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Review

Health impacts of different edible oils prepared from coconut (*Cocos nucifera*): A comprehensive review☆

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ABSTRACT

Background: Edible oils, preferably plant origin are rich sources of fatty acids and other lipophilic antioxidants to the body. Among these, oils derived from the coconut kernel (*Cocos nucifera*) are widely used in India and the Asia Pacific, which includes copra oil (CO), virgin coconut oil (VCO) and refined, bleached and deodorized (RBD) oil. Based on the mode of preparation, their composition and biological effects vary.

Scope and approach: This review compares the physicochemical and biological properties of coconut oils prepared by different methods. The metabolism of coconut oil and its predominant content lauric acid is also explained. This review emphasizes the use of VCO in the prevention and amelioration of several degenerative diseases, including cardiovascular disease and cancers, over CO or RBD oils.

Key findings and Conclusion: There are no evident differences in the fatty acid profiles of CO, VCO and RBD oils. However, they differ in their polyphenol contents, which are reported to be high in VCO, possibly due to less harsh treatment during its preparation. Various epidemiological and clinical reports indicating the health risk of coconut oils could be pertinent to the data on the individuals consumed CO/RBD oil, which lacks polyphenols. Whereas, VCO have antioxidant, anti-inflammatory, lipid-lowering and cytoprotective efficacies, which may be attributed to its higher polyphenolics. Further, emerging studies have indicated that hot-extracted VCO (H-VCO) have a pharmacological advantage over VCO prepared by fermentation. At this juncture, further explorations on the biopharmaceutical potential of VCO have to be undertaken through clinical studies.

1. Introduction

Oils and fats are the part of human dietary regimen since ancient times, of which vegetable oils form an important part. As edible oils are the essential sources of fatty acids, they have an important role in determining the physiological and biochemical environment of the body. Based on the composition they are divided in to polyunsaturated (Eg: sunflower oil), monounsaturated (Eg: mustard oil) and saturated (Eg: palm and coconut oils) fatty acid rich edible oils. Among these, oils produced from coconut (*Cocos nucifera* L.) are extensively used for food and industrial purposes in India and other Asia Pacific countries. However, due to the high saturated fat content, RBD oil has been widely criticized for possible harmful effects, especially on cardiovascular diseases (Eyres, Eyres, Chisholm, & Brown, 2016). Further, consumption of thermally oxidized coconut oil has also shown to exacerbate high fructose-induced fatty liver (Narayanankutty et al., 2017). This has been now explained that loss of polyphenolic antioxidants during CO or RBD oil preparation could be responsible for this ill effects. However, recent evidence from preclinical and limited clinical studies, polyphenol-rich VCO preparations have been reported to exert beneficial effect by increasing functional HDLc level in normal and cardiovascular disease patients (Bandeira, Moreira, Rafael, Marciane, & Valdir, 2017; Cardoso, Moreira, de Oliveira, Raggio Luiz, & Rosa, 2015; Chinwong, Chinwong, & Mangklabruks, 2017).

Coconut oil is generally obtained from the mature coconut kernel by means of mechanical or thermal processes. Due to the high saturated fat content, they are resistant to oxidative modifications such as rancidification as well as thermal oxidation, making them ideal for cooking purpose. Based on the mode of preparation, oils from coconut are of different types which include copra oil (CO), virgin coconut oil (VCO) and coconut testa oil (CTO). Compared to CO and CTO, VCO doesn't undergo any kind of chemical or thermal treatment (Narayanankutty

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et al., 2016). This difference in preparation itself causes changes in their chemical composition as well as physiological activities. This review summarizes the metabolism of coconut oil specifically focusing on the health effects of different edible oils prepared from coconut, with a detailed analysis of the efficacy and possible uses of VCO against several degenerative diseases.

2. Preparation of different oils from coconut

Different coconut oils are produced from different parts of coconut by different means. Copra and RBD oils are produced from dried coconut kernel, with a difference that RBD oil undergoes chemical refinement and bleaching. The brown testa of the coconut is used for the preparation of CTO, which is actually a byproduct of coconut oil preparation. Compared to CO and RBD oils, VCO depends on a "wet method" using fresh coconut milk. As there is no specific method of preparation of VCO has been established, all types of preparations that do not involve refinement and alterations in the oil are considered as virgin.

2.1. Copra oil

Copra is the dried coconut kernel; the fresh coconut kernel is dried in the oven or sunlight and oil is collected by mechanical milling. The oil is collected and sun-dried to remove the moisture content.

2.2. Testa oil

Coconut testa oil is the emerging form, which can be extracted using isopropyl alcohol from the coconut testa (Zhang, Zheng, Duan, & Gui, 2016). According to the authors, CTO is best obtained at a temperature of 60 °C for a period of 3 h with the substrate to solvent ratio of 1:4 and having a yield up to 63–76%. Since the extraction involves chemical solvents, the oil has not yet been widely used for edible purposes.

2.3. Virgin coconut oil

Virgin coconut oil is extracted from the fresh coconut kernel using natural means without the application of high temperature or chemical treatment. Based on the mode of preparation, several types of VCO are available.

2.3.1. Cold extraction (C-VCO)

Cold processing is the method of extraction of VCO without the aid of heat. Here, the coconut milk is subjected to chilling (2–8 °C) overnight and the separated oil is collected by centrifugation, filtered and stored. This is a simpler and cheapest method available.

2.3.2. Hot extraction (H-VCO)

Hot extraction is traditionally used in Southern India for VCO preparation. In this method, the coconut milk is subjected to a moderate temperature of up to 100 °C. The processing lasts for 60 min or until the oil get completely separated from the milk and the oil formed is collected by filtration. This heating process helps to increase the release of bound phenolic acids into the oil and also yield is much higher. The oil prepared in this way is being used conventionally in the Ayurvedic system of medicine for skin ailments, especially in children.

2.3.3. Fermentation technique (F-VCO)

The fermentation method uses bacterial activity to generate VCO has also been proposed. It is mainly of two types-natural fermentation as well as induced fermentation. In the natural fermentation method, the fresh grated coconut kernel is extracted with its water to collect the coconut milk. It is then kept for 24–48 h under room temperature (or up to a temperature of 45 °C) to allow fermentation and separation of oil layer, which is scooped out, filtered and stored (K G Nevin & Rajamohan, 2006). Masyithah (2017) prepared VCO by induced fermentation technique, where they used *Saccharomyces cerevisiae* and *Lactobacillus plantarum* (strain 1041 IAM) for the extraction of VCO from coconut milk. Other than L. *plantarum*, *L. delbrueckii* is also used in the fermentation process. However, studies using induced fermentation are quite rare and VCO produced by natural fermentation method is often regarded as F-VCO.

2.3.4. Enzymatic extraction technique

In the enzymatic process, a mixture of enzymes is used to release the oil portion from the coconut milk. It contains α-amylase, which produces simpler carbohydrates from starch, a protease for removal of plant proteins, polygalacturonase and cellulase for removing cell wall components.

3. Physicochemical properties

3.1. Fatty acid composition

Fatty acid profiles are found to be similar in all the different varieties of coconut oils such as CO, VCO or RBD oil. In CTO, there is comparatively a higher level of unsaturated fatty acids than CO/VCO, with a concomitant reduction in the medium-chain saturated fatty acid (MCFA) (Appaiah, Sunil, Prasanth Kumar, & Gopala Krishna, 2014). The other oils contain high amounts of MCFA (Table 1). Among these, lauric acid (C12:0) forms the predominant fatty acid (45–52%), followed by myristic acid (15–19%) and palmitic acid (10–11%). As indicated by the triglyceride composition of these oils, the major triacylglycerol is formed by tri-lauryl glycerols (22.2–23.9%). On the other hand, CTO contains higher levels of CLaLa (18.7%), followed by trilauryl glycerol (14.3%) and LaLaO (13.4%). As shown in Table 2, other than these, triglycerides of lauric, capric and myristic acids are also present in different types of coconut oils (Appaiah et al., 2014) (Table 3).

3.2. Phenolic composition

Polyphenols are another main group of bioactive compounds

Table 1
Fatty acid composition of virgin coconut oil.

Fatty acid	CO (Sheela et al., 2016)	RBD (Prasanth Kumar & Gopala Krishna, 2015)	VCO (From our Lab reports)	CTO (Appaiah et al., 2014)
Caproic acid (C6:0)	–	0.02	0.6	–
Caprylic acid (C8:0)	9.6	7.24	0.8	1.6–3.9
Capric acid (C10:0)	6.4	5.25	6.4	2.2–3.8
Lauric acid (C12:0)	51.5	50.9	48.5	32.4–42.9
Myristic acid (C14:0)	19.1	21.3	17.6	20.2–20.9
Palmitic acid (C16:0)	6.9	9.22	8.4	11.3–14.1
Stearic acid (C18:0)	1.1	0.38	2.5	1.2–1.6
Linoleic acid (18:1)	4.3	4.81	6.5	12.2–17.8
Linolenic acid (C18:2)	1.1	0.81	1.5	5.3–10.6

Table 2
Phenolic acids of different coconut oil preparations (mg/Kg).

Phenolic acid	CO (Appaiah et al., 2014)	RBD (Seneviratne et al., 2009)	VCO (Seneviratne & Sudarshana Dissanayake, 2008)	CTO (Appaiah et al., 2014)
Total Polyphenols	131.2	618	322	313.9
Protocatechuic acid	–	0.16	–	–
Gallic acid	24.7	–	–	32.1
Hydroxy benzoic acid	7.6	–	–	126.4
Vanillic acid	63.8	–	2.08	–
Syringic acid	17.9	–	0.45	–
p-Coumaric acid	10.0	0.34	2.0	42.1
Caffeic acid	3.1	0.13	3.0	12.8
Ferulic acid	1.7	0.31	3.3	47.5
Cinnamic acid	2.4	–	–	4.1

Table 3
Triacylglycerol composition of different edible oils of coconut.

Type	CO (Appaiah et al., 2014)	RBD (Prasanth Kumar & Gopala Krishna, 2015)	VCO (Marina et al., 2009)	CTO (Appaiah et al., 2014)
CCICI	–	–	0.00–0.44	–
CpCpLa	1.21	1.36	0.41–0.95	1.68
CpCLa	2.24	3.20	3.81–4.11	7.91
CCLa	7.41	11.57	12.7–13.8	12.20
CLaLa	12.18	16.47	17.0–21.1	18.76
LaLaLa	16.23	19.63	22.2–23.9	14.30
LaLaM	16.0	17.27	17.9–19.8	5.04
LaLaO	3.87	2.28	1.72–1.88	13.42
LaMM	13.73	10.6	10.8–12.1	4.91
LaMO	4.62	2.18	1.00–1.24	6.21
LaMP	6.81	5.35	4.72–6.26	0.29
LaOO	–	1.59	0.57–0.74	–
LaPP	1.66	1.93	0.83–1.64	1.43
MOO	1.97	0.68	0.00–0.27	1.10

Cp, caproic; Cl, caprylic; C, capric; La, lauric; M, myristic; P, palmitic; O, oleic.

present in edible oils prepared from coconut. Several studies have analyzed the phenolic content and composition of various types of coconut oils, among which VCO prepared by hot pressing and fermentation methods have higher levels of phenolic antioxidants (Narayanankutty et al., 2016; Seneviratne & Sudarshana Dissanayake, 2008). These studies also indicated the possibility of temperature-dependent release of phenolic acids from coconut kernel to the oil portion. Individual polyphenols present in coconut oil are p-coumaric acid, ferulic acid, caffeic acid, quercetin and catechins and the level of these are found to be higher in F-VCO compared to CO and RBD oil (Illam, Narayanankutty, & Raghavamenon, 2017). It is now being believed that these polyphenolic compounds or their derivatives may be involved in the actual biological properties exhibited by different VCO preparations. Previous epidemiological and clinical studies that projected the health risks of coconut oil are based on data collected from the individuals who consumed either CO or RBD oil (Eyres et al., 2016). This projection is hardly acceptable as polyphenol-rich VCO exhibit beneficial effects against cardiovascular disease in recent randomized control trials (Bandeira et al., 2017; Cardoso et al., 2015; Chinwong et al., 2017). The common polyphenols including phenolic acids of different coconut oils are summarized in Table 2.

4. Digestion and metabolism of coconut oil

The medium chain triglycerides (MCT) are easily hydrolyzed by various lipases in the gastrointestinal tract of humans. Compared to long-chain triglycerides (LCT), MCT rich coconut oils are easily digestible and absorbable (Łoś-Rycharska, Kieraszewicz, & Czerwionka-Szaflarska, 2016). In the colon tissues of rats, the MCTs are readily absorbed through the villi at a faster rate than that of long chain fatty acids (LCFA) and enter the circulation faster (Schönfeld & Wojtczak, 2016). Since coconut oils are rich in lauric acid, the hydrolysis of MCTs yields a sufficient quantity of mono lauryl glycerol which subsequently generates pharmacologically active compounds such as monolaurin (McCarty & DiNicolantonio, 2016).

Like any other lipids, mitochondrial beta-oxidation is the predominant catabolic pathway for MCFAs also. The MCFAs are easily transported across the mitochondrial membrane without any carrier molecule and hence they are rapidly metabolized in the liver (Wang et al., 2018). In mitochondria, medium-chain acyl-CoA dehydrogenase (MCAD) and long-chain acyl-CoA dehydrogenase (LCAD) are responsible for the β-oxidation of MCFAs including lauric acid generating acetyl-CoA (Bonito, Leandro, Ventura, & Guedes, 2016; Tucci, Behringer, & Spiekerkoetter, 2015). The metabolic fate of acetyl-CoA is varied, which includes the formation of ketone bodies such as acetoacetic acid and beta-hydroxybutyric acid (Nonaka et al., 2016). Under an MCT diet, there occurs the formation of a large quantity of ketone bodies, especially in the astrocytes (Thevenet et al., 2016). These ketone bodies formed by the metabolism of MCTs are also found healthy in the prevention of a variety of disorders including colon cancer (Kadochi et al., 2017).

In a separate experiment, Rioux, Daval, Guillou, Jan, and Legrand (2003) have shown that a fraction of the lauric acid in hepatic cells tends to undergo elongation resulting in the formation of myristic acid and palmitic acid, which is later used for N-myristoylation or S- acylation of protein (Udenwobele et al., 2017). N-myristoylation is an important post-transcriptional modification of proteins, which is involved in a variety of cellular functions (Perinpanayagam et al., 2013). Numerous genes associated with apoptosis and autophagy such as Calcium- and integrin-binding protein 1 (CIB1), p21-activated kinase 1 (PAK1) and polo-like kinase 3 (PLK3) are regulated by the N-myristoylation process (Leisner, Freeman, Black, & Parise, 2016; Martin & Hayden, 2015; Thinon, Morales-Sanfrutos, Mann, & Tate, 2016). It is therefore expected that high intake of MCT in the form of coconut oil may possibly regulate the different aspects of cellular proliferation and apoptosis; however, these aspects need further evaluation.

Gluconeogenesis is another important fate of acetyl co-A; where, a high MCT diet has reported to increase gluconeogenesis process in normal rats (Sugiyama, Akter, Morishita, Miura, & Takase, 2015). Considering the dual roles of gluconeogenesis in various disease conditions, especially in carcinogenesis (Balsa-Martinez & Puigserver, 2015; Khan & Chakrabarti, 2015; Khan et al., 2015), the MCT diet-induced activation of gluconeogenesis may influence the cancer cell growth in either way. Therefore, it needs a thorough evaluation of the effect of long-term MCT feeding on gluconeogenesis and its impact on various physiological and pathological processes.

In addition to the beta-oxidation mediated metabolism, MCTs are also metabolized by omega (ω) oxidation to a lesser extent. Cytochrome P450 4A11 is the major enzyme involved in the ω-oxidation of lauric acid together with CYP 4A1 (lauric acid hydroxylase) and CYP 450 2E1 (Uehara et al., 2016). The metabolic conversion of various MCTs by omega oxidation and their products are emerging and thorough studies are necessary in these aspects.

5. Biological and pharmacological effects

In the past few years, clinical studies have revealed that coconut oils are not causing problems instead are healthy (Chinwong et al., 2017; Khaw et al., 2018). Among the different coconut oils, F-VCO is studied in detail than others. Different researchers studied the biological effects of VCO and compared with that of other coconut oils, and also using VCO of different preparation methods.

5.1. Antioxidant and anti-inflammatory activities

Among the various VCO preparations, F-VCO and H-VCO showed higher radical scavenging and inhibition of lipid peroxidation properties than cold extraction methods (Marina, Man, Nazimah, & Amin, 2009; Seneviratne, HapuarachchI, & Ekanayake, 2009). Substantiating the observations, *in vivo* validation studies also showed the antioxidant potentials of different VCO preparations, especially F-VCO. Among the many studies using VCO, K G Nevin and Rajamohan (2006) are the first to report the antioxidant activities in rats. They analyzed the effects of feeding of F-VCO, sunflower oil and groundnut oil on serum and tissue antioxidant status. Results indicated an increase in the activities of enzymatic antioxidants such as catalase and superoxide dismutase which are involved in the detoxification of peroxide and superoxide radicals. In addition to the improvement in antioxidant enzyme activities, VCO also enhances the levels of intracellular reduced glutathione in cell culture as well as animal models, which is actively involved in the phase II detoxification system (Illam et al., 2017). As reported by Narayanankutty et al. (2016), enhancement in the GSH levels in diabetic rats by VCO are thought to be due to the down-regulation of polyol pathway enzymes such as aldose reductase and sorbitol dehydrogenase. Compared to other oils from coconut, studies on the *in vivo* antioxidant activity are still lacking with CTO, though the *in vitro* antioxidant potentials are promising.

Anti-inflammatory activity of VCO is proven several years back and thereafter, various studies have proposed that the anti-inflammatory activity of VCO and its mechanism of action in various experimental models. Studies conducted by Intahphuak, Khonsung, and Panthong (2010) showed the protective effect of F-VCO in granuloma formation in chemically induced ear and paw oedema models. F-VCO also reduced adjuvant-induced arthritis in rats, by downregulating the expressions of cyclooxygenase, inducible nitric oxide synthase and TNF-α (Vysakh et al., 2014). A study conducted by Zakaria, Somchit, et al. (2011) observed that F-VCO efficiently reduce acute inflammation, however in chronic models, it is found to be less effective. Together with anti-inflammatory activities, anti-nociceptive and analgesic activities are also reported for F-VCO (Intahphuak et al., 2010; Zakaria, Somchit, et al., 2011). Other VCO preparations are not yet evaluated for their anti-inflammatory activity.

5.2. Lipid metabolism and cardiovascular disorders

It has been reported that saturated fats are responsible for hyperlipidemia and increased risk for cardiovascular diseases (Hooper, Martin, Abdelhamid, & Davey Smith, 2015; Lin et al., 2005). However, VCO which contain medium chain triglyceride (MCT) is reported to have beneficial effects on lipid metabolism (Resende et al., 2016). According to the studies conducted by K Govindan Nevin and Rajamohan (2004) and (Sheela, Nazeem, Narayanankutty, Manalil, & Raghavamenon, 2016), F-VCO and lauric acid reduce the levels of total cholesterol, triglycerides, and LDL fractions in rats to a better extent, compared to the CO. In separate studies by Govindarajan and Vellingiri (2016); K. G. Nevin and Rajamohan (2009); Shariq et al. (2015), where dietary cholesterol is co-administered with VCO, a similar reduction in the lipid parameters is noticed. Providing insight into the mechanism of action, studies conducted by Arunima and Rajamohan (2012) showed a reduction in the activities of hepatic lipogenic enzymes HMG-CoA reductase, glucose-6-phosphate dehydrogenase and isocitrate dehydrogenase. Inhibition of *de novo* fatty acid synthesis by F-VCO is further explained by the reduction in the expression of genes including acyl CoA carboxylase, fatty acid synthase and its transcriptional regulator sterol regulatory element binding protein (SREBP-1) (Arunima & Rajamohan, 2014). They also observed an increase in the lipolytic enzyme lipoprotein lipase, Carnitine palmitoyltransferase I, acyl CoA oxidase via peroxisome proliferator-activated receptor alpha (PPARα)-dependent pathway (Arunima & Rajamohan, 2012, 2014). Further, clinical studies have reported a reduction in visceral adiposity in healthy volunteers (Liau, Lee, Chen, & Rasool, 2011) and improvement in lipid metabolism in pre-menopausal women (Feranil, Duazo, Kuzawa, & Adair, 2011) as well as coronary artery disease patients (Cardoso et al., 2015).

In addition to the roles played on lipid metabolism, the effect of F-VCO on cardiovascular diseases prevention is further explained by their ability to promote reverse cholesterol transport. In a recent study conducted by Arunima and Rajamohan (2016b) in hepatocytes and macrophages, lauric acid present in F-VCO is found responsible for the reverse cholesterol transport (RCT) by up-regulating the expression of SRB1 and ABCA1 partly by PPAR α - LXR α dependent pathways. Supporting the above, increased levels of lecithin-cholesterol acyl transporter (LCAT) and paraoxonase I activity is improved in rats fed with 8% VCO (Arunima & Rajamohan, 2013). LCAT is an enzyme that sequesters free cholesterol into the core of lipoproteins thereby synthesizing new HDLc particles, whereas paraoxonase is a well-known antioxidant enzyme associated with HDL that protect LDL and HDL from oxidative modifications.

Other than lipid metabolism, factors controlling platelet aggregation and blood coagulation also control the process of development of atherosclerosis (Badimon, Padró, & Vilahur, 2012; Spronk, van der Voort, & ten Cate, 2004). The important molecules involved in the aforementioned processes are fibrin, fibrinogen and thromboxane B2. Treatment with F-VCO reduced the levels of these molecules with a concomitant hike in the prothrombin and activated partial thromboplastin time (Arunima & Rajamohan, 2016a; K. G.; Nevin & Rajamohan, 2008), indicating the antithrombotic activity of F-VCO by modulating both extrinsic and intrinsic coagulation pathways. F-VCO also shows improvement in hypertension and endothelial functioning in thermally oxidized oil fed rats, with a restoration of histomorphometric parameters of aorta and heart (Kamisah et al., 2015; Nurul-Iman, Kamisah, Jaarin, & Qodriyah, 2013).

5.3. Diabetes and insulin resistance

Insulin resistance is one of the most common and important factor involved in the development of metabolic disorders. As a fatty acid source, F-VCO plays an important role in regulating insulin resistance and associated glucose metabolism. Studies have shown that F-VCO effectively reduce hyperglycemia in alloxan-induced diabetic rats (Iranloye, Oludare, & Olubiyi, 2013; Maidin & Ahmad, 2015); whereas Saat, Rosli, and Syakroni (2013) reported the antidiabetic effect of cold pressed VCO in streptozotocin-induced diabetes. Siddalingaswamy, Rayaorth, and Khanum (2011) compared the hot extracted VCO with cold pressed VCO and commercial CO on their protective potential in a streptozotocin-induced diabetic model and there observed a higher efficacy for H-VCO as a hypoglycemic and insulin-sensitizing agent than the other oils. It is also reported that in comparison with copra oil, F-VCO efficiently prevents the development of insulin resistance and dyslipidemia in high fructose-fed rats (Narayanankutty et al., 2016). F-VCO is also shown to exert its protective effects in diabetic nephropathy in animals (Akinnuga et al., 2014). Though the mechanism of antidiabetic effect of VCO preparations has not been evaluated, it is possible that the inhibition of dipeptidyl peptidase-4 (DPP-4) or insulin sensitization by the phenolic compounds play an important role in its antidiabetic activity (Sheela et al., 2017). In addition, inhibition of polyol pathway by these phenolic compounds may also offer protection against secondary diabetic complications such as diabetic nephropathy.

5.4. Cancer prevention and management

There are very limited studies on the anticancer activity of coconut oils. The first study reporting the anticancer effect is done by Craig-Schmidt, White, Teer, Johnson, and Lane (1993), who observed a significant reduction in DMBA induced mammary tumourigenesis in Balb/

c mice treated with a combination of coconut oil with menhaden oil. In a separate study, Enos, et al. (2016) observed that a diet rich in coconut oil efficiently reduce the ulcerative colitis and associated colon cancer incidence in Azoxymethane/Dextran sodium sulphate induced colon cancer model. It has been found that treatment with coconut oil 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 fatty acid in VCO, have recently shown to induce apoptotic changes in various colorectal cancer cells mediated by reactive oxygen species (Fauser, Matthews, Cummins, & Howarth, 2013). Furthermore, lauric acid induces cell cycle arrest in G0/G1 and G2/M phases as well as ROS induced activation of Rho-associated kinase-mediated pathway and p21 dependent apoptosis (Lappano et al., 2017).

5.5. Amelioration of chemotherapy-induced toxicity

In addition to the anticancer effects, VCO by virtue of its strong antioxidant activity has shown to reduce the complications associated with chemotherapy. In an animal model study, Nair, et al. (2016) have shown that F-VCO effectively ameliorates the myelosuppression and disturbed antioxidant status induced by chemotherapeutic drug cyclophosphamide. Similarly, F-VCO has also been shown to reduce the hepato-renal toxicities of methotrexate due to their anti-inflammatory and antioxidants potentials (Famurewa, Aja, et al., 2017; Famurewa et al., 2018; Famurewa et al., 2017). Further, consumption of VCO by breast cancer patients is shown to improve their quality of life by reducing the side effects of chemotherapy such as sleeping difficulties, dyspnea and problems associated with food intake and loss of appetite (Law et al., 2014). Similarly, VCO and salt soda mouthwash reduced problems associated with radiation-induced mucositis in nasopharyngeal carcinoma patients (Cruz et al., 2014).

5.6. Hepatic damage and fatty liver disease

The liver is an important organ dealing with detoxification and elimination of wastes and toxic products. During this processes, damages to the hepatocytes are not uncommon. Several of the drugs and chemical compounds in the diet are harmful to the hepatocyte leading to hepatotoxicity. FVCO has shown to reduce paracetamol-induced toxicity by restoring liver function markers and hepatic morphology (Zakaria, Rofiee, et al., 2011). Similarly, the common antibiotic trimethoprim-sulfamethoxazole induced toxicity is also reduced by cold pressed VCO administration (Otuechere, Madarikan, Simisola, Bankole, & Osho, 2014). Providing the mechanistic basis, it is reported that the protective effect of VCO (cold pressed) is due to the involvement of cytochrome P450 and partly due to its antioxidant activity (Rofiee et al., 2011). It is thus expected F-VCO and H-VCO may exert a similar inhibitory effect on the CYP450 system and its extent of inhibition needs to be further evaluated. Further, our recent study has indicated that F-VCO treatment facilitates the reversal of already developed hepatosteatosis by the restoration of redox balance (Narayanankutty, Palliyil, Kuruvilla, & Raghavamenon, 2018).

5.7. Neurological disorders including Alzheimer's disease

Compared to the studies conducted in other areas of human health and diseases, the effect of virgin coconut oil is less studied with respect to neurological ailments. Nafar and Mearow (2014) are the first to demonstrate the possible beneficial effects of cold pressed virgin coconut oil in the prevention of neurodegenerative disorders. Their results have shown that treatment of cortical neuronal cells with coconut oil improves cell survival by reducing the mitochondrial alterations as a result of amyloid beta-peptide-induced toxicity. Further, the studies conducted by Hu Yang et al. (2015) observed that consumption of a daily dose of 40 mL of VCO by Alzheimer's patients improved their cognitive status. Though the study is found to be promising, the results are dependent on the age, sex as well as the diabetic history of the patients. Later, Fernando, et al. (2015) discussed the possibilities of VCO as a potential therapeutic agent against the neurological disorders including Alzheimer's disease. According to him, as medium chain saturated fat-rich diets are ketogenic, it can have a greater role in the prevention of neurotoxicity. Additionally, the antioxidants in the VCO can also offer higher protective effects compared to other edible oils.

5.8. Effect on skin diseases

Traditionally, H-VCO has been in use as a skin protective agent, especially for newborn babies. By virtue of its high antioxidant potentials and antibacterial activities, VCO is capable of preventing several skin diseases. The initial reports on the efficacy of VCO as a moisturizer in conditions of Xerosis, which is characterized by the rough and scaly skin which causes a defect in its barrier functioning (Agero & Verallo-Rowell, 2004). Evangelista, Abad-Casintahan, and Lopez-Villafuerte (2014) observed the protective effect of cold pressed VCO under pediatric atopic dermatitis, where it is shown to improve the epidermal barrier functioning, subcutaneous infection, and hydration. Other than in skin infections, VCO has also been shown to accelerate the dermal wound healing process, mediated by their antioxidant potentials (K. G. Nevin & Rajamohan, 2010).

5.9. Antibacterial and antiviral activities

VCO is traditionally used as an antibacterial agent. It has been shown that F-VCO possess antibacterial activities against a variety of strains including Candida (Ogbolu, Oni, Daini, & Oloko, 2007) and Staphylococcus (Tangwatcharin & Khopaibool, 2012). VCO also reduces plaque-related gingivitis, which is a bacterial infection induced oral disease (Peedikayil, Sreenivasan, & Narayanan, 2015). Possible antimicrobial activities of VCO may be attributed to lauric acids, which forms the major fatty acids of VCO (Nakatsuji et al., 2009). Monolaurin compounds are the major metabolite which is responsible for its activity (Manohar et al., 2013). There are no similar studies available on the other edible oils from the coconut.

6. Future perspectives

Edible oils from the coconut kernel, especially VCO, have been always in the limelight of scientific research. Though VCO prepared by different methods vary in their phenolic composition and thereby their biological effects, only fermentation processed VCO has been explored in detail. Compared to F-VCO, hot pressed VCO possess higher antioxidant molecules, studies on this oil remain limited. In addition, CTO, a new member of the coconut oil family also remains unexplored. Considering their higher levels of phenolic antioxidants, these oils may prove better than the existing cold pressed and F-VCO preparations.

Further, studies on the bioactivities of edible oils from coconut have established its effects on lipid metabolism including inhibition of cholesterol biosynthesis, upregulation of HDLc levels and improving reverse cholesterol transport mechanisms. Though studies have agreed on the hike in GSH (reduced glutathione) levels, the molecular mechanism is not explored; the possible involvement of nrf2 system is expected. Nrf2, a nuclear transcription factor which is central to redox balance in the body, regulates the expression of various downstream genes including GSH, glutathione peroxidase (GPx) as well as glutathione-S-transferase (GST). These key enzymes are involved in the detoxification of xenobiotics in cells, thereby protecting against the exogenous oxidative insults. Studies in these lines may provide a novel area for research and will effectively explain the future interventions possible through the pathway. In addition to the individual effects of VCO preparations, recent studies have identified that VCO in combination with curcumin act as a natural skin lightening enhancer (Bridgemohan

& Khan, 2014). It is thus possible that the oil may also act as a suitable base for the preparations for several drugs and drug combinations.

7. Conclusion

Coconut has been a rich source of edible oils; the pharmacological and nutritional potentials of which varies based on their mode of preparation. In the recent years, virgin coconut oil is the most studied and has emerged as a functional food and nutraceutical. Compared to RBD oil, which has been previously described as deleterious to human health; VCO, by virtue of their MCFA content and high amounts of phenolic antioxidants shown to exert potential preventive and curative efficacy in several conditions such as hyperlipidemia, fatty liver, diabetes, and cancers. Though there is sufficient pre-clinical support is available, limited clinical studies have been carried out except for few cases of cancers and cardiovascular diseases, which shows promising results. In view of all these, it is suggested that more clinical exploration of VCO have to be made as a preventive, curative as well as adjuvant nutraceutical.

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