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Clinical Study

Influence of Running and Walking on Hormonal Regulators of Appetite in Women

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Nine female runners and ten walkers completed a 60 min moderate-intensity (70% VO2max) run or walk, or 60 min rest in counterbalanced order. Plasma concentrations of the orexogenic peptide ghrelin, anorexogenic peptides peptide YY (PYY), glucagon-like peptide-1 (GLP-1), and appetite ratings were measured at 30 min interval for 120 min, followed by a free-choice meal. Both orexogenic and anorexogenic peptides were elevated after running, but no changes were observed after walking. Relative energy intake (adjusted for cost of exercise/rest) was negative in the meal following running (−194 ± 206 kcal) versus walking (41 ± 196 kcal) (P = 0.015), although both were suppressed (P < 0.05) compared to rest (299 ± 308 and 284 ± 121 kcal, resp.). The average rate of change in PYY and GLP-1 over time predicted appetite in runners, but only the change in GLP-1 predicted hunger (P = 0.05) in walkers. Results provide evidence that exercise-induced alterations in appetite are likely driven by complex changes in appetite-regulating hormones rather than change in a single gut peptide.

1. Introduction

The benefits of exercise in the prevention of chronic diseases including overweight and obesity are well documented. Regular physical activity reduces blood pressure, creates a more favorable lipid profile, and reduces risk for stroke, coronary heart disease, hypertension, and colon cancer [1, 2]. Regular exercise also helps maintain healthy body weight [1] and may aid in weight loss and weight loss maintenance [3]. To help prevent weight gain (or obesity), the 2008 Physical Activity Guidelines for Americans [4], sponsored by the Centers for Disease Control and Prevention and Healthy People 2020, suggests incorporating a minimum weekly total of two and a half hours of moderate-to-vigorous intensity physical activity, spread over most days of the week. Working up to five or more hours per week (−60 min/day) is recommended to gain additional benefits which include weight loss and weight loss maintenance.

Although the aforementioned recommendations, if followed, are likely to have a major impact on health, intervention studies find that exercise without intentional food restriction and/or behavior modification does not effectively promote weight loss, [5, 6], particularly in women [7, 8]. This may be because exercise stimulates a compensatory (relative to the energy expenditure of the activity) or noncompensatory drive to eat that is either biologically—(i.e., altered appetite regulating hormones) or psychologically—(i.e., feeling one deserves dessert after exercising) driven. These studies, however, are not consistent with short-term experimental studies conducted mostly in men which have found reductions in appetite and relative food intake following moderately intense-to-vigorous exercise. This may be because the exercise-induced effect is influenced by factors including the intensity and mode of the exercise [9–11], the sex, and body composition of the exerciser [9, 10]. Several previous studies found that hunger and/or food intake are
suppressed following 30–90 min of intense- but not necessarily light-to-moderate intensity exercise [12–18] including cycling, running, and brisk walking. Others reported increases in hunger and food intake following swimming [19] and exercise calisthenics [20]. Less is known concerning individual differences; however, one study found suppressed hunger and food intake in lean but not overweight women following bicycle exercise [21].

The recent discovery of several gut peptides involved in appetite regulation and energy homeostasis provides an attractive mechanism to explain how exercise reduces hunger/appetite in some conditions and increases it in others. Alterations in circulating ghrelin, the only known orexigenic gut peptide, along with the anorexigenic gut peptides peptide YY (PYY) and glucagon-like peptide-1 (GLP-1) may work in concert to influence exercise-associated alterations in hunger and food intake. Alterations in circulating gut peptides appear to regulate food intake for as long as 24 h and are not specifically controlled by body fat stores. A number of previous studies have found that these peptides are altered by an acute bout of exercise [16, 17, 22–28]; however, the majority of studies evaluated only a single mode of exercise compared to rest. In addition, only a few of these studies simultaneously evaluated both the orexogenic and anorexogenic gut peptides [17, 27, 28], and few included women [23, 27, 28].

The purpose of this study was to assess the effect of a 60-minute bout of exercise on circulating concentrations of gut peptides ghrelin, PYY, and GLP-1; appetite and ad libitum food intake among women. An additional purpose was to assess whether alterations in these gut peptides were associated with alterations in appetite following exercise. Exercise was performed at a moderately hard intensity in two different modes: running and walking. We hypothesized that circulating ghrelin would be suppressed; PYY and GLP-1 concentrations elevated following both modes of exercise compared to rest. Furthermore, we hypothesized that ghrelin concentration would be directly correlated with ratings of hunger and desire to eat and PYY and GLP-1 concentrations would be indirectly correlated.

2. Methods

Nine endurance-trained female runners and ten habitual walkers between the ages of 18–40 were recruited for the study. To qualify, participants had to be in good general health, have normal hemoglobin (between 14.0–18.0 mg/dL) and thyroid status (thyroid stimulating hormone between 0.40–4.50 mlU/L), have regularly occurring menstrual cycles, and be of “low exercise risk” as per the American College of Sports Medicine (ACSM) [29]. The runners had to be currently running at least 32 km/wk, be performing runs of at least 60 min in duration as part of their training regimen, and have maximal aerobic capacity (VO$_{2\text{max}}$) of at least 45 mL/kg/min. The walkers had to be performing walks of at least 60 min in duration three or more days/wk and have a VO$_{2\text{max}}$ of less than 40 mL/kg/min. Participants were excluded if they smoked, were anemic, hyper-or hypo-thyroid, pregnant or postmenopausal, had renal, hepatic, endocrine, gastrointestinal, pulmonary, cardiac, or hematological diseases including high blood pressure (>120/80 mm/Hg at rest), prediabetes/diabetes, demonstrated signs of significant depression, anxiety, other psychological problems, alcoholism or other substance abuse, used prescription or over the counter medications (other than contraceptives), or herbal preparations that can influence metabolism, had food allergies, or were unwilling to consume all foods/beverages provided in the run-in diet. The study was approved by the Institutional Review Board of the University of Wyoming. Volunteers were fully informed of possible risks of all procedures before providing written informed consent.

2.1. Baseline Testing. Approximately two weeks before initiation of the experimental protocol, VO$_{2\text{max}}$ was determined on a motor-driven treadmill (Trackmaster TMX22, Newton, KS, USA) in accordance with ACSM recommendations [29]. For most runners, testing was initiated at 6 mph (0% grade) with the grade increasing by 1% every min until exhaustion. For the walkers, the test was initiated at 3.5 mph (2% grade) with grade increasing by 1% every min until exhaustion. Oxygen consumption (VO$_{2}$) and carbon dioxide production (VCO$_{2}$) were measured continuously using a metabolic cart (ParvoMedics TuneOne 2400, Sandy, UT, USA), and heart rate (HR) was monitored by an electrocardiography machine (Quinton Q-5000, Bothell, WA, USA). Rating of perceived exertion (RPE) was assessed during the last 10 seconds of each stage using the modified Borg Scale [29]. The highest 20-second VO$_{2}$ and respiratory exchange ratio (RER) achieved in the final two min of exercise were recorded as the maximum values. To qualify as an acceptable maximum test, participants had to meet two of the four following criteria: (1) a leveling or plateau of VO$_{2}$ (defined as an increase of <2 mL·kg$^{-1}$·min$^{-1}$ with increased workload); (2) RER ≥ 1.10; (3) maximum heart rate within 10 beats of age predicted maximum [208 – (0.7 × age)] [30]; (4) rating of perceived exertion (RPE) ≥ 17. After a 30 min recovery period, participants underwent a titration run/walk to determine the speed and grade required to elicit an oxygen uptake of 70% VO$_{2}\text{max}$. For descriptive purposes, body composition was measured using dual-energy X-ray absorptiometry (DEXA, GE Lunar Prodigy 8743, Waukesha, WI, USA).

2.2. Experimental Protocol. The study was a counterbalanced, cross-over study where participants completed an exercise and control (rest) test day. A schematic of the study is shown in Figure 1. The two test trials were scheduled in the follicular phase of the participants’ menstrual cycle (between days 1 and 11) and spaced either 2 to 10 days or 1 menstrual cycle (3 to 5 wks) apart. The exercise test day consisted of a 60 min run/walk at 70% VO$_{2}\text{max}$ followed by 2 h of rest, whereas the control day consisted of 3 h of rest. Food intake was controlled for 24 h prior to each test day by providing participants with a controlled diet. The diet provided 2000 kcal (64% carbohydrate, 14% protein, and 22% fat) from commercially available foods and beverages plus an optional additional 200 kcal provided as two 100 kcal snack bars (28.4 g, ~100 kcals; Clif Bar and Company, Berkeley, CA, USA).
Volunteers were asked to consume the foods provided (and nothing in addition other than water) and to return empty wrappers and any food and beverages that could not be consumed.

2.3. Test Days. On both test days, participants consumed a standard breakfast (Boost Smoothie, Clif Builder Bar and 2 cups of water; ~380 kcal; 65% carbohydrate, 20% protein, 15% fat) at 0630 prior to arriving in the laboratory. At 0730, height, weight, and blood pressure were measured. On the control visit, an intravenous indwelling (IV) catheter was inserted into an arm or hand vein and connected to a normal saline solution (0.9% saline solution) that was slowly infused to keep the catheter patent. Blood was drawn immediately before (baseline, preexercise) and immediately after one h of rest (t = 0 min) and every 30 min thereafter for 2 h (t = 30, 60, 90, 120 min). The first 3 cc at each time point draw was presumed to be diluted with saline and was discarded. Resting energy expenditure (REE) and respiratory quotient (RQ) were measured using a metabolic cart (800 lpm pneumatach, ParvoMedics, TrueOne 2400, Sandy, UT, USA) while the subject lay motionless in the supine position, as previously described [31]. The last 20 min of data was used to calculate REE.

On the exercise day, baseline blood was obtained by venipuncture. An IV catheter was inserted immediately following the 60 min run/walk, and blood was drawn on the same schedule as the rest day. For 10 min at the beginning (between 5–15 min after the start) and end (last 5 min) of the run/walk session, VO2, VCO2, RER and RPE, were measured using a metabolic cart (800 lpm pneumatach, ParvoMedics, TrueOne 2400, Sandy, UT, USA). Exercise pace was adjusted, if necessary, during the first 10 min to achieve an oxygen cost as close to 70% VO2max as possible but was not further adjusted. HR was monitored continuously using a portable heart rate monitor (Polar S60i, Polar, Port Washington, NY, USA).

On both test days, hunger and satiety were assessed using 100 mm visual analogue scales (VASs), anchored at each end with a word describing the extremes of the appetite being measured [32]. The scales specifically asked (1) how hungry do you feel? (2) how satisfied do you feel? (3) how full do you feel? (4) how much do you think you can eat? Hunger and satiety ratings were obtained 4 to 5 min before each blood draw (t = 0, 30, 60, 90, and 120) and at 20 min after initiation of the ad libitum meal (see below).

At completion of the exercise and rest sessions (120 min), participants were offered an ad libitum, free-choice meal. The free-choice meal consisted of the following (in weighed portions) attractively and consistently arranged on the dining table: rigatoni pasta (140 g dry, cooked), marinara sauce (140 g), alfredo sauce (140 g), whole-wheat bread (2 slices), white bread (2 slices), hard boiled eggs (2), apples (2), oranges (2), Clif snack bar (2), Clif Builder bar (2), nonfat yogurt (1, 6 oz), regular yogurt (1, 6 oz), individual portions of margarine (4), honey (4), peanut butter (4), assorted jellies (4), lemon-lime Gatorade (2, 20 oz), 2% milk (2, 8 oz), and water (~1500 g). Participants were given 20 min to eat the meal and were instructed to eat until satiety. Participants were not allowed to read or study during the meal or carry backpacks, purses, or coats into the room. They were discretely monitored by the same investigator who worked quietly on a computer in the back of the room (with their back turned toward the participant). Food and water consumption were determined by weighing remaining food (to the nearest 0.1 g) at cessation of eating. By difference, food/beverages consumed were analyzed for total energy, protein, fat, carbohydrate, simple sugars, and fiber using Nutritionist Pro (Axxya Systems, Stafford, TX, USA, 77477). Ad libitum water intake and intake of energy and macronutrients as solids and liquids were also assessed. Relative food intake was calculated by subtracting estimated energy expenditure during the exercise (60 min exercise, 120 min rest) or rest (180 min rest) sessions from the respective free-choice energy intake.

2.4. Blood Samples and Hormone Analysis. Blood samples taken both before exercise and rest (baseline) were analyzed for serum concentrations of progesterone concentration.
using a human solid phase RIA kit (Siemens Diagnostics, Los Angeles, CA, USA). Plasma samples were analyzed for total ghrelin, acylated ghrelin (ghrelin_{acyl}), PYY_{3-36}, GLP-1, glucose, lactate and hematocrit at pre-exercise/rest and for 120 min following exercise and rest were analyzed for total ghrelin, acylated ghrelin (ghrelin_{acyl}), PYY_{3-36}, GLP-1, glucose, lactate, and hematocrit. Blood samples for total ghrelin, ghrelin_{acyl}, PYY_{3-36}, and GLP-1 were collected into EDTA-treated prechilled tubes. Blood collected for analysis of plasma ghrelin_{acyl} was collected into a chilled tube containing 100 µL of 200 mM AEBSF with 200 µL of 1 N HCl added per mL of plasma following centrifugation. Samples collected for analysis of PYY were treated with 150 µL of aprotinin and 40 µL DFP-IV. All plasma samples were cold-centrifuged (2–8°C) for 10 min at 3500 rpm. Aliquots of supernatant were stored in cryovials at −80°C and batch-analyzed in duplicate at study completion by radioimmunoassay using commercially available kits specific for humans (Millipore, St. Charles, MO, USA). Blood samples for analysis of glucose and lactate were collected into 4 mL purple top vacutainers, centrifuged as above, and the plasma was stored at −80°C until analysis. Glucose and lactate were analyzed using a Microstat Multiassay Analyzer (Analox instruments, Lunenburg, MA, USA). Hematocrit was analyzed as a marker of hemoconcentration and hemodilution using an Autocrit Ultra 3 (Clay Adams, Sparks, MI, USA) at each blood draw.

### 2.5. Statistical Approach

A sample size analysis conducted with mean and standard deviation estimates based on preliminary exercise-associated data from Russel et al. [28] in women (n = 10) and Martins et al. [33] in both sexes (n = 6 men, 6 women) for the pre-to post-exercise change in PYY_{3-36} (15 to 25% increase with exercise; SDs proportional to means; ratio of SD to mean = 0.26) and an alpha = 0.05 determined that a sample size of n = 8 was sufficient to detect, with 80% power, a minimal postexercise increase of ~20% (http://www.statsalive.com/). Given this calculation, an n = 9 for each exercise group (i.e., one additional subject per group) was selected. The sample size calculation was not performed using ghrelin or ghrelin_{acyl} due to inconsistent results for ghrelin and lack of published results for ghrelin_{acyl}.

Our first aim was to assess the effect of exercise on circulating concentrations of gut peptides, appetite, and ad libitum food intake among runners and among walkers. Concentrations of the gut peptides (total ghrelin, ghrelin_{acyl}; PYY_{3-36} and GLP-1), and the primary measure of appetite, hunger ratings were measured at baseline and five time points (t = 0, 30, 60, 90, and 120 minutes) following exercise and rest. As secondary outcomes relating to appetite, we also considered ratings of satiety, fullness, and desire to eat. For each subject, we summarized the repeated measures by calculating the slope or the rate of change in outcome per 30-minute interval, following exercise and rest. Using the derived slopes as the outcome, linear regression models were fit to evaluate differences between exercise and rest responses over the entire period of 120 minutes following exercise or rest. We adjusted for baseline levels and included an interaction between exercise/rest and runner/walker group. We calculated robust standard errors that accounted for correlation between exercise and rest measures from the same subject. As a way of capturing total response over all time points, the area under the curve (AUC) was also calculated for the 120 minutes following exercise and rest for the gut peptide concentrations and hunger ratings using the trapezoidal method (GraphPad Prism version 5.02 for Windows, GraphPad Software, San Diego, CA, USA, http://www.graphpad.com/). AUC included, by definition, the area under the curve and above baseline. For ghrelin, ghrelin_{acyl}, and GLP-1 which were observed to dip below baseline in the later postexercise period, negative AUC was also calculated as the area above the curve and below baseline. Paired t-tests were used to evaluate differences between exercise and rest responses over the entire period of 120 minutes after exercise or rest, within runners and within walkers. Ad libitum food intake was measured at a single time point following the ad libitum meal, as absolute energy intake and relative energy intake. Again, paired t-tests were used to compare exercise versus rest within runners and within walkers. As secondary outcomes, we also considered more specific components of energy intake, using three macronutrients: protein, carbohydrate, and fat. As an exploratory analysis relating to this aim, we also assessed the immediate effects of exercise versus rest on gut peptide concentrations and appetite. That is, rather than considering the trajectory of each outcome across all time points from t = 0 to t = 120, we considered only the difference between the measurements at t = 0 and at baseline for exercise versus rest. With these differences as the outcomes, we fit linear regression models, including an interaction between exercise/rest and runner/walker group. Again, we calculated robust standard errors that accounted for correlation between exercise and rest measures from the same subject.

Our second aim was to investigate whether changes in ghrelin, ghrelin_{acyl}, PYY, and GLP-1 were associated with changes in hunger following exercise. For each gut peptide, we fit a linear regression model with the peptide concentration as the predictor of interest and hunger as the outcome. Additionally, we included an interaction between hormone level and runner/walker group and adjusted for baseline hunger rating and exercise/rest period. Again, we calculated robust standard errors to account for multiple measures from the same subject. The other appetite ratings were also assessed as secondary outcomes. Statistical analyses were performed using Stata (version 10) and R statistical software (version 2.10.1). All reported P values were two-sided, with statistical significance taken to be P value < 0.05. There was no adjustment for multiple testing.

### 3. Results

Nineteen volunteers (9 runners and 10 walkers) enrolled in the study. The majority of the runners regularly competed in local road races and two were NCAA division I collegiate runners. The walkers walked regularly either for fitness and weight control, or as cross-training for other activities. None of the walkers, however, were regular runners or joggers.
Data from blood samples are missing for several participants at one or more time points following the exercise or rest periods due to complications from obtaining blood via the indwelling catheter (i.e., occasional clotting inadequate venous return). Data for ghrelinacyl are missing for several time points due to undetectable readings by the RIA.

### 3.1. Baseline Characteristics
The characteristics of the 9 runners and 10 walkers are summarized in Table 1. Table 1(a) shows that the exercise groups were fairly comparable with respect to age and height. Not surprisingly, the runners were leaner than the walkers and had a higher VO2max than the walkers.

Comparing exercise versus rest, Table 1(b) and Figure 3 show unexpected differences at baseline (preexercise or rest) in mean Ghrelinacyl only among runners, whereas mean ghrelin, PYY, and GLP-1 were similar in both groups before exercise and rest periods. Differences in mean appetite ratings were also observed prior to exercise versus rest periods among both runners and walkers.

The energy and macronutrient intakes of the controlled diet were similar (P > 0.05) before exercise and rest in both the runner and walker groups. Runners averaged 1868 ± 380 kcal (14.1 ± 1.7% protein, 64.8 ± 3.2% carbohydrate, and 21.4 ± 2.3% fat) before exercise and 2035 ± 239 kcal (14 ± 0.6% protein, 63.6 ± 1.3% carbohydrate, and 22.4 ± 1.1% fat) before rest. Walkers averaged 1770 ± 400 kcal (14.7 ± 1.5% protein, 63.8 ± 2.7% carbohydrate, and 21.5 ± 2.6% fat) before exercise and 1811 ± 357 kcal (14.9 ± 1.8% protein, 62.3 ± 1.8% carbohydrate, and 22.7 ± 1.5% fat) before rest. In the runners, the exercise test day fell on menstrual cycle day 5.9 ± 3.1, whereas the rest day fell on day 5.2 ± 2.9. In the walkers, exercise and rest days fell on 5.4 ± 3.4 and 4.4 ± 1.5, respectively. Serum progesterone concentrations were less than 2 ng/mL for all participants during both exercise and rest and averaged 0.66 ± 0.2 and 0.64 ± 0.19 for runners and 0.63 ± 0.33 and 0.46 ± 0.3 for walkers during exercise and rest, respectively, confirming follicular status.

### 3.2. Oxygen Uptake, Heart Rate and Energy Expenditure
Absolute VO2, HR, RER, RQ, RPE, and energy expenditure (EE) during the 60 min bout of running or walking and rest along with concentrations of lactate and glucose are shown in Table 2. Runners ran at an average pace of 2.9 ± 0.18 m/s (6.5 MPH), and walkers walked at an average of 1.69 ± 0.1 m/s (3.77 MPH). Absolute VO2 was higher during exercise in runners versus walkers, but relative VO2 was similar (P = 0.624) and averaged 70.4 ± 4.1% and 68.6 ± 6.4% VO2max during the run and walk, respectively. HR was also ~10 bpm higher during exercise in the runners compared to the walkers (172 bpm versus 162 bpm) but was similar between the rest trials. Energy expenditure was approximately 180 kca/average during exercise in runners compared to walkers (483.1 kcal/h versus 305.1 kcal/h) but was similar at rest. Blood lactate concentration was also similarly elevated following the run and the walk.

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### Table 1: Baseline preexercise characteristics for 9 female runners and 10 female walkers. Data are presented as mean ± SD. Body fat percentage by dual-energy X-ray absorptiometry; VO2max: maximal oxygen uptake while running or walking on a motor driven treadmill.

#### (a) Anthropometric characteristics

<table>
<thead>
<tr>
<th></th>
<th>Runners</th>
<th>Walkers</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>23.7 ± 2.4</td>
<td>24.6 ± 6.9</td>
<td>0.70</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>163.9 ± 4.2</td>
<td>164.6 ± 8.6</td>
<td>0.80</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>53.5 ± 3.1</td>
<td>60.0 ± 12.3</td>
<td>0.14</td>
</tr>
<tr>
<td>BMI (kg · m⁻¹)</td>
<td>19.8 ± 1.0</td>
<td>22.1 ± 3.4</td>
<td>0.06</td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>23.0 ± 4.9</td>
<td>35.7 ± 5.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VO2max (mL · kg⁻¹ · min⁻¹)</td>
<td>49.7 ± 3.0</td>
<td>33.9 ± 3.7</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

#### (b) Gut peptide concentrations and appetite ratings before exercise and rest trials

<table>
<thead>
<tr>
<th></th>
<th>Runners</th>
<th>Walkers</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ghrelin</td>
<td>144.7 ± 52.6</td>
<td>167.8 ± 37.0</td>
<td>0.33</td>
</tr>
<tr>
<td>Ghrelinacyl</td>
<td>10.6 ± 8.6</td>
<td>25.4 ± 15.7</td>
<td>0.02</td>
</tr>
<tr>
<td>PYY</td>
<td>45.0 ± 8.9</td>
<td>43.6 ± 10.9</td>
<td>0.67</td>
</tr>
<tr>
<td>GLP-1</td>
<td>42.5 ± 19.5</td>
<td>47.6 ± 15.5</td>
<td>0.59</td>
</tr>
<tr>
<td>Hunger</td>
<td>8.9 ± 9.4</td>
<td>13.6 ± 16.4</td>
<td>0.34</td>
</tr>
<tr>
<td>Satiety</td>
<td>71.8 ± 23.2</td>
<td>77.7 ± 19.3</td>
<td>0.75</td>
</tr>
<tr>
<td>Fullness</td>
<td>64.6 ± 26.4</td>
<td>78.2 ± 15.6</td>
<td>0.29</td>
</tr>
<tr>
<td>Desire to Eat</td>
<td>26.6 ± 25.1</td>
<td>33.4 ± 30.1</td>
<td>0.51</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Exercise*</th>
<th>Rest*</th>
<th>Exercise*</th>
<th>Rest*</th>
<th>P value¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Runners</td>
<td>126.8 ± 42.8</td>
<td>209.4 ± 127.8</td>
<td>0.99</td>
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</tr>
<tr>
<td>Walkers</td>
<td>10.5 ± 6.7</td>
<td>70.2 ± 11.8</td>
<td>0.70</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

¹ Mean ± SD.

Based on paired t-test.
3.3. Blood Concentration, Hormones, and Metabolites

**Blood Concentration.** As shown in Figure 2, total hematocrit was fairly constant over time following both exercise and rest in the runners and walkers. However, because hemocencentration was observed following exercise in quite a few of the walkers and an occasional dilute samples from saline infusion was observed in both groups, blood data were adjusted according to the methods of Dill and Costill [34] and used in all statistical analyses. As a sensitivity analysis, we repeated all analyses using the unadjusted data and obtained similar results (data not shown).

**Ghrelin.** Figure 3 (upper left panel) illustrates the average trajectory over all time points (one time point before exercise or rest and five time point after exercise or rest). Average total ghrelin concentration drifted upward immediately after exercise in the runners and the walkers, and then leveled off in the runners, while demonstrating large variability in the walkers. The concentration remained fairly constant after rest. Table 3(a) reflects these results, indicating that exercise may increase the overall rate of change of ghrelin concentration in runners. While the rate of change after exercise stay close to zero (−2.1 pmol/L per minute), a positive average

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**Table 2:** Oxygen uptake, energy expenditure, and heart rate of 9 female runners and 10 female walkers during 60 min of exercise and 60 min of bed rest. VO₂: volume of oxygen uptake; RER: respiratory exchange ratio; RQ: respiratory quotient; HR: heart rate; RPE: rating of perceived exertion.

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Exercise</th>
<th>Runners*</th>
<th>Walkers*</th>
<th>Runners*</th>
<th>Walkers*</th>
<th>Runners₆₉ versus walkers₆₉†</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂ (L·min⁻¹)</td>
<td>0.23 ± 0.02</td>
<td>0.22 ± 0.04</td>
<td>1.9 ± 0.16</td>
<td>1.39 ± 0.34</td>
<td>0.48 (0.22, 0.73)</td>
<td>0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RER/RQ</td>
<td>0.78 ± 0.05</td>
<td>0.74 ± 0.05</td>
<td>0.90 ± 0.03</td>
<td>0.88 ± 0.04</td>
<td>−0.01 (−0.04, 0.02)</td>
<td>0.570</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Energy expenditure (kcal/h)</td>
<td>65.5 ± 6.7</td>
<td>62.3 ± 11.5</td>
<td>483.1 ± 49.7</td>
<td>324.6 ± 138.1</td>
<td>158.5 (56.2, 260.7)</td>
<td>0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>68.6 ± 24.3</td>
<td>72.3 ± 7.9</td>
<td>171.8 ± 11.3</td>
<td>161.9 ± 16.1</td>
<td>9.9 (−4.17, 24.0)</td>
<td>0.15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RPE</td>
<td>—</td>
<td>—</td>
<td>13.1 ± 1.1</td>
<td>14.1 ± 2.7</td>
<td>−0.97 (−3.13, 1.19)</td>
<td>0.35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactate (mmol/L)</td>
<td>0.76 ± 0.26</td>
<td>0.92 ± 0.4</td>
<td>1.4 ± 0.66</td>
<td>1.0 ± 0.55</td>
<td>0.4 (−0.21, 1.0)</td>
<td>0.19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>5.1 ± 0.56</td>
<td>5.0 ± 0.67</td>
<td>6.7 ± 1.8</td>
<td>5.7 ± 0.66</td>
<td>1.03 (−0.38, 2.4)</td>
<td>0.14</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Mean ± SD.
†Difference in means (95% confidence interval) from two-sample t-test.
VO₂: oxygen uptake; RER: respiratory exchange ratio; RQ: respiratory quotient; HR: heart rate; RPE: rate of perceived exertion.
Table 3: Effect of exercise on (a) rate of change of gut peptide concentrations and hunger ratings over 120 minutes after exercise or after rest, among runners and among walkers and (b) gut peptide concentrations and hunger ratings immediately after exercise or after rest (at t = 0).

(a) Effect of exercise over all time points

<table>
<thead>
<tr>
<th></th>
<th>Runners</th>
<th>Walkers</th>
<th>P value</th>
<th>Runners</th>
<th>Walkers</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Exercise*</td>
<td>Rest*</td>
<td>Exercise effect†</td>
<td>P value</td>
<td>Exercise*</td>
<td>Rest*</td>
</tr>
<tr>
<td>Ghrelin (pmol/L)</td>
<td>10.0 ± 22.9</td>
<td>−2.1 ± 11.7</td>
<td>12.9 (−3.9, 29.7)</td>
<td>0.12</td>
<td>−12.8 ± 33.6</td>
<td>−4.6 ± 23.2</td>
</tr>
<tr>
<td>Ghrelin acyl (pmol/L)</td>
<td>3.6 ± 10.0</td>
<td>−1.4 ± 3.9</td>
<td>2.9 (−7.6, 13.4)</td>
<td>0.56</td>
<td>1.7 ± 4.2</td>
<td>−1.3 ± 4.7</td>
</tr>
<tr>
<td>PYY (pmol/L)</td>
<td>−3.9 ± 2.4</td>
<td>−1.7 ± 1.6</td>
<td>−2.0 (−4.0, −0.095)</td>
<td>0.041</td>
<td>−9.4 ± 8.3</td>
<td>−2.2 ± 1.7</td>
</tr>
<tr>
<td>GLP-1 (pmol/L)</td>
<td>−8.5 ± 7.9</td>
<td>1.5 ± 6.3</td>
<td>−10.7 (−17.0, −4.4)</td>
<td>0.002</td>
<td>−10.2 ± 10.6</td>
<td>6.9 ± 18.1</td>
</tr>
<tr>
<td>Hunger</td>
<td>13.4 ± 5.9</td>
<td>10 ± 4.6</td>
<td>3.6 (−1.5, 8.7)</td>
<td>0.15</td>
<td>9.2 ± 4.9</td>
<td>7.6 ± 4.5</td>
</tr>
<tr>
<td>Satiety</td>
<td>−8.7 ± 7.8</td>
<td>−8.2 ± 7.6</td>
<td>−2.6 (−7.1, 1.8)</td>
<td>0.23</td>
<td>−5.6 ± 6.8</td>
<td>−7.6 ± 3.8</td>
</tr>
<tr>
<td>Fullness</td>
<td>−10.6 ± 6.4</td>
<td>−9.3 ± 3.3</td>
<td>−3.9 (−7.7, −0.16)</td>
<td>0.042</td>
<td>−8.2 ± 4.3</td>
<td>−6.6 ± 5.0</td>
</tr>
<tr>
<td>Desire to Eat</td>
<td>10.2 ± 6.3</td>
<td>8.4 ± 4.3</td>
<td>1.9 (−2.4, 6.1)</td>
<td>0.37</td>
<td>7.9 ± 4.4</td>
<td>8.3 ± 3.7</td>
</tr>
</tbody>
</table>

*Mean ± SD of slopes.
†Estimated effect (95% confidence interval) from linear regression, adjusting for baseline.

(b) Immediate effect of exercise

<table>
<thead>
<tr>
<th></th>
<th>Runners</th>
<th>Walkers</th>
<th>P value</th>
<th>Runners</th>
<th>Walkers</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Exercise*</td>
<td>Rest*</td>
<td>Exercise effect†</td>
<td>P value</td>
<td>Exercise*</td>
<td>Rest*</td>
</tr>
<tr>
<td>Ghrelin (pmol/L)</td>
<td>−3.7 ± 71.1</td>
<td>3.6 ± 43.5</td>
<td>−7.3 (−57.2, 42.6)</td>
<td>0.76</td>
<td>39.2 ± 125.4</td>
<td>−28.7 ± 151.9</td>
</tr>
<tr>
<td>Ghrelin acyl (pmol/L)</td>
<td>7.9 ± 5.7</td>
<td>0.03 ± 9.2</td>
<td>7.9 (−0.9, 16.7)</td>
<td>0.075</td>
<td>−2.3 ± 5.3</td>
<td>4.0 ± 20.5</td>
</tr>
<tr>
<td>PYY (pmol/L)</td>
<td>8.3 ± 12.7</td>
<td>−4.0 ± 5.8</td>
<td>12.3 (3.6, 21.0)</td>
<td>0.008</td>
<td>9.8 ± 18.4</td>
<td>−3.6 ± 13.2</td>
</tr>
<tr>
<td>GLP-1 (pmol/L)</td>
<td>30.3 ± 25.5</td>
<td>−2.2 ± 18.9</td>
<td>32.4 (5.3, 59.5)</td>
<td>0.022</td>
<td>9.8 ± 56.5</td>
<td>1.1 ± 10.5</td>
</tr>
</tbody>
</table>

*Mean ± SD of difference in t0 and baseline.
†Estimated effect (95% confidence interval) from linear regression.
postexercise slope (10.0 pmol/L per 30 minutes) and an overall positive difference between exercise and rest (12.9 pmol/L per minute, 95% CI [−3.9, 29.7], P value = 0.12) are estimated. Meanwhile, for walkers, the larger variability at later time points may obscure the true effect of exercise. Table 3(b) contains results of exploratory analysis relating to immediate effects of exercise. Figure 3 (upper right panel) shows patterns for average Ghrelinacyl concentration that are somewhat similar to those for total ghrelin. Table 3(a) shows little evidence of an exercise effect on the rate of change of Ghrelinacyl concentration over time. There is some indication, however, of a larger immediate increase in Ghrelinacyl concentration after exercise versus after rest among runners (7.9 pmol/L, 95% CI [−0.9, 16.7], P value = 0.075).

The positive AUC (area above baseline) for both total ghrelin and ghrelinacyl was found to be higher following exercise in the runners but not the walkers. Negative AUC (area under baseline) was found to be smaller following exercise versus after rest among runners (7.9 pmol/L, 95% CI [−17.0, −4.4], P value = 0.006) and among walkers (−2.0 pmol/L per 30 minutes, 95% CI [−10.7, −0.3], P value = 0.12). There was also evidence of an immediate effect of exercise among runners, but not in the walkers. The positive AUC for GLP-1 was not different following exercise compared to rest in either the runners or the walkers, however.

**PYY and GLP-1.** In runners, PYY concentration peaked immediately after exercise (Figure 3) then gradually returned to baseline over the 120 min after exercise; whereas in walkers, PYY concentration peaked at 30 min after exercise before returning to baseline 90 min after exercise. Table 3(a) shows evidence of the effect of exercise on the rate of change of PYY, with exercise causing a faster decline in PYY concentration over time among runners (−2.0 pmol/L per 30 minutes, 95% CI [−4.0, −0.9], P value = 0.041) and among walkers (−6.7 pmol/L per 30 minutes, 95% CI [−13.2, −0.14], P value = 0.43), although the effect was not statistically significant in the walkers potentially due to high variability. Immediate effects were also evident, but positive (Table 3(b)), thus reflecting the observed pattern of an immediate rise in concentration followed by a decline among both runners and walkers. The positive AUC for PYY tended to be higher after exercise versus rest in the runners. Negative AUC was also found to be higher after exercise versus rest in walkers (Table 4).

Similar to PYY, GLP-1 concentration peaked immediately after exercise in both runners and walkers returning to pre-exercise concentrations at approximately 30 min after exercise. Unlike PYY, GLP-1 dipped visibly below baseline after 60 min after exercise in both groups (Figure 3). Table 3(a) shows fairly large effects of exercise on the rate of change of GLP-1. Again, exercise caused a faster decline in GLP-1 concentration among both runners (−10.7 pmol/L per 30 minutes, 95% CI [−17.0, −4.4], P value = 0.002) and walkers (−16.5 pmol/L per 30 minutes, 95% CI [−28.0, −5.0], P value = 0.008). There was evidence of an immediate effect of exercise among runners, but not in the walkers. The positive AUC for GLP-1 was not different following exercise compared to rest in either the runners or the walkers, however.
the area below baseline was greater in the walkers (1957 ± 2058 and 1447 ± 3459, \( P = 0.05 \)) but not the runners after exercise versus rest (Table 4).

### 3.4. Hunger and Satiety Ratings.

As shown in Figure 4 (top panel), ratings of "hunger" increased from baseline across the 120 min after exercise or after rest period in both the runners and walkers during the exercise and rest trials. Similar results were observed for desire to eat, that is, how much you think you can eat (data not shown). Ratings of satiety (Figure 4, bottom panel) and fullness (data not shown) tended to decrease from baseline across the 120 min after exercise or after rest period in both groups during both trials. We see from Table 5 that there was not enough evidence to show an effect of exercise on the rate of change over time for any of the four subjective appetite ratings in either group. The AUCs for the four appetite ratings were also not found to be significantly different after exercise versus after rest for either group.

### 3.5. Ad Libitum Food Intake.

As shown in Table 5, there was no evidence of a difference between absolute energy intake and macronutrient intake at the free-choice meal following running versus rest. However, absolute intake tended to be higher following walking compared to rest (73.2 kcal, 95% CI [−110.0, 157.5], \( P = 0.080 \)). Interestingly, walkers (but not runners) tended to consume more protein (in walkers: 4.1 g, 95% CI [2.2, 6.0], \( P = 0.001 \)) following exercise compared to rest (Figure 5).

### 3.6. Relative Energy Intake.

After adjusting for the cost of exercise or rest, relative energy intake was lower following exercise compared to rest in both groups (\( P = 0.001 \)).
3.7. Gut Peptides and Appetite Ratings. In the runners, the change in concentrations of PYY and GLP-1 was predictive of the change in hunger (Table 6). Analogous results were obtained for satiety, fullness, and desire to eat (data not shown). Increases in PYY and GLP-1 were positively associated with satiety and fullness and negatively associated with hunger and desire to eat. In the walkers, there may be some indication of an association between hunger ratings and Ghrelin (0.63 units, 95% CI [−0.19, 1.46], P value = 0.12) and GLP-1 (0.095 units, 95% CI [−0.32, 0.028], P value = 0.095) concentrations.

4. Discussion
The purpose of the current study was to evaluate the effect of 60 min of moderately hard running and walking at the same

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**Figure 4**: Average change in hunger (upper panel) and satiety (lower panel) for runners and walkers during exercise (solid grey) and rest (dashed black). "Pre" represents the time point just before exercise or rest. Vertical bars display the standard error of the mean for hunger and satiety at that time point. Data for fullness and desire to eat are not shown.
Because ghrelinacyl responds more rapidly to glucose infusion and exercise [42]. Thus, measurement of ghrelinacyl, which accounts for is currently the only known orexogenic peptide [35, 36]. Circulating ghrelinacyl was found to predict hunger in runners but not walkers. Interestingly, the average rate of change in the anorexogenic peptides and a trend for an increase in over time was not replaced. Overall, these results suggest that the energy cost of the exercise which was ~38% higher during running versus walking may promote increased ghrelin secretion, perhaps more so in women. Coupled with our finding that neither total ghrelin nor ghrelinacyl correlated with hunger versus walking) may promote increased ghrelin secretion because the signal is dampened by increases in the anorexogenic peptides over the same time point [28].

In contrast to ghrelin, peptide YY and GLP-1 are satiety peptides which are secreted from the endocrine L cells of the distal gastrointestinal tract in response to a mixed meal [47]. Circulating concentrations of both PYY and GLP-1 are low in fasting and increase following meal ingestion [48]. Peripheral infusion of both peptides at physiological concentrations markedly decrease food intake in humans [49, 50] which appears to be additive when infused simultaneously [51]. The action of PYY is thought to be via inhibition of NPY/AgRP neurons and/or stimulation of vagal-afferent nerves, whereas the action of GLP-1 is thought to be via vagal mediation [37]. Both forms of PYY (PYY3-36 and PYY1-36) and GLP-1 are thought to serve as satiety signals, regulating the termination of individual meals [40].

Consistent with our findings, previous studies have found elevations in both PYY [25, 27, 52] and GLP-1 [27, 52] following different modes and intensities of exercise. A study by Ueda and colleagues [16] found that postexercise elevation of PYY but not GLP-1 was dependent on exercise intensity and was elevated to a greater extent following 30 min of cycling at 75% compared to 50% VO2max. In another study, Broom and colleagues [17] found elevated PYY and suppressed hunger in the 2 h after a 60 min bout of

**Table 6: Association of hormone concentrations with hunger ratings.**

<table>
<thead>
<tr>
<th>Hormone (pmol/L)</th>
<th>Runners</th>
<th>P value</th>
<th>Walker</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ghrelin</td>
<td>0.038 (−0.066, 0.14)</td>
<td>0.45</td>
<td>0.033 (−0.022, 0.088)</td>
<td>0.22</td>
</tr>
<tr>
<td>Ghrelinacyl</td>
<td>0.077 (−0.23, 0.38)</td>
<td>0.60</td>
<td>0.63 (−0.19, 1.46)</td>
<td>0.12</td>
</tr>
<tr>
<td>PYY</td>
<td>−1.02 (−1.52, −0.53)</td>
<td>&lt;0.001</td>
<td>−0.034 (−0.28, 0.21)</td>
<td>0.78</td>
</tr>
<tr>
<td>GLP-1</td>
<td>−0.36 (−0.62, −0.11)</td>
<td>0.008</td>
<td>−0.14 (−0.32, 0.028)</td>
<td>0.09</td>
</tr>
</tbody>
</table>

*Estimate (95% confidence interval) from linear regression, adjusting for exercise and baseline hunger.
running at 69% VO₂max compared to both rest and a 90 min bout of resistance exercise. It is important to note, however, that the energy cost was 50% higher in the high-intensity compared to the moderate intense cycling and ~260% greater with the running compared to the resistant training in the aforementioned studies. Thus, our finding that both PYY and GLP-1 were elevated immediately after running, and that only PYY was elevated after walking may also be explained by the greater energy cost of the run, which was ~37% greater than the walk. Interestingly, the average rate of change in PYY and GLP-1 after the run, and the rate of change in GLP-1 after the walk was significantly greater relative to rest, indicating an average downward trend following exercise, particularly for GLP-1 which dipped below baseline in the later postexercise period. While it is possible this dip in GLP-1, which had a more negative AUC in walkers compared to runners, at least partially accounted for the higher (less negative) relative intake in the walkers compared to the runners, future research is needed to affirm that such a role is causal.

Our results concerning *ad libitum* food intake following exercise in women are in agreement with previous studies in both sexes which found either no difference or slightly higher absolute food intake after a bout of exercise compared to a noexercise control, but significantly lower relative energy intake when accounting for the energy cost of exercise [12, 13, 53–55]. Interestingly, in these studies, relative energy intake was lowest (i.e., creating a more negative balance) when exercise intensity was high, and when foods offered in the subsequent *ad libitum* meal were low in fat [13, 53, 54]. Imbeault and colleagues [15], for example, found lower relative energy intake after 34 min of running at 75% VO₂max than after 72 min of walking at 35% VO₂max, which elicited the same energy cost (~485 kcal). King and colleagues [12], who were first to introduce the concept of relative energy intake, have argued the greater relevance of relative rather than absolute energy intake because higher energy intake would be an expected compensatory mechanism of increased energy expenditure through increased physical activity. Thus, if energy intake remains the same following exercise, as in the current study, it can be considered equivalent to a suppression of appetite relative to the intake expected to compensate for the exercise. Unfortunately, the majority of studies, including the current study, have not measured energy intake for a long enough period after exercise to evaluate how compensation for negative energy balance occurs following different modes of exercise like running but not necessarily walking. Total or partial compensation through altered energy intake and reduced energy expenditure are possible and likely, otherwise exercise would result in drastic reductions in body mass/body adiposity.

Although we did not find significant differences in perceived hunger at any point following running or walking compared to rest, small changes in hunger due to exercise rather than time (observed in the nonexercise control condition) may be difficult to detect using available methodology. Indeed, only about half of the studies in men using designs similar to ours have observed differences in hunger using VAS [12, 14, 16, 17, 22, 23, 27, 53, 56], whereas very few studies in women have detected exercise-associated differences [55, 57]. The lack of a strong exercise influence on appetite in all studies may be because VAS are not sensitive enough to detect small changes following exercise using sample sizes typically employed for exercise studies. It also may be that only a small subset of subjects is in tune with biological hunger cues and respond instead to other signals including time of day or time past since the last meal. Mattes [58], for example, observed that food intake often occurred when hunger was low or had not changed acutely. In our studies we did find, however, that VAS track well with changes in both PYY and GLP-1 in runners and tended to track with GLP-1 in walkers which suggests a relation between appetite ratings and satiety peptides even if exercise-induced alterations in appetite were not observed.

The current study used a unique complex modeling approach to evaluate whether changes in the gut peptides tracked with or predict changes in hunger and/or *ad libitum* food intake. Collectively, our findings suggest that changes in PYY and GLP-1 over time tracked indirectly with changes in hunger and desire to eat, and directly with changes in satiety. Interestingly, the change in either total ghrelin or ghrelinacyl did not track with subjective ratings of hunger. This provides additional support for the hypothesis that signals from elevated concentrations of circulating ghrelin may be muted by elevated concentrations of satiety peptides. Given that few [16, 22, 25] previous studies have found clear associations between gut peptide concentrations and appetite following exercise, it is probable that exercise-induced alterations in appetite are driven by complex changes in appetite-regulating hormones rather than a single gut peptide in isolation. A previous study by Martins and colleagues [27], for example, observed an inverse temporal pattern between hunger and both PYY and GLP-1 concentrations during 1 h of exercise but did not describe such a relation following exercise. In contrast, Broom et al. [22] and Ueda et al. [16] observed direct associations between the AUC for plasma ghrelinacyl and hunger, and indirect association between the AUC for GLP and postexercise energy intake. The discrepancy between the findings and published studies may be explained by the different exercise-induced patterns of gut peptide release.

In the current study, we elected to evaluate the effect of walking and running on appetite and gut hormone responses because both weight-bearing activities are recommended for weight loss and weight loss maintenance. Walking, however, is the most common exercise recommended [18] and, unlike running, can be undertaken by the majority of the population because it does not require the fitness base or produce the biomechanical stress of running. Our overall observation that walking did not elicit the same negative energy balance or increase in the satiety hormones as did running, yet promoted a slightly higher postexercise fat and protein intake, suggests that walking may create some challenges for long-term weight loss unless dietary restriction is employed. While our results appear to contradict those of King and Colleagues [18] who observed significantly lower relative energy intakes in men after a 60-min “brisk” walk at a self-selected pace (ranging from 33.8 to 55.5% VO₂max), the apparently discrepant results may help explain why exercise is less effective in promoting weight loss in women compared to men [7, 8].
The mechanism, however, may not be easily identified because Ghrelin_{acyl} was not altered by walking in either study, and King and colleagues [18] unfortunately did not simultaneously measure PYY, GLP or other satiety peptides. In our study, we also observed a curious tendency for Ghrelin_{acyl}, total ghrelin and subjective hunger to be lower when subjects knew that they were going to exercise, which may have interfered with our ability to detect true changes with exercise compared to rest. The increased consumption of fat may be important given that a reversal of the energy deficit induced by previous exercise is noted when high-fat rather than low-fat foods offered after exercise [13, 53, 54]. Finally, from our study design, it is impossible to determine whether our observed differences between running and walking are due to exercise mode or the physiological characteristics of the walkers who were on average fatter and had a lower VO_{2max} (i.e., were less fit) than the runners. Although the current study did not measure any long-acting adiposity hormone such as leptin or insulin, it is possible that these hormones were higher in the walkers. Emerging evidence suggests that long- and short-acting signals interact to alter hypothalamic sensitivity to satiation signals [37] which could ultimately influence eating behavior following exercise. Future studies should consider different modes of exercise along with sex and adiposity differences of the exerciser and measurement of short- and long-acting satiety signals.

Acknowledgments

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