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**Appetite, energy intake and resting metabolic responses to 60 min treadmill running performed in a fasted versus a postprandial state.**

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## **Abstract**

This study investigated the effect of fasted and postprandial exercise on appetite, energy intake and resting metabolic responses. Twelve healthy males (mean±SD: age 23±3 years, body mass index 22.9±2.1 kg.m<sup>-2</sup>, maximum oxygen uptake 57.5±9.7 mL.kg<sup>-1</sup>.min<sup>-1</sup>) performed three 10 h experimental trials (control, fasted exercise and postprandial exercise) in a Latin Square design. Trials commenced at 8 am after an overnight fast. Sixty min of treadmill running at ~70% of maximum oxygen uptake was performed at 0-1 h in the fasted exercise trial and 4-5 h in the postprandial exercise trial. A standardised breakfast was provided at 1.5 h and ad libitum buffet meals at 5.5 and 9.5 h. Appetite ratings and resting expired air samples were collected throughout each trial. Postprandial exercise suppressed appetite to a greater extent than fasted exercise. Ad libitum energy intake was not different between trials, resulting in a negative energy balance in exercise trials relative to control after accounting for differences in energy expenditure (control: 9774±2694 kJ; fasted exercise: 6481±2318 kJ; postprandial exercise: 6017±3050 kJ). These findings suggest that 60 min treadmill running induces a negative daily energy balance relative to a sedentary day but is no more effective when performed before or after breakfast.

**Keywords:** exercise, appetite, energy intake, energy balance, compensation, energy expenditure, substrate metabolism.

## Introduction

Obesity is classified as a body mass index (BMI) equal to or greater than  $30 \text{ kg.m}^{-2}$  and represents a major global health problem as it is associated with an increased prevalence of chronic diseases, including type 2 diabetes, hypertension, dyslipidaemia, and cardiovascular disease (National Institute of Health, 1998). The prevalence of obesity has increased to such an extent that in 2008 an estimated 9.8 % of adult men and 13.8 % of adult women worldwide were classified as obese (Finucane et al., 2011). Obesity is the result of a chronic positive energy balance achieved via a long term surplus of energy intake over energy expenditure. Therefore methods of maximising energy expenditure and/or minimising energy intake are important in combating obesity.

Exercise is an important component of successful long-term weight control programs and is particularly effective when combined with dietary modifications (Franz et al., 2007). Exercise can induce a negative energy balance not only by increasing energy expenditure but also by modulating energy intake (Hubert, King, & Blundell, 1998) as strenuous exercise has been found to acutely suppress hunger in a phenomenon described as 'exercise-induced anorexia' (King & Blundell, 1995; King, Burley, & Blundell, 1994).

The majority of short (1 - 2 d) to medium (7 - 16 d) term intervention studies indicate that acute exercise does not provoke compensatory increases in appetite and food intake or alter macronutrient preferences (Blundell & King, 1999; Blundell, Stubbs, Hughes, Whybrow, & King, 2003; Martins, Morgan, & Truby, 2008). In this regard, recent studies have demonstrated exercise to be more effective than energy restriction in creating an energy deficit without subsequent increases in appetite and energy intake

(Hubert et al., 1998; King et al., 2011a). With this knowledge, research must now focus on maximising the effectiveness of exercise as a method of producing a negative energy balance for weight control. One potential avenue for maximisation is to manipulate the timing of exercise in relation to food consumption as both exercise and feeding influence appetite and subsequent energy intake (King et al., 2011a).

A common strategy to facilitate weight and fat loss is to perform aerobic exercise after an overnight fast. Exercise in the fasted state enhances fat oxidation due to decreased glycogen availability and a lipolytic hormonal environment including reduced plasma insulin concentrations and elevated cortisol and epinephrine concentrations (De Bock et al., 2005; Febbraio, Chiu, Angus, Arkinstall, & Hawley, 2000; Maughan, Fallah, & Coyle, 2010). However, recent evidence suggests that postprandial exercise may be more beneficial for weight control than fasted exercise as a result of more favourable effects on appetite regulation and resting metabolism. In this regard, Cheng, Bushnell, Cannon, & Kern (2009) recently demonstrated that 50 min of cycling at 60 % of  $\text{VO}_2$  max resulted in more prolonged hunger suppression when performed 2 h after a high fat breakfast (70 % fat, 26 % carbohydrate, 4 % protein) rather than after a 12 h overnight fast. Furthermore, previous evidence suggests that post-meal decreases in appetite are attenuated after fasted compared with postprandial exercise (Borer, Wuorinen, Chao, & Burant, 2005). Such findings indicate that energy intake may be lower after postprandial exercise than fasted exercise. However, ad libitum energy intake was not assessed in these studies and it is therefore unknown if such appetite responses would influence energy intake and subsequent energy balance.

Postprandial exercise may also promote metabolic changes that are more beneficial to weight loss than those induced by fasted exercise. Paoli et al. (2011) recently demonstrated that 36 min of treadmill exercise stimulated a greater increase in resting energy expenditure in the 24 h after exercise when performed 40 min after a 673 kcal Mediterranean breakfast (53 % fat, 22 % carbohydrate, 25 % protein) compared with when exercise was completed immediately before breakfast after a 12 h overnight fast. Furthermore, in comparison with exercise in the fasted state, postprandial exercise resulted in higher proportional fat metabolism in the 24 hours after exercise, which has been shown to be predictive of fat loss (Barwell, Malkova, Leggate, & Gill, 2009).

As well as substrate metabolism being influenced by exercise and energy intake, there has recently been renewed interest in the theory that substrate metabolism may itself be a biological determinant of feeding behaviour (Hopkins, Jeukendrup, King, & Blundell, 2011; King et al., 2011b). Evidence from pharmacological studies suggests that fatty acid oxidation may inhibit food intake via the prolongation of post-meal satiety (Gatta et al., 2009; Scharrer & Langhans, 1986). Furthermore, the glycogenostatic theory proposed by Flatt in 1987 suggests that short term feeding behaviour is designed to replenish and maintain glycogen stores (Flatt, 1987). Similarly, higher proportional carbohydrate oxidation during exercise has been suggested to increase subsequent energy intake (Almeras, Lavalley, Despres, Bouchard, & Tremblay, 1995; Kissileff, Pi-Sunyer, Segal, Meltzer, & Foelsch, 1990). However, this finding is inconsistent (Imbeault, Saint-Pierre, Almeras, & Tremblay, 1997; Klausen et al., 1999) and requires further investigation.

The ability of exercise to induce a negative energy balance depends on the energy cost of exercise as well as its ability to modify appetite, energy intake and post-exercise metabolic responses (Tremblay & Therrien, 2006). This study seeks to compare these variables in response to fasted and postprandial exercise in order to elucidate the most beneficial timing of exercise as a method of inducing a negative energy balance. Therefore the primary purpose of the present study was to investigate the effect of 60 min of treadmill running on prolonged appetite, energy intake and resting metabolic responses when performed 1.5 h before or 2.5 h after a high carbohydrate breakfast. We hypothesised that postprandial exercise would stimulate greater increases in resting energy expenditure and induce a more prolonged appetite suppression than fasted exercise, resulting in lower energy intake and a greater negative 24 h energy balance.

## **Methods**

*Participants.* Following the approval of Loughborough University's Ethics Advisory Committee, twelve healthy males (18 - 27 years) gave their written informed consent to participate. Participants were non-smokers, not taking medication, not hypertensive (blood pressure <140/90 mmHg) and were not dieting or undertaking any extreme dietary habits. Participants had no known history of cardiovascular/metabolic disease. The physical characteristics of participants (mean  $\pm$  SD) were as follows: age  $23 \pm 3$  years, body mass index (BMI)  $22.9 \pm 2.1 \text{ kg.m}^{-2}$ , body mass  $70.3 \pm 8.8 \text{ kg}$ , body fat  $13.8 \pm 4.1 \%$ , waist circumference  $78.4 \pm 6.5 \text{ cm}$ , maximum oxygen uptake  $57.5 \pm 9.7 \text{ mL.kg}^{-1}.\text{min}^{-1}$ .

*Participant Screening.* Prior to main trials participants visited the laboratory to undergo screening, familiarization and preliminary anthropometric measurements.

Questionnaires were completed to assess food preferences, health status and habitual physical activity. Height was measured to the nearest 0.1 cm using a stadiometer (Seca Ltd, Germany) and body weight was measured to the nearest 0.1 kg using a digital scale (Seca 770, Seca Ltd, Germany). Body mass index was subsequently calculated. Body density was estimated via subcutaneous fat measurements of the bicep, tricep, subscapular and suprailliac sites (Durnin & Womersley, 1974), taken using skinfold callipers (Baty International, West Sussex, UK) and body fat percentage was ascertained (Siri, 1956). Waist circumference was determined as the narrowest part of the torso between the xiphoid process and the iliac crest. Blood pressure was measured after a 5 min rest in a seated position using an electronic blood pressure monitor (OMRON M5-I, Hoofddorp, Netherlands).

*Exercise Tests.* After familiarisation with the testing equipment, participants completed a 16 min submaximal incremental treadmill running test on a level motorised treadmill (RUNRACE, Techno gym, Gambettola, Italy) in order to determine the relationship between running speed and oxygen consumption. The initial running speed was set between 6.5 and 12 km.h<sup>-1</sup> depending on the fitness level of each participant and was increased by 1 - 1.5 km.h<sup>-1</sup> after the completion of each 4 min stage. Oxygen consumption was determined from expired gas collections taken during the final minute of each stage along with the participant's rating of perceived exertion (RPE) using the Borg scale (Borg, 1973). Heart rate was monitored continuously using short-range radio telemetry (Polar T31; Polar Electro, Kempele, Finland).

After a 20 - 30 min rest, maximal oxygen uptake (VO<sub>2</sub> max) was assessed using an incremental uphill treadmill running test at constant speed to volitional exhaustion

(Jones & Doust, 1996). Run speed was set at the speed corresponding to a heart rate of  $\sim 150 \text{ beats} \cdot \text{min}^{-1}$  or an RPE of 12 on the submaximal exercise test. The test commenced on a level treadmill and the incline increased by 1 % every minute until volitional exhaustion, which was reached within 9 - 12 min. Maximal oxygen consumption was determined from an expired air sample collected during the final minute of the test when participants signalled that they could only continue for an additional 1 min. Heart rate and RPE were monitored throughout the test.

*Experimental Protocol.* Participants performed three experimental trials (control, fasted exercise and postprandial exercise) separated by at least one week in a Latin Square design. Each trial lasted 10 h and commenced at 8 am after an overnight fast of at least 10 h. Participants completed a weighed food diary in the 24 h before the first main trial and replicated this before each subsequent trial. Alcohol, caffeine and strenuous physical activity were not permitted during this period. Participants exerted themselves minimally when travelling to the laboratory and travelled via motorized transport when possible.

During each trial, appetite perceptions (hunger, satisfaction (satiety), fullness and prospective food consumption (PFC)) were assessed at baseline and every 30 min throughout using 100 mm visual analogue scales (Flint, Raben, Blundell, & Astrup, 2000). Environmental temperature and humidity were also measured at these times using a handheld hygrometer (Omega RH85, Manchester, UK). Resting expired air samples were collected in a seated position after 5 min seated rest at: -5 min, 2.25, 3.25, 6.25, 7.25, 8.25, 9.25, and 10 h. Energy expenditure and substrate oxidation rates were calculated from  $\text{VO}_2$  and  $\text{VCO}_2$  values using stoichiometric equations, based on the

assumptions that 1 g of carbohydrate utilises 0.828 L of oxygen and produces 0.828 L of carbon dioxide and 17 kJ of energy, whereas 1 g of fat utilises 1.989 L of oxygen and produces 1.419 L of carbon dioxide and 39 kJ of energy (Frayn, 1983).

The fasted exercise trial (FAST) commenced with a 60 min run on a level treadmill at a speed predicted to elicit 70 % of maximal oxygen uptake. Samples of expired gas were collected every 15 min during exercise to monitor the intensity of the run, with adjustments made to the treadmill speed if necessary. Heart rate and RPE were also assessed at these times. After the run, participants rested for 9 h in the laboratory (sitting reading, working at a desk or watching television). Identical procedures were completed during the postprandial exercise trial (FED) except the run was completed from 4 – 5 h rather than 0 – 1 h. The control trial (CON) was identical to the two exercise trials except that no exercise was performed. In order to estimate net energy expenditure of the run (gross energy expenditure of run minus resting energy expenditure), samples of expired air were collected when resting during the control trial at time points when exercise was taking place in FAST and FED to determine resting energy expenditure. Figure 1 provides an overview of the protocol for the experimental trials.

*Standardised breakfast and ad libitum buffet meals.* Participants were provided with a standardised breakfast at 1.5 h, which consisted of toasted white wheatgerm bread, margarine, strawberry jam, banana and orange juice. The macronutrient content of the meal was 72.9 % carbohydrate, 9.5 % protein, 17.6 % fat. The breakfast provided 30% of the estimated daily energy needs for each individual for a sedentary day. To calculate this, resting daily energy requirements were estimated for each individual

(Mifflin et al., 1990). This value was then multiplied by a physical activity level of 1.4 to represent a sedentary day.

Participants were given 30 min access to ad libitum buffet meals provided at 5.5 and 9.5 h, from which energy and macronutrient intake was covertly monitored. The buffet foods were identical before each meal (Appendix A) and provided diversity in protein, fat and carbohydrate content. Food was presented in excess of expected consumption and participants were told to eat until satisfied and that additional food was available if desired. Participants consumed meals in isolation so that social influence did not affect food selection. Food consumption was determined by weighing buffet items before and after each meal. The energy and macronutrient content of the items consumed was ascertained using manufacturer values. Before the main trials, acceptability of the buffet food items presented was ensured after completion of a food preference questionnaire. The questionnaire required participants to rate preselected food items on a scale ranging from 1 (dislike extremely) to 10 (like extremely). Questionnaires were examined to ensure that food items would be to the taste of each individual. Distaste for the buffet items (rating 5 items < 5) resulted in participant noninclusion.

Upon leaving the laboratory, overnight food consumption was monitored via a weighed food diary. Water was available ad libitum throughout each trial.

*Statistical Analysis.* Data was analysed using Predictive Analytics Software version 18.0 for Windows (SPSS Inc., Somers, NY, U.S.A.). All area under the concentration versus time curve (AUC) calculations were performed using the trapezoidal method. Exercise responses in FAST and FED were compared using Students paired t-tests. One-way repeated measures ANOVA was used to assess trial-based differences in total

energy/macronutrient intake as well as fasting appetite perceptions and fasting and AUC values for resting energy expenditure and substrate oxidation. Repeated measures, two-factor ANOVA was used to examine differences between trials over time for appetite perceptions, energy/macronutrient intake, energy expenditure and substrate oxidation. The Pearson product moment correlation coefficient was used to examine relationships between variables. Assumptions of sphericity in the data were checked, and adjustments in the degrees of freedom for the ANOVA were made using the Greenhouse-Geiser method of correction where appropriate. Where significant trial and interaction effects were found, post-hoc analysis was performed using Bonferroni correction for multiple comparisons. Statistical significance was accepted at the 5 % level. Results in text and tables are presented as mean  $\pm$  SD. Graphical representations of results are presented as mean  $\pm$  SEM to avoid distortion of the graphs.

## **Results**

*Exercise Responses.* Participants completed the 60 min run at  $10.2 \pm 2.5$  km.h<sup>-1</sup> during both FAST and FED. This elicited a mean oxygen consumption equivalent to  $71.1 \pm 2.1$  and  $71.9 \pm 2.7$  % of maximum oxygen uptake and generated a mean heart rate of  $162 \pm 12$  and  $165 \pm 14$  beats.min<sup>-1</sup> in FAST and FED, respectively. The net energy expenditure of the run was  $3247 \pm 423$  kJ ( $776 \pm 101$  kcal) in FAST and  $3234 \pm 435$  kJ ( $773 \pm 104$  kcal) in FED. None of these values differed significantly between exercise trials.

The mean respiratory exchange ratio (RER) during exercise was significantly lower in FAST than FED ( $0.91 \pm 0.03$  vs.  $0.93 \pm 0.03$ ;  $P = 0.008$ ), reflecting a higher proportional contribution of fat and lower contribution of carbohydrate to energy

provision during fasted exercise (FAST:  $31.2 \pm 10.6$  % fat,  $68.8 \pm 10.6$  % carbohydrate; FED:  $22.3 \pm 11.6$  % fat;  $77.7 \pm 11.6$  % carbohydrate).

Mean RPE did not differ between trials ( $13 \pm 1$  and  $12 \pm 1$  in FAST and FED respectively) and indicated that participants perceived the intensity of the run to be 'fairly hard'.

*Baseline Parameters.* No between trial differences existed at baseline for appetite perceptions, energy expenditure or RER (Table 1).

*Appetite.* Two-factor ANOVA revealed a main effect of time (all  $P < 0.0005$ ) and a trial x time interaction (all  $P < 0.03$ ) for each appetite perception assessed (hunger, fullness, satisfaction and PFC) indicating that responses differed over time between the trials (Figure 2). Analysis also revealed a main effect of trial for satisfaction ( $P = 0.037$ ) demonstrating elevated perceptions during FED compared with FAST ( $P = 0.01$ ).

Post-hoc analysis of trial x time interactions revealed a decrease in PFC and an increase in satisfaction during exercise in FAST compared with CON at 0.5 h. Differences in ratings of satisfaction and fullness were apparent at 3.5 h as well as differences in PFC from 2.5 - 3.5 h, demonstrating greater appetite in FAST than FED shortly after breakfast. Each appetite perception differed in FED compared with CON and FAST from 4 – 5.5 h, indicating suppressed appetite during and shortly after exercise in FED (all  $P < 0.05$ ). However, after Bonferroni adjustment only higher satisfaction at 3.5 h and lower hunger at 4.5 h in FED compared with FAST remained significant ( $P < 0.0005$ ).

*Energy and macronutrient intake.* Two-factor ANOVA showed a significant difference in the amount of energy consumed at the separate meals during the course of the trials (main effect of time,  $P < 0.0005$ ). However, this differential was not influenced by the trials (Table 2). For fat, protein and carbohydrate intake, two-factor ANOVA revealed a main effect of time ( $P < 0.0005$ ) indicating that the intake of these macronutrients varied across the meals within each trial. There was no difference in macronutrient intake between trials (Table 3).

*Energy Expenditure and Substrate Utilisation.* Two-factor ANOVA revealed a significant trial, time and trial x time interaction for energy expenditure throughout the trials (all  $P < 0.0005$ ; Figure 3). Energy expenditure was higher during the exercise trials than control ( $P < 0.0005$ ). At individual time points post hoc analysis indicated elevated energy expenditure in FAST than CON at 0.25, 0.5, 0.75, 1, 4.25, 4.5, 6.25, 7.25 and 8.25 h, as well as higher energy expenditure in FAST than FED at 0.25, 0.5, 0.75, 1, 2.25 and 9.25 h (all  $P < 0.05$ ). Energy expenditure was higher in FED than FAST and CON at 4.25, 4.5, 4.75 and 5 h. However, after Bonferroni adjustment only higher energy expenditure in FAST than FED and CON at 0.25, 0.5, 0.75 and 1 h (i.e. during exercise in FAST), and higher energy expenditure in FED than FAST and CON at 4.25, 4.5, 4.75, and 5 h (i.e. during exercise in FED) remained significant (all  $P < 0.0005$ ).

Two-factor ANOVA for RER revealed a significant time and trial x time interaction (both  $P < 0.0005$ ) but no main effect of trial ( $P = 0.273$ ; Figure 4). Post-hoc analysis indicated higher RER in FAST than FED and CON at 0.25, 0.5 and 1 h, as well as higher RER in FED than FAST and CON at 4.25, 4.5, 4.75 and 5 h. At rest, RER was

higher in CON than FAST at 3.25, 4.75, 5 and 6.25 h and in CON than FED at 6.25 and 7.25 h (all  $P < 0.05$ ). However, after Bonferroni adjustment only elevated RER in FAST than FED and CON at 0.25, 0.5 and 1 h (i.e. during exercise in FAST), and higher RER in FED than FAST and CON at 4.25, 4.5 and 4.75, and in FED than FAST at 5 h (i.e. during exercise in FED) remained significant ( $P \leq 0.001$ ).

In order to calculate area under the curve values exclusively for *resting* energy expenditure and RER, data during exercise was substituted for control data from equivalent time points. One-way ANOVA revealed a significant main effect of trial for area under the resting energy expenditure verses time curve from 0 – 5 h, 5 – 10 h and for the total 10 h trial (all  $P < 0.015$ ). Post-hoc analysis indicated higher resting energy expenditure in FAST than CON and FED for each time period (all  $P < 0.05$ ) but these differences were no longer significant after Bonferonni adjustment.

One-way ANOVA revealed a significant main effect of trial for area under the RER verses time curve from 5 – 10 h and for the total 10 h trial (all  $P < 0.025$ ). Post-hoc analysis indicated lower RER in FAST and FED than CON from 5 - 10 h and in FAST than CON for the total trial (all  $P < 0.05$ ). After Bonferonni adjustment, RER during FAST remained lower than CON for 5 – 10 h and the total 10 h trial (all  $P < 0.01$ ).

*Relative energy intake.* After accounting for the energy expenditure induced by exercise, participants remained in energy deficit in both FAST and FED compared with CON (both  $P < 0.0005$ ) but no differences existed between exercise trials (CON  $13,451 \pm 2682$  kJ ( $3215 \pm 641$  kcal); FAST  $10,406 \pm 2289$  kJ ( $2487 \pm 547$  kcal); FED  $9699 \pm 2866$  kJ ( $2318 \pm 685$  kcal)). Relative energy intake was 22.6 and 27.9 % lower than CON in FAST and FED after accounting for the energy cost of exercise. Similar

findings existed after energy intake was adjusted for estimated energy expenditure for the entire 10 h trial based on exercising and resting energy expenditure (CON  $9774 \pm 2694$  kJ ( $2336 \pm 644$  kcal); FAST  $6481 \pm 2318$  kJ ( $1549 \pm 554$  kcal); FED  $6017 \pm 3050$  kJ ( $1438 \pm 729$  kcal); both  $P < 0.0005$ ). Relative energy intake was 33.7 and 38.4 % lower than CON in FAST and FED after accounting for the estimated energy expenditure for the 10 h trial. Closer inspection of the data revealed that 7 of 12 participants exhibited a higher relative energy intake in FAST than FED, while 5 of the 12 participants demonstrating a higher relative energy intake in FED than FAST. All participants exhibited lower relative energy intake in both exercise trials compared with control.

*Correlations between energy intake and other variables.* Differences in relative energy intake between the exercise trials were calculated via the subtraction of FED from FAST values. This difference was positively correlated with body weight ( $r = 0.603$ ;  $P = 0.038$ ) and BMI ( $r = 0.810$ ;  $P = 0.001$ ), indicating greater increases in REI after fasted exercise compared with postprandial exercise as body weight and BMI increased. However, this was not correlated to age,  $\text{VO}_2$  max or body fat percentage. Mean energy intake of the trials was negatively correlated with ‘cognitive restraint of eating’ ( $r = -0.580$ ,  $P = 0.048$ ) as assessed by the three-factor eating questionnaire but was not related to ‘disinhibition’ or ‘hunger’ (Stunkard and Messick 1985). Neither exercising RER nor 10 h mean RER were significantly related to energy or macronutrient intake.

## **Discussion**

The purpose of this investigation was to examine the appetite, energy intake and resting metabolic responses to a prolonged bout of treadmill running when performed under

fasted or postprandial conditions. The primary findings arising from this study were that postprandial exercise invoked a more substantial and prolonged suppression of appetite than fasted exercise. However, this did not result in any differences in energy intake between trials and elicited a negative energy balance relative to control in both exercise trials.

Fasted exercise reduced sensations of prospective food consumption and increased feelings of satisfaction during the exercise bout, which returned to normal levels upon completion. This indicates a suppression of appetite during exercise and is consistent with the phenomenon of 'exercise-induced anorexia' (King & Blundell, 1995; King et al., 1994). Exercise in the postprandial period inhibited post-meal increases in hunger and PFC and decreases in satisfaction and fullness during and immediately after exercise. This is again consistent with exercise-induced anorexia and supports the findings of Cheng et al. (2009) that postprandial exercise has greater appetite suppressive effects than fasted exercise. The mechanisms underlying appetite suppression during exercise are still unclear but recent evidence suggests that exercise-induced changes in episodic appetite regulating gut peptides such as peptide YY, glucagon-like-peptide-1, pancreatic polypeptide and acylated ghrelin may be implicated (Broom, Batterham, King, & Stensel, 2009; King et al., 2011a; Martins, Morgan, Bloom, & Robertson, 2007; Ueda, Yoshikawa, Katsura, Usui, & Fujimoto, 2009).

In addition to exercise-induced changes in appetite, there was also a tendency for increased perceptions of PFC and decreased feelings of satisfaction and fullness in FAST than FED immediately after breakfast, prior to the postprandial exercise bout. This supports the findings of Borer et al. (2005) that the post-meal decline in appetite is

attenuated when fasted exercise is performed prior to breakfast. Although this suggests that a small compensatory rise in appetite may occur after fasted exercise, it is also plausible that appetite perceptions were lower in FED after breakfast due to the anticipation of forthcoming exercise. This finding coupled with the greater and more prolonged appetite suppression during FED exercise, resulted in higher satisfaction during the FED trial than FAST and indicates greater appetite suppression when exercise is performed after, rather than immediately before, breakfast.

However, such differences in appetite did not influence ad libitum energy or macronutrient intake during the remainder of the day. This may be explained by the transient nature of exercise-induced anorexia as appetite was suppressed almost exclusively during and immediately after exercise. This suggests that appetite suppression during exercise has little influence on subsequent energy intake and supports previous evidence that exercise does not provoke short term compensatory changes in food and macronutrient intake (Blundell & King, 1999; Blundell et al., 2003; Martins et al., 2008).

Consistent with a longstanding body of literature, resting energy expenditure and proportional fat oxidation tended to be higher after both exercise sessions compared with the control trial (Poehlman, 1989; Votruba, Atkinson, Hirvonen, & Schoeller, 2002). However, the tendency for higher resting energy expenditure after fasted exercise than postprandial exercise and no difference in resting RER between exercise trials contrasts with recent findings by Paoli et al. (2011). Such differences are likely to be a result of variations in study protocol as exercise was performed 2.25 h into the postprandial period in the present study but only 40 min after breakfast in that of Paoli

et al. (2011). The postprandial increase in energy expenditure in the present study began to subside before the commencement of exercise, whereas exercise in the study conducted by Paoli et al. (2011) corresponded with a substantial postprandial increase in energy expenditure, which was concluded to be the stimulus for an elevation in resting 24 h energy expenditure.

Reductions in resting RER after exercise have been suggested to promote the replenishment of muscle glycogen stores (Hopkins et al., 2011) and a positive relationship has been demonstrated between muscle glycogen utilisation during exercise and post-exercise increases in fat oxidation (Henderson et al., 2007). Despite elevated carbohydrate oxidation during postprandial exercise compared with fasted exercise, it is plausible that muscle glycogen utilisation did not differ between exercise bouts in the present study. Evidence suggests that increases in muscle glycogen utilisation only occur during postprandial exercise when rebound hypoglycaemia occurs (Febbraio & Stewart, 1996; Hargreaves, Costill, Katz, & Fink, 1985). Such rebound hypoglycaemia is unlikely to have occurred during postprandial exercise in the present study due to the substantial time period between breakfast and postprandial exercise. Such similar levels of glycogen depletion during the exercise bouts may explain similar RER values observed at rest during the afternoon of the exercise trials. However, this remains speculative as blood glucose and muscle glycogen content were not measured in the present study.

Despite renewed interest in the idea that substrate metabolism may act as a biological determinant of feeding behaviour (Hopkins et al., 2011; King et al., 2011b), there was no relationship between substrate oxidation and energy/macronutrient intake in the

present study. This adds to the equivocal evidence regarding this theory and suggests that other physiological mediators of appetite and energy intake are likely to be more influential in affecting these variables. It is also plausible that previously recognised relationships between glycogen availability and energy intake may be coincidental with other factors (e.g. hormones) acting to regulate energy intake while also mediating substrate metabolism (Hopkins et al., 2011). This is supported by evidence that peripheral ghrelin infusion increases carbohydrate oxidation as well as increasing appetite and energy intake (Tschöp, Smiley, & Heiman, 2000). Such relationships may be implicated in previous findings that increased carbohydrate oxidation is related to increased energy intake.

The substantial energy deficits incurred during both the fasted and postprandial exercise bouts did not elicit any compensatory increases in energy intake during the remainder of the day, which resulted in a negative 24 h energy balance in both exercise trials compared with control. Although the main determinants of energy balance are behavioural (i.e. exercise energy expenditure and energy intake) (King et al., 2007), the stimulation of resting energy expenditure after exercise further increased the energy gap between control and exercise trials, resulting in energy deficits of 33.7 and 38.4 % in the fasted and postprandial trials relative to control. It has previously been demonstrated that individual differences occur in the energy intake responses to exercise-induced energy deficits (Finlayson, Bryant, Blundell, & King, 2009) and are implicated in the success of weight loss programs (King, Hopkins, Caudwell, Stubbs, & Blundell, 2008; King et al., 2009). However, all participants in the present study incurred a negative energy balance in both exercise trials compared with control. It is plausible that compensatory increases in energy intake may occur in the longer term and

a longer observation period is needed to elucidate such responses. However, this study substantiates evidence that appetite and energy intake responses to exercise are certainly not as immediate or powerful as those to diet-induced energy deficits (Hubert et al., 1998; King et al., 2011a).

This study has a number of notable limitations. Firstly, the population sample comprised of young, physically active, healthy males; therefore the findings may not generalise to other populations such as females, the elderly, sedentary, or overweight and obese populations. Secondly, the exercise undertaken in this study was physically challenging and is unlikely to be an achievable form of exercise for sedentary and clinical populations. Further work should be performed to investigate the effect of exercise on energy balance in overweight and obese populations as this is the population where weight management strategies hold the most clinical relevance. Thirdly, circulating concentrations of appetite-regulating hormones were not measured in the present study and may be prudent in future investigations to elucidate potential mechanisms underlying the appetite and energy intake response to exercise. Finally, energy intake may have been influenced by the different time periods between the cessation of exercise and the ad libitum meals in the fasted and postprandial trials. However, this is unavoidable when controlling for feeding state and manipulating the timing of exercise in relation to breakfast and represents an ecologically valid scenario.

In conclusion, this is the first study to directly compare the ad libitum energy intake response to fasted and postprandial exercise and demonstrates that enhanced appetite suppression during postprandial exercise does not transpire into altered energy intake. Subsequently both fasted and postprandial exercise induced a negative daily energy

balance relative to the control trial but there appears to be no additional benefit of exercising before or after breakfast. This study further supports the use of exercise in weight management programs and highlights the importance of monitoring energy intake as well as the appetite response to exercise.

## References

- Almeras, N., Lavallee, N., Despres, J. P., Bouchard, C., & Tremblay, A. (1995). Exercise and energy intake: effect of substrate oxidation. *Physiology & Behavior*, 57, 995-1000.
- Barwell, N. D., Malkova, D., Leggate, M., & Gill, J. M. R. (2009). Individual responsiveness to exercise-induced fat loss is associated with change in resting substrate utilization. *Metabolism: Clinical and Experimental*, 58, 1320-8.
- Blundell, J. E., & King, N. A. (1999). Physical activity and regulation of food intake: current evidence. *Medicine and Science in Sports and Exercise*, 31, S573-83.
- Blundell, J. E., Stubbs, R. J., Hughes, D. A., Whybrow, S., & King, N. A. (2003). Cross talk between physical activity and appetite control: does physical activity stimulate appetite? *The Proceedings of the Nutrition Society*, 62, 651-61.
- Borer, K. T., Wuorinen, E., Chao, C., & Burant, C. (2005). Exercise energy expenditure is not consciously detected due to oro-gastric, not metabolic, basis of hunger sensation. *Appetite*, 45, 177-81.
- Borg, G. A. (1973). Perceived exertion: a note on “history” and methods. *Medicine and Science in Sports*, 5, 90-3.
- Broom, D. R., Batterham, R. L., King, J. A., & Stensel, D. J. (2009). Influence of resistance and aerobic exercise on hunger, circulating levels of acylated ghrelin, and peptide YY in healthy males. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*, 296, R29-35.

Cheng, M. H., Bushnell, D., Cannon, D. T., & Kern, M. (2009). Appetite regulation via exercise prior or subsequent to high-fat meal consumption. *Appetite*, 52, 193-8.

De Bock, K., Richter, E. A., Russell, A. P., Eijnde, B. O., Derave, W., Ramaekers, M., Koninckx, E., et al. (2005). Exercise in the fasted state facilitates fibre type-specific intramyocellular lipid breakdown and stimulates glycogen resynthesis in humans. *The Journal of Physiology*, 564, 649-60.

Durnin, J. V., & Womersley, J. (1974). Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *The British Journal of Nutrition*, 32, 77-97.

Febbraio, M. A., Chiu, A., Angus, D. J., Arkinstall, M. J., & Hawley, J. A. (2000). Effects of carbohydrate ingestion before and during exercise on glucose kinetics and performance. *Journal of Applied Physiology*, 89, 2220-6.

Febbraio, M. A., & Stewart, K. L. (1996). CHO feeding before prolonged exercise: effect of glycemic index on muscle glycogenolysis and exercise performance. *Journal of Applied Physiology*, 81, 1115-20.

Finlayson, G., Bryant, E., Blundell, J. E., & King, N. A. (2009). Acute compensatory eating following exercise is associated with implicit hedonic wanting for food. *Physiology & Behavior*, 97, 62-7.

Finucane, M. M., Stevens, G. A., Cowan, M. J., Danaei, G., Lin, J. K., Paciorek, C. J., Singh, G. M., et al. (2011). National, regional, and global trends in body-mass index

since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9·1 million participants. *Lancet*, 377, 557-67.

Flatt, J. P. (1987). Dietary fat, carbohydrate balance, and weight maintenance: effects of exercise. *The American Journal of Clinical Nutrition*, 45, 296-306.

Flint, A., Raben, A., Blundell, J. E., & Astrup, A. (2000). Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *International Journal of Obesity and Related Metabolic Disorders: Journal of the International Association for the Study of Obesity*, 24, 38-48.

Franz, M. J., VanWormer, J. J., Crain, A. L., Boucher, J. L., Histon, T., Caplan, W., Bowman, J. D., et al. (2007). Weight-loss outcomes: a systematic review and meta-analysis of weight-loss clinical trials with a minimum 1-year follow-up. *Journal of the American Dietetic Association*, 107, 1755-67.

Frayn, K. N. (1983). Calculation of substrate oxidation rates in vivo from gaseous exchange. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology*, 55, 628-34.

Gatta, B., Zuberbuehler, C., Arnold, M., Aubert, R., Langhans, W., & Chapelot, D. (2009). Acute effects of pharmacological modifications of fatty acid metabolism on human satiety. *The British Journal of Nutrition*, 101, 1867-77.

Hargreaves, M., Costill, D. L., Katz, A., & Fink, W. J. (1985). Effect of fructose ingestion on muscle glycogen usage during exercise. *Medicine and Science in Sports and Exercise*, 17, 360-3.

Henderson, G. C., Fattor, J. A., Horning, M. A., Faghihnia, N., Johnson, M. L., Mau, T. L., Luke-Zeitoun, M., et al. (2007). Lipolysis and fatty acid metabolism in men and women during the postexercise recovery period. *The Journal of Physiology*, 584, 963-81.

Hopkins, M., Jeukendrup, A., King, N. A., & Blundell, J. E. (2011). The relationship between substrate metabolism, exercise and appetite control: does glycogen availability influence the motivation to eat, energy intake or food choice? *Sports Medicine*, 41, 507-21.

Hubert, P., King, N. A., & Blundell, J. E. (1998). Uncoupling the effects of energy expenditure and energy intake: appetite response to short-term energy deficit induced by meal omission and physical activity. *Appetite*, 31, 9-19.

Imbeault, P., Saint-Pierre, S., Almeras, N., & Tremblay, A. (1997). Acute effects of exercise on energy intake and feeding behaviour. *The British Journal of Nutrition*, 77, 511-21.

Jones, A. M., & Doust, J. (1996). Communications to the annual conference of the British association of sport and exercise sciences (BASES). *Journal of Sports Sciences*, 14, 3-104.

King, N. A., & Blundell, J. E. (1995). High-fat foods overcome the energy expenditure induced by high-intensity cycling or running. *European Journal of Clinical Nutrition*, 49, 114-23.

- King, N. A., Burley, V. J., & Blundell, J. E. (1994). Exercise-induced suppression of appetite: effects on food intake and implications for energy balance. *European Journal of Clinical Nutrition*, 48, 715-24.
- King, N. A., Caudwell, P., Hopkins, M., Byrne, N. M., Colley, R., Hills, A. P., Stubbs, J. R., et al. (2007). Metabolic and behavioral compensatory responses to exercise interventions: barriers to weight loss. *Obesity*, 15, 1373-83.
- King, N. A., Caudwell, P., Hopkins, M., Stubbs, J. R., Naslund, E., & Blundell, J. E. (2009). Dual-process action of exercise on appetite control: increase in orexigenic drive but improvement in meal-induced satiety. *The American Journal of Clinical Nutrition*, 90, 921-7.
- King, N. A., Hopkins, M., Caudwell, P., Stubbs, R. J., & Blundell, J. E. (2008). Individual variability following 12 weeks of supervised exercise: identification and characterization of compensation for exercise-induced weight loss. *International Journal of Obesity*, 32, 177-84.
- King, N. A., Horner, K., Hills, A. P., Byrne, N. M., Wood, R. E., Bryant, E., Caudwell, P., et al. (2011b). Exercise, appetite and weight management: understanding the compensatory responses in eating behaviour and how they contribute to variability in exercise-induced weight loss. *British Journal of Sports Medicine*, Epub ahead of print. DOI: 10.1136/bjsm.2010.082495.
- King, J. A., Wasse, L. K., Ewens, J., Crystallis, K., Emmanuel, J., Batterham, R. L., & Stensel, D. J. (2011a). Differential acylated ghrelin, peptide YY3-36, appetite, and food

intake responses to equivalent energy deficits created by exercise and food restriction.

*The Journal of Clinical Endocrinology and Metabolism*, 96, 1114-21.

Kissileff, H. R., Pi-Sunyer, F. X., Segal, K., Meltzer, S., & Foelsch, P. A. (1990). Acute effects of exercise on food intake in obese and nonobese women. *The American Journal of Clinical Nutrition*, 52, 240-5.

Klausen, B., Toubro, S., Ranneries, C., Rehfeld, J. F., Holst, J. J., Christensen, N. J., & Astrup, A. (1999). Increased intensity of a single exercise bout stimulates subsequent fat intake. *International Journal of Obesity and Related Metabolic Disorders: Journal of the International Association for the Study of Obesity*, 23, 1282-7.

Martins, C., Morgan, L. M., Bloom, S. R., & Robertson, M. D. (2007). Effects of exercise on gut peptides, energy intake and appetite. *The Journal of Endocrinology*, 193, 251-8.

Martins, C., Morgan, L., & Truby, H. (2008). A review of the effects of exercise on appetite regulation: an obesity perspective. *International Journal of Obesity*, 32, 1337-47.

Maughan, R. J., Fallah, J., & Coyle, E. F. (2010). The effects of fasting on metabolism and performance. *British Journal of Sports Medicine*, 44, 490-4.

Mifflin, M. D., St Jeor, S. T., Hill, L. A., Scott, B. J., Daugherty, S. A., & Koh, Y. O. (1990). A new predictive equation for resting energy expenditure in healthy individuals. *The American Journal of Clinical Nutrition*, 51, 241-7.

National Institute of Health (1998). Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults--The Evidence Report. National Institutes of Health. *Obesity Research*, 6, 51S-209S.

Paoli, A., Marcolin, G., Zonin, F., Neri, M., Sivieri, A., & Pacelli, Q. F. (2011). Exercising fasting or fed to enhance fat loss? Influence of food intake on respiratory ratio and excess postexercise oxygen consumption after a bout of endurance training. *International Journal of Sport Nutrition and Exercise Metabolism*, 21, 48-54.

Poehlman, E. T. (1989). A review: exercise and its influence on resting energy metabolism in man. *Medicine and Science in Sports and Exercise*, 21, 515-25.

Scharrer, E., & Langhans, W. (1986). Control of food intake by fatty acid oxidation. *The American Journal of Physiology*, 250, R1003-6.

Siri, W. E. (1956). The gross composition of the body. *Advances in Biological and Medical Physics*, 4, 239-80.

Tremblay, A., & Therrien, F. (2006). Physical activity and body functionality: implications for obesity prevention and treatment. *Canadian Journal of Physiology and Pharmacology*, 84, 149-56.

Tschöp, M., Smiley, D. L., & Heiman, M. L. (2000). Ghrelin induces adiposity in rodents. *Nature*, 407, 908-13.

Ueda, S. Y., Yoshikawa, T., Katsura, Y., Usui, T., & Fujimoto, S. (2009). Comparable effects of moderate intensity exercise on changes in anorectic gut hormone levels and energy intake to high intensity exercise. *The Journal of Endocrinology*, 203, 357-64.

Votruba, S. B., Atkinson, R. L., Hirvonen, M. D., & Schoeller, D. A. (2002). Prior exercise increases subsequent utilization of dietary fat. *Medicine and Science in Sports and Exercise*, 34, 1757-65.

**Table 1** - Baseline appetite, resting energy expenditure and resting RER values in the control (CON), fasted (FAST) and postprandial (FED) trials. Values are mean  $\pm$  SD (n = 12). PFC = prospective food consumption, RER = respiratory exchange ratio.

		CON		FAST		FED		<i>P</i>
Hunger	0-100	63	$\pm$ 16	62	$\pm$ 18	61	$\pm$ 18	0.891
Satisfaction	0-100	27	$\pm$ 13	24	$\pm$ 17	25	$\pm$ 16	0.897
Fullness	0-100	23	$\pm$ 15	16	$\pm$ 11	16	$\pm$ 8	0.168
PFC	0-100	73	$\pm$ 10	67	$\pm$ 16	71	$\pm$ 14	0.235
Resting energy expenditure	kJ.min <sup>-1</sup>	4.85	$\pm$ 0.88	5.06	$\pm$ 0.63	4.77	$\pm$ 0.96	0.488
	(kcal.min <sup>-1</sup> )	(1.16	$\pm$ 0.21)	(1.21	$\pm$ 0.15)	(1.14	$\pm$ 0.23)	
Resting RER		0.83	$\pm$ 0.08	0.83	$\pm$ 0.06	0.82	$\pm$ 0.05	0.723

**Table 2** – Ad libitum energy intake during the control (CON), fasted (FAST) and postprandial (FED) trials. Values are mean  $\pm$  SD (n = 12), kJ and (kcal)

		CON			FAST			FED		
Buffet 1 (5.5 – 6 h)	kJ	7205	$\pm$	1707	7540	$\pm$	1515	6820	$\pm$	2238
	(kcal)	(1722	$\pm$	408)	(1802	$\pm$	362)	(1630	$\pm$	535)
Buffet 2 (9.5 – 10 h)	kJ	4448	$\pm$	1703	4548	$\pm$	1774	4732	$\pm$	2013
	(kcal)	(1063	$\pm$	407)	(1087	$\pm$	424)	(1131	$\pm$	481)
Overnight (10 – 24 h)	kJ	1803	$\pm$	908	1565	$\pm$	1197	1377	$\pm$	1000
	(kcal)	(431	$\pm$	217)	(374	$\pm$	286)	(329	$\pm$	239)
Total trial (0 – 24 h)	kJ	13,452	$\pm$	2682	13,652	$\pm$	2385	12,929	$\pm$	2933
	(kcal)	(3215	$\pm$	641)	(3263	$\pm$	570)	(3090	$\pm$	701)

No significant differences between trials.

**Table 3** – Macronutrient intake in the control (upper panel), fasted (middle panel) and postprandial (lower panel) trials. Values are mean  $\pm$  SD (n = 12).

		Fat	Carbohydrate	Protein
Control				
Buffet 1	g	61.1 $\pm$ 25.0	240.2 $\pm$ 57.1	52.8 $\pm$ 17.4
	(%)	(31.9)	(55.8)	(12.3)
Buffet 2	g	40.2 $\pm$ 17.0	142.7 $\pm$ 57.6	32.5 $\pm$ 16.3
	(%)	(34.1)	(53.7)	(12.2)
Overnight	g	14.8 $\pm$ 10.1	62.2 $\pm$ 30.1	12.2 $\pm$ 9.4
	(%)	(30.8)	(57.8)	(11.4)
Total	g	116.1 $\pm$ 36.9	445.1 $\pm$ 84.0	97.5 $\pm$ 27.3
	(%)	(32.5)	(55.4)	(12.1)
Fasted				
Buffet 1	g	65.8 $\pm$ 24.4	245.3 $\pm$ 58.1	57.1 $\pm$ 12.4
	(%)	(32.9)	(54.4)	(12.7)
Buffet 2	g	37.7 $\pm$ 18.0	155.8 $\pm$ 59.9	31.1 $\pm$ 16.6
	(%)	(31.2)	(57.3)	(11.5)
Overnight	g	15.8 $\pm$ 14.6	47.7 $\pm$ 36.1	10.4 $\pm$ 9.0
	(%)	(38.0)	(50.9)	(11.1)
Total	g	119.3 $\pm$ 33.0	448.7 $\pm$ 94.5	98.6 $\pm$ 21.5
	(%)	(32.9)	(55.0)	(12.1)
Postprandial				
Buffet 1	g	53.5 $\pm$ 28.5	236.9 $\pm$ 74.4	50.2 $\pm$ 14.6
	(%)	(29.6)	(58.1)	(12.3)
Buffet 2	g	44.4 $\pm$ 23.3	147.8 $\pm$ 61.9	35.2 $\pm$ 20.9
	(%)	(35.3)	(52.3)	(12.4)
Overnight	g	12.2 $\pm$ 12.3	48.3 $\pm$ 35.1	6.6 $\pm$ 4.7
	(%)	(33.4)	(58.7)	(7.9)
Total	g	110.1 $\pm$ 42.6	433.0 $\pm$ 86.7	92.0 $\pm$ 25.5
	(%)	(32.1)	(56.0)	(11.9)

No significant differences between trials.

**Figure 1.** Schematic representation of the study protocol.

**Figure 2.** Perceptions of hunger (a), satisfaction (b), prospective food consumption (c) and fullness (d) in CON (▼), FAST (●) and FED (○) trials. Values are mean ± SEM (n = 12). Dashed line indicates CON, solid line indicates FAST, dotted line indicates FED. Horizontally shaded rectangle indicates fasted exercise, vertically shaded rectangle indicates postprandial exercise, hatched shaded rectangle indicates standardised breakfast meal, black rectangles indicate ad libitum buffet meals. <sup>a</sup>Higher in FED than FAST, <sup>b</sup>Higher in FAST than FED (both P < 0.0005).

**Figure 3.** Energy expenditure in CON (▼), FAST (●) and FED (○) trials. Values are mean ± SEM (n = 12). Dashed line indicates CON, solid line indicates FAST, dotted line indicates FED. Horizontally shaded rectangle indicates fasted exercise, vertically shaded rectangle indicates postprandial exercise, hatched shaded rectangle indicates standardised breakfast meal, black rectangles indicate ad libitum buffet meals. <sup>a</sup>Higher in FED than FAST, <sup>b</sup>Higher in FAST than FED, <sup>c</sup>Higher in FED than CON, <sup>d</sup>Higher in FAST than CON (all P < 0.0005).

**Figure 4.** Respiratory exchange ratio values in CON (▼), FAST (●) and FED (○) trials. Values are mean ± SEM (n = 12). Dashed line indicates CON, solid line indicates FAST, dotted line indicates FED. Horizontally shaded rectangle indicates fasted exercise, vertically shaded rectangle indicates postprandial exercise, hatched shaded rectangle indicates standardised breakfast meal, black rectangles indicate ad libitum buffet meals. <sup>a</sup>Higher in FED than FAST, <sup>b</sup>Higher in FAST than FED, <sup>c</sup>Higher in FED than CON, <sup>d</sup>Higher in FAST than CON (all P < 0.0005).

Figure 1

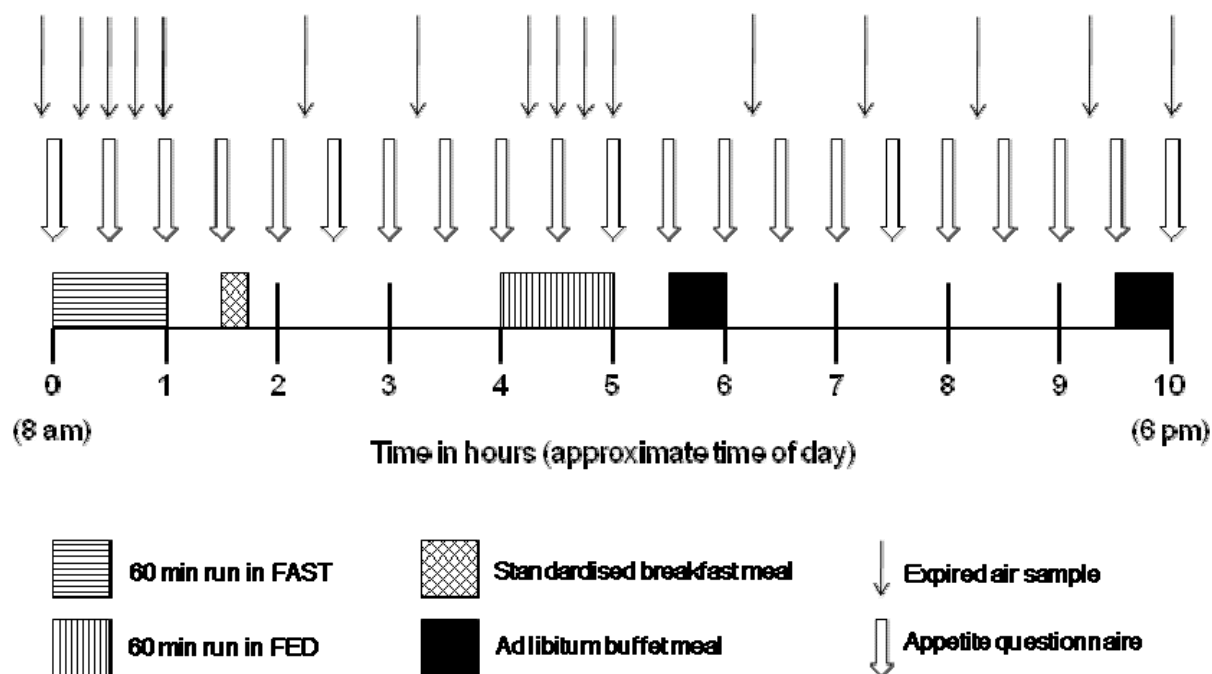
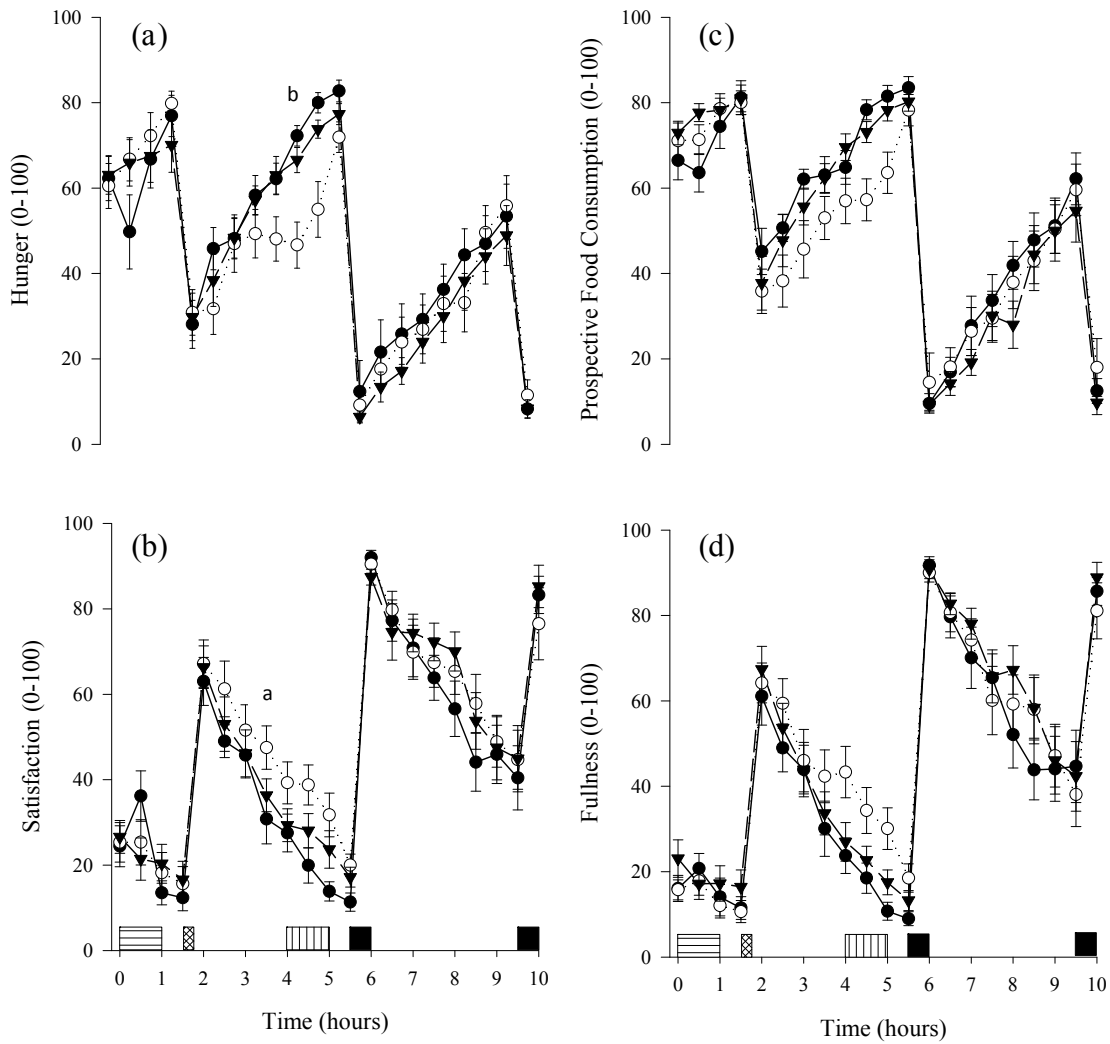
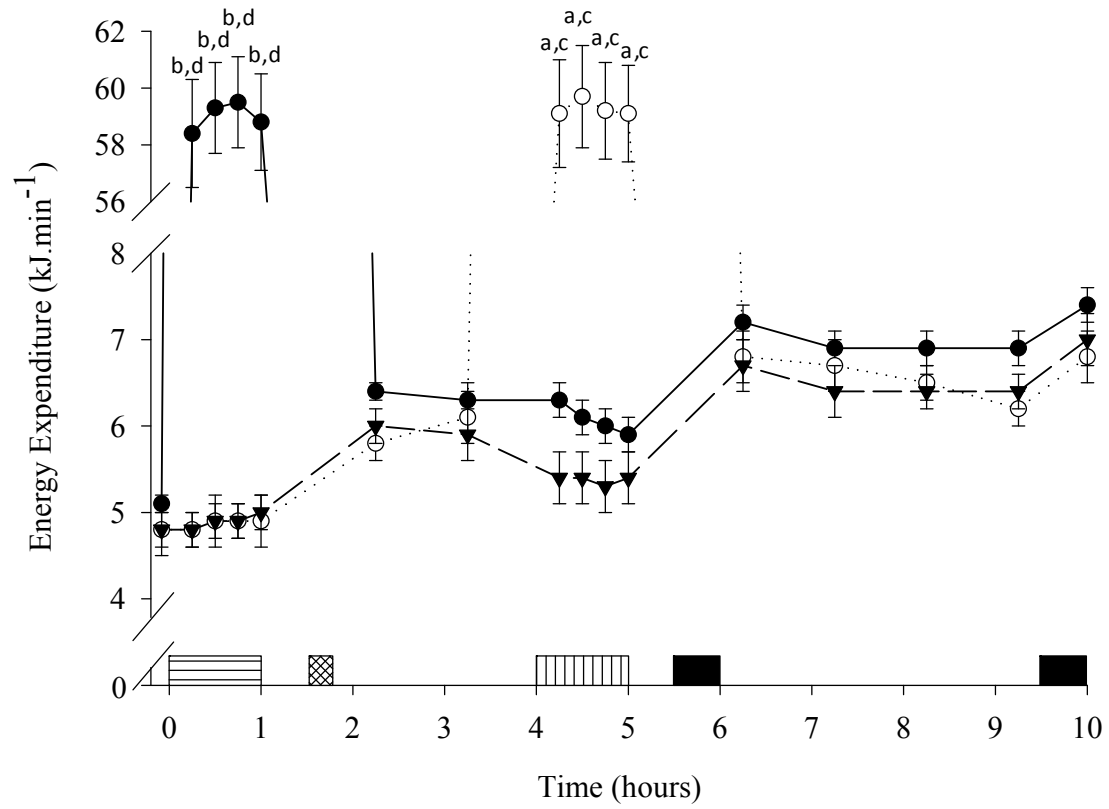


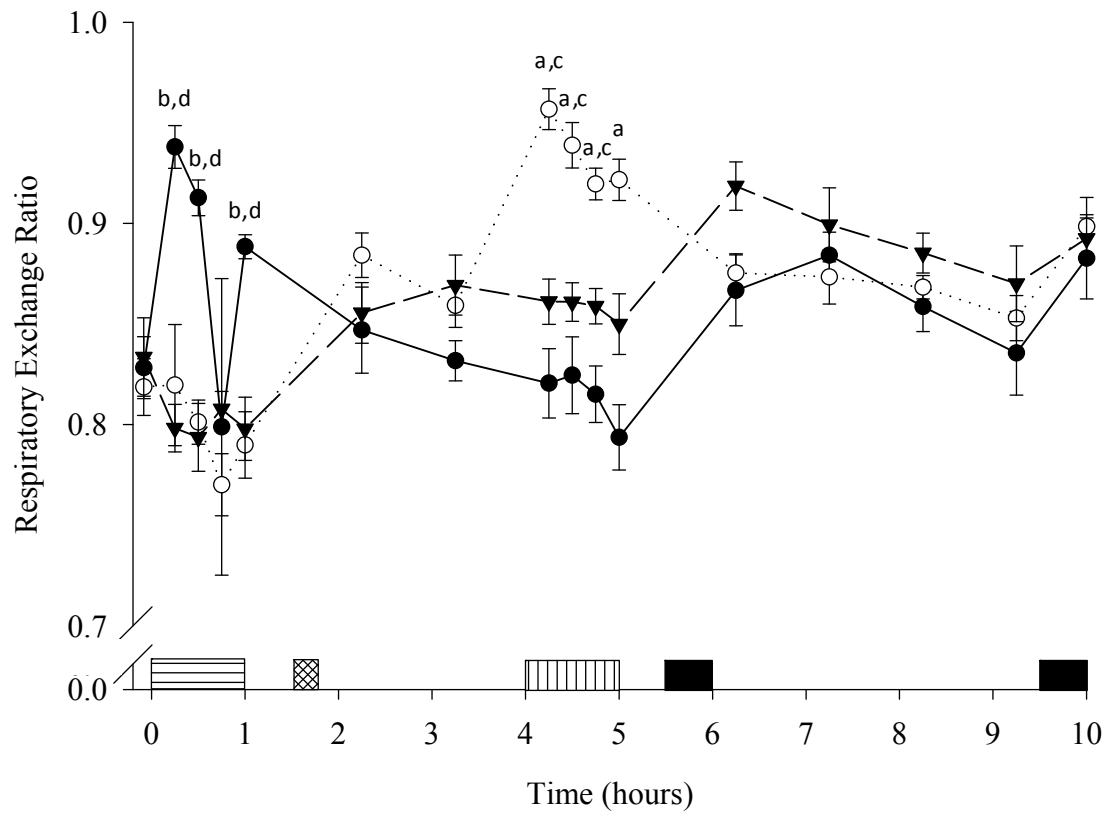
Figure 2



**Figure 3**



**Figure 4**



## **Appendix A. Items presented at buffet meals**

Frosties – Cereal

Cornflakes – Cereal

Coco Pops – Cereal

Milk

Orange Juice

White Bread

Brown Bread

Cheese

Ham

Tuna

Banana

Apple

Orange

Salted Crisps

Butter

Margarine

Mayonnaise

Nutri-Grain Bar

Mars Bar

Cookies

Chocolate Muffins

Plain Muffins

Chocolate Mini-Rolls