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

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

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ABSTRACT

Background

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

Methods

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

Results

Following the 12-week exercise intervention BM [p=.04], BMI [p=.035], WC [p<.001] and fat mass [p=.003] were significantly reduced, and fat-free mass (FFM) significantly increased [p=.003]. Total [p=.028], *ad libitum* [p=.03] and snack box EI [p=.048] were significantly increased and this was accompanied by an increase in hunger [p=.01] and a decrease in fullness [p=.03] before meals. The peptides did not explain changes in appetite [p>.05]. There was no

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

Conclusions

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

Trial registration

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

KEY WORDS

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

BACKGROUND

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

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

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

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

METHODS

Participants

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

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

Design

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

Measures days

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

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

Probe days

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

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

Participants returned to the laboratory four hours later for the *ad libitum* dinner of tomato and herb risotto, garlic bread, salad items, chocolate brownies and water. An *ad libitum* snack box containing an apple, two mandarins, roast ham, cheese, bread, margarine, crisps, chocolate buttons and a vanilla yoghurt was given to participants to take home in the evening. Participants could eat any food items from the snack box but were instructed not to share the

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

174 During probe days visual analogue scales (VAS) were completed immediately before and
175 after meals and periodically between meals to assess subjective appetite sensations using a
176 validated electronic appetite rating system (15). Area under the curve (AUC) was calculated
177 using the trapezoid method for subjective feelings of hunger, fullness, desire to eat and
178 prospective foods consumption throughout the whole day (post-breakfast (0 min), +15 min,
179 +30 min, +60 min, +90 min, +120 min, +180 min, +230 min, pre-lunch (+235 min), post-
180 lunch (+260 min), +300 min, +360 min, +420 min, pre-dinner (+480 min), post-dinner (+500
181 min), +540 min, +600 min).

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

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

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

Non-exercise physical activity

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

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

Exercise intervention

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

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

Blood parameters

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

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

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

Statistical analysis

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

RESULTS

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

Change in body composition, anthropometrics and resting metabolism

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

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

Table 1 around here

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

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

Energy intake

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

Table 2 around here

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

Subjective appetite sensations

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

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

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

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

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

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

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

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

Change in fasting and postprandial appetite-related peptide response

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

Table 3 around here

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

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

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

DISCUSSION

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

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

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

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

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

451 The increase in EI was accompanied by an increase in hunger throughout the day (mainly
452 during the morning) and decreased fullness reflected in AUC for hunger and fullness. The
453 results of the current study are similar to those observed in 'non-responders' in the study by
454 King et al. (6) with respect to change in BM (-0.9 kg), FM (-1.2 kg), EI (+164 kcal) and AUC
455 for hunger and fullness. A possible explanation is that the majority of the participants in the
456 current study are 'non-responders'; they do not achieve the predicted change in body
457 composition calculated from their exercise-induced EE. When the current sample are
458 categorised as 'responders' and 'non-responders' using the method described by King et al.
459 (6), two thirds are classified as 'non-responders'. Participants in the current study had a lower
460 BMI at the start of the study (27.94 kg/m^2 vs. 31.80 kg/m^2) which could explain why their

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

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

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

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

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

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

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

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

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

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

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

CONCLUSIONS

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

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

LIST OF ABBREVIATIONS

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

DECLARATIONS

Ethics approval and consent to participate

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

Consent for publication

Not applicable

Availability of data and material

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

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

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

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742

743 **FIGURES, TABLES AND ADDITIONAL FILES**

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

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

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

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