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Published version

HOPKINS, Mark, BLUNDELL, John E. and KING, Neil A. (2014). Individual variability in compensatory eating following acute exercise in overweight and obese women. British Journal of Sports Medicine, 48 (20), 1472-1476.

Individual Variability in Compensatory Eating Following Acute Exercise in Overweight and Obese Women.

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Key words: Exercise, appetite control, carbohydrate oxidation, energy intake.

Word Count: 2654

ABSTRACT

Background While compensatory eating following acute aerobic exercise is highly variable, little is known about the underling mechanisms that contribute to alterations in exercise-induced eating behaviour.

Methods Overweight and obese women (BMI = 29.6 ± 4.0kg·m^2) performed a bout of cycling individually tailored to expend 400kcal (EX), or a time-matched no exercise control condition in a randomised, counter-balanced order. Sixty minutes after the cessation of exercise, an *ad libitum* test meal was provided. Substrate oxidation and subjective appetite ratings were measured during exercise/time-matched rest, and during the period between the cessation of exercise and food consumption.

Results While *ad libitum* EI did not differ between EX and the control condition (666.0 ± 203.9kcal vs. 664.6 ± 174.4kcal, respectively; *ns*), there was marked individual variability in compensatory energy intake (EI). The difference in EI between EX and the control condition ranged from -234.3 to +278.5kcal. Carbohydrate oxidation during exercise was positively associated with post-exercise EI, accounting for 37% of the variance in EI (r = 0.57; p = 0.02).

Conclusions These data indicate that capacity of acute exercise to create a short-term energy deficit in overweight and obese women is highly variable. Furthermore, exercise-induced CHO oxidation can explain part of the variability in acute exercise-induced compensatory eating. Post-exercise compensatory eating could serve as an adaptive response to facilitate the restoration of carbohydrate balance.

WHAT ARE THE NEW FINDINGS?

- Compensatory eating in overweight and obese women following acute exercise is highly variable, but inter-individual variability is masked when reporting the mean response in food intake.

- While for some individuals there is no compensatory increase in energy intake following exercise, others partially compensate for the increased exercise-induced energy expenditure through increased energy intake.

- Carbohydrate oxidation during exercise influenced post-exercise energy intake, with those demonstrating greater carbohydrate oxidation during exercise experiencing greater post-exercise energy intake.

HOW MIGHT IT IMPACT ON CLINICAL PRACTICE IN THE NEAR FUTURE?

- The disclosure of marked individual variability in post-exercise eating behaviour helps further our understanding of how appetite regulation is affected by acute exercise.

- Future research should focus on further characterising the physiological and behavioural mechanisms that mediate compensatory changes in energy intake, and turn, body weight regulation.

INTRODUCTION

While acute exercise does not stimulate an obligatory rise in post-exercise energy intake (EI) to restore energy balance,[1] marked individual variability exists in post-exercise compensatory eating behaviour in lean[2] and obese females[3]. Energy intake increases in some people and partially compensates for the exercise-induced energy expenditure (ExEE), while others show no evidence of compensation.[2 3] Such variability is consistent with the heterogeneity reported in body weight and eating behaviour changes following long-term exercise in overweight and obese individuals,[4 5] but is concealed in studies reporting the mean response only. However, currently little is known about the underling mechanisms that contribute to alterations in exercise-induced eating behaviour.

While non-homeostatic factors such as food hedonics have been shown to play a role,[2] compensation in EI may also be metabolically driven. Given the tight regulation of carbohydrate (CHO) in the body,[6] CHO metabolism could influence eating behaviour in order to regulate energy and substrate balance. This would be of importance for exercise-induced compensatory eating, as exercise is a potent metabolic stimulus that perturbs nutrient balances.[7] These disturbances may encourage post-exercise compensation as glycogen availability may act as a signal that influences day-to-day EI.[8] However, while a negative CHO balance following dietary and/or exercise manipulation of glycogen has been shown to predict greater *ad libitum* EI,[9-13] findings are equivocal.[14-17]

Given the variability in substrate oxidation during[18] and following exercise,[19] those who rely more heavily on CHO oxidation during/following exercise could be more susceptible to post-exercise compensatory eating (due to an elevated drive to restore CHO balance). Indeed, it has been suggested that poor metabolic flexibility could increase the risk of weight gain, with a positive energy balance in part driven by glycogenostatic feeding.[20] However, evidence of a direct link between substrate oxidation during exercise and post-exercise compensatory eating is limited.[21 22] Therefore, this study aimed to characterise the variability in acute compensatory eating, and examine the role of substrate oxidation during and following exercise in driving such behaviour.

METHODS

Participants

Sixteen pre-menopausal overweight or obese females (mean (± SD) BMI = 29.6 ± 4.0kg·m^2; Table 1) were recruited via poster or email advertisements. Participants were sedentary (≤ 2hrs·wk^{-1} of moderate intensity exercise over the previous six months), weight stable (± 2kg for the previous three months), non-smokers and not taking medication known to effect metabolism or appetite. All participants gave their written informed consent, and the study was approved by the Institute of Psychological Sciences Ethics Board, University of Leeds, UK.

Table 1: Descriptive characteristics of participants.

	Mean (SD)
Age (yrs)	39.3 (10.3)
BMI (kg·m^2)	29.6 (4.0)
Body Mass (kg)	79.8 (12.1)
Waist Circumference (cm)	111.6 (7.7)
RMR (kcal·day^{-1})	1465.1 (165.6)
Maximal Aerobic Capacity (ml·kg^{-1}·min^{-1})	32.0 (9.6)

BMI, body mass index; RMR, resting metabolic rate.

Study Design

Following a preliminary assessment visit, participants performed an exercise (EX) or time-matched no exercise control condition in a randomised, counter-balanced order. Each condition was performed at the same time of day (8-9am) following an overnight fast, and consisted of three phases: 1) pre-treatment, 2) metabolic assessement, and 3) a 60 minute recovery phase. Phases One and Three did not differ between conditions. During EX, Phase Two consisted of cycling (gross ExEE = 400kcal), while during the control condition exercise was substituted for time-matched rest. Substrate metabolism and subjective appetite were measured throughout, and an *ad libitum* test meal was provided at the end of Phase Three (Figure 1). Experimental conditions were separated by one week, with participants maintaining their normal dietary and physical ativity patterns between conditions.

Figure 1 here

Preliminary Assessment

Body composition, resting metabolic rate (RMR) and maximal aerobic capacity were measured in the morning (8-9am) following an overnight fast (10-12hrs). To exclude any residual effects of previous exercise, participants were instructed to avoid strenous exercise 24hrs prior to testing. Resting metabolic rate was measured using an indirect calorimeter fitted with a ventilated hood (GEM, Nutren Technology Ltd, Cheshire, UK), based on The American Dietetic Association guidelines.[23] Resting substrate oxidation was calculated using standard stoichiometric equations[24]. Air-displacement plethysmography (BOD POD Body Composition System, Life Measurement, Inc., Concord, USA) was used to measure body composition, according to manufacturer's instructions. Waist circumference was measured at the narrowest point between the lower costal border and the iliac crest.[25] Finally, maximal aerobic capacity was determined using a validated incremental treadmill test.[26]

Experimental Conditions

At the start of each experimental condition, participants rated their feelings of subjective appetite (hunger, fullness and desire to eat) using 100mm visual analogue scales (VAS). These scales have previously been shown to be valid and reliable.[27] Participants then completed either a bout of cycling, expending 400kcal at 70% of their age predicted maximum heart rate (EX), or an equivalent period of seated rest (control). The required duration of exercise (and therefore seated rest) was calculated using stoichiometric equations[24] based on data from the incremental treadmill test. During exercise/rest, respiratory data was measured continously (Sensormedics Vmax29, Yorba Linda, USA). Data was averaged over time periods that equated to the expenditure of 100 (T_1), 200 (T_2), 300 (T_3) and 400kcal (T_4) during exercise, with the same periods used during time-matched rest.

Following exercise/rest, participants underwent a 60 minute seated recovery period in which respiratory measures were taken at 0-10 (R10), 30-35 (R35) and 50-60 (R60) minutes, and appetite ratings were taken at 0, 10, 35 and 60 minutes. Finally, an *ad libitum* test meal was administered, consisting of risotto (Uncle Ben's Mediterranean Vegetable), yoghurt (Yeo Valley Organic Strawberry) and water. When participants had reached comfortable fullness, a final post-meal appetite rating was taken. Participants ate in isolation and were instructed to eat as much or as little as they

wanted until comfortably full. Food was provided in excess of expected consumption, with participants able to request further food and water if required. Energy and macronutrient intake was calculated by weighing the food before and after consumption, and with reference to the manufacturers' energy values. The mean (± SD) proportions of energy contributed by fat, protein, and carbohydrate in the meal were 24.1 ± 6.0%, 11.2 ± 2.0%, and 64.7 ± 7.6%, respectively. Before commencing the study, prospective participants completed a food preference questionnaire, and if they disliked any of the test foods, they were excluded.

Statistical Analysis

Data are reported as mean ± SD throughout. Statistical analyses were performed using PASW for windows (SPSS, Chicago, Illinois, Version 18.0). Paired sample *t*-tests were used to examine differences in the ExEE and EI. Two-way ANOVAs with repeated measures (time*condition) were performed to examine differences in substrate oxidation and subjective appetite. Where appropriate, Bonferroni corrections were applied to *post-hoc* analysis. Bivariate and partial correlation analyses were used to test for associations between EI, substrate oxidation and appetite, while simple linear regression was used to calculate the proportion of between-subject variance in EI explained by exercise CHO oxidation.

RESULTS

Energy Expenditure & Substrate Oxidation

By design, total energy expenditure for EX (exercise and recovery) was significantly greater than during the control condition (445.2 ± 54.0 vs. 88.4 ± 22.5kcal; $p < 0.001$). Energy expenditure during the exercise phase of EX (Phase Two) was 390.2 ± 16.4kcal, as compared to 39.4 ± 13.3kcal during the control condition ($p < 0.001$). During the recovery phase (Phase Three), energy expenditure did not differ between EX and the control condition (54.4 ± 25.5 vs. 49.0 ± 13.1kcal, respectively; $p = 0.29$; Table 2).

Table 2: Mean (± SD) energy expenditures during the phases of EX and the control condition.

	Mean (SD)
Total Energy Expenditure during EX (kcal)	445.2 (54.0)*****
Total Energy Expenditure during Control (kcal)	88.4 (22.5)
Energy Expenditure during Exercise (kcal)	390.2 (16.4)******
Energy Expenditure during Time-matched Rest (kcal)	39.4 (13.3)
Energy Expenditure during Recovery of EX (kcal)	54.4 (25.5)
Energy Expenditure during Recovery of Control (kcal)	49.0 (13.1)

*Total energy expenditure during EX significantly different from total energy expenditure during the control condition ($p < 0.001$). **Energy expenditure during exercise significantly different from energy expenditure during time-matched rest ($p < 0.001$).

Mean RER during the exercise phase of EX (Phase Two) was 0.86 ± 0.04, while during the equivalent phase of the control condition, RER was 0.80 ± 0.05. Mean CHO and fat oxidation during exercise was 23.30 ± 10.79 and 8.20 ± 2.97$mg \cdot kg^{-1} \cdot FFM \cdot min^{-1}$, respectively. During the equivalent rest phase of the control condition, mean CHO and fat oxidation was 1.56 ± 0.97 and 1.33 ± 0.69$mg \cdot kg^{-1} \cdot FFM \cdot min^{-1}$, respectively. There was a significant main effect of time ($p < 0.001$), condition ($p < 0.001$), and a time*condition interaction ($p < 0.001$) for RER, CHO and fat oxidation, such that these variables were significantly higher during the exercise phase of EX than during the equivalent phase of control (Figure 2). During Phase Three (recovery), RER was significantly lower at R10 during EX ($p = 0.01$), and remained lower throughout Phase Three ($p > 0.05$).

Figure 2 here

Subjective Appetite and Food Intake

There was no difference in EI between EX and the control condition (666.0 ± 203.9 vs. 664.6 ± 174.4kcal, respectively; $p > 0.05$). Significant main effects of time ($p < 0.001$) and condition ($p < 0.001$) were observed for hunger, desire to eat and fullness, but no time*condition interactions were noted ($p > 0.05$). Food intake after EX or the control condition was not related to hunger, desire to eat or fullness ($p > 0.05$). In addition, post-exercise EI following EX was not related to ExEE or exercise duration ($p > 0.05$).

Examination of the individual responses in EI revealed marked between-subject variability (Figure 3). The differences in EI between EX and the control condition were calculated (EX EI – Control EI) to provide an indicator of compensation (EI_{diff}). EI_{diff} ranged from -234.3 to +278.5kcal. Nine participants ate less following EX compared to the control condition (mean reduction: -100.3 ± 60.0kcal), one participant showed no change in EI (-1.5kcal), while six participants ate more (mean increase: +154.3 ± 77.2kcal). To account for differences in exercise-and-rest-induced EE, and to determine the degree of EI compensation, the difference between EE_{diff} (total EE during EX – total EE during control) and EI_{diff} was compared. The net energy deficit created by exercise ranged from -112.6 to -543.3kcal.

Figure 3 here

Substrate Oxidation and Food Intake

Total exercise CHO oxidation (54.4 ± 22.2g) was positively associated with EI following EX ($r = 0.61$; $p = 0.01$), and this relationship remained after controlling for body weight ($r = 0.58$; $p = 0.02$), fat-free mass ($r = 0.56$; $p = 0.02$) or fat mass ($r = 0.60$; $p = 0.01$). Similarly, EI following EX was associated with the rate of CHO oxidation during exercise ($mg \cdot kg^{-1} \cdot FFM \cdot min^{-1}$; $r = 0.57$; $p = 0.02$). There were also significant associations between exercise CHO oxidation and CHO ($r = 0.63$; $p = 0.02$) and protein intake ($r = 0.53$; $p = 0.03$), but not fat intake ($r = 0.43$; $p = 0.09$). However, substrate oxidation during the recovery phase of either condition was not related to EI (Table 2).

Table 3: Correlation coefficients for energy intake (kcal), resting metabolic rate ($kcal \cdot day^{-1}$), resting substrate oxidation ($mg \cdot kg \cdot FFM^{-1} \cdot min^{-1}$) and total carbohydrate and fat oxidation (g) during Phase Two EX and Control.

Variable	***Energy Intake***		
	r	r^2	***P* value**
Total CHO oxidation during exercise of EX	0.61	0.37	$p = 0.01$*
Total fat oxidation during exercise of EX	0.18	0.03	$p = 0.51$
Total CHO oxidation during EX recovery	0.21	0.04	$p = 0.46$
Total fat oxidation during EX recovery	0.35	0.13	$p = 0.19$
Total CHO oxidation during time match rest (Control)	0.21	0.05	$p = 0.43$
Total fat oxidation during time match rest (Control)	0.36	0.13	$p= 0.18$
Total CHO oxidation during Control recovery	0.19	0.01	$p = 0.69$
Total fat oxidation during Control recovery	0.46	0.21	$p = 0.71$
RMR and EI during EX	0.61	0.38	$p = 0.01$*
RMR and EI during Control	0.41	0.17	$p = 0.12$
Resting CHO oxidation and EI during EX	0.11	0.01	$p = 0.99$
Resting fat oxidation and EI during EX	0.01	0.01	$p = 0.99$
Resting CHO oxidation and EI during Control	-0.21	0.04	$p = 0.47$
Resting fat oxidation and EI during Control	0.31	0.09	$p = 0.25$

CHO, carbohydrate; RMR, resting metabolic rate; EI, energy intake; FFM, fat-free mass; EX, exercise condition; Control, no exercise condition. *Correlation is significant at the 0.05 level (2 tailed).

DISCUSSION

This study examined the relationship between substrate oxidation during and following exercise, and variability in post-exercise compensatory eating in overweight and obese women. In line with previous findings, our data demonstrated that the degree of EI compensation in response to acute exercise is highly variable. This heterogeneity was concealed by the mean response, and was not related to differences in body composition or ExEE. This highlights the importance of examining biological and behavioural responses to exercise at the individual level; although acute exercise did not elicit immediate compensation in EI in all individuals, some clearly showed exercise-induced overconsumption. Here, approximately 56% of participants consumed the same or less following exercise, while 38% consumed more following exercise. Although exercise created an acute energy deficit in all individuals, the actual deficit ranged from -112.6 to -543.3kcal. As the capacity of exercise to create a net energy deficit is moderated by compensatory eating, a greater understanding of the mechanisms that confer susceptibility to exercise-induced overconsumption is warranted.

Carbohydrate Oxidation and Post-Exercise Compensatory Eating

In the present study, total exercise CHO oxidation was strongly associated with post-exercise EI during EX, with greater CHO oxidation associated with greater EI. This relationship was not influenced by body composition or habitual diet, and is consistent with the Glycogenostatic theory of feeding[8] and reports that a negative CHO balance is associated with increased EI.[9-13] Greater CHO oxidation during exercise could augment reductions in glycogen stores and increase perturbations to CHO balance, eliciting a greater compensatory drive in EI to restore CHO balance. However, it should be recognised that CHO oxidation during exercise is unlikely to act in isolation (or indeed be the main driver) in promoting exercise-induced compensatory eating. Such compensation will be mediated through a combination of homeostatic (e.g. gastrointestinal peptides) and non-homeostatic factors (e.g. food hedonics[2]). However, the relative contribution of these, or how they interact within a co-ordinated regulatory system, is unclear.

However, as total CHO oxidation during EX was small (57.5 ± 22.3g), the impact of EX on stored glycogen would have been minimal. This is of importance as the pathways through which perturbations to glycogen stores are signalled to the brain to elicit compensatory feeding are unclear.[28 29] Vagal afferent nerve activity has been suggested as the signal between the liver and the central nervous system.[30] However, it cannot be ruled out that the associations between CHO balance and EI[9-13] are co-incidental rather than causal. Indeed, the observed relationship between CHO oxidation and EI may reflect exercise-induced changes in appetite related peptides, which act to regulate EI while mediating substrate metabolism. In the present study, post-exercise substrate oxidation was measured as exercise-induced changes during this period could have influenced subsequent EI. However, substrate oxidation during recovery and EI were not related, despite large variability in post-exercise CHO and fat oxidation. Given the low absolute rates of post-exercise substrate turnover, even after accounting for any exercise-induced elevation, the impact on glycogen concentrations would have been minimal.

Methodological Issues and Areas for Future Research

Although exercise CHO oxidation was also associated with CHO and protein intake following EX, the test meal employed was not designed to examine differences in macronutrient intake. These relationships are likely to be a function of the total energy consumed, rather than substrate-driven macronutrient selection. The test meal employed was designed to detect small (but meaningful) changes in EI, despite marked variability in EI and substrate oxidation. Indeed, buffet meals with a wide selection of highly palatable foods may not be sensitive to small changes in hunger and satiation, as they may promote hedonically driven overconsumption. However, we recognise that compensation to exercise was only measured at one meal, and therefore the effects of exercise on 24hr energy balance cannot be determined. Indeed, changes in EI at a single meal may not alter 24hr EI owing to compensation at subsequent meals.

It should also be noted that this study was conducted in fasted overweight and obese women. The effect of prior nutritional status on compensatory eating is unclear. Although post-prandial exercise has been shown to suppress post-exercise hunger to a greater extent than fasted exercise,[31] these differences are not reflected in subsequent

EI.[17] Sex differences have also been reported in the appetite-related hormonal response to acute exercise,[32] and the efficacy of exercise-induced weight loss.[33] However, recent evidence indicates that when the ExEE is matched, no sex differences exist in the compensatory responses (body composition, EI or appetite) to chronic exercise.[34] Consequently, the effect of gender and prior nutritional status needs to be addressed, as variability in post-exercise compensatory EI has only been examined in fasted women. Furthermore, differences in susceptibility should be explored between lean and obese individuals.[35]

Practical Implications

The disclosure of large variability in post-exercise EI could be dismissed as random variability. However, the clear association with underlying biological variables suggests this is not the case. Rather, this variability reflects a dynamic regulatory system in which physiological mediators can act as drivers of behaviour. Acknowledgment of this variability is of fundamental importance, as without this, a full understanding of how appetite regulation is affected by exercise cannot be achieved. The characterization of susceptible/resistant individuals is needed to further our understanding of the behavioural and biological mechanisms that mediate weight loss, and help explain why some people fail to lose as much weight as expected during exercise-induced weight loss.[36]

Conclusions

These data confirm that the capacity of acute exercise to create a short-term energy deficit in overweight and obese women is highly variable, and is mediated by post-exercise EI. While for some individuals there is no compensatory increase in EI following exercise, others partially compensate for the ExEE through increased EI. While the physiological and behavioural mechanisms behind this variability need further examination, greater CHO oxidation during exercise was associated greater post-exercise EI. As such, the metabolic response to a bout of exercise may mediate acute perturbations to energy balance, with CHO oxidation during exercise providing a potential stimulus for post-exercise compensatory eating.

FIGURE LEGENDS

Figure 1: Schematic representation of the study design. RMR, resting metabolic rate; BC, body composition; WC, waist circumference; VO_2max, maximal aerobic capacity; HRmax, heart rate maximum; TFEQ, Three factor eating questionnaire; RER, respiratory exchange ratio; VAS, visual analogue scale.

Figure 2: Mean changes in RER (Panel A), carbohydrate (Panel B) and fat oxidation (Panel C) during Phases Two and Three of EX and the control condition. CHO, carbohydrate; FFM, fat-free mass; EX, exercise condition; Control, no exercise condition. *Significant difference between EX and the control condition ($p < 0.05$).

Figure 3: Individual variability in compensatory EI (EI_{diff}) following acute exercise. EX, exercise condition; Control, no exercise condition.

ACKNOWLEDGEMENTS

The authors declare no conflicts of interest.

NK and JB receive funding from the Biotechnology and Biological Sciences Research Council (grant number BB/G005524/1).

The authors' responsibilities were as follows: MH, JB and NK conceived the project. MH carried out the data collection and analysed the data. MH, JB and NK all contributed to the writing of the manuscript.

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