

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

Article

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

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18 **Running head:** MCT and satiety: a systematic review

19 **Abbreviations list:** Medium-chain triglycerides (MCT), cholecystokinin (CCK), long-chain
20 triglycerides (LCT), body mass index (BMI), pancreatic polypeptide (PP), gastric inhibitory
21 polypeptide (GIP), peptide YY (PYY), glucagon-like peptide-1 (GLP-1).

22 Data described in the manuscript, code book, and analytic code will be made available upon
23 request.

24

25

26 **PROSPERO database:** registration number: CRD42018092550.

27

28

29 **Abstract**

30 Research has indicated that consuming medium-chain triglycerides (MCT) may be more

31 satiating than consuming long-chain triglycerides (LCT) potentially causing a reduction in

32 energy intake. However not all studies have demonstrated this acute reduction in energy intake

33 and it has yet to be systematically reviewed. Our main objective was to examine how ingestion

34 of MCT influences energy intake, subjective appetite ratings and appetite-related hormones

35 compared to LCT. Web of Science, MEDLINE, CINHAL and Embase were searched for

36 publications comparing the effect of MCT on appetite (commonly hunger, fullness, desire to

37 eat, and prospective food consumption), appetite-related hormones (pancreatic polypeptide

38 (PP), gastric inhibitory polypeptide (GIP), peptide YY (PYY), glucagon-like peptide-1 (GLP-

39 1), neurotensin, leptin, total ghrelin and active ghrelin) and energy intake to LCT. A random-

40 effects meta-analysis was conducted on studies which examined energy intake.

41 Seventeen studies (291 participants) were included in the systematic review, of which 11 were

42 included in the energy intake meta-analysis. Synthesis of combined data showed evidence of a

43 statistically significant moderate decrease in *ad libitum* energy intake after both acute and

44 chronic ingestion of MCT compared to LCT when assessed under laboratory conditions (mean

45 effect size: -0.444, 95% CI -0.808, -0.080, $p < 0.017$), despite little evidence of any effect of

46 MCT on subjective appetite ratings or circulating hormones.

47 The current evidence supports the notion that MCT decreases subsequent energy intake, but

48 does not appear to affect appetite. Further research is warranted to elucidate the mechanisms

49 by which MCT reduce energy intake.

50

51 **Key words:** Medium-chain triglycerides, satiety, appetite, energy intake, systematic review,
52 meta-analysis

53 **Introduction**

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

64 As a result of this, foods with enhanced satiety have gained much attention, both commercially
65 and in research (Hetherington et al. 2013; Chambers, McCrickerd, and Yeomans 2015).
66 Medium-chain triglycerides (MCT) are triglycerides with shorter chain lengths (6-12 carbon
67 atoms long) than 'traditional' long-chain triglycerides (LCT; 12+ carbon atoms long). **Due to**
68 **the shorter chain length of MCT, its consumption results in attenuated release of**
69 **cholecystokinin (CCK) compared to LCT (Feltrin et al. 2007, 2006; Feinle et al. 2001;**
70 **Matzinger et al. 2000; French et al. 2000). CCK is involved in lipid-related satiety (McLaughlin**
71 **et al. 1999), and thus LCT promote satiety via this mechanism.** However, MCT are absorbed
72 much quicker than LCT (Marten, Pfeuffer, and Schrezenmeir 2006) which leads to large
73 amounts of β -oxidation (Bach and Babayan 1982) and the production of β -hydroxybutyrate
74 (Page et al. 2009); a process which is thought to be anorexigenic (Laeger, Metges, and Kuhla
75 2010; Scharrer 1999). Studies have shown decreased appetite and subsequent energy intake

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

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

90

91 **Methods**

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

94 *Search Strategy*

95 The research question of this systematic review was formulated using PICOS (Population,
96 Intervention, Comparison, Outcome, Setting). The population was defined as adults of healthy
97 status excluding overweight or obesity. The intervention was considered to be any investigation
98 examining medium-chain triglycerides or medium-chain fatty acids on appetite and satiety
99 measures. Outcomes incorporated any measure of appetite (i.e. visual analogue scales),
100 physiological markers of appetite regulation (e.g. PYY, ghrelin) and energy intake measures

101 (*ad libitum* meals, diet diaries). There was no restriction to the settings in which studies were
102 conducted.

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

110 *Exclusion criteria*

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

116 *Data Screening*

117 Records were screened for duplicates, which were removed. Potential studies were identified
118 by examining all titles and removing those which did not contain reference to MCT and appetite
119 or energy intake by one reviewer. The abstracts of the remaining titles were read, and full text
120 copies were obtained if they still met the initial criteria. Information on the remaining studies
121 after abstract screening was tabulated by one researcher (TM), and both investigators (TM and
122 MC) discussed the inclusion of the studies until a mutual consensus was met. The following
123 information was extracted from the included into a spreadsheet: authors, date of publication,
124 sample size, participant characteristics (age, sex, body mass index [BMI]), study setting, source
125 and amount of MCT, appetite outcome measures and results.

126 *Quality Checks*

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

132 *Meta-analysis procedures*

133 Due to inconsistent reporting of visual analogue scale data (i.e. presented in a variety of ways,
134 graphical format, as raw data or calculated AUC), and the small number of studies examining
135 appetite hormones (four), only acute *ad libitum* energy intake data was included in the meta-
136 analysis (either at a single meal or over the course of a whole day). The remaining 11 studies
137 were broken down into 20 subgroups, accounting for studies investigating multiple doses of
138 MCT (Rolls et al. 1988, Stubbs and Harbron 1996) or coconut oil (Rizzo et al. 2016), and for
139 studies with multiple investigations (St-Onge et al. 2014). Energy intake (kJ) was measured at
140 both *ad libitum* meals and habitual daily intake. Where needed, reported values were converted
141 to kJ before computation to standardise the units. Meta-analysis software (Comprehensive
142 Meta-Analysis, Version 3, Biostat, Englewood, NJ, USA) was used to conduct a meta-analysis
143 on extracted data. Data inputted included sample sizes, mean energy intake for LCT and MCT
144 trials, their respective SDs, and a correlation coefficient to account for the fact that the included
145 studies were crossover trials ($r = 0.940$, calculated from energy intake data from the studies
146 included in the review). The software computed effect sizes for each study, as well as an overall
147 effect size using a random-effects model (DerSimonian-Laird inverse variance approach).
148 Effect size was calculated as the standardised difference in means, which we interpreted to be
149 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
150 1992). Negative effect sizes indicate decreased consumption in MCT trials/conditions, whereas

151 positive effect sizes indicated LCT led to decreased energy intake. Publication bias was
152 assessed utilising funnel plots and by quantifying Egger's regression intercept. A significant
153 regression indicates the presence of a small study effect (Sterne, Egger, and Moher 2011).

154

155 **Results**

156 *Descriptive*

157 The database search yielded 4,547 results, which was reduced to 3,517 after the removal of
158 duplicates. After the screening of titles and abstracts, 3,302 were removed. Of the remaining
159 216 texts, 17 satisfied the inclusion criteria (**Figure 1.**).

160 Seven studies were conducted in the UK (Clegg, Golsorkhi, and Henry 2013; Clegg et al. 2012;
161 Coleman, Quinn, and Clegg 2016; Kinsella, Maher, and Clegg 2017; Stubbs and Harbron 1996;
162 Rizzo et al. 2016), two in Australia (Feltrin et al. 2004, 2008), two in France (Van Wymelbeke
163 et al. 1998; Van Wymelbeke, Louis-Sylvestre, and Fantino 2001), two in the US (St-Onge et
164 al. 2014; Rolls et al. 1988), and one each in Italy (Barbera et al. 2000), Sweden (Krotkiewski
165 2001), Brazil (Valente et al. 2018), and New Zealand (Poppitt et al. 2010). Participants were
166 28.57 ± 6.20 years of age with a BMI of 23.49 ± 3.42 kg/m², and there was an average of $15 \pm$
167 8 participants per study (means \pm SD). Participants in one study were classified as 'overweight'
168 according to BMI (M. St-Onge et al. 2014), and were classified as 'obese' in one other
169 (Krotkiewski 2001); all others were in the 'normal' BMI category. There was a total of 291
170 participants, of which 107 were male and 184 were female. There were 11 acute feeding studies
171 (Rolls et al. 1988; Clegg, Golsorkhi, and Henry 2013; Coleman, Quinn, and Clegg 2016;
172 Kinsella, Maher, and Clegg 2017; Rizzo et al. 2016; Miriam E. Clegg et al. 2012; M. St-Onge
173 et al. 2014; Valente et al. 2018; Van Wymelbeke et al. 1998; Van Wymelbeke, Louis-Sylvestre,
174 and Fantino 2001; Poppitt et al. 2010), three acute infusion studies (Barbera et al. 2000; Feltrin
175 et al. 2008, 2004), and two chronic dietary intervention studies, of which one examined

176 participants three times across all arms of the intervention and quantified habitual daily energy
177 intake (Stubbs and Harbron 1996) and the other was a comparison of independent matched
178 groups (Krotkiewski 2001). One dietary intervention provided all foods consumed by
179 participants in 14-day long manipulations, where the amount of energy from MCT was altered
180 (Stubbs and Harbron 1996), and the other was a very low calorie diet, with either MCT or LCT
181 was incorporated into the low-calorie formula incorporated into the diet (Krotkiewski 2001).

182 *Measures*

183 Fourteen out of fifteen studies used 100mm visual analogue scales to measure subjective
184 sensations of appetite (St-Onge et al. 2014). Ten studies examined energy intake during at least
185 one subsequent *ad libitum* eating episode after consumption of a meal/preload containing
186 LCT/MCT (Rizzo et al. 2016; Coleman, Quinn, and Clegg 2016; Feltrin et al. 2004; Kinsella,
187 Maher, and Clegg 2017; Poppitt et al. 2010; Feltrin et al. 2008; Rolls et al. 1988; St-Onge et
188 al. 2014; Van Wymelbeke, Louis-Sylvestre, and Fantino 2001; Van Wymelbeke et al. 1998),
189 and one examined daily habitual energy intake after MCT was covertly incorporated into the
190 diet (Stubbs and Harbron 1996). In that study, participants were required to consume all meals
191 in the laboratory, but were allowed to leave and were not required to ‘live’ in the laboratory;
192 and thus *ad libitum* daily energy intake was quantified. Three of those also included diet diaries
193 for subsequent energy intake (Coleman, Quinn, and Clegg 2016; Kinsella, Maher, and Clegg
194 2017; Van Wymelbeke et al. 1998). Four studies examined appetite hormones, including
195 pancreatic polypeptide (PP) (Barbera et al. 2000), CCK (Barbera et al. 2000; Feltrin et al. 2004,
196 2008), gastric inhibitory polypeptide (GIP) (Barbera et al. 2000; Feltrin et al. 2004), peptide
197 YY (PYY) (Feltrin et al. 2008; St-Onge et al. 2014), leptin (St-Onge et al. 2014), glucagon-
198 like peptide-1 (GLP-1) (Feltrin et al. 2004) and both active and total ghrelin (St-Onge et al.
199 2014).

200 *Test lipids*

201 The main results of included studies are shown in **Table 3**. Six studies directly compared MCT
202 to LCT, which acted as a control (Barbera et al. 2000; Clegg, Golsorkhi, and Henry 2013;
203 Feltrin et al. 2004; Rolls et al. 1988; St-Onge et al. 2014; Stubbs and Harbron 1996). Two
204 studies compared MCT and LCT, and also included a low-fat/no-fat control (Feltrin et al. 2008;
205 Krotkiewski 2001; Van Wymelbeke, Louis-Sylvestre, and Fantino 2001). Three studies had
206 multiple fats, including several LCT conditions such as sunflower oil, olive oil and butter
207 (Clegg et al. 2012); olive oil and lard (Van Wymelbeke et al. 1998); and two with another test
208 oil, which was conjugated linoleic acid (Coleman, Quinn, and Clegg 2016) and short-chain
209 triglycerides (Poppitt et al. 2010). Two studies used coconut oil as the source of MCT in the
210 study (Rizzo et al. 2016; Valente et al. 2018), and another study used coconut oil as well as
211 MCT (Kinsella, Maher, and Clegg 2017). For the LCT trials and controls, three studies used
212 sunflower oil (Clegg, Golsorkhi, and Henry 2013; Clegg et al. 2012; Rizzo et al. 2016), two
213 used rapeseed oil (Coleman, Quinn, and Clegg 2016; Kinsella, Maher, and Clegg 2017), two
214 used corn oil (Rolls et al. 1988; St-Onge et al. 2014), one used beef tallow (Poppitt et al. 2010),
215 one used extra virgin olive oil (Valente et al. 2018), one used margarine (Van Wymelbeke,
216 Louis-Sylvestre, and Fantino 2001), one study used an unspecified vegetable oil (Stubbs and
217 Harbron 1996), and three studies (which administered the lipids via infusion and not feeding)
218 used emulsions of oleic and linoleic acid (Barbera et al. 2000), oleic acid (Feltrin et al. 2008),
219 and lauric acid (Feltrin et al. 2004). One study did not specify the LCT used in their study
220 (Krotkiewski 2001). In terms of saturation of LCT, six studies utilised LCT with a mixture of
221 polyunsaturated and monounsaturated acids (Barbera et al. 2000; Rolls et al. 1988; Clegg,
222 Golsorkhi, and Henry 2013; St-Onge et al. 2014; Rizzo et al. 2016; Valente et al. 2018), four
223 used purely monounsaturated fatty acids (Kinsella, Maher, and Clegg 2017; Coleman, Quinn,
224 and Clegg 2016; Stubbs and Harbron 1996; Feltrin et al. 2008), two used mixtures of
225 monounsaturated and saturated fatty acids (Van Wymelbeke, Louis-Sylvestre, and Fantino

226 2001; Poppitt et al. 2010), and two studies used multiple sources of LCT; polyunsaturated and
227 monounsaturated (sunflower oil), monounsaturated (olive oil) and saturated (butter) fatty acids
228 (Clegg et al. 2012), and saturated (lard) or monounsaturated (olive oil) (Van Wymelbeke et al.
229 1998). When accounting for studies that provided multiple doses, the dosage of MCT ranged
230 from 10 g (Poppitt et al. 2010) to 42.4 g (Van Wymelbeke et al. 1998), with an average dose
231 of 23.8 g. Three studies provided 10-15 g (Poppitt et al. 2010; Rolls et al. 2004; Rizzo et al.
232 2016), four provided 20-25 g (Coleman, Quinn, and Clegg 2016; Kinsella, Maher, and Clegg
233 2017; St-Onge and Jones 2002; Rolls et al. 2004), three provided 30-35 g (St-Onge et al. 2014;
234 Van Wymelbeke, Louis-Sylvestre, and Fantino 2001; Rolls et al. 1988) and one provided 40-
235 45 g (Van Wymelbeke et al. 1998).

236 *Outcomes*

237 One out of 11 studies (Feltrin et al. 2004) reported decreased energy intake at an *ad libitum*
238 meal after MCT compared to LCT, although this was only significant in seven studies
239 (Coleman, Quinn, and Clegg 2016; Feltrin et al. 2008; Rolls et al. 1988; St-Onge et al. 2014;
240 Stubbs and Harbron 1996; Van Wymelbeke, Louis-Sylvestre, and Fantino 2001; Van
241 Wymelbeke et al. 1998). The one study that reported decreased intake after LCT compared to
242 MCT reported a significantly lower energy intake after LCT compared to MCT (Feltrin et al.
243 2004). The average energy intake at the *ad libitum* meal in that study after LCT and MCT trials,
244 respectively, was $1,747 \pm 633$ kJ and $4,109 \pm 589$ kJ. Five studies out of 14 reported significant
245 differences in appetite ratings, which were decreased hunger and increased satiety after MCT
246 (Krotkiewski 2001), increased fullness after MCT (Kinsella, Maher, and Clegg 2017),
247 increased satiety after LCT (Barbera et al. 2000), decreased hunger but also decreased desire
248 to eat after infusion of MCT (Feltrin et al. 2004), and increased hunger and decreased fullness
249 after MCT (coconut oil) (Valente et al. 2018). Three studies (Rolls et al. 1988; Feltrin et al.
250 2004; Barbera et al. 2000) reported significant adverse effects, which manifested as gastric

251 aching after the MCT drinks (Rolls et al. 1988), and increased nausea after infusions LCT
252 compared to MCT (Barbera et al. 2000; Feltrin et al. 2004).

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

259 *Meta-analysis*

260 Due to high levels of heterogeneity ($I^2 = 97.0\%$, $Q = 333.9$, $T^2 = 0.355$, $d_f = 10$), a random
261 effects model was chosen (Ades, Lu, and Higgins 2005). Effect size for acute *ad libitum* energy
262 intake ranged from -2.235 to 3.789. Statistics for each individual study are reported in
263 Supplementary table 1. There was a statistically significant moderate decrease in *ad libitum*
264 energy intake after MCT ingestion compared to LCT ingestion (mean effect size: -0.444, 95%
265 confidence intervals -0.808 to -0.080, $N = 11$, $p = 0.017$; Figure 2). Sensitivity analysis showed
266 that the removal of each study had only minor effects on overall effect size, and no effect on
267 significance. In order to further examine and specify the effect of consuming MCT on satiety,
268 a sensitivity analysis was conducted by removing infusion studies. This did not alter the
269 direction of significance, but it did increase the level of significance (mean effect size: -0.681,
270 95% confidence intervals -0.950 to -0.412, $N = 8$, $p < 0.001$). More sensitivity analyses were
271 conducted in order to specify the effect of MCT without the influence of coconut oil. Similarly
272 to the removal of infusion studies, removal of the comparison of LCT to coconut oil increased
273 the size of the effect of MCT on energy intake (mean effect size: -0.529, 95% confidence
274 intervals -0.598 to -0.460, $N = 10$, $p < 0.001$). The funnel plot (**Figure 3.**) along with Egger's

275 regression intercept showed that there were no small study effects (intercept = -1.094, 95%
276 confidence intervals: -11.481 to 9.293, $p = 0.817$).

277

278 **Discussion**

279 *Main results*

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

292 *Energy intake*

293 The present meta-analysis showed that nine out of 10 studies reported decreased energy intake
294 at an acute *ad libitum* meal after ingestion or infusion of MCT, and the only study examining
295 habitual energy intake when MCT was incorporated into the diet also led to decreased energy
296 intake compared to LCT. Whereas the decreased energy intake after MCT consumption wasn't
297 significant in all individual studies, the meta-analysis demonstrated a moderate effect of MCT
298 on energy intake compared to LCT. However, it must be noted that these findings are
299 predominantly limited to the first meal after ingestion of MCT and cannot be extrapolated to

300 further meals. More research is needed to elucidate whether compensation occurs in later
301 meals, or if an energy deficit is achieved. One study did incorporate MCT as part of the habitual
302 diet in different MCT:LCT ratios and found that habitual daily intake was lower after the high
303 MCT:LCT ratio period (Stubbs and Harbron 1996). Where this does corroborate the hypothesis
304 that chronic consumption of MCT decreases overall intake; whether this is due to repeat
305 exposure of MCT or a persistent effect is still not known. Furthermore, as only one study to
306 date has investigated chronic MCT consumption and habitual energy intake, these results
307 require validation.

308 *Appetite*

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

323 *Mechanisms*

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

340 The one study that found greater *ad libitum* energy intake after MCT compared to LCT (Feltrin
341 et al. 2004) compared lauric acid (C12) to decanoic (C10) acid via intraduodenal infusion and
342 observed significant differences in *ad libitum* energy intake. This was accompanied by greater
343 stimulation of CCK and GLP-1 after infusion of C12. This suggests that the longer chain length
344 is more efficacious at decreasing appetite. It has previously been reported incretin responses to
345 infusions of glucose and lipids are not as pronounced as the response to oral ingestion of
346 glucose (Elrick et al. 1964) or lipids (Lindgren et al. 2011). As such, this makes drawing
347 conclusions from infusion studies difficult. It must also be noted that infusion of C12 induced
348 nausea, which may also explain the decreased *ad libitum* energy intake. This increased nausea

349 was also found after the infusion of LCT but not MCT (Barbera et al. 2000), which also may
350 explain increased satiation scores in that study. Only one other study which examined energy
351 intake reported adverse effects, which were in the form of ‘gastric aching’ (Rolls et al. 1988),
352 which also may partly explain the decreased *ad libitum* energy intake after MCT ingestion in
353 that study. In the first study to examine MCT and satiety (Rolls et al. 1988), there was a
354 significant interaction between fatty acid chain length and dosage for gastric aching, suggesting
355 that increased dosage of MCT was linked to stronger adverse effects. However, higher doses
356 have been examined with no adverse effects (Van Wymelbeke, Louis-Sylvestre, and Fantino
357 2001; Van Wymelbeke et al. 1998), and the authors describe that, despite statistically
358 significant differences, absolute differences were small (3.5 mm on a 100 mm scale). Only five
359 of the studies included a rating of nausea (Barbera et al. 2000; Feltrin et al. 2008, 2004; Poppitt
360 et al. 2010; Clegg, Golsorkhi, and Henry 2013), which may also confound the effect observed
361 in our meta-analysis, as (although only two studies reported adverse effects) MCT have been
362 shown to cause GI distress (Jeukendrup et al. 1998; Goedecke et al. 2005) and are generally
363 unpalatable (Clegg 2010; Maher and Clegg 2018).

364 *Methodology*

365 The dosages of MCT provided in the studies included in this review had a large variation (10
366 g (Poppitt et al. 2010) to 42.4 g (Van Wymelbeke et al. 1998) with an average dose of 23.8 g).
367 There did not appear to be a relationship between dose and whether there was an effect on
368 energy intake, as despite the study providing the lowest dose reported no effect (Poppitt et al.
369 2010), another study found significant effects with all three doses administered in their study;
370 the lowest providing 12.04 g of MCT (Rolls et al. 1988). Furthermore, the greatest decrease
371 observed after MCT ingestion occurred after 30 g of MCT was provided in a breakfast and
372 preload study (St-Onge et al. 2014). The optimal dose required to beneficially affect appetite
373 remains elusive. One point that must be taken into consideration is the energy contributed from

374 the MCT compared to the decrease in subsequent energy intake it begets. MCT was
375 administered in a variety of ways in the studies in this review, including duodenal infusions
376 (Barbera et al. 2000; Feltrin et al. 2004, 2008), being added to beverages (Coleman, Quinn, and
377 Clegg 2016; Kinsella, Maher, and Clegg 2017; Rolls et al. 1988; St-Onge et al. 2014), a low
378 calorie formula (Krotkiewski 2001), being added to solid meals (Van Wymelbeke et al. 1998;
379 Van Wymelbeke, Louis-Sylvestre, and Fantino 2001; Valente et al. 2018), being cooked into
380 other foods (Clegg et al. 2012; Poppitt et al. 2010; St-Onge et al. 2014), ice cream (Rizzo et al.
381 2016), and being added into the whole diet (Stubbs and Harbron 1996). One practical limitation
382 that must be considered is the fact that the majority of these studies added the test oils to other
383 foods. however the foods were always kept constant and only fats changed ensuring they were
384 controlled.

385

386 *Limitations*

387 There are several limitations to this review and meta-analysis. The main limitation to
388 acknowledge is the fact that one reviewer reviewed all papers, instead of multiple reviewers
389 screening all titles and a consensus being met. Furthermore, studies were initially excluded
390 based on titles alone, instead of a title and abstract screening process. These two limitations
391 mean that incomplete retrieval of records cannot be ruled out. Only 16 studies were included
392 based on our criteria, of which 11 were included in the meta-analysis of energy intake
393 (consisting of 20 subgroups). This highlights the limited data examining the role of MCT in
394 satiety rather than a limitation of this review, however there are methodological differences in
395 the studies included which do need to be acknowledged. Three studies used coconut oil as the
396 means of administering MCT (Rizzo et al. 2016; Poppitt et al. 2010; Valente et al. 2018). One
397 study included in this review examined the effect of MCT to coconut oil as well as a control
398 LCT oil, and reported that MCT resulted in lower energy intake compared to both LCT and

399 coconut oil (Kinsella, Maher, and Clegg 2017). This could be due to the higher concentration
400 of lauric acid (~50%) (Denke and Grundy 1992) in coconut oil than in MCT oil (1-3%) (Bach
401 and Babayan 1982; Clegg 2017). It has been shown that only 20-30% of lauric acid acts as an
402 MCT, whereas the remainder is packed in chylomicrons as with LCT (Denke and Grundy
403 1992). This implies that coconut oil may not be a suitable method of examining MCT, and this
404 may have affected the results of the meta-analysis. A sensitivity analysis was conducted by
405 removing the one study investigating coconut oil (Rizzo et al. 2016) and the one subgroup that
406 compared coconut oil and LCT (Kinsella, Maher and Clegg 2017), which led to the effect size
407 to increase; which supports the notion that coconut oil is not as effective as MCT at inducing
408 satiety. Two studies, including the only study that reported increased intake after MCT,
409 administered the oils via infusion and not incorporated into a meal (Feltrin et al. 2008, 2004).
410 We did not specify in our criteria that studies included required to have the MCT in a meal,
411 and thus we decided to include these studies, however, the validity of these studies among other
412 feeding studies could be questioned. Furthermore, the one study that reported an increase intake
413 after LCT (Feltrin et al. 2004) compared lauric acid (C12) to decanoic (C10) acid, which is
414 arguably not MCT compared to LCT due to the absorption of lauric acid, as aforementioned.
415 Removal of these studies did not affect the results of the meta-analysis, and thus they have been
416 kept in in order to better represent the available data. However, similar to studies investigating
417 coconut oil, a sensitivity analysis was conducted by removing the two studies which infused
418 MCT (Feltrin et al. 2004, 2008), and this increased the effect size; meaning the inclusion of
419 infusion studies weakened the effect of MCT on energy intake. From a practical standpoint,
420 this further highlights that consumption of MCT leads to suppressed energy intake compared
421 to LCT.

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

425

426 **Conclusion**

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

437

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