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Virgin Coconut Oil and Its Potential Cardioprotective Effects

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Abstract: Emphasis on diet to improve the cardiovascular (CV) risk profile has been the focus of many studies. Recently, virgin coconut oil (VCO) has been growing in popularity due to its potential CV benefits. The chemical properties and the manufacturing process of VCO make this oil healthier than its copra-derived counterpart. This review highlights the mechanism through which saturated fatty acids contribute to CV disease (CVD), how oils and fats contribute to the risk of CVD, and the existing views on VCO and how its cardioprotective effects may make this a possible dietary intervention in isolation or in combination with exercise to help reduce the burden of CVDs.

Keywords: coconut oil; cardiovascular disease; diet; lifestyle intervention

Introduction

Poor diet is well established as a primary cardiovascular disease (CVD) risk factor.[1] In particular, it has been found that total fat, saturated fatty acid (SFA), and monounsaturated fatty acid (MUFA) intake are strong predictors of coronary heart disease (CHD) mortality.[2] Associations have also been found between SFA and incident CVD.[3] This has led to numerous interventions being designed on diet modifications to improve the cardiovascular (CV) risk profile. In general, diets consisting of nonhydrogenated unsaturated fats, whole grains, fruit and vegetables, and omega-3 polyunsaturated fatty acids (ω-3 PUFAs) have been shown to be protective against CVD.[4] Similar findings have been echoed by other large-scale studies and supported by consensus statements.[5] The CV benefits of ω-3 PUFA consumption have been found to be greater than those found with ω-6 PUFA consumption.[6]

Methods

To find animal and human trials of VCO and its cardioprotective benefits, a systematic search was performed in PubMed using the terms *virgin coconut oil, coconut oil, cardiovascular disease, fatty acids,* and *lipids*. The boolean terms AND and OR were also used with various combinations of these terms. The search was limited to English-language articles published through November 2013. In addition, a search was performed in Google Scholar of the gray literature published in this area.

How Do SFAs Contribute to Atherosclerosis?

The process of atherosclerosis begins at the susceptible site of arteries. Imbalances between various anti- and proinflammatory substances following endothelial and vascular intimal stress initiate the process. Development of isolated macrophage

foam cells (type I) lead to the formation of multiple foam cell layers (type II), which over time have isolated extracellular lipid (type III) that was added to these layers.[7] These susceptible sites facilitate faster accumulation of lipids and faster progression of lesions to the development of the lipid core (type IV) and the fibromuscular layers (type V). At this stage, the plaque is extremely vulnerable and can cause a surface defect, hematoma, or thrombosis (type VI). In type VII, calcification predominates in the lesion, and finally, in type VIII, fibrous tissue changes occur.

The high dietary intake of SFA and cholesterol increases the low-density lipoproteins (LDLs), thereby facilitating and potentially accelerating the process of atherosclerosis. The SFAs have been shown to produce LDL particles that are larger in size, more active, and enriched with cholesteryl oleate, thereby propagating the progression of atherosclerosis.[8] All forms of fat (ie, cholesterol, MUFA, and SFA) are responsible for the formation of cholesteryl ester, which is driven by the activation of cholesterol acyltransferase. Esterification of dietary cholesterol results in cholesteryl oleate enrichment of very-low-density lipoproteins (VLDL). Cholesteryl oleate enrichment of LDL has been shown to be the primary culprit in the propagation of the initial steps in the atherosclerotic cascade (ie, type I–III lesions, as mentioned above) by favoring arterial retention and subsequent foam-cell formation, thus leading on to the development of CVD. This phenomenon has been observed in both animal and human studies.

Although it is true that dietary intake of SFA contributes to the progression of atherosclerosis, it is important to remember that there is a significant contribution from various cellular and immunological factors to help further facilitate the process of atherosclerosis.[9]

Oil/Fats and Cardiovascular Disease Risk

Oils rich in saturated fats have been identified as a catalyst promoting atherosclerosis, thereby increasing the risk for CVD. A recent publication from the Global Burden of Disease presented information on the consumption of oil in various regions of the world.[10] It brought to light the global problem of trans fatty acid consumption beginning at a young age and further highlighted how this consumption has now become part of our lifestyle. Given these trends, it is not surprising to find a rise in CVD across the globe.

Fats and oils contain various proportions of both long-chain and medium-chain fatty acids (LCFAs and MCFAs). Oils rich in LCFA contain varying proportions of SFA, PUFA, and MUFA, whereas those rich in MCFA contain medium-chain SFAs (Figure 1).

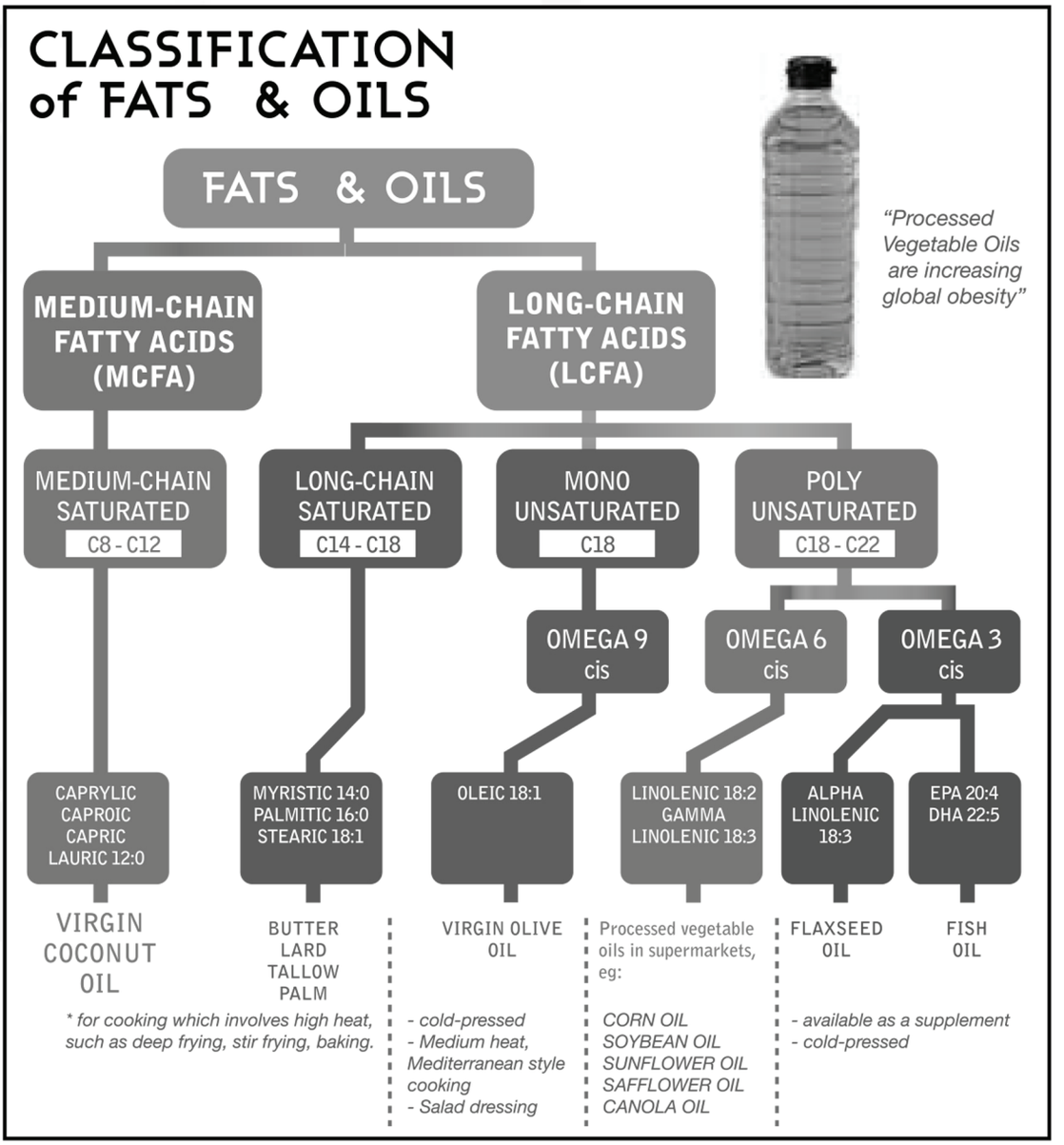
The SFAs have been suggested to increase LDL and total cholesterol.[11] Thus, it is essential to emphasize the "better" oils that have lower SFAs and higher PUFAs. This emphasis ushered in the era of ω-3 PUFAs beginning in the 1980s. The ω-3 and ω-6 PUFAs are essential fatty acids commonly obtained through various dietary sources. The ω-3 PUFAs are available from various dietary sources such as salmon, trout, herring, canola oil, walnuts, and flax seed, whereas ω-6 PUFAs are obtained from soybean oil, corn oil, and safflower oil.[12]

The earliest observation regarding the possible benefits of ω-3 PUFAs occurred among Eskimos, who have a very low CVD- related mortality.[13] An early study by Hay et al[14] found that a diet rich in ω-3 PUFA reduced platelet/vessel-wall interaction in a small group of patients with CHD. This dietary approach has also been found to significantly improve dyslipidemia by reducing VLDL and the production of thromboxane. Subsequently, considerable emphasis has been placed on utilization of oils with higher ω-3 PUFAs and lower SFAs. Globally, the mean intakes of ω-3 PUFA from seafood and plants were 163 mg/day and 1371 mg/day, respectively, with wide regional variations in their consumption.[15] Marine-based ω-3 PUFAs reduce inflammation through various pathways, thereby increasing its antiatherogenic properties.[16] This anti-inflammatory effect of ω-3 PUFAs works toward the stabilization of atherosclerotic plaque. It has been shown that the daily consumption of 1 g of ω-3 PUFA was associated with a 50% reduction in CVD risk.[17] This benefit in reduction of CVD risk is due to antithrombotic effects, improvements in endothelial function, and a reduction in inflammation.[18]

Commercially, there have been parallel efforts to market oils with low SFAs. When various oils were tested to identify the concentration of SFA, it was found that coconut oil led the way with the highest SFA (86%), whereas canola oil had the lowest amount of SFA (7%).[19] This caused a greater push from manufacturers to promote healthy oils, and coconut oil began to be sidelined after being classified as an unhealthy oil.

The need arose for studies to determine if changes in SFA intake and its replacement with other sources of fat or carbohydrates would alter CVD risk. A large prospective trial on women found that a 5% increase in intake of SFA resulted in a 17% increase in CHD risk.[20] Similar findings have been subsequently demonstrated. An increase in palm oil (SFA content 49%) consumption was found to be related to a higher mortality due to CHD in developing countries.[21] Through intentional SFA reduction, a large trial found that

Figure 1. Classification of fats and oils.



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the replacement of 5% SFA with unsaturated fats would reduce CHD risk by 42%.[20] Recent studies have shown that for every 1% of SFA replaced with PUFA, there has been an associated 2% to 3% reduction in the incidence of CHD.[22] Thus, it appears that modification of SFA intake is imperative for control of CVD risk. Nevertheless, this concept is not without controversy.[23–25]

The American Heart Association has provided dietary recommendations for the consumption of ω-3 PUFA for those with and without CHD as well as for individuals requiring triglyceride reduction.[26] The association endorsed supplementation through other sources (eg, prescription supplements) besides diet for those with established CHD due to the high daily recommendations for ω-3 PUFA. A recent paper by Harris et al[27] elucidated recent developments and trends in the use of ω-3 PUFA, including the various formulations available as supplements through prescriptions.

From the above discussion it is clear that there are significant benefits of consuming ω-3 PUFA when accompanied by a reduction in SFA. The inclusion of MCFA also entails significant health benefits. The MCFAs are found in medium chain triglycerides, which contain a mixture of MCFAs

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(ie, C6:0, C8:0, C10:0, and C12:0) that are obtained through the hydrolysis of coconut oil followed by the fractionation of fatty acid.[28] The MCFAs have a smaller molecular size, which makes them more water soluble. The added property of their being a weak electrolyte and their ability to be easily ionized at a neutral pH make them more soluble in body fluids. The smaller molecular weight of the MCFAs facilitates the action of pancreatic lipase and enables absorption of the hydrolyzed products that is nearly as fast as that of glucose. The MCFAs follow the portal venous system, unlike the LCFAs, which follow the lymphatic system. The rapid deposition to the liver reduces the amount of free MCFAs in the blood.[28] The MCFAs are absorbed faster in the intestine, and enter the portal vein, where they bind to albumin and are transported directly to the liver (Figure 2). In the liver, they get oxidized immediately for providing energy, thus the rapid transportation and oxidation of MCFAs in liver reduce the amount of free MCFA in the blood.[28]

The other fatty acids that enter the lymphatic system travel through the peripheral tissues and finally reach the liver, leading to their deposition in tissues and the bloodstream. The transport of MCFAs also differs from that of LCFAs in that there is no sterol absorption and no incorporation into the chylomicrons (crucial steps in the transport of LCFAs). Due to these benefits, MCFAs have been used for various diseases such as fat malabsorption and gallbladder disease. However, in spite of these numerous benefits on fat deposition, the role of MCFAs in CVD prevention did not receive attention until recently with the increasing popularity of VCO. The following sections highlight the importance of VCO and its CVD benefits reported in both animal and human trials.

Coconut Oil and Virgin Coconut Oil

For considerable time, coconut oil has been thought to be a culprit in raising CVD/CHD risk due to its high SFA content.[29] However, it is important to also understand that this oil contains a good amount of MCFA, which has numerous health benefits, as highlighted above.[28,30] Consumption of food rich in MCFA reduces the level of body fat and the CVD/CHD risk. The mechanism through which this occurs was discussed in the previous section. With these known benefits of MCFA, it became necessary to establish methods of oil production that would enhance that the effects of MCFA and thereby bring about CV benefits. However, there is limited information on the use of coconut oil and VCO for CV benefits, which is the emphasis of this review.

It has been found that the process of production could possibly influence the content of coconut oil and thereby have an impact on health outcomes. Traditionally, coconut oil is obtained from copra (ie, the dried kernel of the coconut) through a process of refining, bleaching, and deodorizing, which results in higher levels of free fatty acid. To make this process healthier, the production process was modified, thus ushering in the era of VCO.

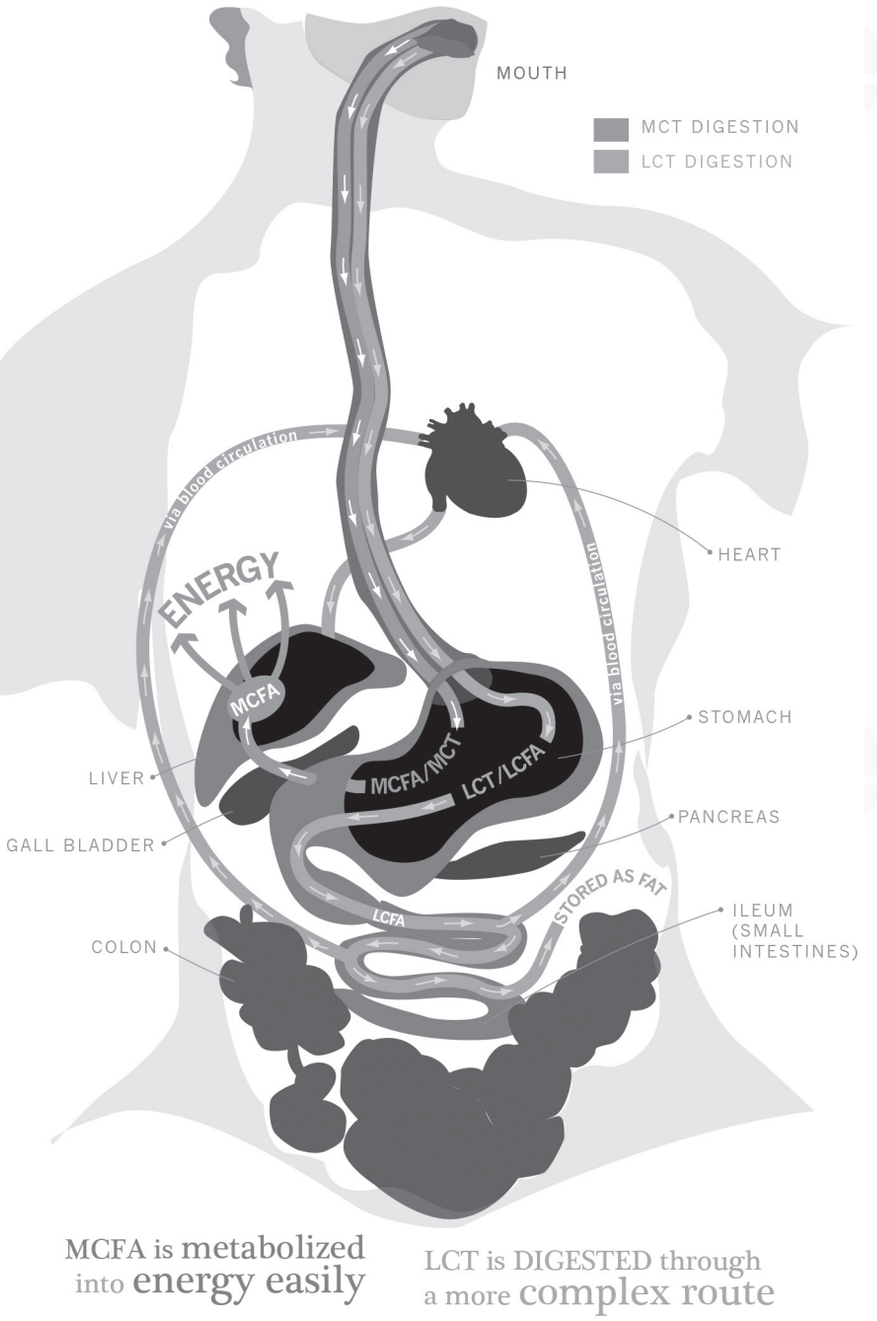
Virgin coconut oil is obtained from the fresh, mature kernel of the coconut by mechanical or natural means, with or without the use of heat and without a chemical refining process.[31] Therefore, the oil that is produced without going through the process of refining, bleaching, and deodorizing, and not altering the nature of the oil, is defined as VCO. This method of production helps preserve the nutritional value of various biologically active substances, which are normally lost during the manufacturing of coconut oil.[32] Virgin coconut oil has traditionally been prepared through fermentation of fresh coconut milk.[33] However, this method of production has resulted in a form of VCO that has poor oil recovery and a characteristic fermented odor. Thus, the wet processing technique of obtaining VCO was developed. This method of production keeps VCO rich in vitamins, minerals, and antioxidants. Technical details regarding the production of VCO are beyond the scope of this paper and are summarized in detail by Marina et al[31] and Gopala Krishna et al.[33]

Virgin Coconut Oil and Cardiovascular Benefits

Coconut has a rich abundance of MCFAs, which are rapidly absorbed in the intestine, thus not allowing for their participation in cholesterol transport[31] and providing a quick source of energy. The MCFAs are an integral component of an energy source for those recovering from chronic illnesses and for infants.[33] Virgin coconut oil has a rich content of MCFAs, consisting of caproic acid, caprylic acid, capric acid, and lauric acid.[34] Apart from the high concentration of MCFAs (59.02% to 62.27%), VCO also contains SFAs (myristic acid, palmitic acid, and stearic acid) and unsaturated fatty acid, both mono- and di-unsaturated fatty acid. These concentrations of SFA and total unsaturated fatty acid range from 28% to 31% and 6.73% to 8.13%, respectively. It is these properties of VCO that make it a potential healthy addition to the normal diet.

The potential benefits of coconut oil have been reported from animal trials of almost 2 decades ago.[35] In particular, VCO has been shown to have a high total phenolic content (11.82–29.18 mg gallic acid equivalents [GAE]/100 g oil), which is responsible for its high antioxidant properties (antioxidant activity ranging from 52.54% to 79.87%).[31,36] The phenolic content was compared with that of conventional

Figure 2. Digestion of medium-chain and long-chain fatty acids.



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coconut oil and was found to be much higher in VCO, thus further supporting the potential health benefits of VCO, which is capable of reducing the lipid peroxidation content.[34,37] The high phenol content is also responsible for normalizing lipids through various pathways.[38] The higher polyphenolic fraction of VCO is responsible for its anti-inflammatory and antioxidant effects, which all work toward the prevention of CVD by preventing the progression of atherosclerosis.[39] Apart from these benefits, VCO has also been found to enhance antithrombotic effects related to inhibition of platelet coagulation and promote anti-inflammatory effects.[40,41]

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Virgin Coconut Oil Preparations

Virgin coconut oil is commercially available and marketed by various companies around the world. Some companies call their VCO "extra virgin," but there are no specific preparation methods for achieving this "extra" status; thus, either "virgin" or "extra virgin" is appropriate for use. Recently, a capsulated version of VCO has been available in the market and has been approved by the Republic of Philippines Food and Drug Administration.

Published Studies

Animal Studies

Studies on the interaction among SFA, ω-3 PUFA, and atherosclerosis have found that VCO improved the lipid profile and prevented LDL oxidation, which is a crucial step in the progression of atherosclerotic plaque formation.[42] Virgin coconut oil supplementation in animals increases antioxidant activity and lowers lipids and thrombotic risk factors when compared with normal coconut oil.[42,43] A recent study comparing VCO with 5-times-heated palm oil found that a diet of VCO alone and VCO with 5-times-heated palm oil resulted in lower blood pressure and higher nitric oxide than in controls and in those receiving only the 5-times-heated palm oil.[44] Although VCO did not have any impact on vascular relaxation, it did decrease endothelial vasoconstriction, which the heated palm oil did not. Apart from these benefits, the blending of coconut oil with ω-3 and ω-6 fatty acids has shown beneficial effects on lipids in animal models.[33] Along with improved lipid profiles, animals fed with VCO were also found to have better coagulation studies, with lower levels of fibrin (10.5 ± 0.50 mg/dL) and better prothrombin time (11.25 ± 0.14 seconds) when compared with copra oil and sunflower oil (13.57 ± 0.53 mg/dL and 10.16 ± 0.16 seconds, and 11.70 ± 0.82 mg/dL and 11.37 ± 0.08 seconds, respectively).[40]

Human Studies

Populations commonly utilizing more coconut in their diet have been found in observational studies to have higher cholesterol levels.[45] However, a case-control study from Indonesia found no change in CVD risk based on the consumption of saturated and unsaturated fatty acids.[46,47] A randomized trial found that a 3-month supplementation of MCFA improved body weight, waist circumference, and lipid profile among overweight diabetic patients.[48] Improvements in insulin sensitivity and serum C-peptide concentrations were also observed in this group. Waist circumference reduction and no worsening of the lipid profile were found in a prospective randomized trial among women with abdominal obesity.[49] A recent prospective open-label trial found a 4-week supplementation regimen of VCO significantly reduced waist circumference (mean difference 2.87 ± 4.95 cm; $P = 0.02$) and improved lipid profile with no accompanying anthropometric changes.[50] Another study also assessed how the consumption of oil altered the lipid profile and the antioxidant levels of patients with and without diabetes.[51]

How Does Virgin Coconut Oil Fare Against Other Healthy Oils?

Up until now, there have been no direct head-to-head comparisons made between the various ω-3 PUFA–rich oils in human trials. Therefore, it is only possible to speculate what the potential benefits may be with regard to the CVD benefits of various oils together. Apart from VCO, there has been a very large emphasis placed on the use of olive oil to help reduce the burden of CVD. Olive oil has been shown to possess 75% MUFA and 15% SFA.[52] Cardioprotective changes, with subjects consuming a diet rich in olive oil along with the Mediterranean type of diet, have led to a reduction in the total burden of CVD (crude hazard ratio, 0.70; 95% CI, 0.53–0.91).[53] However, the change found in body composition (with regard to waist circumference) with VCO would suggest that there are significant benefits of VCO consumption.[54] However, trials comparing VCO with other ω-3 PUFA oils are limited, and therefore it would appear that there is no clear winner among the ω-3 PUFA–rich oils.

Future Recommendations

From the existing body of literature, it appears that there may be health benefits associated with VCO. However, evidence pertaining to VCO use is currently insufficient and of low methodological rigor to definitively recommend dietary modifications to improve CVD risk, reduce SFA, and increase PUFA. Evidence with regard to VCO and its CV benefits are at a very early stage and currently limited to animal studies and a few human trials. Even so, there seems to be initial compelling evidence that VCO may be a beneficial dietary consideration with respect to CV health. To better address this area, future research priorities by nutritionists and preventive cardiology experts should focus on the following areas of research.

In vivo studies determining the biochemical effects of VCO on lipid metabolism and establishing the pathways through which VCO alters lipid metabolism should be performed to help elucidate the biochemical basis for VCO and its cardioprotective effects. Apart from laboratory studies, it

is also important to promote translational research. Studies assessing the potentially important role for MCFA in diet to reduce CVD risk are also an important area for future research.

There is also a strong need for long-term longitudinal prospective cohort studies among various populations and economies to study their food consumption and CV risk profile. Clinical trials need to be designed to assess the effects of increased VCO consumption on the primary and secondary prevention of CHD. In addition to these trials, combining VCO/MCFAs with exercise training among various groups of patients with and without CVD and comparing various ω-3 fatty acid supplementations with VCO and other interventions (eg, other pharmacologic and exercise therapies) are important clinical studies to better establish independent and synergistic roles in decreasing CVD risk.

Conclusion

Virgin coconut oil may have a role to play in reducing the risk of CVD, thereby aiding in controlling the rising global burden of this noncommunicable disease. However, no definitive recommendations can be made given the current available evidence. More research in this area is needed and, based on initial findings, is clearly warranted before policy can be made with regard to methods of production and marketing, keeping in mind the best interest of the general population.

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Conflict of Interest Statement

Abraham Samuel Babu, MPT, Sundar Kumar Veluswamy, MPT, Ross Arena, PhD, PT, Marco Guazzi, MD, PhD, and Carl J. Lavie, MD, have no conflicts of interest to declare.

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