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Perspectives on the cardioprotective, neuroprotective and anti-obesity functions of coconut (*Cocos nucifera* L.)

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ABSTRACT

Widely acclaimed as the "tree of life," the coconut is intrinsically woven into human culture as a source of food, shelter, and medicine. Coconut oil and its derivatives have found diverse applications in both food and industry. Of late, the consumption of coconut in the form of oil and its derivatives has been proven to be beneficial. Taking into account the most recent clinical evidences, an attempt was made to present a concise review of the cardioprotective, neuroprotective, and anti-obesity effects of coconut and its derivatives. Our analysis reveals that research evidence supports the cardioprotective and neuroprotective effects of coconut. Numerous clinical trials have proven the anti-obesity and hypoglycemic effects of coconut oil and products. High contents of myocardial anti-oxidants, and differential metabolism of medium chain fatty acids (MCFAs) provide cardioprotective effects. The ketogenic effect of coconut derived products confers neuroprotective measures and enhanced energy expenditure in the metabolism of MCFAs and polyphenolic anti-oxidants are suggested to offer anti-obesity effects. Nevertheless, further research with more randomized, controlled, large clinical trials that evaluate the optimal dosage and side effects, if any, are warranted. Based on this comprehensive review, it is understood that MCFAs are the key component, apart from the phytochemicals such as polyphenols, tocopherols, and other antioxidants that accord these health benefits. Overall, the health benefits of coconut and its derivatives are perceptible, though there is a need for long-term clinical trials. Also a shift of research focus from coconut fatty acids and oil to other phytochemicals and to design appropriate clinical and epidemiological studies to discover coconut biomolecules of health importance is warranted.

1. Introduction

Coconut palm (*Cocos nucifera* L.) is a versatile tropical crop grown in more than 97 countries. It is associated with human culture and tradition in various ways as a source of food, shelter, and medicine. It provides an array of edible products, namely coconut oil, virgin coconut oil (VCO), tender coconut, coconut water, coconut inflorescence sap, etc. Oils derived from coconut, such as coconut oil and VCO, are known as one of nature's healthiest and purest dietary oils (Beegum et al., 2021). Coconut oil is extracted from the dried kernel (copra), which can be considered as a crude oil; on the other hand, VCO is the pure form of oil, extracted from the wet processing of coconut milk. The various methods of preparation (such as wet processing through hot processing, fermentation, and centrifugation, and dry processing through dry gratings) of VCO and the resultant biochemical attributes of the oil have been described (Ramesh et al., 2020). The multivariate analysis of biochemical features of VCOs revealed that VCO from hot-process belongs to a group with high total phenolic and flavonoid content and consequently strong antioxidant capacity. The fatty acid composition of coconut is the prime biochemical attribute responsible for its potential health benefits. Dorni et al. (2018) have compared the fatty acid profiles of various edible oils and fats and observed that every kind of edible oil show its unique fatty acid profile with significant variations in the contents of each individual fatty acid. The highest total saturated fatty

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Table 1
Bioactive components in coconut-derived oils.

Biochemical features	Virgin coconut oil	Coconut oil
Fatty acid composition (%)		
C6:0 Caproic acid	0.6	0.50
C8:0 Caprylic acid	0.8	6.76
C10:0 Capric acid	6.4	6.37
C12:0 Lauric acid	48.5	47.10
C14:0 Myristic acid	17.6	17.6
C16:0 Palmitic acid	8.4	8.80
C18:0 Stearic acid	2.5	3.03
C18:1 Oleic acid	6.5	6.45
C18:2 Linoleic acid	1.5	1.45
C18:3 Linolenic acid	–	0.27
SFAs (%)	84.8	89.75
MUFAs (%)	6.5	6.45
PUFAs (%)	1.5	1.72
MCFAs (%)	56.3	60.73
Tocopherols (μg/g)	15–30	2–6
Phytosterols (μg/g)	2.5–3.0	0.5–1.0
Total polyphenols content (μg/g)	500–700	150–250
Anti-oxidant potential (%)	75–90	35–45

(Source: Ramesh et al., 2021)

acid (SFA) was observed in coconut oil (ie. 90.84%, 7.24% monounsaturated FA, and 1.90% polyunsaturated FA) among corn oil, cotton seed oil, groundnut oil, sesame oil, palmolein, rice bran oil, safflower oil, soybean oil, sunflower oil and mustard oil. Moreover, 50% of SFA in coconut oil was lauric acid (followed by myristic acid at 21.12%), which is quite unique to coconut oil. As already stated, majority of the fatty acids are medium-chain fatty acids (MCFAs), which offers several health benefits. In addition, these MCFAs, characterized by 6–12 carbon chains, and exhibit good digestibility (Myrie & Jones, 2011) compared to the long-chain triglycerides (LCTs) of other vegetable oils. MCFAs have 6–12 carbon chains and the MCFAs of coconut oil are primarily lauric acid (45.0%–53.0%), caprylic acid (8.0%–9.0%), capric acid (5.0%–7.0%), and caproic acid (0.80%–0.95%) (Prasanth Kumar and Gopala Krishna, 2015; Nasir et al., 2018; Ramesh et al., 2021). In addition to SFAs, coconut oil comprises a small amount of polyunsaturated and monounsaturated fatty acids. These MCFAs, ensures their rapid dietary absorption compared to the long chain fatty acids (LCFAs) found in other vegetable oils (Dayrit, 2015). Lauric acid has a tendency to bypass the lymphatic system and enter the portal venous transport system of fatty acids (Dayrit, 2015). The MCFA diet (in which 28% of total calories and 74% of total fat are from MCFAs) mimics starvation (due to the high consumption of lipids and very low intake of carbohydrates), ultimately reducing blood glucose concentrations. This is because MCFAs are rapidly metabolized in the liver, producing ketone bodies including acetones. These ketone bodies act as an alternative source of energy for the brain cells. They have potential therapeutic effects, especially on the brain. Besides, most ketogenic MCFA are caprylic acid (C8) and capric acid (C10), which are found in coconut oil (Jensen et al., 2018). The effects of MCFAs may be through direct receptor-mediated intracellular pathways and metabolic regulators that can alter the levels of circulating hormones and metabolites, and hence indirectly regulate body metabolism. MCFA-enriched diets could therefore be used to manage metabolic diseases through the modification of gut microbiota, activation of GPR 40 and GPR 84-mediated cellular regulation pathways (Jensen et al., 2018; Roopashree et al., 2021). In addition, possible mechanisms of action of lauric acid against diseases such as diabetes, epilepsy, cancer, etc. have started emerging. Coconut oil and VCO also have tocopherols, phytosterols, phenolics, and antioxidant activity (Marina et al., 2009; Ramesh et al., 2020). The total phenolics range from 0.45 mg/100 g to 2.87 mg/100 g in VCO and 1.95 mg/100 g in coconut oil. The antioxidant capacity of VCO was found to be in the range of 3.87 mM Trolox equivalent (TE) to 11.31 mM TE, and that of coconut oil was 9.01 mM TE (Ramesh et al., 2020). The antioxidant capacity of VCO is attributed to its phenolic compounds (Tinkov et al., 2015). Table 1 shows the bioactive components of coconut and its derivative. All these functional bioactive compounds collectively bestow multiple therapeutic properties, including cardioprotective, neuroprotective, and anti-obesity functions to coconut oil and coconut derivatives. The antimicrobial, oral protective and anti-diabetic functions of coconut and its derivatives have been reviewed elsewhere (Beegum et al., 2021). In this

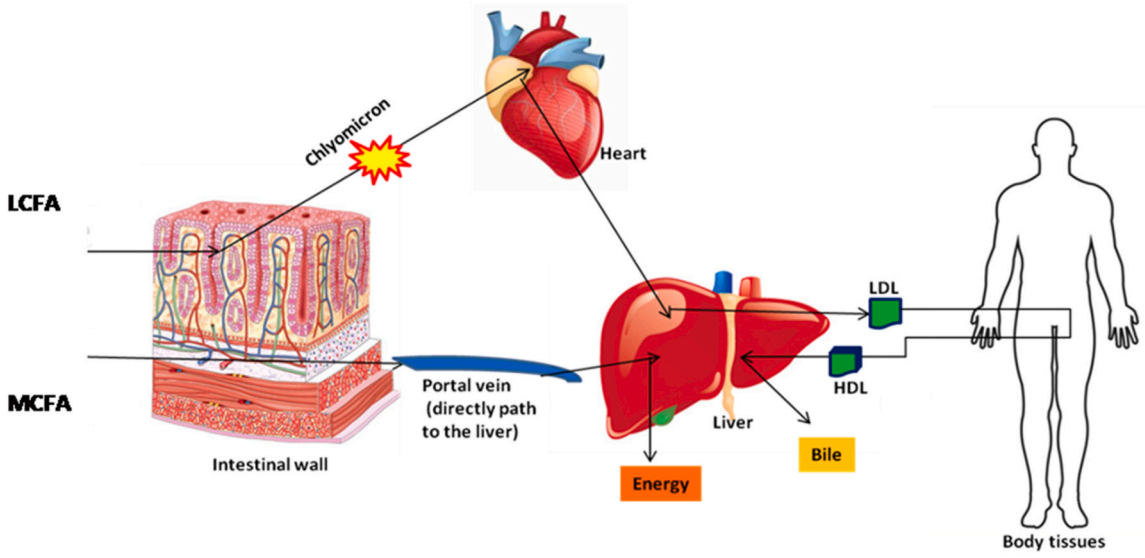

Fig. 1. Metabolic fate of various classes of fatty acids (medium chain fatty acids-MCFAs, long chain fatty acids-LCFAs) derived from coconut oil.

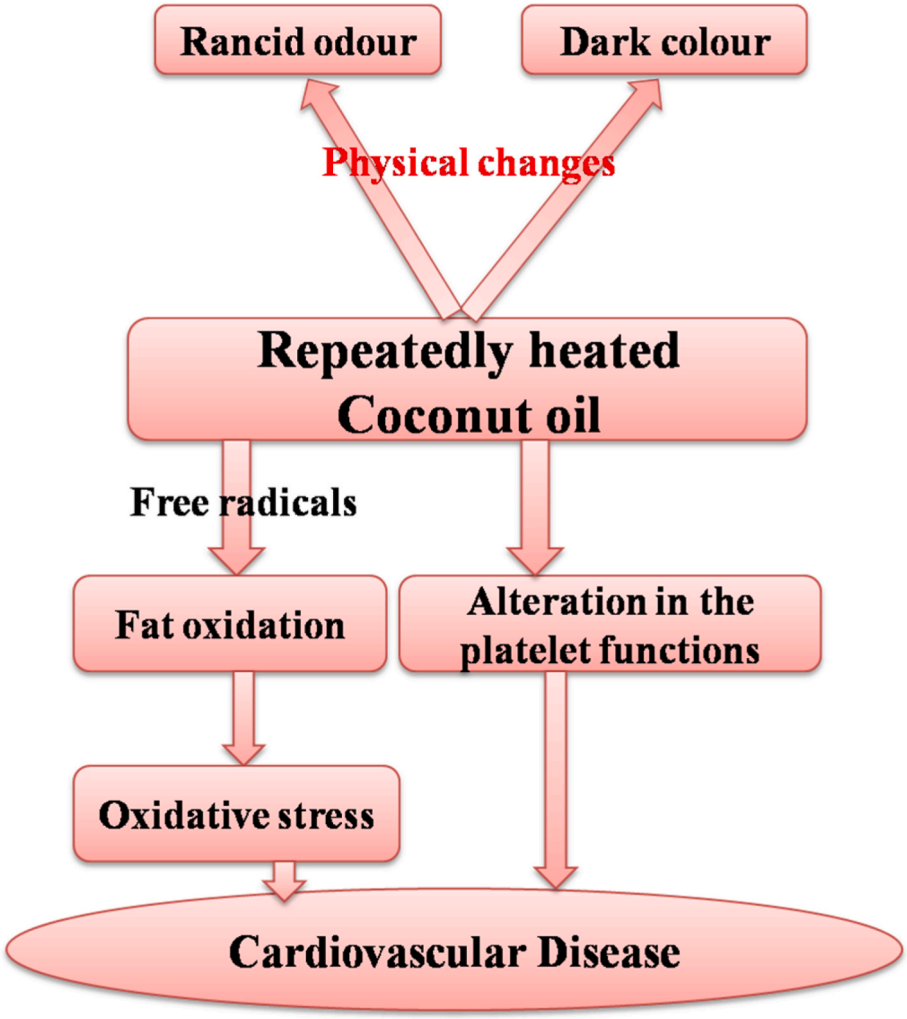

Fig. 2. Fate of repeatedly heated coconut oil and its implications on human cardiovascular health.

backdrop, this review aims to comprehensively examine the cardio-protective, neuro-protective, and anti-obesity functions of coconut and its derivatives, shedding light on their possible mechanisms of action.

2. Coconut derivatives against coronary heart disease (CHD)

Coronary heart disease (CHD) is one of the leading causes of death globally, and a significant number of these deaths are due to coronary artery disease (CAD), caused by atherosclerotic disease due to high lipid levels (Sekhar, Ravinarayan, et al., 2022). It is one of the major health concerns as it accounts for over 30% of all deaths globally (18 million deaths per year) (WHO, 2021). Multiple factors, including elevated blood pressure, high cholesterol, diabetes mellitus, obesity, smoking, lifestyle, age, family genetics, etc., have been ascribed with the development of CHD (Hajar, 2017). Among the various factors, high blood cholesterol levels are attributed to the increased risk of CHD. For many decades, saturated fatty acids (SFA) in the majority of fats and oils have been considered harmful because they have been linked to CHD and stroke. Ancel Keys' hypothesis, based on studies conducted in seven different countries, received widespread publicity in the United States between 1953 and 1957 (Enig, 2010). Because of the extensive endorsement surrounding Key's assumptions, the edible oil industry promoted the polyunsaturated fatty acids (PUFAs) in the oil as a panacea in the US, which led to an increase in consumption of vegetable oil rich in PUFAs by 400% (Ribeiro, 2017). As a result, vegetable oils worldwide have been marketed with low SFAs despite the lack of scientific evidence to support the claim. A case study conducted in Indonesia concluded that the consumption of total fat or saturated fat, including that from coconut was not a predictor for CHD in this food culture, rather the intakes of animal foods, total protein, dietary cholesterol and less plant derived carbohydrates were predictors of CHD (Lipoeto et al., 2004). Moreover, cardiovascular disease (CVD) incidence does not rise due to the consumption of saturated fat, which was strongly supported by a meta-analysis of over 8,00,000 patients (Siri-Tarino et al., 2010). According to a study, involving over 5000 patients (Souza et al., 2015), consumption of SFAs was not associated with CVD. However, coconut oil is characterized by 63% MCFAs, (lauric acid being the most predominant one). MCFAs are differentially metabolized in the human body, because, upon consumption, MCFAs in the blood stream are metabolized for energy production and/or deposited as body fat (Guillot et al., 1993). Conversely, other vegetable oils, which are predominant in long chain fatty acids (LCFAs), necessitate the action of lipoproteins for their transport and are consequently deposited into various organs of the body, including heart vessels (Fig. 1). Coconut oil has a high proportion of SFAs compared to PUFAs, which makes it resistant to oxidative rancidity and thus serves as excellent cooking oil (Gunstone, 2011; Srivastava et al., 2010). The oxidative stability of coconut oil results in fewer free radicals upon storage. Contrarily, PUFA enriched fats generate enough free radicals to ultimately damage the living cells. The high degree of saturation of coconut oil (>90%), precludes the requirement of a hydrogenation process to maintain its stability

Table 2
Clinical studies on cardioprotective effects of coconut products.

S. No.	Coconut derivatives	Key ingredients	Dosages	*In vitro* tests, experimental animals and human data	Characteristics of volunteers in human trials	Adverse reactions, symptoms, and side effects, if any	Inference(s)	Reference
1.	Coconut oil (as cooking oil)	SFAs present in coconut oil	For everyday cooking, the individuals were instructed to use the allocated oils—coconut versus sunflower oils	**Human study:** Body fat, BMI, waist-hip ratio, vasodilatation, and biochemical parameters with a 24-h dietary recall after consuming coconut oil vs. sunflower oil for cooking. A 7-day recall and a diet diary were used to monitor the adherence of study subjects to the assigned oil and food patterns.	200 Medical College patients who used coconut oil and sunflower oil for cooking underwent evaluations every three, six, one, and two years.	Nothing reported	Using coconut oil as cooking oil for two years did not alter the lipid-related cardiovascular risk factors and events in patients under normal medical treatment, despite the higher saturated fatty acid content of oil	Vijayakumar et al. (2016)
2.	Coconut oil	Healthy fats present in coconut oil	The recruited respondents had been using either coconut or sunflower oil as their primary cooking medium for more than six years. The oils provide them with between 13 and 20 percent of their total calories.	**Human study:** Serum lipid profiles (HDL, LDL, and TAG), and GSH (total glutathione), glutathione peroxidase (GPx), and superoxide dismutase (SOD).	140 male subjects (35–65 years old): Coconut oil consumers (35 with a mean age of 44.9 $\pm$ 7), Sunflower oil consumers (35 with a mean age of 45.2 $\pm$ 8.6) Coconut oil consumers with type 2 diabetes (35 with a mean age of 55.1 $\pm$ 8.6), Sunflower oil consumers with type 2 diabetes (35 with a mean age of 53.6 $\pm$ 7.4)	Nothing reported	In comparison to the coconut oil groups, the sunflower oil groups had lower HDL cholesterol concentrations suggesting that habitual consumption of coconut and coconut oil, along with a normal diet, do not specifically contribute to the development of CHD in the people of Kerala.	Sabitha et al. (2009)
3.	VCO against Metabolic syndrome (MetS)	MCFA especially lauric acid	30 mLof VCO in place of the same amount of conventional dietary oil	**Human study:** 30 mL of VCO per day as an alternative to the same amount of oil in the habitual diet to prevent excess energy intake for four weeks. Serum triglyceride levels, fasting blood sugar, blood pressure, and other relevant observations.	For a 4-week intervention, 44 subjects—22 males and 22 female controls—aged 20 to 50 years—with waist circumferences of at least 102 cm (40 inches) for men and at least 88 cm (35 inches) for women, fasting blood sugar (FBS) of at least 100 mg/dl, HDL-C of at least 40 mg/dl, and at least 50 for women, fasting triglyceride (TG) level of at least 150 mg/dl, and blood pressure (BP $>$ 130 and $<$ 85 mmHg—were included in the study.	Levels of TC, LDL-C, and ADMA increased slightly following consumption of VCO. Moreover, there was no improvement in anthropometric traits, blood pressure measurements, or the LDL-C/HDL-C ratio.	By raising HDLC levels and lowering TG and FBS, VCO exhibited positive impacts on MetSWaist circumference and blood pressure remain unchanged.	Nikooei et al. (2021)
4.	Coconut sugar	• Low Glycaemic index • High anti oxidant activity and nutraceutical properties.	1.5g per day for 8 weeks	**Human study:** A placebo-controlled, randomised study involving 19 middle-aged and older adults (mean age 55.3 $\pm$ 2.1 years). Measurements were made before and after an 8-week	19 middle-aged and older adults (MA/O, $\geq$45 yr) Pre- and post-eight-week observations with either 1.5 g/day of coconut sugar or 1.5 g/day of a placebo	Nothing reported	Eight weeks of coconut sugar reduces mechanical stiffness in the CCA and brachial SBP, suggesting a possible cardioprotective benefit in MA/O adults.	Carlini et al. (2023)

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Table 2 (*continued*)

S. No.	Coconut derivatives	Key ingredients	Dosages	*In vitro* tests, experimental animals and human data	Characteristics of volunteers in human trials	Adverse reactions, symptoms, and side effects, if any	Inference(s)	Reference
				period of either 1.5 g/day of coconut sugar or 1.5 g/day of a placebo, including brachial and carotid blood pressure and arterial stiffness.				
5.	VCO	VCO as fat source in the diet may increase lipolysis and decrease lipogenesis in the liver as well as other tissues	30 mL	**Human study:** Observations on body weight, hip and waist circumference, Fat percent for total body, android and gynoid, fat mass, and lean mass.	For 28 days, with a 28-day washout, twelve postmenopausal women (58.8–3.7 years) ingested 30 mL of VCO or sunflower oil.	Due to an increase in cytokines, one participant experienced negative reactions to VCO and elevated inflammation.	When using VCO, TG and the TG/HDL ratio decreased to the ideal range of 2.0 or below, however when using sunflower oil, the parmeters show an increase. VCO is neutral and, when used regularly, may even be helpful for some people. Individual monitoring is necessary since sensitivity to VCO can occur	Harris et al. (2017)
6.	VCO 1.1.1	Antioxidant and membrane stabilizing properties of VCO polyphenols (potent free-radical scavenger)	Polyphenols extracted from VCO administered at 10, 20 and 50 mg/kg body weight	**Animal study:** Serum levels of cholesterol, lipoproteins, and lipids were measured, as well as cardiovascular risk ratios. Superoxide dismutase (SOD) and catalase (CAT) hepatic activity were examined, along with reduced glutathione (GSH), malondialdehyde (MDA), and glutathione concentrations as well as cardiovascular markers.	Two weeks before the concurrent administration of Cd (5 mg/kg) for five weeks, 48 rats weighing between 100 and 200 g were pretreated orally with polyphenols from VCO (10, 20, and 50 mg/kg body weight).	Nothing reported	In addition to stabilizing antioxidant defence systems equivalent to the control group, the co-administration of VCO polyphenol and Cd significantly improved the lipid profile and cardiovascular risk ratios (atherogenic index (AI), coronary risk index (CRI), and cardiovascular risk index (CVRI).	Famurewa and Ejezie (2018)
7.	VCO	• High polyphenol content of VCO and antioxidant activity (80 mg/100 gm oil) compared to CO (64.4 mg/100 gm oil) • Presence of minor constituents including Vitamin E 1.1.2	100–130 g male rats were fed with oil (8%) along with a semi synthetic diet for 45 days (ground nut, coconut oil, VCO and control)	**Animal study:** Body weight gain, tissue analysis, and cholesterol analysis from blood were carried out.	8% consumption combined with a diet that is artificially high in starch, protein, salt, and vitamins	Nothing reported	The concentration of cholesterol in the serum, liver, and heart of the VCO-treated group was significantly lower in VCO-treated animals suggesting the potential of VCO in protecting and lowering lipid levels in serum and tissues and LDL oxidation by physiological oxidants.	Nevin and Rajamohan (2004)
8.	VCO and coconut oil	Beneficial effect of VCO over CO is attributed to the presence of unsaponifiable components, namely polyphenols and vitamin E since there was no major difference in the fatty acid profiles	Male rats (100–120 g) were fed with 8% oils (VCO, coconut oil, olive oil and sunflower oil) along with a semi synthetic diet for 45 days	**Animal study:** After 45 days, animals were fasted overnight and killed. Gain in body weight, lipid analysis from blood and tissue analysis were per formed	Number of animals in each groups (Total 4 groups) were not mentioned. 8% intake along with a synthetic diet containing starch, protein, salt and vitamin mixture	Nothing reported	The amount of lipids in the serum and liver was reduced due to the use of VCO. Dietary VCO lowers the risk of CHD by modifying the synthesis and breakdown of fatty acids in a positive way.	Arunima and Rajamohan (2014)

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Table 2 (*continued*)

S. No.	Coconut derivatives	Key ingredients	Dosages	*In vitro* tests, experimental animals and human data	Characteristics of volunteers in human trials	Adverse reactions, symptoms, and side effects, if any	Inference(s)	Reference
		among VCO and coconut oil. 1.1.3						
9.	VCO	SFA s and Phenolics	35 g/day.	**Human study:** Twenty fit, non-obese male volunteers, weighing 67.5 kg and aged 28 to 50, were enrolled consecutively in a two arm controlled feeding trial for eight weeks, followed by a six-week washout period. The diets in the first arm were prepared using VCO, whereas the second arm used 35 g/day of peanut oil as a control. 1.1.4	Evaluation of effect of VCO on HDL-C and a few anthropometric and biochemical markers linked to cardiovascular health in humans both before and after the feeding trial.	Not in the list. Nonetheless, there was a minor rise in LDL-C and total cholesterol in otherwise healthy, non-obese participants by the end of the eighth week, with no change in body composition or other anthropometric measurements.	When combined with a healthy diet, VCO had no discernible effects on the majority of cardiovascular risk factors. It is necessary to test this effect across longer time periods with larger sample sizes	Jeyakumar et al. (2023 b)

(Vasudevan, 2017). Hence, consumption of MCT-rich coconut oil is suggested to control the rise in body weight and reduce fat deposition, thereby potentially negating the risk of developing CHD. In addition, the non-hydrogenated, natural coconut oil tends to increase high-density lipoprotein (HDL) and therefore protects the heart. Lipid peroxidation products namely malondialdehyde (MDA), conjugated dienes and hydroperoxides are markers to assess the rate of lipid oxidation. Coconut oil consumption results in lower levels of lipid peroxidation products (MDA and conjugated dienes) and higher levels of antioxidants (beta--carotene, Vitamin A and Vitamin C). However, dietary consumption of repeatedly heated coconut oil (6 times repeated heating at 300 °C followed by cooling) can cause a genotoxic and preneoplastic change in the liver as stated by Srivastava et al. (2010). On the other hand, a very low incidence of CVD has been noticed in populations across the world that predominantly consumes coconut oil. Also, coconut oil is found to stimulate the HDL/total cholesterol ratio (Lindeberg et al., 1994; Mendis, 2017). Although the Republic of the Philippines is known to have the highest coconut intake index, the Bicol region of the country, being the largest consumer of coconut in the Philippines, showed the lowest incidence of heart disease in a survey (Dayrit, 2003). Across the globe, similar research evidences have revealed a negative correlation between regular coconut oil consumption and CVD (Babu et al., 2014; Cardoso et al., 2015; Fernando, 2011; Korrapati et al., 2019; Ma & Lee, 2016; Sekhar, Ravinarayan, et al., 2022; Vijayakumar et al., 2016).

Several clinical studies have been conducted to investigate the effect of coconut oil intake on the incidence of CHD. A case study conducted in Kerala, India, analyzed the lipid profiles of 76 CAD patients who consumed coconut oil and sunflower oil over a 2-year period (Vijayakumar et al., 2016), which revealed that though coconut oil is rich in SFAs in comparison to sunflower oil, when used as cooking oil, it did not change the lipid-related cardiovascular risk factors and events in those receiving standard medical care. Similarly, no significant differences were observed in the fatty acid composition of plasma, due to the dietary fat source (either coconut oil or sunflower oil). Also, coconut oil consumption did not elicit changes in the ratio of HDL cholesterol to LDL cholesterol (HDLc/LDLc) in the subjects (Sabitha et al., 2014). Likewise, previous research on the dietary preference for coconut oil and/or its derivatives in conjunction with normal diets found no link between the diet and the development of CHD (Sabitha et al., 2009). Consumption of coconut kernel (55.8 g/head/day) along with coconut oil (38 g/head/-day) resulted in lower total cholesterol and higher HDL cholesterol, lowered the LDLc/HDLc ratio and decreased the triglyceride levels. In addition, the dietary fibre and protein in the kernel add to the health benefits. These observations clearly indicate that coconut and coconut oil consumption as part of a normal diet has no deleterious effect with respect to serum lipids (Rajmohan, 2017). Increased lipid peroxidation is associated with reduced antioxidant status of erythrocytes, make them more susceptible to hemolysis as evident from the study conducted with coconut oil, mustard oil, and sunflower oil that underwent repeated heating. Heating unsaturated fatty acid rich oils causes greater degree of lipid peroxidation than heating oils rich in SFAs such as coconut oil. Compared with heated mustard oil and heated sunflower oil, heated coconut oil in the diet resulted in lower lipid peroxidation in erythrocyte membrane and hemolysis (Chacko & Rajmohan, 2018). A clinical study conducted in New Zealand white rabbit models and with 100 patients clearly indicated that consumption of coconut oil by a cardiac patient during medication is safe (Vijayakumar & Sandhya, 2017). In general, atherogenicity increases due to the oxidation of LDL in the walls of arteries (Loidl et al., 2004). Antioxidants such as beta-carotene (provitamin A) and vitamin C may play a role in the prevention of this type of oxidative damage (Osganian et al., 2003). Coconut oil is less susceptible to heat-induced oxidative decomposition owing to its fatty acids, which are composed mostly of SFAs. Dietary oils are heated to an elevated temperature while cooking and frying food. This in turn leads to oxidative changes (release of hydroperoxide, aldehydes, ketones, etc.) in oil depending on the duration of heating, degree of unsaturation of oil, and extent of aeration (Choe & Min, 2007). The dietary ingestion of thermally oxidized PUFA-rich cooking oils promotes the induction, development, and progression of CVDs (Bordin et al., 2013; Choe & Min, 2007). Heating of coconut oil at 180 °C for a period of 30 min resulted in hardly 1 mmol per litre of toxic aldehyde, whereas the corresponding values for butter (1.5), extra virgin olive oil (3), corn oil (5), and sunflower oil (5) suggest that, among the various edible oils, coconut oil could be considered ideal for cooking, roasting, and frying (Ribeiro, 2017). Dietary intake of culinary oils following the repeated heating caused significant alterations in the platelet functions of cholesterol-fed rats. In fact, oxidative deterioration was pronounced in heated oils (210 °C for 15 h at 3 h per day for 5 days) compared to the unheated oils, but the ill effects were low in heated coconut oil in comparison with heated mustard or sunflower oil (Chinu & Thankappan, 2011). The effects of repeatedly heating coconut oil on cardiovascular disease are depicted in Fig. 2.

VCO, which is derived from coconut oil, and has superior biochemical and functional properties, also showed similar health benefits. VCO

Table 3
Summary on the possible mechanisms for the cardio protective effect of coconut derivatives.

Coconut derivatives/Key substance	Mechanism of cardioprotective effect	References
Coconut oil &VCO: Medium chain fatty acids in coconut	• MCFAs are readily absorbed through the small intestine. • MCFAs are not stored in the tissue as fat because they are transferred to the liver blood flow to be metabolized without carnitine to produce energy rapidly and efficiently. • The cardioprotective effect is attributed to the high content of lauric acid and myristic acid in the fatty acid fraction. • In humans, coconut fat lowers triglyceride levels (kernel at 55.8g/head/day and oil at 38g/head/day), lowers total cholesterol, raises HDL cholesterol, and lowers the LDL/HDL cholesterol ratio.	Guillot et al. (1993) Vijayakumar and Sandhya (2017) Chinwong et al. (2017) Rajmohan (2017).
Coconut oil &VCO: Saturated fatty acids	• Highly resistant to rancidity and oxidative peroxidation. • Regulates weight gain and minimize fat accumulation with MCT-rich coconut oil, eliminating the chances of CHD.	Srivastava et al. (2010) Gunstone (2011). Choe and Min (2007) Bordin et al. (2013) Ribeiro (2017)
VCO: Blood-pressure lowering effect	• VCO (84 mg/100 g oil) and CO (64.4 mg/100 g oil) have higher total phenolic content, which lowers blood pressure. • Higher concentrations of vitamin E and phenolic components (catechin, ferulic acid, P-coumaric acid, and caffeine) have a heart-protective impact.	Shariq et al. (2015)
VCO: Lowering lipid deposition	• B-carotene in VCO lowers the content of lipids and enhances the production of bile acids in the faeces.	Seo et al. (2004) Shariq et al. (2015)
VCO:	• By increasing the excretion of bile acids and neutral sterols in the faeces and competitively blocking the absorption of cholesterol, phytosterols can improve circulating lipid profiles and lower the risk of coronary heart disease. A 10–15% VCO supplementation in diet has positive effect on heart.	Famurewa and Ejezie (2018) Nevin et al., (2004); Arunima and Rajamohan (2013) Arunima and Rajamohan (2014) Subermaniam et al. (2014) Kamisah et al. (2015).
Coconut derivatives	• Elevated quantities of antioxidants, such as beta-carotene, vitamin A, and vitamin C, increase the ratio of HDL to total cholesterol.	Osganian et al. (2003), Mendis (2017)
Coconut kernel: Kernel protein	• Myocardial antioxidants • Cardioprotective effects of coconut kernel protein (50 mg/100 g) may be partially attributed to antioxidant processes, elevation of nitric oxide, and the protein's capacity to block TNF-a and NF-kB activation. • The main protein fraction, globulins, not only has anti-diabetic and *anti*-peroxidative properties, but also a cardioprotective impact. • Albino rats that consumed (8%) coconut protein showed lower incidence of myocardial infarction attributed to high content of L-arginine in protien.	Remya et al. (2013) Mini and Rajmohan (2002)
Repeated heating of oils		
Coconut oil and VCO	• Modifications in the functioning of platelets. • Oxidative degradation occurs in contrast to the oils that are not heated. • When heated coconut oil was added to the diet, lipid peroxidation in the erythrocyte membrane and hemolysis were low in comparison to heated mustard and sunflower oils.	Chinu and Thankappan (2011) Chacko and Rajmohan (2018).

has many heart protective functions. Intake of VCO could reduce total cholesterol (TC), total triglyceride (TG), low density lipoprotein (LDL), and increase high density lipoprotein (HDL) levels in a high carbohydrate/high lipid diet (HCD/HLD) group of rats (Shariq et al., 2015). Furthermore, Nikooei et al. (2021) showed evidence regarding the beneficial effect of VCO (30 mL) on cardiometabolic risk factors (metabolic syndrome (MetS) in adults. The polyphenol fraction of VCO has been shown to prevent LDL-oxidation (Nevin & Rajamohan, 2004; Shariq et al., 2015). Daily consumption of 30 mL of VCO for 8 weeks among 35 healthy volunteers significantly increased the levels of HDL cholesterol (Harris et al., 2017), corroborating the findings of Cardoso et al. (2015). A 4-week VCO supplementation regimen significantly improved the lipid profile without causing anthropometric changes (Liau et al., 2011). The experiment was further extended to investigate any potential side effects of consuming 30 mL of VCO daily. However, consumption of VCO along with dietary supplements, elicited no harmful side effects (Chinwong et al., 2017), owing to the high concentration of lauric and myristic acids in VCO. Famurewa and Ejezie (2018) isolated various polyphenols from VCO and showed that the polyphenols in VCO could be a great source for the development of CVD-preventing supplements in rats. The effect of 10% and 15% VCO in healthy rats for 5 weeks after analyzing the total cholesterol (TC), LDL, and HDL levels revealed that the performance of the rat group fed with 15% VCO was significantly good compared with the group that was fed with 10% VCO. Similar research findings correlating the VCO polyphenol content and CVD risk reduction are made available (Nevin & Rajamohan, 2004; Arunima & Rajamohan, 2014; Subermaniam et al., 2014; Kamisah et al., 2015). In majority of these studies, 0.5 mL–1 mL VCO or 10%–15% of the diet supplement has been used in rodent models. In addition, coconut kernel protein in diet also offers cardioprotective effects (Remya et al., 2013). Furthermore, cardioprotective effect of coconut sugar has been unravelled (Carlini et al., 2023). A recent compilation on the coconut oil and cardiovascular disease risk suggest no or less evidence for risk due to coconut oil (Schwingshackl & Sabrina Schlesinger, 2023). All of these investigations, including the most recent article on VCO, recommended testing in larger sample sizes over longer time periods as a future line of inquiry (Jeyakumar et al., 2023). Hence, it is safe to conclude that consumption of coconut oil per se did not elicit any ill effects in the lipid profiles of the human subjects. Biochemical studies, on the other hand, have shown that MCFAs and the polyphenol fraction of VCO or coconut oil have the potential to promote heart health and maintain a healthy lipid profile. A detailed insight into clinical studies evaluating the cardio protective effect of coconut derivatives is presented in Table 2. In addition, a summary on the cardio protective effect and its mechanisms is furnished in Table 3.

3. Neuroprotective effects of coconut

Cognitive deficits induced due to neurodegeneration are important characteristic features of Alzheimer's disease (AD). Pathological and biochemical observations have revealed that AD is characterized by the accumulation of A (amyloid) peptides in cerebral tissues (Chetelat et al., 2010) and the formation of neurofibrillary tangles in neurons due to phosphorylated tau proteins (Iqbal et al., 2010). These pathological features occur due to abnormal turnover activity of amyloid precursor

Table 4
Clinical studies on neuroprotective functions of coconut products.

S. No.	Coconut derivatives	Key ingredients	Dosages	*In vitro* tests experimental animals and human data	Characteristics, of volunteers in human trials	Adverse reactions, symptoms, and side effects, if any	Inference(s)	Reference
1.	AC-1202 (composed of glycerin and caprylic acid), a form of MCTs, derived using glycerin from vegetable oil and fatty acids from coconut	AC-1202 would elevate serum ketone bodies even in the presence of carbohydrate in the diet. Thus, a mild state of ketosis can be induced.	During the first seven days of the trial, 30 g sachets containing 10 g of AC-1202 were distributed. On Day 8, the dose was increased to two 30 g sachets daily (equivalent to 20 grams AC-1202), which was continued till Day 90. Doses were given every day at breakfast. A washout visit took place on Day 104.	**Human study:** Measurement of cognitive abilities	There were 152 outpatients with a diagnosis of mild to moderate Alzheimer-type dementia (diet remained on approved AD medicines).	GI side effects included diarrhoea in 24.4% of AC-1202 patients and 13.6% of placebo subjects.	Quantities of circulating ketone bodies increased, which may provide neuroprotection. Ketogenic diets also cause several other alterations (raised activity of uncoupling proteins).	Henderson et al. (2009).
2.	**Coconut shreds (**The presence of curcuminoids and MCT in coconut)	Low acetylcholinesterase (AChE) enzyme activity due to co-extract of turmeric powder and coconut shreds	Intranasal administration of supercritical extracts of turmeric and coconut at a dosage of 40 mg/kg	**Animal study:** Animals underwent behavioural assessment. Assessments of the brain's hippocampus region from a biochemical, molecular, and histological perspective. Rat epidermal keratinocyte (REK) and rat glioblastoma (C6) cell lines were used to test the *in vitro* cytotoxicity.	Adult male albino Wistar rats weighing 180–220 g, aged 4–6 weeks. The rats were fed purified water on an as-needed basis along with a conventional commercial laboratory balanced meal consisting of 22.75% protein, 4.63% fats, and 5.35% fibre. Five groups of 10 adult male Wistar rats each, one with coconut and turmeric, were randomly assigned out of the fifty rats.	Nothing reported	When taken intravenously, the SFE co-extract of coconut shreds and turmeric powder enhanced cognitive function, corrected histological changes in the brain, and decreased hippocampal inflammation as measured by proinflammatory cytokine markers.	Sharma et al. (2024)
3.	**Coconut oil**	SFAs	40 mLcoconut oil per day. Calorie intake: 55% carbohydrate, 15% protein and 30% by coconut oil	**Human study**: Analyzed temporal orientation, visuospatial and visuoconstructive abilities, semantic and episodic memory.	44 AD patients (75% female and 25% male) were split into two groups, with 75% female and 25% male in each group. One group consumed a Mediterranean diet supplemented with coconut oil for a period of 21 days. Patients' degrees of cognitive impairment were rated as mild, moderate, or severe.	Nothing reported	The Mediterranean diet enhanced with isocaloric coconut oil appears to enhance cognitive abilities; this was particularly noticeable in women with mild-to-moderate Alzheimer's disease.	De la Rubia Orti et al. (2018)
4.	**VCO**	As a ketogenic agent and antioxidative characteristics	30 mL/day for 24 weeks	**Human study**: Cognition was measured at baseline and after the intervention using the	120 over 65 of them have mild-to-moderate AD	Not mentioned. However, after a 24-week intervention,	VCO improved the MMSE scores in APOE ε4 carriers. Besides, VCO did not compromise lipid	Fernando et al. (2023)

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Table 4 (*continued*)

S. No.	Coconut derivatives	Key ingredients	Dosages	*In vitro* tests experimental animals and human data	Characteristics, of volunteers in human trials	Adverse reactions, symptoms, and side effects, if any	Inference(s)	Reference
				Mini-Mental State Examination (MMSE) and the Clock Drawing Test. Blood samples were drawn, their lipid profiles examined, and their HbA1 C levels measured.		VCO did not enhance cognition in people with mild-to-moderate AD.	profile and HbA1 C levels and is thus safe to consume.	
5.	**VCO**	MCFAs (61%), phenolic compound (49.8 mg eq./kg) and its antioxidant activity (53.7 μg/mL).	The VCO emulsion (20%) in Pasteurized milk. One group received VCO (1.42 mL/kg b.wt.) daily for 60 days, whereas the other group took a daily dosage of dairy formula enhanced with the same quantity of VCO.	**Animal study:** Chemistry, biochemistry, behaviour, histopathology, and immunohistochemistry.	42 Wistar rats weighing between 150 and 200 g were divided into seven groups and given one of three treatments: $AlCl_3$, the usual medication (rivastigmine), the enriched formula, or a combination of these with $AlCl_3$.	The consumption of dairy formula supplemented with VCO can prevent the pain or complaints (feeling unwell) that are linked to swallowing or "drinking" the liquid VCO directly.	The cognitive damage was lessened following administration of a dairy formula supplemented with VCO. The dairy formula enriched with VCO has nearly identical biological effects to that of the unformulated VCO	Khalil et al. (2020)
6.	**VCO**	Antioxidants	• VCO at 62.5 μg/mL for 24 h. • Al $(OH)_3$ (362 μM) + VCO (62.5 μg/mL). • AD-VCO group: RA (10 μM) + BDNF (2.5 ng/mL) + VCO (62.5 μg/mL). • AD-Al-VCO group: Al $(OH)_3$ (362 μM) + RA (10 μM) + BDNF (2.5 ng/mL) + VCO (62.5 μg/mL).	Specific markers of AD, oxidative stress parameters and neurotransmitter-related parameters were measured	*In vitro* study	–	In the study groups, VCO decreased the levels of hyperphosphorylated Tau protein and amyloid beta. Moreover, VCO reduced oxidative stress and enhanced neurotransmitter parameters.	Demirel et al., (2023)
7.	**VCO (Fermented method)**	Anti-inflammatory, antioxidant properties, and modulation of the levels of acetylcholinesterase.	VCO at 100 and 200 mg/kg body weight	For 21 days, 70 male rats weighing between 180 and 300 g were given subcutaneous injections of ethanol at a dose of 2 g/kg every day. Next, behavioural and metabolic tests were performed on the treated rats. Further, exposed to coconut treatments once more.	70 Male Rats were tested for 21 days. 1.1.5	Not reported	Coconut oil fractions and *Punica granatum* hydroalcoholic extract reduced the effects of ethanol on synaptic loss, neuroinflammation, oxidative stress, and neuronal death. Long-term coconut oil consumption offers neuroprotection.	Gavitre et al. (2022)
8.	**Coconut water**	Phytochemicals	CW at 0.02, 0.05, 0.1, and 0.2 mL/20 g body weight	**Animal study**: Different concentrations of coconut water were administered to Swiss mice, and the impact of CW on cognition was evaluated. Nitric oxide (NO), glutathione (GSH), and malondialdehyde (MDA) levels were estimated.	The experiment was run at intervals of 7, 14, and 28 days. Groups 3–6 were given CW at 0.02, 0.05, 0.1, and 0.2 mL/20 g body weight, respectively, whereas Group 1 got distilled water, Group 2 received	A significant increase in GSH (glutathione) levels was observed in CW treated rats.	The latency time, malondialdehyde, and nitric oxide levels were significantly lower in the groups treated with CW for seven days. The study demonstrated how coconut water might lessen oxidative stress and enhance memory in lab mice.	Onasanwo et al., (2020)

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Table 4 (*continued*)

S. No.	Coconut derivatives	Key ingredients	Dosages	*In vitro* tests experimental animals and human data	Characteristics, of volunteers in human trials	Adverse reactions, symptoms, and side effects, if any	Inference(s)	Reference
					lipopolysaccharide (LPS) at 250 μg/kg body weight.			
9.	**Young coconut juice**	Therapeutic effect of young coconut juice: • Presence of amino-acid L-arginine. • Beta-sitosterol • increasing the activity of a lipoprotein lipase in the heart and adipose tissue	Three low doses of YCJ (10, 20 and 40 mL/kg body weight for ten weeks	The brains, liver, kidney, adrenal glands, prostate glands, and seminal vesicles of the 10 week-old rats were removed, weighed, fixed, and paraffin embedded for sectioning and staining. The rats were then euthanized. Serum analysis was carried out.	8 months old male Wistar rats (250–300 g).	After five weeks of a large dose (100 mL) of YCJ feeding, the female rats began to exhibit undesirable side effects, such as the accumulation of glycogen in the liver.	Consuming YCJ (at a maximum of 10 mL per kilogramme of body weight daily) was found to be safe for the lipid, hepatic, and renal profiles and may be a supplement for maintaining neuronal cell density.	Balit et al. (,2018)

protein (APP) (Kanekiyo et al., 2013) or could be attributed to protein mis-folding. Furthermore, age, diet, genetic, lifestyle, and environment factors all contribute to this disease, and diet is thought to be an important factor in reversing the disease (Bhardwaj et al., 2017). The pharmaceutical industry recommends the inhibitors of acetyl cholinesterase as potential drugs to treat AD (Hansen et al., 2008). In this context, coconut oil and its products have gained immense interest in alleviating the symptoms of AD (Chatterjee et al., 2020; Hu, O De, et al., 2015).

An early stage biochemical marker for AD is impaired glucose metabolism of cerebral tissues, which means that glucose, the brain's main fuel, cannot be used effectively. Nonetheless, in the absence of glucose, alternate sources of energy such as ketone bodies could supply the requisite energy to the brain cells during these conditions (Asih et al., 2014). Further, coconut oil and diets that are rich in MCFAs are potential ketone precursors and hence could serve as source of alternative energy to the neuron cells which could not utilize glucose as a source of energy during the progression of AD (Ota et al., 2019). As early as 2009, caprylic acid derived from coconut was used in the treatment of AD (Henderson et al., 2009). Also, Reger et al. (2004), showed the promise of MCT in improving the cognitive functioning of mild AD patients. Besides, the MCT-derived ketone bodies in conferring neuroprotective effects, coconut oil has been shown to have positive effects on AD patients due to various other modes of action such as reversal of age-related mitochondrial disorders (Balietti et al., 2010; Mirzaei et al., 2019; Studzinski et al., 2008), phosphorylation of GSK3 and ERK enzymes, thereby causing the activation of Akt serine/threonine kinase family and extracellular signal-regulated kinase (ERK) and inhibition of glycogen synthase kinase 3 (GSK3) enzymes which causes the promotion of cell survival (Nafar et al., 2017); inhibits the formation of NOD-like receptor family pyrin domain-3 (NLRP3) containing inflammasome, a critical feature of AD; (Mirzaei et al., 2019) impairs the expression of ADP-ribosylation factor-1 (ARF1) thereby inhibiting the secretion of Aβ (Bansal et al., 2019). In addition, lauric acid, a major constituent of coconut oil, potentially ameliorates the lipopolysaccharide-induced oxidative stress, decreasing NO production and affecting the production of pro-inflammatory cytokines in a G-protein-coupled receptor 40-dependent pathway (Nishimura et al., 2018).

Clinical studies have proven the efficacy of coconut oil and its derivatives in enhancing the cognitive ability of subjects (Hu et al., 2015). The MCT preparation containing a MCFA C8:0 (>95%) derived from coconut oil and palm kernel has demonstrated a significant effect in increasing the level of ketone bodies and thus providing neuroprotective effects (Henderson et al., 2009). Administration of VCO in experimental animals improved the anti-oxidant status of brain cells and increased acetylcholine leading to enhanced synapsis and neurotransmission. The influence of VCO is likely mediated through two cholinergic neurons and the microenvironment in the brain. For cholinergic neurons, VCO reduced AChE action, which in turn increased ACh concentration in the brain. The increased ACh plays an important role in effective synaptic transmission during the acquisition of new information and the consolidation of memory within the microenvironment. On the other hand, VCO increased production of antioxidants in the brain, namely superoxide dismutase (SOD), catalase (CAT), glutathione (GSH), and glutathione peroxidase (GPX). The up-regulated antioxidants served as protection against lipid peroxidation (MDA) by oxidative stress (NO) (Rahim et al., 2017). Ingestion of the Mediterranean diet combined with coconut oil (40 mL/day) by AD patients, in particular women, showed a significant improvement in their cognitive functions (De la Rubia Ortí et al., 2018). Along similar lines, oral intake of extra virgin coconut oil (EVCO) improved the cognitive functions of the female patients (Hu, O De, et al., 2015). Dietary interventions such as Keto-Medite approach rich in vitamins and coconut oil in combination with physical activity delayed the onset of AD (Perng et al., 2017). The prophylactic effect of VCO was proven in AD model rats since, VCO administration significantly reduced the levels of glutamate and increased the anti-oxidant status of brain tissues compared to the control animals (Alghamdi, 2018). In Alzheimer's rat models, VCO inhibits the progression of AD through multiple pathways such as reversing the damage to nerve cells, declining the production of tumour necrosis factor-α, significantly reducing the oxidative stress and hypo-phosphorylation of tau proteins (Mirzaei et al., 2019). Nonetheless, VCO did not improve cognition in individuals with mild-to-moderate AD following a 24-week intervention; however, it improved the MMSE scores in APOE-4 carriers. Besides, VCO did not compromise the lipid profile or HbA1C levels and is thus safe to consume, as evident from Fernando et al. (2023). In addition, a recent investigation showed that the supercritical fluid extract of co-extracts of turmeric powder and coconut shreds has greater efficiency in preventing neurodegeneration associated with AlCl3-induced Alzheimer's disease (Sharma et al., 2024). Apart from coconut oil, lower dose of tender coconut juice has been proven to have neuroprotective functions (Balit et al., 2018). A recent literature review on the effects of coconut oil on Alzheimer's disease with emphasis on preclinical studies (Rodrigues et al., 2023) revealed that coconut oil interferes important metabolic pathways that promote neuroinflammation, amyloid plaque formation, neurotransmitter imbalance, and signaling pathway dynamics. Furthermore, the use of coconut oil increased the levels of ketone bodies. To some extent, many clinical trials have already shown

Table 5
Summary on the possible mechanisms for the neuroprotective effect of coconut derivatives.

Active Ingredient and quantity	Neuroprotective effect(s)	References
Coconut oil and VCO: MCFAs	• MCFAs are potential ketone precursors (alternate sources of energy), provide energy to the neuron cells which could not utilize glucose as a source of energy during the progression of AD • Caprylic acid derived from coconut used in the treatment of AD	Asih et al. (2014), Ota et al. (2019)
Coconut oil and VCO: MCFAs	• Positive effects on AD patients due to reversal of age-related mitochondrial disorders phosphorylation of GSK3 and ERK enzymes	Studzinski et al. (2008) Balietti et al. (2010) Mirzaei et al. (2019),
Coconut oil and VCO: MCFAs	• Lauric acid, a major constituent of coconut oil, potentially ameliorates the lipopolysaccharide-induced oxidative stress, decreasing NO (Nitrous oxide) production and affecting the production of pro-inflammatory cytokines in a G-protein-coupled receptor 40-dependent pathway.	Nishimura et al. (2018).
	• MCFA (>95%) from palm kernel and coconut oil has been shown to have a major impact on raising ketone body levels and offering neuroprotective benefits.	Henderson et al. (2009).
VCO: vitamins and antioxidants • Oral administration of α-Tocopherol at 35 mg/kg body weight) in Wistar rats • 1, 5 and 10 g/kg VCO for 31 days by oral gavages in Wistar rats	• Memory-enhancing effect of VCO was comparable to that of Apha T • Apha T is a monophenolic lipid-soluble Vitamin E that protects cell membranes against lipid peroxidation • Memory-enhancing effect of VCO was found to be accompanied by increased cholinergic activity in rat brain. • VCO-enhanced memory was closely associated with changes of antioxidant status in brain. • Vitamin E, has a promising role for clinical manifestations due to oxidative stress. • The hydroxyl group of phenolic compounds may be able to reduce the toxicity of the Alzheimer's Ab peptide.	Bharrhan et al., (2010). Rahim et al. (2017)
Young coconut water	• Young coconut juice contains compounds that resemble oestrogen. • β-sitosterol, which makes up 58% of YCJ's overall composition, together with other sterols including fucosterol, stigmasterol, and fustatrienol, may be the cause of the plant's potent oestrogenic activity by promoting the production of endogenous oestrogens.	Balit et al. (2018) Radenahma et al., (2011)

favorable support for the use of coconut oil as an adjuvant in the treatment of AD. In addition to VCO, it was suggested that the tender nut water of coconut has a potential therapeutic value for AD (Radenahmad et al., 2011). Neuro protective studies on coconut has been conducted using the coconut shreds (Sharma et al., 2024), coconut water (Fernando et al., 2023), young coconut juice (Balit et al., 2018) etc. A few of the most important clinical research on the neuroprotective properties of coconut products are displayed in Table 4. Furthermore, Table 5 provides a summary of on the neuroprotective effect of coconut derivatives and the underlying mechanisms. A pictorial representation on the mode of action has been depicted in Fig. 3. The multi-pronged modulatory effects of coconut oil on mitochondrial irregularities, enhancement of cell survival pathways, amelioration of neurological inflammation and inhibition of Aβ are some of the neuroprotective functions of coconut oil and its derivatives (Ramesh et al., 2021). VCO has been tested the most in animal or human study models among the several coconut derivatives; this may be because it possesses antioxidant and phenolic properties in addition to SFAs. Clinical and epidemiological data reveal the neuroprotective effects of coconut oil when combined with a healthy diet or when adhering to the ketogenic diet. According to these clinical studies and growing body of scientific literature, the best suitable dosage for the coconut oil requires to be determined by further human trials.

4. Coconut oil and obesity

Obesity is an abnormal condition of excessive body fat accumulation, resulting in serious health issues. Co-morbidities associated with obesity include CHD (Pereira et al., 2020), atherosclerotic disease (Pereira et al., 2020), type 2 diabetes (Tinkoy et al., 2015), fatty liver (Fabbrini et al., 2010), hypertension (Klop et al., 2013), atherogenic dyslipidemia (Schaefer, 2002) among others. Dietary fat is a major contributor of obesity because it causes metabolic disorders such as dyslipidemia (Schaefer, 2002). In obese women and men, serum lipid levels are linked to bone marrow fat (Bredella et al., 2013). It is reported that intake of a high fat diet enriched with MCFAs increased the population of beneficial gut microbiota when compared with consumption of a high-fat diet with LCFAs (Machate et al., 2020). The gut microbiome witnessed an increase in production of short chain fatty acids (SCFAs), thus reducing obesity and preventing associated metabolic dysbiosis. MCTs can affect satiety as they are rapidly absorbed (Van Wymelbeke, et al., 2001) and have the potential to increase satiety (Jensen et al., 2020). Among the SFAs, lauric acid contributes least to fat accumulation (Dayrit, 2015). The comparative metabolic effects of MCTs (ingested as butter and coconut oil) and LCTs (as animal fats) in increasing the oxidation of LCFAs revealed that MCTs had the capacity to increase the endogenous oxidation of LCFAs, suggesting their vital role in controlling body weight over the long term (Papamandjaris et al., 2000). In addition to healthy fatty acids, inclusion of functional foods in the diet containing bioactive compounds such as antioxidant vitamins (A, C and E) and phenolic compounds exert positive effects in the lipid and glucose metabolism (Fig. 4). A recent evaluation of effect of coconut oil on lipid metabolism-related diseases and immune response using *in vitro* models revealed that coconut oil can reduce lipid accumulation in hepatocytes and adipocytes as well as is capable of modulating the immune response in gut cells (Machado et al., 2023). High-density lipoprotein, (HDL) absorbs cholesterol and carries it back to the liver. The liver then flushes it from the body. High levels of HDL cholesterol can lower the risk of heart disease and stroke. Diets enriched with VCO resulted in reducing the waist circumference of the subjects (Cardoso et al., 2015). The HDL concentrations increased, thereby aiding the secondary prevention for patients suffering from CAD. Intake of 30 mL of VCO per day, three times a day for four weeks, caused a significant reduction of 2.86 cm (mean) in waist circumference in men (Liau et al., 2000). Similar positive effects of coconut oil (tested for 8 weeks) have been observed when women with a BMI of 30–39.9 kg/m^2 were evaluated for anthropometric and biochemical characteristics (Oliveira-de-Lira et al., 2018). Intake of coconut oil in the diet resulted in enhanced weight loss and a reduced body mass index. The circumference of the waist, waist-to-height ratio, conicity index, and body fat percentage were reduced, thereby supporting anthropometric characteristics as well as reduced biochemical parameters namely glycemia and glycated hemoglobin, resulting in *anti*-obesogenic effects. In rat models, a comparison of the *anti*-obesogenic effects of VCO with orlistat (a commercial anti-obesity drug) and omega-3 fatty acids (both with proven *anti*-obesogenic effects) revealed that VCO demonstrated parallel or even superior *anti*-obesogenic effects to orlistat and omega-3 fatty acids (Adeyemi et al., 2020). The findings of the study suggested that the antioxidant effect of VCO is due to its terpenoid and anthraquinone constituents, which are known to have potent antioxidant property. Similar findings of lowered body weight, and improved structure and function of the heart and liver with VCO diet in Wistar rats were observed by Panchal et al. (2017). VCO has been shown to reduce lipid concentrations in serum and tissues

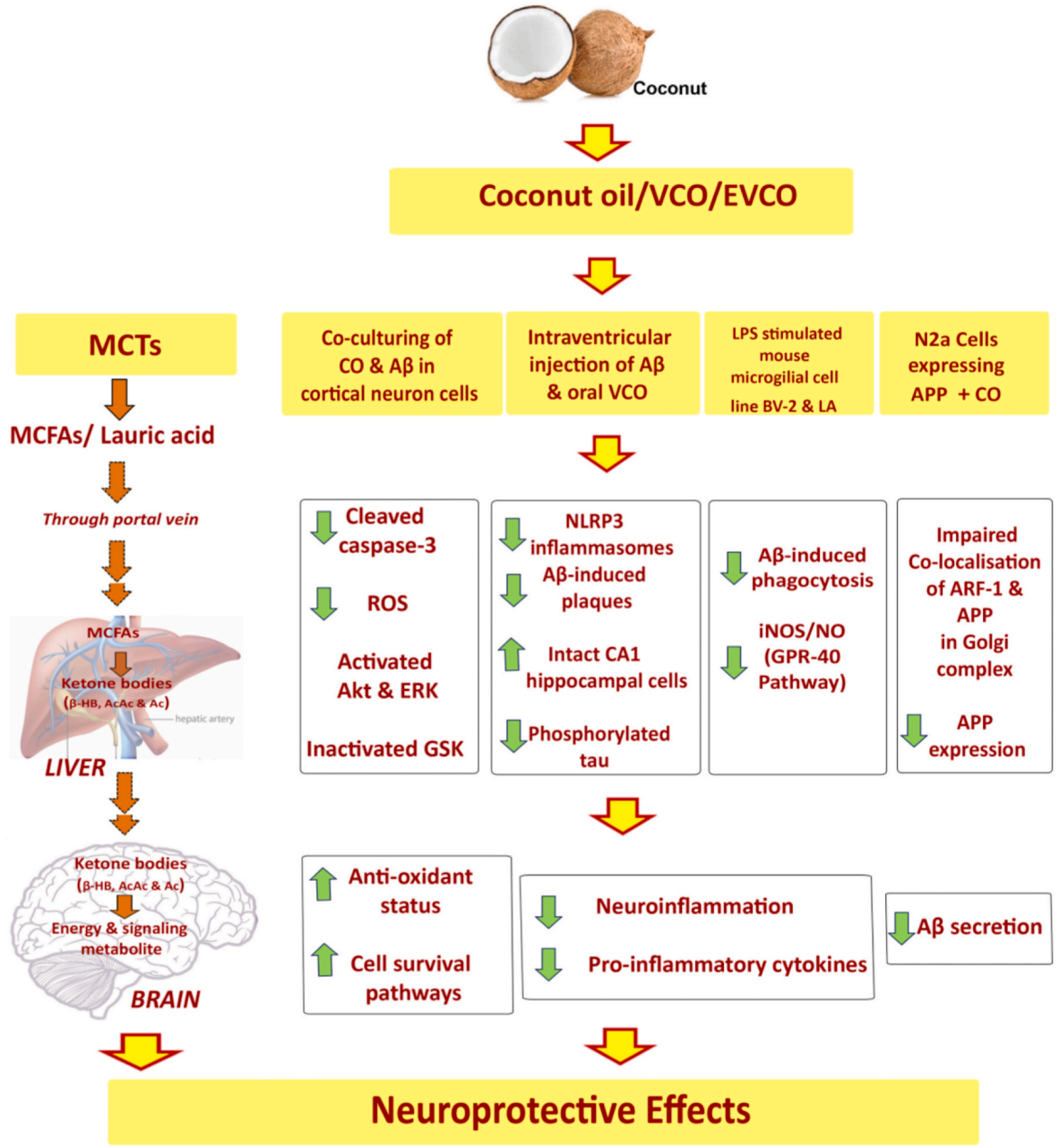

Fig. 3. Schematic representation of neuroprotective functions of coconut oil and its derivative (Source: Ramesh et al., 2021)

(Nevin & Rajamohan, 2004). Dyslipidemia is the imbalance of lipids such as cholesterol, low-density lipoprotein cholesterol, triglycerides, and high-density lipoprotein. This condition can result from diet, tobacco exposure, or genetics and can lead to cardiovascular disease with severe complications. The anti dyslipidemic effect of VCO is due to its high MCTs. These MCTs are known to improve lipid metabolism as well as fatty acid oxidation. This was supported by Assunçao et al. (2009) wherein, 40 women aged between 20 and 40 years were provided a diet with coconut oil. The results demonstrated that the diet did not cause dyslipidemia and rather enhanced abdominal obesity reduction. Furthermore, VCO exerts anti-obesity effect by modulating adiposity and improves hepatic lipid metabolism, leptin and insulin resistance in diet-induced obese rats (de Vasconcelos, 2022). Roopashree et al. (2021) have elaborately discussed the anti-obesity effect of coconut oil with many supporting research findings. The results of the incorporation of coconut oil into an iso-caloric balanced diet indicated positive effects on HDL and total cholesterol to HDL ratio in obese men, thus aiding in weight loss (Vogel et al. (2020). Thus, it appears that the high content of MCFAs in coconut oil could be underpinning the rapid metabolism of fatty acids and its *anti*-obesogenic effects, along with the secondary metabolites, including phenolic profile, which may act in synergism. Other than coconut fatty acids, coconut water is shown to have *anti*--hypolipidaemic, anti-inflammatory hypolipidaemic and liver-protective functions (Mohamad et al., 2017). Dietary consumption of coconut products including coconut milk, coconut oil, coconut water and VCO appears to impart beneficial health effects (Alatawi & Alshubaily, 2021). Table 6 shows some of the recent clinical studies on the anti-obesity effect of coconut products including coconut oil, VCO, coconut water, coconut vinegar, coconut milk and its derivatives. Also, a summary on the possible mechanisms for the anti-obesity effect is presented in Table 7.

To conclude, multiple research findings endorse the usage of coconut products to combat obesity and offer apparent health benefits. Nevertheless, elucidation of physiological and cellular biochemical processes

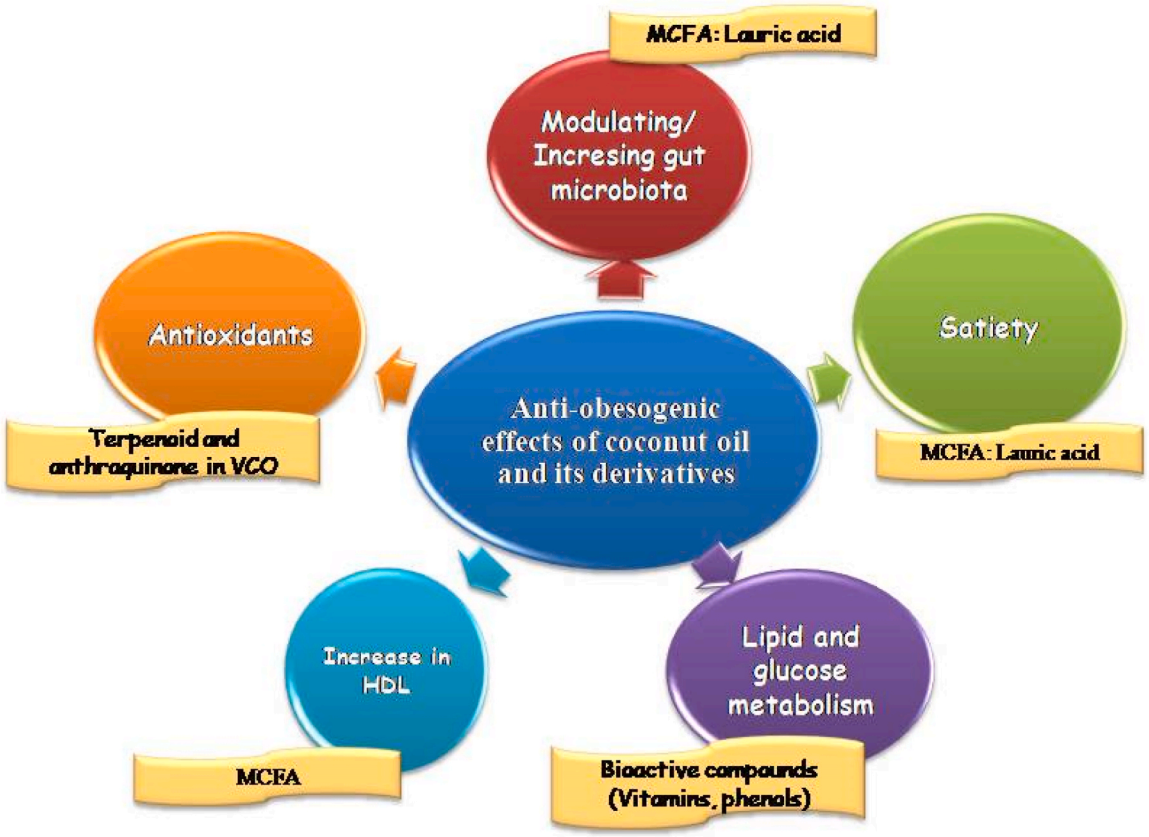

Fig. 4. Anti-obesity functions of coconut oil and its derivatives.

underlying anti-obesity effects of coconut and to exclude any confounding effects resulting from dietary diversity is warranted. Hence, large scale epidemiological investigations followed by clinical trials in this regard are indispensable.

5. Perspectives and conclusion

It is apparent that among the variants of coconut oil namely, VCO, coconut oil and refined, bleached and deodorized (RBD) coconut oil, VCO has been shown to possess antioxidant, anti-inflammatory, lipid-lowering and cytoprotective efficacies, owing to the presence of polyphenol content (Narayanankutty et al., 2018). The relatively high polyphenol content of VCO [11.82–28.19 mg GAE/100g of oil] bestows the oil with very high anti-oxidant properties. Polyphenol constituents of VCO have been shown to inhibit the lipid peroxidation, promote antithrombotic effects, and provide anti-inflammatory effects. These properties of polyphenol thus endow the oil with cardiovascular benefits. Besides, the fatty acid profile of coconut oil and its variants suggests MCFAs predominate (59.02%–62.27%). However, coconut oil also contains SFAs (28–31%) and UFA (6.7–8.3%). The varying proportion of different fatty acids and the differential mechanism of absorption of MCFAs in intestine thereby serving as a source of quick energy and not contributing to the cholesterol transport highlight the uniqueness of coconut oil in conferring cardioprotective effects (Babu et al., 2014). Anti-obesity effects of VCO and extra virgin coconut oil (E-VCO) in animal models reveal that differential metabolic fate of oil derived MCFAs, bypassing the activity of pancreatic lipase but direct transport to liver through hepatic portal vein, prevents accumulation of body fat and body weight gain. In addition MCFAs in E-VCO, has been shown to cause a negative regulation of lipoprotein lipase in adipose tissue and lower the expression of adipogenic transcription factors (TFs), thus aiding in insulin sensitivity and glucose tolerance (Vasconcelos et al., 2022). The phenolic acids in VCO namely rutin, 3,4-dihydroxybenzoic acid, salicylic acid, quercetin, p-coumaric acid, caffeic acid and myricetin are also involved in the regulation of insulin resistance and glycaemic index suggesting another mode of action of coconut derived biomolecules in providing health benefits (Illam, Narayanankutty, & Raghavamenon, 2017). Also, ingestion of E-VCO and VCO has been demonstrated to influence the hepatic lipid metabolism by mobilization of fat, cholesterol and TG from the liver to the blood. It caused greater excretion of faecal cholesterol leading to enhanced energy expenditure and greater mobility of body fat for the biosynthesis of bile acids in liver (Vasconcelos et al., 2022). The concerted action of polyphenols in providing anti-oxidant and anti-inflammatory properties coupled with cardiovascular benefits of coconut oil derived fatty acids could be underlying the numerous health benefits of consumption of oil.

Coconut and its derivatives have grown in popularity in the mainstream media and the digital world, and medical professionals have endorsed their health benefits and recognized coconut oil as highly valuable and healthy. The whole discussion on the health benefits or amelioration of ill effects of cardiovascular diseases, hyperglycaemic conditions, and neuroprotective properties of coconut oil revolves around the oil fatty acid composition alone, conveniently leaving aside other important phytonutrients such as phytosterols, whose health-promoting effects are well established. Hence, in this context, it is crucial to explore the role of coconut oil-derived phytosterols for their nutraceutical effects. Nonetheless, the clinical studies reviewed in this manuscript support the consumption of coconut oil, which is, in fact, a traditional practice too. The results of many of these studies suggest that individuals wishing to use coconut oil in their diets can do so safely, but more studies need to be conducted with larger sample sizes, diverse populations, and more specific clinical markers such as particle size. The small sample size of each group is another limitation of much clinical work. Longer periods of time with a larger sample to certify the

Table 6
Clinical studies on anti-obesity functions of coconut products.

S. No.	Coconut derivatives	Key ingredients	Dosages	*In vitro* tests, experimental animals and human data	Characteristics, of volunteers in human/Animal trials	Adverse reactions, symptoms, and side effects, if any	Inference(s)	Reference
1.	VCO	• Anthraquinone • Terpenoids • The antidyslipidaemic effect is due to its high content of medium chain triglycerides,	80 adult male Wistar rats (170–220 g) VCO at 200 mg/kg B.W, VCO + high fat diet, for 20 weeks.	**Animal study:** Blood samples, hepatic tissue were collected for analysis. • VCO, showed comparable efficacy in reducing TG, LDL-C, and athero-genic index following dietary change in the obese rats. • VCO demonstrated the most potent effect in reducing TC.	80 adult male Wistar rats treated for 20 weeks	Not reported	VCO demonstrated superior actions on malondialdehyde, glutathione peroxidase, and interleukin −6. Thus showed considerable *anti*-obesitogenic effects	Adeyemi, et al. (2020)
2.	**E-VCO**	• Anti-adiposity effect of MCFA • Fatty acids and the phenolic profile of VCO act in synergism	32 Wistar rats (154.56 ± 10.25 g) ±40 days of age.	Analyzed for oral glucose and insulin tolerance tests, Somatic parameters, euthanasia and biological material, fatty acid profile, hepatic oxidative parameters and Histological analysis.	Healthy group with VCO and obese group with VCO at 3000 mg/kg BW)	E-VCO could not improve the oxidative parameters of obese or healthy rats	Significant reduction in body weight gain induced by an obesogenic diet. E-VCO treatment in obese rats reduced deposition of fats, cholesterol, triglycerides and bile acids in the liver, reduced excretion of triglycerides and increased faecal cholesterol excretion.	Vasconcelos et al., 2023
3.	Coconut water vinegar	Gallic and vanillic acids in coconut vinegar: *anti*-hypolipidaemic Effect, anti-inflammatory hypolipidaemic and liver-protective effects	24 male mice of 7 week old were selected. Fed a standard pellet diet and distilled water ad libitum. Vinegar at 0.08 and 2 mL/kg body weight fed in week 24 to the end of week 33	**Animal study:** Changes in the body weight, fat-pad weight, serum lipid profile, expression of adipogenesis-related genes and adipokines in the fat pad, expression of inflammatory-related genes, and nitric oxide levels in the livers. Gut microbiota was also tested.	24 male mice of 7 week were fed with vinegar from 24th week to 3 week.	Nothing reported.	The oral intake of coconut water vinegar significantly reduced the body weight, fat-pad weight, and serum lipid profile of the HFD-induced obese mice in a dose-dependent manner. The HDL level and the ratio of HDL/LDL in the coconut water vinegar-treated groups increased.	Mohammed et al., (2019)
4.	**VCO, Coconut oil, coconut water, coconut milk**	• Possess anti-inflammatory, anti-oxidant, antidiabetes, bactericidal, anti-fungal, and antiviral activities • Phenolic and fatty acid compounds, phospholipids, tocopherols, sterols, proteins, carbohydrates, and volatile compounds in coconut oil and coconut milk	42 male Wistar rats (170–250 g) were treated with VCO (10 mg/kg body weight), refined coconut oil (10 mg/kg body weight), coconut water (4 mL/100 g body weight), coconut milk (4 mL100 g body weight) or metformin (200 mg/kg body weight) respectively for 28 days.	**Animal study**: In the STZ-induced diabetic rat model, diabetic parameters include lipid profile, electrolytes, renal, and urine parameters. • Examined the histological alterations in the renal tissue of diabetic rats.	**42** male Wister rats (170–250 g) were treated for 28 days.	Coconut milk had no significant effect on body weight	The investigated coconut products demonstrated their anti-diabetic potential by acting favourably on the measured diabetes parameters with an efficacy matching that of metformin. Moreover, kidney tissues were significantly shielded by coconut products from the histological alterations caused by diabetes.	Alatawi and Alshubaily (2021)
5.	**VCO**	• SFAs and other phenolic compounds	Every day, a balanced,	Glycemia, lipid profiles, and	29 adults administered	Anthropometric characteristics of	When added to an isoenergetic balanced	Vogel et al. (2020)

(*continued on next page*)

Table 6 (*continued*)

S. No.	Coconut derivatives	Key ingredients	Dosages	*In vitro* tests, experimental animals and human data	Characteristics, of volunteers in human/Animal trials	Adverse reactions, symptoms, and side effects, if any	Inference(s)	Reference
		have the ability to prevent obesity.	isoenergetic meal and one tablespoon (12 mL) of extra virgin coconut oil or soybean oil were given to 29 obese adult men.	anthropometric profiles were assessed at baseline and 45 days following the intervention.	with VCO/ soybean oil for 45 days.	the groups did not alter before or after the intervention	diet, coconut oil may raise HDL cholesterol and lower the TC/HDL cholesterol ratio in obese men.	

Table 7
Summary on the possible mechanisms for the anti-obesity effect of coconut derivatives.

Active Ingredient and quantity	Anti-obesity effect(s)	References
VCO: 3x10mL/day VCO integrated in diabetic regimen.	• Improves the blood glucose and lipid profiles of type 2 diabetics due to its ability to provide rapid energy to body cells. • VCO did not change BMI, fasting serum glucose, insulin, triglyceride, or ketone concentrations in type 2 diabetics, despite higher energy and saturated fatty acid intake compared to the control group. • Beneficial effects may be attributed to the increased polyphenolics and other antioxidants in hot-extracted VCO. • MCT containing 6–12 carbon fatty acids differs from LCT (which have fatty acids of >12 carbon) in that they are absorbed directly into the portal circulation and transported to the liver for rapid oxidation. LCTs, however, are transported via chylomicrons into the lymphatic system, allowing for extensive uptake into adipose tissue.	Siddalingaswamy et al. (2011)
Coconut oil: 10–15 mg/mL of coconut oil	• Coconut oil reduces lipid accumulation in hepatocytes (68%) and adipocytes (42%). • Modulates immune response in gut cells.	Machado et al. (2023)
VCO: (200 g/kg) Wistar rats	• VCO components may induce increased energy expenditure, leading to a lower weight gain. • Lauric acid and myristic acid in VCO are rapidly absorbed in the intestine, even without pancreatic lipase.	Panchal et al. (2017)
VCO: (3000 mg/kg BW), which is equivalent to 484 mg/kg in human	• VCO treatment in a diet-induced obesity models reduced body mass and adiposity indexes, improved resistance to insulin by HOMA-IR, leptin concentrations, and liver function. • VCO reverses changes in lipid profile and hepatic steatosis. • VCO treatment reduced consumption of proteins and sodium and reversed changes in somatic parameters caused by obesity, such as lower body weight gain, adiposity index, and abdominal circumference. • Oxidation of MCFA in muscles justifies the reduction of weight gain in obese rats treated with VCO. • Increase in energy expenditure induced by MCFAs, such as caprylic, capric, and lauric acids • MCFA from VCO and E-VCO negatively regulates the activity of lipoprotein lipase in adipose tissue and decrease the expression of adipogenic transcription factors. • Phenolic compounds such as rutin, 3,4-dihydroxybenzoic acid, salicylic acid, quercetin, p-coumaric acid, caffeic acid, and myricetin might also have contributed to regulating insulin response and glycaemic profile. • The polyphenols, vitamin E, phytosterols, and molecules with antioxidant and anti-inflammatory properties of coconut oils could explain their important role in liver homeostasis. • VCO administration modified the hepatic lipid metabolism, thus inducing mobilization of fat, cholesterol, and hepatic TG reserves into the blood and consequently increasing the excretion of faecal cholesterol.	de Vasconcelos et al. (2022)

beneficial effects should be entrusted. Thus, it can be safely concluded that coconut oil and its derivatives possess cardioprotective, neuroprotective, and anti-obesity properties. Nonetheless, there is a need for more randomized, controlled clinical trials that evaluate the optimal dosage, side effects, etc., so that they can come out as a recommendation in the worldwide diet.

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CRediT authorship contribution statement

P.P. Shameena Beegum: Writing – original draft, Conceptualization. **S.V. Ramesh:** Writing – review & editing, Supervision, Project administration, Investigation, Conceptualization. **Ravi Pandiselvam:** Writing – review & editing, Writing – original draft, Conceptualization. **M. Neema:** Writing – original draft, Visualization, Resources, Data curation. **Daliyamol:** Writing – original draft, Methodology. **M.R. Manikantan:** Visualization, Validation, Supervision. **K.B. Hebbar:** Supervision.

Declaration of competing interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Data availability

Data will be made available on request.

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