

ORIGINAL ARTICLE

Individual variability following 12 weeks of supervised exercise: identification and characterization of compensation for exercise-induced weight loss

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Objective: To identify and characterize the individual variability in compensation for exercise-induced changes in energy expenditure (EE).

Design: Twelve-week exercise intervention.

Subjects: Thirty-five overweight and obese sedentary men and women (body mass index, $31.8 \pm 4.1 \text{ kg m}^{-2}$; age, 39.6 ± 11.0 years) were prescribed exercise five times per week for 12 weeks under supervised conditions.

Measurements: Body weight, body composition, resting metabolic rate (RMR), total daily energy intake (EI) and subjective appetite sensations were measured at weeks 0 and 12.

Results: When all subjects' data were pooled, the mean reduction in body weight ($3.7 \pm 3.6 \text{ kg}$) was significant ($P < 0.0001$) and as predicted, which suggested no compensation for the increase in EE. However, further examination revealed a large individual variability in weight change (-14.7 to $+1.7 \text{ kg}$). Subjects were identified as compensators (C) or noncompensators (NC) based on their actual weight loss (mean NC = $6.3 \pm 3.2 \text{ kg}$ and C = $1.5 \pm 2.5 \text{ kg}$) relative to their predicted weight loss. C and NC were characterized by their different metabolic and behavioural compensatory responses. Moderate changes in RMR occurred in C ($-69.2 \pm 268.7 \text{ kcal day}^{-1}$) and NC ($14.2 \pm 242.7 \text{ kcal day}^{-1}$). EI and average daily subjective hunger increased by $268.2 \pm 455.4 \text{ kcal day}^{-1}$ and $6.9 \pm 11.4 \text{ mm day}^{-1}$ in C, whereas EI decreased by $130 \pm 485 \text{ kcal day}^{-1}$ and there was no change in subjective appetite ($0.4 \pm 9.6 \text{ mm day}^{-1}$) in NC.

Conclusion: These results demonstrate that expressing the exercise-induced change in body weight as a group mean conceals the large inter-individual variability in body weight and compensatory responses. Individuals who experience a lower than predicted weight loss are compensating for the increase in EE.

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Introduction

Exercise is frequently compared with diet, diet and exercise combined and pharmacological interventions as a means of reducing or maintaining body weight. Most studies evaluating the efficacy of exercise to promote body weight loss tend to report the mean data and overlook the inter-individual variability. It is unlikely that a fixed dose of exercise will be

effective to the same extent in all individuals. Similar to medical interventions, a failure of exercise to produce significant reductions in body weight is assumed to be a lack of effectiveness of the exercise treatment *per se*. The phenomenon of variability in response to drug treatment is well established in the clinical environment, such that, in general, drugs are effective only in 25–60% of patients.¹ Therefore, if an analogy is made between an exercise intervention and a dose of drug treatment, it is intuitive that the effectiveness of exercise on weight loss will also vary. Part of the variability in the effectiveness could be accounted for by compliance.^{2,3} However, even when compliance is near perfect, the effectiveness of exercise will be undermined by compensatory responses that could potentially offset the energy deficit.

The impact of exercise on weight loss has variable success because some individuals recruit adaptive mechanisms to

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oppose the negative energy balance resulting from the imposed exercise.⁴ Compensatory adaptive responses will oppose the exercise-induced energy deficit. Partial compensation for exercise-induced energy deficits is detectable over 2 weeks, and is slow and variable between individuals.⁵ Mayer *et al.*⁶ claimed that 'the regulation of food intake functions with such flexibility that an increase in energy output due to exercise is automatically followed by an equivalent increase in caloric intake'. Epstein and Wing⁷ also stated that '...exercise may stimulate the appetite so that persons who exercise increase their eating and do not lose as much weight as expected' and '...a person who exercises in the early evening may go to sleep earlier or require more rest in the evening...'. Given that energy intake (EI) and activity energy expenditure (EE) are two major behavioural determinants of body weight, their independent and combined compensatory responses could undermine the exercise-induced energy expenditure (ExEE). Although most of the evidence from acute studies suggests that there is no automatic exercise-induced increase in EI,^{5,8} there is evidence that weight loss is associated with increased motivation to eat following longer term negative energy balance interventions.⁹⁻¹³ Compensatory reductions in EE could also oppose any perturbations in energy balance. Compensatory adjustments in exercise and nonexercise activity, for example a failure to maintain a 100% compliance with the exercise regime,¹⁴ and a reduction in physical activity in the nonexercise time, could contribute to a lower than predicted weight loss. In addition to behaviourally mediated compensatory responses, metabolic responses could oppose an imposed energy deficit. It is known that when obese individuals lose weight, resting metabolic rate (RMR) decreases.¹⁵⁻¹⁷ This may also occur in response to exercise and would clearly help to offset any exercise-induced energy deficit.

It is important to identify and characterize the various components of energy balance that may undermine weight loss so that appropriate weight management strategies can be employed. For some people, exercise is an unsuccessful method of weight control. Therefore, compensatory responses could render some individuals resistant to the theoretical weight loss benefits of exercise. Additionally, individuals who fail to lose weight in response to exercise interventions may vary in the type of compensatory mechanism observed.

The concept of resistance and variability to weight gain has been discussed previously.¹⁸ The classic genetic studies conducted by Claude Bouchard¹⁹ were instrumental in identifying the variability in response to over-feeding interventions in twins. It has also been demonstrated that there is a large inter-individual variability in improvements in maximal aerobic capacity (VO_{2max}) in responses to exercise interventions.^{20,21} Therefore, the phenomenon of variability in the VO_{2max} response to exercise interventions and variability in body weight to dietary interventions has been documented before. However, the phenomenon of

variability in the changes in exercise-induced body weight has not been exposed fully. It is a relatively new concept to consider individuals who could be resistant or susceptible to exercise-induced weight loss. The identification of such individuals would be helpful in devising appropriate treatment strategies.

It is likely that individual variability will exist in the compensatory response to the exercise-induced increase in EE by volitionally altering their behaviour and/or automatic physiological changes. Therefore, compensators (C) could be defined as those who defend their body weight by adjusting for the exercise-induced increase in EE. Adjustment for the increase in EE (that is, compensation) could result from changes in both EI and EE.²² Some individuals will be predisposed to compensatory responses that render them resistant to the weight loss benefits theoretically associated with an exercise-induced increase in EE. The aim of this study was to examine the individual variability in weight change after a medium-term, supervised exercise intervention. We hypothesized that the extent and degree of compensation will vary between individuals, and explain why some individuals experience a lower than predicted weight loss.

Methods

Subjects

Thirty-five overweight and obese sedentary men ($n=10$) and women ($n=25$) with mean body mass index = $31.8 \pm 4.1 \text{ kg m}^{-2}$ and age = 39.6 ± 11.0 years were recruited for this study. All subjects gave their consent to take part in the study, and ethical approval was obtained from the Institute of Psychological Sciences Ethical committee.

Exercise intervention

Participants were subjected to a 12-week exercise programme that was individually designed to expend 500 kcal per session at approximately 70% HR maximum of 5 days per week. All exercise sessions were supervised in the research unit. Subjects wore a POLAR (S610, POLAR, Finland) heart rate monitor during each exercise session. Participants could choose from a selection of exercise modes: bicycle ergometers, stepping machines, rowing ergometers and treadmills. To account for changes in VO_{2max} and body weight, a submaximal VO_{2max} test was performed every 4 weeks to recalculate the exercise duration and intensity to prescribe the 500 kcal EE. Expired air was collected and analysed using indirect calorimetry (Sensormedics Vmax29, Conshuhocken, PA, USA). Indirect calorimetry was performed every 4 weeks to assess the EE of the prescribed exercise session. This information was used to calculate the weekly energy cost of the exercise sessions, which was eventually used to estimate the total ExEE.

Probe measurements

A range of anthropometric, behavioural and metabolic measurements were performed at week 0 (baseline) and week 12. Subjects arrived at the laboratory following an overnight fast. Measurements were taken in the following order after voiding. RMR was measured (GEM, Nutren Technology Ltd, Cheshire, UK) with subjects laying supine for 45 min under a ventilated hood. Diastolic and systolic blood pressures and resting heart rate were also measured in the supine position following 45 min of rest. This was performed using a Ormeron digital blood pressure cuff. Body weight and body composition were measured using the InBody Bioelectrical Impedance Analysis Systems (Inbody 3.0, Biospace, Seoul, Korea). Height was measured using a stadiometer (Seca, Leicester, UK).

Test meal day procedure

Twenty-four-hour energy and macronutrient intake were measured using *ad libitum* lunch and dinner test meals on a single day at weeks 0 and 12. On the morning of week 0, participants selected from an *ad libitum* breakfast. The amount and type of breakfast consumed on the test meal day in week 0 was recorded and fixed for the test meal day at week 12. Following the fixed breakfast, subjects were provided with *ad libitum* lunch and dinner meals; each meal was separated by 4 h. The macronutrient composition of the lunch (% energy 7.4, 54.3 and 38.3) and dinner (% energy 20.3, 31.2 and 48.5) for protein, fat and carbohydrate, respectively, was fixed. Participants ate alone in a cubicle and were instructed to eat to a comfortable level of fullness. On test meal days during weeks 0 and 12, subjective appetite sensations were measured immediately before and after meals, and hourly between meals using an electronic appetite rating system. This system has been validated^{23,24} and used extensively in appetite studies.^{25,26}

Treatment of data and statistical analysis

C and noncompensators (NC) were identified by comparing their predicted weight loss with actual weight loss. We estimated that a loss of 1 kg in body weight (assuming 70:30 fat/lean tissue) is equivalent to 7700 kcal.²⁷ Therefore, on the basis of the EE calculated for each individual separately, the predicted weight loss was calculated from the ExEE using the following equation:

$$\text{expected weight loss (kg)} = \frac{\text{Total ExEE}}{7700}$$

A participant was labelled a C if their actual weight loss was less than their predicted weight loss. A participant was labelled a NC if their actual weight loss was more than or equal to their predicted weight loss. Therefore, participants were identified as C or NC based on their predicted weight loss relative to their actual weight loss. This was based on the assumption that if actual weight loss was less than predicted weight loss, the individual compensated for the exercise-induced increase in EE.

Paired *t*-tests were used to compare values at week 0 with week 12 for the whole group and within each of the compensator and noncompensator groups. Independent *t*-tests were used to compare differences between the two groups.

Results

Whole group

When all 35 subjects' data were pooled, there was a significant reduction in mean body weight (3.7 ± 3.6 kg) and body fat (3.7 ± 2.6 kg) after 12 weeks of exercise (lowest $t = 6.09$, d.f. = 29; $P < 0.001$). The mean group weight loss of 3.7 kg was very similar to the weight loss predicted from ExEE data. Therefore, using the group mean there was no indication that any participants compensated for the exercise-induced increase in EE.

Individual variability: compensators and noncompensators

There was a large variability in weight and fat changes, ranging from -14.7 to $+1.7$ kg and -9.5 to $+2.6$ kg for BW and BF, respectively (see Figure 1). On the basis of the predicted weight loss associated with the exercise EE, participants were divided into two groups. By comparing the actual to predicted weight change, NC ($n = 17$) were identified as losing equal, or more than the predicted weight loss, and C ($n = 18$) were identified as losing less than the predicted weight loss, or actually gaining weight. There was no marked difference in the ratio of males/females between the two groups (NC 4:13 and C 6:12).

There was no significant difference in the measured mean gross ExEE (C = 2393 ± 547 kcal week⁻¹ and NC = 2272 ± 542 kcal week⁻¹; $t = 1.12$, d.f. = 33, NS) or the proportion of prescribed sessions attended (C = 84 and NC = 82%; $t = 0.12$, d.f. = 33, NS) between NC and C.

At baseline, there were no significant differences in any of the characteristics (see Table 1) between NC and C. However, there was a trend for the NC to have higher initial body weight and body mass index. By definition, NC lost more body weight and fat mass than C. The mean decreases in body weight and fat mass were 6.3 ± 3.2 and 5.3 ± 2.2 kg for NC, and 1.5 ± 2.5 and 2.1 ± 2.3 kg for C. When expressed as a proportion of the initial body weight, NC lost significantly a higher % body weight compared with C (6.9 ± 3.5 and $1.6 \pm 2.0\%$; $t = 5.56$, d.f. = 33; $P < 0.001$). C experienced an increase in fat-free mass (0.47 ± 1.51 kg) compared with a decrease in NC (-0.89 ± 2.12 kg); the differential in fat-free mass changes between the two groups was significant ($t = 2.2$, d.f. = 33; $P < 0.05$). Although the NC (-7.1 ± 3.6 cm) experienced a greater reduction in waist circumference compared with the C (-2.7 ± 2.9 cm), the difference between the two groups was not significant ($t = 0.27$, d.f. = 32, NS).

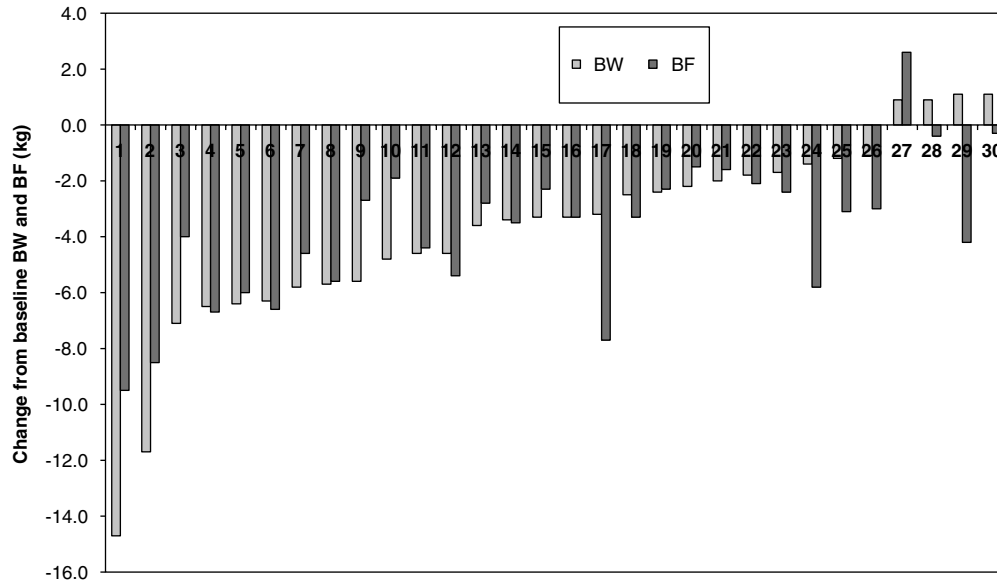


Figure 1 Individual body weight and fat mass changes after 12 weeks of imposed exercise. Each pair of histograms represents one participant.

Table 1 Mean (\pm s.d.) baseline characteristics of C and NC

	NC (n = 17)	C (n = 18)
Age (years)	39.5 \pm 13.3	38.2 \pm 9.0
BMI (kg m ⁻²)	33.1 \pm 4.7	30.7 \pm 2.9
Weight (kg)	92.2 \pm 10.9	88.4 \pm 10.4
% Body fat	37.2 \pm 7.9	32.7 \pm 8.0
VO ₂ max (ml kg ⁻¹ min ⁻¹)	28.4 \pm 5.8	28.8 \pm 5.7

Abbreviations: BMI, body mass index; C, compensators; NC, noncompensators. No significant difference between the NC and C for any baseline characteristic.

Behavioural and metabolic responses

There was no significant change in total daily test meal EI between weeks 0 and 12 when all data were pooled ($+92.8$ kcal day⁻¹). NC and C differed in the direction of their changes in EI. NC decreased EI by -130.0 ± 485 kcal day⁻¹, whereas C increased EI by 268.2 ± 455 kcal day⁻¹. The difference in EI between the two groups was statistically significant ($t = 2.35$, d.f. = 33; $P < 0.05$). Examination of the macronutrient composition revealed that there was a significant increase ($+2.0\%$) in the proportion of energy from fat at week 0 compared with week 12 ($t = -2.24$, d.f. = 17; $P < 0.05$).

The subjective sensation of hunger was different between NC and C at week 12 (see Figure 2 and Table 2). C experienced greater hunger at week 12 compared with NC. There was a trend for area under the curve hunger to increase from baseline to week 12 (26.7 ± 2.4 and 33.6 ± 2.8 mm), whereas NC experienced a negligible change in area under the curve hunger at week 12 (29.7 ± 2.4 and 30.1 ± 3.0 mm). An independent *t*-test revealed that the delta (week 0–week 12) area under the curve hunger was not statistically significant between C and NC; however, it approached significance ($t = 1.41$, d.f. = 33; $P = 0.075$).

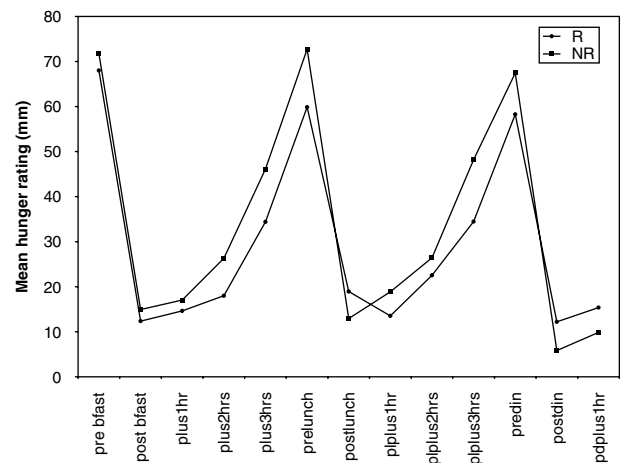


Figure 2 Profiles of subjective states of hunger at week 12 in compensators and noncompensators.

NC showed no marked change in RMR (14.2 ± 242.4 kcal day⁻¹) after 12 weeks, whereas C showed a mild, but nonsignificant decrease in RMR (-60.2 ± 298.3 kcal day⁻¹). A paired comparison revealed that there was no significant difference between the changes in RMR between the two groups.

Discussion

This study has identified several important issues and implications for exercise as a means of weight management. First, examining the overall group mean weight loss could lead to the misinterpretation that individuals do not

Table 2 Mean (\pm s.d.) energy and macronutrient intakes at weeks 0 and 12 for the whole group, C and NC

	EI (kcal)		% Fat		% Protein		% CHO	
	Week 0	Week 12	Week 0	Week 12	Week 0	Week 12	Week 0	Week 12
Whole group	2309 (495.2)	2384.1 (647.4)	32.1 (3.3)	33.3 (3.8)	12.7 (1.0)	12.6 (1.1)	55.2 (3.4)	54.2 (3.8)
NC	2269.7 (657.8)	2139.8 ^a (577.0)	31.5 (2.3)	31.7 (4.2)	12.7 (1.1)	12.4 (1.2)	55.8 (4.2)	55.9 (2.7)
C	2346.6 (411.4)	2614.8 ^a (559.8)	32.6 ^b (3.5)	34.9 ^b (3.6)	12.7 (0.9)	12.6 (1.1)	54.7 (2.1)	52.6 (4.2)

Abbreviations: C, compensators; NC, noncompensators. ^aSignificant difference in change from week 0 between NC and C. ^bSignificant increase from week 0 in C only.

compensate for the exercise-induced increase in EE, and that individuals experience the same degree of effectiveness of exercise. Second, supervised exercise produces large inter-individual variability in body weight and fat changes, and some individuals do compensate, hence lose less than the predicted amount of body weight. Finally, despite a marked increase in ExEE, some individuals experience weight gain. Therefore, these data highlight the importance of examining the individual variability in response to imposed exercise in addition to reporting pooled mean data.

In addition to identifying individuals who lose less weight than predicted, the present study has advanced our understanding of the phenomenon of individual variability by characterizing the underlying compensatory mechanisms. Partial or overcompensation for ExEE will influence the weight loss associated with an energy deficit. Although various studies have demonstrated individual variability in response to exercise and dietary interventions,^{19–21,28–32} this is the first study to attempt to characterize the causes of variability. Speculations about potential compensatory responses have been proposed and discussed previously,^{22,33} however, these data actually demonstrate the effect. Donnelly and Smith³³ stated that ‘Surprisingly little information is available for the ability of exercise to reduce risk of overweight and obesity for men and women consuming ad libitum diets from randomized controlled trials in which exercise is supervised, the energy expenditure of exercise is known, and the interactive components of energy balance are measured’. The novelty, and hence the strength, of this study is that the exercise was supervised and clamped. Therefore, unlike unsupervised exercise interventions, any variability in weight loss cannot be explained by differences in exercise compliance. In addition, these data develop the understanding of the role of exercise in weight management by actually demonstrating that behavioural and metabolic compensatory responses explain lower than expected weight loss. These data provide evidence to explain why some individuals are less successful than others at achieving weight loss. The identification and characterization of compensatory responses to exercise could be used to improve the effectiveness of exercise.

Using a predicted weight loss criterion we were able to identify C and NC. We do not claim that our procedure of identifying C and NC is perfect. We acknowledge that the

identification of C and NC is based on the assumption that the composition of exercise-induced weight loss is 70:30 (fat/lean tissue). First, this ratio is based on evidence from dietary-induced weight loss,²⁷ and second there will be inherent inter-individual variability in the ratio of body composition changes. Therefore, the variability observed in absolute weight loss could simply be an artefact of variability in the ratio of fat mass/fat-free mass changes. For completeness, we calculated and compared the responders and nonresponders both in terms of body weight and the energy equivalence of body composition changes. One problem with the energy equivalence of body composition changes is that it also involves assumptions inherent in the calculations made. The basic message of the data was independent of the method used to calculate the response to exercise. The body composition issues require more detailed treatment and further examination.

It is likely that metabolic and/or behavioural adjustments occurred to offset the increase in EE. However, these data suggest that the adaptations were more behavioural than metabolic. Indeed, the compensatory increase in EI could contribute to an explanation why the C lost less weight than predicted. We acknowledge the need to be cautious about making assumptions from the episodic test meal intake data and inferring changes in habitual intake. The test meal intake measures in this study can be considered as ‘assays’ of eating behaviour, which form snapshots of food intake. We accept that the test meal intakes may not reflect habitual EI.³⁴ However, the measure serves as an indicator of the system to exhibit compensatory changes in EI, and has been shown to detect differences in activity-induced compensation previously.³⁵

Compensatory increase in EI demonstrates the capacity of eating to undermine perturbations in energy balance.³⁶ Previous evidence indicates that individuals vary in their compensatory changes in EI.^{37,38} However, in a previous study, C and NC were categorized based on changes in EI alone, not body weight.³⁸ In addition, Woo and Pi-Sunyer³⁹ have also shown that individuals show partial compensation for an increase in EE. In the present study, although nonsignificant, there was a trend for the C to experience an increase in hunger at week 12. Previous evidence shows that weight loss produces an increase in the subjective drive to eat.^{9,10} It is not clear whether the increase in hunger

caused EI to increase or whether it was a result of the selective nutrient-specific shift of increasing fat intake. It is unlikely that both contributed. Very few studies provide evidence for the capacity of exercise to alter macronutrient preference.^{40,41} However, several studies have demonstrated that the beneficial effects of exercise on energy balance can be completely reversed when physical activity is associated with high-fat, energy-dense foods and diets.^{42–44}

These data also demonstrate that differences in metabolic responses could account for variability in weight changes. While the C and NC displayed a difference in the direction of changes in RMR, the absolute change after 12 weeks within each group was relatively small. However, the accumulative metabolic changes could contribute to long-term energy balance.^{15,16} Of course, the potency of the increase in EI experienced by the C could serve as a more significant contributor to restoring energy balance compared with less marked changes in RMR.⁵ However, this does not undermine the contribution of metabolic adjustments in response to weight loss. Indeed, there is evidence to demonstrate that lower than predicted reductions in RMR associated with body weight loss could account for an increased resistance to weight loss in response to energy deficits.¹⁷ Unlike behavioural changes such as increased EI, changes in RMR can be considered as exclusively automatic; that is, they are beyond the volitional control of the individual.²²

A key feature of this study is the attempt to characterize individuals who lose less than the predicted weight by identifying metabolic and behavioural compensatory responses. These individuals could be predisposed to compensatory responses that render them resistant to the weight loss benefits theoretically associated with an exercise-induced increase in EE. It is possible that these individuals defend their body weight independent of the circumstance. An individual's portfolio of metabolic and behavioural compensatory responses could independently and collectively cancel out the predicted exercise-induced weight loss. Indeed, those participants who gained weight demonstrated that it can be completely reversed by overcompensating. Furthermore, those who lost more weight than predicted (for example, 14.7 kg) could be under-compensating by reducing food intake.

The inter-individual variability in weight loss and identification of weight gainers in response to exercise-induced increased EE has important implications for the use of exercise as a method of weight loss. These data demonstrate that exercise may not be the most appropriate method of weight management for everyone. The individual variability reported here demonstrates the need to treat people as individuals. It also highlights the importance of determining the mechanisms that may explain the variability. In this regard, the key issue is how to change the more resistant (for example, C) to a more susceptible profile for weight management, and to use this to determine an optimal intervention. Treatment such as controlled dietary intake (for example, prescribed portion control, macronutrient

intake, timing of eating episodes) in addition to exercise could improve the compensators' responsiveness.

There are data to demonstrate that lower than expected exercise-induced weight loss is associated with reductions in exercise compliance and therefore ExEE.^{14,45} Therefore, it is intuitive to assume that individuals who lose less than the predicted amount of body weight could be doing less exercise, hence not meeting their prescribed EE. However, one strength of the present study is that all the exercise sessions were supervised in the laboratory and the EE was measured. All subjects completed a minimum of 80% of their prescribed exercise. We also showed that there was no significant difference in ExEE between the C and NC. Therefore, we can be certain that any variability in weight change is not explained by lower ExEE.

An important note is that the C can be considered as successful, because they did experience some weight loss; however, it was less than predicted. Therefore, it is important to stress that the C should not be labelled unsuccessful. Indeed, they have achieved (on average) some weight loss and they experienced other health benefits such as increased VO_{2max} and reduced resting heart rate, blood pressure (data not reported here) and waist circumference. They are interesting physiologically and psychologically because they have lost less weight—in some cases much less—than the theoretical maximum. In actual weight loss regimes, we know that this small response leads to frustration, resentment and to self-labelling as hopeless cases. This should not be the case. It is possible that C behave as NC in response to alternative interventions intended to create a negative energy balance.

The NC had higher, but not significant, body weight and body fat at baseline. Also, the NC lost a higher proportion of body weight when expressed relative to baseline body weight. Previous evidence has shown that baseline body fat is an important predictor of the EI and body weight responses.⁴⁶ It has also been suggested that body fat serves as an energy buffer, and the EI compensatory response to exercise will depend on whether lean body mass is threatened.⁵ Therefore, we acknowledge this difference in initial body weight and fat at baseline—but this difference did not account for the variability. There is some evidence to suggest that men respond better to exercise compared with women, with respect to weight loss.^{47–49} Therefore, one possible explanation for the variability in weight loss could be gender. However, our data did not reveal any difference in compensatory responses between men and women.

In conclusion, these results demonstrate large inter-individual variability in body weight changes to the same volume of imposed exercise; both the magnitude and direction varied. Some individuals do not experience the beneficial effects of exercise on body weight. The identification and characterization of the various compensatory responses to exercise are useful for explaining the variability and could be used to improve the effectiveness of exercise.

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References

- 1 Wilkinson GR. Drug metabolism and variability among patients in drug response. *N Engl J Med* 2005; **352**: 2211–2219.
- 2 Laurance BD, McGuinness I, Ridley M, Goldswain P. Nutritional supplements after hip fracture: poor compliance limits effectiveness. *Clin Nutr* 2003; **22**: 47–50.
- 3 Manninen V, Elo MO, Frick MH, Haapa K, Heinonen OP, Heinsalmi P *et al*. Lipid alterations and decline in the incidence of coronary heart disease in the Helsinki Heart Study. *JAMA* 1998; **260**: 641–651.
- 4 Stubbs RJ, Hughes DA, Johnstone AM, Whybrow S, Horgan GW, King N *et al*. Rate and extent of compensatory changes in energy intake and expenditure in response to altered exercise and diet composition in humans. *Am J Physiol Regul Integr Compar Physiol* 2004; **286**: R350–R358.
- 5 Blundell JE, Stubbs RJ, Hughes DA, Whybrow S, King NA. Cross talk between physical activity and appetite control: does physical activity stimulate appetite? *Proc Nutr Soc* 2003; **62**: 651–661.
- 6 Mayer J, Marshall NB, Vitale JJ, Christensen JH, Mashayekhi MB, Stare FJ. Exercise, food intake and body weight in normal and genetically obese adult mice. *Am J Physiol* 1954; **177**: 544.
- 7 Epstein LH, Wing RR. Aerobic exercise and weight. *Addict Behav* 1980; **5**: 371–388.
- 8 King NA, Tremblay A, Blundell JE. Effects of exercise on appetite control: implications for energy balance. *Med Sci Sports Exerc* 1997; **29**: 1076–1089.
- 9 King NA, Hester J, Gately PJ. The effect of a medium-term activity- and diet-induced energy deficit on subjective appetite sensations in obese children. *Int J Obes Rel Metab Dis* 2007; **31**: 334–339.
- 10 Drapeau V, King N, Hetherington M, Doucet E, Blundell J, Tremblay A. Appetite sensations and satiety quotient: predictors of energy intake and weight loss. *Appetite* 2007; **48**: 159–166.
- 11 Keim NL, Stern JS, Havel PJ. Relation between circulating leptin concentrations and appetite during a prolonged, moderate energy deficit in women. *Am J Clin Nutr* 1998; **68**: 794–801.
- 12 Heini A, Kirk KA, Lara-Castro C, Weinsier RL. Relationship between hunger-satiety feelings and various metabolic parameters in women with obesity during controlled weight loss. *Obes Res* 1998; **6**: 225–230.
- 13 Doucet E, Imbeault P, St-Pierre S, Almeras N, Mauriege P, Richard D *et al*. Appetite after weight loss by energy restriction and a low-fat diet-exercise follow-up. *Int J Obes Rel Metab Dis* 2000; **24**: 906–914.
- 14 Donnelly JE, Hill JO, Jacobsen DJ, Potteiger J, Sullivan DK, Johnson SL *et al*. Effects of a 16-month randomized controlled exercise trial on body weight and composition in young, overweight men and women. *Arch Intern Med* 2003; **163**: 1343–1350.
- 15 Elia M. Energy expenditure in the whole body. In: Kinney JMT (ed). *Energy Metabolism: Tissue Determinants and Cellular Corollaries*. Raven Press Ltd: New York, USA, 1992, pp 19–49.
- 16 Leibel RL, Rosenbaum M, Hirsch J. Changes in energy expenditure resulting from altered body weight. *N Engl Med* 1995; **332**: 621–628.
- 17 Doucet E, Imbeault P, St-Pierre S, Almeras N, Mauriege P, Despres JP *et al*. Greater than predicted decrease in energy expenditure during exercise after body weight loss in obese men. *Clin Sci* 2003; **105**: 89–95.

- 18 Blundell JE, Stubbs RJ, Golding C, Croden F, Alam R, Whybrow SL *et al*. Resistance and susceptibility to weight gain: individual variability in response to a high-fat diet. *Physiol Behav* 2005; **86**: 614–622.
- 19 Bouchard C, Tremblay A, Despres JP, Nadea A, Lupien PJ, Theriault G *et al*. The response to long-term overfeeding in identical twins. *N Engl J Med* 1990; **22**: 1477–1482.
- 20 Bouchard C. Individual differences in response to regular exercise. *Int J Obes* 1995; **19** (Suppl 4): S5–S8.
- 21 Hautala AJ, Kiviniemi AM, Makikallio TH, Kinnunen H, Nissila S, Huikuri HV *et al*. Individual differences in the responses to endurance and resistance training. *Eur J Appl Physiol* 2006; **96**: 535–542.
- 22 King NA, Caudwell P, Hopkins M, Byrne NM, Colley R, Hills AP *et al*. Metabolic and behavioural compensatory responses to exercise interventions: barriers to weight loss. *Obesity* 2007; **15**: 1373–1383.
- 23 Delargy HJ, Lawton CL, Smith FC, King NA, Blundell JE. Electronic Appetite Rating System (EARS): validation of continuous automated monitoring of motivation to eat. *Int J Obes* 1996; **20**: 104.
- 24 Stubbs RJ, Hughes DA, Johnstone AM, Rowley E, Reid C, Elia M *et al*. The use of visual analogue scales to assess motivation to eat in human subjects: a review of their reliability and validity with an evaluation of new hand-held computerized systems for temporal tracking of appetite ratings. *Br J Nutr* 2000; **84**: 405–415.
- 25 King NA, Lluch A, Stubbs RJ, Blundell JE. High dose exercise does not increase hunger or energy intake in free living males. *Eur J Clin Nutr* 1997; **51**: 478–483.
- 26 King NA, Craig SA, Pepper T, Blundell JE. Evaluation of the independent and combined effects of xylitol and polydextrose consumed as a snack on hunger and energy intake over 10 d. *Br J Nutr* 2005; **93**: 911–915.
- 27 Forbes GB, Kreipe RE, Lipinski B. Body composition and the energy cost of weight gain. *Hum Nutr Clin Nutr* 1982; **36C**: 485–487.
- 28 Leon AS, Gaskill SE, Rice T, Bergeron J, Gagnon J, Rao DC *et al*. Variability in the response of HDL cholesterol to exercise training in the HERITAGE Family Study. *Int J Sports Med* 2002; **23**: 1–9.
- 29 Tremblay A, Poehlman ET, Nadeau A, Dussault J, Bouchard C. Heredity and overfeeding-induced changes in submaximal exercise VO_2 . *J Appl Physiol* 1987; **62**: 539–544.
- 30 Tremblay A, Poehlman ET, Despres JP, Theriault G, Danforth E, Bouchard C. Endurance training with constant energy intake in identical twins: changes over time in energy expenditure and related hormones. *Metabolism* 1997; **46**: 499–503.
- 31 Snyder KA, Donnelly JE, Jabobsen DJ, Hertner G, Jakicic JM. The effects of long-term, moderate intensity, intermittent exercise on aerobic capacity, body composition, blood lipids, insulin and glucose on overweight females. *Int J Obes Rel Metab Dis* 1997; **21**: 1180–1189.
- 32 Levine JA, Lanningham-Foster LM, McCrady SK, Krizan AC, Olson LR, Kane PH *et al*. Interindividual variation in posture allocation: possible role in human obesity. *Science* 2005; **307**: 584–586.
- 33 Donnelly JE, Smith BK. Is exercise effective for weight loss with *ad libitum* diet? Energy balance, compensation and gender differences. *Exerc Sport Sci Rev* 2005; **33**: 169–174.
- 34 Hill AJ, Rogers PJ, Blundell JE. Techniques for the experimental measurement of human eating behaviour and food intake: a practical guide. *Int J Obes Rel Metab Dis* 1995; **19**: 361–375.
- 35 Long SJ, Hart K, Morgan LM. The ability of habitual exercisers to influence appetite and food intake in response to high- and low-energy preloads in man. *Br J Nutr* 2002; **87**: 517–523.
- 36 Blundell JE, King NA. Effects of exercise on appetite control: loose coupling between energy expenditure and energy intake. *Int J Obes* 1998; **22**: 1–8.
- 37 Stubbs RJ, Sepp A, Hughes DA, Johnstone AM, King N, Horgan G *et al*. The effect of graded levels of exercise on energy intake and

- balance in free-living women. *Int J Obes Rel Met Dis* 2002; **26**: 866–869.
- 38 Stubbs RJ, Sepp A, Hughes DA, Johnstone AM, Horgan GW, King N *et al*. The effect of graded levels of exercise on energy intake and balance in free-living men, consuming their normal diet. *Eur J Clin Nutr* 2002; **56**: 129–140.
- 39 Woo R, Pi-Sunyer FX. Effect of increased physical activity on voluntary intake in lean women. *Metabolism* 1985; **34**: 836–841.
- 40 Janssen GME, de Graaf CJJ, Saris WHM. Food intake and body composition in novice athletes during a training period to run a marathon. *Int J Sports Med* 1989; **10**: S17–S21.
- 41 Wood PD, Haskell WL, Blair SN, Williams PT, Krauss RM, Lindgren FT *et al*. Increased exercise level and plasma lipoprotein concentrations: a one-year, randomized, controlled study in sedentary, middle-aged men. *Metabolism* 1983; **32**: 31–39.
- 42 King NA, Blundell JE. High-fat foods overcome the energy expenditure due to exercise after cycling and running. *Eur J Clin Nutr* 1995; **49**: 114–123.
- 43 Tremblay A, Almeras N, Boer J, Kranenbarg EK, Despres JP. Diet composition and postexercise energy balance. *Am J Clin Nutr* 1994; **59**: 975–979.
- 44 Murgatroyd PR, Goldberg GR, Leahy FE, Gilsenan MB, Prentice AM. Effects of inactivity and diet composition on human energy balance. *Int J Obes Rel Metab Dis* 1999; **23**: 1269–1275.
- 45 Byrne NM, Meerkin JD, Laukkanen R, Ross R, Fogelholm M, Hills AP. Weight loss strategies for obese adults: personalized weight management program vs. standard care. *Obesity* 2006; **14**: 1777–1788.
- 46 Lim CL, Lee LK. The effects of 20 weeks basic military training program on body composition, VO₂max and aerobic fitness of obese recruits. *J Sports Med Phys Fitness* 1994; **34**: 271–278.
- 47 Donnelly JE, Hill JO, Jacobsen DJ, Potteiger J, Sullivan DK, Johnson SL *et al*. Effects of a 16-month randomized controlled exercise trial on body weight and composition in young, overweight men and women: the Midwest Exercise Trial. *Arch Int Med* 2003; **163**: 1343–1350.
- 48 Westerterp KR, Meijer GAL, Janssen EME, Saris WHM, Ten Hoor F. Long-term effect of physical activity on energy balance and body composition. *Br J Nutr* 1992; **68**: 21–30.
- 49 Gleim GW. Exercise is not an effective weight loss modality in women. *J Am Col Nutr* 1993; **12**: 363–367.