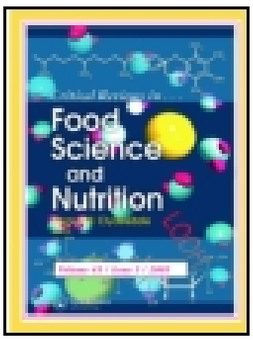

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REVIEW

A systematic review and meta-analysis of medium-chain triglycerides effects on acute satiety and food intake

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ABSTRACT

Research has indicated that consuming medium-chain triglycerides (MCT) may be more satiating than consuming long-chain triglycerides (LCT) potentially causing a reduction in energy intake. However not all studies have demonstrated this acute reduction in energy intake and it has yet to be systematically reviewed. Our main objective was to examine how ingestion of MCT influences energy intake, subjective appetite ratings and appetite-related hormones compared to LCT. Web of Science, MEDLINE, CINHAL, and Embase were searched for publications comparing the effect of MCT on appetite (commonly hunger, fullness, desire to eat, and prospective food consumption), appetite-related hormones (pancreatic polypeptide (PP), gastric inhibitory polypeptide (GIP), peptide YY (PYY), glucagon-like peptide-1 (GLP-1), neurotensin, leptin, total ghrelin and active ghrelin) and energy intake to LCT. A random-effects meta-analysis was conducted on studies which examined energy intake. Seventeen studies (291 participants) were included in the systematic review, of which 11 were included in the energy intake meta-analysis. Synthesis of combined data showed evidence of a statistically significant moderate decrease in *ad libitum* energy intake after both acute and chronic ingestion of MCT compared to LCT when assessed under laboratory conditions (mean effect size: −0.444, 95% CI −0.808, −0.080, $p < 0.017$), despite little evidence of any effect of MCT on subjective appetite ratings or circulating hormones. The current evidence supports the notion that MCT decreases subsequent energy intake, but does not appear to affect appetite. Further research is warranted to elucidate the mechanisms by which MCT reduce energy intake.

KEYWORDS

Medium-chain triglycerides; satiety; appetite; energy intake; systematic review; meta-analysis

Introduction

Overweight and obesity are defined as the accumulation of excess body fat which may lead to impaired health (World Health Organisation 2018). Despite the well-reported risks of increased body fat, including type 2 diabetes, coronary heart disease, some cancers, and stroke (National Statistics 2016), overweight and obesity are still increasingly prevalent. In 2016, more than 1.9 billion adults were overweight globally and 650 million of these were obese; figures which have nearly tripled since 1975 (World Health Organisation 2018). These conditions are caused by a chronic energy surplus from either excessive energy intake or inadequate energy expenditure (Hill, Wyatt, and Peters 2012). It is known that adherence to dietary interventions aiming to reduce bodyweight is low due to feelings of hunger (Franklin et al. 1948), meaning that the target weight loss is not always achieved.

As a result of this, foods with enhanced satiety have gained much attention, both commercially and in research (Chambers, McCrickerd, and Yeomans 2015; Hetherington et al. 2013). Medium-chain triglycerides (MCT) are triglycerides with shorter chain lengths (6-12 carbon atoms long) than 'traditional' long-chain triglycerides (LCT; 12+ carbon atoms long). Due to the shorter chain length of MCT, its consumption results in attenuated release of cholecystokinin (CCK) compared to LCT (Feinle et al. 2001; Feltrin et al. 2007, 2006; French et al. 2000; Matzinger 2000). CCK is involved in lipid-related satiety (McLaughlin et al. 1999), and thus LCT promote satiety via this mechanism. However, MCT are absorbed much quicker than LCT (Marten, Pfeuffer, and Schrezenmeir 2006) which leads to large amounts of β-oxidation (Bach and Babayan 1982) and the production of β-hyroxybutyrate (Page et al. 2009); a process which is thought to be anorexigenic (Laeger, Metges, and Kuhla 2010; Scharrer 1999). Studies have shown decreased appetite and subsequent energy intake after a preload (Rolls

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Data described in the manuscript, code book, and analytic code will be made available upon request.
PROSPERO database: registration number: CRD42018092550.

Table 1. Keywords included in database search strategy.

Medium chain Triglycerides	Satiety		Human
MCT	Appetite	Diar*	*In vivo*
Medium chain fatty	Hunger	*Ad lib**	Man
MCFA	Satiety	Calori* intake	Woman
Caproic	Fullness	Energy density	Men
C6:0	Desire to eat	Hormon*	Women
Hexanoic	Prospective food consumption	Peptide*	Volunteer*
Caprylic	Nausea	Gut	Participant
C8:0	Time to meal request	Peptide YY	Lean
C10:0	TTMR	Tyrosine	Healthy
Lauric	Motivation to eat	PYY	Overweight
C12:0	Palatability	Ghrelin	Obese
Dodecanoic	Food reward	Acylated	
Coconut*	Hedonic	GLP*	
Palm kernel oil	Liking	Glucagon-like peptide*	
	Wanting	Pancreatic polypeptide	
	Food intake	Oxyntomodulin	
	Food consumption	OXM	
	Energy intake	Cholescystokinin	
	Compensation	CKK	
	Protein intake	Leptin	
	Fat intake	Ketone*	
	Carbohydrate intake	Beta-hydroxybutyrate	
	Macronutrient*	Butyrate	
	Diet	BHB	
	Diary	Satiety hormone*	

et al. 1988), breakfast (Coleman, Quinn, and Clegg 2016; Kinsella, Maher, and Clegg 2017; Wymelbeke et al. 1998) or lunch (Wymelbeke, Louis-Sylvestre, and Fantino 2001) containing MCT. This is not a universal finding however, as some studies have reported no difference in energy intake after meals containing MCT or a control oil (Poppitt et al. 2010; St-Onge et al. 2014), and have even shown increased feelings of hunger after MCT-based meals (Valente et al. 2018).

Clearly, the findings surrounding MCT and satiety are mixed. It is therefore important to systematically determine whether MCT ingestion results in greater satiety and decreased energy intake. Thus, this review aims to assess if there is sufficient evidence to support the hypothesis that MCT can increase satiety in comparison with LCT. Specifically, the objectives are to examine if the consumption of MCT decreases energy intake in subsequent eating episodes, if MCT ingestion favorably alters subjective sensations of appetite (i.e. increased fullness and decreased hunger/desire to eat), and to compile the data on the effects of MCT on circulating hormones involved in appetite regulation.

Methods

This review is reported according to the PRISMA guidelines (Moher et al. 2009), and is registered in the PROSPERO database (registration number: CRD42018092550).

Search strategy

The research question of this systematic review was formulated using PICOS (Population, Intervention, Comparison, Outcome, Setting). The population was defined as adults of healthy status excluding overweight or obesity. The intervention was considered to be any investigation examining medium-chain triglycerides or medium-chain fatty acids on appetite and satiety measures. Outcomes incorporated any measure of appetite (i.e. visual analog scales), physiological markers of appetite regulation (e.g. PYY, ghrelin) and energy intake measures (*ad libitum* meals, diet diaries). There was no restriction to the settings in which studies were conducted.

The databases Web of Science, MEDLINE, CINHAL and Embase were searched for studies in the English language between 1970 and 2018 comprising of all human participants using the strategy ("medium chain triglycerides" AND "satiety" AND "human"). The last search was run on 14 May 2018. Previous systematic reviews were screened to identify relevant subject headings and key words to include within each subject category. Reference lists from the resulting articles were also screened to identify any additional articles. Table 1 shows a full list of the specific key words.

Exclusion criteria

Studies were excluded if they did not examine MCT or medium chain fatty acids (or a product containing either) and subjective measurement of appetite sensations or energy or food intake. Studies were also excluded if they were conducted in animals, or if they contained individuals outside the age range of 18-70 years, or if they did not include an LCT arm that was matched in calories and composition to an MCT arm.

Data screening

Records were screened for duplicates, which were removed. Potential studies were identified by examining all titles and removing those which did not contain reference to MCT and appetite or energy intake by one reviewer. The abstracts of the remaining titles were read, and full text copies were obtained if they still met the initial criteria. Information on the remaining studies after abstract screening was tabulated by one researcher (TM), and both investigators (TM and MC) discussed the inclusion of the studies until a mutual

Table 2. Analysis of bias using the Cochrane Collaboration's Tool for assessing risk of bias.

Study	Random sequence generation	Allocation concealment	Blinding of participants	Blinding of personnel	Blinding of outcome assessment	Incomplete outcome data	Selective reporting	Other sources of bias
Barbera (2000)	Unclear	High	Low	Unclear	High	High	High	Unclear
Clegg, Golsorkhi, and Henry(2013)	Unclear	High	Unclear	Unclear	High	High	High	Unclear
Clegg et al. (2012)	Unclear	High	Low	High	High	High	High	Unclear
Coleman, Quinn, and Clegg (2016)	Unclear	High	Low	High	High	High	High	Unclear
Feltrin et al. (2008)	Unclear	Low	Low	Low	Low	High	High	Unclear
Feltrin et al. (2004)	Unclear	Low	Low	Low	Low	High	High	Unclear
Kinsella, Maher, and Clegg (2017)	Unclear	High	Low	High	High	High	High	Unclear
Krotkiewski (2001)	High	High	Low	Low	Low	High	High	Unclear
Poppitt et al. (2010)	Unclear	High	Low	High	High	High	Unclear	Low
Rizzo et al. (2016)	Unclear	High	Low	High	High	High	High	Unclear
Rolls et al. (1988)	Low	High	Low	Low	Low	High	High	Unclear
St-Onge et al. (2014)	Low	High	Low	High	High	Low	Low	Unclear
Stubbs and Harbron (1996)	Unclear	High	Unclear	Unclear	Unclear	Unclear	High	Unclear
Valente et al. (2018)	Low	High	Low	High	High	Low	Low	Unclear
Van Wymelbeke et al. (1998)	Low	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear
Van Wymelbeke, Louis-Sylvestre, and Fantino (2001)	Low	High	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear

consensus was met. The following information was extracted from the included into a spreadsheet: authors, date of publication, sample size, participant characteristics (age, sex, body mass index [BMI]), study setting, source and amount of MCT, appetite outcome measures and results.

Quality checks

Risk of bias was assessed within the individual studies using the Cochrane Collaboration's Tool (Higgins et al. 2011). Selection bias, reporting bias, performance bias, detection bias, attrition bias and other sources of bias (such as funding etc.) were assessed. Eligible studies were included regardless of risk of bias. Table 2 details risk of all sources of bias for each study.

Meta-analysis procedures

Due to inconsistent reporting of visual analog scale data (i.e. presented in a variety of ways, graphical format, as raw data or calculated AUC), and the small number of studies examining appetite hormones (four), only acute *ad libitum* energy intake data was included in the meta-analysis (either at a single meal or over the course of a whole day). The remaining 11 studies were broken down into 20 subgroups, accounting for studies investigating multiple doses of MCT (Rolls et al. 1988; Stubbs and Harbron 1996) or coconut oil (Rizzo et al. 2016), and for studies with multiple investigations (St-Onge et al. 2014). Energy intake (kJ) was measured at both *ad libitum* meals and habitual daily intake. Where needed, reported values were converted to kJ before computation to standardize the units. Meta-analysis software (Comprehensive Meta-Analysis, Version 3, Biostat, Englewood, NJ, USA) was used to conduct a meta-analysis on extracted data. Data inputted included sample sizes, mean energy intake for LCT and MCT trials, their respective SDs, and a correlation coefficient to account for the fact that the included studies were crossover trials ($r = 0.940$, calculated from energy intake data from the studies included in the review). The software computed effect sizes for each study, as well as an overall effect size using a random-effects model (DerSimonian-Laird inverse variance approach). Effect size was calculated as the standardized difference in means, which we interpreted to be trivial at <0.2, small at 0.2–0.3, moderate at 0.4–0.8, and large at >0.8, as per Cohen (Cohen 1992). Negative effect sizes indicate decreased consumption in MCT trials/conditions, whereas positive effect sizes indicated LCT led to decreased energy intake. Publication bias was assessed utilizing funnel plots and by quantifying Egger's regression intercept. A significant regression indicates the presence of a small study effect (Sterne, Egger, and Moher 2011).

Results

Descriptive

The database search yielded 4,547 results, which was reduced to 3,517 after the removal of duplicates. After the screening of titles and abstracts, 3,302 were removed. Of the remaining 216 texts, 17 satisfied the inclusion criteria (Figure 1).

Seven studies were conducted in the UK (Clegg, Golsorkhi, and Henry 2013; Clegg et al. 2012; Coleman, Quinn, and Clegg 2016; Kinsella, Maher, and Clegg 2017; Rizzo et al. 2016; Stubbs and Harbron 1996), two in Australia (Feltrin et al. 2004, 2008), two in France (Wymelbeke, Louis-Sylvestre, and Fantino 2001; Wymelbeke et al. 1998), two in the US (Rolls et al. 1988; St-Onge et al. 2014), and one each in Italy (Barbera 2000), Sweden (Krotkiewski 2001), Brazil (Valente et al. 2018), and New Zealand (Poppitt et al. 2010). Participants were 28.57 ± 6.20 years of age with a BMI of $23.49 \pm 3.42\ kg/m^2$, and there was an average of 15 ± 8 participants per study (means ± SD). Participants in one study were classified as 'overweight' according to BMI (M. St-Onge et al. 2014), and were classified as 'obese' in one other (Krotkiewski 2001); all others were in the 'normal' BMI category. There was a total of 291 participants, of which 107 were male and 184 were female. There were 11 acute feeding studies (Clegg, Golsorkhi, and Henry 2013; Clegg et al. 2012; Coleman, Quinn, and Clegg 2016; Kinsella, Maher, and Clegg 2017; Poppitt et al. 2010; Rizzo et al. 2016; Rolls et al. 1988; St-Onge et al. 2014; Valente et al. 2018; Wymelbeke, Louis-Sylvestre, and Fantino 2001; Wymelbeke et al. 1998), three

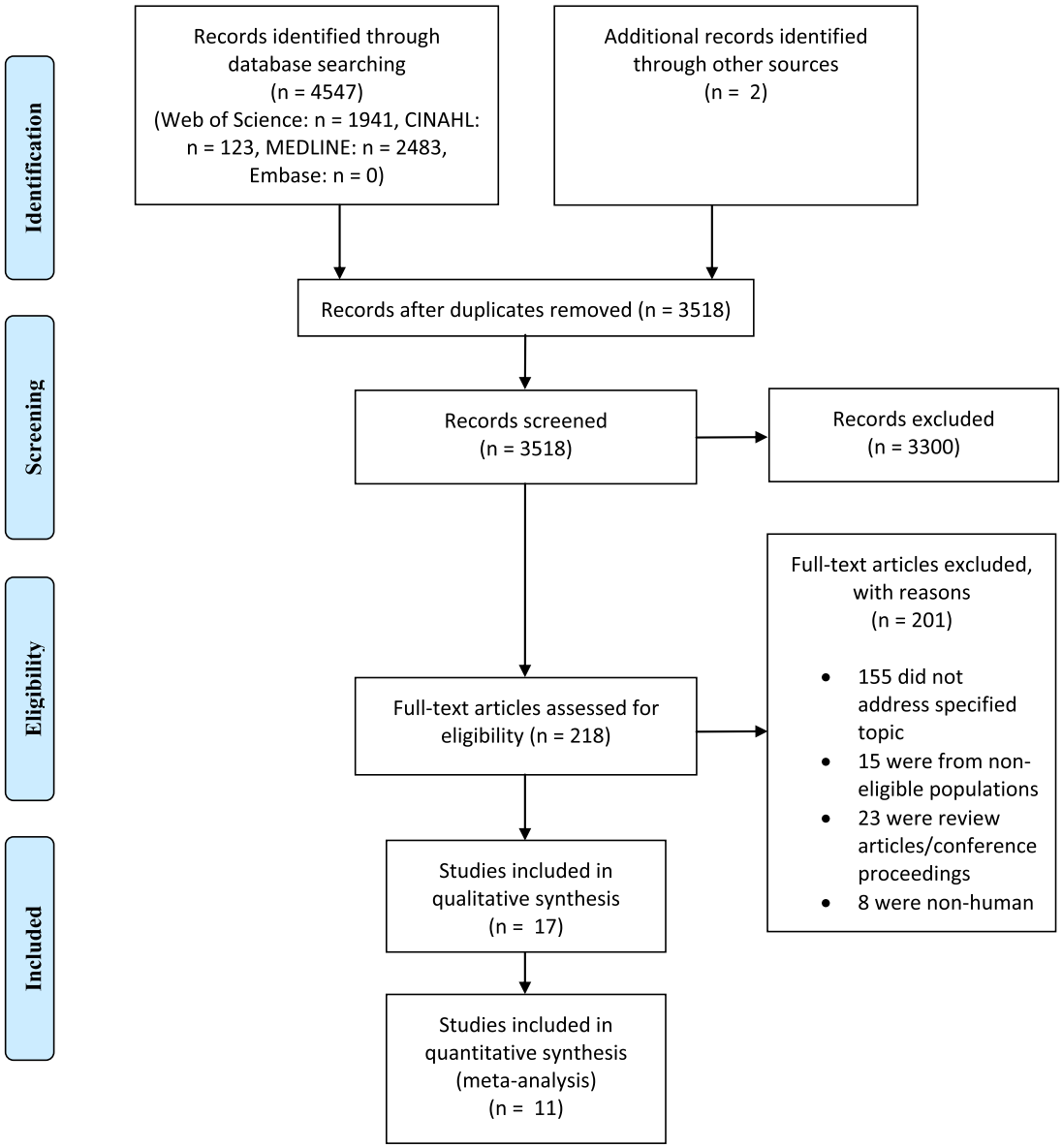

Figure 1. Flowchart of methodology used for identifying studies included in the systematic review.

acute infusion studies (Barbera 2000; Feltrin et al. 2008, 2004), and two chronic dietary intervention studies, of which one examined participants three times across all arms of the intervention and quantified habitual daily energy intake (Stubbs and Harbron 1996) and the other was a comparison of independent matched groups (Krotkiewski 2001). One dietary intervention provided all foods consumed by participants in 14-day long manipulations, where the amount of energy from MCT was altered (Stubbs and Harbron 1996), and the other was a very low calorie diet, with either MCT or LCT was incorporated into the low-calorie formula incorporated into the diet (Krotkiewski 2001).

Measures

Fourteen out of fifteen studies used 100 mm visual analog scales to measure subjective sensations of appetite (St-Onge et al. 2014). Ten studies examined energy intake during at least one subsequent *ad libitum* eating episode after consumption of a meal/preload containing LCT/MCT (Coleman, Quinn, and Clegg 2016; Feltrin et al. 2004, 2008; Kinsella, Maher, and Clegg 2017; Poppitt et al. 2010; Rizzo et al. 2016; Rolls et al. 1988; St-Onge et al. 2014; Wymelbeke, Louis-Sylvestre, and Fantino 2001; Wymelbeke et al. 1998), and one examined daily habitual energy intake after MCT was covertly incorporated into the diet (Stubbs and Harbron 1996). In that study, participants were required to consume all meals in the laboratory, but were allowed to leave and were not required to 'live' in the laboratory; and thus, ad libitum daily energy intake was quantified. Three of those also included diet diaries for subsequent energy intake (Coleman, Quinn, and Clegg 2016; Kinsella, Maher, and Clegg 2017; Wymelbeke et al. 1998). Four studies examined appetite hormones, including pancreatic polypeptide (PP) (Barbera 2000), CCK (Barbera 2000; Feltrin et al. 2004, 2008), gastric inhibitory polypeptide (GIP) (Barbera 2000; Feltrin et al. 2004), peptide YY (PYY) (Feltrin et al. 2008; St-Onge et al. 2014), leptin (St-Onge et al. 2014), glucagon-

like peptide-1 (GLP-1) (Feltrin et al. 2004) and both active and total ghrelin (St-Onge et al. 2014).

Test lipids

The main results of included studies are shown in Table 3. Six studies directly compared MCT to LCT, which acted as a control (Barbera 2000; Clegg, Golsorkhi, and Henry 2013; Feltrin et al. 2004; Rolls et al. 1988; St-Onge et al. 2014; Stubbs and Harbron 1996). Two studies compared MCT and LCT, and also included a low-fat/no-fat control (Feltrin et al. 2008; Krotkiewski 2001; Wymelbeke, Louis-Sylvestre, and Fantino 2001). Three studies had multiple fats, including several LCT conditions such as sunflower oil, olive oil and butter (Clegg et al. 2012); olive oil and lard (Wymelbeke et al. 1998); and two with another test oil, which was conjugated linoleic acid (Coleman, Quinn, and Clegg 2016) and short-chain triglycerides (Poppitt et al. 2010). Two studies used coconut oil as the source of MCT in the study (Rizzo et al. 2016; Valente et al. 2018), and another study used coconut oil as well as MCT (Kinsella, Maher, and Clegg 2017). For the LCT trials and controls, three studies used sunflower oil (Clegg, Golsorkhi, and Henry 2013; Clegg et al. 2012; Rizzo et al. 2016), two used rapeseed oil (Coleman, Quinn, and Clegg 2016; Kinsella, Maher, and Clegg 2017), two used corn oil (Rolls et al. 1988; St-Onge et al. 2014), one used beef tallow (Poppitt et al. 2010), one used extra virgin olive oil (Valente et al. 2018), one used margarine (Wymelbeke, Louis-Sylvestre, and Fantino 2001), one study used an unspecified vegetable oil (Stubbs and Harbron 1996), and three studies (which administered the lipids via infusion and not feeding) used emulsions of oleic and linoleic acid (Barbera 2000), oleic acid (Feltrin et al. 2008), and lauric acid (Feltrin et al. 2004). One study did not specify the LCT used in their study (Krotkiewski 2001). In terms of saturation of LCT, six studies utilized LCT with a mixture of polyunsaturated and monounsaturated acids (Barbera 2000; Clegg, Golsorkhi, and Henry 2013; Rizzo et al. 2016; Rolls et al. 1988; St-Onge et al. 2014; Valente et al. 2018), four used purely monounsaturated fatty acids (Coleman, Quinn, and Clegg 2016; Feltrin et al. 2008; Kinsella, Maher, and Clegg 2017; Stubbs and Harbron 1996), two used mixtures of monounsaturated and saturated fatty acids (Poppitt et al. 2010; Wymelbeke, Louis-Sylvestre, and Fantino 2001), and two studies used multiple sources of LCT; polyunsaturated and monounsaturated (sunflower oil), monounsaturated (olive oil) and saturated (butter) fatty acids (Clegg et al. 2012), and saturated (lard) or monounsaturated (olive oil) (Wymelbeke et al. 1998). When accounting for studies that provided multiple doses, the dosage of MCT ranged from 10 g (Poppitt et al. 2010) to 42.4 g (Wymelbeke et al. 1998), with an average dose of 23.8 g. Three studies provided 10–15 g (Poppitt et al. 2010; Rizzo et al. 2016; Rolls et al. 2004), four provided 20–25 g (Coleman, Quinn, and Clegg 2016; Kinsella, Maher, and Clegg 2017; Rolls et al. 2004; St-Onge and Jones 2002), three provided 30–35 g (Rolls et al. 1988; St-Onge et al. 2014; Wymelbeke, Louis-Sylvestre, and Fantino 2001) and one provided 40–45 g (Wymelbeke et al. 1998).

Outcomes

One out of 11 studies (Feltrin et al. 2004) reported decreased energy intake at an *ad libitum* meal after MCT compared to LCT, although this was only significant in seven studies (Coleman, Quinn, and Clegg 2016; Feltrin et al. 2008; Rolls et al. 1988; St-Onge et al. 2014; Stubbs and Harbron 1996; Wymelbeke, Louis-Sylvestre, and Fantino 2001; Wymelbeke et al. 1998). The one study that reported decreased intake after LCT compared to MCT reported a significantly lower energy intake after LCT compared to MCT (Feltrin et al. 2004). The average energy intake at the *ad libitum* meal in that study after LCT and MCT trials, respectively, was 1,747 ± 633 kJ and 4,109 ± 589 kJ. Five studies out of 14 reported significant differences in appetite ratings, which were decreased hunger and increased satiety after MCT (Krotkiewski 2001), increased fullness after MCT (Kinsella, Maher, and Clegg 2017), increased satiety after LCT (Barbera 2000), decreased hunger but also decreased desire to eat after infusion of MCT (Feltrin et al. 2004), and increased hunger and decreased fullness after MCT (coconut oil) (Valente et al. 2018). Three studies (Barbera 2000; Feltrin et al. 2004; Rolls et al. 1988) reported significant adverse effects, which manifested as gastric aching after the MCT drinks (Rolls et al. 1988), and increased nausea after infusions LCT compared to MCT (Barbera 2000; Feltrin et al. 2004).

Only four studies examined blood parameters in response to the oils which showed LCT led to increased postprandial concentrations GIP, neurotensin, PP (Barbera 2000), CCK (Barbera 2000; Feltrin et al. 2008, 2004), PYY (Feltrin et al. 2008) and GLP-1 (Feltrin et al. 2004). Conversely, one study showed that relative to MCT, LCT led to increased postprandial leptin and PYY, and no effect on GLP-1 or total ghrelin, but active ghrelin concentrations were reduced to a lesser extent after MCT (St-Onge et al. 2014).

Meta-analysis

Due to high levels of heterogeneity ($I^2 = 97.0\%$, $Q = 333.9$, $T^2 = 0.355$, $d_f = 10$), a random effects model was chosen (Ades, Lu, and Higgins 2005). Effect size for acute *ad libitum* energy intake ranged from −2.235 to 3.789. Statistics for each individual study are reported in Supplementary Table 1. There was a statistically significant moderate decrease in *ad libitum* energy intake after MCT ingestion compared to LCT ingestion (mean effect size: −0.444, 95% confidence intervals −0.808 to −0.0.80, $N = 11$, $p = 0.017$; Figure 2). Sensitivity analysis showed that the removal of each study had only minor effects on overall effect size, and no effect on significance. In order to further examine and specify the effect of consuming MCT on satiety, a sensitivity analysis was conducted by removing infusion studies. This did not alter the direction of significance, but it did increase the level of significance (mean effect size: −0.681, 95% confidence intervals −0.950 to −0.412, $N = 8$, $p < 0.001$). More sensitivity analyses were conducted in order to specify the effect of MCT without the influence of coconut

Table 3. Characteristics and main outcomes of studies comparing LCT to MCT included in systematic review.

References	Participants			Study information				Outcomes		
	n	Gender (M/F)	Characteristics	Study design	Lipids used	Test meal	Time between test meal and *ad libitum* meal	Energy Intake	Postprandial Appetite Method – analysis Parameters	Hormone Measures
Barbera (2000)	9	6/3	Age: 20-56 y	Randomized crossover duodenal infusion study	0.9% Saline *then*20% LCT emulsion 20% MCT emulsion	Infusions Total energy: 250.6 kJ	NA	NA	VAS – mm Duration: immediately after infusions Satiation* ↑ in LCT Fullness Abdominal Bloating Nausea* ↑ in LCT Pain	Somatostatin PP* ↑ in LCT CCK* ↑ in LCT GIP* ↑ in LCT Neurotensin* ↑ in LCT
Clegg, Golsorkhi, and Henry (2013)	7	1/6	Age: 26 ± 4 y W: 62.5 ± 7.5 kg H: 1.69 ± 0.09 m	Randomized acute crossover postprandial feeding study	Sunflower oil (18.40 g) MCT oil (20.00 g	Fried breakfast containing: Pepper and sunflower oil Pepper and MCT oil Chili and sunflower oil Chili and MCT oil Total energy: 449 kJ	NA	NA	Duration: 240 min Hunger Fullness Desire to Eat Prospective Food Consumption *Gastrointestinal comfort* Nausea Belching Bloatedness Headache Cramping	NA
Clegg et al. (2012)	10	2/8	Age: 26 ± 4 y W: 61.8 ± 9.6 kg H: 1.70 ± 0.09 m	Randomized acute crossover postprandial feeding study	Sunflower oil (24.40 g) Olive Oil (24.40 g) Butter (26.90 g) MCT oil (24.30 g) Control (no oil added)	Pancakes Total energy: 2523 kJ	NA	NA	Duration: 240 min Hunger Fullness Desire to Eat Prospective Food* ↑ in butter compared to LCT Consumption *No other differences between MCT and any LCT condition*	NA
Coleman, Quinn, and Clegg (2016)	19	7/12	Age: 31 ± 18 y W: 68.6 ± 11.7 kg H: 1.69 ± 0.11 m	Randomized acute crossover postprandial feeding study	Vegetable oil (22.00 g) CLA and vegetable oil (5.00 g CLA, 16.00 g vegetable oil) MCT (25.00 g)	Mango and passionfruit smoothie Total energy: 1323 kJ	300 min	*Ad libitum* lunch CON: 3343 ± 870 kJ CLA: 3051 ± 789 kJ MCT: 3051 ± 766 kJ Diet diary CON: 4905 ± 1919 kJ* CLA: 2931 ± 1346 kJ* MCT: 2708 ± 1314 kJ Total days intake CON: 8248 ± 2790 kJ CLA: 5891 ± 2135 kJ* MCT: 5759 ± 2080 kJ*	Duration: 60 min Hunger Fullness Desire to Eat Prospective Food Consumption	NA
Feltrin et al. (2008)	13	13/0	Age: 26 ± 2 y BMI: 22.9 ± 0.6 kg/m^2	Randomized double-blind crossover duodenal infusion study	0.9% saline (control) Oleic acid (C18, 4.88 g) Lauric acid (C12, 4.52 g)	Infusions at a rate of 1.569 kJ min^{-1} Total energy: 100 kJ	60 min	*Ad libitum* brunch CON: 5293 ± 385 kJ C18: 4745 ± 335 kJ C12: 5226 ± 301 kJ†	VAS – mm Duration: 60 min Hunger Fullness Nausea Bloating	CCK*† ↑ in both C18 and C12 compared to control ↑ in C18 compared to C12 PYY*† ↑ in both C18 and C12 compared to Con. ↑ in C18 compared to C12
Feltrin et al. (2004)	8	8/0	Age: 24 ± 4 y BMI: 22.0 ± 1.6 kg/m^2	Randomized double-blind crossover duodenal infusion study	CON (distilled water) Decanoic acid (C10) Lauric acid (C12)	Infusions at a rate of 1.569 kJ min^{-1} Total energy: 141 kJ	90 min	*Ad libitum* brunch Control: 4604 ± 464 kJ C10: 4109 ± 589 kJ C12: 1747 ± 633 kJ†	VAS – mm Duration: 90 min Hunger† ↓ in Lauric acid Fullness Desire to eat† ↓ in Lauric acid Prospective Food Consumption Nausea Bloating	CCK*† ↑ in both C10 and C12 compared to control ↑ in C12 compared to C10 GIP* ↑ in C12 compared to both C10 and control.
Kinsella, Maher, and Clegg (2017)	24	6/18	Age: 28 ± 6 y W: 64.5 ± 8.5 kg H: 1.68 ± 0.07 m BMI: 22.9 ± 2.4 kg/m^2	Randomized acute crossover postprandial feeding study	CON: Vegetable oil (23.00 g) CO (26.00 g) MCT oil (25.00 g)	*Mango and passionfruit smoothie* Total energy: 1456 kJ	180 min	*Ad libitum* lunch CON: 7023 ± 2084 kJ CO: 6738 ± 2099 kJ* MCT: 6011 ± 2397 kJ† Total days intake CON: 12518 ± 2995 kJ CO: 11338 ± 2284 kJ* MCT: 10722 ± 3841 kJ*	VAS – AUC Duration: 180 min Hunger Fullness† ↑ in MCT Desire to Eat Prospective Food Consumption	NA

Krotkiewski (2001)	66	0/66	*Low-fat group* Age: 45 ± 3 y W: 94.6 ± 2.8 kg H: 1.69 ± 0.04 m BMI: 33.1 ± 0.6 kg/m^2 *LCT group* Age: 43 ± 4 y W: 95.6 ± 2.4 kg H: 1.68 ± 0.04 m BMI: 34.1 ± 0.5 kg/m^2 *MCT group* Age: 43 ± 4 y W: 95.5 ± 2.6 kg H: 1.69 ± 0.04 m BMI: 34.1 ± 0.3 kg/m^2	Group matched 4-week dietary intervention	*Very low-calorie diet (2420.9 kJ/d) with either* Low fat (control) (2.70 g) LCT (7.92 g) MCT (8.91 g)	NA	NA	NA	Duration: 120 min Hunger* ↓ in MCT Fullness* ↑ in MCT Appetite* ↓ in MCT Prospective Food Consumption	NA
Poppitt et al. (2010)	18	18/0	Age: 27 ± 9 y W: 73.4 ± 10.0 kg BMI: 22.8 ± 1.8 kg/m^2	Randomized acute crossover postprandial feeding study	High-LCT: Beef tallow (52 g) High-MCT: Coconut oil (10 g coconut oil, 42 g LCT) High-SCT: Soft fraction milk fat (3 g SCT, 7 g MCT, 42 g LCT)	Savoury breakfast muffins Total energy: 3315 kJ	210 min	*Ad libitum* lunch High-LCT: 4490 ± 324 kJ High-MCT: 4422 ± 306 kJ High-SCT: 4406 ± 366 kJ	VAS – mm Duration: 390 min Hunger Fullness Prospective Food Consumption, Nausea	NA
Rizzo et al. (2016)	36	0/36	Age: 29.7 ± 4 y W: 66.4 ± 4.9 kg H: 1.50 ± 0.06 m BMI: 21.7 ± 0.1 kg/m^2	Randomized acute crossover postprandial feeding study	High CO: 75%CO:25%SO Equal: 50:50 High SO: 75%SO:25%CO	*Ice cream containing different ratios of CO and SO* Total energy: 1130 kJ	45 min	*Ad libitum* dinner High-CO: 7881 ± 504 kJ Equal: 8190 ± 503 kJ High-SO: 8287 ± 518 kJ *Ad libitum* snacks High-CO: 2069 ± 276 kJ* Equal: 1548 ± 180 kJ High-SO: 1573 ± 201 kJ	VAS - AUC Duration: 480 min Hunger Fullness Desire to Eat Prospective Food Consumption Satisfaction	NA
Rolls et al. (1988)	24	0/24	*Dieters* Age: 27 ± 2 y W: 68.9 ± 4.5 kg H: 1.69 ± 0.02 m *Non-dieters* Age: 28 ± 2 y W: 55.3 ± 1.7 kg H: 1.67 ± 0.01 m	Randomized acute crossover postprandial feeding study	418.4 kJ (11.11 g LCT, 12.04 g MCT) 836.8 kJ (22.22 g LCT, 24.09 g MCT) 1255.2 kJ (33.33 g LCT, 31.14 g MCT)	*30% fat liquid preload. Three doses of each* Energy density of the drinks: 3.47 kJ/100 mL	30 min	*Ad libitum* lunch (non-dieters) LCT: 418.4 kJ: 563.2 ± 54.6 g 836.8 kJ: 495.6 ± 64.4 g 1255.2 kJ: 510.3 ± 71.5 g MCT: 418.4 kJ: 535.3 ± 71.2 g* 836.8 kJ: 455.7 ± 56.4 g* 1255.2 kJ: 413.5 ± 52.1 g* *Ad libitum* lunch (dieters) 418.4 kJ: 590.5 ± 38.1 g 836.8 kJ: 466.8 ± 73.8 g 1255.2 kJ: 483.3 ± 65.3 g MCT: 418.4 kJ: 539.6 ± 66.0 g 836.8 kJ: 446.8 ± 47.2 g 1255.2 kJ: 432.8 ± 42.1 g	VAS – mm Duration: 30 min Hunger Desire to Eat Stomach Fullness Gastric aching* ↑ in MCT	NA
St-Onge et al. (2014)	17	*Study 1* 10/0 *Study 2* 7/0	*Study 1* Age: 29.7 ± 4 y W: 87.1 ± 1.7 kg H: 1.76 ± 0.01 m BMI: 28.2 ± 0.01 kg/m^2 *Study 2* Age: 39.6 ± 2.1 y W: 91.9 ± 5.1 kg H: 1.79 ± 0.04 m BMI: 28.4 ± 0.5 kg/m^2	Two randomized, acute crossover postprandial feeding studies	*Study 1:* LCT: 20.0 g MCT: 20.0 g *Study 2:* 3h after breakfast LCT: 10.0 g MCT: 10.0 g	*Study 1* Muffin and orange juice Total energy: 2671 kJ *Study 2* Liquid meal replacement, and a yoghurt preload Total energy: 2510 kJ (breakfast) 728 kJ (preload)	*Study 1* 180 min *Study 2* 60 min	*Ad libitum* lunch Study 1 LCT: 2773 ± 532 kJ MCT: 2548 ± 457 kJ Study 2 LCT: 3369 ± 769 kJ MCT: 2227 ± 616 kJ*	NA	*Study 1* No significant differences *Study 2* Active ghrelin* ↑ in MCT Leptin* ↑ in MCT *Combined analysis* Leptin* ↑ in MCT PYY* ↑ in MCT

(*continued*)

Table 3. Continued.

References	Participants			Study information				Outcomes		
	n	Gender (M/F)	Characteristics	Study design	Lipids used	Test meal	Time between test meal and *ad libitum* meal	Energy Intake	Postprandial Appetite Method – analysis Parameters	Hormone Measures
Stubbs and Harbron (1996)	6	6/0	Age: 27 ± 4 y W: 63.3 ± 7.3 kg H: 1.72 ± 0.05 m	Randomized crossover study, each arm consisting of a 14-day dietary manipulation	High energy (639 kJ/100 g) diets containing 62:28:10 percentage fat, carbohydrate and fat. Ratios of MCT:LCT were: Low MCT: 1:2 (35.8 g/1000g of food) Medium MCT: 1:1 (55.5 g MCT per 1000g of food) High MCT: 2:1 (73.2 g MCT per 1000g of food)	NA	NA	*Daily habitual energy intake* Low MCT: 13.5 ± 1.8 MJ Medium MCT: 13.7 ± 1.6 MJ High MCT: 12.4 ± 1.5 MJ†	VAS – mm Duration: 15 min after each meal Pleasantness of food Satisfaction* ↑ In High MCT	NA
Valente et al. (2018)	15	0/15	Age: 27 ± 1 y BMI: 27.7 ± 0.4 kg/m^2	Randomized acute crossover postprandial feeding study	25.00 mL EVOO 25.00 mL VCO	White bread and a strawberry milkshake Total energy: 2067 kJ	NA	NA	VAS – iAUC Duration: 240 min Hunger* ↑ in VCO Fullness* ↓ in VCO Satisfaction* ↓ in VCO Desire to eat something sweet Desire to eat something salty Desire to eat something savoury Desire to eat something fatty	NA
Van Wymelbeke et al. (1998)	12	12/0	Age: 21 ± 2 y BMI: 21.7 ± 1.6 kg/m^2	Randomized acute crossover postprandial feeding study	70 kJ fat substitute *or* 1460 kJ from different fats: Saturated LCT (from 42 g lard) Monounsaturated LCT (from 40 g olive oil) MCT (from 43 g of Ceres MCT oil)	Pasta and tomato sauce Total energy: 3130 kJ (LCT-S, LCT-U, MCT) 1740 kJ (Sub)	*Time to meal request* *Breakfast to lunch* Sub: 252.0 ± 10.0 mins LCT-S: 285.0 ± 14.3 mins LCT-U: 273.0 ± 9.8 mins MCT: 286.0 ± 16.6 mins *Lunch to dinner* Sub: 350.0 ± 11.9 mins LCT-S: 334 ± 15.5 mins LCT-U: 331.0 ± 16.8 mins MCT: 320 ± 8.0 mins	*Ad libitum* lunch Sub: 3715 ± 246 kJ LCT-S: 3798 ± 207 kJ LCT-U: 3278 ± 328 kJ MCT: 3100 ± 277 kJ Diet diary Sub: 8028 ± 558 kJ LCT-S: 8522 ± 466 kJ LCT-U: 8787 ± 500 kJ MCT: 8414 ± 466 kJ	VAS – mm Duration: 150 min (breakfast to lunch) Duration: 240 min (lunch to dinner) Hunger	NA
Van Wymelbeke, Louis-Sylvestre, and Fantino (2001)	10	10/0	Age: 22 ± 0.6 y BMI: 21.9 ± 1.7 kg/m^2	Randomized acute crossover postprandial feeding study	40 kJ fat substitute (Sub) 32 g LCT 35 g MCT 53 g CHO and 8 g LCT (CHO)	Potato flakes, mashed carrots, apple sauce Total energy: 3510 kJ (LCT, MCT, CHO) 2380 kJ (Sub)	*Time to meal request* Sub: 362.6 ± 18.0 mins LCT: 364.3 ± 11.0 mins MCT: 372.4 ± 13.0 mins CHO: 413 ± 22 mins	*Ad libitum* dinner Sub: 4375 ± 262 kJ LCT: 4143 ± 390 kJ* MCT: 3602 ± 258 kJ* CHO: 4351 ± 373 kJ	VAS – iAUC Duration: 300 min Hunger	NA

LCT: long-chain triglycerides; MCT: medium-chain triglycerides; PP: pancreatic polypeptide; CCK: cholecystokinin; GIP: gastric-inhibitory peptide; CON: control, CLA: conjugated linoleic acid; PYY: peptide YY; CO: coconut oil; SCT: short-chain triglycerides; SO: sunflower oil; EVOO: extra virgin olive oil; VCO: virgin coconut oil; CHO: carbohydrate; AUC: area under the curve; iAUC: incremental area under the curve; VAS: visual analog scale; mm: millimeter; NA: Not assessed.

↑Denotes increased or greater; ↓ denotes decreased or lesser.

*Different from Control/LCT or lowest dose/concentration ($p < 0.05$).

†Different from all other trials ($p < 0.05$).

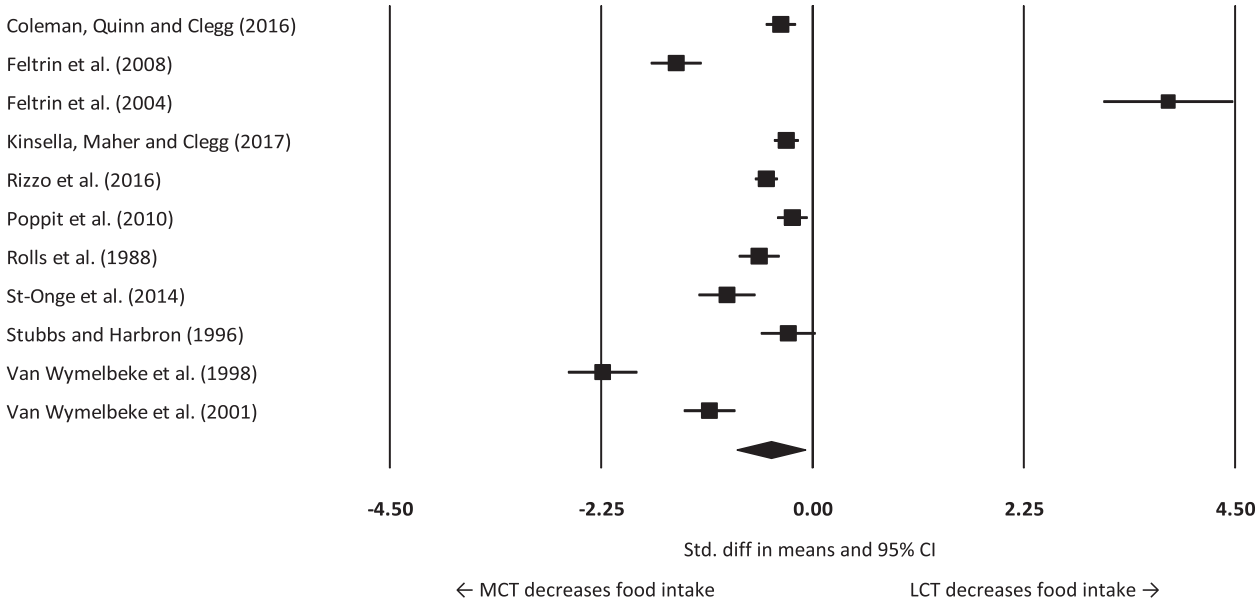

Figure 2. Forrest plot of effects sizes (means ± 95% confidence intervals) for studies examining the effect of MCT compared with LCT on energy intake using a random effects model.

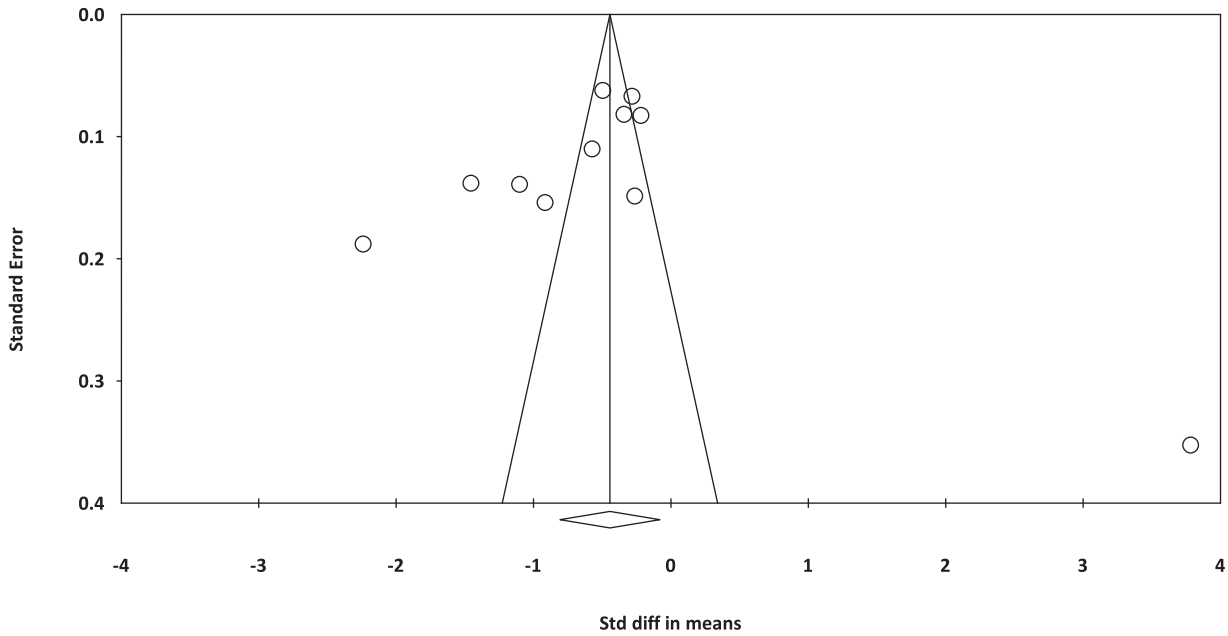

Figure 3. Funnel plot of standard error by standard difference in means for studies evaluating the influence of MCT on energy intake (acute *ad libitum* intake: $n = 10$; habitual daily intake: $n = 1$).

oil. Similarly, to the removal of infusion studies, removal of the comparison of LCT to coconut oil increased the size of the effect of MCT on energy intake (mean effect size: −0.529, 95% confidence intervals −0.598 to −0.460, $N = 10$, $p < 0.001$). The funnel plot (Figure 3) along with Egger's regression intercept showed that there were no small study effects (intercept = −1.094, 95% confidence intervals: −11.481 to 9.293, $p = 0.817$).

Discussion

Main results

Prior to this review, MCT had been identified as potentially having more satiating properties than LCT, but studies investigating this are sparse and have found equivocal findings. Understanding how MCT may affect appetite may have implications for weight management, as feelings of hunger are known to the linked to the low rates of adherence commonly seen in dietary strategies (Franklin et al. 1948; Heymsfield et al. 2007). Whereas it is well known that protein is the most satiating of the macronutrients and fat the least, a significant portion of energy in the western diet comes from fat, and therefore methods to increase the satiety response to fat has implications for weight management strategies. The purpose of this review was to examine the appetite responses and energy intake after meals containing either MCT or LCT. It was hypothesized that MCT would increase satiety compared to LCT. The analyses show that MCT suppress energy intake compared to LCT, and this

appears to be independent of changes in subjective sensations of appetite and alterations in gut peptide hormones.

Energy intake

The present meta-analysis showed that nine out of 10 studies reported decreased energy intake at an acute *ad libitum* meal after ingestion or infusion of MCT, and the only study examining habitual energy intake when MCT was incorporated into the diet also led to decreased energy intake compared to LCT. Whereas the decreased energy intake after MCT consumption wasn't significant in all individual studies, the meta-analysis demonstrated a moderate effect of MCT on energy intake compared to LCT. However, it must be noted that these findings are predominantly limited to the first meal after ingestion of MCT and cannot be extrapolated to further meals. More research is needed to elucidate whether compensation occurs in later meals, or if an energy deficit is achieved. One study did incorporate MCT as part of the habitual diet in different MCT:LCT ratios and found that habitual daily intake was lower after the high MCT:LCT ratio period (Stubbs and Harbron 1996). Where this does corroborate the hypothesis that chronic consumption of MCT decreases overall intake; whether this is due to repeat exposure of MCT or a persistent effect is still not known. Furthermore, as only one study to date has investigated chronic MCT consumption and habitual energy intake, these results require validation.

Appetite

Despite reported alterations in energy intake, this appears to have occurred without any reporting of an effect on subjective appetite responses, indicating that MCT suppresses *ad libitum* energy intake without a concomitant change of feelings of hunger. As aforementioned, this requires further investigation as there is a lack of studies investigating energy intake beyond a single *ad libitum* meal or a single day. Extraction of subjective sensation data was challenging due to the inconsistent reporting of raw values (i.e. only represented in graphical format), and so these were not included in the meta-analysis. Inspection of the results (Table 3) shows that the majority of studies do not report significant differences in any subjective sensation parameter, and when a difference is reported it is not consistent in all parameters in the study (Barbera 2000; Clegg et al. 2012; Kinsella, Maher, and Clegg 2017; Stubbs and Harbron 1996; Valente et al. 2018). The only study to show consistent changes in subjective sensations of appetite incorporated MCT into the diet as part of a very low-calorie diet for 4 weeks (Krotkiewski 2001). It is possible that acute feedings of MCT do not alter perceptions of appetite, but repeated exposure may do so.

Mechanisms

Only four studies examined appetite-related hormones, and so drawing conclusions from these studies are mere speculation; however, secretion of CCK, GIP, PP or GLP-1 appears to be more potent after LCT than MCT. Additionally, another study showed that active ghrelin may be suppressed to a lesser extent than after LCT. MCT have been shown to increase stomach concentrations of acylated ghrelin, as MCT and MCFA are directly used for the acylation of ghrelin (Nishi et al. 2005), which may explain the suppression by LCT. Ghrelin is the only appetite hormone known to stimulate hunger (Wren et al. 2000), whereas CCK, GIP, PP and GLP-1 are involved in promoting satiety and satiation (Batterham et al. 2002; Flint et al. 2001; Gibbs, Young, and Smith 1973; Kissileff et al. 1981; Perry and Wang 2012). Taken together, this implies that MCT exert its anorectic affect through non-hormone mediated mechanisms, however the paucity of data makes this speculation. MCT have been shown to delay gastric emptying (Clegg et al. 2012), despite MCFA being absorbed at a much quicker rate than LCFA (Bach and Babayan 1982). MCT consumption also leads to the production of the ketone body of β-hyroxybutyrate, which may also be anorexigenic (Laeger, Metges, and Kuhla 2010). Future studies should include these measures in their protocols in order to shed further light on these mechanisms.

The one study that found greater *ad libitum* energy intake after MCT compared to LCT (Feltrin et al. 2004) compared lauric acid (C12) to decanoic (C10) acid via intraduodenal infusion and observed significant differences in *ad libitum* energy intake. This was accompanied by greater stimulation of CCK and GLP-1 after infusion of C12. This suggests that the longer chain length is more efficacious at decreasing appetite. It has previously been reported incretin responses to infusions of glucose and lipids are not as pronounced as the response to oral ingestion of glucose (Elrick et al. 1964) or lipids (Lindgren et al. 2011). As such, this makes drawing conclusions from infusion studies difficult. It must also be noted that infusion of C12 induced nausea, which may also explain the decreased *ad libitum* energy intake. This increased nausea was also found after the infusion of LCT but not MCT (Barbera 2000), which also may explain increased satiation scores in that study. Only one other study which examined energy intake reported adverse effects, which were in the form of 'gastric aching' (Rolls et al. 1988), which also may partly explain the decreased *ad libitum* energy intake after MCT ingestion in that study. In the first study to examine MCT and satiety (Rolls et al. 1988), there was a significant interaction between fatty acid chain length and dosage for gastric aching, suggesting that increased dosage of MCT was linked to stronger adverse effects. However, higher doses have been examined with no adverse effects (Wymelbeke, Louis-Sylvestre, and Fantino 2001; Wymelbeke et al. 1998), and the authors describe that, despite statistically significant differences, absolute differences were small (3.5 mm on a 100 mm scale). Only five of the studies included a rating of nausea (Barbera 2000; Clegg, Golsorkhi, and Henry 2013; Feltrin et al. 2008, 2004; Poppitt et al. 2010), which may also confound the effect observed in our meta-analysis, as (although only two studies reported adverse effects) MCT have been shown to cause GI distress (Goedecke et al. 2005; Jeukendrup et al. 1998) and are generally unpalatable (Clegg 2010; Maher and Clegg 2019).

Methodology

The dosages of MCT provided in the studies included in this review had a large variation (10 g (Poppitt et al. 2010) to 42.4 g (Wymelbeke et al. 1998) with an average dose of 23.8 g). There did not appear to be a relationship between dose and whether there was an effect on energy intake, as despite the study providing the lowest dose reported no effect (Poppitt et al. 2010), another study found significant effects with all three doses administered in their study; the lowest providing 12.04 g of MCT (Rolls et al. 1988). Furthermore, the greatest decrease observed after MCT ingestion occurred after 30 g of MCT was provided in a breakfast and preload study (St-Onge et al. 2014). The optimal dose required to beneficially affect appetite remains elusive. One point that must be taken into consideration is the energy contributed from the MCT compared to the decrease in subsequent energy intake it begets. MCT was administered in a variety of ways in the studies in this review, including duodenal infusions (Barbera 2000; Feltrin et al. 2004, 2008), being added to beverages (Coleman, Quinn, and Clegg 2016; Kinsella, Maher, and Clegg 2017; Rolls et al. 1988; St-Onge et al. 2014), a low calorie formula (Krotkiewski 2001), being added to solid meals (Valente et al. 2018; Wymelbeke, Louis-Sylvestre, and Fantino 2001; Wymelbeke et al. 1998), being cooked into other foods (Clegg et al. 2012; Poppitt et al. 2010; St-Onge et al. 2014), ice cream (Rizzo et al. 2016), and being added into the whole diet (Stubbs and Harbron 1996). One practical limitation that must be considered is the fact that the majority of these studies added the test oils to other foods. however, the foods were always kept constant and only fats changed ensuring they were controlled.

Limitations

There are several limitations to this review and meta-analysis. The main limitation to acknowledge is the fact that one reviewer reviewed all papers, instead of multiple reviewers screening all titles and a consensus being met. Furthermore, studies were initially excluded based on titles alone, instead of a title and abstract screening process. These two limitations mean that incomplete retrieval of records cannot be ruled out. Only 16 studies were included based on our criteria, of which 11 were included in the meta-analysis of energy intake (consisting of 20 subgroups). This highlights the limited data examining the role of MCT in satiety rather than a limitation of this review, however there are methodological differences in the studies included which do need to be acknowledged. Three studies used coconut oil as the means of administering MCT (Poppitt et al. 2010; Rizzo et al. 2016; Valente et al. 2018). One study included in this review examined the effect of MCT to coconut oil as well as a control LCT oil, and reported that MCT resulted in lower energy intake compared to both LCT and coconut oil (Kinsella, Maher, and Clegg 2017). This could be due to the higher concentration of lauric acid (~50%) (Denke and Grundy 1992) in coconut oil than in MCT oil (1-3%) (Bach and Babayan 1982; Clegg 2017). It has been shown that only 20-30% of lauric acid acts as an MCT, whereas the remainder is packed in chylomicrons as with LCT (Denke and Grundy 1992). This implies that coconut oil may not be a suitable method of examining MCT, and this may have affected the results of the meta-analysis. A sensitivity analysis was conducted by removing the one study investigating coconut oil (Rizzo et al. 2016) and the one subgroup that compared coconut oil and LCT (Kinsella, Maher, and Clegg 2017), which led to the effect size to increase; which supports the notion that coconut oil is not as effective as MCT at inducing satiety. Two studies, including the only study that reported increased intake after MCT, administered the oils via infusion and not incorporated into a meal (Feltrin et al. 2008, 2004). We did not specify in our criteria that studies included required to have the MCT in a meal, and thus we decided to include these studies, however, the validity of these studies among other feeding studies could be questioned. Furthermore, the one study that reported an increase intake after LCT (Feltrin et al. 2004) compared lauric acid (C12) to decanoic (C10) acid, which is arguably not MCT compared to LCT due to the absorption of lauric acid, as aforementioned. Removal of these studies did not affect the results of the meta-analysis, and thus they have been kept in in order to better represent the available data. However, similar to studies investigating coconut oil, a sensitivity analysis was conducted by removing the two studies which infused MCT (Feltrin et al. 2004, 2008), and this increased the effect size; meaning the inclusion of infusion studies weakened the effect of MCT on energy intake. From a practical standpoint, this further highlights that consumption of MCT leads to suppressed energy intake compared to LCT.

These limitations should be taken into consideration for future research examining this topic, and also shows the small number of appropriate studies examining the effect of MCT on appetite and energy intake.

Conclusion

The present meta-analysis indicates a moderate reduction in energy intake after consumption of MCT, predominantly at single *ad libitum* meals, but also total daily energy intake after daily incorporation of MCT into the diet. Whether this reduction persists past the first meal after consumption of MCT remains to be elucidated. The systematic review indicates that there is no effect of MCT on subjective sensations of appetite. Further work is required to confirm the role of appetite hormones in the satiety response to MCT, but there is currently no evidence to suggest a hormonal role of MCT satiety. Due to the small total number of studies, coupled with the fact not all included a feeding component and only four examined hormonal responses to MCT, this paper calls for more studies examining MCT and satiety incorporating these measures, as well as appropriate sources of MCT.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Author contributions

T.M. and M.E.C. designed the manuscript, developed the search terms and inclusion and exclusion criteria. T.M. did the searches and both authors screening the final papers. TM did the analysis and wrote the paper. MC had responsibility for the final content.

Abbreviations

MCT	Medium-chain triglycerides
CCK	cholecystokinin
LCT	long-chain triglycerides
BMI	body mass index
PP	pancreatic polypeptide
GIP	gastric inhibitory polypeptide
PYY	peptide YY
GLP-1	glucagon-like peptide-1

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