

**Women with a low satiety phenotype show impaired appetite control and greater resistance to weight loss.**

BUCKLAND, Nicola J <<http://orcid.org/0000-0002-3667-6438>>, CAMIDGE, Diana, CRODEN, Fiona, MYERS, Anna <<http://orcid.org/0000-0001-6432-8628>>, LAVIN, Jacquelynn H, STUBBS, R James, BLUNDELL, John E and FINLAYSON, Graham

Available from Sheffield Hallam University Research Archive (SHURA) at:

<http://shura.shu.ac.uk/24928/>

---

This document is the author deposited version. You are advised to consult the publisher's version if you wish to cite from it.

**Published version**

BUCKLAND, Nicola J, CAMIDGE, Diana, CRODEN, Fiona, MYERS, Anna, LAVIN, Jacquelynn H, STUBBS, R James, BLUNDELL, John E and FINLAYSON, Graham (2019). Women with a low satiety phenotype show impaired appetite control and greater resistance to weight loss. *British Journal of Nutrition*, 1-22.

---

**Copyright and re-use policy**

See <http://shura.shu.ac.uk/information.html>

1 **Women with a low satiety phenotype show impaired appetite control and greater resistance to**  
2 **weight loss.**

3 Nicola J. Buckland<sup>1</sup>, Diana Camidge<sup>2</sup>, Fiona Croden<sup>2</sup>, Anna Myers<sup>3</sup>, Jacquelynne H. Lavin<sup>4</sup>, R. James  
4 Stubbs<sup>2</sup>, John E. Blundell<sup>2</sup> & Graham Finlayson<sup>2</sup>

5 <sup>1</sup>Department of Psychology, University of Sheffield, Cathedral Court, 1 Vicar Lane, Sheffield, S1  
6 2LT.

7 <sup>2</sup>Human Appetite Research Unit, Appetite Control and Energy Balance Group, School of Psychology,  
8 University of Leeds, Leeds, LS2 9JT.

9 <sup>3</sup>Sheffield Hallam University, Centre for Sport and Exercise Science, Collegiate Hall, Sheffield, S10  
10 2BP

11 <sup>4</sup>Nutrition and Research Department, Slimming World, Alfreton, Derbyshire, DE55 4RF.

12

13 **Corresponding author:** Dr Nicola Buckland, email: n.buckland@sheffield.ac.uk, telephone: (+44)  
14 114 222 6508.

15

16 **Shortened title:** Satiety phenotypes, appetite and weight loss

17

18 **Key words:** satiety phenotypes, weight loss, energy density, food intake, food preferences, appetite  
19 control

ACCEPTED MANUSCRIPT

20 **Abstract**

21 This trial compared weight loss outcomes over 14-weeks in women showing low or high  
22 satiety responsiveness [low or high satiety phenotype (LSP, HSP)] measured by a  
23 standardized protocol. Food preferences and energy intake after low and high energy density  
24 (LED, HED) meals were also assessed. Ninety-six women (n = 52 analysed;  $41.24 \pm 12.54$   
25 years;  $34.02 \pm 3.58 \text{ kg/m}^2$ ) engaged in one of two weight loss programs underwent LED and  
26 HED laboratory-test days during weeks 3 and 12. Preferences for LED and HED-foods  
27 (Leeds Food Preference Questionnaire) and *ad libitum* evening meal and snack energy intake  
28 (EI) were assessed in response to equi-caloric LED- and HED-breakfasts and lunches.  
29 Weekly questionnaires assessed control over eating and ease of adherence to the program.  
30 Satiety quotients based on subjective fullness ratings post-LED and HED breakfasts  
31 determined LSP (n=26) and HSP (n=26) by tertile splits. Results showed that the LSP lost  
32 less weight and had smaller reductions in waist circumference compared to HSP. The LSP  
33 showed greater preferences for HED-foods, and under HED-conditions, consumed more  
34 snacks (kcal) compared to HSP. Snack EI did not differ under LED-conditions. LSP reported  
35 less control over eating and reported more difficulty with program adherence. In conclusion,  
36 low satiety responsiveness is detrimental for weight loss. LED meals can improve self-  
37 regulation of EI in the LSP, which may be beneficial for longer-term weight control.

## 38 Introduction

39 In 2015, 63% of UK adults were overweight or obese<sup>(1)</sup>. In efforts to control body  
40 weight, two thirds of women have reported a recent weight loss attempt<sup>(2)</sup>. Weight loss in  
41 response to such attempts varies<sup>(3)</sup>, and few individuals achieve long term weight loss<sup>(4-6)</sup>.  
42 Individuals who have attempted weight loss report that hunger is one of the main challenges  
43 to losing weight<sup>(7, 8)</sup>. As such the ability to detect appetite sensations may impact the success  
44 of a weight loss attempt.

45 There is variability in the extent to which individuals are able to detect changes in  
46 appetite sensations after eating<sup>(9, 10)</sup>. The satiety quotient (SQ) has been used to measure the  
47 degree to which individuals feel satiated in response to a meal (satiating efficiency) (meals are  
48 often calibrated to estimated individual daily energy needs<sup>(11)</sup>). The SQ measures changes in  
49 subjective appetite sensations following a fixed-energy meal. Higher SQ scores (greater  
50 satiating efficiency) have been found to correspond with lower energy intake (EI) in  
51 laboratory and free-living settings<sup>(12, 13)</sup>. Based on SQ scores, individuals can be categorised  
52 as either low or high satiety behavioural phenotypes (LSP, HSP)<sup>(11, 14, 15)</sup>. These satiety  
53 phenotypes have been shown to differ on psychological<sup>(11, 14)</sup>, metabolic<sup>(14)</sup> and behavioural  
54 outcomes<sup>(11)</sup>. For instance, compared to the HSP, the LSP is associated with greater trait  
55 disinhibition (tendency to eat opportunistically)<sup>(10, 11)</sup>, lower craving control, greater  
56 preferences to eat high fat foods [as indicated with The Leeds Food Preference Questionnaire  
57 (LFPQ)<sup>(16)</sup>] and greater meal EI<sup>(14)</sup>. As such, the evidence suggests that the LSP are less able  
58 to control their appetite and are susceptible to overconsumption compared to HSP.

59 Therefore, it is important to identify strategies that promote satiety in the LSP and  
60 prevent overconsumption. Low energy density (LED) foods have been identified as a food  
61 associated with increased satiation and satiety<sup>(17-19)</sup>. Whether LED meals improve LSP's  
62 acute appetite control is unknown; to date, studies have only compared LSP's and HSP's  
63 appetite responses to one meal<sup>(11, 14)</sup>. To our knowledge, no studies have compared appetite  
64 responses to LED and high energy dense (HED) meals in the satiety phenotypes. In terms of  
65 appetite responses in women engaged in weight loss, it is important to assess not only  
66 subjective appetite and intake, but also implicit preferences for high fat food. Dietary energy  
67 reductions have been shown to increase the rewarding value and appeal of foods<sup>(20, 21)</sup>, which  
68 may impair dietary control. It is currently unknown whether LED foods can prevent such  
69 hedonic motivations previously found in the LSP<sup>(11)</sup>.

70 Moreover, the impact of the LSP on weight loss is unclear. One study in men reported  
71 that the LSP lost less body weight after a 16-week diet compared to the HSP<sup>(15)</sup>. Whereas  
72 another study using male and female participants reported no effects of the LSP on weight  
73 change<sup>(22)</sup>. As such, further studies which investigate specific samples (e.g. women only) and  
74 types of weight loss programs followed are needed to confirm the role that the LSP has on  
75 weight loss.

76 This study characterised women as LSP or HSP and compared weight loss and  
77 changes in body composition after a 14-week weight loss program (Slimming World, UK or  
78 NHS Live Well program). Food intake and food preferences (liking and wanting) in response  
79 to LED and HED meals in LSP and HSP were also assessed in the laboratory. Additionally,  
80 the study compared LSP's and HSP's self-reported appetite control during the program. It  
81 was hypothesised that compared to the HSP, the LSP would lose less body weight and body  
82 fat, have smaller reductions in waist and hip circumference, exhibit weaker appetite control  
83 under HED test conditions compared to LED test conditions, and report weaker appetite  
84 control during the program.

## 85 **Methods**

### 86 *Participants*

87 The study was conducted as a secondary analysis from data collected for a trial that is  
88 reported in more detail elsewhere<sup>(19)</sup> (ClinicalTrials.gov #NCT02012426). The current  
89 analysis differs to the previous analyses (which reported effects for the overall sample), by  
90 focusing specifically on satiety phenotypes. Based on previous research<sup>(15)</sup> power calculations  
91 in G\*Power with an  $\alpha$  of 0.05 and power of 0.80 showed that a sample size of 54 participants  
92 would be sufficient to detect significant differences in weight change between satiety  
93 phenotypes<sup>(23)</sup>. Ninety-six women who were overweight or obese and had recently enrolled in  
94 a weight loss program were recruited. Participants were recruited from Slimming World, UK  
95 groups<sup>(24)</sup> ( $n = 49$ ) and the University of Leeds population and local area ( $n = 47$ ). Only  
96 volunteers who had recently enrolled in the Slimming World, UK program were recruited to  
97 the Slimming World arm of the trial. Following recruitment, this group continued with the  
98 Slimming World, UK program. Participants recruited from the University of Leeds and local  
99 area followed the NHS Live Well program<sup>(25)</sup>. Further details about each program have been  
100 previously reported<sup>(19)</sup>. In brief, Slimming World, UK is a group-based commercial weight  
101 management program. The program advocates ad libitum intake of LED foods and controlled

102 amounts of higher energy dense foods. The NHS Live Well program is an online program  
103 which recommends a daily 600 kcal deficit and provides dietary and physical activity advice.

104 Volunteers who indicated confounding health issues, were taking medications that  
105 affect appetite or weight, had received bariatric surgery, indicated an inability to eat the study  
106 foods or follow study procedures were excluded (for full exclusion criteria see<sup>(19)</sup>). The study  
107 was approved by the University of Leeds, School of Psychology ethics committee.  
108 Participants provided written informed consent and received £250 upon study completion.

#### 109 *Design, measures and procedure*

110 At week 1, body weight and height were measured (by a Slimming World, UK group  
111 leader or University researcher using a stadiometer and electronic scales,) and participants  
112 started their weight loss program. During weeks 2 and 14, participants attended a morning  
113 session at the University of Leeds, Human Appetite Research Unit, and under standardised  
114 controlled procedures (overnight fast, 24-hour alcohol abstinence and no physical activity on  
115 the morning of the session; compliance was checked upon arrival) the following measures  
116 were assessed: body weight and body composition [body fat, percentage (%) body fat and fat-  
117 free mass assessed using air plethysmography (Bodpod, Concord, California, USA) in  
118 minimal clothing], waist and hip circumference (measured by researcher, average of two  
119 measures), RMR (indirect calorimeter, GEM; Nutren Technology Ltd), resting blood pressure  
120 and heart rate (Omron M10-IT digital blood pressure cuff) and psychometric traits (cognitive  
121 restraint, trait disinhibition and trait hunger using the Three Factor Eating Questionnaire<sup>(26)</sup>).  
122 Other measures, specifically relevant to the larger study were also recorded but not reported  
123 here<sup>(19)</sup>.

124 To assess appetite control in response to energy density manipulations, early on in the  
125 program (week 3) participants attended the unit under standardised controlled procedures  
126 mentioned above (but with instructions to maintain similar levels of physical activity across  
127 days), and in a repeated-measures design were provided with LED or HED meals. Condition  
128 order was counter-balanced across participants and each condition was separated by a  
129 minimum of 7-days in both weeks 3 and 12<sup>(27)</sup>. The energy density manipulations were  
130 repeated later on in the program (week 12). During the interval between conditions (both at  
131 the early late phase of the program), participants completed weighed food diaries and wore a  
132 physical activity monitor (SenseWear Armband; BodyMedia, Inc., Pittsburgh, PA) which  
133 assessed total physical activity and sleep duration, as has previously been described<sup>(28)</sup>. The

134 number of days between participants starting the weight loss program and completing the  
135 measures session and test meal probe days were matched across program type. Thus,  
136 participants from the Slimming World, UK and NHS Live Well program had been engaged in  
137 a weight loss program for the same duration when body weight and body composition ( $M$ :  $21$   
138  $\pm 6$  days) and appetite control ( $M$ :  $27 \pm 7$  days) were assessed. A diagram of the overall study  
139 timeline has been reported here<sup>(19)</sup>.

#### 140 *Energy density*

141 On test meal days, participants were provided with either a day of LED ( $\leq 0.8$  kcal/g)  
142 or HED foods ( $\geq 2.5$  kcal/g) across breakfast, lunch, an evening meal and evening snacks.  
143 Across both LED and HED conditions, the breakfast and lunch provided 50% of total daily  
144 energy needs (based on  $RMR \times 1.4$  sedentary physical activity levels). The evening meal and  
145 evening snacks were served to *ad libitum* (for more details see<sup>(19)</sup>). Foods were sourced from  
146 a UK supermarket except for the LED evening meal (beef chilli con carne) which was  
147 provided by Slimming World, UK and used in all LED test sessions (regardless of weight  
148 loss program being followed). Energy density was manipulated by using LED and HED  
149 versions of products. For fixed meals, participants were required to eat the entire portion. For  
150 the evening meal, participants were instructed to help themselves to as much or as little of the  
151 food as they liked and to eat until they felt they had eaten enough. For snacks, participants  
152 were instructed to help themselves to as much or as little of the foods as they liked, to avoid  
153 eating other foods and to avoid sharing the snacks. Meals were served four hours apart and  
154 took place in the research unit. Participants could leave the research unit between meals but  
155 were instructed to fast and consume water only during this period. Bottled water was  
156 provided to improve compliance. After each meal, participants rated meal palatability  
157 (appeal, pleasantness and satisfaction) on 100-mm visual analogue scales (VAS). Participants  
158 took snacks home and returned left over packaging the next day so that intake could be  
159 assessed.

#### 160 *Food intake and food preferences*

161 To determine food intake, meals were covertly weighed pre- and post-consumption.  
162 Weight intake was converted to EI using food composition tables<sup>(29)</sup> and manufacturers'  
163 nutritional information. Meal and snack intake were summed to provide total day intake.

164 Implicit and explicit food preferences to LED- and HED-foods were assessed pre- and  
165 post-lunch using the validated LFPQ (for details see<sup>(16)</sup>). Participants were presented with

166 sweet and savoury, LED- and HED-foods on screen, and to assess explicit liking, participants  
167 rated the pleasantness of each food. To assess implicit wanting, participants completed a  
168 forced-choice task, whereby the food images were paired so that every image from each of  
169 the four food types (LED/HED, sweet/savoury) were compared to every other type over  
170 repeated trials (food pairs). Participants were instructed to respond as quickly and accurately  
171 as possible to indicate the food they most wanted to eat at that time. Reaction times were  
172 recorded and used to compute mean response times for each food type after adjusting for  
173 frequency of selection. Mean LED-food scores were subtracted from mean HED-food scores  
174 to provide a bias score for HED- versus LED-foods. Higher scores indicate greater preference  
175 for HED- relative to LED-foods.

#### 176 *Satiety quotient (SQ)*

177 During the LED and HED test meal days, participants rated subjective fullness  
178 sensations on 100-mm VAS immediately pre- and post- each meal and at hourly intervals  
179 (“How hungry do you feel right now”, ‘0 = not at all’; ‘100 = extremely’)<sup>(27)</sup>). The SQ was  
180 calculated using the average fullness scores collected at pre- and 180-minutes post-breakfast  
181 on the LED and HED probe days administered in the early phase of the program. Fullness  
182 ratings were used because of the appetite sensations (e.g. hunger, desire to eat), fullness is the  
183 strongest predictor of EI, and it has been argued that fullness is the easiest sensation to detect  
184 due to its links with physical gastric distension<sup>(12)</sup>. Tertile splits were conducted on appetite  
185 ratings recorded on the early probe days only to prevent weight loss over the program  
186 confounding the satiety phenotype categorisation<sup>1</sup>. There was good internal reliability  
187 between scores (Cronbach  $\alpha = 0.65$ ). The SQ was calculated using the following formula:

$$188 \quad \text{SQ (mm/kcal)} = \left[ \frac{\text{180-minutes post-breakfast fullness (mm)} - \text{fasting fullness (mm)}}{\text{Breakfast energy intake (kcal)}} \right] \times 100$$

189

#### 190 *Appetite control during the program*

191 Self-reported appetite control was assessed outside the unit with questionnaires each  
192 week. Participants were instructed to complete questionnaires on the same day and time each  
193 week. Participants rated control over eating, ability to adhere to the program’s food choices,  
194 adherence to the program overall and ease of adhering to the program on 100-mm VAS

<sup>1</sup>SQ scores obtained at the early (HSP: 12.64  $\pm$ SD 3.40; LSP: 1.05  $\pm$ SD 2.76) and late phases of the program (HSP: 9.59  $\pm$ SD 6.16; LSP: 4.61  $\pm$ SD 5.79) were significantly correlated,  $r = .44$ ,  $p = .001$ .



195 (“How much do you feel IN CONTROL of what you're eating?”; “Have you felt able to stick  
196 to your plan regarding your food choices?”; “How WELL have you managed to stick with the  
197 weight control program?”; “How EASY do you find it to stick to your weight control  
198 program?”).

### 199 *Statistical analyses*

200 Raw SQ scores from the early probe days in the full sample were initially included as  
201 a covariate in an ANCOVA examining changes in body weight between weeks 1 and 14  
202 controlling for programme type. The week x SQ interaction was significant,  $p=.003$ ,  $\eta p^2=.11$   
203 and as such further analyses of SQ (comparisons of LSP and HSP) were conducted using  
204 point estimates of lower and upper tertile SQ-scores. Scores  $<4.5$  were classified as LSP;  
205 scores  $>8.5$  were classified as HSP. These cut-off points are similar to those used in previous  
206 research<sup>(14)</sup>. Participants scoring 4.6 to 8.5 were unclassified and not included in further  
207 analyses or figures to facilitate interpretation and visualisation of findings.

208 Outcomes were assessed in participants who completed the study with eligible data  
209 (completers analysis). For body weight and body composition outcomes, separate intention to  
210 treat analyses (ITT) using last observation carried forward were also conducted to account for  
211 participants that did not complete the study, provided that data was available (no data was  
212 available for participants who withdrew before completing early test meal sessions)<sup>(30)</sup>. To  
213 assess data collected from the SenseWear armbands, proprietary algorithms available in the  
214 SenseWear software were used (SenseWear Professional software version 8.0, algorithm  
215 v5.2). Total physical activity was calculated by summing the amount of time spent in  
216 activities  $>1.5$  METs.

217 A Chi-Squared test showed that participants from each weight loss program were  
218 evenly distributed across the satiety phenotypes [LSP: Slimming World  $n = 12$ , NHS Live  
219 Well  $n = 14$ ; HSP: Slimming World  $n = 13$ , NHS Live Well  $n = 13$ ;  $X(1)=0.78$ ,  $p=.78$ ].  
220 Program type and percentage weight change up to the week 2 measures session was included  
221 as a covariate in all analyses except for t-tests and unless specified. For concision, results are  
222 reported for covariates only when covariates were significant.

223 To compare the characteristics of the satiety phenotypes at week 1, ANCOVAs were  
224 conducted. Mixed-ANCOVAs were used to compare changes in body weight and  
225 composition between satiety phenotypes. To control for starting body weight and  
226 composition, percentage change in body weight outcomes between satiety phenotypes were

227 compared. Mixed ANCOVAs were used to compare food intake and food preferences in the  
228 satiety phenotypes under LED and HED conditions. To assess appetite control during the  
229 program mixed ANOVAs were used to compare ratings between satiety phenotypes across  
230 weeks. Significant interactions were explored with t-tests unless specified. Averages from  
231 early and late probe days were computed where necessary. Results were considered  
232 significant if  $p < 0.05$  except for tests with multiple comparisons, whereby a more  
233 conservative  $p$ -value was used to account for multiple comparisons (0.05 divided by the  
234 number of comparisons). The analysis reports results for the comparison between LSP and  
235 HSP only. Overall changes over weeks for each outcome have previously been reported for  
236 the full sample<sup>(19)</sup>. Data are presented as means  $\pm$  standard deviation (95% confidence  
237 intervals: lower, upper) unless specified. For concision, when multiple results are reported,  
238 the most conservative  $p$ -value is provided. Partial eta squared ( $\eta^2$ ) is reported for effect sizes  
239 and interpreted as 0.01 small, 0.06 moderate and 0.14 as large<sup>(31)</sup>. Analyses were conducted  
240 in Statistical Package for Social Science (IBM SPSS, version 24).

## 241 **Results**

### 242 *Sample characteristics*

243 Of the 96 participants (age:  $41.24 \pm 12.54$  years; BMI:  $34.02 \pm 3.58$  kg/m<sup>2</sup>), ten  
244 withdrew and six were excluded (ineligible  $n=3$ <sup>2</sup>, extreme weight gain  $n=1$ , broken leg  $n=1$ ;  
245 medical condition  $n=1$ ). One participant could not be classified to a satiety phenotype due to  
246 missing appetite ratings. The remaining 79 participants were classified as LSP ( $n=26$ ), HSP  
247 ( $n=26$ ) or unclassified ( $n=27$ ). Data from four other participants were available for ITT  
248 analyses (LSP  $n=2$ , HSP  $n=1$ , unclassified  $n=1$ ).

249 Baseline characteristics for the LSP and HSP that completed the trial are shown in  
250 Table 1. By definition, the LSP's SQ was significantly lower compared to the HSP. With the  
251 exception of blood pressure, no baseline outcomes significantly differed between satiety  
252 phenotypes. The LSP had significantly greater resting systolic and diastolic blood pressure  
253 that remained significant when controlling for body weight and body mass index (BMI).

---

<sup>2</sup>Two were long term members of Slimming World, UK and led group sessions, and one had a confounding health issue identified after study enrolment.

254 *Changes in body weight and body composition*

255 Results for changes in body weight and body composition did not differ between  
256 completers and ITT analyses unless stated (see Table 2). The HSP lost significantly more  
257 weight compared to the LSP as qualified by a significant week x phenotype interaction on  
258 body weight ( $p=.02$ ,  $\eta p^2=0.10$ ) (approached significance in the ITT model,  $p=.09$ ,  $\eta p^2=0.05$ )<sup>3</sup>.

259 For body composition outcomes, data was missing for 8 participants due to a technical  
260 fault (LSP n=7). In response to the technical fault, 4 participants' (LSP n = 1) data was  
261 collected in weeks 1 and 14 with bioelectrical impedance (model BC418MA, Tanita, Europe,  
262 UK) and due to the consistent method of assessment in both weeks the data was retained in  
263 the analysis. Changes in fat mass and %fat did not significantly differ between satiety  
264 phenotypes ( $p=.16$ ,  $\eta p^2=0.05$ )<sup>4</sup>. In completers, there was a significant week x satiety  
265 phenotype interaction on fat free mass ( $p=.04$ ,  $\eta p^2=0.10$ ) (non-significant for ITT,  $p=.09$ ,  
266  $\eta p^2=.06$ ), but post hoc comparisons did not reveal any significant differences between  
267 phenotypes ( $p=.06$ ). Waist reductions were significantly greater for the HSP compared to the  
268 LSP (week x satiety phenotype interaction on waist circumference,  $p=.02$ ,  $\eta p^2=.12$ ) and  
269 remained significant when controlling for starting waist circumference ( $p=.02$ ,  $\eta p^2= 0.13$ ).  
270 Changes in hip circumference did not significantly differ between satiety phenotypes ( $p=.10$ ,  
271  $\eta p^2=0.06$ ).

272 *Food intake and food preferences*

273 Snack and total day intake data were missing for two participants due to non-returned  
274 snacks (LSP n=1). The LSP's and HSP's mean energy intake for fixed meals, evening meals  
275 and evening snack are shown in Figure 1. Evening meal and total day EI did not significantly  
276 differ between satiety phenotypes ( $p=.07$ ,  $\eta p^2=0.07$ ), but LSP's snack EI was significantly  
277 greater compared to the HSP ( $p=.02$ ,  $\eta p^2=0.11$ ). There was a significant condition x satiety  
278 phenotype interaction on snack intake ( $p=.04$ ,  $\eta p^2=0.09$ ), which showed that under LED  
279 conditions, LSP's snack energy intake did not differ to HSP's snack energy intake [mean  
280 difference:  $63 \pm \text{SEM } 43$  kcal (24, 149),  $p=.15$ ]. Whereas, under HED conditions, LSP's

---

<sup>3</sup> Percentage weight change at week 2 was a significant predictor of weight change at week 14 (%) ( $p<0.001$ ,  $\eta p^2=0.40$ ). Greater weight loss at week 2 was associated with significantly greater weight loss at week 14 ( $r = .71$ ,  $p<0.001$ )

<sup>4</sup>Percentage weight change at week 2 was a significant covariate of changes in percentage body fat (completers and ITT) and body fat mass at week 14 (ITT only). Greater weight loss at week 2 was associated with greater reductions in body fat mass and percentage body fat at week 14 ( $r = .42$ ,  $p = 0.004$ )

281 snack intake was  $289 \pm \text{SEM } 133$  kcal (22, 556) higher compared to HSP's snack intake  
282 ( $p=.03$ ).

283 For gram intake, snack, evening meal and total day gram intake did not differ between  
284 satiety phenotypes ( $p=.05$ ,  $\eta p^2=0.08$ ). There was a significant condition x satiety phenotype  
285 interaction on evening meal gram intake ( $p=.003$ ,  $\eta p^2=0.17$ ), but post hoc comparisons failed  
286 to reach significance ( $p=.16$ ). No other condition x satiety phenotypes interactions on gram  
287 intake were significant and there were no significant covariates for gram intake.

288 For food preferences, independent of programme type<sup>5</sup>, the LSP showed a greater fat  
289 bias for HED-foods compared to the HSP who showed a greater bias for LED-foods,  $p=.007$ ,  
290  $\eta p^2=0.18$  [explicit liking: LSP:  $9.01 \pm \text{SEM } 3.48$  (1.96, 16.06), HSP:  $-5.98 \pm \text{SEM } 3.57$  (-13.20,  
291 1.25); implicit wanting: LSP:  $17.10 \pm \text{SEM } 7.08$  (2.76, 31.44), HSP:  $-14.26 \pm \text{SEM } 7.26$  (-  
292 28.95, 0.44)].

293 Breakfast and lunch meal palatability ratings did not differ between the satiety  
294 phenotypes ( $p=.23$ ,  $\eta p^2=0.03$ ). Across conditions, the LSP rated the evening meals as less  
295 appealing, less pleasant and less filling compared to the HSP ( $p=.03$ ,  $\eta p^2=0.10$ ) (program type  
296 was a significant covariate for appeal and pleasantness,  $p=.03$ ,  $\eta p^2=0.09$ ). Satisfaction ratings  
297 for the *ad libitum* evening meal did not differ between phenotypes ( $p=.09$ ,  $\eta p^2=0.06$ )  
298 (program type was a significant covariate of evening meal satisfaction,  $p=.04$ ,  $\eta p^2=0.09$ ) (see  
299 Table S1).

### 300 *Appetite control during the program*

301 Compared to the HSP, the LSP felt significantly less in control over what they were  
302 eating, less able to adhere to the program generally and to the food choices encouraged by the  
303 program, and found the program more difficult to follow (see Table 3).

### 304 *Food diaries, sleep and physical activity*

305 Analysis of the food diaries completed at the start and end of the program showed  
306 energy intake did not differ between satiety phenotypes [LSP:  $6881 \pm \text{SEM } 322$  KJ/day (6233,  
307 7530); HSP:  $6254 \pm \text{SEM } 322$  KJ/day (5606, 6902;  $n=25$ ),  $p=.18$ ,  $\eta p^2=0.04$ <sup>6</sup>]. Analysis of the  
308 physical activity monitors worn at the start and end of the program also showed that sleep  
309 duration (LSP:  $7.06 \pm \text{SEM } 0.19$  hours/day [6.67, 7.45]; HSP:  $6.97 \pm \text{SEM } 0.17$  hours/day

<sup>5</sup>Programme type was a significant covariate for liking and wanting ( $p=0.03$ ,  $\eta p^2=0.12$ )

<sup>6</sup>Food diary data  $n = 50$ , missing data due to non-returned diaries (LSP  $n = 1$ ; HSP  $n = 1$ )

310 [6.63, 7.32],  $p=.73$ ,  $\eta p^2=.003$ ) and total physical activity did not differ between phenotypes  
311 (LSP:  $4.29 \pm \text{SEM } 0.45$  hours/day [3.38, 5.21], HSP:  $4.65 \pm \text{SEM } 0.39$  hours/day [3.85, 5.45],  
312  $p=.56$ ,  $\eta p^2=0.01$ )<sup>7</sup>.

### 313 Discussion

314 In this study over a 14 week weight management program, the LSP lost less weight  
315 and had smaller reductions in waist circumference compared to the HSP. Changes in body fat  
316 mass, %fat mass, fat-free mass and hip circumference did not significantly differ between  
317 phenotypes. On test meal days, under HED conditions, the LSP consumed significantly more  
318 energy from snacks compared to the HSP. Under LED conditions, EI did not significantly  
319 differ between LSP and HSP. Additionally, across conditions, the LSP showed a greater drive  
320 for HED-foods compared to the HSP who showed a preference for LED-foods on the LFPQ.  
321 The LSP also reported less control over eating, and found the weight loss program more  
322 difficult to adhere to compared to the HSP.

323 Lower weight loss in the LSP is consistent with one previous study in men, which  
324 reported that the LSP lost less weight over 16-weeks compared to the HSP<sup>(15)</sup>. The  
325 differences in weight loss between satiety phenotypes were similar across studies (current  
326 study: -3.1% versus -6.4%, previous study: -3.3 to -4.3 % versus -5.4 to -6.6 %). Thus, the  
327 current findings confirm that the LSP is linked with poorer weight loss outcomes, and  
328 extends this finding to women. Yet, not all studies have reported that the LSP is linked with  
329 less weight loss, with one study reporting no effects<sup>(22)</sup>. To explain the mixed findings it has  
330 been suggested that the LSP may be particularly influential when participants are following a  
331 satiating diet, and less influential when the LSP are following an energy restricted diet<sup>(22)</sup>.  
332 The current findings do not add support to this explanation as some participants were  
333 following an energy restricted program. Therefore, while the current study reported effects in  
334 a women-only sample, it remains unclear which aspects of the sample or program may affect  
335 the extent to which the LSP will influence weight loss. Nevertheless, the impact of the LSP  
336 on appetite control and weight loss reported here, are consistent with previous research  
337 highlighting that managing appetite control is one of the main challenges to weight loss<sup>(7)</sup>.  
338 The current findings extend previous research by confirming that there are particular  
339 individuals who are least able to detect sensations of fullness, and ultimately have greater  
340 difficulty losing weight. This finding has important implications for weight management

---

<sup>7</sup>Physical activity and sleep total n = 39 participants (LSP n = 17). Missing physical activity and sleep data due to invalid data [ $<5$  days (including  $<1$  weekend day)] (n = 11) or technical issues (n = 2).

341 strategies. For example, weight management programs could screen participants in the early  
342 phases of the program to identify individuals who report a weak ability to detect fullness  
343 sensations, and offer additional support or dietary strategies that promote satiety (e.g. low  
344 energy density strategies) to optimise weight loss. Future research should assess whether such  
345 additional support provided to the LSP can optimise weight loss in this group.

346 However, it is also important to note that there were no significant changes in body  
347 composition between the LSP and HSP. The lack of significant differences in body  
348 composition could be due to a low sample size because body composition data could not be  
349 collected for a sub-sample of participants. It could also be due to body fat being measured in  
350 week 2 and not at the start of the weight loss program. The weight change (%) at week 2 was  
351 a significant covariate of weight change and changes in fat mass and percentage body fat at  
352 the end of the program. Thus, significant differences between phenotypes for changes in body  
353 fat might have been observed if it had been possible to assess body fat at the start of the  
354 weight loss program.

355 Findings from the test meal days suggest that the lower weight loss in the LSP was  
356 due to weaker appetite control. The LSP exhibited a greater drive for HED-foods and under  
357 HED conditions consumed more snacks (kcal) compared to the HSP. This corroborates  
358 previous research which reported that the LSP exhibited a greater drive for high fat-foods and  
359 consumed more energy compared to the HSP(11). Other research has also shown that the  
360 LSP show psychological characteristics linked with overeating such as greater night eating  
361 symptoms, external hunger(14) and trait disinhibition(10, 11). Moreover, in this study during  
362 the weight loss program, the LSP reported less control over eating and more difficulty  
363 adhering to the program compared to the HSP. It seems that for the LSP, detecting fullness  
364 sensations and controlling EI is more challenging compared to the HSP, and over time this  
365 leads to less weight loss. These findings are important because while previous research has  
366 shown that the LSP is linked with less weight loss, this study provides support that the  
367 inferior weight loss is due to weaker appetite control in LSP, as indicated by objective and  
368 self-report measures. Of note, unlike previous research(10, 11) the LSP did not score  
369 significantly higher on trait disinhibition compared to the HSP. While there was a trend for  
370 the LSP to score higher compared to the HSP, this may not have been significant because  
371 trait disinhibition was measured at week 2 of the weight loss program. Trait disinhibition can  
372 decrease during weight loss attempts(32), thus it might be that measuring trait disinhibition at  
373 week 2, rather than at the start of the program minimised the opportunity to observe

374 significant differences between satiety phenotypes. Additionally, the food diaries did not  
375 reveal differences in self-reported intake (possibly due to underreporting and imprecision of  
376 self-reported dietary intakes<sup>(33, 34)</sup>). But, the lack of differences in objectively assessed  
377 physical activity and sleep duration, add support that the differences in weight loss between  
378 satiety phenotypes were attributable to LSP's weaker appetite control.

379 For the first time, this study compared LSP's and HSP's appetite response to meals  
380 varying in energy density. Previous research has only examined appetite responses in the  
381 satiety phenotypes to one type of meal, where energy density has not been manipulated (e.g.  
382 <sup>(11, 14)</sup>). The current findings showed that the LSP only consumed greater EI compared to the  
383 HSP when consuming HED foods, not LED foods. Thus, the LSP may be most susceptible to  
384 overconsumption when consuming HED foods, while LED foods can prevent excessive EI in  
385 LSP. This has important implications for our obesogenic environment where energy dense  
386 foods are readily available<sup>(35)</sup>. Indeed, under LED conditions, the LSP consumed more grams  
387 of food compared to the HSP, but evening meal and snack EI did not differ. These findings  
388 suggest that LED meals provide an effective strategy for the LSP to eat larger quantities of  
389 food without consuming excessive energy.

390 Interestingly, at the start of the trial the LSP had greater resting systolic and diastolic  
391 blood pressure compared to the HSP (albeit, average values were still within clinically  
392 normal ranges<sup>(36)</sup>), even after controlling for starting body weight and BMI. As far as we are  
393 aware, no other studies have reported differences in blood pressure between the satiety  
394 phenotypes. Caution is needed interpreting this difference as blood pressure can vary due to a  
395 number of factors beyond satiety phenotypes, but greater blood pressure is consistent with the  
396 characteristics of the LSP or low satiating efficiency profiles that previous studies have  
397 identified. For instance, stress, intake of high fat foods, overconsumption and shorter sleep  
398 durations are factors associated with high blood pressure that previous research has identified  
399 in the LSP<sup>(11-14, 37)</sup>. More research is needed to support and explain this finding, but it  
400 indicates that the LSP may be associated with wider health implications.

401 There are a number of limitations with this study which mean the findings should be  
402 interpreted with caution. Firstly, due to restrictions on accessing and recruiting volunteers,  
403 the study could not obtain baseline appetite measures prior to engagement in the Slimming  
404 World, UK or NHS Live Well weight loss programs. This is especially of concern because  
405 participants were recruited from two different weight loss programs. Whilst, prior % weight  
406 change during the program (and program type) was controlled for in the analyses, it remains

407 possible that the first weeks of the programs affected appetite responses and the satiety  
408 phenotype grouping rather than the grouping being based on underlying appetite traits per se.  
409 Therefore, study findings need to be interpreted with caution and future research should  
410 include true baseline appetite measures and recruit from one weight loss program to confirm  
411 the role of satiety phenotypes on weight loss. It is also important to note that tertile splits  
412 were conducted on the data meaning that 27 unclassified participants were not included in the  
413 data analyses. Tertile splits were used to be consistent with previous research to allow for  
414 cross study comparisons. However, even though an ANCOVA identified raw SQ scores as a  
415 significant covariate on body weight change, it is not clear whether the estimated effect  
416 applied to the unclassified group. This is important as the unclassified group also had a BMI  
417 classified overweight or obese, and research needs to identify effective strategies for weight  
418 management for this group as well as for the LSP. The study design was also limited by the  
419 absence of a control group not engaged in weight loss. It would be useful to compare weight  
420 changes, food preferences and food intake in response to energy density manipulations in a  
421 group not engaged in weight loss. Also, the *ad libitum* meals provided access to only LED- or  
422 HED-foods. The LSP might have opted for HED-foods if they were available in the LED  
423 conditions, especially as the LSP showed a high drive for HED-foods across both conditions  
424 as measured by the LFPQ. Further research could provide a selection of LED- and HED-food  
425 options at the *ad libitum* evening meal and assess food choice and intake. Methods to assess  
426 weight also varied with participants being weighed on scales during week 1 and weighed  
427 under standardised conditions (fasted) using air plethysmography in week 2 and 14. However  
428 all participants underwent these mixed methods of assessment and as such, the resulting  
429 variance was unlikely to have differed between the satiety phenotypes. Additionally, appetite  
430 control was assessed behaviourally and it would be useful for future research to incorporate  
431 biomarkers of appetite control to further characterise the LSP and HSP. Menstrual phase  
432 (date of last cycle and average cycle length) was assessed during study screening and of the  
433 completed responses, at the start of the weight management program there did not appear to  
434 be a difference in the number of LSPs and HSPs in the follicular or luteal phases. However, a  
435 number of participants did not provide complete answers or reported either irregular or no  
436 menstruation (n = 30) meaning no formal analyses on this data could be reported. Therefore,  
437 future studies should collect more information on menstrual phase and control for its possible  
438 influence on appetite control on the test meal days and weight change<sup>(38, 39)</sup>. Finally, the study  
439 was slightly underpowered by two participants and the body composition analyses were  
440 conducted on a sub-sample of participants. As such, replication of these study findings in



441 larger samples and different populations, along with systematic reviews and meta-analyses of  
442 multiple studies are recommended before informed conclusions about the impact of satiety  
443 phenotypes on weight loss can be drawn.

#### 444 **Conclusion**

445 The ability to resist the drive to eat varies from person to person. This can be measured  
446 by the strength of satiety responsiveness. Low satiety responsiveness is detrimental for  
447 weight loss but LED dietary strategies may improve appetite control in the LSP. Further  
448 research exploring these satiety behavioural phenotypes is highly warranted.

#### 449 **Acknowledgements**

450 This trial was registered on ClinicalTrials.gov #NCT02012426. We thank the women who  
451 participated in this trial. We also grateful to Dr Stephen Whybrow (University of Aberdeen)  
452 for analysing the food diaries. The current datasets are available from the corresponding  
453 author on reasonable request.

#### 454 **Financial support**

455 The trial was funded by Slimming World UK. Slimming World UK supported the design of the test  
456 meals and recruitment. The funder had no role in the analysis or writing of this article.

#### 457 **Conflict of interest**

458 JL is employed by Slimming World, UK; JS consults for Slimming World through the University of  
459 Leeds consulting service. All other authors have no conflicts of interest.

#### 460 **Authorship:**

461 NB, JB, JS and GF designed the research; JL supported the design of the meals and  
462 recruitment; NB, DC and FC conducted the trial; AM integrated and processed the physical  
463 activity data. NB performed statistical analyses and wrote the manuscript. All authors read  
464 and approved the final manuscript.

465

466

467 **References**

- 468 1. Public Health England. Health matters: obesity and the food environment 2017. Available  
469 from: [https://www.gov.uk/government/publications/health-matters-obesity-and-the-food-](https://www.gov.uk/government/publications/health-matters-obesity-and-the-food-environment/health-matters-obesity-and-the-food-environment--2)  
470 [environment/health-matters-obesity-and-the-food-environment--2](https://www.gov.uk/government/publications/health-matters-obesity-and-the-food-environment/health-matters-obesity-and-the-food-environment--2). Accessed: June 2017.
- 471 2. Wardle J & Johnson F (2002) Weight and dieting: examining levels of weight concern in  
472 British adults. *Int J Obes* **26**, 1144-9.
- 473 3. Teixeira PJ, Going SB, Sardinha LB et al. (2005) A review of psychosocial pre-treatment  
474 predictors of weight control. *Obes Rev.* **6**, 43-65.
- 475 4. Crawford D, Jeffery RW, French SA (2000) Can anyone successfully control their weight?  
476 Findings of a three year community-based study of men and women. *Int J Obes* **24**, 1107-10.
- 477 5. Wing RR, Hill JO (2001). Successful weight loss maintenance. *Annu. Rev. Nutr.* **21**, 323-41.
- 478 6. Mann T, Tomiyama AJ, Westling E et al. (2007) Medicare's Search for Effective Obesity  
479 Treatments: Diets Are Not the Answer. *Am. Psychol.* **62**, 220-33.
- 480 7. Stubbs J, Brogelli D, Pallister C et al. (2012) Behavioural and motivational factors associated  
481 with weight loss and maintenance in a commercial weight management programme. *Open Obes J.* **4**,  
482 35-43.
- 483 8. Womble LG, Williamson DA, Greenway FL et al. (2001) Psychological and behavioral  
484 predictors of weight loss during drug treatment for obesity. *Int J Obes.* **25**, 340-5.
- 485 9. Drapeau V, Hetherington M, Tremblay A (2011). *Impact of Eating and Lifestyle Behaviors on*  
486 *Body Weight: Beyond Energy Value*. Handbook of Behavior, Food and Nutrition 693-706.
- 487 10. Barkeling B, King NA, Naslund E, et al. (2007) Characterization of obese individuals who  
488 claim to detect no relationship between their eating pattern and sensations of hunger or fullness. *Int J*  
489 *Obes.* **31**, 435-9.
- 490 11. Dalton M, Hollingworth S, Blundell J et al. (2015) Weak Satiety Responsiveness Is a Reliable  
491 Trait Associated with Hedonic Risk Factors for Overeating among Women. *Nutrients* **7**, 7421-36.
- 492 12. Drapeau V, Blundell J, Therrien F, et al. (2005) Appetite sensations as a marker of overall  
493 intake. *Br. J. Nutr.* **93**, 273-80.
- 494 13. Drapeau V, King N, Hetherington Met al. (2007) Appetite sensations and satiety quotient:  
495 Predictors of energy intake and weight loss. *Appetite* **48**, 159-66.
- 496 14. Drapeau V, Blundell J, Gallant AR et al. (2013) Behavioural and metabolic characterisation  
497 of the low satiety phenotype. *Appetite* **70**, 67-72.
- 498 15. Arguin H, Tremblay A, Blundell JE et al. (2017) Impact of a non-restrictive satiating diet on  
499 anthropometrics, satiety responsiveness and eating behaviour traits in obese men displaying a high or  
500 a low satiety phenotype. *Br. J. Nutr.* **118**, 750-60.
- 501 16. Finlayson G, King N, Blundell J (2008). The role of implicit wanting in relation to explicit  
502 liking and wanting for food: Implications for appetite control. *Appetite* **50**, 120-7.
- 503 17. Stubbs J, Ferres S, Horgan G (2000). Energy density of foods: Effects on energy intake. *Crit.*  
504 *Rev. Food Sci. Nutr.* **40**, 481-515.
- 505 18. Bell EA, & Rolls BJ (2001). Energy density of foods affects energy intake across multiple  
506 levels of fat content in lean and obese women. *Am J Clin Nutr.* **73**, 1010-8.
- 507 19. Buckland NJ, Camidge D, Croden F, et al. (2018) A Low Energy-Dense Diet in the Context  
508 of a Weight-Management Program Affects Appetite Control in Overweight and Obese Women. *J.*  
509 *Nutr.* **148**, 798-806.
- 510 20. Epstein LH, Truesdale R, Wojcik A, et al. (2003) Effects of deprivation on hedonics and  
511 reinforcing value of food. *Physiol Behav.* **78**, 221-7.

- 512 21. Goldstone AP, de Hernandez CGP, Beaver JD et al. (2009) Fasting biases brain reward  
513 systems towards high-calorie foods. *Eur J Neurosci.* **30**,1625-35.
- 514 22. Drapeau V, Jacob R, Panahi S et al. (2019) Effect of Energy Restriction on Eating Behavior  
515 Traits and Psychobehavioral Factors in the Low Satiety Phenotype. *Nutrients* **11**, 245.
- 516 23. Faul F, Erdfelder E, Lang A-G, et al. (2007) G\*Power 3: A flexible statistical power analysis  
517 program for the social, behavioral, and biomedical sciences. *Behav Res Methods* **39**, 175-91.
- 518 24. Stubbs RJ, Morris L, Pallister C et al. (2015) Weight outcomes audit in 1.3 million adults  
519 during their first 3 months' attendance in a commercial weight management programme. *BMC Public*  
520 *Health* **15**, 882
- 521 25. NHS-Choices. Start the NHS weight loss plan 2015. Available from:  
522 <http://www.nhs.uk/Livewell/weight-loss-guide/Pages/losing-weight-getting-started.aspx>. Accessed  
523 January 2014.
- 524 26. Stunkard AJ & Messick S (1985) The three-factor eating questionnaire to measure dietary  
525 restraint, disinhibition and hunger. *J Psychosom Res* **29**, 71-83.
- 526 27. Blundell J, de Graaf C, Hulshof T et al. (2010) Appetite control: methodological aspects of  
527 the evaluation of foods. *Obes. Rev.* **11**, 251-70.
- 528 28. Myers A, Gibbons C, Finlayson G, Blundell J (2017). Associations among sedentary and  
529 active behaviours, body fat and appetite dysregulation: investigating the myth of physical  
530 inactivity and obesity. *Br J Sports Med* **51**,1540-4.
- 531 29. Finglas PM, Roe MA, Pinchen HM et al. (2015) *McCance and Widdowson's The*  
532 *Composition of Foods*. Seventh summary edition ed. Cambridge: Royal Society of Chemistry.
- 533 30. Elobeid MA, Padilla MA, McVie T et al. (2009) Missing Data in Randomized Clinical Trials  
534 for Weight Loss: Scope of the Problem, State of the Field, and Performance of Statistical Methods.  
535 *Plos One* **4**, e6624.
- 536 31. Field A. (2005) *Discovering Statistics using SPSS (introducing statistical methods series)*.  
537 Second Edition ed: Sage Publications Ltd.
- 538 32. Butryn ML, Thomas JG, Lowe MR (2009) Reductions in Internal Disinhibition During  
539 Weight Loss Predict Better Weight Loss Maintenance. *Obesity* **17**, 1101-3.
- 540 33. Poppitt SD, Swann D, Black AE et al. (1998) Assessment of selective under-reporting of food  
541 intake by both obese and non-obese women in a metabolic facility. *Int J Obes* **22**, 303-11.
- 542 34. Heitmann BL, Lissner L (1995) Dietary underreporting by obese individuals-is it specific or  
543 non-specific? *Br Med J.* **311**, 986-9.
- 544 35. Wadden TA, Brownell KD, Foster GD (2002) Obesity: Responding to the Global Epidemic.  
545 *J. Consult. Clin. Psychol.* **70**, 510-25.
- 546 36. National Health Service. High blood pressure (hypertension) (2016). Available from:  
547 <https://www.nhs.uk/conditions/high-blood-pressure-hypertension/>. Accessed June 2017.
- 548 37. McNeil J, Drapeau V, Gallant AR et al. (2013). Short sleep duration is associated with a  
549 lower mean satiety quotient in overweight and obese men. *Eur J Clin Nutr.* **67**,1328-30.
- 550 38. Geiker NRW, Ritz C, Pedersen SD, Larsen TM, Hill JO, Astrup A. (2016). A weight-loss  
551 program adapted to the menstrual cycle increases weight loss in healthy, overweight,  
552 premenopausal women: a 6-mo randomized controlled trial. *Am J Clin Nutr.* **104**,15-20.
- 553 39. Brennan IM, Feltrin KL, Nair NS, Hausken T, Little TJ, Gentilcore D, et al. (2009). Effects of  
554 the phases of the menstrual cycle on gastric emptying, glycemia, plasma GLP-1 and insulin,  
555 and energy intake in healthy lean women. *Am. J. Physiol. Gastrointest. Liver Physiol.* **297**,  
556 G602-G10.
- 557

558 **List of Figures**

559 **Figure 1.** Mean ( $\pm$ SEM) energy intake under low and high energy density (LED, HED)  
560 conditions in the low and high satiety phenotypes (LSP, HSP).

ACCEPTED MANUSCRIPT

**Table 1.** Mean  $\pm$  SD (95% confidence intervals) baseline characteristics for the low and high satiety phenotypes.

	Low satiety phenotype (n = 26)	High satiety phenotype (n = 26)	<i>p</i>	$\eta p^2$
<b>SQ (mm/kcal)</b>	1.05 $\pm$ 2.76 (-0.06, 2.16)	12.64 $\pm$ 3.40 (11.27, 14.02)	<.001	.77
<b>Week 2 weight change (%)</b>	-2.12 $\pm$ 1.64 (-2.79, -1.46)	-2.97 $\pm$ 1.60 (-3.61, -2.32)	.06	.07
<b>Age (years)</b>	39.31 $\pm$ 11.33 (34.73, 43.88)	44.54 $\pm$ 12.06 (39.67, 49.41)	.14	.05
<b>Height (m)</b>	1.65 $\pm$ 0.06 (1.63, 1.68)	1.63 $\pm$ 0.08 (1.60, 1.66)	.43	.01
<b>Weight (kg)</b>	94.42 $\pm$ 13.39 (89.02, 99.83)	90.99 $\pm$ 13.72 (85.36, 96.44)	.56	.01
<b>BMI (kg/m<sup>2</sup>)</b>	34.41 $\pm$ 3.61 (32.95, 35.86)	33.99 $\pm$ 3.40 (32.61, 35.36)	.84	.01
<b>Fat mass (kg)<sup>a</sup></b>	43.52 $\pm$ 11.50 (37.98, 49.07)	40.92 $\pm$ 9.60 (36.96, 44.88)	.35	.02
<b>% Fat<sup>a</sup></b>	45.89 $\pm$ 6.97 (42.54, 49.25)	45.92 $\pm$ 4.59 (44.02, 47.81)	.81	.01
<b>FFM (kg)<sup>a</sup></b>	50.25 $\pm$ 6.58 (47.08, 53.42)	47.34 $\pm$ 5.69 (45.00, 49.69)	.35	.02
<b>RMR (kcal/day)</b>	1750 $\pm$ 280 (1637, 1863)	1628 $\pm$ 243 (1533, 1722)	.23	.03
<b>Waist (cm)<sup>b</sup></b>	109.64 $\pm$ 13.36 (104.12, 115.15)	108.21 $\pm$ 11.26 (103.46, 112.97)	.73	.01
<b>Hip (cm)</b>	118.12 $\pm$ 11.15 (113.61, 122.62)	116.75 $\pm$ 10.23 (112.61, 120.80)	.99	.00
<b>Systolic (mmHg)<sup>c</sup></b>	122.44 $\pm$ 13.71 (116.91, 127.98)	111.76 $\pm$ 12.15 (106.74, 116.78)	.01	.13
<b>Diastolic (mmHg)<sup>c</sup></b>	84.29 $\pm$ 11.16 (79.78, 88.80)	75.58 $\pm$ 8.63 (72.02, 79.14)	.01	.14
<b>Heart rate (bpm)<sup>d</sup></b>	63.96 $\pm$ 8.35 (60.52, 67.40)	61.82 $\pm$ 9.12 (58.05, 65.59)	.69	.01
<b>Fasting glucose<sup>d</sup></b>	4.84 $\pm$ 0.78 (4.51, 5.16)	4.90 $\pm$ 0.64 (4.64, 5.17)	.81	.01
<b>TFEQ Restraint</b>	9.50 $\pm$ 3.17 (8.22, 10.78)	8.69 $\pm$ 3.33 (7.35, 10.04)	.15	.04
<b>TFEQ Disinhibition</b>	10.54 $\pm$ 3.18 (9.25, 11.82)	9.92 $\pm$ 2.92 (8.74, 11.10)	.99	.00
<b>TFEQ Hunger</b>	7.23 $\pm$ 3.54 (5.80, 8.66)	5.96 $\pm$ 3.14 (4.69, 7.23)	.50	.01

Note.

<sup>a</sup>LSP n = 19; HSP n = 25.

<sup>b</sup>LSP n = 25; HSP n = 24.

<sup>c</sup>HSP n = 25; Comparisons controlled for weight loss program and percentage weight change at week 2.

<sup>d</sup>LSP n = 25; HSP n = 25.

BMI = body mass index.

SQ = satiety quotient.

Week 2 weight change refers to percentage weight change since starting the weight loss programme and the measures session completed in week 2.

TFEQ = Three Factory Eating Questionnaire.

Comparisons between the low and high satiety phenotype.

\**p* < .05 different from LSP, controlling for week 1 body weight and body mass index.

\*\*\**p* < .001 different from LSP

**Table 2.** Mean  $\pm$  SD (95% confidence intervals) changes in study outcomes for the low and high satiety phenotypes in completers and last observation carried forward analyses (LOCF).

	<b>n</b>	<b>Low satiety phenotype</b>	<b>High satiety phenotype</b>	<b>p</b>	<b><math>\eta^2</math></b>
<b>% weight change</b>					
Completers	52	-3.11 $\pm$ 3.43 (-4.49, -1.72)	-6.35 $\pm$ 4.23 (-8.05, -4.64)	0.02	0.10
LOCF	55	-3.19 $\pm$ 3.39 (-4.53, -1.85)	-5.88 $\pm$ 4.50 (-7.63, -4.14)	0.08	0.06
<b>Weight (kg)</b>					
Completers	52	-2.89 $\pm$ 3.08 (-4.13, -1.64)	-5.71 $\pm$ 3.65 (-7.19, -4.23)	0.02	0.10
LOCF	55	-2.97 $\pm$ 3.04 (-4.17, -1.76)	-5.28 $\pm$ 3.93 (-6.80, -3.76)	0.08	0.06
<b>Fat mass (kg)</b>					
Completers	44 <sup>a</sup>	-0.91 $\pm$ 2.02 (-1.88, 0.07)	-2.69 $\pm$ 3.19 (-4.01, -1.37)	ns	0.01
LOCF	47 <sup>a</sup>	-0.93 $\pm$ 1.97 (-1.85, -0.01)	-2.28 $\pm$ 3.42 (-3.63, -0.93)	ns	0.01
<b>Percentage fat</b>					
Completers	44 <sup>a</sup>	-0.64 $\pm$ 1.41 (-1.32, 0.04)	-1.60 $\pm$ 2.68 (-2.71, -0.49)	ns	0.01
LOCF	47 <sup>a</sup>	-0.60 $\pm$ 1.38 (-1.25, 0.04)	-1.35 $\pm$ 2.75 (-2.44, -0.26)	ns	0.01
<b>Fat free mass (kg)</b>					
Completers	44 <sup>a</sup>	0.22 $\pm$ 1.20 (-0.36, 0.79)	-0.42 $\pm$ 1.09 (-0.88, 0.03)	0.04	0.10
LOCF	47 <sup>a</sup>	0.13 $\pm$ 1.23 (-0.45, 0.70)	-0.39 $\pm$ 1.08 (-0.82, 0.04)	ns	0.06
<b>Waist Circumference (cm)</b>					
Completers	49 <sup>b</sup>	-0.66 $\pm$ 3.97 (-2.30, 0.98)	-3.30 $\pm$ 2.84 (-4.50, -2.10)	0.01 <sup>c</sup>	0.13
LOCF	49	-0.66 $\pm$ 3.97 (-2.30, 0.98)	-3.30 $\pm$ 2.84 (-4.51, -2.10)	0.01 <sup>c</sup>	0.13
<b>Hip Circumference (cm)</b>					
Completers	52	-0.21 $\pm$ 4.86 (-2.18, 1.75)	-2.54 $\pm$ 4.28 (-4.27, -0.81)	ns	0.06
LOCF	55	0.28 $\pm$ 4.78 (-1.61, 2.17)	2.19 $\pm$ 4.33 (0.51, 3.87)	ns	0.04

*Note.*

Negative values indicate decreases between weeks.

All comparisons controlled for weight loss program (Slimming World, UK or NHS Live Well program) and weight change at week 2 (%).

<sup>a</sup>For fat mass, percentage fat mass and fat free mass, data was missing from eight participants due to a fault with the BodPod.

<sup>b</sup>Missing data from three participants due to measurement issues (low satiety phenotype n = 1).

<sup>c</sup>Remained significant when controlling for starting waist circumference ( $p < .05$ ).

**Table 3.** M  $\pm$  SEM (95% confidence intervals) self-reported appetite control during the program for the low and high satiety phenotypes.

	<b>Low satiety phenotype</b>	<b>High satiety phenotype</b>	<b><i>p</i></b>	<b><i><math>\eta p^2</math></i></b>
How much do you feel IN CONTROL of what you're eating?	50.3 $\pm$ 4.6 (40.9, 59.7)	73.0 $\pm$ 4.7 (63.4, 82.7)	0.01	0.19
Have you felt able to stick to your plan regarding your food choices?	43.6 $\pm$ 4.1 (35.3, 51.9)	61.9 $\pm$ 4.2 (53.4, 70.5)	0.01	0.18
How WELL have you managed to stick with the program?	39.8 $\pm$ 4.3 (31.0, 48.6)	60.1 $\pm$ 4.4 (51.0, 69.1)	0.01	0.18
How EASY do you find it to stick to your weight control program?	46.6 $\pm$ 4.8 (36.8, 56.4)	66.0 $\pm$ 5.0 (55.9, 76.1)	0.05	0.12

*Note.*

There was missing data for 17 participants due to non-returned questionnaires; total sample size n = 35 (Low satiety phenotype, n = 18).

Responses ranged from '0 = not at all' to '100 = very'.

All comparisons controlled for weight loss program (Slimming World, UK or NHS Live Well program) and weight change at week 2 (%).

ACCEPTED MANUSCRIPT

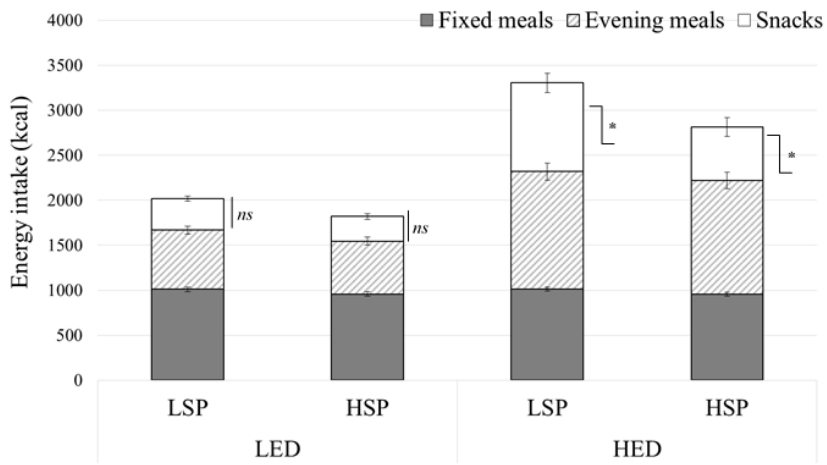


Figure 1.

\* $p < 0.05$  between LSP and HSP

ACCEPTED MANUSCRIPT