

The influence of resistance and aerobic exercise on hunger, circulating levels of acylated ghrelin and peptide YY in healthy males

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Running head

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ABSTRACT

Resistance (muscle strengthening) exercise is a key component of exercise recommendations for weight control yet very little is known about the effects of resistance exercise on appetite. We investigated the effects of resistance and aerobic exercise on hunger and circulating levels of the gut hormones acylated ghrelin and peptide YY (PYY). Eleven healthy male students: age 21.1 ± 0.3 y, body mass index 23.1 ± 0.4 kg/m², maximum oxygen uptake 62.1 ± 1.8 mL/kg/min (mean \pm SEM) undertook three, 8-h trials, 1) resistance exercise: a 90 min free weight lifting session followed by a 6.5 h rest period, 2) aerobic exercise: a 60 min run followed by a 7 h rest period, 3) control: an 8 h rest, in a randomised crossover design. Meals were provided 2 and 5 h into each trial. Hunger ratings and plasma concentrations of acylated ghrelin and PYY were measured throughout. Two-way ANOVA revealed significant ($P < 0.05$) interaction effects for hunger, acylated ghrelin and PYY indicating suppressed hunger and acylated ghrelin during aerobic and resistance exercise and increased PYY during aerobic exercise. A significant trial effect was observed for PYY indicating higher concentrations on the aerobic exercise trial than the other trials (8 h area under the curve: control 1411 ± 110 , resistance 1381 ± 97 , aerobic 1750 ± 170 pg/mL 8 h). These findings suggest ghrelin and PYY may regulate appetite during and after exercise but further research is required to establish whether exercise induced changes in ghrelin and PYY influence subsequent food intake.

Key words

Acylated ghrelin, appetite, exercise, hunger, peptide YY

INTRODUCTION

Body weight is regulated by a balance between food intake and energy expenditure (19). Exercise is an effective method of increasing energy expenditure (2) and it may, paradoxically, lead to a short term hunger suppression (6, 7, 24, 28, 29). This relationship between exercise and hunger has led investigators to study the role of gut hormones in mediating exercise-induced hunger changes. The majority of studies have focused on aerobic (cardiovascular) exercise (31) with only three studies examining the effects of resistance (muscle strengthening) exercise and these have reported contradictory effects (21, 33, 47). Resistance exercise is a key component of exercise recommendations for weight control (2) and public health (23, 42) thus it is important to clarify the effects of resistance exercise on hunger and gut hormones.

The effect of acute exercise bouts on total plasma ghrelin concentrations are controversial with studies reporting no changes either during or post-exercise (10, 15, 26, 27, 32, 39, 43, 45, 47), as well as increases (14, 17, 25, 44) and decreases (21, 33, 48, 50). Acylation of ghrelin is thought to be essential for appetite regulation because only the acylated form of the hormone can cross the blood-brain barrier (41). Thus, measurements of total ghrelin may mask important changes in acylated ghrelin. Currently, little is known about the influence of exercise on acylated ghrelin. Recently, we have shown that plasma acylated ghrelin is suppressed during vigorous treadmill running (9) while another recent study has reported increases in acylated ghrelin after five consecutive days of aerobic exercise (1 h per day) (36).

Less is known regarding the response of other gut hormones to exercise. Several studies have demonstrated increases in fasting and postprandial levels of the satiety hormone pancreatic polypeptide (PP) after aerobic exercise (38) but there is a paucity of data on peptide YY (PYY). There are two main circulating forms of PYY; PYY₁₋₃₆ and PYY₃₋₃₆ (11) both have been shown to reduce food intake when administered peripherally (13) however, PYY₃₋₃₆ is more potent than PYY₁₋₃₆. Only one study has investigated the response of PYY to an acute bout of exercise (39). In this study

subjects consumed a standardised breakfast and one hour later cycled for 60 min at 65% of their maximal heart rate. Plasma PYY concentrations were elevated during but not after exercise while the satiety hormones glucagon-like peptide-1 (GLP-1) and PP were elevated during and for a short while (up to 60 min) after exercise. Another recent study (12) has demonstrated an increased GLP-1 response to feeding after five consecutive days of aerobic exercise (1 h/day). Collectively, these findings suggest that hunger is inhibited during and for a short while after aerobic exercise.

Several limitations are apparent in the research literature regarding exercise and gut hormones. Most studies have measured gut hormone responses in the fasted state and for relatively short periods. Few studies have assessed post-exercise gut hormone responses to feeding over a prolonged period or attempted to relate these responses to changes in hunger. Moreover, no studies have examined acylated ghrelin and PYY responses to resistance exercise. This requires attention since ghrelin plays an important role in meal initiation (1) and PYY infusion, at normal postprandial concentrations, suppresses hunger in humans (3). Thus, we sought to investigate the acute effects of resistance and aerobic exercise on hunger, acylated ghrelin and total PYY in healthy male fasted subjects. In addition, we evaluated the effects of resistance exercise and aerobic exercise on meal-stimulated changes in hunger, acylated ghrelin and total PYY at two time points (2 h and 5 h post-exercise) to gain insights into the longer term effects of exercise on these parameters.

MATERIALS AND METHODS

Subjects

Loughborough University's Ethics Advisory Committee approved the study. Eleven healthy physically active, Caucasian males aged 19 to 23 yrs gave their written informed consent to participate. Subjects were non-smokers, not taking any medication, weight-stable for three months prior to the study and had no food allergies. The physical characteristics of the subjects were: age 21.1 ± 0.3 yrs, body mass index (BMI) 23.1 ± 0.4 kg/m², waist

circumference 78.5 ± 1.1 cm, maximum oxygen uptake 62.1 ± 1.8 mL/kg/min (4.6 ± 0.1 L/min).

Preliminary tests

Orientation session. Subjects attended the laboratory for an initial session during which anthropometric data were collected and they were familiarised with treadmill running and weight lifting. After this session subjects returned to the laboratory on two further occasions to complete weight lifting tests and on one further occasion to complete two treadmill running tests.

Weight lifting tests. A 12-repetition maximum test was completed for each of the 10 resistance exercises employed in the study. The order in which each exercise was performed was: squat, dumbbell lateral raise, bench press, upright row, lunges, bicep curl, barbell pullover, seated shoulder press, triceps extension and bent over row. On a separate visit subjects undertook a 90 min familiarisation session where they completed a full weight lifting session: three sets of 12 repetitions of 10 different weight lifting exercises at 80% of 12 repetition max.

Treadmill running tests. Subjects completed a 16-min submaximal treadmill running test and a maximum oxygen uptake test on a motorised treadmill as described previously (9). These tests were performed on the same day with a 30 min rest between tests. Expired air samples were collected into Douglas bags during these tests for the determination of oxygen consumption and carbon dioxide production (9). The results of the two tests were used together to determine the running speed required to elicit 70% of maximum oxygen uptake.

Main trials

One week after completing the preliminary exercise tests subjects undertook a counterbalanced randomised three-way crossover study with an interval of 7 days between each study day. The three trials were: resistance exercise (weight lifting), aerobic exercise (treadmill running) and control. For two days before the first main trial participants recorded their weighed food intake using a food record diary. The same food intake was consumed for the two days prior to subsequent trials. Participants were also asked to refrain

from vigorous exercise and ingesting caffeine or alcohol 24 h prior to the main trials. On trial days participants arrived at the laboratory between 08:00 and 09:00 having fasted for 10 h. Water was permitted *ad libitum* during this time.

Resistance exercise trial. At the start of this trial subjects completed a free weight session for 90 min performing three sets of 12 repetitions of 10 different weight lifting exercises at 80% of 12 repetition max. Participants were given 3 min to complete each set. On completion of the 12 repetitions, participants rested for the remainder of the 3 min. Exercises were completed in the order described for the preliminary tests. All sets for one exercise were completed before moving onto the next exercise. An expired air sample was taken for 3 min during the third set for each exercise. After the session, participants rested for 6.5 h. The short duration intermittent nature of weight lifting invalidates the assumptions of indirect calorimetry and therefore energy expenditure during weight lifting was estimated as 5.047 kcal (21.1 kJ) per litre of oxygen consumed (40). This reflects the assumption that energy was derived from carbohydrate rather than fat and assumes no protein oxidation during exercise. This assumption may not be entirely valid but was used to provide an approximation.

Aerobic exercise trial. At the start of this trial participants ran on the treadmill for 60 min at a speed predicted to elicit 70% of maximum oxygen uptake. One minute expired air samples were collected into Douglas bags at 14-15, 29-30, 44-45 and 59-60 min during the run. Oxygen consumption and carbon dioxide production were determined from expired air samples as described previously (9). Energy expenditure was predicted from oxygen consumption and carbon dioxide production values using indirect calorimetry (20). Ratings of perceived exertion were recorded during each expired air collection using the Borg scale (8). After the run, participants rested for 7 h.

Control trial. For the control trial participants rested for the entire duration of the trial.

Test meals

Participants were fed a test meal 2 h and 5 h into each trial (approximately 11:00 and 14:00 respectively). Meals consisted of white bread, butter, mayonnaise, Cheddar cheese, potato

crisps, whole milk and milk shake powder. The macronutrient content of the meals was 33% carbohydrate, 11% protein and 56% fat. The energy content was 3230 kJ for a 70 kg person. The amount of food consumed was adjusted for each participant based on their bodyweight and kept constant throughout all three trials. Participants were encouraged to consume the meal within 15 min and kept to the same start and finish times on all trials. Water was available *ad libitum* during trials.

Ratings of perceived hunger

Ratings of perceived hunger were assessed by means of a validated visual scale which ranged from 0 “not hungry” to 15 “very hungry” (10). Hunger measurements were recorded at baseline, 0.5, 0.75 and 1 h and every 30 min thereafter for the duration of each trial.

Blood sampling

In each main trial venous blood samples were collected into pre-cooled 9 mL EDTA monovettes (Sarstedt, Leicester, U.K.) at 0, 0.75, 1.5, 2, 2.5, 3, 4, 5, 5.5, 6, 7 and 8 h. In the control trial and the aerobic exercise trial all samples were collected using a cannula (Venflon, Becton Dickinson, Helsinborg, Sweden) which was inserted into an antecubital vein. In the weight lifting trial the first two blood samples (0 and 0.75 h) were collected by venepuncture and the remaining samples were collected using a cannula inserted into an antecubital vein. All blood samples were collected whilst subjects lay in a semi-supine position with the exception of the 0.75 h sample during the running trial, this sample was collected while subjects straddled the treadmill. The EDTA monovettes were spun at 1681 g (4000 revs/min) for 10 min in a refrigerated centrifuge (Burkard, Hertfordshire, U.K.) at 4°C. The plasma supernatant was then aliquoted into Eppendorf tubes. These were stored at -80°C for analysis of total PYY, glucose and insulin at a later date.

Separate venous blood samples were drawn into 4.9 mL monovettes at 0, 0.75, 1.5, 2, 2.5, 5, 5.5 and 8 h for the determination of plasma acylated ghrelin concentration. These monovettes contained EDTA and p-hydroxymercuribenzoic acid to prevent the degradation of acylated ghrelin by protease. The monovettes were spun

at 1287 g (3500 revs/min) for 10 min in a refrigerated centrifuge at 4°C. The supernatants were then aliquoted into storage tubes and 100 µL of 1 M hydrochloric acid was added per mL of plasma. Samples were then spun at 1287 g (3500 revs/min) for 5 min in a refrigerated centrifuge at 4°C before being transferred into Eppendorf tubes and stored at -80°C for analysis later.

At each acylated ghrelin blood sampling point, duplicate 20 µL blood samples were collected into micropipettes for the measurement of haemoglobin concentration and triplicate blood samples were collected into heparinised micro haematocrit tubes for the determination of haematocrit. Haemoglobin and haematocrit values were used to assess plasma volume changes (16).

Blood biochemistry

To eliminate inter-assay variation, samples from each participant were analyzed in the same run. Plasma acylated ghrelin concentrations were determined by enzyme linked immunoassay (ELISA) (SPI BIO, Montigny le Bretonneux, France). The within batch coefficient of variation (CV) was 4.8%. Total PYY was measured by ELISA (Diagnostic System Laboratories, Texas, USA). The within batch CV was 1.2%. Plasma insulin concentrations were determined by ELISA (Mercodia, Uppsala, Sweden). The within batch CV was 3.3%. Plasma glucose concentrations were determined by enzymatic, colorimetric methods (Randox Laboratories Ltd., County Antrim, UK) with the aid of an automated centrifugal analyzer (Cobas Mira Plus; Roche, Basel, Switzerland). The within batch CV was 3.3%.

Statistical Analysis

Data were analyzed using the Statistical Package for Social Sciences (SPSS) software version 14.0 for Windows (SPSS Inc, Chicago, IL, USA). Area under the curve (AUC) values were calculated using the trapezoidal rule. One-way ANOVA and Bonferroni post-hoc tests were used to assess differences between fasting and AUC values across trials. Two-way ANOVA was used to examine differences between trials over time. Where significant interactions were found, between trial differences at each time

point were examined using one-way ANOVA and Bonferroni post-hoc tests. The Pearson product moment correlation coefficient was used to examine relationships between variables. Statistical significance was accepted at the 5% level. Plasma volume changes did not differ significantly between trials and the unadjusted values are reported. Results are given as mean \pm SEM.

RESULTS

Exercise responses

The total weight lifted during the 90 min resistance exercise session was $10,568 \pm 621$ kg. The gross energy expenditure from resistance exercise was estimated to be 1473 ± 114 kJ. The mean percentage of maximum oxygen uptake elicited during aerobic exercise was $69 \pm 2\%$ and the mean respiratory exchange ratio (RER) was 0.92 ± 0.01 . Average heart rate during running was 167 ± 3 beats/min and the median rating of perceived exertion (RPE) was 15 i.e. 'hard' (range 13-17). Gross energy expenditure during aerobic exercise was 3832 ± 97 kJ with $27 \pm 4\%$ of energy provided from fat and $73 \pm 4\%$ of energy provided from carbohydrate. For comparison gross energy expenditure during the first hour of the control trial was 363 ± 24 kJ, the mean RER value during this time was 0.84 ± 0.03 with $47 \pm 11\%$ of energy provided from fat and $53 \pm 4\%$ of energy provided from carbohydrate. Energy expenditure during running was higher than energy expenditure in resistance exercise which in turn was higher than energy expenditure during an equivalent (90 min) period of rest during the control trial ($P < 0.0005$ for each).

Hunger

Figure 1 displays the delta (difference from baseline) scores for hunger on the three trials (top panel) and the raw scores for hunger (bottom panel). Fasting hunger did not differ significantly between trials. There was an effect of trial ($P < 0.037$), an effect of time ($P < 0.001$) and a trial \times time interaction ($P < 0.001$) for hunger indicating that responses differed over time between trials.

On the control trial hunger increased prior to the first test meal. In response to consuming the first

test meal ($t = 2$ h) hunger scores fell and returned to baseline just prior to the second test meal ($t = 5$ h). Post-second meal hunger scores decreased and remained suppressed until the end of the study period ($t = 8$ h).

Hunger scores were reduced by resistance exercise and this reduction became significant at the 0.75 h time point compared with the control trial (top panel on Figure 1). After exercise hunger scores increased but remained suppressed compared with the control trial in the pre-meal interval. However, after consumption of the test meal no further differences between the resistance and control trials were apparent.

Hunger scores were reduced by aerobic exercise from the first time point assessed during exercise ($t = 0.5$ h) throughout the exercise period. After exercise hunger scores increased in the pre-meal interval but remained significantly suppressed compared with the control trial until initiation of the first test meal ($t = 2$ h). After consumption of the first test meal there were no differences between control and aerobic exercise trials.

Aerobic exercise resulted in a greater suppression of hunger than resistance exercise at 0.75 h and 1 h. Calculation of the AUC for hunger for the pre-prandial period (0 to 2 h) revealed that aerobic exercise significantly reduced hunger in comparison with the control trial (Table 1). No significant trial differences were observed when AUC values for the entire study period were assessed.

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Plasma acylated ghrelin and total PYY

Fasting acylated ghrelin concentrations did not differ significantly between trials. There was an effect of time ($P < 0.001$) and a trial \times time interaction ($P = 0.035$) for acylated ghrelin indicating that compared with the control trial, values were suppressed at 0.75 h and 1.5 h in the resistance exercise trial and at 0.75 h in the aerobic exercise trial (Figure 2). Pre-prandial (0 to 2 h) AUC values were significantly lower during the resistance exercise trial than the control trial (Table 1).

Fasting PYY concentrations did not differ significantly between trials. There was a main effect of trial ($P = 0.002$), a main effect of time ($P < 0.0005$) and a trial \times time interaction ($P = 0.029$) for PYY indicating higher values on the aerobic exercise trial than both the control ($P = 0.020$) and resistance exercise trials ($P = 0.017$) (Figure 2). These findings were confirmed when analysing AUC values (Table 1). There were no significant differences between the control and resistance exercise trials.

INSERT FIGURE 2 NEAR HERE

Glucose and insulin

Fasting plasma glucose concentrations did not differ significantly between trials. There was a main effect of trial, a main effect of time and a trial \times time interaction (all $P < 0.0005$) for glucose indicating higher values on the aerobic exercise trial than both the control trial ($P = 0.025$) and the resistance exercise trial ($P = 0.003$) (Figure 3, bottom panel). These findings were confirmed by analysis of the AUC values for glucose (Table 1).

Fasting plasma insulin concentrations did not differ significantly between trials. There was a main effect of time ($P < 0.0005$) but no significant trial or interaction effects (Figure 3, top panel). Pre-prandial (0 to 2 h) AUC values were higher on the resistance exercise trial than the control trial. There were no other significant differences when comparing insulin AUC values (Table 1).

INSERT FIGURE 3 NEAR HERE

Correlations

Baseline plasma acylated ghrelin and PYY concentrations were not significantly correlated with BMI, waist circumference, maximum oxygen uptake, fasting hunger, fasting glucose concentration or fasting insulin concentration. Acylated ghrelin and PYY concentrations at other time points were not consistently correlated with each other or with corresponding hunger, glucose and insulin values.

DISCUSSION

This study demonstrates that: 1) hunger is suppressed during and for a short while after

resistance and aerobic exercise, 2) acylated ghrelin is suppressed during resistance and aerobic exercise, 3) PYY is increased during and after aerobic exercise. In particular the suppression of hunger and acylated ghrelin during resistance exercise and the increase in PYY for a prolonged period after aerobic exercise are novel findings.

The finding that hunger is suppressed during and immediately after vigorous treadmill running is consistent with previous studies indicating that strenuous (around 60% of maximum oxygen uptake and above) aerobic exercise transiently suppresses appetite (6, 9, 29, 39). The hunger response to resistance exercise has not previously been examined and the present findings suggest a similar although slightly attenuated response in comparison with vigorous running. One possible explanation for this attenuation is the lower energy expenditure during resistance exercise. Another possibility is that the attenuated responses are due to the intermittent nature of resistance exercise and the lower gut disturbance compared with running.

The current study confirms our previous findings that treadmill running suppresses acylated ghrelin and extends them by demonstrating acylated ghrelin suppression during resistance exercise. It is perhaps surprising that acylated ghrelin concentrations were not elevated towards the end of the exercise trials since energy intake was not increased in these trials to compensate for the energy expended during exercise. These data are consistent with the recent finding that post-exercise ghrelin responses may be independent of energy balance (22) and lend support to previous research indicating that acute exercise does not increase energy intake in the short term i.e. one to two days after exercise (6, 7, 24, 28, 29). It would be of interest to examine acylated ghrelin concentrations the day after exercise to assess whether values are elevated in response to a short term negative energy balance.

Only one previous study has examined the PYY response to exercise (39). This study observed elevations in PYY during a one-hour cycling bout. These elevations were not maintained post-exercise. In the present study PYY concentrations were increased significantly during treadmill running. Moreover, after cessation of exercise total plasma PYY

concentrations remained elevated prior to consuming the first meal and following meal ingestion. By the end of the observation period, however, PYY concentrations did not differ among the three trials. Although PYY was not elevated post-exercise in the study of Martins and colleagues (39) they did observe a transient elevation in GLP-1 after exercise. Another recent study (12) has demonstrated an increased GLP-1 response to feeding after five consecutive days of aerobic exercise (1 h/day). Collectively these findings suggest that aerobic exercise exerts a transient, hormone mediated, inhibition of appetite.

The lack of change in PYY in response to weight lifting is perplexing in light of the change in acylated ghrelin with weight lifting. It is possible that the energy expenditure induced by weight lifting was insufficient to evoke a change in PYY. Alternatively, the lack of gut upheaval and/or a lower perception of stress during weight lifting in comparison with hard continuous running may be an explanation. A limitation of the present study was that total PYY was measured rather than PYY₃₋₃₆. The majority of studies examining circulating PYY have reported total PYY levels using assays which detect both the PYY₁₋₃₆ and PYY₃₋₃₆ (5, 34, 35, 37, 38). Currently there is only one assay which is specific for PYY₃₋₃₆ form and this requires the addition dipeptidyl peptidase IV (DPP-IV) inhibitor to the blood. As we did not add DPP-IV inhibitor we are unable to measure PYY₃₋₃₆. However, we and others have previously shown that PYY₃₋₃₆ is the predominant form both in the fed and fasted states and in lean and obese subjects (4, 30). Moreover, we have shown a high positive correlation ($r = 0.98$, $P < 0.001$) between total PYY and PYY₃₋₃₆ (49). Whilst future studies need to be undertaken with DPP-IV inhibitor added to enable the assessment of PYY₃₋₃₆ available evidence suggests that total PYY measurements reflect changes in PYY₃₋₃₆.

Glucose and insulin were measured in the present study because they may interact with ghrelin and PYY. The glucose elevation observed during aerobic exercise might explain the suppression of acylated ghrelin (46) but glucose was not elevated when ghrelin was suppressed in resistance exercise. In the resistance exercise trial, an elevation in pre-prandial insulin coincided with a decline in pre-

prandial ghrelin supporting a regulatory role for insulin (18). Further research is required to determine the true significance of these findings.

PERSPECTIVES AND SIGNIFICANCE

Previous studies have shown that aerobic exercise can cause a transient suppression of appetite that lasts from several hours to two or more days. The mechanism for this effect is unknown and the effects of resistance exercise on appetite are uncertain. The present findings confirm a transient (1 to 2 h) suppression of appetite during and after aerobic and resistance exercise. The findings suggest that ghrelin may mediate this suppression for both forms of exercise. There was an elevation in PYY during and after aerobic exercise and this may possibly contribute to appetite suppression. Further research is required to determine how long exercise induced changes in gut hormones persist and whether the changes have any effect on energy intake. A better understanding of the role of exercise in appetite regulation may lead to a more effective prescription of exercise for weight control.

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DISCLOSURE

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Table 1: Area under the curve values for hunger, plasma acylated ghrelin, total PYY, glucose and insulin (mean \pm SEM). Findings were analysed using one-way analysis of variance and Bonferroni post-host tests.

	Pre-prandial 0 to 2 h	Postprandial 2 to 5 h	Postprandial 5 to 8 h	Total 0 to 8 h
Hunger	(units 2 h)	(units 3 h)	(units 3 h)	(units 8 h)
Control	19 \pm 2	19 \pm 3	13 \pm 2	51 \pm 6
Resistance	12 \pm 2	16 \pm 2	12 \pm 2	40 \pm 5
Aerobic	9 \pm 1 ^a	16 \pm 3	14 \pm 3	39 \pm 6
Ghrelin	(pg/mL 2 h)	(pg/mL 3 h)	(pg/mL 3 h)	(pg/mL 8 h)
Control	228 \pm 62	304 \pm 95	279 \pm 101	811 \pm 257
Resistance	169 \pm 55 ^a	258 \pm 67	269 \pm 75	696 \pm 196
Aerobic	188 \pm 68	287 \pm 102	261 \pm 101	736 \pm 270
PYY	(pg/mL 2 h)	(pg/mL 3 h)	(pg/mL 3 h)	(pg/mL 8 h)
Control	229 \pm 29	459 \pm 44	724 \pm 55	1411 \pm 110
Resistance	232 \pm 43	498 \pm 35	651 \pm 47	1381 \pm 97
Aerobic	324 \pm 54 ^a	663 \pm 79 ^a	763 \pm 59	1750 \pm 170 ^{ab}
Glucose	(mmol/L 2 h)	(mmol/L 3 h)	(mmol/L 3 h)	(mmol/L 8 h)
Control	9.9 \pm 0.2	15.5 \pm 0.3	15.1 \pm 0.4	40.5 \pm 0.7
Resistance	9.7 \pm 0.2	11.9 \pm 0.5	14.9 \pm 0.5	38.8 \pm 1.1
Aerobic	10.5 \pm 0.4 ^b	16.5 \pm 0.4 ^{ab}	16.1 \pm 0.4	43.1 \pm 0.9 ^{ab}
Insulin	(pmol/L 2 h)	(pmol/L 3 h)	(pmol/L 3 h)	(pmol/L 8 h)
Control	39 \pm 5	272 \pm 36	258 \pm 21	568 \pm 45
Resistance	58 \pm 7 ^a	288 \pm 26	220 \pm 49	565 \pm 60
Aerobic	46 \pm 5	289 \pm 25	237 \pm 23	572 \pm 46

Notes: Aerobic exercise was performed for the first hour of the pre-prandial period (0-1 h); resistance exercise was performed for the first 90 minutes of the pre-prandial period (0-1.5 h); the units are area under the curve values over 2 hours, 3 hours, 3 hours and 8 hours respectively for columns 1 to 4.

^a Different from control $P < 0.05$

^b Different from resistance $P < 0.05$

LEGENDS FOR FIGURES

FIGURE 1

Delta (i.e. change from baseline) hunger scores (top panel) and absolute hunger scores (bottom panel) during the three trials (mean \pm SEM, $n = 11$). Lightly shaded rectangle indicates the treadmill run, open rectangle indicates weight lifting, black rectangles indicate consumption of the test meals.

^a Control different from aerobic exercise $P < 0.05$, ^b control different from resistance exercise $P < 0.05$,

^c aerobic exercise different from resistance exercise $P < 0.05$. Error bars are omitted from some trials for clarity.

FIGURE 2

Plasma concentrations of acylated ghrelin (top panel) and total PYY (bottom panel) during the three trials (mean \pm SEM, $n = 11$). Lightly shaded rectangle indicates the treadmill run, open rectangle indicates weight lifting, black rectangles indicate consumption of the test meals. ^a Control different from aerobic exercise $P < 0.05$, ^b control different from resistance exercise $P < 0.05$, ^c aerobic exercise different from resistance exercise $P < 0.05$. Error bars are omitted from some of trials for clarity.

FIGURE 3

Plasma concentrations of insulin (top panel) and glucose (bottom panel) during the three trials (mean \pm SEM, $n = 11$). Lightly shaded rectangle indicates the treadmill run, open rectangle indicates weight lifting, black rectangles indicate consumption of the test meals. ^a Aerobic exercise different from control $P < 0.05$, ^b Aerobic exercise different from resistance exercise $P < 0.05$. Error bars are omitted from some trials for clarity.

Figure 1

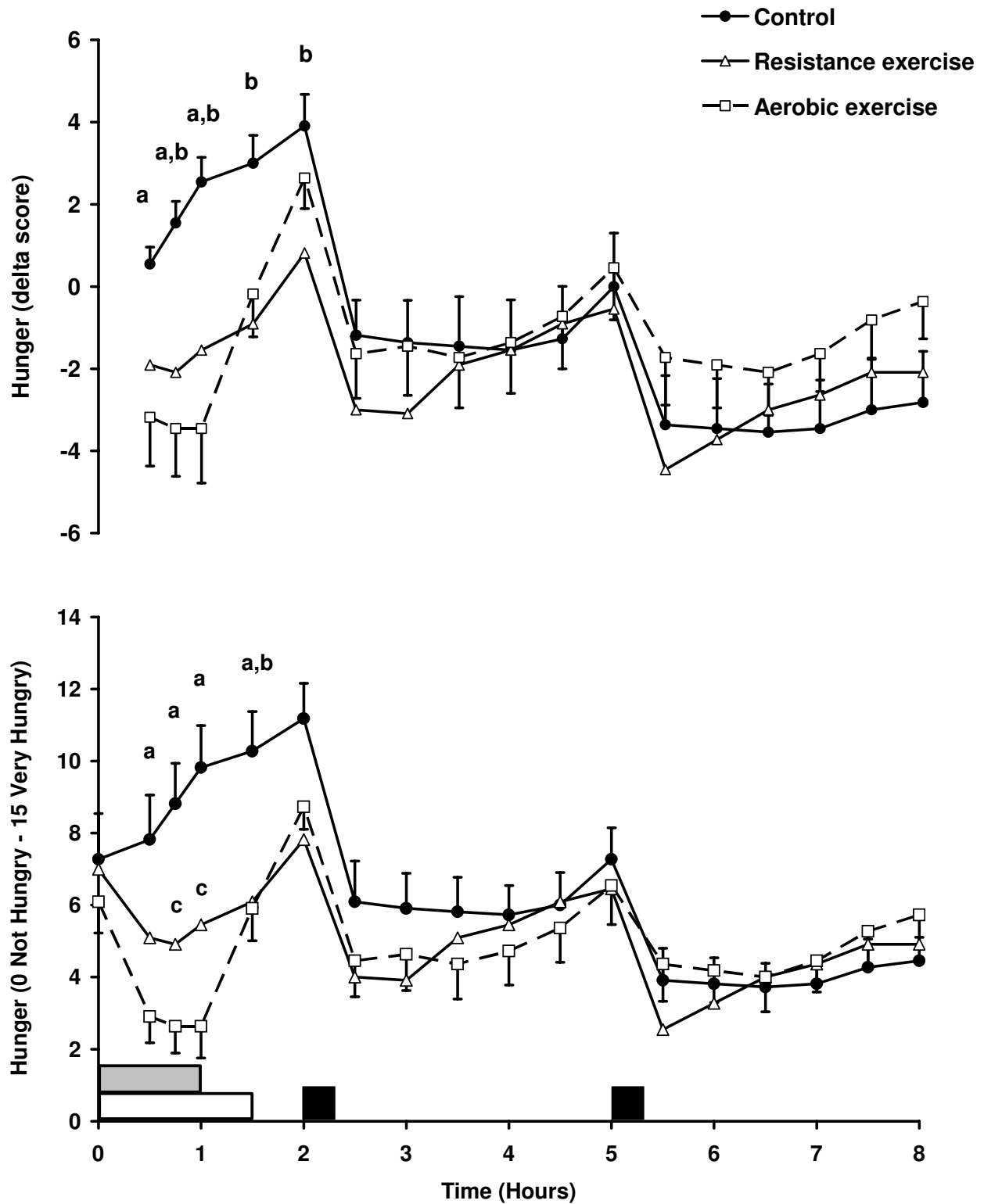


Figure 2

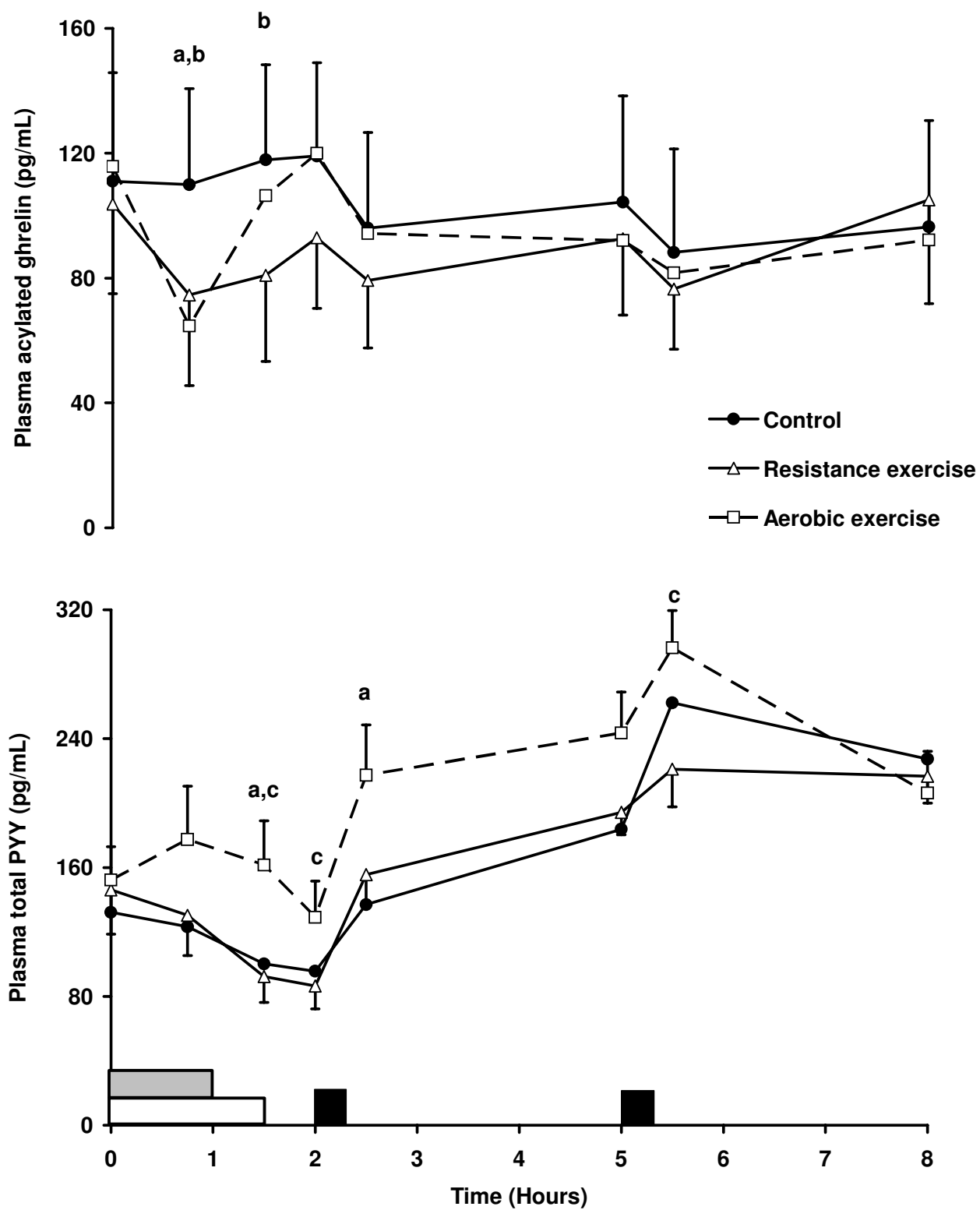


Figure 3

