

## Research Summary for Coconut fats

1: J Nutr. 2002 Mar;132(3):329-32. Related Articles, Links

**Physiological effects of medium-chain triglycerides: potential agents in the prevention of obesity.**

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**SUMMARY:** Medium chain fatty acids (MCFA) are readily oxidized in the liver. Animal and human studies have shown that the fast rate of oxidation of MCFA leads to greater energy expenditure (EE). Most animal studies have also demonstrated that the greater EE with MCFA relative to long-chain fatty acids (LCFA) results in less body weight gain and decreased size of fat depots after several months of consumption. Furthermore, both animal and human trials suggest a greater satiating effect of medium-chain triglycerides (MCT) compared with long-chain triglycerides (LCT). The aim of this review is to evaluate existing data describing the effects of MCT on EE and satiety and determine their potential efficacy as agents in the treatment of human obesity. Animal studies are summarized and human trials more systematically evaluated because the primary focus of this article is to examine the effects of MCT on human energy metabolism and satiety. Hormones including cholecystokinin, peptide YY, gastric inhibitory peptide, neurotensin and pancreatic polypeptide have been proposed to be involved in the mechanism by which MCT may induce satiety; however, the exact mechanisms have not been established. From the literature reviewed, we conclude that MCT increase energy expenditure, may result in faster satiety and facilitate weight control when included in the diet as a replacement for fats containing LCT.

2: Life Sci. 1998;62(14):1203-15. Links

**Medium chain fatty acid metabolism and energy expenditure: obesity treatment implications.**

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**SUMMARY:** Fatty acids undergo different metabolic fates depending on their chain length and degree of saturation. The purpose of this review is to examine the metabolic handling of medium chain fatty acids (MCFA) with specific reference to intermediary metabolism and postprandial and total energy expenditure. The metabolic discrimination between varying fatty acids begins in the GI tract, with MCFA being absorbed more efficiently than long chain fatty acids (LCFA). Subsequently, MCFA are transported in the portal blood directly to the liver, unlike LCFA which are incorporated into chylomicrons and transported through lymph. These structure based differences continue through the processes of fat utilization; MCFA enter the mitochondria independently of the carnitine transport system and undergo preferential oxidation. Variations in ketogenic and lipogenic capacity also exist. Such metabolic discrimination is supported by data in animals and humans showing increases in postprandial energy expenditure after short term feeding with MCFA. In long term MCFA feeding in animals, weight accretion has been attenuated. These differences in metabolic handling of MCFA versus LCFA are considered with the conclusion that MCFA hold potential as weight loss agents.

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3. Lipids 2009;44(7):593-601.

### Effects of dietary coconut oil on the biochemical and anthropometric profiles of women presenting abdominal obesity.

Abstract: The effects of dietary supplementation with coconut oil on the biochemical and anthropometric profiles of women presenting waist circumferences (WC) >88 cm (abdominal obesity) were investigated. The randomised, double-blind, clinical trial involved 40 women aged 20-40 years. Groups received daily dietary supplements comprising 30 mL of either soy bean oil (group S; n = 20) or coconut oil (group C; n = 20) over a 12-week period, during which all subjects were instructed to follow a balanced hypocaloric diet and to walk for 50 min per day. Data were collected 1 week before (T1) and 1 week after (T2) dietary intervention. Energy intake and amount of carbohydrate ingested by both groups diminished over the trial, whereas the consumption of protein and fibre increased and lipid ingestion remained unchanged. At T1 there were no differences in biochemical or anthropometric characteristics between the groups, whereas at T2 group C presented a higher level of HDL (48.7 +/- 2.4 vs. 45.00 +/- 5.6; P = 0.01) and a lower LDL:HDL ratio (2.41 +/- 0.8 vs. 3.1 +/- 0.8; P = 0.04). Reductions in BMI were observed in both groups at T2 (P < 0.05), but only group C exhibited a reduction in WC (P = 0.005). Group S presented an increase (P < 0.05) in total cholesterol, LDL and LDL:HDL ratio, whilst HDL diminished (P = 0.03). Such alterations were not observed in group C. It appears that dietetic supplementation with coconut oil does not cause dyslipidemia and seems to promote a reduction in abdominal obesity

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### COCONUT OIL: Atherogenic or Not?

(What therefore causes Atherosclerosis?)

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SUMMARY: According to the universally accepted Lipid-Heart Theory, high saturated fats cause hypercholesterolemia and coronary heart disease. Coronary morbidity and mortality are said to be highest in the countries and peoples consuming the highest amounts of saturated fats. Coconut oil, with its saturated medium chain fats, has been especially condemned for this reason. The true facts are just the opposite. The countries consuming the highest amounts of coconut oil – the Polynesians, Indonesians, Sri Lankans, Indians, Filipinos – have not only low serum cholesterol but also low coronary heart disease rates – morbidity and mortality. The reason why coconut oil cannot be atherogenic is basic. Coconut oil consists predominantly of 65% medium chain fatty acids (MCFAs) and MCFAs are metabolized rapidly in the liver to energy and do not participate in the biosynthesis and transport of cholesterol. Coconut oil, in fact, tends to raise the HDL and lower the LDL:HDL ratio. Coco oil is not deposited in adipose tissues and therefore does not lead to obesity. It is primarily an energy supplier and as fast a supplier of energy as sugar. MCFAs therefore differ in their metabolism from all the long chain fatty acids, whether saturated or unsaturated.

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The pathogenesis of atherosclerosis has recently taken a complete paradigm shift – from a simple deposition of cholesterol and cholesterol esters to an inflammatory condition where numerous genetically dependent factors – dyslipoproteinemias, dysfunctions of endothelial and other cells leading to invasions of the subendothelial region by macrophages, smooth muscle cells, leukocytes and T cells – all interplay in a scenario still not fully understood.

This will be discussed at length and whatever role fat deposition plays appears late in atherogenesis and secondary to oxidation process and the overriding role of the dysfunctional endothelium. Coconut oil has no role at all to play in this highly complex and still ill understood process.