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

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Running Head: Exercise, compensatory responses and energy balance

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## Abstract

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

5 **Design:** 12 week exercise intervention.

**Subjects:** 35 Overweight and obese sedentary men and women (BMI;  $31.8 \pm 4.1 \text{ kg/m}^2$  age;  $39.6 \pm 11.0 \text{ yr}$ ) were prescribed exercise five times per week for 12 weeks under supervised conditions.

10 **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  
15 in weight change ( $-14.7$  to  $+1.7 \text{ kg}$ ). Subjects were identified as compensators (C) or non-compensators (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 ( $69.2 \pm 268.7 \text{ kcal/d}$ ) and NC ( $-14.2 \pm 242.7 \text{ kcal/d}$ ). EI and average daily  
20 subjective hunger increased by  $268.2 \pm 455.4 \text{ kcal/d}$  and  $6.9 \pm 11.4 \text{ mm/d}$  in the C, whereas EI decreased by  $130 \pm 485 \text{ kcal/d}$  and no change in subjective appetite ( $0.4 \pm 9.6 \text{ mm/d}$ ) in NC ( $P < 0.05$ ).

**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  
25 weight and compensatory responses. Individuals who experience a lower than predicted weight loss may be compensating for the increase in energy expenditure.

30 Key words: exercise, compensation, energy balance

## 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 only effective in 25-60% of patients (Wilkinson 2005). 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 (Laurence et al, 2003; Mannin et al, 1998). However, dissimilar to drug treatments, the effectiveness of exercise will be undermined by compensatory responses which could potentially offset the energy deficit.

The impact of exercise on weight loss has variable success because some individuals recruit adaptive mechanisms to oppose the negative energy balance resulting from the imposed exercise (Stubbs et al, 2004). Compensatory adaptive responses will oppose the exercise-induced energy deficit. Partial compensation for exercise-induced energy deficits is detectable over two weeks, and is slow and variable between individuals (Blundell et al 2003). Mayer 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” (Mayer, 1954). Epstein and Wing 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...” (Epstein and Wing, 1980). Given that energy intake and activity energy expenditure are two major behavioural determinants of body weight, their independent and combined compensatory responses could undermine the exercise-induced energy expenditure. Although most of the evidence from acute studies suggests that there is no automatic exercise-induced

increase in EI (see King et al, 1997 and Blundell et al, 2003 for reviews), there is evidence that weight loss is associated with increased motivation to eat following longer term negative energy balance interventions (King et al, 2005; Drapeau et al, 2006; Keim et al, 1998; Heini *et al*, 1998; Doucet et al, 2002). Compensatory reductions in EE could also oppose any perturbations in energy balance. Compensatory adjustments in exercise and non-exercise activity, for example a failure to maintain a 100% compliance (Donnelly et al, 2003) with the exercise regime, and a reduction in physical activity in the non-exercise time, could contribute to a lower than predicted weight loss. In addition to behaviourally mediated compensatory responses (increases in food intake and decreases in non-exercise physical activity), metabolic responses could oppose an imposed energy deficit. It is known that when obese individuals lose weight, resting metabolic rate decreases (Elia 1999, Leibel et al, 1995; Doucet et al, 2003). 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 which 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 (Blundell et al, 2005). The classic genetic studies conducted by Claude Bouchard were instrumental in identifying the variability in response to over-feeding interventions in twins (e.g., Bouchard et al, 1990). 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 (e.g., Bouchard 1995; Hautala et al, 2006). 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. Hence, it is a relatively new concept to consider individuals resistant or susceptible to exercise-induced weight loss.

The aim of this study was to examine the individual variability in weight change after a medium term exercise intervention. We hypothesize that the extent and degree of compensation will vary between individuals, and explain why some individuals experience a lower than predicted weight loss. 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.

## **Method**

### **Subjects**

35 overweight and obese sedentary men and women (BMI;  $31.8 \pm 4.1$  and age;  $39.6 \pm 11.0$ ) were recruited for this study. All subjects gave their consent to take part in the study and  
5 ethical approval was obtained from the Institute of Psychology Ethical committee.

### **Exercise Intervention**

Participants were subjected to a 12 week exercise program that was individually designed to expend 500 kcal per session at approximately 70% HR max 5 days per week. All  
10 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, a rowing ergometer and treadmills. To account for changes in  $VO_{2max}$  and body weight, a sub-maximal  $VO_{2max}$  test was performed every 4 weeks to re-calculate the exercise  
15 duration and intensity to prescribe the 500kcal energy expenditure. Expired air was collected and analysed using indirect calorimetry (Sensormedics Vmax29, USA). Indirect calorimetry was performed every 4 weeks to assess the energy expenditure of the prescribed exercise session. This information was used to calculate the weekly energy cost of the exercise sessions.

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### **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. Resting metabolic  
25 rate was measured (GEM – UK) with subjects laying supine for 45 minutes under a ventilated hood. Diastolic and systolic blood pressure and resting heart rate were also measured in the supine position following 45 minutes 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). Height was measured using a stadiometer (Seca Leicester, uk).

### **Test meal day procedure**

24 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 on at week 12. Following the fixed breakfast subjects were provided with *ad libitum* lunch and dinner meals, and a snack box for the remainder of the day (each meal was separated by 4 hours). The protein, fat and carbohydrate composition of the lunch and dinner meals was 7.4%, 54.3%, 38.3%, and 20.3%, 31.1%, 48.5% respectively. The composition of the snack box was 5.4%, 43.2% and 51.4%. The composition of the lunch, dinner and snack box meals was fixed at weeks 0 and 12. 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 (EARS). This system has been validated (Delargy et al, 1995) and used extensively in appetite studies (e.g. King et al, 1997, King et al, 2003; Stubbs et al, 2000).

### **20 Treatment of data and statistical analysis**

Compensators (C) and non compensators (NC) were identified by comparing their predicted with actual weight loss. We estimated that a loss of 1kg in body weight (assuming 70:30 fat: lean tissue) is equivalent to 7700 kcal (Forbes et al, 1992). Therefore, based on individual energy expenditure the predicted weight loss was calculated from the exercise-induced energy expenditure (ExEE) using the following equation:

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

A participant was labeled a compensator if their actual weight loss was less than their predicted weight loss. A participant was labeled as a non compensator 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 relative to their actual weight loss. This was based on the assumption that if actual weight loss was less than predicted 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 non-compensator 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 ( $3.7 \pm 3.6$  kg) and body fat ( $3.7 \pm 2.6$ kg) after 12 weeks of exercise (highest  $t$ ,  $df = 29$   $P < 0.001$ ). However, the changes in body weight ranged from a loss of 14.7 to a gain of 1.7 kg. The mean group weight loss of 3.7kg matched the predicted weight loss (3.7 kg) from exercise energy expenditure data. Therefore, using the group mean there was no indication that any participants compensated for the exercised-induced increased EE.

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### Individual variability: compensators and non-compensators

There was a large variability in weight and fat changes, ranging from  $-14.7$  to  $+1.7$ kg and  $-9.5$  to  $+2.6$  for BW and BF respectively (see figure 1). Based on the predicted weight loss associated with the exercise energy expenditure participants were divided into two groups. By comparing the actual to predicted weight change non-compensators (NC;  $n=17$ ) were identified as losing equal to, or more than the predicted weight loss, and compensators (C;  $n=18$ ) identified as losing less than the predicted weight loss, or gaining weight.

There was no significant difference in the mean gross exercise-induced EE (C= $2393 \pm 547$  kcal/wk and NC= $2272 \pm 542$  kcal/wk;  $t = 1.12$   $df = 33$   $P = 0.86$ ) or the proportion of prescribed sessions attended (C=84 and NC=82%;  $t = 0.12$ ,  $df = 33$   $P = 0.91$ ) between NC and C.

*Figure 1 and Table 1 about here*

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 BMI. By definition NC lost more body weight and fat mass than C. The mean decreases in body weight and fat mass were  $6.3 \pm 3.2$ kg and  $5.3 \pm 2.2$ kg for NC, and  $1.5 \pm 2.5$ kg and  $2.1 \pm 2.3$ kg for C. When expressed as a proportion of initial body weight, NC lost significantly a higher % body weight compared with C ( $6.9 \pm 3.5$  % and  $1.6 \pm 2.0$ %;

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df = 33, P = 0.001). C experienced an increase in lean mass ( $0.47 \pm 1.51\text{kg}$ ) compared with a decrease in NC ( $-0.89 \pm 2.12\text{kg}$ ); the differential in lean mass change between the two groups was significant ( $t = 2.2$  df = 33 P = 0.035).

## 5 *Behavioural and metabolic responses*

There was no significant change in total daily test meal EI between weeks 0 and 12 when the group's data were pooled ( $+92.8\text{kcal}$  P = 0.412). NC and C differed in the direction of their changes in EI. NC decreased EI by  $-130.0 \pm 485\text{kcal/d}$  whereas C increased EI by  $268.2 \pm 455\text{kcal/d}$ . The difference in EI between the two groups was statistically significant ( $t = 2.35$ , df = 33, P = 0.025). Examination of the macronutrient composition revealed that the increase in EI could be explained by a significant increase ( $+2.0 \pm \%$ ) in the proportion of energy from fat at week 0 compared with week 12 ( $t = 2.24$  df = 17 P =  $<0.05$ ).

*Table 2 about here*

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The subjective sensation of hunger was different between NC and C at week 12 (see Figure 2). C experienced greater hunger at week 12 compared with NC. There was a trend for area under the curve (AUC) hunger to increase from baseline to week 12 ( $26.7 \pm 2.4\text{mm}$  and  $33.6 \pm 2.8\text{mm}$ ) whereas NC experienced a negligible change in AUC hunger at week 12 ( $29.7 \pm 2.4$  and  $30.1 \pm 3$ ). An independent t-test revealed that the delta (week 0-week12) AUC hunger was not statistically significant between C and NC, however, it approached significance ( $t = 1.41$ , d.f. = 33,  $p = 0.075$ ).

*Figure 2 about here*

25

NC showed no marked change in RMR ( $-14.2 \pm 242.4\text{kcal/d}$ ) after 12 weeks whereas C showed a mild, but non-significant decrease in RMR ( $60.2 \pm 298.3\text{kcal/d}$ ). A paired comparison revealed that there was no significant difference between the change in RMR between the two groups.

## Discussion

This study has identified several important issues and implications for exercise as a means of weight management. Firstly, examining the overall group mean weight loss could lead to the misinterpretation that individuals do not compensate for the exercise-induced increase in EE, and that individuals experience the same degree of effectiveness of exercise. Secondly, 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 exercise-induced energy expenditure some individuals experience weight gain. Therefore, these data highlight the importance of examining the individual variability in response to imposed exercise, rather than 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 over-compensation for exercise-induced EE will influence the weight loss associated with an energy deficit (King 2006). Although various studies have demonstrated individual variability in response to exercise and dietary interventions (Leon et al 2002; Tremblay et al, 1987; Tremblay et al, 1997; Bouchard et al, 1990; Bouchard 1995; Snyder et al, 2003; Levine et al, 2005; Hautula et al, 2006), this is the first study to attempt to characterize the causes of variability. Furthermore, it provides evidence to explain why some individuals are less successful than others at achieving the predicted weight loss. Using a predicted weight loss criterion we were able to identify compensators and non-compensators. 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 periodic test meal intake 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 energy intake (Hill et al, 1995). 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 (Long et al, 2002).

Compensatory increase in EI demonstrates the capacity of eating to undermine perturbations in energy balance (Blundell et al, 2003). Previous evidence indicates that individuals vary in their compensatory changes in EI (Stubbs et al, 2002). However, Stubbs et al defined compensators and non-compensators based on changes in EI alone; not body weight. In addition, Woo et al have also shown that individuals show partial compensation for an increase in EE (Woo et al 1985). Although non-significant, there was a trend for the C to experience and increase in hunger at week 12. Previous evidence shows that weight loss produces an increase in the subjective drive to eat (King et al, 2005; Drapeau et al, 2006). It is not clear whether the increase in EI was caused by an increase in hunger or solely due to the nutrient specific effect of increasing fat intake. Very few studies provide evidence for the capacity of exercise to alter macronutrient preference (Jansenn et al, 1990; Wood et al, 1985). However several studies have demonstrated that the beneficial effects of exercise on EB can be completely reversed when physical activity is combined with high-fat, energy dense foods and diets (King et al, 1994; Tremblay et al, 1994; Murgatroyd et al, 1999).

These data also demonstrate that difference in metabolic responses could account for variability in weight changes. Whilst the C and NC experienced 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 (Leibel et al, 1995; Elia, 1999). 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 (Blundell et al, 2005). 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 (Doucet et al, 2003). 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 (King, 2006).

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. 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 over-compensation. 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 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 (e.g. compensators) to more susceptible to weight management, or at least determine which intervention (e.g. dietary) is more effective. Treatment such as reduced dietary intake 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 exercise-induced EE (Donnelly et al, 2003; Byrne et al, 2006). 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 energy expenditure. However, one strength of the present study is that all the exercise sessions were supervised in the laboratory and the energy expenditure was measured. All subjects completed a minimum of 80% of their prescribed exercise. We also showed that there was no significant difference in exercise-induced EE between the C and NC. Therefore, we are confident that any variability in weight change is not explained by lower than prescribed EE.

The non-compensators 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. We are also aware that previous evidence has shown that

baseline body fat is an important predictor of the EI and body weight responses (Lim and Lee, 1994). It has also been suggested that body fat serves as an energy buffer and the EI compensatory response to exercise will depend on when lean body mass is threatened (Blundell et al, 2003). Therefore, we acknowledge this difference in initial body weight and fat at baseline – but this difference did not account for the variability.

### ***Definition of compensators and non-compensators***

Due to the lack of a universally accepted definition of successful weight loss, labeling of the groups who lose or gain weight is ambiguous. The issue of establishing a definition of success associated with exercise-induced weight loss (and maintenance) was addressed over 20 years ago (Colvin and Olson, 1983). Wing and Hill (2001) have defined success by suggesting that it must include both measurements of magnitude and duration of weight loss. Others have categorized individuals based on their responses to exercise (Weinsier et al, 2002) and dietary interventions (Levine et al, 1999). However, there is still a need to have a clear criterion-related definition of the success and effectiveness of weight loss interventions. Snyder et al (2003) used a dichotomous approach to identify responders and non-responders, based on the absolute direction of their fat change. That is, individuals who gained fat mass were non-responders, while those who lost fat mass were responders; independent of the magnitude of the change. Therefore, individuals experiencing fat mass gain and loss of 0.2kg were identified as a non-responder and responder respectively; despite a relatively small differential of 0.4kg between them. We feel that our predicted weight criterion is more robust because it is objective and individually assessed. We also feel justified in labeling them as C or NC based on the assumption of compensatory responses, rather than classifying them on the quality or success of their body weight response. Indeed, the non-compensators can be considered as successful, because they did experience some weight loss – however, it was less than predicted. Therefore, we acknowledge that the NC should not be considered as unsuccessful or non-responders; simply that they experience lower than predicted weight loss, probably due to compensatory responses. We do not claim that our procedure of identifying C and NC is water-tight. However, we believe that it provides an alternative,

more objective approach. We propose that there is a need for an improved method of classifying the quality of response to exercise.

5 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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### **Figure and Table legends**

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

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

Table 1 – Mean ( $\pm$  SD) baseline characteristics of compensators and non-compensators. No significant difference between the Nc and C for any baseline characteristic.

Table 2 – Mean ( $\pm$  SD) energy and macronutrient intakes at weeks 0 and 12 for the whole group, compensators and non-compensators.

\*Significant difference in change from week 0 between NC and C.

\*\*Significant increase from week 0 in C only

Table 1

	Non-Compensators (n=17)	Compensators (n=18)
Age (years)	39.5±13.3	38.2±9.0
BMI (kg/m <sup>2</sup> )	33.1±4.7	30.7±2.9
Weight (kg)	92.2±10.9	88.4 ±10.4
% Body fat	37.2±7.9	32.7±8.0
VO <sub>2</sub> max (ml/kg/min)	28.4±5.8	28.8±5.7

Table 2

	<b>EI (Kcal)</b>		<b>% Fat</b>		<b>% Protein</b>		<b>% CHO</b>	
	<b>Wk0</b>	<b>Wk12</b>	<b>Wk0</b>	<b>Wk12</b>	<b>Wk0</b>	<b>Wk12</b>	<b>Wk0</b>	<b>Wk12</b>
<b>Whole group</b>	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)
<b>Non compensator NC</b>	2269.7 (657.8)	2139.8* (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)
<b>Compensator C</b>	2346.6 (411.4)	2614.8* (559.8)	32.6** (3.5)	34.9** (3.6)	12.7 (0.9)	12.6 (1.1)	54.7 (2.1)	52.6 (4.2)

Figure 2

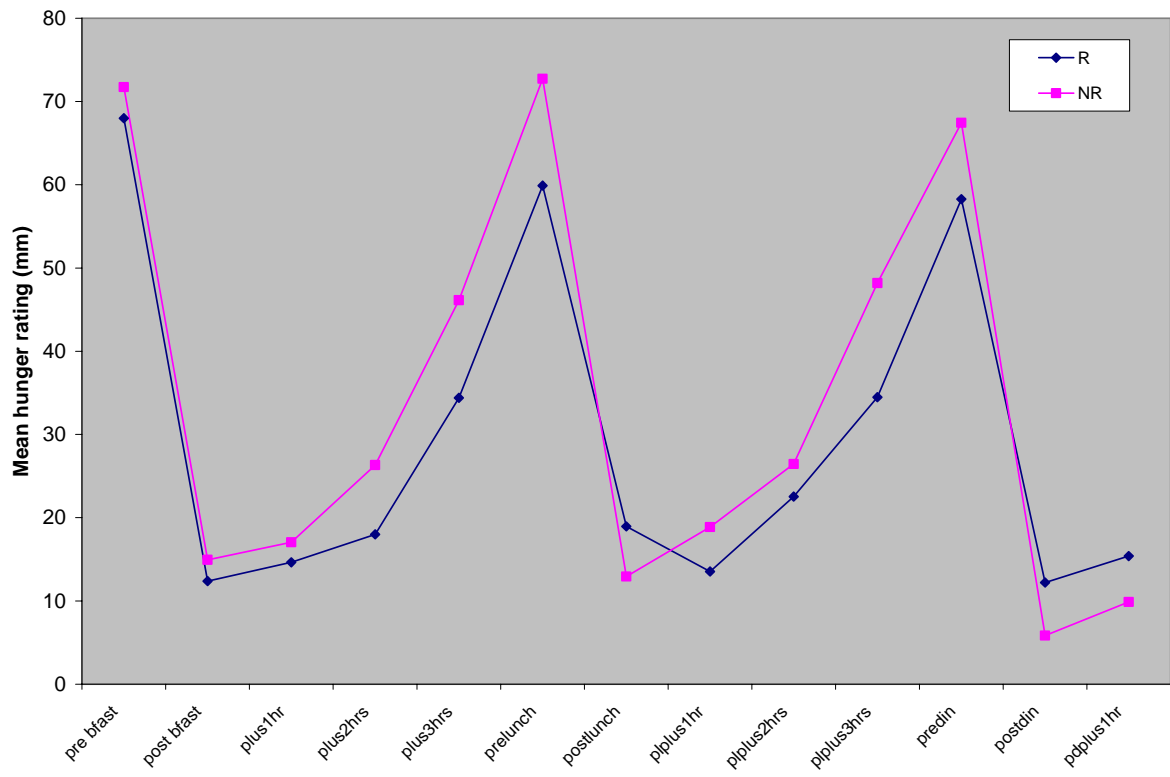


Figure 1.

