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1 Full title: Structured, aerobic exercise reduces fat mass and is partially compensated through
2 energy intake but not energy expenditure in women

3 Short title: Compensatory EI and EE after structured exercise in women

4 ¹Anna Myers*, ²Michelle Dalton, ³Catherine Gibbons, ³Graham Finlayson & ³John Blundell

5 ¹Centre for Sport and Exercise Science, Faculty of Health and Wellbeing, Sheffield Hallam
6 University

7 ²School of Social and Health Sciences, Leeds Trinity University, Leeds, United Kingdom

8 ³Appetite Control and Energy Balance Research, School of Psychology, Faculty of Medicine
9 and Health, University of Leeds, Leeds, United Kingdom

10

11 *Corresponding Author:

12 Dr Anna Myers
13 Centre for Sport and Exercise Science
14 Sheffield Hallam University
15 Sheffield
16 S10 2BP

17 a.myers@shu.ac.uk

18 0114 225 2274

19

20 Michelle Dalton (m.dalton@leedstrinity.ac.uk), Catherine Gibbons

21 (C.Gibbons@leeds.ac.uk), Graham Finlayson (G.S.Finlayson@leeds.ac.uk) John Blundell

22 (J.E.Blundell@leeds.ac.uk),

23 **ABSTRACT**

24 **Background**

25 Exercise-induced weight loss is often less than expected and highly variable in men and
26 women. Behavioural compensation for the exercise-induced energy deficit could be through
27 energy intake (EI), non-exercise physical activity (NEPA) or sedentary behaviour (SB). We
28 investigated this issue in women.

29 **Methods**

30 Twenty-four overweight [body mass index (BMI) $M=27.9 \text{ kg/m}^2$, $SD=2.7$] women [age
31 $M=33.1$ years, $SD=11.7$] completed 12-weeks of supervised exercise (5x500kcal per week) in
32 a non-randomised pre-post intervention study. Body mass (BM), waist circumference (WC),
33 body composition, resting metabolic rate (RMR), total daily EI, individual meals, appetite
34 sensations and appetite-related peptides were measured at baseline (week 0) and post-
35 intervention (week 12). Free-living physical activity (PA) and SB were measured (SenseWear)
36 at baseline, week 1 and 10 of the exercise intervention, and at post-intervention (week 13).

37 **Results**

38 Following the 12-week exercise intervention BM [$p=.04$], BMI [$p=.035$], WC [$p<.001$] and fat
39 mass (FM) [$p=.003$] were significantly reduced, and fat-free mass (FFM) significantly
40 increased [$p=.003$]. Total [$p=.028$], ad libitum [$p=.03$] and snack box EI [$p=.048$] were
41 significantly increased and this was accompanied by an increase in hunger [$p=.01$] and a
42 decrease in fullness [$p=.03$] before meals. The peptides did not explain changes in appetite

43 [p>.05]. There was no compensatory reduction in NEPA [p>.05] and no increase in SB, rather
44 there was a decrease in SB during the exercise intervention [p=.03].

45 **Conclusions**

46 Twelve-weeks of supervised aerobic exercise resulted in a significant reduction in FM and an
47 increase in FFM. Exercise increased hunger and EI which only partially compensated for the
48 increase in energy expenditure. There was no evidence for a compensatory reduction in NEPA
49 or an increase in SB. Dietary intervention, as an adjunct to exercise, may offset the
50 compensatory increase in EI and result in a greater reduction in BM.

51 **Trial registration**

52 Our trial was retrospectively registered on the International Standard Randomised Controlled
53 Trials Registry (ISRCTN78021668, 27th September 2016) and can be found here:
54 <https://doi.org/10.1186/ISRCTN78021668>

55 **KEY WORDS**

56 Exercise, appetite control, weight loss, compensation, non-exercise physical activity, sedentary
57 behaviour

58

59

60 **BACKGROUND**

61 There is much discussion about the role of physical activity (PA) and/or exercise for reducing
62 obesity and promoting weight maintenance. The scepticism surrounding the efficacy of PA for
63 weight management arises from the observation that weight loss as a result of exercise
64 interventions is often less than expected (1) and the belief that increased exercise-induced
65 energy expenditure (EE) is automatically countered by an increase in energy intake (EI) (2).
66 Despite this, observational studies demonstrate that habitual PA is associated with lower body
67 mass (BM) and fat mass (FM) (3, 4). Furthermore, experimental studies have shown that
68 structured exercise results in reduced BM and FM, often with an increase or preservation of
69 fat-free mass (FFM) (5-7). Exercise and/or PA is also a strong predictor of weight loss
70 maintenance (8). The evidence demonstrates that exercise is an integral component of weight
71 management interventions (5).

72 Despite significant reductions in average BM and FM with exercise, weight loss is often less
73 than the theoretically predicted reduction based on the exercise-induced EE, even when
74 adherence to the exercise intervention is strictly supervised and monitored and compliance is
75 high (1, 7). This less than theoretically predicted weight loss could, in part, be due to the use
76 of overly simplistic and static predictive equations that do not account for dynamic
77 physiological adaptations to weight loss and therefore overestimate the weight loss resulting
78 from a particular exercise-induced energy deficit (9). Additionally, compensation in response
79 to the energy deficit generated by the exercise regime would attenuate weight loss. This
80 compensation could arise through an increase in EI (7, 10), or compensation that acts to reduce
81 total daily EE such as a decrease in non-exercise physical activity (NEPA) or an increase in
82 sedentary behaviour (SB) (or subtle combinations of all these components of energy balance)
83 (11, 12). The literature regarding changes in EI, NEPA and SB in response to structured

84 exercise is conflicting and many studies lack accurate and reliable measures of EI, EE, NEPA
85 and SB (13, 14).

86 This study applied objective methodology to assess the influence of an exercise regime on EI
87 (food intake, appetite sensations and appetite-related peptides) and EE (PA and SB outside of
88 the structured exercise) in women. The specific objective was to examine whether a 12-week
89 supervised, structured aerobic exercise regime generated compensation through appetite,
90 NEPA or SB.

91 **METHODS**

92 **Participants**

93 Thirty-two overweight or obese inactive women were recruited to take part in the study. Only
94 women were recruited to reduce unwanted variability in the design. Of those 32 participants,
95 24 women aged 33.1 years (SD = 11.7) with a body mass index (BMI) of 27.9 kg/m² (SD =
96 2.7) completed the study. The following reasons were given for participant dropouts: did not
97 like exercise (week 1; n=1); exercise related injury (week 4; n=1); did not comply with
98 procedures (week 4; n=1); personal reasons (week 6; n=1); no reason provided (week 7; n=1);
99 time commitment of exercise too much (week 10; n=2); illness (week 12; n=1). Participants
100 were recruited from the University of Leeds, UK, and surrounding area using posters and email
101 mailing lists. An online screening survey was completed to assess the eligibility of potential
102 participants based on the following criteria: women aged 18-55 years, BMI between 25.0 and
103 34.9 kg/m², not currently dieting to lose weight, inactive (less than 150 min/week of moderate-
104 to-vigorous PA (MVPA) assessed by questionnaire), no increase in PA in previous four weeks,
105 weight stable (no significant weight loss ($\geq 5\%$) in the previous 6 months), non-smokers, not

106 taking any medication or have any medical condition known to affect metabolism or appetite,
107 and acceptance of the study foods (≥ 3 liking of study foods on 7-point Likert scale). All
108 participants provided written informed consent before taking part in the study. The study
109 procedures and all study materials were reviewed and approved by the National Research
110 Ethics Service Committee Yorkshire & the Humber (ref: 09/H1307/7).

111 **Design**

112 This study was a non-randomised pre-post study with a 12-week supervised aerobic exercise
113 intervention. Anthropometrics, body composition and resting metabolic rate (RMR) were
114 taken before (week 0) and at the end of the exercise intervention (week 12). Participants also
115 completed two probe days prior to the exercise intervention (week 0) commencing and two in
116 the final week (week 12) of the exercise intervention to assess eating behaviour and
117 subjective appetite sensations. On both measures and probe days, the participants arrived at
118 the research unit between 07:00 and 09:00 following a 10 hour fast (no food or drink except
119 water). Free-living PA and SB were measured before (week -1), during (week 1 and week 10)
120 and after (week 13) the intervention.

121 **Measures days**

122 A range of measurements were performed at week 0 (baseline) and week 12. Participants
123 arrived at the laboratory following an overnight fast. RMR was measured (GEM, NutrEn
124 Technology Ltd, Cheshire, UK) with participants laying supine for 40 min during which
125 expired air was collected using a ventilated hood system. VO_2 and VCO_2 values were
126 sampled every 30 seconds. The average of the final 30 min values was deemed to be the
127 RMR expressed as kcal/d. BM and body composition (fat mass (FM) and fat-free mass

128 (FFM)) were measured using the BODPOD (Body Composition Tracking System, Life
129 Measurement, Inc., Concord, USA) which uses air displacement plethysmography.
130 Participants wore tight clothing and a swim cap to allow for an accurate measure of body
131 volume. Height was measured using a stadiometer (Seca Ltd., Birmingham, UK) and waist
132 circumference (WC) was measured horizontally in line with the umbilicus.

133 **Probe days**

134 Twenty-four hour EI and subjective appetite sensations were measured during the probe day
135 visits. Participants were provided with an individually fixed energy breakfast (25% of
136 measured RMR) of muesli and milk and a choice of tea, coffee or water and were instructed
137 to consume all food and drink within 10 min. The macronutrient composition of the breakfast
138 was fixed at 55%, 30% and 15% for carbohydrate, fat and protein, respectively. Participants
139 remained in the laboratory between breakfast and lunch and were able to use a desktop
140 computer/laptop, listen to music or read.

141 Four hours after breakfast, an ad libitum lunch consisting of chilli with rice, and strawberry
142 yoghurt with double cream was provided with water. Participant were then free to leave the
143 laboratory between lunch and dinner but were not allowed to consume any food or drink
144 except the bottle of water provided.

145 Participants returned to the laboratory four hours later for the ad libitum dinner of tomato and
146 herb risotto, garlic bread, salad items, chocolate brownies and water. An ad libitum snack box
147 containing an apple, two mandarins, roast ham, cheese, bread, margarine, crisps, chocolate
148 buttons and a vanilla yoghurt was given to participants to take home in the evening.

149 Participants could eat any food items from the snack box but were instructed not to share the

150 foods. Participants returned the snack box containing any uneaten foods and food packaging
151 the following day. All of the ad libitum meals were presented in excess of expected
152 consumption and participants were instructed to eat until they reached a comfortable level of
153 fullness. EI was calculated by weighing foods to the nearest 0.1 g before and after
154 consumption and using energy equivalents for protein, fat and carbohydrate of 4, 9 and 3.75
155 kcal/g, respectively, and nutritional information from the manufacturers' food labels.

156 During probe days visual analogue scales (VAS) were completed immediately before and
157 after meals and periodically between meals to assess subjective appetite sensations using a
158 validated electronic appetite rating system (15). Area under the curve (AUC) was calculated
159 using the trapezoid method for subjective feelings of hunger, fullness, desire to eat and
160 prospective foods consumption throughout the whole day (post-breakfast (0 min), +15 min,
161 +30 min, +60 min, +90 min, +120 min, +180 min, +230 min, pre-lunch (+235 min), post-
162 lunch (+260 min), +300 min, +360 min, +420 min, pre-dinner (+480 min), post-dinner (+500
163 min), +540 min, +600 min).

164 EI and subjective appetite sensations were averaged across the two baseline probe days and
165 the two post-intervention probe days to provide a single measure of EI and subjective appetite
166 sensations at both time points. Data were averaged in this way because, as part of a wider
167 project, the two probe days involved the consumption of a novel yoghurt or a calorie and
168 energy matched control yoghurt immediately after breakfast. As the two different yoghurts
169 had no effect on any of the outcome measures in this study, we included it as part of the total
170 breakfast intake and averaged the probe days at baseline and post intervention to give a more
171 robust pre and post intervention measure.

172 **Free-living physical activity, sedentary behaviour and energy expenditure**

173 Free-living PA, SB and EE were measured using the SenseWear Armband mini (SWA;
174 BodyMedia, Inc., Pittsburgh, PA), as has previously been described (3). Measures were
175 completed before the exercise intervention (week -1), week 1 and week 10 of the exercise
176 intervention and post-intervention (after the exercise intervention was complete; week 13).
177 Participants wore the SWA at all times apart from when showering, bathing or swimming, this
178 included wearing the SWA during structured exercise sessions. Participants wore the SWA on
179 the posterior surface of their upper non-dominant arm for a minimum of 22 hours/d for 7-8
180 days. The SWA measures motion (triaxial accelerometer), galvanic skin response, skin
181 temperature and heat flux. Proprietary algorithms available in the accompanying software
182 (SenseWear Professional software version 8.0, algorithm v5.2) calculate EE and classify the
183 intensity of activity. SB was classified as <1.5 METs, light 1.5-2.9 METs, moderate 3-5.9
184 METs and vigorous >6 METs (16). Moderate and vigorous PA was grouped together to form
185 one MVPA category to correspond with the guidelines for PA. Activity EE was calculated by
186 summing the energy expended in activities >1.5 METs. PA and SB variables were expressed
187 as average min/d and activity EE was expressed as average kcal/d by dividing the total min/d
188 or kcal/d recorded during the whole wear period by the number of days participants wore the
189 SWA. For a wear period to be valid there had to be ≥ 5 days of valid data (≥ 22 hours/d)
190 including ≥ 1 weekend day (17). The SWA has been shown to accurately estimate time spent
191 in sedentary, light and moderate activities, total EE, EE at rest and EE during free-living light
192 and moderate intensity PA (18-21).

193 **Non-exercise physical activity**

194 The duration of weekly prescribed exercise was averaged over 7 days for week 1 (M = 47.30
195 min/d, SD = 6.96) and week 10 (M = 40.16 min/d, SD = 5.83) of the exercise intervention.
196 Average structured exercise minutes per day was then subtracted from time spent in MVPA

197 per day measured using the SWA during week 1 and week 10 of the exercise intervention to
198 determine NEPA MVPA. Similarly, the five day exercise-induced EE (2500 kcal) was
199 averaged over 7 days (357.14 kcal/d) and subtracted from activity EE measured using the SWA
200 during week 1 and week 10 of the exercise intervention to determine NEPA activity EE.

201 **Exercise intervention**

202 Participants were required to exercise at the laboratory exercise facility five times per week for
203 12-weeks. Each exercise session was individually tailored to expend 500 kcal at 70% of their
204 HR maximum (2500 kcal/wk). Participants completed a maximal treadmill fitness test and
205 expired air was collected and analysed using indirect calorimetry (SensorMedics Vmax29,
206 California, USA) to calculate EE during exercise. Standard stoichiometric equations were used
207 with respiratory data (VO_2/VCO_2) to calculate the energy expended at 70% HR maximum (22).
208 To account for changes in fitness and BM, a further VO_2 max test was performed during week
209 six of the intervention to recalculate the exercise duration required to expend 500 kcal at 70%
210 HR maximum. Compliance with the exercise intervention was monitored and tracked daily
211 using HR monitors (S610, POLAR, Finland) to ensure the correct intensity and duration of
212 exercise was achieved. Participants could choose from a selection of exercise equipment:
213 bicycle ergometers, cross-trainers, rowing ergometers and treadmills. Participants could attend
214 the laboratory exercise facility between 7 am and 7 pm Monday – Friday. The facility could
215 accommodate up to 6 participants exercising at any one time. The target total EE over the 12-
216 week exercise intervention was 29,000 kcal for each participant. If participants missed an
217 exercise session for any reason they were required to make up the time they had missed by
218 exercising for longer on other days or exercising away from the laboratory over the weekend
219 providing they recorded their exercise session with the HR monitor. Participants were excluded

220 from the study on a case by case basis if they repeatedly missed exercise sessions and it was
221 deemed unrealistic to make up the exercise they had missed.

222 **Blood parameters**

223 Venous blood samples were collected into 10ml syringes and then transferred to EDTA-
224 containing Monovette tubes. These tubes contained a mixture of inhibitors (dipeptidyl
225 peptidase IV (DPP4) inhibitor (10µl/ml blood), aprotinin (50µl/ml blood) and pefabloc SC
226 (50µl/ml blood)) to prevent degradation of the peptides to be measured. Samples were drawn
227 at eight time points during the morning of the probe day at 0 min and after breakfast at +15
228 min, +30 min; +60 min; +90 min; +120 min; +180 min and +230 min for the measurement of
229 metabolic and appetite peptide levels. After collection, samples were centrifuged for 10
230 minutes at 4°C and 4000 rpm. Samples were immediately pipetted into Eppendorf tubes and
231 stored at -80°C awaiting analysis. Insulin, acylated ghrelin, peptide YY (PYY) and glucagon-
232 like peptide 1 (GLP-1) were analysed in this study. Total PYY was measured due to feasibility.
233 Because the overwhelming composition of circulating total PYY is known to be PYY3–36, the
234 present PYY (total) assay effectively measured PYY3–36. A previous study showed an
235 essentially perfect correlation between this PYY (total) assay and a PYY3–36 selective
236 radioimmunoassay. The relevant antibodies for PYY (total) used in the present study (originally
237 from Linco, St. Charles, Missouri), have been used by others to demonstrate the effects of
238 PYY3–36 (23). The inter- and intra- assay coefficients of variations were 6.35% and 6.2% for
239 insulin, 3.81% and 5.3% for leptin, 4.24% and 4.05% for GLP-1, 4.91% and 5.9% for PYY
240 (total) and 5.12% and 4.45% for acylated ghrelin, respectively.

241 Only a subset of participants completed the postprandial blood samples. Reasons for missing
242 peptide data included unsuccessful cannulation, and participants' unwillingness to take part in

243 this part of the study. All samples that were drawn, were analysed and have been included in
244 the manuscript.

245 **Statistical analysis**

246 Data are reported as mean \pm SD throughout, unless otherwise stated. Statistical analysis was
247 performed using IBM SPSS for Windows (Chicago, Illinois, Version 21) and significance was
248 set at $p < .05$. All variables were checked for outliers and normality was assessed using the
249 Shapiro-Wilk test. Changes in anthropometrics, body composition and RMR from baseline to
250 post-intervention were assessed using paired sample t-tests. To examine changes in EI, free-
251 living PA, SB, NEPA and activity EE in response to structured aerobic exercise, one-way
252 repeated measures ANCOVA were performed with baseline BMI entered as a covariate and
253 reported where significant. Change in subjective appetite sensations and appetite hormones
254 from baseline to post-intervention were assessed using two-way ANCOVA (Week*Time) with
255 effects of baseline BMI reported where significant. Where appropriate Greenhouse-Geisser
256 probability levels were used to adjust for sphericity. Post hoc comparisons using Bonferroni
257 adjustments were used if statistical significance was detected. Because of the large individual
258 variations in fasting levels of metabolic and appetite hormones, the change from baseline was
259 computed at each time point for each individual for all of the variables. Simple linear regression
260 was also performed to identify whether differences in exercise-induced EE or change in total
261 EI explained the variation in body composition change between participants. The last
262 observation carried forward (LOCF) method was used to account for missing data for the eight
263 participants who dropped out of the study. The analyses that were conducted on the completer
264 dataset were repeated on the LOCF dataset. Results were reported only when LOCF analyses
265 differed from completer analyses.

266 **RESULTS**

267 The prescribed total EE over the 12-week exercise intervention was 29,000 kcal for each
268 participant. The mean total measured exercise-induced EE was 28,792.3 kcal (SD = 872.96),
269 which was 99.3% of the prescribed EE.

270 **Change in body composition, anthropometrics and resting metabolism**

271 Paired sample t-tests revealed there was a significant reduction in BM [$t(23) = 2.18, p = .04$],
272 BMI [$t(23) = 2.25, p = .035$], WC [$t(23) = 4.60, p < .001$] and FM [$t(23) = 3.36, p = .003$] and
273 a significant increase in FFM [$t(23) = 3.35, p = .003$], see **Table 1**.

274 Assuming 1 kg of BM (70:30 fat/lean tissue) is equivalent to 7,700 kcal (24), the predicted
275 sample average weight loss resulting from the total exercise-induced energy deficit (28,792.29
276 kcal) was 3.74 kg. The observed weight loss was less than the predicted weight loss (22.19%
277 of predicted) indicating compensation for the exercise-induced energy deficit occurred. There
278 was no significant change in RMR from baseline to week 12 [$p = .304$], see **Table 1**.

279 ****Table 1 around here****

280 There was considerable variability in weight loss and body composition change between
281 participants. Seventeen participants lost weight, one participant remained the same and six
282 participants gained weight following the 12-week supervised aerobic exercise intervention.
283 Changes in BM ranged from -4.3 kg to +3.1 kg (see figure 1). Of the 24 participants, 20 reduced
284 their FM, one remained the same and three gained FM with changes ranging from -4.4 kg to
285 +4.9 kg. Two participants had unfavourable changes in both FM (increased) and FFM

286 (decreased). Total exercise-induced EE did not explain the variation in BM change [$F(1, 22) =$
287 $1.259, p = .274, R^2 = .054$], FM change [$F(1, 22) = 2.418, p = .134, R^2 = .099$] or FFM change
288 [$F(1, 22) = 1.475, p = .237, R^2 = .063$].

289 **Energy intake**

290 Paired sample t-tests revealed participants total EI during week 12 probe days was significantly
291 higher compared with total EI during baseline probe days [$t(23) = 2.35, p = 0.028$].
292 Furthermore, ad libitum EI (lunch, dinner and snack box EI combined) [$t(23) = 2.31, p = .03$]
293 and snack box EI [$t(23) = 2.09, p = .048$] were also higher at week 12. However, there was no
294 significant difference in lunch [$p = .998$] or dinner [$p = .194$] EI, see **Table 2**. When these
295 analyses were adjusted for baseline BMI (ANCOVA), there was no effect of BMI and no
296 interaction between BMI and the intervention.

297 ****Table 2 around here****

298 As with body composition change, there was considerable variability in total EI change from
299 baseline to week 12 between participants. Ten participants decreased their EI, whereas 14
300 participants increased their EI. Change in total EI ranged from -581.5 kcal/d to +763.9 kcal/d.
301 Change in total EI did not explain the variation in BM change [$F(1, 22) = 0.583, p = .453, R^2$
302 $= .026$], FM change [$F(1, 22) = 1.336, p = .260, R^2 = .057$] or FFM change [$F(1, 22) = 1.065, p$
303 $= .313, R^2 = .046$].

304 **Subjective appetite sensations**

305 There was no significant difference between baseline and week 12 fasting hunger ratings [$t(23)$
306 = 1.64, $p = .12$]. There was a main effect of week [$F(1, 23) = 7.82$, $p = .01$] with hunger being
307 higher (when measured over the whole day) at week 12 ($M = 25.58$ mm, $SD = 16.49$) compared
308 with baseline ($M = 21.68$ mm, $SD = 17.11$). Pairwise comparisons with Bonferroni adjustments
309 revealed VAS hunger ratings were significantly higher during the post-intervention probe days
310 compared with baseline immediately post-breakfast [$t(23) = 2.08$, $p = .049$], 15 min [$t(23) =$
311 2.65, $p = .014$], 30 min [$t(23) = 2.63$, $p = .015$], 90 min [$t(23) = 2.20$, $p = .038$], immediately
312 post-lunch [$t(23) = 2.33$, $p = .029$], immediately post-dinner [$t(23) = 2.63$, $p = .015$] and 600
313 min [$t(23) = 3.01$, $p = .006$]. There was also a main effect of time [$F(2.69, 61.95) = 66.99$, $p <$
314 $.001$] but no week*time interaction [$F(6.12, 140.70) = 0.73$, $p = .63$], see **Figure 2a**. Paired
315 sample t-tests revealed there was a significant increase in AUC for hunger [$t(23) = 2.61$, $p =$
316 $.016$] throughout the whole day from baseline to week 12.

317 There was no significant difference between baseline and week 12 fasting fullness ratings [$t(23)$
318 = 1.03, $p = .32$]. There was a main effect of week [$F(1, 23) = 5.55$, $p = .03$], with fullness being
319 lower (when measured over the whole day) at week 12 [$M = 56.12$ mm, $SD = 19.54$] compared
320 with baseline [$M = 60.06$ mm, $SD = 19.71$]. Pairwise comparisons with Bonferroni adjustments
321 revealed VAS fullness ratings were significantly lower during the week 12 probe days
322 compared with baseline at 30 min [$t(23) = 2.17$, $p = .040$], 180 min [$t(23) = 2.65$, $p = .014$],
323 immediately post-lunch [$t(23) = 2.78$, $p = .011$], immediately post-dinner [$t(23) = 2.49$, $p =$
324 $.021$] and at 600 min [$t(23) = 2.41$, $p = .024$]. There was also a main effect of time [$F(4.26,$
325 $97.99) = 75.28$, $p < .001$] but no week*time interaction [$F(7.54, 173.32) = 0.58$, $p = .78$], see
326 **Figure 2b**. Paired sample t-tests revealed there was a significant decrease in AUC for fullness
327 [$t(23) = 2.18$, $p = .04$] throughout the whole day from baseline to week 12. The results of these
328 analyses did not change when controlling for baseline BMI (ANCOVA).

329 **Change in free-living physical activity, sedentary behaviour and non-exercise physical**
330 **activity**

331 When the structured exercise sessions were included in the SWA data during the week 1 and
332 10 measurement period, the amount of time spent in MVPA was significantly different between
333 the four different time points [$F(3, 66) = 18.57, p < .001$]. Post hoc tests revealed MVPA was
334 significantly higher during the first and tenth week of the exercise intervention compared to
335 baseline and post-intervention [$p < .05$], see **Figure 3a**. Similarly, activity EE differed
336 significantly between the different time points [$F(3, 66) = 17.16, p < .001$]. Post hoc tests
337 revealed activity EE was also significantly higher during the first and tenth week of the exercise
338 intervention compared with baseline and post-intervention [$p < .05$], see **Figure 3a**.

339 A repeated measures ANCOVA revealed that there was a significant difference in mean
340 sedentary time between the different time points [$F(3, 66) = 3.32, p = .03$]. Post hoc tests
341 revealed that there was a significant increase in sedentary time between the first week of
342 exercise and the week following the completion of the exercise intervention [$p = .02$]. When
343 the repeated measures ANCOVA was conducted on the LOCF dataset [$F(3, 93) = 5.11, p =$
344 $.002$], there was a significant decrease in SB from baseline to week 1 [$p = .043$] and baseline
345 to week 10 [$p = .047$] of the exercise intervention. The increase in sedentary time between the
346 first week of exercise and the week following the completion of the exercise intervention
347 remained significant [$p = .02$]. There was no covariate effect of baseline BMI and no interaction
348 between BMI and the intervention.

349 Sleep, sedentary time, light PA and MVPA are collinear which means an increase in one
350 category of activity would lead to a decrease in at least one other. The sum of the change in
351 sleep, sedentary time and light PA (all categories excluding MVPA) between baseline and

352 week 1 and baseline and week 10 was calculated to identify whether the increase in structured
353 MVPA displaced these activities rather than displacing MVPA that participants already
354 performed as part of their daily routines. The sum of all the activity categories other than
355 MVPA between baseline and week 1 was -59.61 min/d (SD = 43.89) and between baseline and
356 week 10 was -41.19 min/d (SD = 51.70). Change in MVPA from baseline to week 1 was +50.20
357 min/d (SD = 37.96) and from baseline to week 10 was +42.63 min/d (SD = 49.87). Structured
358 MVPA appears to displace sleep, SB and light PA but not NEPA MVPA.

359 When the structured exercise was removed from the SWA data during week 1 and week 10 of
360 the exercise intervention there was no significant difference between baseline, week 1, week
361 10 and post-intervention NEPA MVPA [$F(3, 66) = 0.05, p = .99$] or NEPA activity EE [$F(3,$
362 $66) = 0.87, p = .46$], see **Figure 3b**. NEPA MVPA ranged from 85.8 min/d to 88.7 min/d and
363 NEPA activity EE ranged from 864.4 kcal/d to 760.1 kcal/d.

364 **Change in fasting and postprandial appetite-related peptide response**

365 There was a significant decrease in fasting insulin levels from baseline to post-intervention, as
366 shown in **Table 3**. There was no significant difference in fasting acylated ghrelin, PYY or GLP-
367 1 between baseline and post-intervention [$p > .05$].

368 ****Table 3 around here****

369 Postprandial profiles for insulin, acylated ghrelin, PYY, and GLP-1 at baseline and post-
370 intervention are displayed in **Figure 4**. There was a main effect of week for PYY [$F(1, 17) =$
371 $9.14, p = .008$] which was higher post-intervention (M = 51.19 ng/L, SD = 21.93) compared
372 with baseline (M = 35.96 ng/L, SD = 16.36). Post hoc tests using the Bonferroni correction

373 revealed that PYY was significantly higher during the post-intervention probe day at +30 min
374 [$p = .002$], +60 min [$p = .003$], and +90 min [$p = .041$]. There was a main effect of time [$F(2.01,$
375 $34.23) = 17.24, p < .001$] and a significant week*time interaction [$F(3.00, 51.06) = 3.17, p =$
376 $.032$].

377 There was no main effect of week for insulin [$F(1, 17) = 1.29, p = .272$], acylated ghrelin [$F(1,$
378 $16) = 0.21, p = .651$] or GLP-1 [$F(1, 17) = 0.23, p = .642$]. There was a significant main effect
379 of time for insulin [$F(1.31, 22.24) = 67.35, p < .001$], acylated ghrelin [$F(1.98, 31.65) = 64.34,$
380 $p < .001$] and GLP-1 [$F(2.01, 34.19) = 34.50, p < .001$], however there was no week*time
381 interaction for insulin [$F(2.81, 47.68) = 0.96, p = .417$], acylated ghrelin [$F(3.23, 51.72) = 1.16,$
382 $p = .335$] or GLP-1 [$F(2.80, 47.67) = 1.36, p = .268$].

383 **DISCUSSION**

384 The 12-week exercise intervention resulted in a significant reduction in BM and FM, refuting
385 claims from some academics that exercise/PA does not promote weight loss (25). However,
386 weight loss was less than predicted and there was considerable variability in weight change
387 between individuals ranging from -4.3 kg to +3.1 kg. Less than predicted weight loss and large
388 individual variability in weight change have previously been reported in response to increased
389 exercise (1, 7). Total exercise-induced EE throughout the intervention (99.3% of prescribed on
390 average) did not contribute to the variability in weight change, thus ruling out the possibility
391 that the variability was due to adherence to the exercise intervention.

392 It has been suggested that exercise-induced EE will be compensated for through increased EI
393 or decreased NEPA to offset the negative energy balance, rendering exercise futile for weight
394 loss (26, 27). The exercise-induced energy deficit in the current study was not fully

395 compensated for as participants did in fact lose weight on average. However, partial
396 compensation was evident as participants lost less weight than predicted when calculated based
397 on the exercise-induced energy deficit. When calculated the increase in EI between baseline
398 and post-intervention probe days was repeated every day for 12-weeks the accumulated
399 increase in EI would be approximately 15,000 kcal. This is approximately half of the EE due
400 to exercise; thereby effectively reducing the exercise potency by 50%. It is also worth noting
401 that the static Wishnofsky predictive equation (24) for estimating weight loss is simplistic and
402 does not account for adaptations in other components of energy balance as a result of an energy
403 deficit (for example, increased EI, physiological reductions in RMR, an increase in FFM or a
404 decrease in NEPA) and could lead to overestimation of predicted weight loss (28).
405 Furthermore, the 1 kg of BM is equivalent to 7700 kcal rule (1 kg of BM consists of 70% fat
406 and 30% FFM) is based on short-term low-calorie diets and is not directly applicable to the
407 change in body composition induced by exercise. Indeed, in the current study, and others (29),
408 there was in fact a significant increase in FFM.

409 It was hypothesised that EI would increase post-intervention in response to increased exercise
410 as has previously been demonstrated (7, 10). Indeed, there was a significant increase in total,
411 ad libitum and snack box EI at week 12. While some studies show no change in EI, these are
412 often unsupervised and rely on self-report measures of EI (30). When calculated as a
413 proportion of the energy expended per exercise session, the increase in EI represented
414 compensation of 36%, which is similar to the 30% compensation observed by Whybrow et al.
415 (10). The participants in the Whybrow study were lean men and women and would be
416 expected to compensate for a negative energy balance more readily as they have less of a
417 'buffer' (FM) than overweight or obese individuals. That could explain why the degree of
418 compensation is similar in both studies despite the present study being considerably longer.

419 Participants had more FM in the current study and therefore compensation may not occur as
420 quickly as would be expected in lean individuals. It has previously been noted that BM
421 regulation is asymmetrical; a positive energy balance (and weight gain) is well tolerated
422 whereas a negative energy balance (and weight loss) is strongly defended against (31). This
423 study, together with previous research (32), provides further support for the asymmetry of
424 BM regulation evidenced by the compensatory increase in EI to defend against weight loss in
425 response to a prolonged period of increased exercise-induced EE. A strength of this study is
426 the objective measurement of 24 hour EI, however, it is acknowledged that using episodic
427 test meal intake to infer changes in habitual intake has limitations (33). Rather, probe day
428 measures of EI can be viewed as assays for eating behaviour and give an indication of
429 compensatory appetite responses to perturbations in energy balance that are free from
430 external influences (34). Similar test meals and probe day procedures to those reported in the
431 current study have previously been shown to detect exercise-induced compensation in eating
432 behaviour (7).

433 The increase in EI was accompanied by an increase in hunger throughout the day (mainly
434 during the morning) and decreased fullness reflected in AUC for hunger and fullness. The
435 results of the current study are similar to those observed in ‘non-responders’ in the study by
436 King et al. (6) with respect to change in BM (-0.9 kg), FM (-1.2 kg), EI (+164 kcal) and AUC
437 for hunger and fullness. A possible explanation is that the majority of the participants in the
438 current study are ‘non-responders’; they do not achieve the predicted change in body
439 composition calculated from their exercise-induced EE. When the current sample are
440 categorised as ‘responders’ and ‘non-responders’ using the method described by King et al.
441 (6), two thirds are classified as ‘non-responders’. Participants in the current study had a lower
442 BMI at the start of the study (27.94 kg/m² vs. 31.80 kg/m²) which could explain why their

443 weight loss response was less pronounced than that observed in a previous study (6).
444 Furthermore, the study by King et al. (6) included men and men have been shown to exhibit a
445 greater weight loss in response to exercise than women (35, 36). However, this is not a
446 universal finding (37). The current findings in women should not be assumed to generalise to
447 men and further research is required to verify this.

448 Greater compensation in NEPA, rather than changes in EI, have previously been reported in
449 response to increased exercise (38). In the current study, SWA data was initially analysed
450 with structured exercise included in the data collected during week 1 and 10 of the exercise
451 intervention. When MVPA and activity EE were compared across the four time points
452 (baseline, week 1, week 10 and post-intervention) participants spent significantly more time
453 in MVPA and had significantly higher activity EE during week 1 and week 10 compared with
454 baseline and post-intervention. Total compensation in NEPA would be apparent if, for
455 example, MVPA and activity EE did not increase during the exercise intervention. MVPA
456 and activity EE returned to baseline values when PA was measured post-intervention. This
457 demonstrates that participants did not maintain their increased PA levels once the
458 intervention ended. Post-interventions PA levels similar to baseline have previously been
459 highlighted (39-42).

460 There was no evidence for a compensatory increase in SB. In fact, SB was lower in the weeks
461 during the exercise intervention, but only the difference between week 1 of the exercise
462 intervention and post-intervention reached statistical significance. This suggests that the
463 structured exercise displaced some sedentary time. This is in contrast with previous research
464 that suggests that interventions need to specifically target reductions in SB to change
465 sedentary time (12). Indeed, the magnitude of the reduction in SB may have been greater with
466 a specific component of the intervention to target reduced SB in the current study. Further

467 examination of activity monitor data suggests structured exercise also displaces some sleep
468 time and light PA, but the difference in sleep and light PA at the different time points
469 throughout the intervention were not significant. The sum of the difference in sleep, SB and
470 light PA between baseline and week 1 and baseline and week 10 was greater than the change
471 in MVPA (in the opposite direction) at the same time points. Furthermore, when the
472 prescribed exercise was removed from SWA data during week 1 and 10, the remaining
473 NEPA MVPA was remarkably similar to baseline and post-intervention values (<3 minutes
474 difference between all four time points) and there was no significant difference in NEPA
475 activity EE across the four time points. Taken together, these findings suggest that increasing
476 MVPA through a structure exercise intervention displaces time spent sleeping, sedentary and
477 in light PA but not NEPA MVPA. This is in agreement with previous studies (40, 42) and a
478 recent systematic review that concluded no statistically or clinically significant mean change
479 in NEPA occurs during exercise training (11).

480 Appetite-related peptides were measured in this study in order to determine if any exercise-
481 induced changes could be related to adjustments in fasting or postprandial gastrointestinal
482 signalling. However, the peptides did not account for changes in subjective appetite
483 sensations or in EI. PYY was higher on average during post-intervention probe days,
484 however this was not coupled with a decrease in hunger or an increase in fullness as might be
485 expected. In fact, there was a significant increase in hunger and decrease in fullness post-
486 intervention. There was no change in postprandial profiles for insulin, acylated ghrelin or
487 GLP-1 in the present study. Acute studies suggest an exercise intensity of at least 65% $\dot{V}O_2$ is
488 required to induce changes in appetite related peptides (43, 44). However, the present
489 findings are not comparable due to the assessment of longer-term exercise training. There
490 was a significant decrease in fasting insulin from baseline to post-intervention. As insulin

491 levels are proportional to FM it is likely the reduction in insulin was driven by the reduction
492 in FM following the exercise intervention. However, some studies have demonstrated
493 improved insulin sensitivity following exercise interventions independent of weight loss/body
494 composition changes whilst others have demonstrated improvements only occur with weight
495 loss (45). The relative importance of exercise and weight loss remains unclear and it is
496 possible both contributed to the reduction in fasting insulin levels in the present study. These
497 findings, while novel in this context, suggest that the changes in appetite are more likely due
498 to changes in body composition rather than changes in appetite peptides, as has previously
499 been proposed (46). It is possible that a greater change in body composition would be
500 required to see concomitant changes in appetite peptides.

501

502 The quasi-experimental design used in the present study allows certain inferences to be made
503 from the presence or lack of changes in compensatory EI and EE behaviours before and after
504 medium-term exercise training. However due to the single non-randomised sample it is not
505 possible to rule out that the effects reported here would not have been seen after 12 weeks of
506 rest (with the two conditions randomised). Future confirmation of these findings using a
507 randomised controlled trial design would be valuable.

508 On average there was a significant increase in EI from baseline to post-intervention providing
509 a plausible explanation for the less than predicted weight loss. However, change in total EI
510 did not explain the variation in BM change. Laboratory measures of EI do not reflect the
511 turbulence of the free-living environment in which eating behaviour is more haphazard and
512 cannot be captured. Indeed, it is possible that the measure of EI obtained from the probe days
513 may not reflect participants eating habits in the free-living environment.

514 It must also be acknowledged that participants' menstrual cycle was not recorded and
515 therefore could not be included as a covariate in analyses. Since there does not seem to be

516 any discernible differences between sexes in the appetite and eating behaviour response to
517 acute and longer-term exercise interventions (37, 47), the authors think it is unlikely that the
518 menstrual cycle had a major impact on the study outcomes.

519 Finally, it is worth emphasising that exercise alone is clearly not the most effective way to
520 lose weight, particularly when compared to standard behavioural interventions in which
521 participants may lose 5-10% of weight. The present study demonstrates that exercise can
522 produce modest fat loss without additional dietary assistance. However, the compensatory
523 increase in energy intake observed suggests that an additional dietary intervention would
524 support an even greater weight (fat) loss.

525 **CONCLUSIONS**

526 Overweight women took part in an exercise intervention which comprised five mandatory
527 sessions of aerobic exercise per week for 12-weeks. No constraint was placed on other free-
528 living behaviour (activity or eating) during the 12-weeks. Therefore, participants were able to
529 demonstrate compensation for the energy expended in exercise by an adjustment of their food
530 intake or the amount of SB or free-living PA. At the end of 12-weeks there was a significant
531 decrease in FM and an increase in FFM indicating that the exercise regime had been effective
532 and had generated a significant impact on body composition. However, there was considerable
533 individual variability and the changes in body composition were smaller than could have been
534 expected on the basis of the total energy expended through exercise (actual weight loss was
535 22.19% of predicted). Compensation for the exercise induced EE was detected in a significant
536 increase in EI but no increase in SB or decrease in free-living PA. In fact, the exercise actually
537 displaced SB. The effect of exercise on FM could be amplified by the addition of a dietary
538 strategy designed to prevent a compensatory increase in EI.

539 Despite finding a short-term increase in EI during laboratory probe days, the magnitude of this
540 effect was not sufficient to fully explain the difference between predicted and observed weight
541 loss. While food intake in the laboratory setting provides a plausible objective marker of
542 changes in free-living intake, it may not reflect absolute levels of energy consumed during the
543 intervention. Therefore it is not possible to decisively conclude from the present findings that
544 compensation for the exercise was due to EI alone. Future studies using other comprehensive
545 measures of EI and EE are needed to corroborate the present results. Moreover, future studies
546 should investigate how weight status (lean, overweight, obese), the amount of exercise applied
547 (volume, intensity) and the periodicity of exercise (frequent small bouts or fewer large bouts)
548 effect the relationship between exercise and behavioural consequences. Considering an effect
549 on EI, it is known that this end point is influenced by body composition (FM and FFM). These
550 variables are also influenced by exercise, therefore any effect of exercise may be mediated
551 indirectly via changes in body composition or directly through some mechanism involved in
552 cellular metabolism.

553 **LIST OF ABBREVIATIONS**

554 ANCOVA, analysis of covariance; BM, body mass; BMI, body mass index; EE, energy
555 expenditure; EI, energy intake; FFM, fat-free mass; FM, fat mass; HR, heart rate; LOCF, last
556 observation carried forward; MVPA, moderate-to-vigorous physical activity; NEPA, non-
557 exercise physical activity; PA, physical activity; RMR, resting metabolic rate; SB, sedentary
558 behaviour; SD, standard deviation; SWA, SenseWear Armband mini; VAS, visual analogue
559 scale; WC, waist circumference.

560 **DECLARATIONS**

561 **Ethics approval and consent to participate**

562 All participants provided written informed consent before taking part in the study. The study
563 procedures and all study materials were reviewed and approved by the National Research
564 Ethics Service Committee Yorkshire & the Humber (ref: 09/H1307/7).

565 **Consent for publication**

566 Not applicable

567 **Availability of data and material**

568 The datasets used and/or analysed during the current study are available from the corresponding
569 author on reasonable request.

570 **Competing interests**

571 The authors declare that they have no competing interests.

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575 **Authors' contributions**

576 AM, MD, CG, GF and JB designed research; AM, MD and CG conducted research; AM
577 analysed data; AM, CG, GF and JB discussed data analysis and interpretation of the data; AM
578 and JB wrote manuscript. All authors approved the final manuscript.

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581

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- 724

725 **FIGURES, TABLES AND ADDITIONAL FILES**

726 Figure 1. Individual variability in BM change between participants.

727 Figure 2. VAS (a) hunger and (b) fullness ratings during baseline (BL) and post-intervention
728 (PI) probe days (error bars are standard error). * = $p < .05$, indicates significant difference
729 between baseline and post-intervention.

730 Figure 3. Time spent in MVPA and activity EE before (baseline; BL), during the 12-week
731 exercise intervention (week 1 and 10) and after the exercise intervention (post-intervention; PI)
732 measured using the SWA with structured exercise included (a) and removed (b) from the data
733 (n=23), ** = $p < .01$, *** = $p < .001$.

734 Figure 4. Postprandial profiles for insulin (a), acylated ghrelin (b), PYY (c), and GLP-1 (d) at
735 baseline (BL) and post-intervention (PI; n=18), * = $p < .05$, ** = $p < .01$.