

Medicine & Science IN Sports & Exercise

The Official Journal of the American College of Sports Medicine

www.acsm-msse.org

. . . Published ahead of Print

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Accepted for Publication: 26 April 2015

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Running title: Breakfast omission and exercise performance

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This research was supported by the National Institute for Health Research (NIHR) Diet, Lifestyle & Physical Activity Biomedical Research Unit based at University Hospitals of Leicester and Loughborough University. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health. The authors would like to declare no conflicts of interest. The results of the present study do not constitute endorsement by the American College of Sports Medicine.

Abstract

Introduction: Breakfast omission may reduce daily energy intake. Exercising fasted impairs performance compared to exercising after breakfast, but the effect breakfast omission has on evening exercise performance is unknown. This study assessed the impact of omitting breakfast on evening exercise performance, as well as within-day energy intake. **Methods:** Ten male, habitual breakfast eaters completed two trials, in randomised, counterbalanced order. Subjects arrived at the laboratory overnight fasted, and either consumed or omitted a 733 ± 46 kcal (3095 ± 195 kJ) breakfast. *Ad-libitum* energy intake was assessed at 4.5 h (lunch) and 11 h (dinner). At 9 h subjects completed 30 min cycling exercise at $\sim 60\%$ VO_2peak , followed by a 30 min maximal cycling performance test. Food was not permitted for subjects once they left the laboratory after dinner until 08:00 the following morning. Acylated ghrelin, GLP-1₍₇₋₃₆₎, glucose and insulin were assessed at 0, 4.5 and 9 h. Subjective appetite sensations were recorded throughout. **Results:** Energy intake was 199 ± 151 kcal greater at lunch ($P < 0.01$) after breakfast omission compared to breakfast consumption and tended to be greater at dinner after consuming breakfast ($P = 0.052$). Consequently, total *ad-libitum* energy intake was similar between trials ($P = 0.196$), with 24 h energy intake $19 \pm 5\%$ greater after consuming breakfast ($P < 0.001$). Total work completed during the exercise performance test was 4.5% greater after breakfast (314 ± 53 kJ vs. 300 ± 56 kJ; $P < 0.05$). Insulin was greater during BC at 4.5 h ($P < 0.05$), with no other interaction effect for hormone concentrations. **Conclusions:** Breakfast omission might be an effective means of reducing daily energy intake, but may impair performance later that day, even after consuming lunch.

Key words: Appetite, Energy restriction, Energy balance, Meal omission, Ghrelin, GLP-1

Introduction

Maintenance of a stable body weight is achieved through careful management of energy balance, with weight gain occurring due to a chronic surplus of energy intake above energy expenditure. Refraining from eating at a prescribed meal time will inevitably create an energy deficit, and breakfast omission is a frequently cited method of reducing energy intake (40). Regular breakfast consumption has been recommended as part of a “healthy balanced diet” (24) and individuals who regularly consume breakfast tend to have a lower BMI (3) and reduced prevalence of several chronic diseases including type-2 diabetes (26).

Traditionally, recommendations for regular breakfast consumption have been based on correlational studies that associate a lower BMI with regular breakfast consumption (3). However, these findings do not infer causality as individuals who regularly consume breakfast have often been shown to exhibit healthy lifestyle factors, such as increased physical activity (6) and improved dietary profiles (14). Therefore it is difficult to elucidate whether improved weight control is mediated by breakfast consumption *per-se*.

Acute intervention studies have generally found that the omission of breakfast induces increased feelings of hunger over the morning, leading to greater energy intake in the first meal following breakfast omission (19,22). However, energy intake over the course of the day rarely results in complete compensation for the energy omitted at breakfast, consequently reducing daily energy intake (2,19,22,25,30). Although this is not a universal finding as Astbury *et al.* (1) found that energy omitted at breakfast was fully compensated for at an *ad-libitum* lunch meal, and Farshchi *et al.* (11) found energy intake to be greater following breakfast omission compared to breakfast consumption. Whilst investigating a similar topic, one of these studies utilised a liquid pre-load

between breakfast and lunch to determine the hormonal response to breakfast omission (1) and the other balanced energy intake by providing cereal and milk at either 07:00 or 12:00, representing breakfast consumption and omission, respectively (11). These differences in design may explain the contradictory findings in these studies.

Lifestyle interventions that combine both dietary restriction and exercise have been shown to be more effective for long term sustainable weight loss and maintenance (12). Therefore it is important to consider the effect that a given dietary intervention has on physical activity and the ability to perform exercise, as this will influence the magnitude of energy deficit that can be achieved. Recently it was reported that daily energy intake was reduced by approximately 2250 kJ during a 6 week period of breakfast omission, however this deficit was offset by concomitant decreases in habitual energy expenditure of approximately 1850 kJ (2). The inclusion of structured exercise during periods of energy restriction may have the potential to somewhat offset this decline in habitual energy expenditure, if exercise performance and/or adherence is not affected as a result of breakfast omission.

A working lifestyle may restrict time for exercise to early mornings or evenings. Evening exercise classes have been associated with increased alertness, enthusiasm and reduced effort than morning classes (23), suggesting that evening exercise may be the more acceptable option and may improve long-term adherence to an exercise program. Furthermore, some athletes have been reported to compete or train without the consumption of breakfast (34) and it is important to consider what the effects of breakfast omission are for individuals aiming to achieve peak exercise performance. Whilst it is well established that exercise performance is compromised in the fasted compared to post-prandial state (32,33), no studies have attempted to determine whether exercise performed later in the day is affected by the prior omission of breakfast.

Therefore the aim of this investigation was to examine the impact of breakfast omission/consumption on subsequent energy intake and evening exercise performance 4 h after provision of an *ad-libitum* lunch. We hypothesised that total 24 h energy intake (including breakfast) would be reduced by breakfast omission and that exercise performance would not be different between trials

Methods

Subjects

After ethical approval, subjects completed a medical screening questionnaire and provided written informed consent. Subjects were 10 healthy, weight stable (self-reported), recreationally active (<10 h·week⁻¹) males (age: 22 ± 3 y, weight: 73.1 ± 9.7 kg, height: 1.76 ± 0.05 m, BMI: 23.5 ± 3.2 kg·m⁻², Body fat: 17 ± 6 %). Subjects regularly consumed breakfast and were not restrained, disinhibited or hungry eaters determined after completion of a three-factor eating questionnaire (35).

Preliminary trials

Subjects completed three preliminary trials. During the first trial; height (to nearest 0.1 cm), and weight (to nearest 0.02 kg) were measured, and body fat percentage was estimated using skin-fold callipers (10). A discontinuous incremental exercise test was also performed on an electrically braked cycle ergometer (Lode Corival, Groningen, Holland) to determine peak oxygen consumption (VO_{2peak}). Increments lasted for 4 min, were separated by ~5 min rest and

increased until volitional exhaustion. Expired air was collected into a Douglas bag during the last min of each increment. Heart rate was measured (Polar Beat, Kempele, Finland) and subjects rated their perceived exertion (RPE) on a 6-20 point scale, at the end of each increment. Expired air samples were analysed for oxygen and carbon dioxide concentration (Servomex, Crowborough, UK), volume (Harvard Dry Gas Meter, Harvard Ltd, Edenbridge, UK) and temperature (Edale, Cambridge, UK).

During the second preliminary trial, subjects were fully familiarised with the experimental protocol (described in detail below), with the exception that subjects were permitted to come and go from the laboratory between feeding periods and the exercise protocol. On the third preliminary trial, subjects completed the exercise protocol for a second time.

Pre-trial standardisation

In the 48 h preceding the first experimental trial, subjects completed a weighed food diary, replicating this in the 48 h preceding the second trial. Strenuous exercise and alcohol intake were not permitted during this period. Subjects travelled to and from the laboratory via motorised transport, arriving in the morning following an overnight fast of ≥ 10 h.

Protocol

Subjects completed two experimental trials; breakfast consumption (BC) and breakfast omission (BO). Trials were separated by at least 7 days, conducted at the same time of day, on the same day of the week and were administered in a randomised, counterbalanced order. Subjects were

aware that the aims of the study were to assess the effect of breakfast omission on appetite, energy intake and exercise performance, but were not aware of the hypothesis.

Subjects arrived at the laboratory at ~07:30, were weighed and a fasted blood sample was collected by venepuncture of an antecubital vein, after a 30 min period of supine rest (0 h). Baseline measures of subjective appetite sensations on a visual analogue scale were obtained before participants received either a standardised breakfast (BC) or no breakfast (BO). After breakfast (0.5 h) subjects rested quietly in the laboratory. A second blood sample was drawn at 12:30 (4.5 h), following which a multi-item *ad-libitum* lunch buffet was served consisting of cold, ready-to-eat foods. Upon termination of the meal, subjects again rested in the laboratory. At 17:00 (9 h) a blood sample was drawn before subjects began the exercise protocol (described below). One hour after completion of the performance test (11 h), an *ad-libitum* pasta test meal was served. Following the test meal (11.5 h), subjects were transported home and were instructed not to eat or drink anything other than plain water until they went to bed. Subjects returned to the laboratory after an overnight fast the following morning at 08:00 (24 h) for body mass measurement and to complete a subjective appetite sensations questionnaire. *Ad-libitum* water and low-energy squash was available on request throughout the study period, and was provided with each buffet meal.

Ad-libitum meals

Each *ad-libitum* meal was provided in excess of expected consumption and more food was available on request. The lunch meal consisted of cooked meats, cheese, bread, butter, mayonnaise, salad, fruit, crisps, cereal bars and biscuits (Tesco, Cheshut, UK). The dinner meal

consisted of pasta, cheese, tomato sauce and olive oil (Tesco, Cheshut, UK), was homogenous in nature providing $8.01 \pm 0.04 \text{ kJ}\cdot\text{g}^{-1}$ (14, 33 and 53% of energy provided by protein, fat and carbohydrate, respectively), and was served as previously described (5). Meals were served in an isolated feeding laboratory with no interaction between subjects and investigators. Subjects were given 30 min to consume each meal and were explicitly instructed to eat until they felt 'comfortably full and satisfied'. The amount consumed at each meal was quantified by weighing the food before and after consumption, with macronutrient content of foods ascertained from manufacturer values.

Exercise performance

Subjects began exercise at 17:00 (9 h) and initially performed 30 min steady state cycling at a workload of $\sim 60\% \text{ VO}_{2\text{peak}}$. After 30 min, subjects completed a performance test, during which they were instructed to complete as much work as possible in 30 min. The workload was set at $75\% \text{ VO}_{2\text{peak}}$ and subjects were able to manipulate the workload by pressing up or down on the bikes control unit. The control unit was completely covered, so that subjects received no feedback related to the workload completed and subjects were not provided any encouragement, although they were able to see the time remaining. During the steady state exercise, expired air was collected between 14-15 and 29-30 min, and heart rate and RPE was obtained at the end of each collection. During the performance test, workload and heart rate were recorded every 5 min and RPE every 10 min. Energy expenditure and substrate utilisation were calculated from VO_2 and VCO_2 values using stoichiometric equations (13).

Standardised breakfast meal

During BC subjects were provided with a standardised breakfast meal of 25% estimated daily energy requirements, determined by multiplying resting metabolic rate (RMR) (27) by a physical activity level of 1.7, to account for the exercise component of the trial. Breakfast consisted of crisped rice cereal, semi-skimmed milk, wholemeal bread, margarine, strawberry jam and orange juice (Tesco, Cheshunt, UK), and amounted to 3095 ± 195 kJ, with 11, 17 and 72 % of energy derived from protein, fat and carbohydrate, respectively. During BO, subjects were provided with a bolus of water for breakfast equal to that contained within the BC trial. Subjects were instructed to consume the entire meal gradually over the 30 min period.

Subjective Appetite Sensations

Subjects rated their hunger, fullness, desire to eat (DTE) and prospective food consumption (PFC) on 100 mm visual analogue scales at 0, 0.5, 2.5, 4.5, 5, 7, 9, 10, 11, 11.5, 13, and 24 h. Verbal anchors of ‘not at all/ none at all’ and ‘extremely/ no desire at all/ a lot’ were placed at 0 and 100 mm, respectively.

Blood sampling and analysis

Blood samples (12 mL) were drawn after 30 min of supine rest, at 0 h (baseline), 4.5 h (pre-lunch) and 9 h (pre-exercise) via venepuncture of an antecubital vein. Five mL of blood was immediately mixed with 50 μ l Dipeptidyl-peptidase 4 inhibitor (DPP4-010, Merck Millipore, Watford, UK) and dispensed into an EDTA tube ($1.75 \text{ mg}\cdot\text{mL}^{-1}$), for determination of active

glucagon like peptide 1 (GLP-1₇₋₃₆) by ELISA (EGLP-35K, Merck Millipore, Watford, UK). Two and a half mL of blood was dispensed into an EDTA tube (1.75 mg·mL⁻¹) containing 10 µL·mL⁻¹ blood of a solution of potassium phosphate buffer (PBS) (0.05 M), *P*-hydroxymercuribenzoic acid (PHMB) (0.05 M) and sodium hydroxide solution (NaOH) (0.006 M) for determination of acylated ghrelin concentration by ELISA (A05106, Bioquote Ltd, York, UK). Two and a half mL of blood was dispensed into an EDTA tube (1.75 mg·mL⁻¹) for measurement of blood glucose concentration (GOD-PAP method, Randox Laboratories Ltd, Crumlin, UK) and insulin concentration by ELISA (DX-EIA-2935, Immunodiagnostic Systems, Boldon, UK) .

All samples were centrifuged at 1750g for a total of 15 min in a refrigerated centrifuge (4°C). After 10 min of centrifugation, the supernatant (1 mL) of the PHMB/PBS/NaOH treated blood was combined with 1 M HCl (100 µL) before all samples were centrifuged for a further 5 min. The supernatant of each sample was then removed and stored at -20°C until frozen and then transferred to -80°C for later analysis.

A separate 2 mL of blood was collected into an EDTA tube and used for the determination of haemoglobin (via the cyanmethaemoglobin method) and haematocrit (via micro-centrifugation) and used to estimate changes in plasma volume relative to baseline (9).

Statistical analysis

Data was analysed using SPSS 21.0 (SPSS Inc., Somers, NY, USA). Area under the curve (AUC) values were calculated using the trapezoidal method and were averaged over time. Correction of plasma measures for changes in plasma volume did not alter the results so the

unadjusted values are presented. All data were checked for normality of distribution using a Shapiro-Wilk test. Data containing one factor were analysed using a t-test or Wilcoxon signed-rank test, as appropriate. Data containing two variables were analysed using a two-way ANOVA, and where appropriate followed by Bonferroni-adjusted paired t-tests or Bonferroni-adjusted Wilcoxon signed-ranks, as appropriate. Data sets were determined to be significantly different when $P < 0.05$. Data was found to be normally distributed, with the exception of all subjective appetite sensations, acylated ghrelin and GLP-1₍₇₋₃₆₎ and were subject to non-parametric statistical analysis. However, data has been presented as means \pm standard deviation for consistency throughout, unless stated otherwise.

Results

Energy and macronutrient intake

A breakfast of 3095 ± 195 kJ was provided during BC. Subsequent total *ad-libitum* energy intake was 11685 ± 1893 kJ compared to 11329 ± 2117 kJ, for BO and BC, respectively ($P=0.196$). At lunch, energy intake was greater during BO (5804 ± 1817 kJ) than BC (4970 ± 1987 kJ; $P < 0.01$), whereas at dinner, there was a tendency for greater energy intake during BC (6359 ± 1631 kJ) than BO (5882 ± 1443 kJ; $P=0.052$). Including breakfast, total energy intake was $19 \pm 5\%$ greater during BC (14424 ± 2255 kJ) than BO (11685 ± 1893 kJ) (Fig. 1).

Carbohydrate ($P < 0.05$) and fat ($P < 0.05$) intake was greater at lunch during BO compared to BC, but there was no difference in protein ($P=0.142$) or fibre ($P=0.314$) intake. The dinner meal was homogenous in nature; therefore macronutrient selection could not be gauged from this meal.

Including breakfast, total carbohydrate, protein and fibre intake were greater ($P<0.01$) and fat intake tended to be greater ($P=0.068$) during BC compared to BO (Table 1).

Subjective appetite sensations

All appetite sensations (hunger, fullness, DTE and PFC) showed a main effect of trial ($P<0.05$), time ($P<0.001$) and an interaction effect ($P<0.001$; Fig. 2). Subjects reported increased hunger, DTE and PFC, as well as lower fullness, in the post-breakfast period (0.5-4.5 h) during BO compared to BC ($P<0.01$). Subjects also reported increased fullness at 7 h during BO compared to BC ($P<0.05$). For AUC analysis, data was divided into 3 sections; breakfast to lunch (0-4.5 h), lunch to dinner (5-11 h) and post dinner (11.5-24 h). These analyses revealed differences between trials for all subjective appetite variables between breakfast and lunch ($P<0.01$). Fullness was also increased between lunch and dinner during BO compared to BC ($P<0.05$; Table 2).

Steady state exercise and performance test

Total work completed during the performance test was greater during BC (314 ± 53 kJ) than BO (300 ± 56 kJ; $P<0.05$; Fig. 3). There was no effect of trial order on exercise performance ($P=0.297$). During the 30 min steady state period, energy expenditure was greater during BO (1407 ± 210 kJ) than BC (1330 ± 191 kJ; $P<0.05$). Fat oxidation was also greater during BO compared to BC ($P<0.05$), but there was no difference in carbohydrate oxidation between trials ($P=0.126$). Average heart rate was higher during BO (155 ± 9 bpm) than BC (151 ± 8 bpm;

$P < 0.001$) during steady state, but was not different during the performance test ($P = 0.397$). There were no differences in RPE either during the 30 min steady state preload ($P = 0.464$) or the performance test ($P = 0.712$).

Blood parameters

Plasma glucose ($P < 0.05$), insulin ($P < 0.001$), acylated ghrelin ($P < 0.001$) and GLP-1₍₇₋₃₆₎ ($P < 0.05$) all showed a main effect of time. There were no main effects of trial or interaction effects for plasma glucose ($P \geq 0.201$), acylated ghrelin ($P \geq 0.189$) or GLP-1₍₇₋₃₆₎ ($P \geq 0.056$). There was an interaction effect for insulin ($P < 0.01$), with higher insulin concentrations at 4.5 h during BC than BO ($P < 0.01$), while insulin concentrations tended to be higher at 9 h during BO compared to BC ($P = 0.073$; Table 3).

Discussion

The primary aim of this investigation was to determine the effect of breakfast omission/consumption on subsequent energy intake and evening exercise performance. It was found that total work completed over a 30 min cycling performance test was reduced by approximately 4.5% following breakfast omission. Whilst energy intake was increased at lunch, this study also observed no difference in total *ad-libitum* energy intake between trials, resulting in a reduced total 24 h energy intake after breakfast omission. From a weight management perspective, occasional breakfast omission could be used as a viable means of energy restriction in habitual breakfast consumers, although this may slightly impair exercise performance. Further study is

required to determine whether breakfast omission can be used chronically to assist with long term weight management.

The global increase in the prevalence of obesity has coincided with a gradual decline in breakfast consumption (15), with epidemiological evidence suggesting that those who regularly omit breakfast have a higher BMI than those who regularly consume breakfast (3). However, due to a number of confounding factors, including variations in activity patterns (6) and dietary profiles (14), there is a lack of causal data linking breakfast eating behaviour with energy balance. The results of the current investigation demonstrate that the total energy restricted at breakfast is not accurately compensated for over an acute 24 h period, resulting in a net energy deficit of 2738 kJ. These findings are comparable with those of Levitsky and Pacanowski (22), who found total energy intake was reduced by approximately 1883 kJ following the omission of an *ad-libitum* breakfast meal. Similarly, 7 days consecutive breakfast omission was found to reduce energy intake by $670 \text{ kJ}\cdot\text{d}^{-1}$ on average compared to 7-days consecutive breakfast consumption (30). Taken collectively, data from these acute investigations suggest that, contrary to popular belief, breakfast omission does not lead to elevated energy intake over the course of the day, and as such there is potential for breakfast omission to be used in successful weight management strategies.

Consistent with previous findings, energy intake at lunch was greater during BO than BC (1,19,22,30). Following the omission of breakfast, subjective appetite sensations were elevated throughout the morning compared to when breakfast was consumed (Fig. 2), and accordingly energy intake at lunch was increased by approximately 16%. However, this modest increase in energy intake ($745 \pm 604 \text{ kJ}$) only partially compensated for the energy deficit created by the omission of the breakfast meal ($3095 \pm 195 \text{ kJ}$), and as such subjects remained in energy deficit

throughout the afternoon. Similar to the findings in the current study, Levitsky and Pacanowski (22) reported elevations in hunger following the omission of an *ab-libitum* breakfast meal, leading to increased energy consumption at lunch. Hubert *et al.* (19) found that reducing breakfast energy intake by 1824 kJ resulted in an average elevation in energy intake at lunch of 500 kJ. The average compensation at lunch for breakfast omission is remarkably consistent between these studies, with the current investigation revealing 24% compensation at lunch, compared to 22% (22) and 26% (19) previously reported.

Concentrations of the orexigenic hormone acylated ghrelin and the anorexigenic hormone GLP-1₍₇₋₃₆₎ are thought to respond to fluxes in energy balance (8,17), and stimulate a behavioural response. In the current study, the increase in appetite observed throughout the morning period may have caused an increase in energy consumption during the time between breakfast and lunch in free-living conditions, as was found previously (25). Acylated ghrelin and GLP-1₍₇₋₃₆₎ were only measured 4 h after breakfast consumption/omission and immediately prior to exercise so the dynamic response of these hormones to feeding may have been missed. Following lunch, no differences were observed in subjective appetite sensations, which may suggest no difference in gut hormone concentrations. Accordingly, the appetitive responses to breakfast omission appear to be transient, and do not influence energy intake following the provision of lunch.

Whilst there is general agreement in the literature that breakfast omission reduces daily energy intake, two investigations contest these findings. Astbury *et al.* (1) found that the provision of a 1080 kJ breakfast was completely compensated for in the no breakfast condition at an *ad-libitum* lunch meal. This study was designed primarily to investigate the effect of breakfast on gastrointestinal hormonal regulation of food intake and incorporated a liquid pre-load between breakfast and lunch that may have influenced energy intake at lunch. Additionally, the provision

of a low energy breakfast (10% of daily energy requirements) has previously been shown to be more accurately compensated for at subsequent meals than higher energy breakfasts (31). Farshchi *et al.* (11) aimed to investigate whether the timing of breakfast consumption affected subsequent energy intake. Over a 2 week period, subjects either consumed cereal and milk at a traditional breakfast time (7-8am) or later in the day (12-12:30pm), which ensured that the energy provided was consistent across both interventions. Energy intake was found to be greater following breakfast omission compared to breakfast consumption. This was likely due to the experimental design, which does not necessarily represent typical practise for those utilising breakfast omission as a method of weight management.

It is well documented that consuming breakfast improves exercise performance in the morning compared omitting breakfast, i.e. exercising fasted (32,33). The current study found that exercise performance was also compromised in the evening following breakfast omission in the morning, despite consuming lunch 4.5 h before exercise. Eating breakfast is highly encouraged in the literature to maximise carbohydrate stores prior to competition (38), as glucose availability may be a limiting factor due to glycogen depletion (7). In particular, liver glycogen stores, which are important for blood glucose maintenance during exercise, have been shown to decrease by ~40% following an overnight fast (36). Provision of a high carbohydrate breakfast will help replenish liver glycogen (16), and has been shown to increase muscle glycogen concentrations in the vastus lateralis by 11-17% (4,37). A recent study reported that 73% of female college athletes regularly omitted breakfast, resulting in suboptimal daily carbohydrate and energy intakes (34). This was also shown in the present study, as carbohydrate intake prior to exercise was reduced during BO compared to BC (148 ± 65 vs. 259 ± 73 g), which may have influenced glucose availability and reduced exercise performance. It appears breakfast may play a central role in

meeting daily carbohydrate requirements for exercising individuals and that consumption of breakfast might be important in order to maximise exercise performance throughout the whole day.

Fat oxidation was greater during the 30 min steady state exercise period in BO. Increasing fat oxidation has been suggested to be beneficial for reducing fat mass and may also promote carbohydrate sparing, potentially improving performance (20). However, there was no difference in carbohydrate oxidation between trials therefore it is unlikely that glycogen sparing occurred during BO. Accordingly, energy expenditure was greater during BO, which may be attributable to an increase in dietary induced thermogenesis induced by greater energy intake at the previous *ad-libitum* lunch meal. An increased contribution of dietary induced thermogenesis to energy expenditure may also explain the higher heart rate observed during BO. Following food intake, the splanchnic tissues require an increase in blood supply to assist with the digestion and absorption of nutrients. Therefore, during sub-maximal exercise, an increase in cardiac output is required to meet the oxygen requirements of both the skeletal muscle and splanchnic tissues (39). Another indicator of sympathetic nervous activity is noradrenaline, which has been shown to peak after breakfast, with an attenuated response at subsequent feeding periods (29). Following the omission of breakfast, lunch becomes the first meal of the day. It could be considered that the sympathetic nervous response to feeding was greater following lunch during BO compared to BC, thus heart rate was increased to a greater extent during steady state exercise. Noradrenaline also increases lipolysis (21) and may explain the elevation in fat oxidation during the steady state exercise on BO.

A limitation with any research that investigates breakfast omission is the difficulty in blinding subjects to the study intervention. In the multifactorial 'central governor theory' model of fatigue described by Noakes (28), subject awareness of the study intervention may lead to an expectation

in regard to exercise performance, and performance may decline as a result. This may be particularly pertinent with the current study as all subjects were habitual breakfast consumers, so the withdrawal of breakfast in the morning may have produced a particularly strong expectation of reduced performance. This may partially account for the findings in this study.

It has recently been shown that the omission of breakfast over a 6 week period has a negative effect on physical activity levels, reducing habitual physical activity thermogenesis on average by $1850 \text{ kJ}\cdot\text{d}^{-1}$ compared to when breakfast was consumed (2). Physical activity of this nature is difficult to manipulate or avoid as the nutritional intervention seemingly imposes a subconscious restriction on energy expenditure. Incorporating structured exercise into weight management programs may offset the magnitude of this deficit somewhat, provided adherence to exercise isn't affected. Whilst exercise performance might be important to maximise energy expenditure, the difference in exercise performance observed in the current study had a negligible influence on energy balance. Energy expenditure during the 30 min preload was ~ 80 kJ greater during BO, which was offset by an estimated reduction of energy expenditure of ~ 70 kJ during BO, assuming a cycling efficiency of 20% (18). Therefore net energy expenditure during exercise was almost identical between trials (2898 ± 307 (BC) vs. 2905 ± 307 (BO) kJ; $P=0.834$).

In conclusion, the results of the present study demonstrate that occasionally omitting breakfast may be an effective method of reducing energy intake over a 24 h period in habitual breakfast consumers. However, exercise performance may be compromised throughout the whole day following the omission of breakfast in the morning. Therefore, for those concerned with maximising training and/or competition performance breakfast omission might impair performance or interfere with training adaptation.

Acknowledgements

This research was supported by the National Institute for Health Research (NIHR) Diet, Lifestyle & Physical Activity Biomedical Research Unit based at University Hospitals of Leicester and Loughborough University. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health.

Conflict of interests

The authors would like to declare no conflicts of interest. The results of the present study do not constitute endorsement by the American College of Sports Medicine.

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Figure legends

Figure 1. Energy intake (kJ) at each *ad-libitum* meal and over 24 h during BC (■) and BO (□). Left panel displays mean values with vertical error bars representing standard deviation. Right panel shows individual subjects energy intake response at each *ad-libitum* meal. † indicates values are different to BC ($P<0.05$).

Figure 2. Subjective sensations of hunger (A), fullness (B), desire to eat (DTE) (C) and prospective food consumption (PFC) (D) during BC (■) and BO (○). Data points are means with vertical error bars representing standard error of the mean. White rectangle indicates standard meal feeding, vertical hatched rectangles indicate an *ad-libitum* meal, and black rectangle indicates exercise period. All appetite variables showed a main effect of time. † indicates values are significantly different between trials ($P<0.05$).

Figure 3. Work completed (kJ) during the exercise performance test. Left panel displays mean work completed during BC (■) and BO (□) with vertical error bars representing standard deviation. Right panel displays individual subject's performance during BC (■) and BO (○). † indicates values are significantly different to BC ($P<0.05$).

Figure 1

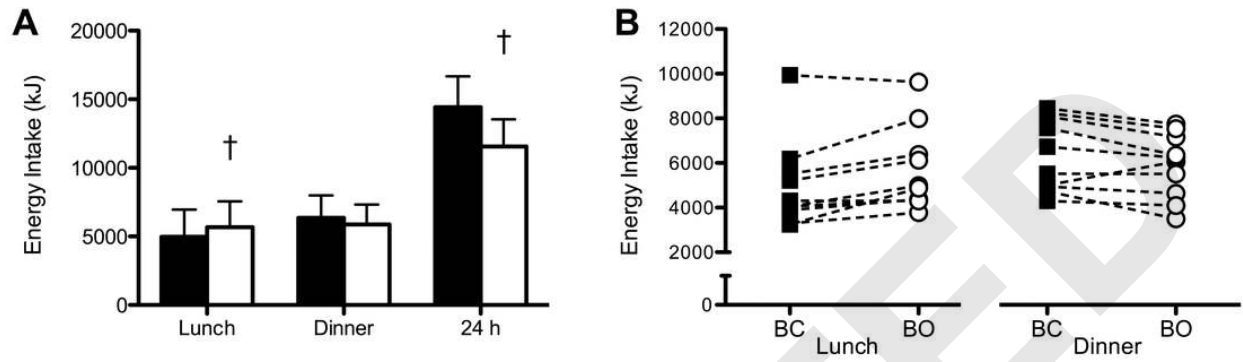


Figure 2

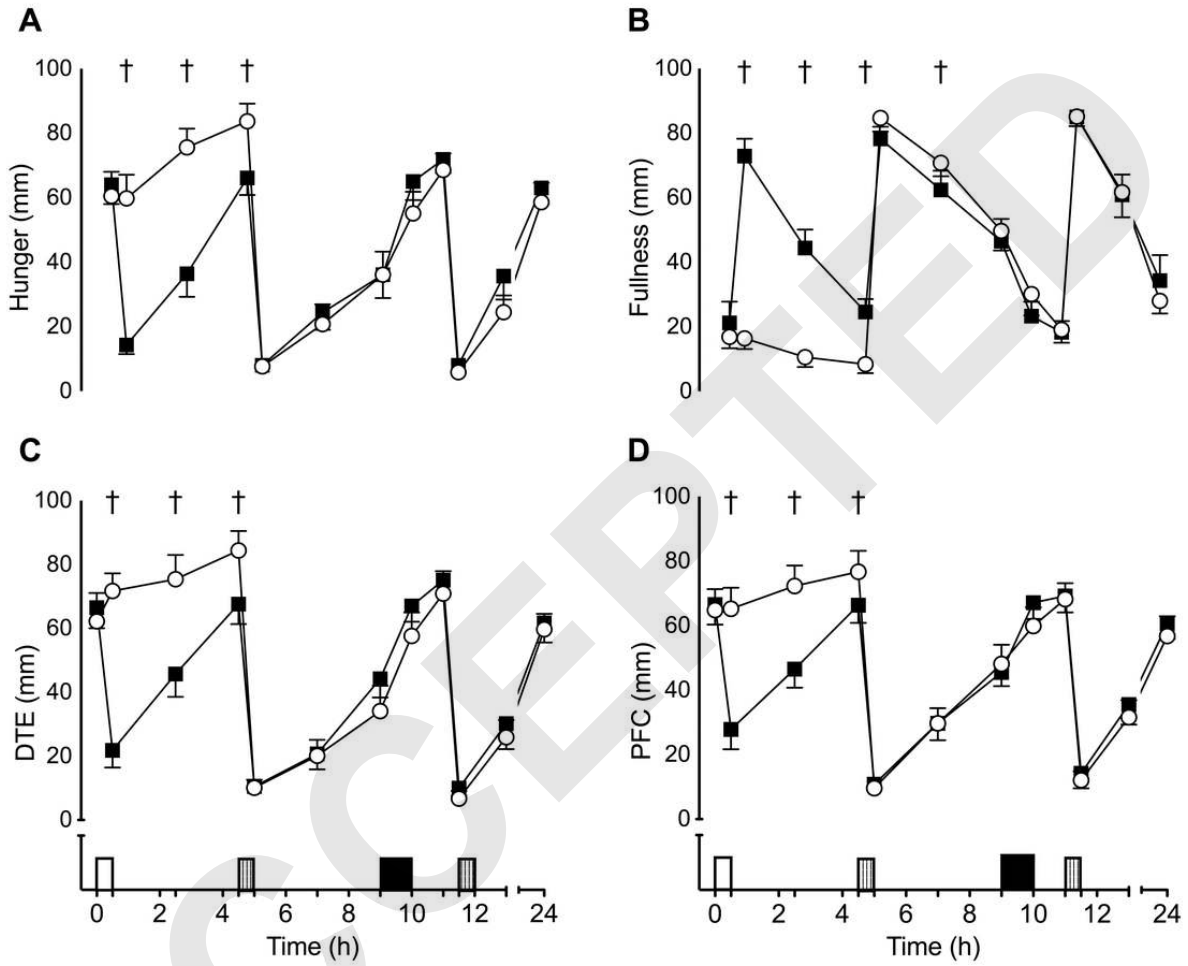


Figure 3

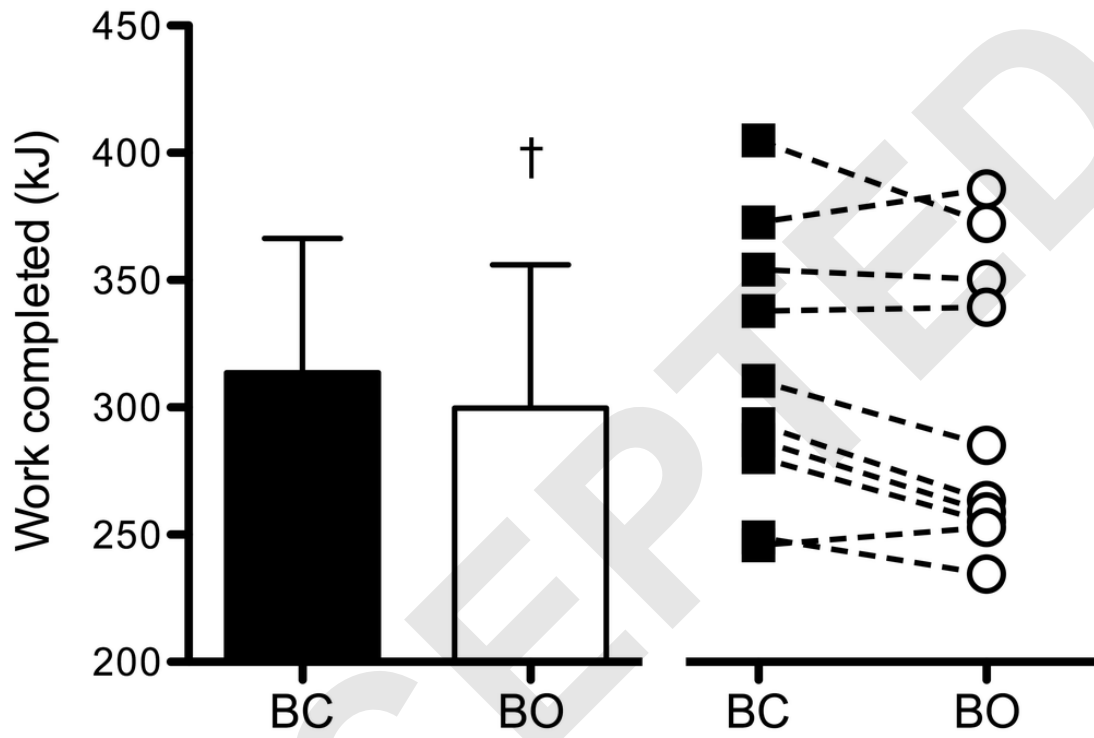


Table 1. Carbohydrate (CHO), protein (PRO), fat, fibre and water intake over the course of the each trial.

| | Energy (kJ) | CHO (g) | PRO (g) | FAT (g) | FIBRE (g) | WATER (ml) |
|------------------|--------------------|----------------|----------------|----------------|------------------|-------------------|
| Breakfast | | | | | | |
| BC | 3095 ± 195 | 130.3 ± 8.2 | 19.5 ± 1.2 | 13.9 ± 0.9 | 4.5 ± 0.3 | 625 ± 39 |
| BO | 0 ± 0 | 0 † | 0 † | 0 † | 0 † | 625 ± 39 |
| Lunch | | | | | | |
| BC | 4970 ± 1987 | 128.5 ± 69.0 | 44.3 ± 22.8 | 52.7 ± 20.2 | 10.2 ± 4.5 | 814 ± 211 |
| BO | 5804 ± 1878 † | 148.1 ± 65.1 † | 50.2 ± 22.2 | 63.3 ± 23.9 † | 11.1 ± 4.2 | 894 ± 207 |
| Dinner | | | | | | |
| BC | 6359 ± 1631 | 194.2 ± 49.8 | 53.6 ± 13.7 | 55.9 ± 14.3 | 9.7 ± 2.5 | 477 ± 121 |
| BO | 5882 ± 1443 | 179.6 ± 44.1 | 49.5 ± 12.2 | 51.7 ± 12.7 | 9.0 ± 2.2 | 443 ± 108 |
| Total | | | | | | |
| BC | 14424 ± 2255 | 453.0 ± 80.9 | 117.4 ± 24.9 | 122.5 ± 19.7 | 24.4 ± 5.5 | 3395 ± 627 |
| BO | 11685 ± 1893 † | 327.8 ± 78.3 † | 99.7 ± 25.0 † | 115.1 ± 17.6 | 20.1 ± 5.5 † | 3335 ± 489 |

Data are means ± standard deviations. † indicates values significantly different to BC ($P < 0.05$).

Please note that the dinner meal was homogenous in nature, therefore macronutrient intake is proportional to volume consumed.

Table 2. Time averaged area under the curve for each appetite variable.

| | Post breakfast (0-4 h) | Post lunch (5-10.5 h) | Post dinner (11-24 h) |
|-----------|--|-----------------------|-----------------------|
| | Hunger ($\text{mm}\cdot\text{h}^{-1}$) | | |
| BC | 38 ± 15 | 39 ± 13 | 44 ± 16 |
| BO | 72 ± 18 [†] | 35 ± 16 | 37 ± 14 |
| | Fullness ($\text{mm}\cdot\text{h}^{-1}$) | | |
| BC | 47 ± 13 | 56 ± 13 | 49 ± 17 |
| BO | 12 ± 9 [†] | 62 ± 12 [†] | 46 ± 15 |
| | DTE ($\text{mm}\cdot\text{h}^{-1}$) | | |
| BC | 45 ± 18 | 41 ± 13 | 41 ± 15 |
| BO | 76 ± 21 [†] | 35 ± 16 | 38 ± 11 |
| | PFC ($\text{mm}\cdot\text{h}^{-1}$) | | |
| BC | 47 ± 16 | 44 ± 12 | 44 ± 13 |
| BO | 71 ± 20 [†] | 43 ± 13 | 40 ± 15 |

Data are means ± standard deviations. [†] values are significantly different to BC ($P < 0.05$).

Table 3. Plasma concentrations of glucose, insulin, acylated ghrelin and GLP-1₍₇₋₃₆₎ over the course of the trial during BC and BO.

| | Pre-breakfast (0 h) | Pre-lunch (4.5 h) | Pre-exercise (9 h) |
|-----------|---|--------------------------|---------------------------|
| | Glucose (mmol·L⁻¹) | | |
| BC | 5.33 ± 0.18 | 4.89 ± 0.42 * | 5.27 ± 0.39 |
| BO | 5.18 ± 0.25 | 4.91 ± 0.33 * | 5.13 ± 0.67 |
| | Insulin (μU·mL⁻¹) | | |
| BC | 15.0 ± 4.4 | 16.1 ± 5.8 | 24.2 ± 6.8 * |
| BO | 13.9 ± 3.5 | 10.7 ± 4.1 †* | 30.7 ± 11.5 * |
| | Acylated Ghrelin (pg·mL⁻¹) | | |
| BC | 108 ± 114 | 115 ± 65 | 92 ± 90 |
| BO | 97 ± 99 | 118 ± 121 * | 71 ± 94 * |
| | GLP-1₍₇₋₃₆₎ (pmol/L⁻¹) | | |
| BC | 7.22 ± 6.06 | 9.85 ± 9.30 | 8.51 ± 7.29 |
| BO | 6.61 ± 6.41 | 6.55 ± 6.82 | 12.99 ± 12.26 * |

Normal and non-normally distributed data are presented as means ± standard deviations for consistency. † indicates values are significantly different to BC; * indicates values are significantly different compared to baseline ($P < 0.05$).