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# Behavioural compensatory adjustments to exercise training in overweight women

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**Abstract**

**Purpose:** To examine extent to which changes in non-exercise physical activity contribute to individual differences in body fat loss induced by exercise programs.

**Methods:** Thirty four overweight/obese sedentary women (age: 31.7 ± 8.1 years, BMI: 29.3 ± 4.3 kg m$^{-2}$) exercised for 8 weeks. Body composition, total energy expenditure (TEE), exercise EE (ExEE), activity EE (AEE) calculated as energy expenditure of all active activities minus ExEE, sedentary EE (SEDEE), sleeping EE (SEE), and energy intake were determined before and during the last week of the exercise intervention.

**Results:** Over the 8-week exercise program net ExEE was 30.2 ± 12.6 MJ and based on this, body fat loss was predicted to be 0.8 ± 0.2 kg. For the group as a whole, change in body fat (-0.0 ± 0.2 kg) was not significant but individual body fat changes ranged from -3.2 kg to +2.6 kg. Eleven participants achieved equal or more than the predicted body fat loss and were classified as 'Responders' and 23 subjects achieved less than the predicted fat loss and were classified as 'Non-responders'. In the group as a whole, daily TEE was increased by 0.62 ± 0.30 MJ (p<0.05) and the change tended to be different between groups (Responders, +1.44 ± 0.49 MJ; Non-responders, +0.29 ± 0.36 MJ, p=0.08). Changes in daily AEE of Responders and Non-responders differed significantly between groups (Responders, +0.79 ± 0.50 MJ; Non-responders, -0.62 ± 0.39 MJ, p<0.05). There were no differences between Responders and Non-responders for changes in SEDEE and SEE or energy intake.

**Conclusion:** Overweight and obese women who during exercise intervention achieve lower than predicted fat loss are compensating by being less active outside exercise sessions.

**Keywords:** exercise, energy balance, overweight women

**Introduction**

***Paragraph Number 1*** Increasing total energy expenditure (TEE) by increasing physical activity is an important component of many lifestyle interventions aimed at reducing obesity and its complications. Such increases in TEE should aid body fat and body weight loss, provided all other variables affecting energy balance are kept constant. However, exercise-induced perturbations to energy balance may initiate behavioural compensatory adjustments and either alter food intake (24,27,33,34,36) or cause a reduction in normal daily activities (11,16,21,36). This compensation for the exercise-induced energy deficit may explain why exercise alone often does not result in successful weight loss in obese and overweight individuals (12).

***Paragraph Number 2*** Despite the commonly reported and accepted notion that the effectiveness of exercise in inducing body fat loss is low, an accumulating body of evidence suggests that the inter-individual variability in body weight and fat changes in response to an exercise intervention is large, and that participants of exercise intervention studies can broadly be separated into "Responders" – i.e. those who achieve a body fat loss in response to exercise, and "Non-responders" – i.e. those who fail to achieve a body fat reduction in response to exercise (3,22,23). This suggests that studies investigating exercise-induced compensatory mechanisms should focus on individual variability rather than consider body fat or body weight changes in the group as a whole. In addition, such studies should ensure adherence to prescription of exercise since variability in the effectiveness of exercise in relation to body fat loss could be accounted for difference in compliance (8,10).

***Paragraph Number 3*** Data evaluating individual responsiveness to exercise induced fat loss are very limited. The recent study of King and colleagues (23) investigated compensatory responses to a supervised and well-controlled exercise program in overweight men and women in relation to individual variability. The authors reported that participants who experienced a lower than predicted weight loss demonstrated a compensatory increase in

their energy intake over the course of the intervention and that those who lost more weight than predicted decreased energy intake, although there was no overall difference before and after the intervention for the group as a whole. Although compensating for exercise-induced energy disturbance could also include alterations in physical activity in non-exercise time (11,16,21,34), this was not investigated in the aforementioned study.

**Paragraph Number 4** The aim of the present study was therefore to examine the extent to which changes in physical activity outside of the exercise intervention and energy intake contribute to individual differences in body fat loss induced by exercise training programs. The volume of exercise used was based on current exercise recommendations (18) and compliance to the prescribed exercise was ensured by supervision of all exercise sessions.

**Methods**

*Subjects*

**Paragraph Number 5** Thirty-four overweight or obese women were recruited for this study (Table 1). All participants were non-smokers, apparently healthy and were not taking any form of medication. Participants were required to be weight stable for at least two months prior to testing, and sedentary (less than one hour of planned physical activity per week and sedentary job). All participants gave written consent prior to inclusion to the study, which was approved by the Ethics Committee of the Faculty of Biomedical and Life Sciences, at the University of Glasgow.

*Study Design*

**Paragraph Number 6** Participants performed an 8-week supervised exercise program, undertaking 150 minutes of exercise per week at heart rates (HR) ranging from 135 to 145 beats min$^{-1}$, which corresponded to 72 to 77 % of their age predicted maximum HR. An 8-week intervention period was chosen to ensure that the trial was long enough to induce measurable changes in body composition, but short enough to ensure high exercise compliance. During the week preceding the exercise program (baseline) and during the last

week of the exercise program (week 8), participants were required to wear HR monitors during all waking hours, record activities and dietary intake in physical activity and food intake diaries. On the day of the first and the last exercise session, measurements of body composition, resting metabolic rate (RMR), and lactate threshold (LT) were obtained, and the individual relationship of oxygen uptake ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) to HR during different states was determined.

*Determination of LT and prediction of $\dot{V}O_2max$*

**Paragraph Number 7** Following a familiarisation session, participants undertook an incremental cycle ergometer test to determine LT and predict $\dot{V}O_2max$. Participants were asked to exercise on a cycle ergometer (Ergomedic 873, Monark, Sweden) at gradually increasing intensities starting at 50W with a graded increase of 15W every 5 minutes. Heart rate was continuously recorded by short-range telemetry (Polar S610i, Polar Electro Oy, Kempele, Finland). At the end of each 5-minute stage, rate of perceived exertion was indicated by the subject on the Borg scale (4) and a finger prick capillary blood sample was taken, which was used for lactate measurement according to protocol of Maughan (26). During the final minute of each stage an expired air sample was collected and analysed through a gas analyser (1440 Gas Analyser, Servomex, UK). The test was terminated once the participant reached 85% of their age-predicted maximum HR. Lactate threshold (32) was determined and $\dot{V}O_2$ max was predicted by extrapolation of the HR against $\dot{V}O_2$ plot to age-predicted maximum HR.

*Determination of the relationship of $\dot{V}O_2$ and $\dot{V}CO_2$ to HR during active and inactive conditions*

**Paragraph Number 8** The approach described by Moon and Butte was used to establish the relationship between $\dot{V}O_2$ and $\dot{V}CO_2$ to HR during active and inactive conditions. This method combines HR and physical activity measures with non-linear and discontinuous models to calculate energy expenditure and shows good agreement with energy expenditure measurements made using room calorimetry (30). Participants were asked to avoid strenuous

activities on the day of testing and the day prior to testing. The individual relationships between HR and $\dot{V}O_2$, and HR and $\dot{V}CO_2$ were determined by analysis of expired air samples collected while participants performed activities categorised as "inactive" or "active" (30). The inactive stage involved subjects sitting still for 30 minutes, while active stages included activities representative of physical activity in habitual daily life such as standing still, standing whilst swaying arms, slow and faster walking on a treadmill at self-selected speeds, and finally cycling at gradually increasing intensities beginning at 50W and reaching 105-120W depending on individual fitness level. Individual regression analyses on the relationships obtained for HR vs $\dot{V}O_2$ and HR vs $\dot{V}CO_2$ were performed to obtain coefficients ($a_1$, $a_2$, $a_3$, $a_4$, $b_1$, $b_2$, $b_3$, $b_4$) specific to each subject: $\dot{V}O_2 = a_1 + b_1$ x $HR^3$, and $\dot{V}CO_2 = a_2 + b_2$ x $HR^3$ for inactive activities; and $\dot{V}O_2 = a_3 + b_3$ x HR, and $\dot{V}CO_2 = a_4 + b_4$ x HR for active activities [30]. The coefficients generated from this procedure were used for energy expenditure (EE) calculations, described in the section below.

*Recording of Physical Activity*

***Paragraph Number 9*** All activities carried out during the week leading to the exercise program and during week 8 were written down by participants in a 24-hour physical activity diary with 5-minute accuracy for seven consecutive days (31). This diary approach has been shown to have high reproducibility for the assessment of energy expenditure (intraclass correlation coefficient = 0.96) (5) and is often used as used as the criterion measure in the validation of physical activity questionnaires (31), although in the present study it was only used for the classification of activity type. Activities were defined as: sleeping; sitting; standing; walking; self-care; driving; and exercise, in the diaries and were divided by researcher into three classifications: "sleeping", "inactive" (sitting including driving) and "active" (all activities excluding sitting, driving and sleeping). Volunteers were also asked to record any miscellaneous activities that did not belong to the specific categories described above. Miscellaneous activities were again classified according to the level of activity (e.g. watching TV would be classified as an inactive activity, while washing the dishes would be classified as an active activity). Participants completed 88% of the available time in their

activity diaries. For missing data, an inactive or active activity classification was assigned using the researchers' judgment, according to the nature of activities undertaken immediately prior to and following the period of missing data, and from heart rate over the missing data period.

*Exercise intervention*

**Paragraph Number 10** All participants undertook 150 minutes of supervised exercise per week. To enable us to address a secondary question concerning whether frequency of exercise influenced the extent of fat loss in response to exercise training, participants were randomly assigned to one of two patterns of exercise: exercising twice per week for the duration of 75 minutes (Pattern A, n=18) or exercising five times per week for the duration of 30 minutes (Pattern B, n=16). The participants who exercised for the duration of 75 minutes were allowed one break for the duration of 5 minutes. Exercise sessions were all performed under laboratory conditions on friction braked cycle ergometers (Ergomedic 873, Monark, Sweden). The intensity of the exercise was individually set at 90% of the LT for the first two weeks followed by 95% of the LT for the next two weeks. Lactate threshold was reassessed after 4 weeks of the intervention; exercise intensity was 90% of the new LT for the next 2 weeks and 95% of this value for the final 2 weeks of the programme. Heart rate was continuously recorded through short-range telemetry HR monitors (Polar S610i, Polar Electro Oy, Kempele, Finland). Exercise sessions were performed at a time convenient for participants and were supervised by a researcher.

*Anthropometry*

**Paragraph Number 11** Measurements of body mass body fat and fat free mass were taken using leg-to-leg bioelectrical impedance scales (TBF-300, TANITA, Cranlea, UK), a technique which has been reported to similarly detect changes in body composition in response to diet and/or exercise interventions when compared to gold standard reference methods (29,35). To determine the test-retest reliability of our body composition measurements, we measured body composition in 30 women on two occasions, at an interval

of 2 days, using the bioelectrical impedance scales. The mean ± SD difference in fat mass between measurements was 0.05 ± 0.68 kg (0.16 ± 2.12%) and the mean ± SD difference in fat free mass was 0.07 ± 0.77 kg (0.14 ± 1.61%). Height and waist circumference were determined using standard protocols (25).

*Resting Metabolic Rate (RMR)*

**Paragraph Number 12** Following a familiarisation session, RMR was measured in the morning after a 12-hour fast and 24-hour abstention from exercise using a ventilated hood system (Deltatrac, Datex Instrumentation Corporation, Helsinki, Finland). Participants were asked to drive to the laboratory and where possible to minimize movement. On arrival at the laboratory volunteers were escorted to a quiet, semi-darkened room where they lie quietly for 10 minutes before measurement began with their arms at their sides and their legs straight and uncrossed, on the examination bed. A ventilated hood was then placed over the participant's head to allow analysis of expired gas. The person was monitored throughout to ensure that sleeping, talking and excess movement did not occur. After 10 minutes supine rest, measurements of $\dot{V}O_2$ and $\dot{V}CO_2$ were made every 60 seconds for 30 minutes and RMR was calculated using indirect calorimetry equations described by Frayn and Macdonald (13). The first 10 minutes of data collected were excluded from RMR calculations to ensure steady state values were used.

*Calculation of Energy Expenditure (EE)*

**Paragraph Number 13** Activities recorded in the 7-day physical activity diaries were categorised by researchers into sleeping, inactive and active activities and then inactive and active activities were time-matched with the HR monitoring data collected during waking hours. The mean value of HR for inactive and active categories was calculated and used to determine corresponding $\dot{V}O_2$ and $\dot{V}CO_2$ using the coefficients produced from the relationship between HR-$\dot{V}O_2$ and HR-$\dot{V}CO_2$ relevant to inactive and active activities (30). The rate of EE of inactive and active activities was then calculated by indirect calorimetry (13). Total energy expenditure (TEE) was calculated as the sum of active EE (AEE) which

included EE of all active activities except EE of exercise sessions; sedentary EE (SEDEE); and sleeping EE (SEE), which was defined as 95% of RMR (15). Gross exercise energy expenditure (ExEE) was calculated using coefficients produced from the relationship between HR-$\dot{V}O_2$ and HR-$\dot{V}CO_2$ obtained during the submaximal test. Net ExEE was calculated by subtracting RMR equivalent for the exercise time from gross ExEE.

*Measurement of Energy Intake*

**Paragraph Number 14** During the week leading to the exercise program and during week 8 participants were instructed to keep a food diary for seven consecutive days which involved weighing all food and drink consumed on electronic scales and recording the weight and time of consumption in the diary (1). Instructions were provided in addition to a visual demonstration by the researcher to show how to use the scales and the diary. The participants were advised to maintain their normal dietary intake. The Diet 5 computer software package (Diet 5, Robert Gordon University, Aberdeen, UK) was then used to analyse the food diaries and determine macronutrient and micronutrient intake for each participant.

*Classification of subjects as Responders and Non-responders*

**Paragraph Number 15** Change body mass in response to the exercise training intervention is the sum of change in lean mass and change in fat mass. As the energy density of fat is 39.4 MJ/kg and the energy density of lean tissue is 3.7 MJ/kg (8), the energy imbalance associated with change in body mass (assuming no change in bone mass) is given by:

$$\Delta\text{Energy balance (MJ)} = \Delta\text{fat mass (kg)} \times 39.4 + \Delta\text{fat free mass (kg)} \times 3.7$$

Thus;

$$\Delta\text{fat mass (kg)} = (\Delta\text{Energy balance (MJ)} - \Delta\text{fat free mass (kg)} \times 3.7)/39.4$$

Thus, the expected change in fat mass in response to the exercise training program can be calculated from the total net ExEE ($\Delta$Energy balance) and the change in fat-free mass. A comparison of predicted fat loss with actual fat loss was used to determine the extent to which compensation had occurred. Participants achieving less than predicted fat loss were classified

as Non-responders, and those achieving more than or equal to their predicted fat loss were classified as Responders.

*Statistical Analysis and Power Calculations*

***Paragraph Number 16*** Statistical analysis was performed using Statistica (version 6.0, StaSoft Inc., Oklahoma). Data were tested for normality using the Anderson-Darling test before statistical analysis and those with distribution significantly different from normal were logarithmically transformed. Data are presented as mean ± SEM, unless otherwise stated. Differences between the two groups at baseline were compared using unpaired t-tests. Changes in all variables from baseline to post-intervention assessment were compared by two-way ANOVA (group x time) with repeated measures on the 'time' factor. The group x time interaction term was used to determine whether subjects from the group classified as Responders and the group classified as Non-responders responded differently to the intervention and *post hoc* Tukey test was used to identify changes within groups. Uni and multi-variate regression analysis was performed to determine whether behavioural compensatory factors (i.e. TEE, AEE, SEDEE, SEE and energy intake) were significant predictors of the extent of change in fat mass over the entire group of responders and non-responders combined. Chi-Square analysis was performed to determine whether exercise pattern influenced the distribution of responders and non-responders. The activity EE was 4.6 ± 1.7 MJ/week at base line (week 0) and 4.4 ± 1.3 MJ/week at the end of exercise program (week 8) and the SD for the difference in AEE between week 8 and week 0 was 12.6 MJ/week. Based on these data, the present study with 11 Responders and 23 Non-responders, had sufficient statistical power to detect a difference of 9.3 MJ/week in AEE change with 85 % power.

**Results**

***Paragraph Number 17*** Compliance with the exercise intervention was 100%, with all participants completing 1200 minutes of supervised exercise over the 8-week exercise

intervention. Participants expended 30.2 ± 12.6 MJ and thus were predicted to achieve a body fat loss of 0.8 ± 0.2 kg. However, when the group was considered as a whole, there was no significant change in body fat (-0.0 ± 0.2 kg) over the course of the intervention (Table 2). Further examination of the data revealed large individual variability in body fat changes ranging from -3.2 kg to +2.6 kg (Figure 1). Eleven of the participants lost more than or equal to their predicted fat loss and were classified as Responders, while 23 of them lost less than their predicted fat loss and were classified as Non-responders (Figure 1). There were no differences in body fat loss between participants assigned to exercise Pattern A and Pattern B (Pattern A, -0.25 ± 0.40 kg; Pattern B, 0.14 ± 0.33 kg; ANOVA: degrees of freedom = 1, F-ratio = 0.573, $p = 0.45$ for interaction) and the pattern of exercise did not significantly influence the distribution of Responders and Non-responders (Pattern A: 11 Non-responders, 7 Responders; Pattern B: 12 Non-responders, 4 Responders, Chi Square $p = 0.39$).

***Paragraph Number 18*** There were no significant differences in any of the measured baseline variables between Responders and Non-responders (Table 1). By definition, body fat responses to exercise program differed significantly between Responders and Non-responders with Responders reducing (by 5.6%) and Non-responders increasing (by 1.9%) in body fat (degrees of freedom = 1, F-ratio = 45.03, $p < 0.0005$ for interaction) (Table 2). Waist circumference decreased by 4.0% for the group as a whole ($p < 0.01$), with no difference between Responders and Non-responders. Maximal oxygen uptake increased by 35% (degrees of freedom = 1, F-ratio = 70.61, $p < 0.0005$ for main effect) and $VO_2$ at lactate threshold increased by 12% (degrees of freedom = 1, F-ratio = 5.376, $p = 0.028$ for main effect) in the group as a whole with no difference between Responders and Non-responders. Exercise training had no effect on RMR (Table 2).

***Paragraph Number 19*** Over the 8 weeks of the exercise program Responders and Non-Responders expended a similar amount of energy (Responders, 28.55 ± 2.14 MJ; Non-responders, 30.29 ± 1.76 MJ; degrees of freedom = 1, F-ratio = 0.032, $p = 0.86$). Although daily TEE increased by 0.62 ± 0.30 MJ (degrees of freedom = 1, F-ratio = 7.101, $p = 0.012$

for main effect) in the group as a whole, the increase in TEE tended to be higher in Responders than Non-responders (Responders, +1.44 ± 0.49 MJ; Non-responders, +0.29 ± 0.36 MJ, degrees of freedom = 1, F-ratio = 3.328, p = 0.078 for interaction) (Figure 2). Changes in daily AEE, reflecting changes in physical activity outside exercise sessions, were significantly different between groups and differed in direction (Responders, +0.79 ± 0.50 MJ; Non-responders, -0.62 ± 0.39 MJ, degrees of freedom = 1, F-ratio = 4.347, p = 0.046 for interaction) (Figure 2), but did not differ according to exercise pattern (Pattern A, 0.06 ± 0.45 MJ; Pattern B, -0.47 ± 0.49 MJ; degrees of freedom = 1, F-ratio = 0.636, p = 0.43 for interaction). There were no differences between Responders and Non-responders for changes in SEDEE (Responders, -0.32 ± 0.31 MJ; Non-responders, -0.13 ± 0.32 MJ,) and SEE (Responders, 0.05 ± 0.09 MJ; Non-responders, 0.07 ± 0.05 MJ).

*Paragraph Number 20* In the group as a whole, the exercise program induced a significant (p<0.05) increase in energy intake by 9.7%, although changes in the individual macronutrients (carbohydrate, protein, fat) did not achieve statistical significance. There were no significant differences between Responders and Non-responders in energy, fat, carbohydrate or protein intake (Table 2). Six participants out of 11 in the group of Responders and nine participants out of 23 in the group of Non-responders reported energy intakes of less than 1.3 x RMR through the 8 weeks of the intervention. This proportion did not differ significantly between the Responder and Non-responder groups (Chi Square, p = 0.40).

*Paragraph Number 21* In both uni- and multi-variate regression analysis, change in AEE was the only significant behavioural predictor of change in fat mass (r = -0.36, p = 0.045), explaining 13% of the variance of change in fat mass in response to the intervention.

**Discussion**

*Paragraph Number 22* The main finding of this study is that individual variability in body weight and fat changes in overweight healthy women in response to a supervised and well-controlled exercise program is, at least in part, related to individual differences in

compensatory changes in EE of physical activity outside exercise sessions. In addition to the evidence that overweight individuals who experience a lower than predicted weight loss are compensating by an increase in energy intake (23), our data suggest that success of exercise programs in relation to body fat loss could conceivably be increased by the employment of strategies directed towards the prevention of exercise-induced compensatory behaviours.

*Paragraph Number 23* Although previous evidence suggests that compensatory reduction in physical activity in non-exercise time may serve as a barrier to exercise induced body mass and body fat loss (11,16,21,34), this was the first study aiming to examine whether direction and extent of change in this compensatory behaviour differ between individuals and thus contribute to the inter-individual variability seen in body mass and body fat changes during exercise interventions (22,23). We found that the change in AEE, which included EE of all active activities except EE of exercise intervention, was significantly different between Responders – i.e. those who achieved more than or equal to their predicted fat loss and Non-responders – i.e. those who lost less than predicted fat loss. Indeed, in comparison to the AEE at baseline, the daily AEE measured during the final week of exercise programme decreased by approximately 0.62 MJ in Non-responders and increased by 0.79 MJ in Responders. Furthermore, change in AEE was a significant predictor of change in fat mass for the group as a whole.  Thus, our data indicate that lower than predicted weight and body fat loss seen in Non-responders can be attributed, at least in part, to a reduction in physical activity outside exercise sessions and implies that direction of the AEE response may be different between those who achieve and those who do not achieve body fat loss.

*Paragraph Number 24* There is a widely accepted notion that, during exercise programs, the exercise-induced energy deficit at some critical point triggers an increase in energy intake (28). However, the recent study of King et al (23) investigating mechanisms responsible for individual variability in body mass and body fat changes during exercise programs in overweight individuals and measuring energy intake changes from *ad libitum* lunch and dinner meals, reported that over the course of exercise intervention some of the participants

increased and others decreased their energy intake, and that differences in energy intake changes contributed to the individual variability in body mass and body fat loss. In contrast, we found that, the exercise-induced change in energy intake was not significantly different between those who lost less and those who lost more than predicted body fat. We appreciate that energy intake assessment using 7-day weighed intake measurements used in our study may be less precise than the measurements made in study of King et al (23) and, as is the case in all studies using weighed food records, comparison of energy intake between baseline and during last week of exercise intervention could potentially be confounded by inaccuracies in data collection (19) and underreporting (17,20). Indeed, consistent with the existing evidence that, in obese individuals, underreporting commonly lies within the range of 20-50% (17,20), we found that 40% of the participant of this study reported energy intake less than RMR x 1.3. On the other hand, it is important to note that in the present study the volume of exercise was substantially lower in comparison to the exercise volume in the study of King et al (23) (~4 MJ/week vs ~10 MJ/week). Thus, it is possible that energy intake compensatory responses to exercise may be influenced by extent of the energy balance perturbation and that that lower ExEE in the present study was below the threshold required for a compensatory response. This suggestion requires further investigation and, findings of such studies may contribute to the design of the exercise programmes that provide more favourable body fat and body weight changes.

*Paragraph Number 25* Although the capacity of behavioural responses to compensate for disturbances in energy balance are expected to be more powerful than metabolic responses (22), we appreciate that lower that expected body weight and fat loss seen in this and other supervised and controlled exercise training studies (23) may be explained not only by behavioural but also metabolic compensatory responses. For example, it has been reported that in men residing at an isolated experimental station in a highly controlled environment, imposition of an exercise-induced energy deficit of 4.2 MJ per day for 84 days, with constant energy intake, led to reductions in body weight ranging from 3 to 12 kg, which is unlikely to be fully explained by differences in compensatory activity between participants (6). One

metabolic factor which may contribute to the difference in response to exercise training is change in RMR. However, in agreement with the study of King et al (23), we found that changes in RMR between the start and end of the intervention did not differ between Responders and Non-responders groups. The identification and characterisation of metabolic compensatory responses requires further research.

*Paragraph Number 26* In this study, overweight and obese but otherwise healthy females undertook an exercise training program based on current exercise recommendations (18), exercising for 150 minutes per week at exercise intensity corresponding HR ranging from 135 to 145 beats min$^{-1}$, which corresponded to 72 to 77% of age-predicted maximum HR.  In participants who did not reduce physical activity outside exercise sessions, this volume of exercise led to a significant reduction in adiposity. It is important to note that most of the subjects, despite having quite low initial fitness levels, found this volume of exercise to be achievable and enjoyable. Therefore, when combined with advice how to eliminate behavioural compensatory responses this level of exercise can be recommended for the reduction of overweight and obesity.

*Paragraph Number 27* The data obtained in this study demonstrate that regardless of the direction and extent of body fat changes all participants experienced health benefits as a result of the exercise program. We found that waist circumference was reduced by approximately 4 cm in both Responders and Non-responders. This suggests that even under conditions of no body fat loss, exercise may induce favourable fat redistribution. This is of great importance since increased abdominal adiposity is thought to reflect visceral fat surrounding the internal organs (29), which can pose a high risk of chronic disease such as heart disease and type 2 diabetes (9). Additionally, both groups benefited from the exercise programme by increasing their $\dot{V}O_2$ max by approximately 0.74 l·min, thus improving their cardiorespiratory fitness, another important predictor of good health (14).

***Paragraph 28*** In the present study, the group as a whole did not lose a significant amount of body fat in response to the exercise intervention, which included 150 minutes of moderate intensity exercise per week. This contrasts somewhat with a recent study from Church and colleagues, who in a 6-month trial found that sedentary, overweight postmenopausal women who undertook ~72 or ~136 minutes of exercise per week had actual weight losses which did not differ significantly from predicted weight losses, whereas women who undertook ~194 minutes of exercise per week lost less weight than predicted, suggesting that whether compensation occurs is related to exercise dose (7). No differences in step-counts outside of exercise were observed between the three exercise doses (7). However, it is important to consider that over 50% of women undertaking ~136 minutes of exercise lost less weight than predicted in that study, indicating that the extent of compensation differed markedly between individuals at any given exercise dose (7). In our study of relatively young, overweight/obese women, individual differences in changes in AEE explained 13% of the variance in the extent of exercise-induced fat loss; however King and colleagues found that dietary compensation contributed to individual variability in weight loss on a group of middle-aged men and women (23) – an effect that we did not see in the present study. Thus, it appears that both the mechanisms, and the magnitude, of behavioural compensation to induced exercise differ substantially between individuals, and this information is lost when group mean values are considered. This highlights the importance of considering data at the individual, rather than group, level to obtain a more complete understanding of factors influencing the extent of fat loss in response to exercise.

***Paragraph Number 29*** The main limitations to this study, which are common to the majority of reports in this field, relate to the measurement of behavioural compensation variables. Issues related to the potential under-reporting of dietary intake (17,20) have been highlighted

above, but it important also recognise that the extent of underreporting is relatively appears to be relatively consistent within an individual (2), implying that differences in dietary intake between two observation points (e.g. changes from intake from baseline to post-intervention) are likely to be determined with greater accuracy than absolute dietary intakes at a single time-point.  Thus, the repeated-measures design in the present study may have attenuated the magnitude of this potential error.  There is no gold-standard technique for assessing components of energy expenditure in free-living individuals, as the gold-standard method for measurement of total energy expenditure – the doubly-labelled water method – does not allow for calculation of separate activity components.  We used a combination of HR monitoring and physical activity diaries to determine components of energy expenditure outwith the exercise intervention.  This approach has been shown to have greater accuracy than HR monitoring alone, agreeing well with room-calorimetry measurements (30), but the use of a self-report diary for classification of active and inactive domains could conceivably introduce errors.  In addition, the study, at 8 weeks, was relatively short-term and further investigations are needed to determine whether differences in physical activity compensation are predictive of the extent of fat loss in response to an exercise intervention over the longer term.  Further study is also needed to determine the effects of different exercise doses on behavioural compensation at the individual level.  A final limitation of the study relates to statistical power.  As it was not possible to predict the number of participants who would be classed as Responders and Non-responders until completion of the study, it was difficult to perform an *a priori* power calculation.  A retrospective power calculation indicated that the study had sufficient power to detect a difference in AEE between the Responder and Non-responder groups, however, the study was slightly underpowered to reveal a significant difference in TEE between the groups: the tendency for a difference in TEE may have become significant with a larger number of participants.

***Paragraph Number 31*** In conclusion, our data confirms that there is a large degree of inter-individual variability in body fat loss in response to an exercise training intervention and indicated that, in overweight women, compensatory reductions in EE of physical activity outside exercise intervention can contribute to the failure of exercise to successfully induce fat loss.

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**Titles and legends to figures**

**Figure 1** Individual predicted and actual change in body fat mass. Each pair of histograms represents one individual. Participants who achieved less than predicted fat loss were classified as Non-responders, and those who achieved more than or equal to their predicted fat loss were classified as Responders.

**Figure 2** Exercise induced changes in daily total energy expenditure (TEE), activity energy expenditure (AEE) calculated as EE of all active activities except exercise EE (ExEE), sedentary energy expenditure (SEDEE) and sleeping energy expenditure (SEE) in Responders and Non-responders. * significant ($p< 0.05$) difference for change between groups.

**Table 1** Subject characteristics at baseline

| | Whole Group | Responders | Non-responders |
|---|---|---|---|
| Age (years) | 31.7 ± 8.1 | 34.0 ± 6.9 | 30.7 ± 8.6 |
| Body mass* (kg) | 78.9 ± 13.2 | 75.7 ± 6.8 | 80.5 ± 15.3 |
| BMI* (kg·m$^{-2}$) | 29.3 ± 4.4 | 28.2 ± 2.0 | 29.9 ± 5.1 |
| Fat mass* (kg) | 31.7 ± 9.6 | 30.0 ± 5.2 | 32.4 ± 10.8 |
| Waist circumference* (cm) | 91.3 ± 10.3 | 91.2 ± 6.8 | 91.3 ± 11.6 |
| TEE (MJ/d) | 9.43 ± 1.66 | 8.50 ± 0.91 | 9.80 ± 1.76 |
| AEE (MJ/d) | 4.59 ± 1.72 | 3.98 ± 0.53 | 4.83 ± 1.97 |
| SEDEE (MJ/d) | 2.77 ± 0.91 | 2.50 ± 0.79 | 2.88 ± 0.95 |
| SEE (MJ/d) | 2.12 ± 0.41 | 2.03 ± 0.18 | 2.16 ± 0.47 |
| RMR* (MJ/d) | 5.95 ± 0.71 | 5.70 ± 0.46 | 6.05 ± 0.78 |
| $\dot{V}O_2$ max (l/min$^{-1}$) | 2.07 ± 0.38 | 2.06 ± 0.33 | 2.08 ± 0.40 |
| $\dot{V}O_2$ at LT (l/min$^{-1}$) | 1.36 ± 0.24 | 1.32 ± 0.22 | 1.41 ± 0.24 |
| Energy intake (MJ/d) | 8.31 ± 2.13 | 7.95 ± 1.96 | 8.45 ± 2.22 |
| Fat intake (MJ/d) | 2.79 ± 0.71 | 2.73 ± 0.60 | 2.82 ± 0.76 |
| Carbohydrate intake (MJ/d) | 4.17 ± 1.36 | 3.98 ± 1.22 | 4.25 ± 1.44 |
| Protein intake (MJ/d) | 1.34 ± 0.46 | 1.25 ± 0.29 | 1.38 ± 0.49 |
| Alcohol (MJ/d) | 0.11 ± 0.03 | 0.15 ± 0.04 | 0.07 ± 0.02 |

Abbreviations: BMI, body mass index; TEE, total energy expenditure; AEE, activity energy expenditure; SEDEE, sedentary energy expenditure, SEE, sleeping energy expenditure; RMR, resting metabolic rate; $\dot{V}O_2$ max: maximal oxygen consumption; LT, lactate threshold. Values are mean ± SD. * statistical analysis performed on logarithmically transformed data.

**Table 2** Responses to exercise programme

| | Whole Group | Responders | *Non-responders* |
|---|---|---|---|
| Body mass* (kg) | -0.15 ± 0.28 | -1.85 ± 0.46 [a] | 0.65 ± 0.20 [a,b] |
| BMI* (kg·m$^{-2}$) | -0.05 ± 0.11 | -0.65 ± 0.22 [a] | 0.23 ± 0.07 [b] |
| Fat mass* (kg) | -0.04 ± 0.24 | -1.75 ± 0.19 [a] | 0.62 ± 0.20 [a, b] |
| Waist circumference* (cm) | -3.66 ± 0.44 [a] | -4.02 ± 0.76 [a] | -3.52 ± 0.55 [a] |
| RMR (MJ/d) | 0.15 ± 0.08 | 0.09 ± 0.17 | 0.17 ± 0.09 |
| $\dot{V}O_2$ max (l/min) | 0.74 ± 0.07 [a] | 0.77± 0.10 [a] | 0.72 ± 0.15 [a] |
| $\dot{V}O_2$ at LT (l/min) | 0.17 ± 0.07 [a] | 0.24 ± 0.12 [a] | 0.14 ± 0.09 [a] |
| Energy intake (MJ/d) | 0.98 ± 0.43 [a] | 0.86 ± 0.75 | 1.03 ± 0.53 |
| Fat intake (MJ/d) | 0.35 ± 0.15 | 0.17 ± 0.22 | 0.43 ± 0.19 |
| Carbohydrate intake (MJ/d) | 0.33 ± 0.26 | 0.35 ± 0.51 | 0.33 ± 0.30 |
| Protein intake (MJ/d) | 0.25 ± 0.11 | 0.14 ± 0.22 | 0.29 ± 0.13 |
| Alcohol intake (MJ/d) | 0.02 ± 0.00 | 0.03 ± 0.00 | 0.01 ± 0.00 |

Abbreviations: BMI, body mass index; RMR, resting metabolic rate; $\dot{V}O_2$ max, maximal oxygen consumption; LT, lactate threshold. Values are mean ± SEM. [a] significant difference from baseline, p< 0.05. [b] significant difference for change between groups, p< 0.05.* statistical analysis performed on logarithmically transformed data
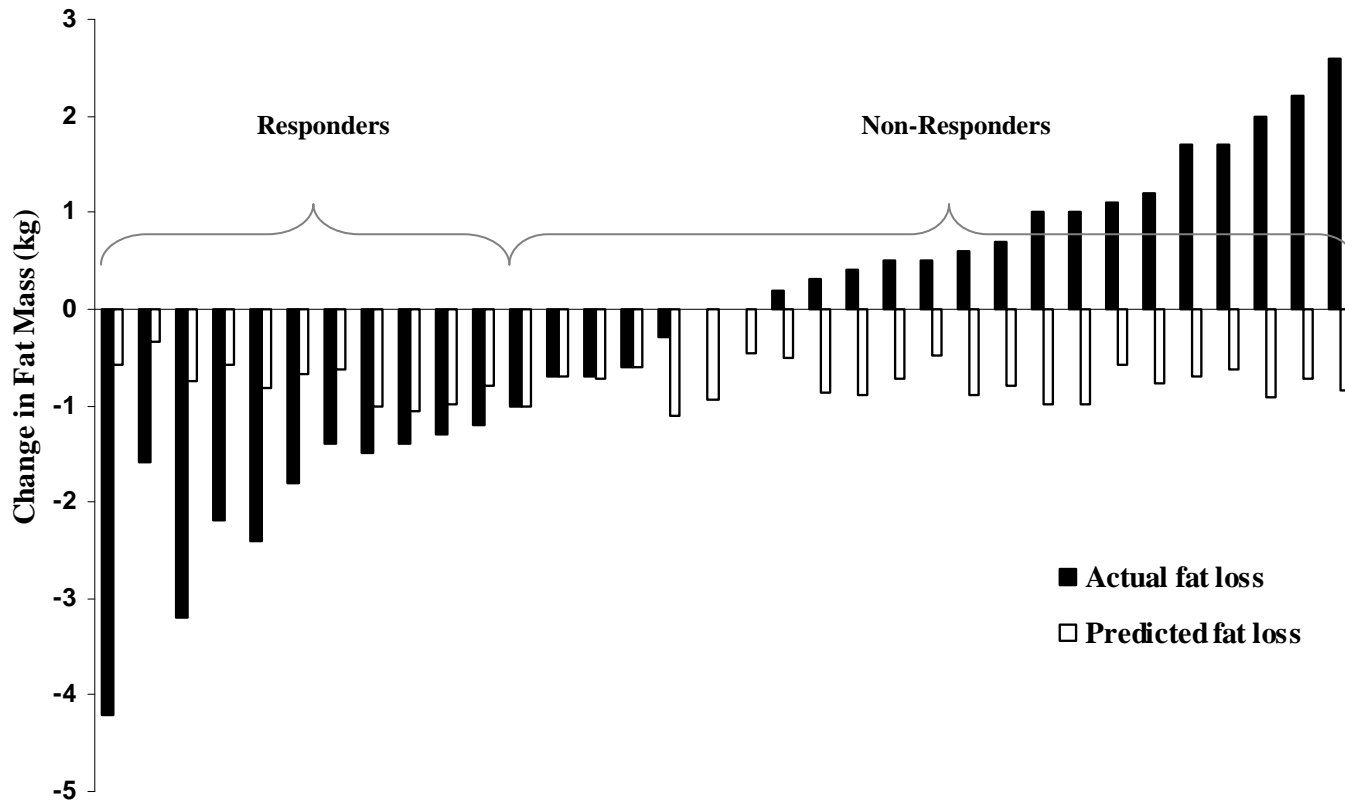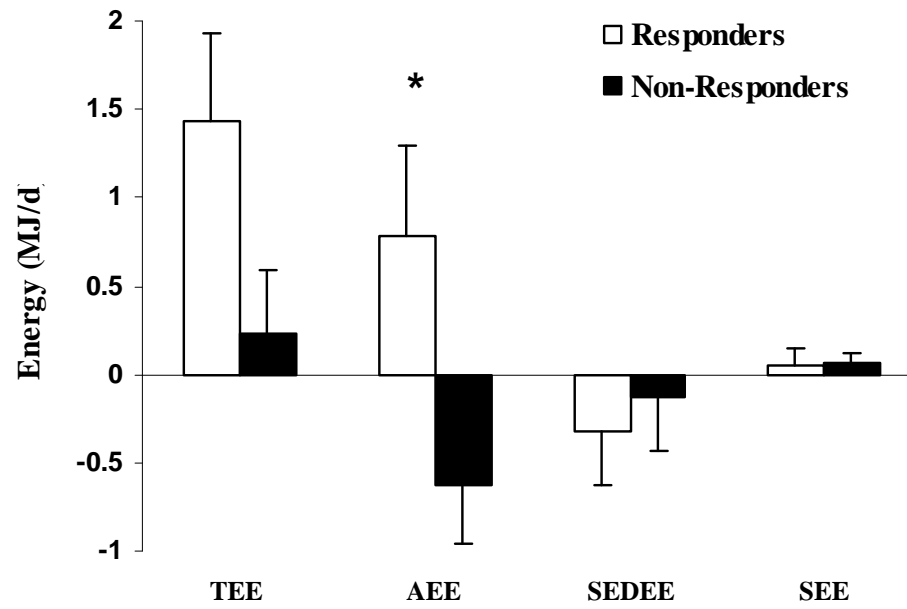
Figure 1

Figure 2