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REVIEW ARTICLE

Can Coconut Oil Promote Non-Alcoholic Steatohepatitis and Metabolic Syndrome? A Timely Review

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Abstract

Metabolic syndrome (MS) and non-alcoholic steatohepatitis (NASH) have been associated with bad eating habits. Supplementation with coconut oil (CO) has been proposed to control these conditions, as some authors suggest that CO provides weight loss and cardiovascular protective effects. However, the effectiveness and safety of emerging diets, such as CO, remain uncertain. This review focuses on the utilization of CO in the control of MS and NASH, besides in addition to describing this cardiovascular and weight loss effects, with special attention to the possible unfavourable impacts on human health, based on clinical and experimental studies.

Keywords

Coconut oil, High-fat diet, Steatohepatitis, Metabolic syndrome

Abbreviations

MS: Metabolic Syndrome; NAFLD: Non-Alcoholic Fatty Liver Disease; NASH: Non-Alcoholic Steatohepatitis; CO: Coconut Oil; HDL: High-Density Lipoprotein; LDL: Low-Density Lipoprotein; VLDL: Very Low-Density Lipoprotein; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; TNF-α: Tumour Necrosis Factor

Introduction

Metabolic syndrome (MS), a disease associated with inadequate diet and sedentary lifestyle, has been considered a significant cause of morbidity in a globalized society [1]. MS corresponds to a group of alterations comprising obesity, visceral fat, hyperglycemia, hypertriglyceridemia, and hypertension. Other manifestations of MS include increased serum levels of total cholesterol, reduction of high-density lipoprotein (HDL), and augment of low-density lipoprotein (LDL). In addition to being correlated with a higher number of cardiovascular events, MS is one of the main risk factors for the development of non-alcoholic fatty liver disease (NAFLD) [2].

NAFLD includes a broad spectrum of hepatic abnormalities, which may range from steatosis to non-alcoholic steatohepatitis (NASH) [3]. In this context, NASH can be identified by the presence of significant fibrosis and necroinflammatory activity, in addition to steatosis. Balloon degeneration, Mallory hyaline corpuscles and nuclear vacuolation of hepatocytes can also be found in NASH [4]. One of the primary mechanisms involved in the development of NASH includes the association of hepatocellular lipid deposits, oxidative stress, and inflammation [5].

Coconut oil (CO) is extracted from coconut fruits (Cocos nucifera) and represents a font of saturated fat containing high levels of lauric acid, a medium chain fatty acid [6]. In recent years, health professionals and laypeople have disseminated uncertain information about CO consumption to control MS and NASH. Those who encourage CO supplementation argue that it provides weight loss and cardiovascular protective effects [7].

The purpose of this review is to verify the effectiveness and safety of CO in the control of MS and NASH, with special attention to the possible unfavourable impacts on human health, based on clinical and experimental studies.

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CO and MS

Concerning about MS, previously, the medium-chain saturated fatty acids, such as CO, has been associated with cardiovascular protection mainly by augmenting the HDL levels [8]. Lauric acid, a significant component of CO, has been associated with a decrease in the ratio of total cholesterol and HDL, suggesting that CO may result in a protective effect against the development of cardiovascular diseases [9]. However, the ability of CO to promote atherosclerosis through other pathways has also been questioned [8]. Some studies indicate that excessive consumption of coconut oil may be associated with the development of MS [6]. Moreover, recent studies associated diets containing CO with health damage, mainly due to cardiovascular complications related to the rising levels of LDL and VLDL and reduced HDL levels [10,11]. In addition, the role of CO in controlling obesity and hepatic steatosis, a hepatic manifestation of MS, remains controversial [12]. In a study was observed that a saturated fat diet containing CO increased the plasma triglyceride and free fatty acid levels in mice [13].

A single clinical trial that compared the consumption of extra-virgin coconut oil, butter and extra-virgin olive oil showed that low-density lipoprotein cholesterol (LDL) levels were significantly increased on butter, when compared with coconut oil and with olive oil, with no differences in change of LDL in coconut oil compared with olive oil. Moreover, coconut oil significantly increased high-density lipoprotein cholesterol (HDL) when compared with butter or olive oil. Furthermore, there were no significant differences in changes in weight, BMI, central adiposity, fasting blood glucose, systolic or diastolic blood pressure among any of the three intervention groups [14]. Nevertheless, the majority of randomized controlled trials show that the intake or supplementation of coconut oil increases an LDL and total cholesterol, besides to decreases an HDL when compared with other vegetable oils [6].

CO and NASH

The literature remains contradictory about diets using CO and hepatic steatosis. Some studies have reported the improvement of hepatic steatosis [15-17], while others have demonstrated the induction or augment of hepatic steatosis even without progression to NASH with the use of CO in the diet [13,18-20]. There is also a reference that the consumption of CO is incapable of promoting NASH [21].

A recent study showed that virgin coconut oil associated with a high-fat diet-induced to metabolic dysfunctions, adipose inflammation, and hepatic lipid accumulation, with increased in a hepatic cholesterol and triglycerides levels, besides of decreased in adiponectin and leptin levels, with an augmented in tumour necrosis factor (TNF)-α levels and an intense oxidative stress [22].

The damaging effects on the liver caused by CO consumption are because the fact that CO is a medium chain saturated fatty acid. This kind of fatty acid tends to slightly increase circulating insulin levels and promote lipogenesis due to insulin/glucagon imbalance. In addition, since medium chain, saturated fatty acids remain in free form after absorption, without significant incorporation into the transport lipoproteins [23]; they can be directly taken to the liver through the portal vein, promoting an imbalance in lipogenesis and hepatic lipid metabolism. These two mechanisms together make medium chain saturated fatty acids potent agents in the induction of hepatic steatosis, leading to oxidative stress and inflammation [24]. In addition, lauric acid increases LDL levels, since it plays a central role as a substrate for apolipoprotein (apo) A1 and apo B synthesis, which are the key molecules in LDL particles [6].

Another pathway that may be associated with hepatic steatosis is the involvement of the intestinal microbiota in the modulation of lipid metabolism. In detail, fat-rich diets have been linked to a differential impact of lipid sources in intestinal microbiota populations [25]. Recently, Patrone, et al. (2018) evaluated the effects of a high-fat diet with CO on the intestinal microbiota of mice. They observed that mice supplemented with CO showed a significant intestinal dysbiosis, which was correlated with increased cholesterol, triglycerides, weight gain, fatty tissue deposition and changes in leptin expression [26]. Therefore, different mechanisms, although correlated, may have participated in the lipid-metabolic changes that resulted in NASH observed in our study.

CO and Cardiovascular Effects

After an extensive review of clinical trials, observational and epidemiological studies on the cardiovascular impacts of CO consumption, Eyres, et al. (2016) concluded that CO could raise cholesterol including LDL and in some cases triglycerides levels, with values comparable to other saturated fats. Thus, the authors have preconized that CO should not be viewed as a heart-healthy food and should be limited in the diet [10]. After this review, the American Heart Association also conducted a study with controlled trials, wherein CO was found to stimulate LDL levels, being able to thick arterial walls resulting in damaging cardiovascular effects [11]. In accordance, the Brazilian Society of Endocrinology and Metabology and Brazilian Association for the Study of Obesity and Metabolic Syndrome stand against the therapeutic use of CO for weight loss, considering such conduct have no scientific evidence of efficacy and present potential risks to the health, mainly cardiovascular damages [27].

CO and Slimming

Even though there is controversy regarding the efficacy of CO as a slimming agent, the use of CO has

become widespread among people who want to lose weight. Moreover, CO effects of inducing weight loss remain controversial. There was described that the dietary supplementation with CO in humans promotes a reduction in abdominal obesity, but with a tendency to increase insulin resistance [28,29]. CO also potentiated the conjugated linoleic acid in lipolysis induction and lipogenesis diminution in mice [30], resulting in weight loss [31]. On the other hand, an observational study in 1981 reported that Pacific Island inhabitants in consuming higher amounts of CO were heavier and presented subscapular skin folds major [32]. Recently, there was showed that CO could not alter body weight, glucose metabolism, liver triglyceride levels and transaminase levels [12]. The current report indicates a lack of consistent evidence on the coconut oil intakes or supplementation, satiety, and weight loss. Given both the publicity and the increased consumption of coconut oil, further research, particularly long-term clinical trials, in this area are warranted [7,33].

Vyas, et al. (2012) have reported that the decrease in abdominal fat or only the non-increase in body weight observed with the use of saturated medium-chain fatty acid may be related not to fat elimination but to the relocation of fat to the visceral site, which was mistakenly believed has been eliminated [34]. According to this hypothesis, it's supposed that CO may thin the silhouette or lead to weight loss at the beginning of the diet, but over time, the fat cannot really be eliminated and remains deposited in some organs, such as liver, causing hepatic steatosis and ultimately NASH. Going further, the prolonged use of CO, first slimming effects can be reversed in weight gain. Even though there is controversy regarding the efficacy of CO as a slimming agent, the use of CO remains widespread among people who want to lose weight, without any knowledge about the adverse effects, as MS and NASH [10,11,22,33]. Also, up till now, studies have shown that CO consumption (intake or supplementation) does not enhance thermogenesis [6,35], as has been popularly triggered.

Conclusion

In conclusion, the current review indicates a lack of consistent evidence correlating CO intake or supplementation with induction of satiety and weight loss. But the most critical finding was that most studies showed the relationship between excessive CO consumption and consequences compatible with MS and NASH, in addition to producing deleterious cardiac effects, such as the development of atherosclerosis. Thus, the popular use of CO should be frankly discouraged. It must be considered that new clinical studies are indispensable to test the efficacy and safety of emerging diets, such as CO.

Conflict of Interest and Funding

The authors declare no competing financial interest.

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