



Clegg, ME. (2017) 'They say coconut oil can aid weight loss, but can it really?', *European Journal of Clinical Nutrition* 

DOI:

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1	They say coconut oil can aid weight loss, but can it really?
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14	Running title: Coconut oil for weight loss
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16	Keywords: Coconut oil, MCT oil, satiety, energy expenditure, body weight
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18	Conflict of interest.
19	The author declares no conflict of interest.
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## 29 Abstract

There has in recent years, been much media speculation and consumer interest in the beneficial satiating properties of consuming coconut oil and it's potential to aid weight loss. However the media has primarily cited studies using MCT oil. The current perspective looks at the research that is available on coconut oil. It examines if and how MCT related research can be applied to coconut oil and if there is potential for coconut oil to aid weight loss. The current report indicates a lack of consistent evidence on the topic of coconut oil, satiety and weight loss. Given both the publicity and the increased consumption of coconut oil further research, particularly long term clinical trials, in this area are warranted. 

57 Coconut oil has gained considerable popularity in recent years with coconut oil in food and beverages accounting for 26% of food and drink new product launches in 2012, this is an 58 increase from 15% in 2008 (1). Coconut oil exports across Asia have also grown 3.3% 59 annually over the past five years, according to the Asian Pacific Coconut Community (2). 60 61 This is not surprising given the numerous ways that coconut oil has been identified by the media, to potentially improve our lives. Articles are wide ranging promoting adding it to stir-62 fries, baking with it and even adding it to coffee (3, 4). Many media articles promote the 63 consumption of coconut oil for weight loss, advocating similar health benefits to that of 64 65 medium chain triglycerides (MCT). This has contributed to an increase in intake of coconut oil in recent years (5). Coconut oil is said to aid weight loss through a combination of 66 increased energy expenditure and satiety induced by MCT. MCT are dietary triglycerides 67 68 with fatty acids chains that are 6 to 12 carbon atoms in length (6). MCT have a smaller molecular weight than long chain triglycerides. This allows them to be more rapidly and 69 70 completely hydrolysed, and absorbed when there are decreased intraluminal concentrations 71 of pancreatic enzymes and bile salts (7). During digestion, MCT are converted to medium-72 chain fatty acids (MCFA) and transported directly in the portal venous system to the liver, as 73 opposed to being transported as chylomicrons in the lymphatic system like LCT (8). MCT 74 therefore bypass peripheral tissues, including adipose tissue, which makes them less likely to be deposited into the adipose tissue via the actions of hormone-sensitive lipase (6). 75 76 Finally, MCFA can cross the mitochondrial membrane of the liver and muscle independently of the acylcarnitine transfer system, this makes them a more readily available energy source 77 78 that is likely to be utilised more rapidly (9).

79

It has been proposed that MCT can affect satiety via a number of mechanisms; however a lot is still unknown. Potential mechanisms include the production of ketones due to the increased acetyl-CoA influx which is necessary to oxidize fatty acids (10). Furthermore, Van Wymelbeke et al (11) and Rolls et al (12) indicate that the increase in satiety maybe due to the rapid rate of absorption of MCT. Where LCT result in two peaks during absorption; the

initial peak at the point of ingestion and a second delayed peak at the beginning of the next
meal, MCT are fully absorbed at the point of ingestion (13). Hence, MCT may contribute to
satiation due to complete absorption mechanism.

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89 The evidence for both increases in diet induced thermogenesis (14-24) and reduced food intake (11, 12, 25-29) following the consumption of MCT has been well documented, with 90 interventions using MCT oil indicating that there is potential for it to help aid weight loss (10, 91 30-33) (Table 1). However it needs to be emphasised that MCT oil and coconut oil are not 92 93 the same thing. Lauric acid (carbon chain length 12) is found in much larger quantities in coconut oil, making up 47.7 % of the total fat, where no lauric acid is found in MCT oil. Other 94 MCFA in coconut oil are capric acid (C10- 5.5%), caprylic acid (C8-7.6%) and caproic acid 95 96 (C6 - 0.52%) (34). There is some debate as to whether lauric acid is a MCT or not and this 97 is demonstrated in how it is utilized in the body. Unlike with pure MCT oil containing fatty acids of shorter carbon length (C6-C10), only twenty to thirty percent of lauric acid is taken 98 99 directly to the liver to be used as energy via the portal vein (35). This means that in total only 100 ~23.16% of the coconut oil contains MCTs that is absorbed and metabolised in the same 101 way as pure MCT oil.

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103 Studies on satiety and MCT have shown that 3g is not sufficient to have an effect on satiety 104 (26, 27). However this is in contrast to a study by Rolls et al (12) that showed a dose as low as 2.9g (100kcal containing 24% MCT) reduced food intake in dieters but had no effect in 105 106 non-dieters. This obviously has practical implications as it is dieters that are most likely to want to see the satiating effects. The other studies that have shown an effect have used 107 much higher doses of ~25g (11, 25, 29). Studies looking at energy expenditure have shown 108 doses of 5g have the ability to increase postprandial thermogenesis (18). However, similar to 109 satiety the majority of other studies tended to use much larger doses of greater than 20g (17, 110 21, 36). For weight loss, similarly doses of 5g of MCT for 12 week resulted in significant 111 112 decreases in body fat, subcutaneous and visceral fat (31).

This shows that doses as low as 5g and perhaps 3g may have an effect on satiety and body 114 weight; however the majority of the research has used MCT amounts much higher than this. 115 Nonetheless, if a dose as low of 5g MCT per day is sufficient to have beneficial effects on 116 117 weight management then 21.6g of coconut oil would be required to obtain sufficient amounts of MCFA. Current UK guidelines limit the intake of saturated to a maximum of just 21g in 118 females and 31g in males (19-64 years) (37). Hence at the lowest dose of coconut oil known 119 to have an effect on body weight, people will have reached or almost reached their total 120 121 saturated fat intake for the day.

122

123 Two studies examining the effects of coconut oil compared to LCTs reported no increase in 124 satiety and no effect on food intake (38, 39) (Table 2). Poppit et al (39) found no difference in visual analogue scale ratings of satiety or differences in ad libitum food intake at lunch 125 following the consumption of either coconut oil (containing 10g MCT), high short chain 126 triglyceride (3g SCT, 7g MCT) (from soft fraction milk fat) or long chain triglycerides (from 127 128 tallow). Rizzo et al (38) found that in a dinner meal following ice-cream with varying 129 quantities of coconut oil there was trend towards reduced consumption with the coconut oil, however this was compensated for later when there was a significant increase in snack 130 consumption resulting in no overall difference between the ice-creams. The amounts of 131 coconut oil used here are 7.5g coconut oil (high dose) consisting of only ~4.8 g MCT 132 (carbons 6-12). This study should be commended on giving the coconut in a realistic form 133 (ice-cream) and amount. However compared to the amounts seen in the many of the MCT 134 studies the dose is very low. The authors conclude that the differences and trends observed 135 may reflect the differences in the absorption and metabolism of the two ice-creams. A third 136 study published across three papers providing 14 days of coconut oil, found no effect on 137 total energy expenditure or thermic effect of feeding. However they did find an increase in 138 basal metabolic rate after 7 days but not 14 days and an increase in endogenous long chain 139

saturated fatty acid oxidation after 14 days (19, 20, 24). In this study, caprylic and capric acid
made up 7.9% of the energy intake of the diet and lauric acid made up 17.7%.

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143 Currently there appears to be a lack of research in this area and to the best of the author's 144 knowledge there are no studies looking at the effect of coconut oil on weight loss and none comparing it to MCT oil. Further work is needed in this area to confirm these preliminary 145 calculations however indications would suggest that the use of coconut oil as a practical 146 147 means of increasing satiety are not credible. The research available on the use of coconut 148 oil on satiety and energy expenditure is limited and particularly there have been no long term clinical trials looking at the effects on weight loss. Given both the publicity and the increased 149 150 consumption of coconut oil further research in this area is warranted.

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Effects on satiety		
Study	Effect seen	Amount given
(Coleman, Quinn et al. 2016)	The MCT meal decreased food intake over the entire day. There significant differences in ad libitum food intake or satiety from VAS scores.	Smoothies with either 5 g CLA and 16g vegetable oil, 25 g MCT or 22 g vegetable oil
(Kovacs, Westerterp-Plantenga et al. 2001) (Kovacs, Westerterp-Plantenga et al. 2001) (Rolls, Gnizak et al. 1988)	2 weeks of with HCA & HCA combined with MCT did not ↑ satiety or ↓ El compared to PLA in subjects losing BW. HCA & HCA combined with MCT did not result in ↑ satiety, FO, 24 h EE or BW loss compared to PLA, in subjects losing BW. In the non-dieting group MCT at all doses decreased caloric intake in the lunch. Dieters were unresponsive to the type of dietary fat.	3 meals & four snacks daily with either no addition to diet (PLA), 500 mg HCA (HCA), or 500 mg HCA & 3 g MCT Controlled diet with no addition (PLA), 500 mg HCA (HCA), or 500 mg HCA & 3 g MCT for 2 weeks 3 doses (100, 200, and 300 kcal) of preloads of two complete liquid diets containing either 30% long-chain triglycerides (LCT) or 24% MCT with 6% LCT. This works out at 2.9g, 5.8g and 8.7g MCT.
(Stubbs and Harbron 1996)	EI was $\downarrow$ on the HMCT diet. Mean values were 13.50, 13.67, & 12.43 MJ/d on the LMCT, MMCT & HMCT diets. Food intake followed parallel. By day 14 BW changes amounted to +0.45, +0.41 & -0.03 kg, respectively.	3 HF diets for 3 days each. MCT to LCT was 1:2, 1:1 & 2:1 on the low-, medium- & high-MCT diets
(Van Wymelbeke, Himaya et al. 1998)	The addition of fats to the high-CHO breakfasts did not alter hunger but delayed the request for lunch compared with LF breakfast. Free-choice lunch was $\downarrow$ after MCT breakfast. Blood glucose & insulin were $\downarrow$ after the 3 fat breakfasts followed by larger $\uparrow$ in glucose & insulin 30 min after lunch.	4 high-CHO breakfasts (1670 kJ) supplemented either with a fat substitute (70 kJ) or 1460 kJ fat as monounsaturated LCT, saturated LCT or MCT.
(Van Wymelbeke, Louis- Sylvestre et al. 2001)	CHO oxidation was $\downarrow$ after the MCT & LCT lunches where FO was $\uparrow$ after the MCT & LCT lunches. The dinner request was delayed after the CHO lunch. FI at dinner was $\downarrow$ after the MCT lunch than after the Sub & CHO lunches, but the dinner meal request was not delayed.	1 lunch was a basic 2310-kJ meal containing 40 kJ fat substitute (Sub). The 3 other lunches were the same but contained 1200 kJ LCT, MCT or 900 kJ CHO + 300 kJ LCT
Effect on energy expenditure Study	Effect seen	Amount given
(Alexandrou, Herzberg et al.	Days 1 & 7 = between diets for resting metabolic rate or mean	45% CHO, 40% fat, 15% pro. The diets had either 60.81%
(Alexandrou, Herzberg et al. 2007) (Binnert, Pachiaudi et al. 1998)	postprandial EE. Days 1 & 7 FO was ↑ with MCT diet LCT oxidation was ↓ in obese & negatively correlated with fat mass. Plasma dietary TAG-derived LCFA were ↓ in the obese & negatively related to fat mass & positively to LCT oxidation. The proportion of MCTs oxidized was ↑ in both groups compared to LCTs	or 1.11% of fat energy from MCT. 30g of olive oil or $30 g - (50% olive oil, 50% MCT)$ mixed with lemon juice
(Clegg, Golsorkhi et al. 2013)	Differences in DIT existed between the chilli-MCT oil and chilli-	Breakfast containing either chilli and 20g MCT oil, chilli

(Nosaka, Maki et al. 2003)	The MCT diet demonstrated significant ↓ in BF, subcutaneous & visceral fat after 12 weeks. There were no differences in measured blood parameters	2100-2400 kcal/day including 65-73 g/day total fat (27.9- 31.2 energy %) Diet contain 14g test margarine with 5g MCT or LCT for 12 weeks
(Krotkiewski 2001)	MCT group had a greater decrease in body weight during the first 2 weeks and hunger feelings were less intense while satiety was higher. Differences were observed during the first 2 weeks of treatment and declined after that	Isoenergetic (578.5 kcal) VLCD) enriched with MCT or LCT (8.0 and 9.9 g/100 g) or a low-fat (3 g/100 g) and high-carbohydrate regimen
(Han, Deng et al. 2007	MCT group had across-time ↓ in BW, WC, insulin resistance & serum cholesterol as well as ↑ in serum C-peptide. LCT group did not. Changes were associated with an involuntary ↓ in EI.	Either MCT or corn oil (LCT). The test oil (18 g/d) was administered as part of daily food intake for 90 days
Effect on weight loss Study	Effect seen	Amount given
(St-Onge, Ross et al. 2003)	Upper body adipose tissue ↓ to a greater extent with functional oil compared olive oil. EE and FO was ↑ on day 2 with FctO compared with OL.	Weight maintaining diet. 40% of energy as fat, (75% primarily as functional oil or olive oil) for 28 days
	oil or 100% beef tallow) for 27 days	BC change with MCT consumption, although not statistically different, was consistent with the shifts in EE.
(St-Onge, Bourque et al. 2003)	LCT did not. Plasma TAG ↑ 68% after LCT but not MCT. Weight maintaining diet. 75% of fat as treatment fat (67% MCT	EE & FO were ↑ during MCT than LCT consumption. The
	LCT. MCT ↑ plasma beta-hydroxybutyrate & serum insulin but	(corn)
(Seaton, Welle et al. 1986)	insulin, & free fatty acids did not differ between meals Postprandial oxygen consumption ↑ 12% after MCT & 4% after	400 kcal meals with either 48g MCT oil & 45g LCT oil
(Scalfi, Coltorti et al. 1991)	PPT was ↑ in both groups after MCTs. Postprandial glucose,	or LCT 38 g LCT or 30 g MCTs plus 8 g LCT
(Kasai, Nosaka et al. 2002)	CHO & protein balances. However, after the LF meal lipid balance was negative PPT was ↑ after 5M5L & 10M compared to 10L. Ingestion of 5 g MCT caused ↑ PPT compared to LCT.	Study 1 meals had 10 g MCT (10M), 5 g MCT & 5 g LCT (5M5L) or 10 g LCT (10L). Study 2 meals had 5 g of MCT
(Flatt, Ravussin et al. 1985)	When MCT were consumed, plasma glucose & insulin were ↓ & plasma FFA ↑ during the first 2 h. Amounts of substrates oxidized was similar in each case, this resulted in comparable	482 kcal meal, 482 kcal meal plus 50 g of LCT margarine; or 482 kcal meal plus 40 g MCT & 10 g LCT margarine
(Dulloo, Fathi et al. 1996)	pepper-sunflower oil and pepper-MCT oil. EE ↑ with ↑ MCT:LCT ratio. No differences were observed in RQ or in urinary nitrogen losses, but 24-h urinary noradrenaline was ↑ with MCT.	Combinations of MCT & LCT totalling 30g/day consumed with habitual diet in 3 equal parts in the ratio of MCT: LCT (g/g) 0:30, 5:25, 15:15 & 30:0.
	sunflower oil, between chilli-MCT oil and pepper-sunflower oil and between pepper-sunflower oil and pepper-MCT oil. There was a significant difference in fat oxidation between the	and sunflower oil, bell pepper and sunflower oil or bell pepper and MCT oil

(St-Onge and Bosarge 2008)	MCT oil resulted in $\downarrow$ BW, trunk fat mass, total fat mass, & intraabdominal adipose tissue than olive oil.	18-24 g/d of MCT oil or olive oil as well as weekly weight loss counselling for 16 weeks
(St-Onge and Jones 2003)	BW ↓ 1.03 kg with MCT compared to 0.62 kg with LCT. Men with ↓ initial BW had a greater ↑ in EE with MCT consumption relative to LCT on day 28 but not day 2. Similar results for FO on day 28.	Diets rich in either MCT or LCT for 4 weeks
(Tsuji, Kasai et al. 2001)	BW & BF $\downarrow$ by wk 4, 8 & 12 in both groups. In volunteers with BMI $\geq$ 23 kg/m <sup>2</sup> BW $\downarrow$ more in the MCT than the LCT group, the loss of BF was $\uparrow$ in the MCT group than the LCT group at 8 wk. the change in area of subcutaneous fat was $\uparrow$ in the MCT than the LCT group at wk 4, 8 & 12.	9218 kJ/d & 60 g/d of total fat. The energy, fat, PRO & CHO intakes did not differ significantly between the groups. MCT group consumed 9.28g MCT per day for 12 weeks
(Yost and Eckel 1989)	Weight loss, serum ketones, & nitrogen balance were not different between groups. The MCT group ↑ their glucose requirement to maintain euglycemia during the clamp after weight loss whereas the LCT group had a ↓ requirement.	Hypocaloric MCT or LCT diets consisting of 47% CHO, 22% PRO & 31% fat. For the MCT group for 4 or 12 weeks
EE = energy expenditure	EI = energy intake	BC = body composition
BW = body weight	RQ = respiratory quotient	BF = body fat
WC = waist circumference	PPT = post prandial thermognesis	TEF = thermic effect of feeding
FO = fat oxidation	CHO = carbohydrate	BMR = basal metabolic rate
PRO = protein	f = female	m = male

Table 1: List of studies using medium chain triglycerides divided into effects on satiety, energy expenditure and weight loss and the effect seen and amount given.

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Study	Effect seen	Amount given
(Poppitt, Strik et al. 2010)	No significant effect of fatty acid chain length on satiety ratings or energy intake at an ad libitum meal.	SCT- (dairy fats), MCT- (coconut oil) and LCT- enriched (beef tallow) test breakfasts (3.3 MJ) containing 52 g lipid (58 en% fat). 15g coconut oil was used to provide 10g MCT.
(Rizzo, Masic et al. 2016)	Participants ate significantly less fat at dinner after the 75% coconut ice cream. There was no difference in intake at the dinner though a trend towards reduced consumption with increases in coconut oil. Calorie intake from snacks was found to be significantly lower after low coconut oil ice-cream, There was no effect of condition on satiety VAS data over the day.	10g of fat in ice-cream consisting of either 25% coconut oil and 75% sunflower oil; 50% coconut oil and 50% sunflower oil; 75% coconut oil and 25% sunflower oil
(Papamandjaris, White et al. 2000)	No difference in exogenous LCSFA as a function of diet on day 7. On day 14, $\uparrow$ LCSFA was observed in those fed the MCT vs LCT diet.	Weight maintaining diet. 80% of fat was either butter & coconut oil (MCT) or beef tallow (LCT) for 14 days
(Papamandjaris, White et al. 1999)	TEE on the MCT diet did not differ from the LCT diet. BMR was $\uparrow$ on the MCT diet on day 7, but not day 14.	Weight maintaining diets. 80% of fat was either 26% MCT & 74% LCT or 2% MCT & 98% LCT for 14 days.
(White, Papamandjaris et al. 1999)	On day 7 BMR was $\uparrow$ on the MCT compared to the LCT diet. EE on day 7 was $\uparrow$ with the MCT diet than the LCT diet. No differences in the TEF were evident between diets.	Weight maintaining diets. Each meal contained 40% energy as fat (80% of which was treatment fat) for 14 days

Table 2: List of studies examining the effects of coconut oil on satiety and energy expenditure and the effect seen and amount given.