Effect of reducing portion size at a single meal on daily energy intake, appetite and gastrointestinal hormones in overweight and obese adults

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Abstract

Background—Larger portion sizes (PS) are associated with greater energy intake (EI), but little evidence exists on the appetitive effects of PS reduction.

Objective—To investigate impact of reducing breakfast PS on subsequent EI, postprandial gastrointestinal hormone responses and appetite ratings.

Design and Methods—Randomised crossover design (n=33 adults; mean BMI 29kg/m^2) providing a set breakfast based on 25% of gender-specific estimated daily energy requirements; PS reduced by 20% and 40%. EI measured at an *ad libitum* lunch (240mins) and snack (360mins), and by weighed diet diaries until bed. Blood was sampled until lunch in 20 participants. Appetite ratings were measured using visual analogue scales.

Results—EI at lunch (control:2930±203; 20% reduction:2853±198; 40% reduction:2911±179kJ) and over the whole day except breakfast (control:7374±361; 20% reduction:7566±468; 40% reduction:7413±417kJ) did not differ. Postprandial PYY, GLP-1, GIP, insulin and fullness profiles were lower and hunger, desire to eat and prospective consumption higher following 40% reduction compared to control. Appetite ratings profiles, but not hormone concentrations were associated with subsequent EI.

Conclusions—Smaller portions at breakfast led to reductions in gastrointestinal hormone secretion but did not affect subsequent energy intake, suggesting small reductions in portion size may be a useful strategy to constrain EI.

Keywords

Portion size; appetite; gastrointestinal hormones; energy intake

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Physical Activity Questionnaire (IPAQ)[10]), unable to eat test foods, and not regularly consuming breakfast (breakfast   3/week).

A sample size of 33 was recruited to give 83% power to detect a minimum difference of 500kJ lunch EI between any pair of experimental conditions assuming an SD of 950kJ[3,11,12]. Biochemical measures were collected in a sub-group of 20 participants.

Recruitment and screening

Participants were recruited from the community, for a study investigating the "relationship between diet and metabolism". Height, weight, waist circumference, body composition (Tanita body composition analyser BC-418MA), and resting metabolic rate (RMR; IS Gem204 with GEMNutrition2008.4 software) were measured. Participants completed the EAT-26, Zung depression scale, IPAQ and the Three Factor Eating Questionnaire (TFEQ) measuring dietary restraint, disinhibition, and hunger traits[13] and fasting plasma glucose was assessed. Participants were asked to maintain their usual exercise and dietary habits during the study.

Study visits

Participants fasted overnight (11h prior to each visit) and were asked to refrain from alcohol and avoid strenuous exercise the 24h before each study day. Provision of the test breakfast marked time zero. Subsequent EI was measured by pre- and post-meal weighing of an *ad libitum* lunch (240min) and afternoon snack (360min), plus a weighed diet diary to record the remainder of the day's intake. Visual analogue scale (VAS) questionnaires rating palatability and portion size were given during breakfast and lunch. Appetite ratings were measured using VAS questionnaires at 30min intervals until lunch, then immediately after and at 300 and 360min, then hourly. In a subgroup of 20 participants, blood samples were collected at fasting and 30, 60, 120, 180 and 240mins for the analysis of peptide tyrosine tyrosine 3-36 (PYY_{3-36}), total glucagon-like peptide-1 (GLP-1), total glucose-dependent insulinotropic peptide (GIP), glucose and insulin (Figure 1).

At the end of the study participants were fully debriefed on the study aims and asked to verbally report if they had noticed the portion size manipulation between visits. They were reimbursed for travel expenses and given an honorarium. Ethical approval for the study was obtained from Cambridgeshire 2 Research Ethics Committee in November 2010 (Ref: 10/H0308/99) and participants gave informed written consent. The study was conducted at Medical Research Council Human Nutrition Research (MRC HNR) between January 2011 and September 2012.

Study foods

The study breakfast and lunch provided 35% energy from fat, 18% from protein and 47% from carbohydrates[14]. The breakfast consisted of a wheat-based breakfast cereal with semi-skimmed milk, scrambled egg, ham, brown toast and butter, and orange juice. For men and women respectively, the control breakfast provided 3310kJ and 2540kJ; the 20% reduction breakfast provided 2650kJ and 2030kJ; the 40% reduction breakfast provided 1990kJ and 1520kJ. The *ad libitum* lunch consisted of a single course amorphous meal of pasta, minced

beef, tomato sauce, mixed vegetables and grated cheese. The lunch provided 8275kJ(men) or 6350kJ(women). The *ad libitum* snack consisted of ten digestive biscuits (plain cookies) on a plate(3250kJ).

The completed diet diaries and weighed consumption at lunch and snack for each study day were used to calculate energy intake using MRC HNR's dietary assessment system.

Questionnaires

The appetite VAS questionnaires rated hunger, fullness, desire to eat and prospective consumption, and also included five distractor questions on mood. The palatability questionnaire used VAS to rate palatability parameters, as distractor questions, and portion size. The VAS questionnaires asked participants to mark a horizontal line measuring 100mm with the ends labelled with the extremes of each sensation (e.g. "Not at all" and "Extremely"). The portion size question was "How large is the size of the portion?" anchored with "Extremely small" and "Extremely large". The distance from the left end to the mark was measured to the nearest millimetre.

Analytic methods

Blood samples were separated on collection and plasma stored at -80°C. Plasma collected on EDTA and treated with dipeptidyl peptidase-IV (DPP-IV) inhibitor immediately on collection (10μl DPP-IV inhibitor/ml of blood) were analysed for PYY_{3-36} by radioimmunoassay (Millipore®, Massachusetts, USA) (interassay CVs: 15% at 84pg/ml; 7% at 217pg/ml), at University College Hospital, London; total GLP-1 using an electrochemical luminescence immunoassay kit on the MesoScale Discovery® multi-array assay platform (Maryland, USA) (CVs: 16.4%, 11.9% and 11.6% at 5.4pg/ml, 29pg/ml and 83pg/ml respectively), at Core Biochemical Assay Laboratory (CBAL), Cambridge; and total GIP using an enzyme-linked immunosorbent assay (Millipore®, Massachusetts, USA) (CVs: 6.1%, 3.3%, 2.3% and 1.8% at 26pg/ml, 50pg/ml, 134pg/ml and 166pg/ml respectively), at Cambridge Institute for Medical Research. Plasma collected on fluoride oxalate were analysed for glucose using a Dimension® clinical chemistry system (Siemens, Newark, USA) (CVs: 1.69%, 2.23% and 2.56% at 6.23mmol/L, 3.09mmol/L and 18.88mmol/L respectively), at MRC HNR. Plasma collected on lithium heparin were analysed for insulin on a 1235 AutoDELFIA® automatic immunoassay analyzer using a two-step time resolved fluorometric assay (Perkin Elmer Life Sciences, Wallac Oy, Turku, Finland) (CVs: 3.1%, 2.1%, 1.9% and 2.0% at29pmol/L, 79.4pmol/L, 277pmol/L and 705pmol/L respectively) at CBAL, Cambridge.

Statistical analysis

Mixed effects models for continuous responses[15] were used for analysis, which extend standard linear regression to account for within-person variation through random effects. EI and perceived PS at breakfast were modelled with PS condition as the explanatory variable, controlling for gender and BMI. Dietary restraint, disinhibition and hunger, were tested for inclusion as covariates, but were omitted for no effects on the associations of interest.

The effect of PS condition on biochemical measures and appetite ratings was assessed by the interaction between condition and time, which estimated differences at each time-point. Area under the curve (AUC) was calculated using the trapezoidal rule for the time periods of fasting to the pre-lunch time-point for biochemical measures and appetite ratings, and over the whole day for appetite ratings. Models of whole-day appetite ratings AUC included PS condition as the explanatory variable, controlling for time over which appetite ratings were made.

Models predicting EI at lunch included explanatory variables of either the pre-lunch or AUC for each biochemical measure or appetite rating, and controlled for condition, gender and BMI. Similar models assessed the relationship between the whole-day AUC of appetite rating with whole day EI (except breakfast), also controlling for time over which appetite ratings were made.

To examine the relationship between biochemical measures and appetite ratings, appetite ratings were modelled separately with each biochemical measure as the explanatory variable. Time, a quadratic term for time, condition, gender and BMI were covariates.

Potential carry-over and sequence effects, gender, BMI and age, unless specified above as included *a priori,* were omitted as covariates as there were no effects on the associations of interest. To account for correlation induced by multiple observations/individual (three visits), a random intercept was incorporated into the models. The models for biochemical and appetite ratings profiles as outcomes had two levels of clustering due to repeated sampling time-points and the crossover design. Therefore, a random intercept and slope for time were added to model within-individual variation. Models were fitted using maximum likelihood estimation and likelihood ratio tests were used for model comparison. Plots of residuals were used to check the goodness of fit for each outcome. Insulin and GIP data were transformed (natural logarithm and square root respectively) for analyses, for a symmetrical distribution. All analyses used STATA®12.0 software (StataCorp, Texas, USA). Statistical significance was set at $p<0.05$. Data are presented as mean±SEM unless indicated otherwise.

Results

Participant characteristics

The characteristics of the study participants are shown in Table 1.

Energy intake

EI was not different between conditions at lunch (control: 2930±203; 20% reduction condition: 2853±198; 40% reduction condition: 2911±179kJ) or over the whole day except breakfast (control: 7374±361; 20% reduction condition: 7566±468; 40% reduction condition: 7413±417kJ) (Figure 2). Daily EI including breakfast was 10287±395kJ, 9897±491kJ and 9161±437kJ in control, 20% reduction and 40% reduction conditions respectively.

Biochemical measures

Figure 3 shows the postprandial profiles for each of the gastrointestinal hormones. Compared to control, there was a reduction in PYY in 40% reduction condition at 120 and 240mins($p<0.05$), but no condition-time interaction for 40% reduction compared to 20% reduction condition($p>0.076$), or 20% reduction compared to control($p>0.42$). Compared to control, GLP-1 was lower in 40% reduction condition at all postprandial time-points($p<0.05$). GLP-1 was also lower in 40% reduction condition compared to 20% reduction at 180mins($p=0.038$), but there was no condition-time interaction for 20% reduction condition compared to control($p>0.056$). GIP was lower in 40% reduction condition compared to control at all postprandial time-points($p<0.001$), and compared to 20% reduction condition at 30, 120, 180 and 240mins($p<0.05$). GIP was lower at 120, 180 and 240mins in 20% reduction condition compared to control($p<0.05$).

Glucose and insulin profiles are shown in Figure 4. There was no condition-time interaction for glucose($p>0.2$ for all comparisons). There was a condition-time interaction such that insulin was less in 40% reduction condition compared to control and 20% reduction condition at 120 and 180($p<0.05$), and compared to control at 240mins($p<0.01$)There was no condition-time interaction for 20% reduction condition compared to control($p>0.083$).

Appetite ratings

Figure 5 shows the appetite ratings. Hunger was greater in 40% reduction condition at all time-points from 30-240min ($p<0.006$) compared to control, and at all postprandial time-points compared to 20% reduction condition ($p<0.021$). There was no condition-time interaction for 20% reduction condition compared to control ($p>0.291$). Fullness was lower in 40% reduction condition at all time-points from 20-180mins($p<0.019$) compared to control and at30($p=0.017$) and 90min($p=0.003$) compared to 20% reduction condition. Also fullness was lower in 20% reduction condition compared to control at 60($p=0.041$) and 120mins($p=0.040$). Desire to eat ratings were greater in 40% reduction condition at all time-points postprandially($p<0.023$) compared to control, and at all time-points from 20-210min ($p<0.037$) compared to 20% reduction condition. There was no condition-time interaction for 20% reduction condition compared to control($p>0.223$). Prospective consumption was greater in 40% reduction condition compared to control at all time-points postprandially($p<0.011$) and compared to 20% reduction condition, at 120($p=0.018$) and 150min($p=0.027$). There was no condition-time interaction for 20% reduction condition compared to control($p>0.068$).

AUCs over the whole day for hunger, desire to eat and prospective consumption were greater in 40% reduction condition compared to control, and smaller for fullness (hunger $\beta=2423.9,p=0.025$; fullness $\beta=-4857.9,p=0.001$; desire to eat $\beta=3832.5,p=0.001$; prospective consumption $\beta=3427.9,p=0.001$). AUC for prospective consumption ratings was greater in 20% reduction condition compared to control($\beta=2284.1,p=0.025$), but AUC for hunger($p=0.232$), fullness($p=0.136$), and desire to eat($p=0.118$) did not differ. There were no differences in hunger or fullness when comparing 20% and 40% reduction conditions (data not shown).

Predictors of energy intake

Most of the biochemical measures did not predict EI at lunch (p>0.137) (Table 2). However, AUC (p=0.032) and pre-lunch (p=0.049) measures of PYY were positively associated with EI at lunch. AUCs and pre-lunch measures of hunger, desire to eat and prospective consumption were positively associated with lunch EI (p<0.02). Pre-lunch fullness was negatively associated with lunch EI (p<0.002), but fullness AUC was not (p=0.085). AUCs for hunger, desire to eat and prospective consumption, but not fullness (p=0.469), were positively associated with EI over the day (p<0.026).

Biochemical measures and appetite ratings associations

GLP-1, GIP, glucose and insulin were negatively associated with hunger, desire to eat, and prospective consumption, and positively associated with fullness (p<0.012). PYY was not associated with any of the appetite ratings (p>0.068) (Table 3).

Perceived portion size

The ratings of portion size at breakfast were different between conditions. Portion size was perceived to be smaller in 40% reduction condition compared to both control (β=-15.6, p<0.001) and 20% reduction (β=-10.8, p<0.001), and portion size was perceived to be smaller in 20% reduction condition compared to control(β=-4.8, p=0.049) (portion size rating: control 53±2mm; 20% reduction 48±3mm; 40% reduction 37±3mm. However at debriefing only two participants specifically reported noticing the change in PS at breakfast. None of the participants were concerned about the blinding of the aims of the study and consented to data inclusion.

Discussion

Reducing PS at a single meal altered psychological and biological markers of appetite, but there was no energy compensation later in the day. EIs at lunch were strikingly consistent in this standardised laboratory setting. These findings indicate reducing PS of a prepared meal could lead to a net reduction in daily EI. However, the effect on gastrointestinal hormones and appetite ratings, particularly after the 40% reduction in PS, questions the sustainability of this strategy to constrain EI.

There were very few differences in the profiles for PYY and GLP-1 between control and 20% reduction conditions. Moreover, there were few differences in the profiles when comparing 20% and 40% reduction conditions suggesting that the responses in these biochemical measures may not be sensitive to the smaller change in PS (660kJ men and 510kJ women). Indeed, all previous studies where a reduction in energy load has led to attenuated PYY[16,17], GLP-1[18,19], or insulin[20,21] profiles, used energy changes between 920-2096kJ. However, the present study showed distinct differences between all conditions in the postprandial profiles for GIP showing that it is sensitive to energy changes in a clear dose response manner, reflecting its important role as an incretin hormone for the regulation of insulin secretion.

Interestingly, the ratings of perceived PS of the breakfast were different between conditions, although at debriefing most participants reported not noticing the meal manipulation. The effect size for the difference between perceived PS ratings was considerably smaller when comparing control *versus* 20% reduction than 20% *versus* 40% reduction conditions (β=-4.8; β=-10.8), although the absolute difference in energy was the same. This difference is likely due to either the relative difference between PS being different (20% for control-20% reduction comparison, and 25% for 20%-40% reduction comparison), or due to the Weber-Fechner law, whereby the ability to perceive stimulus change is proportional to the logarithm of the magnitude of the stimulus[22]. Thus, as the reference portion size in the first comparison (control *versus* 20% reduction) was larger than the second (20% *versus* 40% reduction), the change in PS detectable for the first pairing would have been larger than the second. It is possible that the perception of how much is provided, and thus consumed, could affect appetite ratings. The smaller effect size of perceived PS between control and 20% reduction conditions could in part account for fewer differences in appetite ratings between these conditions.

Postprandial biochemical responses were poor predictors of subsequent EI, consistent with much of the existing evidence[23–25]. However, appetite ratings tended to predict EI at lunch and the rest of the day. This is in agreement with some[12,26–28], but not all[29,30], previous studies. The mixed evidence likely reflects the subjective nature of the perception of appetite which leads to measurement variability, but differences are more easily detected in crossover than parallel design studies[31]. Although associations between appetite ratings and EI in the present study were highly significant, the effect sizes were small. This, coupled with relatively small differences in postprandial appetite ratings response to the manipulated meal, could in part explain the lack of compensation for the changes in energy. In contrast with the known function of PYY, where exogenous administration reduces EI[16,32,33], there was a small but significant positive effect of AUC and pre-lunch PYY on subsequent EI. However, the effect decreased after adjustment for additional participant characteristics, indicating it may be confounded by other factors. Thus there is uncertainty about these present findings relating to PYY. In contrast to the clear exogenous effect, endogenous postprandial responses in PYY were not associated with subsequent EI[12,24,34], possibly as exogenous PYY tends to be supra-physiological[12].

GLP-1, glucose and insulin were positively related to fullness and negatively related to hunger, desire to eat and prospective consumption consistent with previous research[21,23,35–37], indicating that these biochemical measures are likely to play roles in the perception of appetite sensations. However, some studies have found no relationship, or mixed results, between glucose or insulin and appetite ratings[23,28], possibly because they have reported correlations between the mean AUC or peak values rather than examining within-person relationships. Previous findings with respect to the relationship between postprandial PYY and appetite are mixed, including positive associations between PYY and perceived fullness[35,38], while others, consistent with the present findings, have found no associations[12,37,39], or associations in lean but not obese participants[34]. Thus, the robustness of the association of endogenous PYY with appetite ratings is questionable. It is unclear whether GIP plays a role in influencing appetite and EI[40], however the present findings showed GIP was associated with appetite ratings. The distinct similarity between

GIP and appetite ratings profiles may have led to these associations, but causality cannot be assumed. The lack of association between GIP and subsequent lunch EI is in agreement with the perspective that GIP does not influence EI.

The present findings support the concept that reducing the PS of pre-prepared meals or unit foods could constrain EI and contribute to prevention of weight gain. However as weight control advice is inherently overt, it is important to establish whether similar effects are seen when participants are aware of the reduction in PS.

There are several limitations to this study. It was conducted in a laboratory setting and, although the specific hypothesis was concealed, participants were aware of their eating behaviour being observed. The frequency and type of food provided at lunch was fixed, thus only the amount could vary potentially limiting compensation by removing some of the environmental cues that are profuse in a free-living environment and can influence EI. This setting also prevented any self-initiated eating episodes between breakfast and lunch. Some of the appetite and hormone profiles suggest effects of PS reduction may have diminished over time and compensation might be seen in a free-living environment during this period. The use of a different control PS (in terms of percentage of estimated daily energy requirements) may alter the relative effects of a percentage reduction. The overweight and obese sample may limit comparison with previous work, however it was most appropriate to study portion size reduction in this group as those with an elevated BMI are most in need of interventions to constrain energy intake, and are to date an understudied population in this field. The study was conducted over a single day and it is possible that a longer period of consuming portions set to provide energy below requirements could lead to adaptation and energy compensation. Future studies should attempt to examine PS reduction in a more realistic setting and with prolonged exposure to smaller portions.

Conclusions

Reductions in PS led to lower EI, despite changes in biological and behavioural measures that tend to favour energy compensation. Although the effect size was small, if sustained this will be of public health benefit in constraining energy intake.

Acknowledgements

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What is already known about this subject

- Larger portion sizes are linked with increased energy intake.

- There are strong homeostatic mechanisms to guard against low energy intakes. There is a paucity of data of the effects of smaller portions on appetite control systems and energy intake.

What this study adds

- New evidence of the effects of smaller portions on gastrointestinal hormone responses to food.

- Data on the effect of portion size reductions on appetite ratings and subsequent energy intake in overweight and obese adults.

- Support for portion size reduction as a potential strategy to constrain energy intake.
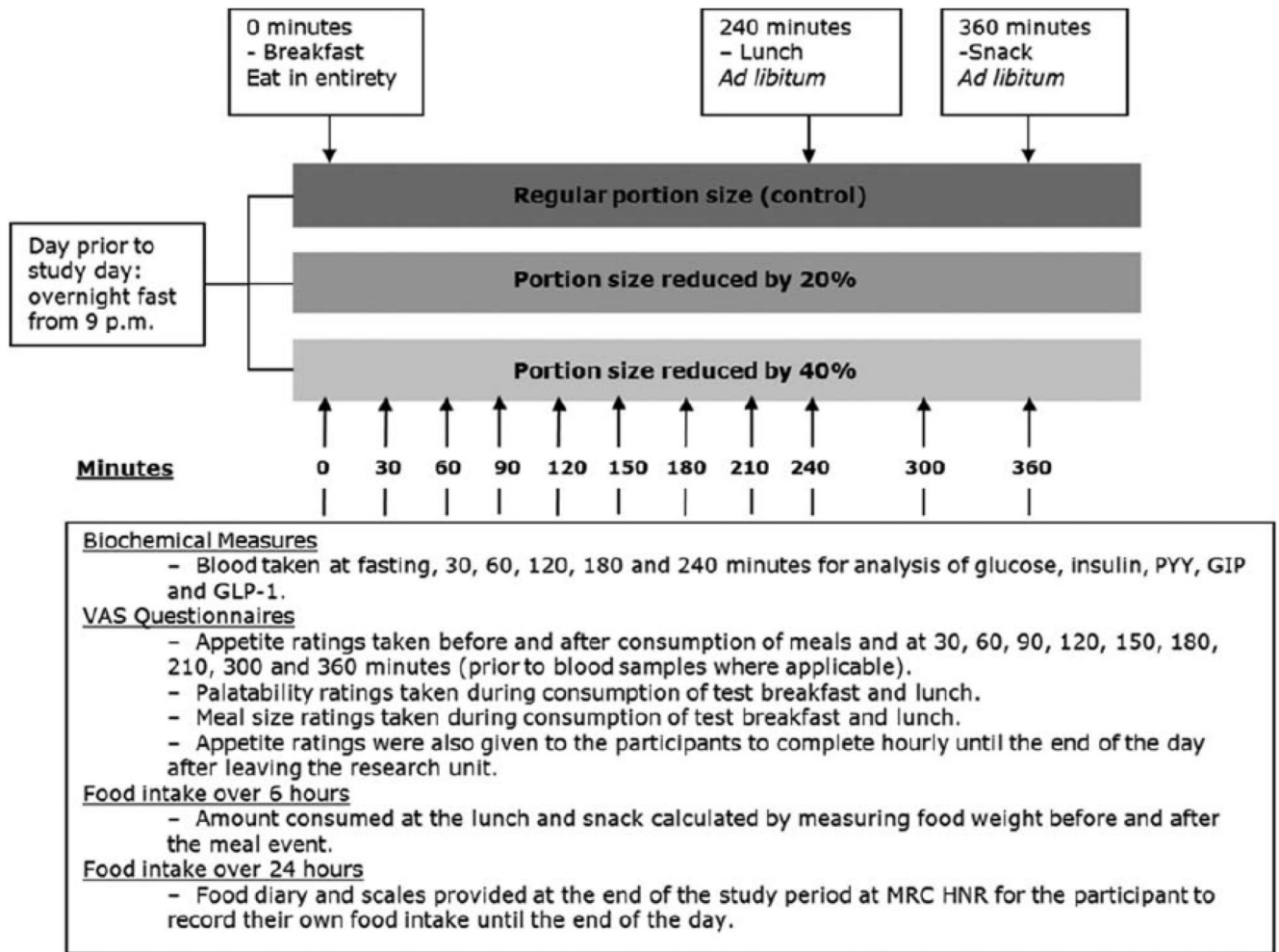
Figure 1.
Overview of the time points for meals and measurements taken during a study day (GIP: glucose-dependent insulinotropic peptide; GLP-1: glucagon-like peptide 1; MRC HNR: Medical Research Council Human Nutrition Research; PYY: peptide tyrosine tyrosine; VAS: visual analogue scales).
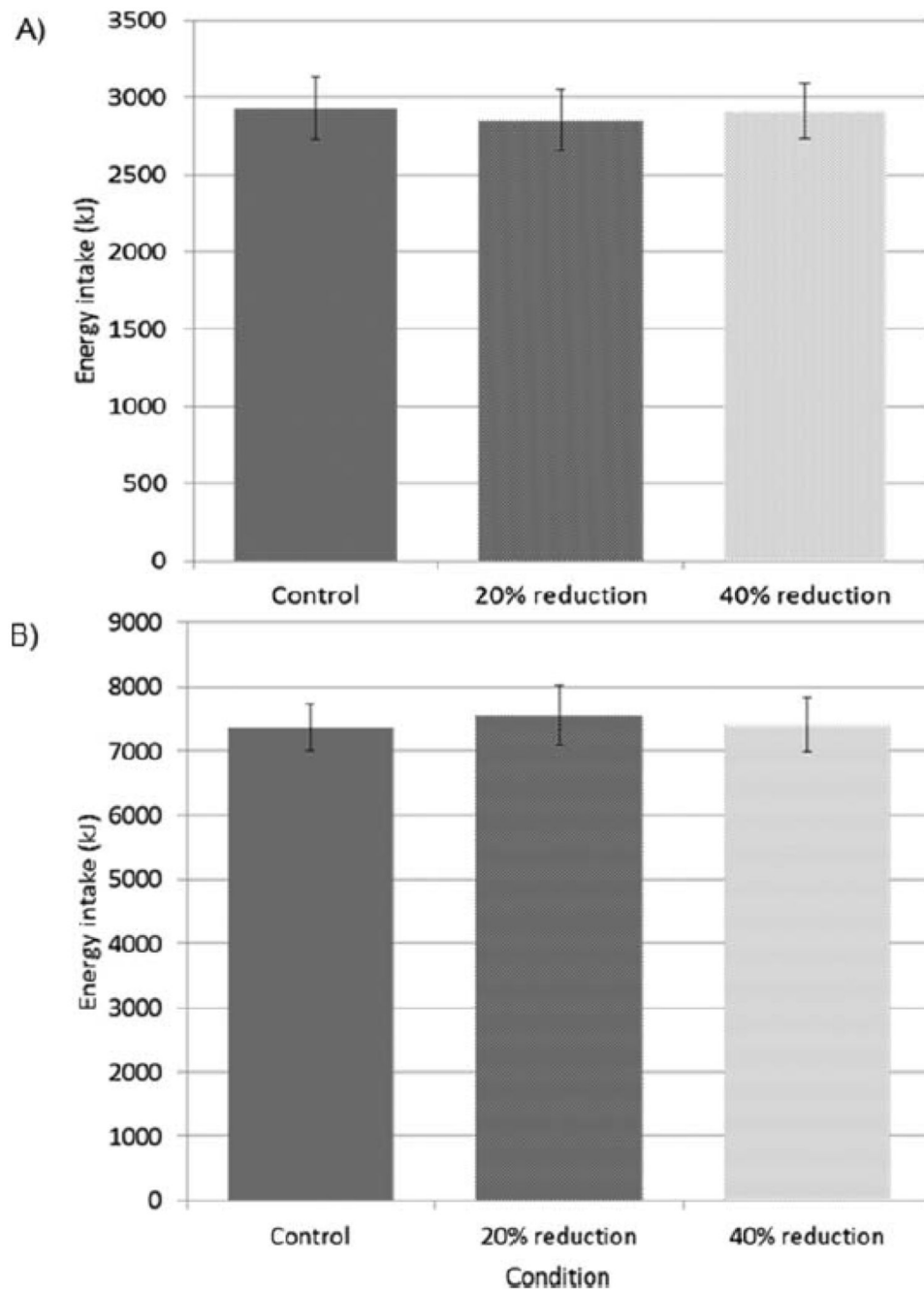
Figure 2.
Mean (± SEM) energy intake at A) lunch were not different between conditions (control vs. 20% reduction, β=-76.6, p=0.429; 20% reduction vs. 40% reduction, β=58.2, p=0.547; control vs. 40% reduction, β=-18.3, p=0.850); and B) over the whole day except breakfast (control vs. 20% reduction, β=192.3, p=0.555; 20% reduction vs. 40% reduction, β=-152.8, p=0.639; control vs. 40% reduction, β=39.5, p=0.904).
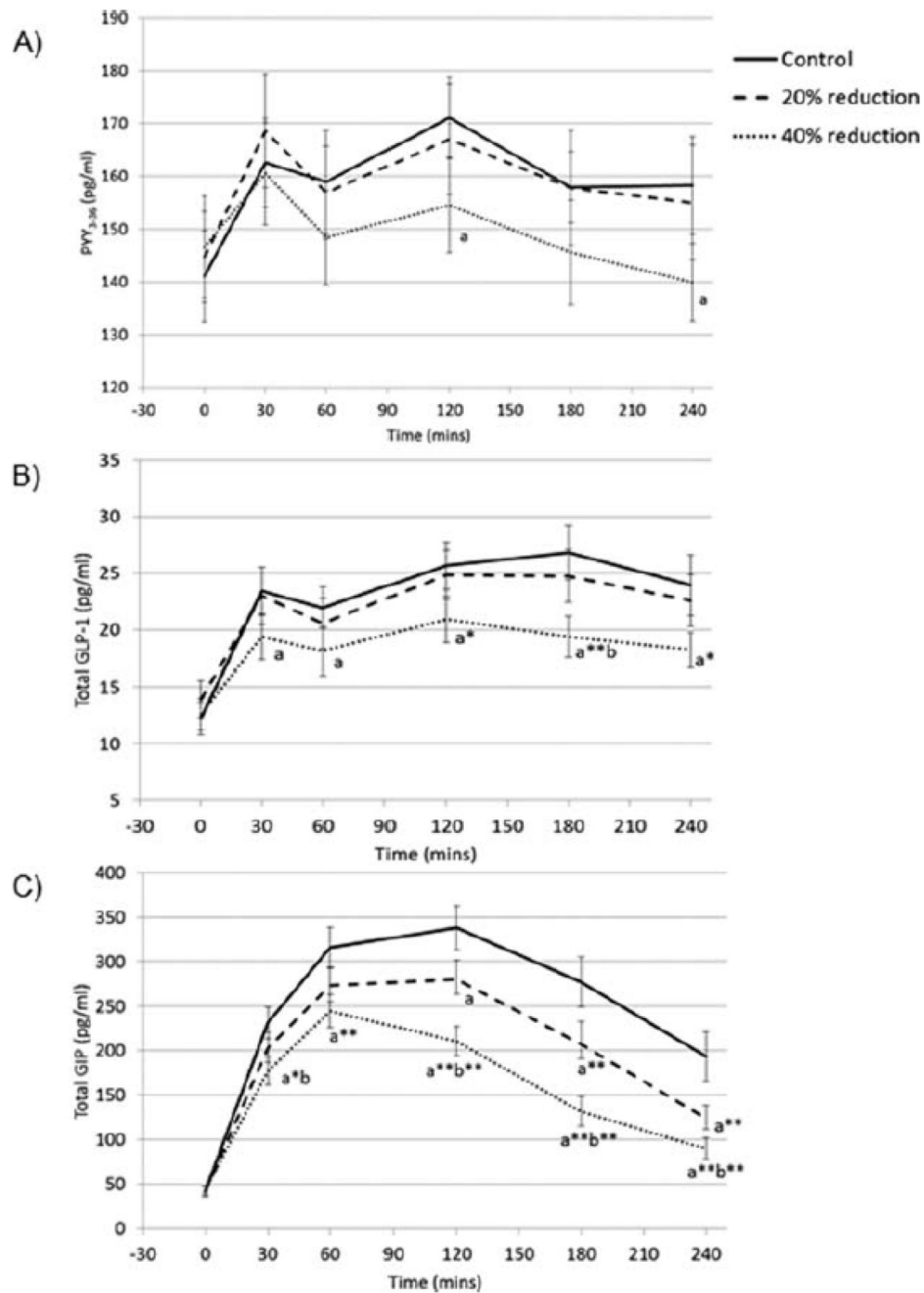
Figure 3.
Postprandial response (mean ± SEM) of A) plasma PYY_{3-36}, B) plasma total GLP-1, and C) plasma total GIP, according to condition.
'a' indicates the mean of the condition is significantly different to the mean of the control condition at that time point. 'b' indicates the mean is significantly different to the mean of the 20% reduction condition at that time point (mixed effects models): $p<0.05$. Addition of * to the letter indicates $p<0.01$ and ** indicates $p<0.001$.
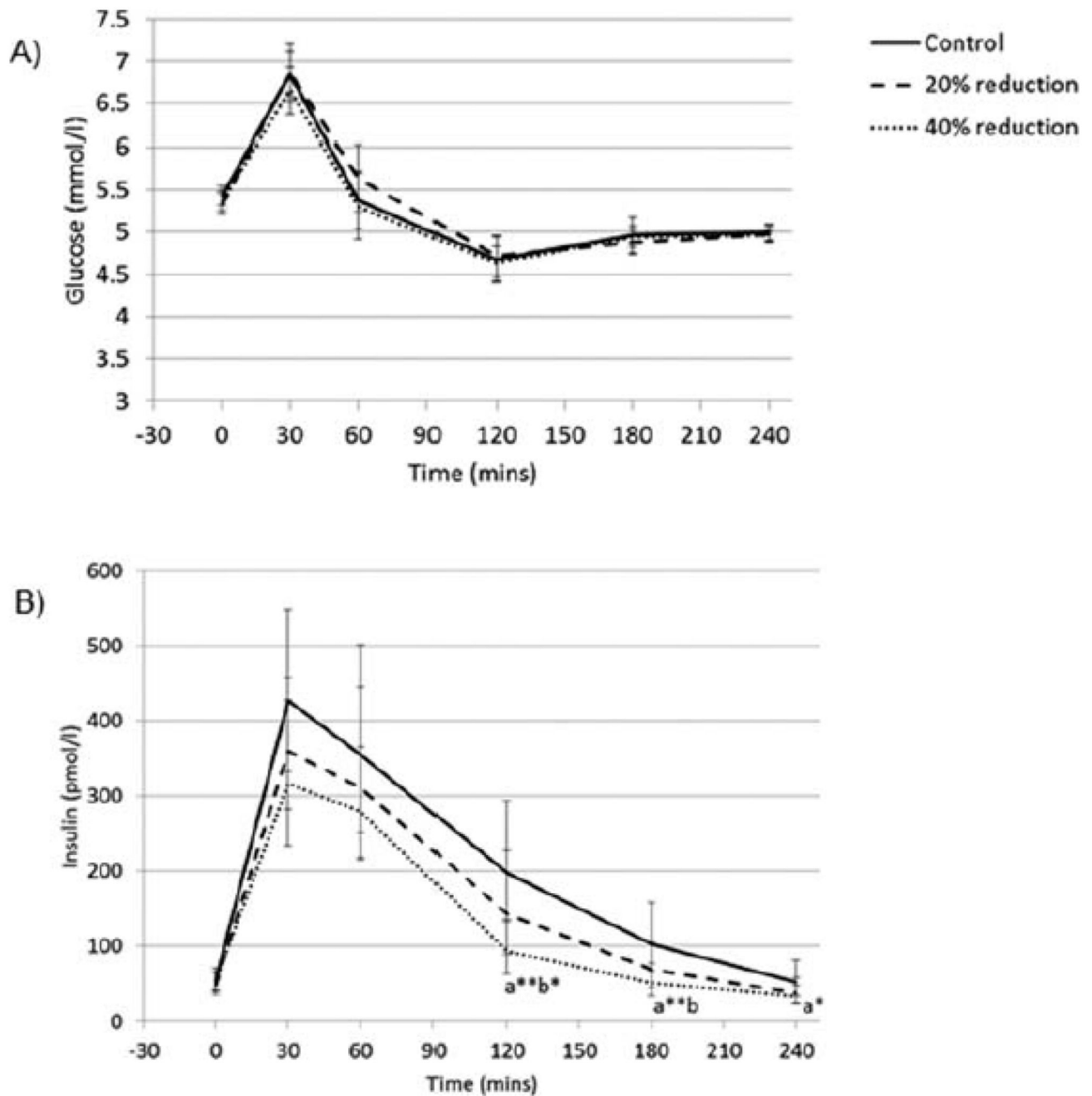
Figure 4.
Postprandial response of A) plasma glucose (mean ± SEM), and B) plasma insulin
(geometric mean ± 95% confidence intervals), according to condition.
'a' indicates the mean of the condition is significantly different to the mean of the control
condition at that time point. 'b' indicates the mean is significantly different to the mean of
the 20% reduction condition at that time point (mixed effects models): $p<0.05$. Addition of *
to the letter indicates $p<0.01$ and ** indicates $p<0.001$.
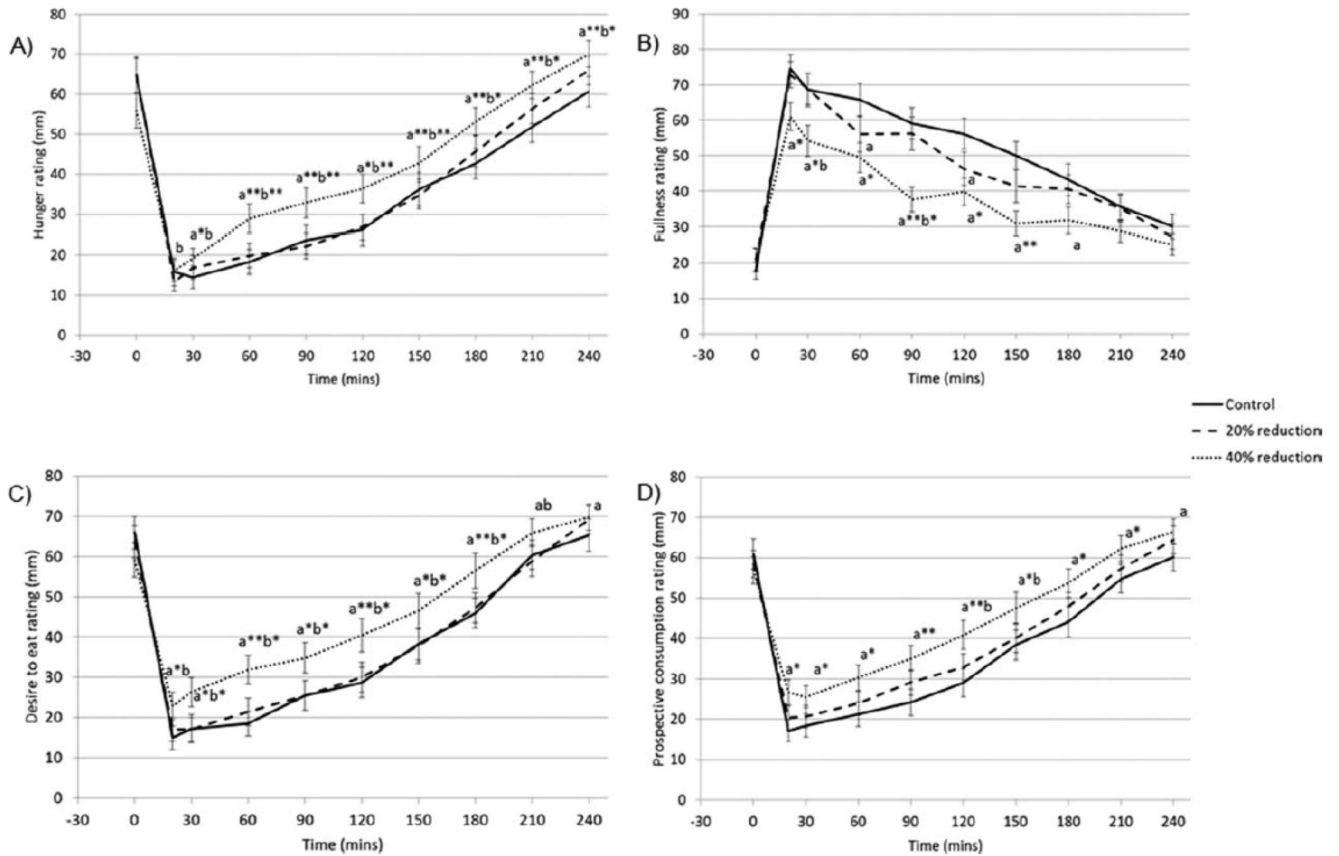
Figure 5.
Postprandial ratings (mean ± SEM) for A) perceived hunger, B) perceived fullness, C) perceived desire to eat, and D) perceived prospective consumption, according to condition. 'a' indicates the mean of the condition is significantly different to the mean of the control condition at that time point. 'b' indicates the mean is significantly different to the mean of the 20% reduction condition at that time point (mixed effects models): $p < 0.05$. Addition of * to the letter indicates $p < 0.01$ and ** indicates $p < 0.001$.

Table 1

Participant characteristics.

Participant characteristic	All participants (n=33)	Blood sample subgroup (n=20)	Non-blood subgroup (n=13)
Number of men/women	15/18	9/11	7/6
Height (m)	1.69 ± 0.01	1.69 ± 0.01	1.71 ± 0.03
Weight (kg)	83.8 ± 1.5	82.9 ± 2.1	85.3 ± 2.0
BMI (kg/m^2)	29.0 ± 0.4	29.0 ± 0.5	29.2 ± 0.8
Age (years)	42.5 ± 2.0	40.8 ± 2.5	45 ± 3.4
Dietary restraint	7.2 ± 0.7	6.5 ± 0.9	8.2 ± 1.1
Disinhibition	6.7 ± 0.6	6.5 ± 0.7	6.9 ± 1.1
Hunger trait	6.3 ± 0.7	6.2 ± 0.8	6.5 ± 1.1
RMR (kJ/day)	6594 ± 160	6704 ± 224	6425 ± 220
Fasting glucose (mmol/L)	4.8 ± 0.1	4.7 ± 0.1	4.9 ± 0.1
Body fat (%)	32.8 ± 1.5	31.9 ± 1.8	34.2 ± 2.6
Vigorous physical activity (mins per week)	65 ± 13	55 ± 14	80 ± 24
Moderate physical activity (mins per week)	142 ± 21	173 ± 29	94 ± 26
Walking (mins per week)	254 ± 30	270 ± 37	231 ± 53

Mean ± SEM.
BMI: Body Mass Index. RMR: Resting metabolic rate.

Table 2

Estimated regression coefficients to measure associations between biochemical measures and appetite ratings (predictor variables) with energy intake at lunch and over the whole day apart from breakfast (outcome variables), from mixed effects models.

	AUC as predictor		Pre-lunch measure as predictor	
Predictor of lunch EI	**Regression coefficient (SE)**	**p-value**	**Regression coefficient (SE)**	**p-value**
Biochemical measure				
PYY	**0.029 (0.014)**	**0.032**	**4.442 (2.257)**	**0.049**
GLP-1	0.019 (0.071)	0.790	15.95 (11.17)	0.154
GIP	2.666 (4.446)	0.549	39.08 (33.06)	0.237
Glucose	-0.916 (0.710)	0.197	-365.5 (245.8)	0.137
Insulin	-197.0 (259.8)	0.448	-157.0 (168.7)	0.352
Appetite rating				
Hunger	**0.091 (0.022)**	**<0.001**	**11.96 (3.934)**	**0.002**
Fullness	-0.029 (0.017)	0.085	**-10.43 (3.389)**	**0.002**
Desire to eat	**0.087 (0.018)**	**<0.001**	**8.788 (3.783)**	**0.020**
Prospective consumption	**0.100 (0.022)**	**<0.001**	**19.21 (4.384)**	**<0.001**

Predictor of whole day EI	Regression coefficient (SE)	p-value		
AUC appetite rating				
Hunger	**0.057 (0.025)**	**0.026**		
Fullness	-0.016 (0.021)	0.469		
Desire to eat	**0.057 (0.023)**	**0.013**		
Prospective consumption	**0.068 (0.025)**	**0.007**		

AUC: area under the curve. EI: energy intake. SE: standard error.

Area under the curve was calculated for between the fasting and pre-lunch time points for predicting energy intake at lunch. Area under the curve for the whole day was calculated for predicting energy intake over the whole day apart from breakfast. Each predictor was analysed in a separate mixed effects model.

Values are given to 4 significant figures. Those in bold are significant.

Table 3

Estimated regression coefficients to measure associations between biochemical measures (predictor variables) and appetite ratings (outcome variables) from baseline to the pre-lunch time-point, from mixed effects models.

| | Appetite rating | | | | | | | |
| | Hunger | | Fullness | | Desire to eat | | Prospective consumption | |
Biochemical measure	Regression coefficient (SE)	p-value	Regression coefficient (SE)	p-value	Regression coefficient (SE)	p-value	Regression coefficient (SE)	p-value
PYY	-0.032 (0. 038)	0.409	0.041 (0.041)	0.315	-0.018 (0.040)	0.650	-0.028 (0.031)	0.366
GLP-1	**-0.494 (0.172)**	**0.004**	**0.631 (0.186)**	**0.001**	**-0.442 (0.176)**	**0.012**	**-0.421 (0.138)**	**0.002**
GIP	**-3.271 (0.373)**	**<0.001**	**3.357 (0.416)**	**<0.001**	**-3.143 (0.379)**	**<0.001**	**-2.629 (0.305)**	**<0.001**
Glucose	**-6.650 (1.058)**	**<0.001**	**6.058 (1.186)**	**<0.001**	**-5.493 (1.087)**	**<0.001**	**-4.396 (0.884)**	**<0.001**
Insulin	**-14.07 (1.227)**	**<0.001**	**14.33 (1.391)**	**<0.001**	**-13.63 (1.250)**	**<0.001**	**-11.86 (0.990)**	**<0.001**

SE: standard error.

Each predictor was analysed in a separate model.

Values are given to 4 significant figures. Those in bold are significant.