Breakfast Consumption Affects Appetite, Energy Intake, and the Metabolic and Endocrine Responses to Foods Consumed Later in the Day in Male Habitual Breakfast Eaters1–3

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Abstract

The effects of breakfast consumption on energy intake and the responses to foods consumed later in the day remain unclear. Twelve men of healthy body weight who reported regularly consuming breakfast (mean ± SD age 23.4 ± 7.3 y; BMI 23.5 ± 1.7 kg/m2) completed 2 trials using a randomized crossover design. Participants were provided with a 1050-kJ liquid preload 150 min after consuming a standardized breakfast (B) (10% daily energy requirement and 14, 14, and 72% energy from protein, fat, and carbohydrate, respectively), or no breakfast (NB). Blood glucose and serum insulin responses to the preload (area under the curve) were higher in the NB condition (P < 0.05). Plasma FFA responses to the preload were higher in the NB condition (P < 0.01). Plasma glucagon-like peptide 1 (P < 0.01) and plasma peptide Y (P < 0.05) responses were higher after the preload in the B condition. Desire to eat, fullness, and hunger ratings collected immediately prior to consuming the preload were all different from the fasting values in the NB condition (P < 0.05). Thus, immediately prior to consuming the preload, the fullness rating was lower and hunger and desire to eat ratings were higher in the NB condition (P < 0.05). Energy intake at the lunchtime test meal was ~17% lower in the B condition (P < 0.01). In conclusion, missing breakfast causes metabolic and hormonal differences in the responses to foods consumed later in the morning as well as differences in subjective appetite and a compensatory increase in energy intake. J. Nutr. doi: 10.3945/jn.110.128645.

Introduction

The increasing global prevalence of obesity is recognized as a major public health problem due to the increased risk of developing multiple chronic diseases (1–3). Regularly eating breakfast is recommended as one of the strategies that may help individuals achieve and maintain a healthy body weight (4). Despite this advice, the proportion of people who report regularly consuming breakfast is declining (5,6), which has coincided with the dramatic increase in overweight and obesity.

Numerous epidemiological studies report that regular breakfast eaters have a lower BMI (7–14), and regular breakfast consumption is demonstrated by those who are successful at maintaining their weight loss (15). However, it is currently unclear whether the link between breakfast consumption and body weight is mediated through differences in total energy intake or is a result of other lifestyle factors associated with breakfast consumption (16).

Some studies have reported no difference in daily energy intake between those who regularly consume breakfast compared with those who do not (17,18), whereas others reported that the daily energy intake of regular breakfast eaters is greater (11,12,19,20) despite their having a lower BMI (11,20). Clearly, it is possible that these associations may be influenced by under-reporting of energy intake by overweight participants (21) or by reverse causality, with the overweight omitting breakfast in an attempt to reduce energy intake (16).

Nevertheless, it has been proposed that omitting breakfast can disrupt the distribution of daily energy intake, resulting in increased consumption of foods later in the day (22,23), including more snacking between meals (23). We previously examined the effect of omitting breakfast on appetite and energy intake in regular breakfast consumers (24). Ten women of healthy body weight ate a standard breakfast or omitted breakfast for 14 d in a randomized crossover design. Daily energy intake was lower during the breakfast eating period, although there were no differences in subjective appetite responses to a standard test meal. However, the metabolic and subjective appetite responses to foods consumed following breakfast were not investigated in that study.

The purpose of this study was to compare the effects of consuming breakfast with omitting breakfast on energy intake at
a lunchtime test meal, subjective appetite responses, and the metabolic and endocrine responses to foods consumed later in the day in men who regularly eat breakfast.

Participants and Methods

Participants

All participants were recruited from the staff and students of the Queen’s Medical Centre, Nottingham and the University of Nottingham via poster advertisement (Table 1).

The sample size required to detect a difference of 500 kJ in intake at the lunchtime test meal between conditions with a 2-tailed α of 0.05 and β of 0.8 was n = 12.

Inclusion criteria were: 1) healthy weight (BMI = 19–25 kg/m²); 2) male; 3) self-reported regular breakfast eater (consuming ready-to-eat cereal or continental style breakfast on ≥ 5 of 7 days); 4) 18–45 y; and 5) no history of serious disease or currently taking any medications. Current dieters, those with a recent weight loss or gain (± 3 kg in the past 6 mo), restrained eaters (defined by a score ≥ 7 for restraint on the Three Factor eating Questionnaire [25]), and those with symptoms of clinical depression (defined by a score ≥ 10 on the Beck Depression Inventory [26]) were excluded from the study.

All participants were recruited and studied at the School of Biomedical Sciences, University of Nottingham UK between January and March 2007. Ethical approval for the study was granted by the University of Nottingham Medical School Ethics Committee and all participants provided informed consent to take part before commencing the study.

Study protocol

All participants (n = 12) completed 2 experimental visits to the laboratory in a randomized crossover design; visits were scheduled at least 7 d apart. Participants were advised to continue their usual food and activity pattern between the study visits but were asked to refrain from vigorous physical activities and consuming alcohol and acetaminophen-containing products for a 24-h period prior to each laboratory visit. A menu of foods was provided for participants to consume as their evening meal the day prior to each experimental trial. On the study day, participants reported to the laboratory at 0745 h. On arrival, a cannula was inserted retrograde into a dorsal hand vein under local anesthetic for the collection of arterialized venous blood samples. A fasting blood sample was collected and baseline ratings of appetite obtained using paper-based visual analogue scales (VAS). Participants were then either served a standardized breakfast (B) consumed over 20 min or were supplied with no breakfast (NB) (continued to fast), after which they rested for 150 min. After 150 min, a further blood sample was collected and participants completed subjective appetite ratings before receiving a standard liquid preload, which they consumed within 15 min. Then further appetite ratings were completed immediately and at 30, 60, and 90 min later and blood samples collected at 15-min intervals. At 90 min, participants were provided with a pasta-based test meal with the instructions to eat as much as they wished until they felt comfortably full. The participants voluntarily terminated the test meal and appetite ratings were collected immediately and at 30, 60, 90, and 120 min later and blood sampling resumed at 15-min intervals. After 120 min, participants were free to leave the laboratory with no further restrictions. Participants consumed water ad libitum during each visit.

Screening

Weight and height were measured using standard methods as previously reported (24). A fasting blood sample was collected for clinical chemistry and hematology tests. Participants were supplied with a diary in which they were asked to record food intake and activity pattern over a 3-d period (2 weekdays and 1 weekend day) prior to the experimental visits.

Test meals

Evening meal. The evening meal the day before each trial was designed to be equivalent to ~30% daily energy requirement (DER) with 16, 35, and 48% energy from protein, fat, and carbohydrate, respectively [equivalent to UK average intakes; National Diet and Nutrition Survey (27)]. This meal was eaten at ~2000 h the evening prior to each experimental visit and no other foods or drinks (other than water) were consumed until participants arrived at the laboratory the next morning.

Breakfast. The breakfast supplied (B condition only) consisted of Rice Krispies (Kelloggs) and semiskimmed milk (1.7% fat; cereal:milk ratio was ~30 g:125 mL) equivalent to 10% of the participant’s DER. Each breakfast contained 14, 14, and 72% energy from protein, fat, and carbohydrate, respectively.

Preload. Preloads were 400 mL 1.050-kJ vanilla-flavored drinks providing 20, 39, and 41% preload energy from protein, carbohydrate, and fat, respectively. Preloads were prepared using instantized whey protein isolate (Bi-Pro, Davisco), maltodextrin (Cerestar) and double cream and flavored with 2 mL of artificial vanilla flavoring (Supercook). Preloads were made up to 400 mL with cold water, and 1500 mg of acetaminophen (Numark) was dissolved into each preload to provide an index of the rate of gastric emptying (28). The appearance of acetaminophen in the blood correlates well with gastric emptying as assessed by scintigraphy (29). Preloads were given in covered opaque containers and consumed through a straw to minimize visual and olfactory stimuli, which could influence appetite.

Lunchtime test meal. The ad libitum test meal provided at lunch consisted of pasta, cheddar cheese, olive oil, tomato, and basil sauce (Mars UK). Cooked, cooled pasta was mixed with the other ingredients and refrigerated in covered dishes until being heated in a microwave oven when required. Participants were given portions of ~500 g and instructed to consume as much as they wished until they felt comfortably full. Once the initial portion was consumed, it was removed and another was provided. This was repeated until participants indicated they felt comfortably full and terminated the meal. Any remaining food was removed by the experimenter. Energy and macronutrient intake was determined from the weight of food consumed (the test meal contained ~657 kJ/100 g; 13, 38, and 49 energy% provided by protein, fat, and carbohydrate, respectively).

Subjective appetite ratings. Appetite was assessed using validated paper-based VAS (30). The question asked at each time point was “How (rathing) do you feel,” where the rating was “a strong desire to eat,” “full,” “hungry,” “nauseous,” or “thirsty.” The terms “extremely” or “not at all” were anchored at either end of a 100-mm vertical line printed below each question. Participants placed a small mark intersecting the horizontal line to correspond to their response to each question. All ratings were scored on a scale from 0 to 100 using a steel rule to measure to the closest millimeter where the vertical mark intersected the horizontal line, so that higher scores indicated a greater subjective sensation.

Blood sampling. Participants placed a hand in a hot air-warmed, ventilated perspex box (30–53°C) to open arterio-venous anastomoses and a cannula (Venflon 18G; Becton Dickinson) was inserted retrograde into a dorsal hand vein under local anesthetic for arterialized blood sampling.

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**Table 1** Participant characteristics

<table>
<thead>
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<th>Characteristic</th>
<th>Value</th>
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<tr>
<td>Age, y</td>
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<tr>
<td>Weight, kg</td>
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<td>Body Mass Index, kg/m²</td>
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<td>Energy Intake, MJ/d</td>
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<td>Protein, % energy</td>
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<td>Fat, % energy</td>
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<td>Alcohol, % energy</td>
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<tr>
<td>DER, MJ/d</td>
<td>10.8 ± 1.00</td>
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1 Values are means ± SD, n = 12.
2 Daily energy intake, assessed using the 3-d food diary completed during screening.
3 DER calculated using the 3-d activity diary and Compendium of Physical Activities (59) completed during screening.
4 Abbreviations used: B, breakfast; DER, daily energy requirement; GLP-1, glucagon-like peptide 1; NB, no breakfast; PYY, peptide Y; VAS, visual analogue scale.
Samples were drawn from a 3-way tap; the first 2 mL of each sample was discarded to avoid contamination with the saline used to maintain patency. Blood was dispensed into lithium heparin tubes containing 50 μL EGTA, and EDTA tubes (containing 75 μL of aprotinin, Trasylol, Bayer) for mixing and immediate centrifugation or a serum separating tube and allowed to clot for 30 min at room temperature before centrifugation. Centrifugation was performed for 10 min at 3000 g at 4°C and the plasma/serum was removed and stored at −80°C for later analysis.

**Blood analysis.** Blood glucose was measured immediately (Hemacue). The lithium heparin plasma was analyzed for FFA (ASC-ACOD method, Wako NEFA C; Wako), glucagon-like peptide 1 (GLP-1), and peptide Y (PYY) (32,33) and the EDTA plasma was analyzed for total ghrelin (LINCO Research) by RIA. Serum insulin was measured by RIA (Coat-A-Count Insulin, Euro Diagnostic Products) and acetaminophen by ELISA (Cambridge Life Science).

**Statistical analysis**

SPSS software (version 13; SPSS) was used for data entry and analysis. All data are presented as mean ± SEM unless otherwise stated. A 2-tailed, paired Student’s t test was used to compare energy intakes at the test meals.

VAS and blood/plasma metabolites/hormones were analyzed by separately considering the preload and test meal responses. Preload responses were analyzed using repeated-measures ANOVA on 2 factors (breakfast × time) if significant main effects were obtained; paired t tests were used to determine the location of the difference. Test meal responses were analyzed using ANOVA on 2 factors (breakfast × time); energy intake at the test meal was used as a covariate to control for the individual differences in intake. If significant main effects were obtained, 2-tailed paired t tests were used to determine the location of any differences. Differences were considered significant at P < 0.05.

**Results**

**Baseline and pre-preload values**

Fasting values of glucose, insulin, FFA, GLP-1, PYY, and ghrelin did not differ between conditions. Pre-preload concentrations of glucose, insulin, PYY, and ghrelin did not differ from their corresponding fasting values in either condition and these values were not significantly different between the conditions (Table 2).

Pre-preload plasma FFA and plasma GLP-1 concentrations differed from the corresponding fasting values in the NB trial only (P < 0.05). Therefore, plasma FFA concentrations were higher and plasma GLP-1 concentrations were lower in the NB trial compared with the B trial immediately prior to the consumption of the preload (P < 0.05) (Table 2).

**Blood glucose and serum insulin responses**

Blood glucose and serum insulin responses to the preload both demonstrated main effects of time (P < 0.01) and breakfast × time interaction (P < 0.01) (Fig. 1A,B). Blood glucose and serum insulin concentrations increased rapidly after consuming the preload in both trials, before returning to fasting concentrations. Blood glucose concentrations were higher in the NB condition at 30, 45, and 75 min post-preload (P < 0.05) (Fig. 1A). However, there were no significant differences in blood glucose or serum insulin concentrations immediately prior to the lunchtime test meal between trials. The incremental AUC above pre-preload concentrations for blood glucose was greater in the NB trial (77.8 ± 14.8 mmol/L × 90 min) compared with the B trial (54.5 ± 11.3 mmol/L × 90 min) (P < 0.05) and the serum insulin incremental AUC was higher in the NB trial (1.60 ± 0.216 mmol/L × 90 min) compared with the B trial (1.53 ± 0.238 mmol/L × 90 min) (P < 0.05).

Blood glucose and serum insulin responses to the test meal using energy intake at the test meal as a covariate in the analysis demonstrated main effects of time only (P < 0.01). Blood glucose and serum insulin concentrations increased in both conditions following the test meal. After the peak post-lunch values, blood glucose concentrations declined in both conditions before rising slightly to a plateau (~5.9 mmol/L) in both trials for the remainder of the protocol. Serum insulin concentrations gradually declined following the post-lunch peak and continued to decline in both conditions for the remainder of the protocol.

**FFA.** FFA responses to the preload demonstrated a main effect of time (P < 0.01) and a breakfast × time interaction (P < 0.01) (Fig. 1C). FFA concentrations declined after consuming the preload in both trials. Concentrations were higher in the NB condition 15 and 30 min post-preload (P < 0.05). After the nadir, plasma FFA concentrations began to increase in both trials, with FFA concentrations higher in the B condition at 75 min post-preload (P < 0.05) and immediately prior to the lunchtime test meal (P < 0.05).

There were main effects of time (P < 0.05) and breakfast (P < 0.05) for the FFA responses to the test meal when energy intake at the meal was used as a covariant in the analysis. Following the test meal, FFA concentrations continued to rise in both trials, gradually increasing until sampling ceased.

**GLP-1.** Plasma GLP-1 responses to the preload demonstrated a breakfast × time interaction (P < 0.05) (Fig. 2A). Following the consumption of the preload, plasma GLP-1 concentrations increased in both trials before returning to pre-preload concentrations. Plasma GLP-1 concentrations were higher in the B trial at 15 min post-preload (P < 0.05); however, plasma GLP-1 concentrations did not significantly differ immediately prior to the lunchtime test meal.

### Table 2

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<td></td>
<td>BL</td>
<td>PL</td>
<td>NB</td>
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<tr>
<td>Blood glucose, mmol/L</td>
<td>4.8 ± 0.1</td>
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<td>Serum insulin, pmol/L</td>
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<td>Plasma FFA, mmol/L</td>
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<td>0.5 ± 0.06</td>
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<td>Plasma acetaminophen, μmol/L</td>
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<td>1.7 ± 0.2</td>
<td>1.7 ± 0.3</td>
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<td>Plasma GLP-1, pmol/L</td>
<td>14.3 ± 0.3</td>
<td>15.8 ± 4.1</td>
<td>14.7 ± 2.5</td>
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<td>Plasma PYY, pmol/L</td>
<td>17.8 ± 1.2</td>
<td>16.4 ± 3.0</td>
<td>17.5 ± 2.6</td>
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<td>Plasma ghrelin, pmol/L</td>
<td>879 ± 49.2</td>
<td>927 ± 50.8</td>
<td>912 ± 69.5</td>
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</table>

*Values are means ± SEM, n = 12. *Different from BL, P < 0.05; *different from corresponding B, P < 0.05.

1. BL, baseline (fasting) sample; PL, sample collected immediately prior to the preload, 150 min after breakfast in the B trial, and its corresponding time point in the NB trial.
There was a breakfast × time interaction \( (P < 0.01) \) for plasma GLP-1 responses to the test meal when energy intake at the test meal was used as a covariant in the analysis. Following consumption of the lunchtime test meal, GLP-1 concentrations increased rapidly in both trials; the post-lunch peak plasma GLP-1 concentrations were higher in the NB trial (49.4 ± 7.2 pmol/L) compared with the B trial (35.2 ± 2.5 pmol/L) \( (P < 0.05) \), after which GLP-1 concentrations declined to similar concentrations in both trials.

There was a breakfast × time interaction for plasma PYY responses to the preload \( (P < 0.05) \) (Fig. 2B). Following the consumption of the preload, plasma PYY concentrations increased in the B trial before gradually returning to pre-preload concentrations and plasma PYY concentrations in the NB remained at a plateau of ~15 pmol/L. Post-preload peak concentrations were higher in the B trial (24.6 ± 2.8 pmol/L) compared with the NB trial (22.5 ± 2.0 pmol/L) \( (P < 0.05) \). Plasma PYY concentrations were higher in the B trial at 15 and 30 min post-preload \( (P < 0.05) \).
however, plasma PYY concentrations did not significantly differ between trials immediately prior to the lunchtime test meal.

There was a main effect of time only ($P < 0.01$) for plasma PYY responses to the test meal when energy intake at the test meal was used as a covariant in the analysis. Following the test meal, plasma PYY concentrations increased rapidly in both trials. Following post-lunch peak values, concentrations remained elevated at ~30 pmol/L in both trials for the remainder of the protocol.

**Ghrelin.** The plasma ghrelin concentration was measured only in the samples collected prior to the ad libitum lunchtime test meal (Fig. 2C).

The plasma ghrelin response to the preload demonstrated a main effect of time only ($P < 0.001$). Following the consumption of the preload, plasma ghrelin concentrations declined in both trials before gradually increasing again in both trials. The plasma ghrelin concentrations immediately prior to the lunchtime test meal did not differ between trials. The minimum plasma ghrelin

**FIGURE 2** Plasma GLP-1 (A), PYY (B), and ghrelin (C) responses to a liquid preload and an ad libitum lunchtime test meal ($A, B$ only) by healthy men in the B and NB trials. The preload was consumed 150 min after the breakfast and the lunchtime test meal was consumed ad libitum 90 min after the preload. Values are means ± SEM, $n = 12$. *Different from B, $P < 0.05$. BL represents the baseline (fasting) sample, PL represents the samples collected immediately prior to the preload, 150 min after breakfast in the B trial, and its corresponding time point in the NB trial.
concentrations reached were lower in the B trial (730 ± 41.9 pmol/L) than in the NB trial (660 ± 51.9 pmol/L) (P < 0.05).

**Gastric emptying.** Serum acetaminophen responses to the preload displayed a main effect of time only (P < 0.01). Following the preload, serum acetaminophen concentrations increased rapidly in both trials with no significant difference between the trials for the maximum concentration reached or the time to reach the maximum concentration. However, the rate of increase tended to be greater in the B trial (P = 0.1) (data not shown).

**Energy intake.** Energy intake at the ad libitum test meal was lower in the B trial (4.90 ± 0.455 MJ) compared with the NB trial (5.76 ± 0.403 MJ) (P < 0.01).

The calculated combined energy intake (breakfast + preload + ad libitum test meal) did not differ between the B (6.8 ± 0.455 MJ) and NB (6.83 ± 0.403 MJ) trials.

**Subjective appetite ratings.** The fasting rating of hunger and fullness did not differ between trials. Hunger ratings were lower and fullness ratings were higher in the B trial than in the NB trials immediately pre-preload (P < 0.05). There was a main effect of time for fullness (P < 0.001) and hunger (P < 0.01) ratings in response to the preload (Fig. 3). Hunger ratings were suppressed, whereas fullness ratings increased in both trials following the consumption of the preload. This initial response was followed by a gradual return to pre-preload values in both trials. There was a main effect of breakfast for hunger (P < 0.01) and fullness ratings (P < 0.05) in response to the preload. Hunger ratings were significantly lower and fullness ratings were higher in the B trial compared with the NB trial immediately after the preload had been consumed (0 min) (P < 0.05).

Ratings of fullness and hunger both showed a main effect of time (P < 0.01) in response to the test meal when energy intake at the meal was used as a covariant in the analysis. Following consumption of the test meal, ratings of hunger decreased, whereas ratings of fullness increased. Following this initial response, both ratings gradually returned in the direction of pretest meal values. Hunger and fullness ratings did not differ between trials.

Ratings of thirst and nausea displayed significant main effects of time only in response to the preload and a main effect of time only for responses to the lunchtime test meal when energy intake at the test meal was used as a covariate in the analysis. Ratings of desire to eat displayed similar responses to hunger ratings (Supplemental Fig. 1).

**Discussion**

Our aim in this study was to investigate the effects of breakfast consumption on subjective appetite, energy intake, and the responses to a standard liquid preload. Snacking is more common among individuals who do not regularly consume breakfast (23); therefore, the findings from the present study provide useful information on the differential metabolic and endocrine responses to a...
snack consumed with or without a preceding breakfast. A breakfast consumed at 0830 h may be followed or replaced by a mid-morning snack at 1100 h and lunch at 1230 h, explaining the 150-min interval between breakfast and preload and the 90-min interval between the preload and ad libitum test meal used in this paradigm.

In this study, energy intake at the lunchtime test meal was reduced (~17%) and participants reported feeling fuller and less hungry in response to the preload when they had consumed breakfast. Furthermore, there were significant differences in the metabolic and endocrine responses. These findings support previous reports of beneficial effects of breakfast consumption, enhancing appetite regulation, and control of short-term food intake (34,35).

Although there is consistent evidence of an association between breakfast consumption and healthy body weight (16), there is limited evidence of an explanatory biological mechanism. Missing breakfast increased total daily energy intake, promoting a positive energy balance and longer term weight gain in some (7–12,14,36), but not all (11,18,19,36,37), studies. We did not measure food intake during the free-living part of this experiment; therefore, we were unable to determine whether breakfast affected total daily intake. However, when our participants omitted breakfast at a single occasion, they compensated for the “missed” energy by increasing energy intake at lunchtime. Previous studies reported an increase in voluntary food intake following the omission of breakfast (38–40). Omitting breakfast (even on a single occasion) may disrupt the distribution of energy intake throughout the day, resulting in fewer but larger meals being consumed, even if there is no overall effect on daily energy intake.

The increased energy intake at lunchtime on the no-breakfast day was associated with higher feelings of hunger and desire to eat and lower feelings of fullness during the mid-morning period despite having consumed a 1050-kJ preload in both trials.

Participants were required to consume the breakfast and liquid preload within fixed times as in previous studies (41,42) and all were able to do so. Altering the consumption rate of standard-sized test meals did not influence subjective appetite, blood glucose, gut peptide concentrations, or voluntary intake at a subsequent test meal (43,44), and thus any differences in the rate of consumption were unlikely to have significantly affected the study outcomes.

The increase in self-reported nausea following consumption of the preload was observed in both trials, and the differences between the trials were small and insignificant. Nevertheless, these differences in nausea may have influenced other subjective appetite ratings.

The differences observed in appetite ratings may be related to the gut hormone responses. Enhanced suppression of ghrelin is associated with increased hunger (45) and increases in GLP-1 and PYY are associated with reduced subjective appetite and energy intake (46,47).

The higher blood glucose and serum insulin responses to the standard liquid preload when breakfast was omitted are consistent with the responses observed following a mixed macronutrient test meal after a prolonged period of fasting (48), despite the fasting period in this study (NB) being substantially shorter. Furthermore, although both glucose and insulin responses to the preload were higher when breakfast was omitted, the enhancement of glucose was ~3 times greater than that of insulin. This suggests a degree of insulin resistance in response to the liquid preload following the omission of breakfast, which is consistent with previous reports that increased inter-meal interval, prolonged periods of fasting, and omitting breakfast cause a degree of insulin resistance (24,49,50). However, the present findings indicate that missing breakfast at a single occasion leads to insulin resistance in response to foods consumed at the next meal. Additionally, the insulin resistance displayed by those who habitually miss breakfast (24) may be driven by the accumulated differences in the responses to foods consumed later in the day.

The reduced insulin sensitivity in response to the preload when breakfast was omitted may be due to differences in FFA concentrations when the preload was consumed. Immediately before the preload, FFA concentrations were low on the breakfast day and high when breakfast was omitted. The elevated FFA levels on the no-breakfast day were likely to impair insulin stimulated glucose utilization (51) and glucose disposal (52,53) after the preload.

Schlundt et al. (23) demonstrated that breakfast consumers lost more weight when they did not eat breakfast, and those who did not regularly consume breakfast lost more weight when they were made to eat a standard breakfast, which suggests that changing the usual eating pattern may have a greater effect than the effect of breakfast per se in those aiming to lose weight. In the present study, we investigated the effect of omitting breakfast in those who consumed breakfast on a regular basis as opposed to another recent study that investigated the effect of reintroducing breakfast to those who skip breakfast (40). Both studies reported similar effects in regard to energy intake; i.e. overall energy intake did not differ when participants consumed breakfast compared with when no breakfast was eaten. However, neither study definitively showed that the observed responses were a result of breakfast per se, because both studies incorporated changes to the participants’ habitual eating pattern. Limited data exist with respect to this issue (40) and it is unclear whether the responses to breakfast may differ between these groups. Future investigations comparing the effects of breakfast on appetite and energy intake in regular consumers with those who do not regularly consume a breakfast meal are warranted.

Many studies investigating appetite responses to various food manipulations use the preload-test meal paradigm, with many providing preloads to participants in a fasted state (54–57). The majority of foods, however, are consumed in the postprandial state. The results of the present study demonstrate that the appetite and metabolic responses to a standard preload may vary considerably depending on whether it is consumed in the postprandial or fasted state. These effects should be carefully considered, especially because the effects of the tested foods may vary depending on when they are consumed.

We undertook this controlled experimental study to investigate possible biological mechanisms that might explain a causal relationship between breakfast consumption and obesity. Familiar food items were provided to the participants for breakfast and the size of the breakfast was individualized with respect to energy requirement based on body weight and physical activity. The relatively small breakfast (10% DER) could be regarded as a limitation, because it is unlikely to represent a typical breakfast consumed by healthy males (58). However, the amounts used were based on manufacturers’ recommended serving sizes. The study was statistically powered to detect a difference in ad libitum energy intake at the lunchtime test meal, which was the primary outcome variable. A further limitation is that the study was not adequately powered to detect differences in other study outcomes, such as subjective appetite and gut hormone responses. Nevertheless, these findings provide insight into the effects of breakfast on appetite and energy intake and suggest that further research into the biological mechanisms of breakfast consumption on appetite regulation and energy intake is warranted.
In summary, it appears that regular breakfast eaters who do not consume breakfast compensate by increasing the consumption of foods later in the day. Furthermore, the prior consumption of breakfast affects the metabolic and endocrine responses to the foods consumed during the remainder of the day. Together, these findings may help explain the association between regular breakfast consumption and the maintenance of a healthy body weight.

Acknowledgments

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