# Evidence Paper

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# COCONUT OIL AND THE HEART

This paper summarises the evidence which forms the basis of the Heart Foundation's position on coconut oil and heart health

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# Introduction

Coconut flesh and coconut milk or cream have traditionally been used in a range of food cultures. They are increasingly used in Western countries as ethnic dishes have become more prominent. More recently, coconut oil has been heavily marketed as an extremely healthy oil. This is in contrast to traditional dietary advice which recommends limiting coconut intake due to its high saturated fat content (92% saturated fat).

It is timely for the Heart Foundation to review the evidence for coconut and extracted oils (including hydrogenated coconut oil) since the recent marketing push mentioned above in both the USA and Australasia has resulted in people substituting coconut oil for the currently recommended unsaturated plant oils.

## Aim of the review

The aim of this evidence paper is to summarise the literature on coconut flesh, coconut cream and milk, and coconut oil and their impact on heart health; and subsequently to provide recommendations on their use as part of a Western style dietary pattern.

This paper will:

- discuss the composition of coconut and its products
- define medium chain fatty acids and medium chain triglycerides
- show the compositional differences between coconut oil and medium chain triglycerides (MCTs)
- summarise the scientific papers on coconut and coconut products from clinical trials in humans and population-based studies, in the context of evidence-based statement on fats by the FAO and WHO.

**Composition of coconut and products included in this review**

Coconut products included in the review were refined, bleached and deodorised (RBD) coconut oil, virgin coconut oil (VCO), hydrogenated coconut oil (HCNO), coconut flakes/flesh and coconut cream. Coconut water was not included as it has a very different nutrient profile and is not relevant to the issue of saturated fat and coronary heart disease. Coconut flour is a relatively recent niche product that is low in fat but high in dietary fibre. It is being used as an interesting culinary ingredient for dairy-free and gluten-free diets, but is not a major topic for this review due to the lack of scientific papers covering this ingredient. Clinical reports of its effect on glycaemic index may be of relevance when evaluating the effects of consuming whole coconut without separating the oil (Trinidad et al., 2004; Trinidad et al., 2003). The composition of coconut and its products containing fat are shown in Table 1.

**Table 1. Composition of Coconut Products**

| Per 100g product | Water (g) | Energy (kJ) | Protein (g) | Fat (g) | Sat Fat (g) | CHO (g) | Fibre (g) |
|---|---|---|---|---|---|---|---|
| Raw coconut flesh | 45 | 1470 | 3.2 | 36 | 33 | 3.6 | 7.7 |
| Coconut desiccated | 2 | 2530 | 5.6 | 62 | 58 | 6.1 | 19.2 |
| Coconut cream (canned) | 71 | 858 | 1 | 20 | 19 | 3.7 | 0.6 |
| Coconut oil (EV or RBD) | 0 | 3700 | 0 | 100 | 92 | 0 | 0 |
| Hydrogenated coconut oil (CF 92) | 0 | 3700 | 0 | 100 | 100 | 0 | 0 |
| Coconut water | 92 | 89 | 0.3 | 0 | 0 | 5 | 0 |
| Coconut flour | 2 | 1854 | 18 | 15 | 14 | 21 | 38 |

Refer to the Glossary for product definitions and descriptions.

## Preparation and use of coconut and coconut products

Coconut oil is largely used for edible purposes, although it is a key component of quality soaps. It is prepared domestically by heating coconut milk until clear oil separates. If done under rigorous conditions, this can be sold as VCO.

The commercial extraction of oil from dried coconut flesh, or copra as it is known, is one of the oldest seed crushing industries in the world. Harvested coconuts are dehusked and split and the resulting flesh is air or kiln dried down to low moisture levels to produce copra. Copra is not consumed as a foodstuff but is further processed to produce coconut oil. Copra processing methods range from simple village processes to modern high-pressure expellers and prepress or solvent extraction plants. Throughput can be more than 500 tonne of copra per day.

Crude coconut oil, as it is ordinarily prepared by hot pressing in tropical countries, is a colourless to pale brownish yellow oil. In temperate climates, or air conditioning, it appears as a greasy somewhat white or yellowish solid fat that has a melting point range between 20° and 26°C. Until refined it has a pronounced odour of coconut due to the high acidity and oxidation that occurs during drying and storage. Coconut oil is often then refined, bleached and deodorised using standard vegetable oil processing technology. This produces a colourless, stable and bland oil, low in acidity that is used for many purposes in the food industry. Refined and deodorised coconut oil has a relatively low smoke point of 170°C compared to commonly used frying fats such as palm and tallow which have smoke points over 230°C (Man & Hussin, 1998).

Generally the smoke point is inversely proportional to the free fatty acid level or acid value, but coconut smokes at a lower temperature due to the short chain fatty acids in the triglyceride molecule. The high level of saturated fatty acids and low level of unsaturated fatty acids means that the oil is highly

resistant to oxidation and polymerisation unlike unsaturated oils. Highly polyunsaturated vegetable oils are easily oxidised especially on frying and this is one area where in small amounts coconut oil, with its high stability and resistance to oxidation, has a small advantage. So coconut oil is acceptable for one use shallow pan frying but is not really suitable for continuous deep fat frying (Srivastava et al., 2010).

Virgin coconut oil is prepared from fresh coconuts without drying and is claimed to contain natural antioxidants such as polyphenols in concentrations similar to olive oil (Marina, Che Man, & Amin, 2009). No human clinical trial reports were found on VCO where the effects of the natural antioxidants could be evaluated. Extraction methods include cold-pressing and aqueous, centrifugal extraction using the same process as olive oil. Virgin coconut oil, if produced to a quality standard, has a low acidity, pale colour and is mild and pleasant in flavour. It is growing in popularity and consumer demand.

Hydrogenated coconut oil (also known as confectionery fat 92) is used as a stable, higher melting point fat in biscuit fillings and confectionery. It does not contain any trans fatty acids, contrary to several reports in the literature as it comprises approximately 100% saturated fatty acids which are produced by the conversion of the 7% unsaturated fatty acids in coconut oil into saturated fatty acids (stearic acid).

## Fats and fatty acids in coconut

The major component of fats is triglycerides. Triglycerides are molecules made up of a glycerol backbone with three fatty acids attached. The triglycerides in coconut oil contain a mixture of the fatty acids shown in Table 2.

**Table 2. Major Fatty Acids in Coconut Oil (descending order)**

| Fatty Acid | Name | Percentage |
|---|---|---|
| C12:0 | Lauric | 45 – 48% |
| C14:0 | Myristic | 14 – 18% |
| C16:0 | Palmitic | 7.5 - 9.5% |
| C18:1 | Oleic | 6 - 8.2% |
| C8:0 | Caprylic | 7.8% |
| C10:0 | Capric | 7.6% |
| C18:0 | Stearic | 5.0% |
| C18:2 | Linoleic | 1 –2% |

Depending on their length, fatty acids are termed short, medium or long chain *fatty acids*. Saturated fatty acids with C4 and C6 carbon chain lengths are short chain fatty acids. Carbon chain lengths of C8 and C10 are medium-chain fatty acids, although some scientists have included lauric acid (C12) in this classification. Long chain fatty acids have traditionally included C12 up to C18 carbon chain length. Very long chain fatty acids are C20 to C24.

As well as the fatty acids themselves being termed short, medium or long chain, when they are combined into a triglyceride they are also termed short, medium or long chain *triglycerides*.

The technical definitions surrounding medium-chain fatty acids (MCFAs) and resultant medium chain triglycerides (MCTs) can be confusing to the lay person, and they are also frequently misinterpreted by researchers. Much of the evidence used to support the health benefits of coconut comes from historical research on MCTs. There are three issues with this assumption which will be addressed:

- whether lauric acid (the main fatty acid in coconut) is a MCFA;
- whether the triglycerides in coconut are MCTs; and
- whether research on MCT oils can be extrapolated to coconut as a food.

## Is lauric acid a medium chain fatty acid (MCFA)?

Lauric acid (C12:0) is the main type of fatty acid in coconut oil. It is frequently classified as a MCFA. In chemical terminology it can be described as either a medium-chain or a long-chain fatty acid; however it behaves more like a long-chain fatty acid in biological systems.

MCFAs defined as C8:0 and C10:0 (not including C12:0) behave differently from long chain fatty acids (LCFA) in every aspect of metabolism from absorption to catabolism. By virtue of their smaller molecular size, MCFAs are relatively soluble in water. The water solubility at 20°C is 68mg/100 ml for C8:0 versus 0.72mg/100ml for C12:0. They are more water soluble than LCFAs and are solubilised in the aqueous phase of the intestines without forming micelles, therefore undergoing faster absorption than LCFA. The fact that MCFAs are weak electrolytes and are highly ionised at neutral pH further increases their solubility in biological fluids. The marked difference in solubility occurs at chain length >10, thus lauric acid (C12:0) behaves more like a LCFA.

**Are the triglycerides in coconut medium chain triglycerides (MCTs)?**

Whilst lauric acid is often misleadingly termed a MCFA, when it is in the form of a natural fat it is combined with a random distribution of all the other fatty acids in coconut oil. These include myristic and palmitic fatty acids which are the chief saturated fatty acids implicated in cholesterol elevation (FAO, 2010).

Coconut is similar in composition to palm kernel oil and in fact the two oils are almost interchangeable in the food industry. They are both termed “tropical oils” or classified as the “lauric” oils due to the predominance of this fatty acid in their compositions.

When lauric fatty acids are combined with glycerol to make triglycerides (the components of fats and oils), the resulting triglycerides are of higher molecular weight and differing metabolism to the lower molecular weight triglycerides of C8 and C10 which have been well characterised in metabolic studies. The accepted definition of medium chain triglycerides in nutritional and clinical papers applies only to these synthesised esters of the C8 and C10 fatty acids. Thus, the triglycerides in coconut cannot be classed as MCTs.

## Can research on MCTs be extrapolated to coconut oil?

Medium chain triglycerides were introduced into clinical nutrition in the 1950s for the dietary treatment of malabsorption syndromes because of their rapid absorption and solubility. A recent review by Kasai et al. (2006) examining their effect on body weight confirms the definition of MCTs as being composed of C8 and C10 fatty acid containing triglycerides.

Medium chain triglycerides are used for nutritional and other commercial purposes, and are derived from tropical oils such as coconut and palm kernel oils. In the process of producing MCTs, these oils are hydrolysed to fatty acids and glycerol. The glycerol is drawn off from the resultant mixture, and the medium-chain fatty acids are fractionally distilled, separating them from the major component lauric acid. The MCFA fraction used commercially is mainly comprised of the 8 carbon caprylic or octanoic acid and the 10 carbon capric or decanoic acid. There are much smaller amounts of the six carbon caproic or hexanoic acid and the 12 carbon lauric acid in the commercial products.

## MCTs and their absorption

When MCTs are digested they are split by pancreatic lipase in the gut to produce fatty acids and a diglyceride.  In the case of mixed triglycerides the C8:0 and C10:0 are liberated preferentially. Marten, Pfeuffer, and Schrezenmeir (2006) showed that in humans MCTs did not produce any change in pancreatic secretion, whereas with LCTs there was a significant overall increase.

The molecular weight of MCTs is smaller than the molecular weight of long-chain triglycerides (LCTs). This facilitates the action of pancreatic lipase. Consequently, MCTs are hydrolysed both faster and more completely than LCTs. The products of MCT hydrolysis are absorbed faster than those of long chain triglycerides and as fast as glucose (Bach & Babayan, 1982).

Subsequent to absorption, MCFA are secreted directly into the portal circulation without undergoing re-esterification in the enterocyte. Due to their

water solubility, MCFA do not require albumin for transportation from capillaries to peripheral tissues. Similar to LCFA, MCFA also undergo oxidation in mitochondria but do not require carnitine for mitochondrial transport. All of these factors result in rapid metabolism of MCFA from ingested MCT and form the basis for their use in clinical nutrition and parenteral nutrition as well as their proposed emerging role in weight management.

Despite many positive claims about the neutral effects of pure MCTs on lipid levels, Cater, Heller, and Denke (1997) reported the converse effect. They found that although MCTs (composed of medium-chain fatty acids 8:0 and 10:0) have long been described as having neutral effects on serum cholesterol concentrations, experimental evidence supporting this claim is limited. In a randomised, crossover, metabolic-ward study, they compared the lipid effects of a natural food diet supplemented with either MCTs, palm oil, or high oleic acid sunflower oil in nine middle-aged men with mild hypercholesterolemia. Rather than having a neutral effect, MCT oil produced total cholesterol concentrations that were not significantly different from those produced by palm oil (MCT oil: 5.87 +/- 0.75 mmol/L; palm oil: 5.79 +/- 0.72 mmol/L) but significantly higher than that produced by high oleic acid sunflower oil (5.22 +/- 0.52 mmol/L). Low-density-lipoprotein (LDL)-cholesterol concentrations paralleled those of total cholesterol. MCT oil tended to result in higher triglyceride concentrations than either palm oil or high oleic acid sunflower oil, but this difference was not significant. There were no differences in high-density-lipoprotein cholesterol concentrations. The palmitic acid and total saturated fatty acid content of plasma triglycerides in the MCT oil diet was not significantly different from that in the palm oil diet. On the basis of percentage of energy, this reference suggests that medium-chain fatty acids have one-half the potency of palmitic acid at raising total and LDL-cholesterol.

## Comparison of coconut oil to medium-chain triglycerides

**Figure 1: MCT oil and virgin coconut oil at ambient temperature in NZ**


The photograph in Figure 1 illustrates a key difference between coconut oil and MCTs. True MCTs are liquid at room temperature so show a marked differentiation with coconut oil which is solid at room temperature, like all other saturated fats. Coconut oil contains some MCFA, but it does not contain significant MCTs by definition. MCTs have a total carbon number of C24:0 to C30:0, whereas only around 4% of triglycerides in coconut are of this length. Table 3 shows that whilst MCT oil (as manufactured and used in specialist applications) is 95% MCT, coconut oil by analysis only contains up to 4%. Thus, coconut oil is not composed of MCTs.

**Table 3: Comparison of the properties of coconut oil, medium-chain triglycerides and butterfat**

| Fatty acids | Coconut oil | Medium chain triglycerides | Butterfat |
|---|---|---|---|
| Butyric 4:0 | 0 | 0 | 4.3 |
| Caproic 6:0 | 1 | <2 | 2.3 |
| Caprylic 8:0 | 9 | 50-80 | 1.4 |
| Capric 10:0 | 7 | 20-50 | 2.8 |
| Lauric 12:0 | 47 | <3 | 3.1 |
| Myristic 14:0 | 16.5 | <1 | 9 |
| Palmitic 16:0 | 7.5 | 0 | 22 |
| Stearic 18:0 | 3 | 0 | 15 |
| Oleic 18:1 cis | 6.4 | 0 | 26 |
| Elaidic 18:1 trans | 0 | 0 | 5 |

| Fatty acids | Coconut oil | Medium chain triglycerides | Butterfat |
|---|---|---|---|
| Linoleic 18:2 | 1.5 | 0 | 1.9 |
| Total saturated | 92 | 100 | 60 |
| Triglycerides carbon number range* | Range C28-C52 | Range C24-C32 | Range C28-C54 |
| C24-C30 | <4% | 95% | <1% |
| Mean molecular weight | 638 | 512 (low) | 690 |
| Physical characteristics | Solid at ambient | Liquid at all temperatures | Solid at ambient |

The composition of coconut oil is substantively different to that of MCTs. Coconut oil therefore needs to be judged on its own merits in rigorous human studies. Studies that extrapolate an effect of coconut based on research in MCTs were therefore not counted as evidence for this review. Several papers consulted (see references) in the production of this review refer to coconut oil as being composed predominantly of MCTs and extrapolate conclusions based on clinical work done on these synthesised products over many years. This is a common theme running through a great deal of the published work and on well publicised websites.

## Saturated fat

Coconut oil is composed of 92% saturated fatty acids combined as triglycerides. Intake of saturated fat has long been held as one of the major risk factors in coronary heart disease. The general stance on saturated fat and heart health and recent data shows replacement of SFA with PUFA reduces CHD risk (Micha & Mozaffarian, 2010). A full review of the impact of saturated fat on heart disease will be available in the Heart Foundation's fats and fatty acids position paper.

In summary, based on the expert report of the FAO and WHO (FAO, 2010), there is convincing evidence that:

- Replacing SFA (C12:0–C16:0) with polyunsaturated fatty acids (PUFA) decreases LDL cholesterol concentration and the total/HDL cholesterol ratio.
- A similar but lesser effect is achieved by replacing these SFA with monounsaturated fatty acids (MUFA).

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* This is the sum of the number of carbons on the three fatty acid chains which make up the triglyceride molecule

Based on coronary heart disease (CHD) morbidity and mortality data from epidemiological studies and controlled clinical trials (using CHD events and death):

- There is **convincing** evidence that replacing SFA with PUFA decreases the risk of CHD.
- There is a possible positive relationship between SFA intake and increased risk of diabetes.

A recent review on the effects of type of dietary fat and chronic disease risk found that there was *convincing* evidence that partial replacement of SFA with PUFA decreases the risk of CVD, especially in men. This finding was supported by an association with biomarkers of PUFA intake; the evidence of a beneficial effect of dietary total PUFA, n-6 PUFA, and linoleic acid (LA) on CVD mortality was *limited suggestive* (Schwab et al., 2014).

However, one of the main arguments proposed by coconut oil proponents is that the saturated fats in coconut oil behave atypically or that constituents in the whole food prevent any negative effects.

Individual saturated fatty acids (SFA) have different effects on the concentration of plasma lipoprotein cholesterol fractions. For example, lauric (C12:0), myristic (C14:0) and palmitic (C16:0) acids increase LDL cholesterol whereas stearic (C18:0) has no effect (FAO, 2010). Katan, Zock, and Mensink (1994) also reviewed the effects of fats and fatty acids in mixed diets. Their work is similar to that reported below, where myristic has the highest cholesterol raising property followed by palmitic and lauric, with stearic acid appearing to be neutral.

Lauric (C12), myristic (C14) and palmitic (C16) acids have generally been regarded as the three cholesterol raising fatty acids and the major plasma lipoprotein fraction affected is LDL. Palmitic is quantitatively the most significant since it is the principal saturated fatty acid in most diets occurring widely in meat products and plant oils.

In a carefully controlled metabolic ward study, Denke and Grundy (1992) compared liquid formula diets rich in lauric, palmitic, and oleic acids. Both lauric and palmitic acids were associated with higher levels of total and LDL cholesterol when compared with oleic acid but the rise in LDL cholesterol on the high lauric acid diet was about two thirds of that on the high palmitic acid diet.

A meta-analysis on 60 selected trials by Mensink, Zock, Kester, and Katan (2003) and currently being updated, found that lauric acid greatly increased total cholesterol but that the ratio of total to HDL cholesterol was decreased. They called for further research to assess the effects of these fats on actual

disease outcomes. They also found that the TC:HDL ratio was more favourably affected by replacing SFA with cis-unsaturated fatty acids.Their conclusions as to the TC:HDL cholesterol ratio was that whilst it could not replace outcomes based on disease or mortality, it was a more sensitive predictor than the levels of individual cholesterol types viewed singularly. They also concluded that whilst coconut oil had a favourable effect on the ratio compared to butter or carbohydrates, it did not exclude the possibility that coconut oil may promote cardiovascular disease through other pathways as yet unknown. Although no conclusions relating to unsaturated oils as a comparator were made they reported that all biomarkers, including lipid levels were no substitute for studies that employ disease or mortality as outcomes. The inhibition of HDL activity by saturated fat as proposed by Nicholls (2006) may fit with alternative but unexplained mechanisms involving endothelial function.

There is vigorous debate around lauric acid's effect on raising HDL-cholesterol – the assumed strength may actually be a weakness. The general acceptance is that HDL-cholesterol is protective against heart disease yet recent research is showing that the reality is more complex.

As discussed in a recent review on HDL functionality and inflammation (Nicholls et al., 2006), the question about HDL is not simply how much is present but also how effective the HDL is. Lauric acid and the other longer-chain saturated fatty acids all raise HDL levels but is it giving rise to 'good' HDL that lowers heart disease risk or non-functional HDL that does nothing? This study conducted in Sydney was the first to show that the quality of HDL could be changed by the food we eat. It showed that feeding saturated fat impaired the anti-inflammatory effects of HDL i.e. it lowered HDL functionality.

## Evidence review

This paper reviews evidence for coconut products and cardiovascular disease (or its risk factors) from intervention and epidemiological studies in humans.

Studies and articles were excluded from this review if they were letters, conference abstracts, opinion pieces, non-systematic reviews or books. Articles were further excluded if outcomes were not relevant to cardiovascular disease; research related to medium-chain triglyceride oils (rationale is provided above); were animal or ex vivo studies; related to coconut water; did not relate to edible coconut or coconut products; related to the history of coconut production or use; were not related to consumption of coconut oil, coconut milk or coconut cream; coconut or a coconut product was not the main intervention or focus of analysis; coconut was part of a mixed intervention and it was not possible to determine its individual impact; the study was in a specialist clinical population e.g. patients with liver cirrhosis; it related to consumption in infant formula; outcomes such as lipid profiles were a minor part of the experimental results and were not a major objective of the

study; it was also a drug trial; or the study investigated the effect of lauric acid or its derivatives rather than coconut.

Studies and reviews that promote coconut oil as having a beneficial or neutral effect on heart health were all found to fall under the above criteria.

Searches were conducted in the Scopus and Medline databases and bibliographies in published literature and on websites promoting coconut oil were examined. The few papers and studies (n=23) identified for inclusion in this review demonstrates the paucity of quality studies, reviews or meta-analyses that examine the effects of coconut in its own right. Even fewer have studied the effects of consumption of coconut or coconut products on cardiovascular disease outcomes.

## Reviews and meta-analyses

None identified that met inclusion criteria.

## Clinical trials and intervention studies

Only (n=10) clinical trials on human subjects were identified that met our criteria. The strongest evidence is obtained from randomised controlled trials (RCTs) as these types of study minimise bias and can demonstrate cause and effect.

### Coconut and serum lipids

Cox, Mann, Sutherland, Chisholm, and Skeaff (1995) conducted a randomised crossover trial to assess the effects of coconut oil, butter and safflower oil on lipids and lipoproteins on moderately hypercholesterolemic individuals. Twenty eight participants (13 men, 15 women) followed three 6-week experimental diets of similar macronutrient distribution with the three different test fats providing 50% total dietary fat. Fat as a percentage of energy was 35-37%. All three groups received intervention. Subjects followed each experimental diet for six weeks. They found that both butter and coconut oil raised total cholesterol and LDL significantly more than safflower oil and that butter raised both outcome measurements the most ($p<0.001$). There was no statistically significant difference in HDL between groups. There was no significant difference for males and females in serum triglyceride levels.

Cox et al. (1998) carried out a sequential feeding experiment on 41 healthy Pacific people to assess the effects of coconut oil, butter, and safflower oil on lipids and lipoproteins. The subjects were fed each of the three test diets for six weeks. They confirmed the findings of Cox et al (1995) in that total cholesterol (TC) and LDL levels were highest on the butter diet and lowest on the safflower diet, with coconut in between the two ($p<0.01$). Plasma triglyceride levels were reduced from 1.98 mmol/L in all three test groups with the coconut oil group showing the lowest concentration at 1.61 mmol/L compared with the safflower oil at 1.77 mmol/L, however the differences were not statistically significant.

Reiser et al. (1985) conducted a randomised crossover trial with medical students that assessed the effects of coconut oil, safflower oil, and beef fat on fasting plasma lipid and lipoproteins. Nineteen male subjects consumed two or three of the diets for five weeks with their normal diet in between. The test fat provided 60% of energy from fat, with total fats providing 35% of energy. The coconut oil diet resulted in higher total cholesterol, HDL, and LDL than beef fat or safflower oil. Triglycerides were lowered by coconut when compared to beef fat and were not altered significantly when compared to safflower oil.

A randomised feeding experiment with 60 Sri Lankans over a 62-week period assessed the effects of lowering coconut and saturated fat intake compared with partial replacement of saturated fat from coconut with polyunsaturated fat from soybean and sesame oil (Mendis, Samarajeewa, & Thattil, 2001). Both intervention diets were low-fat (20% total energy versus 24% total energy). It was found that reducing the intake or replacing a portion of the saturated fat in the diet of Sri Lankans with unsaturated fat resulted in improved serum lipoprotein profiles compared to baseline. There was a 24-27% decrease in TC:HDL ratio ($p<0.002$) at 12 months compared with baseline. The only adverse effect was a small increase (8.2%) in triglycerides in the group supplemented with soybean and sesame oil. However, the group that showed this had some of the total fat replaced by carbohydrate. An earlier paper detailing the effects of replacing coconut fat with some soybean to increase the PUFA:SFA ratio reported a beneficial effect on triglyceride levels (Mendis & Kumarasunderam, 1990).

Fisher, Blum, Zannis, and Breslow (1983) compared corn oil with coconut oil in mixed diets on 9 male participants. Test fats contributed 31% total energy and test diets were administered for 9 days. Coconut oil was found to significantly elevate TC, VLDL, LDL, HDL and triglycerides.

In contrast to the findings on coconut oil, a single feeding trial involving coconut flour seems to indicate potential beneficial effects on cardiovascular disease risk factors. Trinidad et al. (2004) conducted a double-blind randomised crossover feeding trial with 21 human participants with moderately raised serum cholesterol to assess the cholesterol lowering effects of coconut flour. The experimental feeding periods were two weeks long with two breaks between different test foods. They compared corn flakes with oat bran flakes and corn flakes with added coconut flour. Coconut flesh reduced total serum cholesterol, LDL, and serum TG compared to controls. The authors conclude that this effect was due to the fibre content of the coconut product used.

### Coconut and waist circumference/obesity

Obesity is an increasingly prevalent metabolic disorder affecting all Western type populations and is of particular importance in New Zealand. Diets high in energy have been identified as a key problem in obesity onset leading to cardiovascular disease. There have been several reports in the literature

claiming that the use of MCT oil is beneficial in reducing weight in iso-caloric diets (Kasai et al 2006). This has led to an interest in the potential impact of coconut on body weight.

Liau, Lee, Chen, and Rasool (2011) conducted a four-week open-label uncontrolled pilot study with 20 Malay men and women who had a BMI of 23 or greater, to investigate the potential impact of 30ml per day virgin coconut oil on body weight. Overall, there was a statistically significant reduction in waist circumference of 2.87cm (95% CI 0.55, 5.18) from baseline. However, when this was broken down by gender, there was only a statistically significant reduction in waist circumference in men. No other measures of body weight, lipid, renal or liver function reached statistical significance. Changes in diet during the study were not assessed. The author concluded that extra virgin coconut oil with its relatively high concentration of C8 and C10 fatty acids would be oxidised in the body more readily than other fats and not be stored in adipose tissue.

### Inflammation and oxidative stress biomarkers

Oxidative stress has been recognised as an important trigger in the development of cardiovascular disease. A paper by Sabitha, Vaidyanathan, Vasudevan, and Kamath (2009) describes a form of case-control study conducted with middle-aged Indian men with and without type 2 diabetes who self-reported typically consuming coconut oil or sunflower oil as 13 to 20% of their total energy intake. Lipid profiles and oxidative stress parameters were compared between the groups and did not show any statistically significant differences between coconut oil and sunflower oil.

Homocysteine and other inflammatory markers have been suggested as key biomarkers for cardiovascular disease. A high level of homocysteine makes a person more prone to endothelial injury which leads to vascular inflammation which in turn may lead to atherogenesis, which can result in ischaemic injury. Whilst detection of homocysteine may be linked to cardiovascular disease, lowering homocysteine levels may not improve outcomes. The paper by Voon, Ng, Lee, and Nesaretnam (2011) with 45 healthy young Malaysian adults examined a range of biomarkers including homocysteine and postprandial lipid levels after consuming a meal with different fat compositions. The diets were moderate in protein (20% of energy) with the test fats (palm, coconut and olive oil) providing 67% of total fat which in turn was 30% of energy. The levels of homocysteine and other inflammatory biomarkers such as C-reactive protein were not significantly different in all three test fats. Lipid profiles showed that the coconut oil group had the highest total cholesterol and LDL cholesterol levels. TC: HDL ratios were not significantly different among the three test diets.

## Epidemiological evidence/population studies

Indigenous populations who consume significant amounts of coconut flesh and milk include those of India, Sri Lanka, Philippines, Polynesia and Melanesia. Their health statistics are often quoted as evidence that consuming coconut oil poses no risk from cardiovascular disease. However, observational studies in these population groups cannot show causation and are prone to confounding, because many different factors can simultaneously affect a specific health outcome or indicator. Furthermore, cross-sectional studies cannot show a temporal sequence because measures are only taken at one point in time. They are highly prone to recall bias and reverse causation. Observational studies also suffer from inherent limitations in dietary assessment methods, with a strong bias towards underestimation of habitual energy intake. There were (n=13) observational studies identified and discussed below.

The Pukapuka and Tokelau island study by Prior, Davidson, Salmond, and Czochanska (1981) found a low incidence of cardiovascular disease in the populations of these two islands despite a large portion of their energy intake and dietary fat intake being from coconut flesh. It has been reported that the diets of these two populations were low in sugar and high in fibre rich foods resulting in low cholesterol levels (4.5mmol/L and 4.6mmol/L). It is interesting to note that most indigenous populations have consumed either coconut flesh or squeezed coconut cream. The extraction and use of coconut oil in edible applications is a relatively recent phenomenon. Also, since that time a large move towards the Western diet has occurred with imports of unhealthy foods such as corned beef, fast food and processed ingredients leading to a huge increase in obesity and poor health.

A series of studies were conducted on Tokelauans who had migrated to and lived in New Zealand for approximately seven years. Lipid profiles and diets for 1,200 NZ residents were compared with 800 people still living in the Islands. For the males studied, along with changes in diet and lifestyle, their plasma total cholesterol concentration increased by around 0.4 mmol/L, their LDL cholesterol by 0.4 mmol/L, their HDL reduced by 0.06 and their TC:HDL increased by 0.5 to around 4.4 mmol/L. (Stanhope, Sampson, & Prior, 1981). For the people on Tokelau 50% of their energy intake was from fat predominantly coconut either as grated coconut flesh or as coconut cream. Their diet was coconut, breadfruit and fish. Coconut oil was not consumed per se. For the migrants who moved to NZ, their whole diet changed to consuming a lot more dairy products, meat and sugar and less fish. This study reported 40 years ago, is often cited as showing that coconut oil is a healthy choice. The total changes in diet and lifestyle, together with the inconclusive results would make such a conclusion invalid.

The Kitava studies (S Lindeberg, Berntorp, Nilsson-Ehle, Terént, & Vessby, 1997; S. Lindeberg & Lundh, 1993; S. Lindeberg, Nilsson-Ehle, & Vessby, 1996) examined cross-sectional age relations of cardiovascular risk factors of

203 Melanesian people between the ages of 20 and 86 in Papua New Guinea. Coconut is a staple food of the Kitavans and they have a very low incidence of cardiovascular disease. Their fat intake (mainly from coconut) as a percentage of energy was low at 21%. Their diet consisted of whole coconut, tubers, fish and fruit. Their average total cholesterol was reported as 4.7 mmol/L with LDL cholesterol being 3.1 mmol/L. Life expectancy in this study was most highly correlated with BMI, the mean of which for those studied was 20.

Contrary to the findings of the Kitava, Pukapuka and Tokelau studies are studies of the Sri Lankan population which also has coconut as the primary source of their dietary fat intake. Counter to what has been found in Pacific Island populations; there is a relatively high incidence of coronary heart disease in Sri Lanka (Mendis et al., 2001).

It has been hypothesised that the differences observed between these populations are due to what constitutes the rest of their traditional diets (Dibello 2009). The majority of the Pacific Island populations, such as Tokelau and Pukapuka Island, traditionally ate no processed foods, had a diet high in fruit and vegetables, with the main protein source being fish**.** The original participants of the Kitava study also had low BMI and an active lifestyle with no major influences from a Western diet.

A cross-sectional study of 723 American Samoans and 785 Samoans living in the Samoan islands compared people consuming a traditional diet which included coconut products, with a Western style diet and related diet type to the incidence of metabolic syndrome. It was found that the Western diet was associated with an increased incidence of metabolic syndrome and therefore an increased risk of developing cardiovascular disease. These authors did not measure total cholesterol or LDL cholesterol but looked at a variety of measurements such as BMI and blood pressure. They concluded that the results of this study provide evidence for the potential protective effect of a neo-traditional eating pattern in American Samoa and Samoa. This dietary profile was characterised by a high intake of coconut products and seafood and low intake of processed foods, including potato chips, rice, and soft drinks. On the other hand, a more Western dietary pattern was positively associated with the presence of metabolic syndrome (DiBello et al., 2009).

Coconut consumption is associated with higher levels of serum HDL in epidemiological studies and claims of coconut being beneficial to heart health have been attributed to this effect. An analysis of the results of a longitudinal cohort study of 1,839 Filipino women aged 35-69 by Feranil, Duazo, Kuzawa, and Adair (2011) reveals that while HDL levels did indeed rise with an increase in coconut oil intake, total cholesterol and LDL-C also rose. The serum triglyceride levels of all the participants rose with increasing coconut oil intake. The ratio of TC:HDL was unaffected by coconut intake. These results do not indicate either a beneficial nor detrimental effect on serum lipid profiles when

viewed as a whole. The impact on sub- fractions of LDL and HDL has not been investigated.

There are other population studies from Africa looking at obesity and dyslipidemia, (Njelekela et al., 2002; Njelekela et al., 2003), from Indonesia and India looking at coronary heart disease (Kumar, 1997; Lipoeto, Agus, Oenzil, Wahlqvist, & Wattanapenpaiboon, 2004) and India (Beegom & Singh, 1997) which examined hypertension. It is not clear from these studies whether coconut in the diet has any positive or negative effects on cardiovascular disease and its risk factors due to confounding factors between study groups.

## Summary

In summary, while the level of evidence on coconut itself and risk factors for heart disease is mostly poor quality, the evidence suggests that consumption of coconut oil raises total cholesterol, HDL and LDL, although in clinical trials this did not raise them as much as butter.

In the clinical trials included in this review, the effects of coconut oil on triglyceride levels versus unsaturated oils were generally not significant.

As many of the studies are older, the impact on the ratio of total cholesterol to HDL cholesterol was often not reported. No convincing evidence was discovered during the literature search that demonstrated that consumption of coconut oil as opposed to unsaturated oils led to improved lipid profiles and a decreased risk of CHD.

The evidence that coconut oil is super-healthful is not convincing and these claims appear to be more testimonials than clinical evidence.

There is very limited evidence on disease outcomes. Neither the American Heart Association (AHA) (American Heart Association, 2013) nor the U.S. Government's 2010 Dietary Guidelines (USDA. & HHS., 2010) suggest that coconut oil is any better or preferable over other saturated fats. Coconut oil, like all saturated fats, should be limited to 7%-10% of calories because it can increase the risk for heart disease, according to the AHA and 2010 Dietary Guidelines.

The studies reviewed have all indicated that the consumption of coconut products such as flesh and coconut milk which contain fibre, together with sufficient polyunsaturated fats (particularly from fish) and in the absence of excessive calories from carbohydrates, may not pose a risk for heart disease in traditional diets.

When compared to other fat sources, coconut oil does not raise total cholesterol (TC) and LDL cholesterol (LDL-C) to the same extent as butter, but does increase TC and LDL-C levels to a greater extent than vegetable oils and is comparable to palm olein oil.

# Recommendations

Indigenous populations who consume traditional diets with coconut flesh and milk along with fish and vegetables (unsaturated fats and fibre) combined with a physically active lifestyle are unlikely to be at risk of cardiovascular disease from the consumption of coconut products. The situation for indigenous populations who eat a traditional diet is vastly different to that of people consuming a typical "Western" style diet.

For other populations, coconut oil is 92% saturated and nothing in the literature disputes the fact that it acts as a saturated fat and raises total cholesterol, LDL cholesterol and HDL cholesterol.

Coconut oil does not behave the same as MCT's and it is totally erroneous and scientifically wrong to call coconut oil an MCT and thus any analogies comparing coconut oil with clinical work on MCT's are void.

## Use as an occasional culinary oil

Like all food, if small quantities of coconut products are enjoyed in food recipes, then this is unlikely to be a problem. They need not be eliminated from the diet but should be consumed in moderation. This can be done by including unsaturated oils of high nutritional quality with the cooking recipe and reducing the amount of coconut oil or cream. Some suggested culinary oils with their fatty acid composition are seen in Table 4. This has the dual benefit of reducing the saturated fat and increasing the levels of unsaturated fatty acids.

**Table 4. Fatty acid composition of culinary oils**

| Culinary oils (cold pressed) | Saturated fat | Monounsaturated fat | Polyunsaturated fat |
|---|---|---|---|
| Olive oil | 15 | 80 | 5 |
| Avocado oil | 12 | 80 | 8 |
| Flaxseed oil | 8 | 18 | 74 |
| Walnut oil | 10 | 21 | 67 |
| Macadamia nut oil | 13 | 83 | 4 |
| Hazelnut oil | 9 | 80 | 11 |
| Hempseed oil | 9 | 12 | 78 |
| Canola oil | 8 | 60 | 32 |
| Pumpkin seed oil | 20 | 30 | 50 |
| RBD rice bran oil | 25 | 37 | 38 |

## Use of coconut cream/milk

To reduce the energy and saturated fat content, use half the usual amount of coconut milk or cream in the recipe or choose lite coconut milk or coconut cream. If wanting a thick and creamy consistency, thicken with cornflour.

Because of the increasing incidence of obesity, overweight and diabetes in NZ and the high saturated fat intake (39% of energy from fat), consumers should replace some of the saturated fat in their diet. A variety of fruits, coloured vegetables, lean meat, fish, legumes, pulses, nuts, appropriate unsaturated oils, reduced fat dairy products and whole grain cereals can be combined in various ways in suitable dietary regimes such as a cardio protective diet.

### Coconut in traditional diets

Most Pacific countries are food-dependent. A part of good governance for any population is the freedom to choose from a range of nutritious foods. It appears that many people do not get that choice. Diets that consist mainly of locally produced foods have been shown to prevent and reduce obesity, and the Pacific communities of Hawaii and Tonga have attempted to put this into practice. Pacific Island populations have not normally consumed coconut oil *per se* instead coconut oil is consumed as a component of coconut flesh or coconut milk.

The Pacific Island communities have had their traditional diets modified enormously and high fat and high sugar processed foods have been incorporated into their local culinary culture with adverse effects on obesity and health. Making changes in the food supply to improve access and availability of locally produced traditional foods may allow taste preferences to be met and may be a better option than encouraging behaviour change alone.

## Conclusion

For consumers living in New Zealand who are on a Western style diet, based on current evidence it would be inadvisable to switch from unsaturated oils to coconut oil. It is likely that this would lead to less favourable lipid profiles and so a potential increased risk from CHD. Consumers who are using a lot of coconut oil due to the current fad would be well advised to either limit its use, or to blend in some cold-pressed monounsaturated oil such as olive, avocado or canola oil. Although it may be a better choice than butter, coconut oil cannot be recommended as a suitable alternative to non-hydrogenated vegetable oils.

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## GLOSSARY

**Coconut flesh** Extracted from mature coconuts not young coconuts used in the production of coconut water.

**Coconut cream** The liquid squeezed from the grated coconut flesh with coconut milk theoretically being a fat reduced coconut cream by dilution.

**Copra** The dried coconut flesh from mature coconuts, which is used in the extraction of the crude coconut oil prior to refining. It is dried either by sunshine or by heating in ovens using the burning husks of the coconut as fuel.

**Coconut flour** A gluten free "flour" that is essentially dried coconut in powdered form. It is made from the coconut solids that are left over after the production of coconut cream or milk.

**Desiccated coconut** Desiccated coconut is a grated, dried (3% moisture content max.), and unsweetened fresh meat of a mature coconut.

**Coconut Water** The clear to translucent liquid present inside young green coconuts

**Extra virgin coconut oil or VCO** Coconut oil extracted from fresh coconut with no further refining. Product is low in FFA and has a sweet and pleasant natural coconut odour.

**Refined, bleached and deodorised coconut oil** Crude oil extracted from copra that has been fully refined to produce water white, bland and stable oil.

**Fully hydrogenated coconut oil also known as Confectionery Fat 92, Copha or Kremelta** This product contains no unsaturated fatty acids (including trans) with the low amounts of linoleic and oleic acids being converted to stearic acid. The melting point is 33-36 ° C as compared to unhydrogenated oil with a melting point of 26° C.This is a common item of commerce available to food manufacturers and in retail shops. It is used in confectionery and biscuit fillings.

**Examples of Saturated Fatty Acids**

| Common name | Chemical structure | *C:D* |
|---|---|---|
| Caprylic acid | $CH_3(CH_2)_6COOH$ | 8:0 |
| Capric acid | $CH_3(CH_2)_8COOH$ | 10:0 |
| Lauric acid | $CH_3(CH_2)_{10}COOH$ | 12:0 |
| Myristic acid | $CH_3(CH_2)_{12}COOH$ | 14:0 |
| Palmitic acid | $CH_3(CH_2)_{14}COOH$ | 16:0 |

| | | |
|---|---|---|
| Stearic acid | $CH_3(CH_2)_{16}COOH$ | 18:0 |
| Arachidic acid | $CH_3(CH_2)_{18}COOH$ | 20:0 |
| Behenic acid | $CH_3(CH_2)_{20}COOH$ | 22:0 |
| Lignoceric acid | $CH_3(CH_2)_{22}COOH$ | 24:0 |

**Triglycerides (Triacylglycerols)** All dietary fats are composed of triglycerides which are the common term used in this review instead of the more technically correct triacylglycerols. In the gut, ingested fats are hydrolyzed to FFA, monoglycerides, lysophospholipids, and free cholesterol, the hydrolysis products are absorbed by the enterocytes of the intestinal wall.

# Appendix: Excluded papers

**Extrapolated effects and claims made regarding coconut based on MCT, lauric acid, or monolaurin research, or palm kernel oil**

Temme, E. H., Mensink, R. P., & Hornstra, G. (1996). Comparison of the effects of diets enriched in lauric, palmitic, or oleic acids on serum lipids and lipoproteins in healthy women and men. *The American Journal of Clinical Nutrition, 63*(6), 897-903.

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**Animal or in vitro studies**

Nevin, K. G., & Rajamohan, T. (2004). Beneficial effects of virgin coconut oil on lipid parameters and in vitro LDL oxidation. *Clin Biochem, 37*(9), 830-835. doi: 10.1016/j.clinbiochem.2004.04.010

Padmakumaran Nair, K. G., Rajamohan, T., & Kurup, P. A. (1999). Coconut kernel protein modifies the effect of coconut oil on serum lipids. *Plant Foods Hum Nutr, 53*(2), 133-144.

Zakaria, Z. A., Somchit, M. N., Mat Jais, A. M., Teh, L. K., Salleh, M. Z., & Long, K. (2011). In vivo antinociceptive and anti-inflammatory activities of dried and fermented processed virgin coconut oil. *Med Princ Pract, 20*(3), 231-236. doi: 10.1159/000323756

**Coconut oil was a minor component of the trial or was used in conjunction with drugs or other food ingredients in controlled feeding experiments**

Abbey, M., Noakes, M., Belling, G. B., & Nestel, P. J. (1994). Partial replacement of saturated fatty acids with almonds or walnuts lowers total plasma cholesterol and low-density-lipoprotein cholesterol. *The American Journal of Clinical Nutrition, 59*(5), 995-999.

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Myhrstad, M. C. W., Narverud, I., Telle-Hansen, V. H., Karhu, T., Lund, D. B., Herzig, K.-H., . . . Ulven, S. M. Effect of the fat composition of a single high-fat meal on inflammatory markers in healthy young women. *British Journal of Nutrition, 106*(12), 1826-1835.

**Were from non-peer reviewed or non-reputable journals or were not studies themselves**

Amarasiri, W. A., & Dissanayake, A. S. (2006). Coconut fats. *Ceylon Med J, 51*(2), 47-51.

DebMandal, M., & Mandal, S. (2011). Coconut (Cocos nucifera L.: Arecaceae): In health promotion and disease prevention. *Asian Pacific Journal of Tropical Medicine, 4*(3), 241-247. doi: http://dx.doi.org/10.1016/S1995-7645(11)60078-3

**Research studies where outcomes such as lipid profiles were a minor part of the experimental results and were not the major objective of the study**

Dela Paz, C., Jimeno, C., Sy, R., Punzalan, F., & Dela Pena, P. (2010). The effect of virgin coconut oil on lipid profile and fasting blood sugar: A phase I clinical trial. *Philippine Journal of Internal Medicine, 48*(2).

**Used non-food extracts of the coconut plant**

Rinaldi, S., Silva, D. O., Bello, F., Alviano, C. S., Alviano, D. S., Matheus, M. E., & Fernandes, P. D. (2009). Characterization of the antinociceptive and anti-inflammatory activities from Cocos nucifera L. (Palmae). *J Ethnopharmacol, 122*(3), 541-546. doi: 10.1016/j.jep.2009.01.024

**Used subjects who were a specialist clinical population**

Assuncao, M. L., Ferreira, H. S., dos Santos, A. F., Cabral, C. R., Jr., & Florencio, T. M. M. T. (2009). Effects of dietary coconut oil on the biochemical and anthropometric profiles of women presenting abdominal obesity. *Lipids, 44*(7), 593-601.