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Coconut oil – a nutty idea?

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The increasing popularity of edible coconut products, such as coconut oil and coconut water, is difficult to miss. Coconut oil, in particular, has been endorsed by celebrities and chefs for use in cooking and baking, with fashion models reportedly eating it in an attempt to speed up their metabolic rate (*The Telegraph* 2015a). According to leading market research providers Mintel, coconut oil in food and beverages accounted for 26% of food and drink new product launches in 2012, and one in ten 16–24 year-olds currently buys coconut oil (Mintel 2015). Whole Foods reportedly sold six tonnes of coconut oil across the UK in the month of February 2015 (*The Telegraph* 2015a).

Recipe books, advertisements and some journal articles are claiming that coconut oil is a cure-all product that has weight reduction, cholesterol-lowering, wound healing and immune system, energy and memory-boosting effects and can be used to treat Crohn's disease, irritable bowel syndrome, thyroid conditions, diabetes, as well as Alzheimer's and Parkinson's diseases (DebMandal & Mandal 2011; Positivemed.com 2015). Such claims have led to a significant amount of press coverage (*The Independent* 2014, 2015b, *The Daily Mail* 2015b; *The Telegraph* 2015a), comment from the scientific community (Cunningham 2011; DeDea 2012; Inayat *et al.* 2013; Varteresian & Lavretsky 2014) and the inclusion of coconut oil within food products as a perceived health-boosting ingredient, with companies using it as a unique selling point. Additionally, a recent story suggested that coconut oil produces fewer aldehydes than unsaturated oils when heated and is therefore better for health (*The Telegraph* 2015b), a claim that was based on unpublished data.

As a beauty product, coconut oil has been advocated as being useful for frizzy hair, stretch marks, as a moisturiser and make-up remover. Swilling around the mouth has even been encouraged to prevent tooth decay (*The Independent* 2015b). Whilst topical use of coconut oil and indeed the consumption of coconut water [containing around 20 calories per 100 g and providing small quantities of micronutrients (USDA 2015)] are unlikely to be of concern for public health, high consumption of saturated fatty acid-laden coconut oil may well be. In America, for example, big-brand food manufacturers have started selling fat spreads made with coconut oil (Foodnavigator.com 2015). Considering the widespread use of fat spreads as an everyday product, is this a good idea?

What is coconut oil?

Coconut oil is a colourless to brown-yellow edible oil derived from mature coconuts. Standard coconut oil is normally produced by firstly drying the kernel (to produce something known as copra) and secondly refining, bleaching and deodorising the extracted oil. So-called virgin coconut oil is instead made via a 'wet process', either being extracted from coconut milk or from fresh kernel which is not subjected to drying or chemical refining (Babu *et al.* 2014). Coconut oil comprises 99.9% fatty acids; of these, 91.9% are saturated fatty acids (SFA), 6.4% are monounsaturated fatty acid acids (MUFA) and 1.5% are polyunsaturated fatty acids (PUFA), and coconut oil contains no dietary cholesterol (PHE 2015). The individual fatty acid composition of coconut oil can be seen in Figure 1. The principal fatty acids are lauric (C12:0), myristic (C14:0) and palmitic (C16:0) acids. Virgin coconut oil has been found to contain up to seven times higher concentrations of polyphenols than standard coconut oil, with total polyphenol contents of up to 80 mg gallic acid equivalents/100 g oil reported in virgin coconut oil (a figure comparable to extra virgin olive oil), although concentrations differ depending on coconut variety (Nevin & Rajamohan 2004a; Seneviratne & Sudarshana Dissanayake 2008; Marina *et al.* 2009). The lower levels in standard coconut oil are likely to be due to minor components being destroyed during the manufacturing process and also because polyphenols are polar compounds and therefore have a higher affinity for liquid coconut milk and fresh copra as opposed to dried copra

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(Seneviratne & Sudarshana Dissanayake 2008). Despite the difference in concentrations, the mixture of phenolics present (including ferulic, p-coumaric, caffeic, gallic and syringic acids and catechin) (Seneviratne & Sudarshana Dissanayake 2008; Seneviratne *et al.* 2009) is thought to be largely the same in standard and virgin coconut oils (Seneviratne & Sudarshana Dissanayake 2008). Phenolic composition has been characterised only in a small number of publications and further verification is needed, particularly in terms of quantities. Of the compounds identified so far, all are found in a variety of other plant foods. For example, ferulic acid is present in much higher quantities in wholegrain bread flour (72 mg/100 g vs. 0.3 mg/100 g reported in virgin coconut oil), catechin is present in much higher quantities in cocoa (108 mg/100 g vs. 0.3 mg/100 g reported in virgin coconut oil) and p-coumaric acid is higher in dried dates (5.8 mg/100 g vs. 0.2 mg/100 g reported in virgin coconut oil) (Neveu *et al.* 2010). Various biological effects of coconut oil, such as blood pressure and cholesterol lowering, reduction in low-density lipoprotein cholesterol (LDL-C) oxidation and potential as an Alzheimer's treatment, as have been reported in animal and *in vitro* studies, have been attributed to the phenolic content (Nevin & Rajamohan 2004a; Nurul-Iman *et al.* 2013; Fernando *et al.* 2015). Coconut oil also contains small amounts of vitamin E (0.66 mg/100g) and vitamin K (1 μg/100 g) (PHE 2015).

Saturated fatty acids and plasma lipids

Historically, coconut oil has been described as 'one of the most potent agents for elevating serum cholesterol level' (The Inter-Society Commission for Heart Disease Resources 1970). This is due to its very high saturated fatty acids content (up to around 92 g/100 g), which exceeds other edible fats (see Fig. 2), including butter that contains around 52 g/100 g (PHE 2015). The plasma lipid raising potential of saturated fatty acids and the positive association between plasma cholesterol and heart disease risk have long been established in the literature (Keys *et al.* 1965; Castelli *et al.* 1992; Hu *et al.* 2001). This is the rationale behind current UK dietary guidelines (and similar guidelines around the world) for cardiovascular disease (CVD) prevention, which recommend that saturated fatty acid intake should be limited to no more than 11% of food energy, which, in the context of average energy needs, equates to a maximum of 30 g per day for men, 20 g for women and less for children (DH 1991).

The link between saturated fat and heart disease has been recently questioned and debated at length in the press (*The Daily Mail* 2015a; *The Independent* 2015a), due to the publication of some journal articles concluding that there is no association between the two (Siri-Tarino *et al.* 2010; Malhotra 2013; Chowdhury *et al.* 2014). This topic is currently being examined by a BNF Task Force report on cardiovascular disease (due for publication in 2017) and the UK Scientific Advisory Committee on Nutrition. The view that the consumption of saturated fatty acids is not detrimental for health, largely based on observational evidence, has been used to support the use of coconut oil.

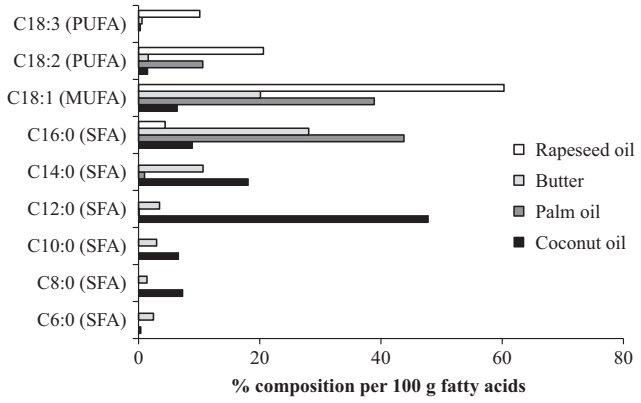

Figure 1 Comparison of the fatty acid composition of selected edible oils and fats. SFA, saturated fatty acid; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid. Data source: McCance and Widdowson's Composition of Foods (PHE 2015)

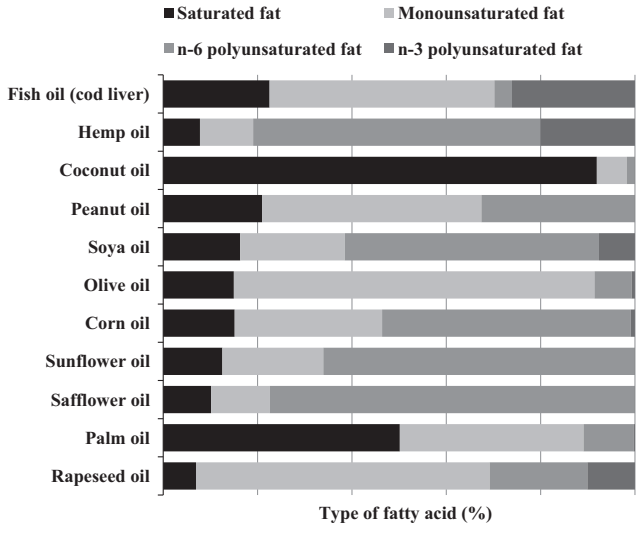

Figure 2 Fatty acid composition of edible oils. Data source: McCance and Widdowson's Composition of Foods (PHE 2015)

Cardiovascular risk markers – observational evidence

It is important to note that claims relating to potential health benefits of coconut oil are often based solely on animal or *in vitro* studies (Nevin & Rajamohan 2004a, 2008; Hayatullina *et al.* 2012; Nurul-Iman *et al.* 2013; Wang *et al.* 2015), or human studies feeding one component of coconut oil rather than the whole food (Inayat *et al.* 2013). Overall, human studies on coconut oil itself are limited and largely non-UK based. A handful of observational studies have examined the prevalence of CVD risk markers in populations for whom coconut represents an important part of the diet. A study of 1839 Filipino women using 24-hour dietary recall reported that coconut oil consumption was not associated with raised levels of triacylglycerides (TAG), LDL-C or with ratio of total cholesterol to high-density lipoprotein cholesterol (TC/HDL-C) (Feranil *et al.* 2011). In pre-menopausal women, the highest tertile of coconut oil intake was associated with higher total cholesterol levels compared to the lowest tertile, and moderate and high intakes were associated with significantly higher HDL-C levels. However, these relationships were not identified in post-menopausal women and the paper did not detail how low, moderate and high intakes were classified in terms of amounts of oil. In a comprehensive study of two island populations of Polynesians (436 Pukapukans and 939 Tokelauans), for which coconuts are a staple food, carried out in the years 1964–1971 (Prior *et al.* 1981), all foods consumed by a subsection of randomly selected families (13 families in Pukapuka and 18 families in Tokelau) over a 7-day period were weighed and recorded by researchers and dishes were analysed chemically to determine fatty acid composition. Pigs and chickens on the islands also had high intakes of coconuts, which added to the saturated fatty acid content of animal foods in the diets. Twenty-four-hour recall was also used to obtain dietary data, though only in a subsample of individuals aged 25–54 years [n = 165 Pukapukans (74% of the age group) and n = 77 in Tokelauans (46% of the age group)]. In the Tokelauan diet, coconuts supplied 63% of energy and the diet contained 54% energy from fat and 48% energy from saturated fatty acids. In the Pukapukan diet, coconuts contributed 34% of energy and the diet contained 37% energy from fat and 28% energy from saturated fatty acids. The groups with the highest mean cholesterol concentrations among the Pukapukans were males aged 35–44 years (4.71 mmol/l) and females aged 55–64 years (5.03 mmol/l), and among Tokelauans were males aged 45–54 years (5.7 mmol/l) and females aged 55–64 years (6.4 mmol/l). For comparison, in the UK, the average diet comprises 34.6% energy from fat and 12.6% energy from saturated fatty acids (PHE 2014) and total cholesterol levels are estimated to be 5.5 mmol/l in women and 5.4 mmol/l in men (WHO 2009). The fact that total cholesterol was higher in the Tokelau population than in Pukapukans could indicate a deleterious effect of higher coconut consumption on cholesterol; however, as dietary data were not obtained for all participants, blood samples were not taken in the same year as dietary measurements and the study was observational in design, it is difficult to draw firm conclusions from these data. It was observed that among Tokelauan individuals who migrated to New Zealand, LDL-C levels increased, despite the percentage of energy from fat decreasing from 57% to 43%. This finding is often used in support of coconut oil consumption due to the low incidence of vascular disease in the indigenous populations. However, this scenario is, of course, completely uncontrolled and does not account for other dietary and lifestyle factors which may have impacted on the lipid profile of these migrants.

A case–control study carried out in South India found no differences in coconut oil intake between coronary heart disease patients and healthy matched controls (Kumar 1997). Average daily intakes of coconut oil were 13.6 ml among patients and 12.5 ml among controls, around 2–3 teaspoons. However, subjects had a mean age of 57 years and were asked to recall their dietary habits from age 15 years onwards, a methodology of questionable validity. Similarly, a study of diabetic cases and controls in South India found no significant differences in lipid profile or antioxidant enzymes between individuals who used coconut oil in cooking, contributing 13–20% of their total energy intake, and those consuming similar amounts of sunflower oil (Sabitha & Vasudevan 2010).

In a study of 71 endarterectomy samples (atherosclerotic plaques removed from artery linings), it was reported that despite coconut oil consumers having significantly higher plasma myristic acid concentrations than sunflower oil consumers, this was not reflected in plaque fatty acid composition (Palazhy *et al.* 2012). However, interestingly, the author reported that the atheromas mostly comprised saturated fatty acids, in stark contrast to earlier studies which had reported that unsaturated fatty acids were the principle component (Lausada *et al.* 2007).

Neither plasma nor plaque lauric acid concentrations were significantly higher in the coconut oil group; however, capric acid was identified in some plaques, providing evidence of the deposition of medium-chain fatty acids (MCFAs) in tissues (see 'Medium-chain triglycerides' section below). Plaque kinetics are complex and fatty acid composition is not restricted to dietary sources. The authors speculated that medium-chain triglycerides (MCTs) may be converted to longer-chain SFAs before plaque incorporation. As dietary fatty acid intake has been associated with atherosclerotic plaque stability (Calder 2012), the effect of coconut oil within this context is certainly of relevance but it is important to note that the study was uncontrolled and the duration and amount of oil consumed varied between subjects.

Although many papers cite observational data as principal evidence for the effects of coconut oil, as always with this type of study a cause and effect relationship cannot be established. In addition, the methods of dietary analysis used in all but one of the aforementioned observational studies (Prior *et al.* 1981) relied on recall and self-report which can lead to misreporting and therefore biased data (Castro-Quezada *et al.* 2015). Robust randomised controlled trial (RCT) evidence is likely to provide more insight.

Cardiovascular risk markers – randomised controlled trials

The majority of the RCTs in this area have focused on lipid-modifying effects of coconut oil, usually in comparison with other dietary fatty acids. The duration of the dietary interventions has ranged from 7 days to 12 months, with many studying the effects after 4 weeks of coconut oil consumption. A summary of the evidence can be seen in Tables 1 and 2.

Most of the RCT evidence available demonstrates that coconut oil raises total cholesterol, LDL-C and HDL-C and removing coconut oil from the diet reduces total cholesterol and LDL-C with varying effects on HDL-C depending on the study. This is perhaps unsurprising considering the known cholesterol-raising effects of coconut oil's principal saturated fatty acids lauric, myristic and palmitic acids (Flock & Kris-Etherton 2013). The TC/HDL-C ratio has been advocated as a marker of CVD risk (Mensink *et al.* 2003), with a lower ratio being more favourable, but almost all of the RCTs presented here do not specifically report this as an outcome. Based on its fatty acid composition, coconut oil is predicted to decrease TC/HDL-C ratio when replacing mixed fats in an average US diet isoenergetically (Mensink *et al.* 2003), although to a lesser extent than olive, soya bean and rapeseed oil (Mensink *et al.* 2003). However, the value of measuring HDL-C as a risk marker, without taking into account its functionality in relation to cholesterol efflux, has recently been called into question. Therefore, simply reporting that coconut oil raises HDL-C may not necessarily indicate a protective effect; the picture may be more complex (Escola-Gil *et al.* 2015). Despite a combined participant number of around 500, the individual study participant numbers in human RCTs investigating coconut oil and plasma lipids are small, which can reduce statistical power. Furthermore, the study designs vary greatly in terms of dosage, duration, delivery vehicle and the comparator, making it difficult to compare the results between studies. Non-randomised studies of a sequential design with no or an insufficient wash-out period may also be subject to carry-over effects, lessening the reliability of the results.

Whilst lipid profile is a very significant marker of future health outcomes (Cromwell *et al.* 2007), there is a paucity of studies investigating the effect of coconut oil on additional biomarkers such as blood pressure, vascular function and inflammation. One postprandial study reported a reduction in flow-mediated dilatation after the consumption of coconut oil vs. safflower oil, which just failed to reach significance, suggesting a possible decrease in blood vessel elasticity (Nicholls *et al.* 2006), whereas two longer-term studies reported no significant difference in blood pressure effects between coconut oil and soya bean oil after 7 days (Ganji & Kies 1996) or coconut oil and no intervention after 3 months (Cardoso *et al.* 2015). Furthermore, two studies examining the impact of a 3-month period of supplementation with 6 g coconut oil per day versus the same dose of fish oil in renal transplant patients reported no significant differences (Kooijmans-Coutinho *et al.* 1996) and significantly raised blood pressure (van der Heide *et al.* 1993) in the coconut oil groups; however, the composition of the coconut oil had been modified in both cases to contain 63% caprylic and 36% capric acids, meaning that these results cannot be extrapolated to whole coconut oil. An RCT ($n = 45$) comparing palm olein, coconut oil and virgin olive oil at 20% of energy for 5 weeks reported no significant differences in the inflammatory markers TNF-α, IL-1β, IL-6 and IL-8, high-sensitivity C-reactive protein and interferon-γ but significant increases in plasma $PGF_{1\alpha}$ and proinflammatory LTB_4 after the consumption of palm olein and coconut oil compared to virgin olive oil (Voon *et al.*

Table 1 Randomised controlled trials conducted in humans investigating the effect of coconut oil consumption on plasma lipids

Study	Subjects	Dose of coconut oil	Duration	Comparator	Effect of coconut oil vs. comparator	Additional information
Ganji & Kies 1996;	*n* = 10 (five men and five women) Normolipidaemic	20%E	7 days	Soya bean oil	↑TC ↑LDL-C ↑TAG	All meals provided under controlled conditions
Mendis & Kumarasunderam 1990;	*n* = 25 men Healthy	30%E	8 weeks	Soya bean oil	↑TC	All meals provided under controlled conditions
Assuncao *et al.* 2009;	*n* = 40 women Abdominal obesity (WC > 88 cm)	14%E	12 weeks	Soya bean oil	↑HDL-C	Free-living
Fisher *et al.* 1983;	*n* = Nine men Normolipidaemic	31%E	18 days	Corn oil	↑TC ↑IDL-C + LDL-C ↑HDL-C ↑TAG	75% of diet consumed as liquid formula
Schwab *et al.* 1995;	*n* = 15 women Healthy	4%E	4 weeks	Palm oil	No significant differences	Free-living
Cox *et al.* 1995;	Cox *et al.* 1995; *n* = 28 (13 men and 15 women) Mild hypercholesterolaemic	18%E	6 weeks	Butter	↓TC ↓LDL-C	Free-living
				Safflower oil	↑TC ↑LDL-C ↑HDL-C in women only	
Cox *et al.* 1998;	*n* = 41 (24 men and 17 women) Normolipidaemic	18%E	6 weeks	Butter	↓LDL-C	Free-living
				Safflower oil	↑TC ↑LDL-C ↑HDL-C	
Reiser *et al.* 1985;	*n* = 19 men Normolipidaemic	21%E	5 weeks	Beef fat	↑TC ↑LDL-C ($P = 0.08$) ↑HDL-C ↓TAG	Two meals per day consumed under controlled conditions
				Safflower oil	↑TC ↑LDL-C ↑HDL-C	
Hegsted *et al.* 1965;	*n* = Ten men (per test diet) Normolipidaemic	22%E and 38%E	4 weeks	Beef fat, safflower oil, olive oil, cocoa butter, corn oil, fish oil and mixtures thereof	Butterfat: ↑TC by 60–65% at 38% energy Coconut oil: ↑TC by 41% at 22%E ↑TC by 56% at 38%E	All meals provided under controlled conditions. Butterfat was the most potent cholesterol-raising agent. Coconut oil was the second most potent

Table 1 *Continued*

Study	Subjects	Dose of coconut oil	Duration	Comparator	Effect of coconut oil vs. comparator	Additional information
Muller *et al.* 2003;	*n* = 25 women Healthy	34%E (Diet 1 – High saturated fatty acids)	3 weeks	Diet 2 – low fat and low saturates (high carbohydrate) Diet 3 – high unsaturated fatty acids	↑HDL-C ↓TAG ↑TC ↑LDL-C	Diet 3 resulted in the most favourable lipid profile Free-living
Liau *et al.* 2011;	*n* = 20 (13 women and 7 men) Obese (using Malaysian criteria, BMI ≥ 25 kg/m^2)	30 ml per day	4 weeks	No comparison group	↑HDL-C No significant differences from baseline	Free-living
Cardoso *et al.* 2015	*n* = 114 (92 in the coconut oil group and 22 in the control group) Coronary artery disease patients	13 ml per day	3 months	Carbohydrates	↑HDL-C	Free-living

%E, percentage of dietary energy; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; IDL-C, intermediate-density lipoprotein; HDL-C, high-density lipoprotein cholesterol; TAG, triacylglycerol; WC, waist circumference. Effects are statistically significant ($P < 0.05$) unless otherwise stated.

2015). It is impossible to determine the impact of coconut oil on inflammatory status from a single study, especially due to the extreme complexity of the inflammatory system and its role in CVD. There is, however, some evidence that saturated fatty acids are proinflammatory (Santos *et al.* 2013; Murphy *et al.* 2015).

As mentioned above, the link between saturated fatty acids, plasma lipids and CVD risk has recently been challenged and is indeed complex. Not all saturated fatty acids are equal in terms of hypercholesterolaemic potential, with stearic acid, for example, thought to have little effect, but a large body of evidence has shown that the principal fatty acids within coconut oil, namely lauric, myristic and palmitic acids, consistently raise total cholesterol and LDL-C (Mensink *et al.* 2003; WHO 2008; Hunter *et al.* 2010). Furthermore, food matrices appear to have an impact. For example, the consumption of dairy products appears to be either neutral or protective against CVD (Gibson *et al.* 2009), despite lauric, myristic and palmitic acids making up around 40% of the fatty acids present and dairy foods being one of the main sources of saturated fatty acids in the UK diet (PHE 2014). The reason for this is yet to be fully elucidated but evidence suggests this may be due to the high micronutrient density (Christensen *et al.* 2009) or effects on gut microflora (St-Onge *et al.* 2000). The French government has chosen to provide separate guidance for intakes of different fatty acids to reflect their differing biological effects (AFFSA 2010). This includes the recommendation that intakes of lauric, myristic and palmitic acids should be limited to less than 8% of energy due to their atherogenic potential. In terms of consumer understanding and action, advice from the UK government (DH 1991) and European and international bodies (WHO 2008; EFSA 2010) to limit intake of all saturated fatty acids may be easier for the general public to implement. This is particularly true as all foods provide a mixture of different fatty acids, making reduced intake of foods high in saturated fatty acids in general easier than attempting to reduce the intake of individual saturated fatty acids. Saturated fatty acids are not an essential part of the human diet. A recent Cochrane review including 15 RCTs with a total of 59 000 participants found that cutting down saturated fatty acid intake led to a 17% reduction in the risk of CVD (Hooper *et al.* 2015). Importantly, this review and several others indicate that replacing saturated fatty acids with small amounts of unsaturated fatty acids produces more favourable plasma lipid profiles than replacing with

Table 2 Randomised controlled trials conducted in humans investigating the effect of removing coconut oil from the diet on plasma lipids

Study	Subjects	Baseline dose of coconut oil	Duration	Replacer	Effect of replacing coconut oil	Additional information
Mendis *et al.* 1989	*n* = 16 men Healthy	100 g coconut + 10 ml coconut oil per day	6 weeks	10 ml corn oil + 10 g cow's milk powder per day	↓TC ↓LDL-C ↓HDL-C ↑TAG	All foods supplied
Ng *et al.* 1991	*n* = 83 (22 women and 61 men) Healthy	24%E	5 weeks	Corn oil	↓TC ↓LDL-C ↓HDL-C ↓TAG	All foods supplied Corn oil had double the TC and LDL-C-lowering potential as palm oil
				Palm oil	↓TC ↓LDL-C ↓HDL-C	
Mendis *et al.* 2001	*n* = 60 (42 men and 18 women)	Phase 1: 17.8%E reduced to 9.3%E	8 weeks	Carbohydrate (total kcal decreased)	↓TC ↓LDL-C	Free-living Changes at the end of dietary phases are reported relative to baseline (17.8%E coconut oil)
		Phase 2: 9.3%E reduced to 4.7%E	12 months	Group 1: carbohydrate (total kcal decreased)	↓TC ↓LDL-C ↑HDL-C ↓TC:HDL ratio	
				Group 2: soya bean and sesame oils (total kcal decreased)	↓TC ↓LDL-C ↑HDL-C ↑TAG ↓TC:HDL ratio	

%E, percentage of dietary energy; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; TAG, triacylglycerol. Effects are statistically significant ($P < 0.05$).

carbohydrates (Micha & Mozaffarian 2010). Meta-analyses of observational studies concluding that there is no relationship between saturated fatty acid intake and CVD risk did not differentiate between studies according to which nutrients had been used to substitute saturated fatty acids in the diet. Analysing these types of studies all together thus creates a biased end result.

Medium-chain triglycerides

Advocates of coconut oil claim that some of the specific saturated fatty acids present in coconut oil, those of medium-chain length, actually confer health benefits (Babu *et al.* 2014). MCTs are mixed triglycerides of saturated fatty acids with a chain length of 8–10 carbons [*i.e.* octanoic acid (caprylic, C8:0) and decanoic acid (capric, C10:0)] (Marten *et al.* 2006). Due to their relatively simple digestion and absorption, MCTs have been used since the 1950s for clinical purposes such as total parenteral nutrition and pre-term infant formula (Marten *et al.* 2006). In addition, the results from animal studies and some human studies have suggested that the consumption of MCTs promotes weight loss, increases satiety resulting in reduced daily food intake and increases energy expenditure, compared to other types of fatty acids at a range of doses of up to 40% of energy, though subject numbers in these studies are often small (St-Onge & Jones 2002). Hexanoic acid (caproic acid, C6:0) and odecanoic acid (lauric acid, C12:0) are sometimes also referred to as MCFAs. However, there is disagreement as to whether they should be classified in this way. If both of these fatty acids are included in the definition, coconut oil could be described as containing around 65% MCFAs. However, it seems unlikely that lauric acid is oxidised immediately in the liver, a characteristic of C8–C10 fatty acids, because of its potent cholesterol-raising potential (Mensink *et al.* 2003). MCT preparations used clinically and those used in the vast majority of human studies investigating potential biological effects, such as increasing energy expenditure, consist predominantly of caprylic (C8:0) and capric acids (C10:0) (Nagao & Yanagita 2010; Costa *et al.* 2012). Considering that lauric acid represents around 48% of the fatty acid content present in coconut oil and caprylic and capric acids only around 14% combined, studies feeding purified oils consisting of only the latter two are of little direct relevance to understanding the effects of coconut oil.

Weight loss

A very small number of studies have reported the effects of coconut oil on bodyweight in humans. An observational study of Pacific island populations consuming high amounts of coconuts revealed much higher amounts of lauric and myristic acids, the two main fatty acids present within coconut oil, in the adipose tissue than that of New Zealand Europeans who consume a more Western style diet (10–12% vs. 0.3% and 16–17% vs. 4.2%, respectively) (Prior *et al.* 1981). Of the two populations studied, the Tokelauans, who consumed higher amounts of coconut (63% of energy derived from coconut vs. 34% in the Pukapukan diet), were heavier and had larger subscapular skin folds.

In an RCT, 40 free-living women aged 20–40 years were instructed to consume 30 ml soya bean or coconut oil per day instead of their usual cooking oil for 12 weeks (Assuncao *et al.* 2009). Both groups experienced a reduction in body mass index (BMI). However, the groups had also been instructed to walk for 50 minutes per day and follow a healthy dietary pattern, and both groups consumed approximately 10% less calories than at baseline. Only the coconut oil group had a reduced waist circumference at the end of the study (a reduction of 1.4 cm). Though the authors used 24-hour dietary recall at the beginning and end of the study period to assess compliance, the exact amounts of coconut oil consumed by subjects were not reported. In a single-arm open-label study, 13 women and seven men aged 24–51 years, with a mean BMI of 32.5 kg/m^2, consumed 30 ml virgin coconut oil per day for 4 weeks (Liau *et al.* 2011). A statistically significant reduction in waist circumference (2.61 $\pm$ 2.17 cm) occurred in male subjects. A reduction of 3.00 $\pm$ 6.03 cm also occurred in women but this was non-significant due to the large variation in the data. Importantly, this study had no control group and very small sample size so firm conclusions cannot be drawn. In a recent non-randomised study of patients with coronary artery disease, 92 individuals consumed 13 ml coconut oil per day for 3 months and 22 served as controls (Cardoso *et al.* 2015). Both groups followed general healthy dietary advice for a 3-month run-in period and the 3-month intervention. The two groups consumed equal amounts of energy because, for the coconut oil group, the energy consumed as coconut oil was offset by reduction in the intake of carbohydrates. Statistically significant differences of -1.9 cm in waist circumference and 0.1 mmol/l in HDL were observed in the coconut oil group compared with the control. However, this study

has many methodological flaws including unequal group sizes, unclear treatment allocation methodology and the fact that subjects in the coconut oil group were instructed to consume the oil 'with fruit or on its own', introducing a further confounder.

Explanations for why coconut oil may lead to weight loss include the ideas that MCTs are 'instantly' metabolised by the body rather than being stored; MCTs are more satiating than longer-chain triglycerides; and MCT consumption speeds up basal metabolic rate. As explained previously, the vast majority of studies suggesting that MCTs may promote weight loss due to the effects on energy expenditure cannot be extrapolated to coconut oil due to the fact that coconut does not comprise 100% capric and caprylic acids, unlike purified MCT oils. Additionally, a small satiety study ($n = 18$) revealed that the effect of a coconut oil fatty acid-rich meal on hunger and fullness ratings and amount of food consumed at a subsequent *ad libitum* meal did not differ to the effect of isoenergetic meals enriched with fatty acids from beef tallow or dairy foods (Poppitt *et al.* 2010). To our knowledge, there is no reported evidence of coconut oil increasing metabolic rate in humans. Overall there is insufficient, good quality evidence at present to conclude that the consumption of coconut oil leads to a reduction in adiposity.

Immune-modulating effects

The 'immune-boosting' properties of coconut oil claimed by coconut oil advocates have been attributed to the saturated fatty acid lauric acid, which is present in human breastmilk (Newburg & Walker 2007), and monolaurin (also known as glycerol monolaurate and variations thereof), a monoglycerol ester of lauric acid which is said to be produced *in vivo* after the consumption of coconut oil (Matonis 2014). Both monolaurin and lauric acid reportedly have antibacterial and antiviral properties but this has mostly been demonstrated *in vitro* (Schlievert & Peterson 2012; Tangwatcharin & Khopaibool 2012; Hess *et al.* 2014; Huang *et al.* 2014) or in animal studies (Strandberg *et al.* 2010; Manohar *et al.* 2013). Monolaurin has generally recognised as safe (GRAS) status in the US, is used as an emulsifier in food products and sold as a supplement. Some human evidence suggests that monolaurin may be effective at preventing infections when used topically (Strandberg *et al.* 2009; Wang *et al.* 2014). However, how much monolaurin is produced endogenously through lauric acid metabolism and the benefits of this *in vivo* remains unknown. To our knowledge, no human studies have demonstrated antimicrobial effects of coconut oil *per se* in humans.

Cognitive effects

Claims relating to coconut oil and the treatment for Alzheimer's disease are largely based on animal studies and a small number of human studies which have reported that a product comprising predominantly of caprylic acid (C8:0), one of the more minor fatty acids in coconut oil (present at around 7%, see Fig. 1), may improve cognitive function in patients (Reger *et al.* 2004; Henderson *et al.* 2009). The mechanism of action is thought to be related to the induction of mild ketogenesis, something which may alleviate the low cerebral metabolic rate of glucose use, which is a feature of Alzheimer's disease. Individual case studies have reported similar findings (Farah 2014; Newport *et al.* 2015). However, to our knowledge, there are no human studies directly looking at the effects of coconut oil on brain function in either healthy people or those suffering from cognitive impairment.

Conclusions

Until recently, coconut oil was not commonly consumed as part of the UK diet, with no reported use by any of the participants of the 2008–2009 and 2011–2012 *National Diet and Nutrition Survey* (PHE 2014). However, the flurry of cookery books dedicated to coconut oil alone, published from 2013 to present, and new products containing coconut oil in the market are likely to have increased our consumption. Coconut oil contains, on average, 90% saturated fatty acids, of which 75% are LDL-C and total cholesterol-raising lauric, myristic and palmitic acids. Such quantities are huge compared to all other food sources (see Figs 1 and 2). The British Heart Foundation and UK Department of Health advise that due to its high saturated fatty acid content, coconut oil should be consumed only in small quantities and that sources of unsaturated fatty acids are a better choice for everyday use (NHS Choices 2015; British Heart Foundation). The *Change4Life* website (2015) lists coconut oil within a category of foods labelled 'leave these on the shelf!'. Not only does using coconut oil in place of other fats go against dietary guidelines which recommend reducing intake of saturated fatty acids (DH 1991), but, at nine calories per gram, pure fats are the most calorie-dense food source. Therefore, consuming large amounts of any oil may promote positive energy balance and weight gain.

Unlike olive oil, for example, which is MUFA rich and backed by a large body of evidence supporting its health benefits (Psaltopoulou *et al.* 2011; Schwingshackl & Hoffmann 2014) as well as a European Food Safety Authority (EFSA)-approved health claim (Agostoni *et al.* 2011), and rapeseed oil, an omega-3 fatty acid-rich component of the Nordic dietary pattern (Nordic Council of Ministers 2012), there is little evidence to support any health-promoting effects of coconut oil. The effect of coconut oil on CVD risk markers other than plasma lipids is largely unreported in humans, although saturated fatty acids in general have been shown to be deleterious (Vafeiadou *et al.* 2012; Murphy *et al.* 2015). Due to existing knowledge regarding saturated fatty acids and heart disease, evidence presented here suggesting that coconut oil raises plasma lipids and a lack of large, well-controlled human studies published in peer-reviewed journals demonstrating clear health benefits of coconut oil, frequent use of coconut oil should not be advised. So, if you spot coconut oil high up the ingredient list, think twice before eating the product on a regular basis.

Conflict of interest

The authors have no conflicts of interest to disclose.

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