiving CNO) showed favorable effects without any serious side effects.[104] Further, a study was conducted with 40 HIV subjects with $CD4^+$ T lymphocyte counts of less than 200 cells μL^{-1} divided into two as VCO group (45 mL daily) and control group (no VCO). The VCO group showed significantly higher average $CD4^+$ T lymphocyte counts compared to control group after 6 weeks.[110] The demonstrated antiviral ability of CNO due to the presence of lauric acid has prompted scientists to start clinical experiments on its potential as a treatment for nCoV-2019.[111]

OTHER PROPERTIES

Moisturizing skin and hair

It is found that CNO has a beneficial impact on external parts of the body such as hair and skin. People in the tropics have used CNO as a natural moisturizer for centuries. In Ayurvedic medicine, CNO has been used to treat numerous skin disorders which include wound healing and microbial infections.[19] Nevin and Rajamohan[19] have shown the beneficial effect of VCO for the healing of dermal wounds in rats. Further, CNO is applied as a remedy to heal the pain of burn wounds.[2] A comparative study done to determine the efficacy of VCO and mineral oil as therapeutic moisturizers for mild to moderate xerosis showed that both oils had considerable hydration ability on the skin and increased the skin surface lipid levels. Grading of xerosis by the investigators and visual analogue scales used by patients showed greater improvement for CNO than mineral oil.[112]

CNO is found to have a strong affinity for hair proteins and it easily penetrates the hair shaft due to its low molecular weight and straight linear chain.[43] Supporting this, a study done using mineral oil, CNO and sunflower oil on prevention of hair damage showed that CNO was the only oil to show a marked decline in protein loss for both damaged and undamaged hair when it was used as a pre-washed or post-washed product. This observation was explained in terms of the FA composition of CNO. Lauric acid, the principal FA, leads to CNO being a hair-enriching oil compared to other oils tested.[43] Ruetsch *et al.*[113] investigated on the penetration of CNO and mineral oil into human hair fibers. The study showed a higher penetration of CNO compared to mineral oil. The observation was supported by the polar nature of CNO compared to mineral oil due to the presence of MCFAs and a certain amount of MCTs. Moreover, CNO is principally a lauric acid triglyceride oil. Thus, the lower molecular weight (lower than 1000 Da) of the oil means that it easily penetrates the hair shaft. Also, that study indicated that CNO provides better hair protection from damage by hygral fatigue.

CONCLUSIONS

CNO is a tropical oil that is consumed in many Asian region countries. Though it was criticized for its adverse health effects in the early days due to the presence of SFAs, recent studies claim the positive health effects of CNO consumption owing to the presence of MCTs. Recent *in vivo* and *in vitro* studies reveal the ability of CNO to suppress certain adverse health issues such as cancer, hepatosteatosis, HIV and diabetes. Furthermore, CNO is found to possess antioxidant, antiobesity, antimicrobial and anti-inflammatory properties. However, the findings on hypocholesterolemic and the cardioprotective effects of CNO are inconclusive. Several studies based on Asian communities support the cardioprotective effect of CNO, but the real value of CNO is obscure due to the limited number of research studies done in Western countries. Hence, properly planned long-term studies may help in understanding the impact of CNO consumption on cardiac health.

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CONFLICT OF INTEREST

The authors have declared that there is no conflict of interest.

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