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

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Broom DR, Batterham RL, King JA, Stensel DJ. Influence of resistance and aerobic exercise on hunger, circulating levels of acylated ghrelin and peptide YY (PYY). Eleven healthy male subjects: age 21.1 ± 0.3 yr, body mass index 23.1 ± 0.4 kg/m², maximum oxygen uptake 62.1 ± 1.8 ml·kg⁻¹·min⁻¹ (means ± SE) 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: a 8-h rest, in a randomized 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 1,411 ± 110, resistance 1,381 ± 97, aerobic 1,750 ± 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.

APPENDIX

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 is controversial, with studies reporting no changes either during or postexercise (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/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 standardized breakfast and 1 h 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 postexercise 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

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aerobic exercise on meal-stimulated changes in hunger, acylated ghrelin, and total PYY at two time points (2 h and 5 h postexercise) 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 yr gave their written informed consent to participate. Subjects were nonsmokers, not taking any medication, weight-stable for 3 mo prior to the study, and had no food allergies. The physical characteristics of the subjects were age, 21.1 ± 0.3 yr; body mass index (BMI), 23.1 ± 0.4 kg/m²; waist circumference, 78.5 ± 1.1 cm; and maximum oxygen uptake, 62.1 ± 1.5 ml·kg⁻¹·min⁻¹ (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 familiarized 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 familiarization session in which they completed a full weight-lifting session: three sets of 12 repetitions of 10 different weight-lifting exercises at 80% of session in which they completed a full weight-lifting session: three sets of 12 repetitions of 10 different weight-lifting exercises at 80% of maximum oxygen uptake. On completion of the 12 repetitions, subjects 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)l 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 (~1100 and 1400, 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 3,230 kJ for a 70-kg person. The amount of food consumed was adjusted for each participant based on their body weight 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, 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 precooled 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. 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 while subjects lay in a semisupine 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 1,681 g (4,000 revs/min) for 10 min in a refrigerated centrifuge (Burkard, Hertfordshire, UK) at 4°C. The plasma supernatant was transferred to 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 1,287 g (3,500 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 milliliter of plasma. Samples were then spun at 1,287 g (3,500 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 hemoglobin concentration, and triplicate blood samples were collected into heparinized microhematocrit tubes for the determination of hematocrit. Hemoglobin and hematocrit values were used to assess plasma volume changes (16).

**Blood Biochemistry**

To eliminate interassay variation, samples from each participant were analyzed in the same run. Plasma acylated ghrelin concentrations were determined by 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, Webster, TX). The within-batch CV was 1.2%. Plasma insulin concentrations were determined by ELISA (Merckodia, Uppsala, Sweden). The within-batch CV was 3.5%. Plasma glucose concentrations were determined by enzymatic, colorimetric methods (Randox Laboratories, 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 ver. 14.0 for Windows (SPSS, Chicago, IL). 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 means ± SE.

**RESULTS**

**Exercise Responses**

The total weight lifted during the 90-min resistance exercise session was 10,568 ± 621 kg. The gross energy expenditure from resistance exercise was estimated to be 1,473 ± 114 kJ. The mean percentage of maximum oxygen uptake elicited during aerobic exercise was 69 ± 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 perceived exertion received a median rating of 15, i.e., “hard” (range 13–17). Gross energy expenditure during aerobic exercise was 3,832 ± 97 kJ, with 27 ± 4% of energy provided from fat and 73 ± 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 ± 11% of energy provided from fat and 53 ± 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) and the raw scores for hunger (bottom). 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 × 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 (Fig. 1, top). After exercise, hunger scores increased but remained suppressed compared with the control trial in the premeal 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 premeal 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 preprandial period (0 to 2 h) revealed that aerobic exercise significantly reduced hunger compared with the control trial (Table 1). No significant trial differences were observed when AUC values for the entire study period were assessed.

**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 × 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 (Fig. 2). Preprandial (0 to 2 h) AUC values were significantly lower during the resistance exercise trial than both the control trial (Fig. 2). Preprandial (0 to 2 h) AUC values were confirmed when analyzing AUC values (Table 1). There were no significant differences between the control and resistance exercise trials.

**Table 1. Area under the curve values for hunger, plasma acylated ghrelin, total PYY, glucose, and insulin**

<table>
<thead>
<tr>
<th></th>
<th>Preprandial 0 to 2 h</th>
<th>Postprandial 2 to 5 h</th>
<th>Postprandial 5 to 8 h</th>
<th>Total 0 to 8 h</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hunger</strong></td>
<td>(units 2 h)</td>
<td>(units 3 h)</td>
<td>(units 3 h)</td>
<td>(units 8 h)</td>
</tr>
<tr>
<td>Control</td>
<td>19±2</td>
<td>19±3</td>
<td>12±2</td>
<td>51±6</td>
</tr>
<tr>
<td>Resistance</td>
<td>12±2</td>
<td>16±2</td>
<td>12±2</td>
<td>40±5</td>
</tr>
<tr>
<td>Aerobic</td>
<td>9.5±1*</td>
<td>16±3</td>
<td>14±3</td>
<td>39±6</td>
</tr>
<tr>
<td><strong>Ghrelin</strong></td>
<td>(pg/ml 2 h)</td>
<td>(pg/ml 3 h)</td>
<td>(pg/ml 3 h)</td>
<td>(pg/ml 8 h)</td>
</tr>
<tr>
<td>Control</td>
<td>228±62</td>
<td>304±95</td>
<td>279±101</td>
<td>811±257</td>
</tr>
<tr>
<td>Resistance</td>
<td>169±55*</td>
<td>258±67</td>
<td>269±75</td>
<td>696±196</td>
</tr>
<tr>
<td>Aerobic</td>
<td>188±68</td>
<td>287±102</td>
<td>261±101</td>
<td>736±270</td>
</tr>
<tr>
<td><strong>PYY</strong></td>
<td>(pg/ml 2 h)</td>
<td>(pg/ml 3 h)</td>
<td>(pg/ml 3 h)</td>
<td>(pg/ml 8 h)</td>
</tr>
<tr>
<td>Control</td>
<td>232±43</td>
<td>498±44</td>
<td>724±55</td>
<td>1411±110</td>
</tr>
<tr>
<td>Resistance</td>
<td>324±54*</td>
<td>663±79*</td>
<td>761±59</td>
<td>1750±170†</td>
</tr>
<tr>
<td>Aerobic</td>
<td>105±0.4†</td>
<td>16.5±0.4†</td>
<td>16.1±0.4</td>
<td>43.1±0.9†</td>
</tr>
<tr>
<td><strong>Glucose</strong></td>
<td>(mmol/l 2 h)</td>
<td>(mmol/l 3 h)</td>
<td>(mmol/l 3 h)</td>
<td>(mmol/l 8 h)</td>
</tr>
<tr>
<td>Control</td>
<td>9.9±0.2</td>
<td>15.5±0.3</td>
<td>15.1±0.4</td>
<td>40.5±0.7</td>
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<tr>
<td>Resistance</td>
<td>9.7±0.2</td>
<td>11.9±0.5</td>
<td>14.9±0.5</td>
<td>38.8±1.1</td>
</tr>
<tr>
<td>Aerobic</td>
<td>39±5</td>
<td>272±36</td>
<td>258±21</td>
<td>568±45</td>
</tr>
<tr>
<td><strong>Insulin</strong></td>
<td>(pmol/l 2 h)</td>
<td>(pmol/l 3 h)</td>
<td>(pmol/l 3 h)</td>
<td>(pmol/l 8 h)</td>
</tr>
<tr>
<td>Control</td>
<td>58±7</td>
<td>288±26</td>
<td>220±49</td>
<td>585±60</td>
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<tr>
<td>Resistance</td>
<td>46±5</td>
<td>289±25</td>
<td>237±23</td>
<td>572±46</td>
</tr>
</tbody>
</table>

Values are expressed as means ± SE. Findings were analyzed using one-way ANOVA and Bonferroni post hoc tests. PYY, peptide YY. Aerobic exercise was performed for the first hour of the preprandial period (0–1 h); resistance exercise was performed for the first 90 min of the preprandial period (0–1.5 h); the units are area under the curve values over 2 h, 3 h, 3 h and 8 h, respectively, for columns 1 to 4. *Different from control $P < 0.05$. †Different from resistance $P < 0.05$.

**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 × 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 ($P = 0.003$) (Fig. 3, bottom). 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 (Fig. 3, top). Preprandial (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).

**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
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 toward 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 postexercise ghrelin responses may be independent of energy balance (22) and lend support to previous research that indicated acute exercise does not increase energy intake in the short term, i.e., 1 to 2 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 1-h cycling bout. These elevations were not maintained postexercise. 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 postexercise 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 compared 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 that is specific for PYY3-36 form, and this requires the addition dipeptidyl peptidase IV (DPPIV) inhibitor to the blood. As we did not add DPPIV 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). While future studies need to be undertaken with DPPIV 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 preprandial insulin 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, and 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 compared 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.

**Fig. 3.** Plasma concentrations of insulin (top) and glucose (bottom) during the three trials (mean ± SE, n = 11). Lightly shaded rectangle indicates the treadmill run, open rectangle indicates weight lifting, black rectangles indicate consumption of the test meals. *Aerobic exercise different from control treadmill run, open rectangle indicates weight lifting, black rectangles indicate consumption of the test meals. *Aerobic exercise different from control treadmill run, open rectangle indicates weight lifting, black rectangles indicate consumption of the test meals. **Aerobic exercise different from control treadmill run, open rectangle indicates weight lifting, black rectangles indicate consumption of the test meals. *Aerobic exercise different from control treadmill run, open rectangle indicates weight lifting, black rectangles indicate consumption of the test meals. **Aerobic exercise different from control treadmill run, open rectangle indicates weight lifting, black rectangles indicate consumption of the test meals. *Aerobic exercise different from control treadmill run, open rectangle indicates weight lifting, black rectangles indicate consumption of the test meals. **Aerobic exercise different from control treadmill run, open rectangle indicates weight lifting, black rectangles indicate consumption of the test meals. **Aerobic exercise different from control treadmill run, open rectangle indicates weight lifting, black rectangles indicate consumption of the test meals. **Aerobic exercise different from control treadmill run, open rectangle indicates weight lifting, black rectangles indicate consumption of the test meals.
coincided with a decline in preprandial 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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