Acute aerobic exercise differentially alters acylated ghrelin and perceived fullness in normal-weight and obese individuals

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Submitted 29 April 2013; accepted in final form 8 July 2013

Heden TD, Liu Y, Park Y, Dellsperger KC, Kanaley JA. Acute aerobic exercise differentially alters acylated ghrelin and perceived fullness in normal-weight and obese individuals. J Appl Physiol 115: 680–687, 2013. First published July 11, 2013; doi:10.1152/japplphysiol.00515.2013.—Adiposity alters acylated ghrelin concentrations, but it is unknown whether adiposity alters the effect of exercise and feeding on acylated ghrelin responses. Therefore, the purpose of this study was to determine whether adiposity [normal-weight (NW) vs. obese (Ob)] influences the effect of exercise and feeding on acylated ghrelin, hunger, and fullness. Fourteen NW and 14 Ob individuals completed two trials in a randomized counterbalanced fashion, including a prior exercise trial (EX) and a no exercise trial (NoEX). During the EX trial, the participants performed 1 h of treadmill walking (55–60% peak O2 uptake) during the evening, 12 h before a 4-h standardized mixed meal test. Frequent blood samples were taken and analyzed for acylated ghrelin, and a visual analog scale was used to assess perceived hunger and fullness. In NW individuals, EX, compared with NoEX, reduced fasting acylated ghrelin concentrations by 18% (P = 0.03), and, in response to feeding, the change in acylated ghrelin (P = 0.02) was attenuated by 39%, but perceived hunger and fullness were unaltered. In Ob individuals, despite no changes in fasting or postprandial acylated ghrelin concentrations with EX, postprandial fullness was attenuated by 46% compared with NoEX (P = 0.05). In summary, exercise performed the night before a meal suppresses acylated ghrelin concentrations in NW individuals without altering perceived hunger or fullness. In Ob individuals, despite no changes in acylated ghrelin concentrations, EX reduced the fullness response to the test meal. Acylated ghrelin and perceived fullness responses are differentially altered by acute aerobic exercise in NW and Ob individuals.

GHRELIN IS A 28-AMINO ACID PEPTIDE hormone predominately produced by P/D1 cells in the fundus of the stomach and has numerous biological functions (29). Initially, ghrelin was discovered as a growth-hormone secretagogue (29). Since its discovery, ghrelin has also been shown to stimulate appetite (1, 37), stimulate gastric acid secretion (13), reduce cardiac afterload and increase cardiac output without altering heart rate (36), and increase adiposity and weight gain (49). Plasma ghrelin secretion increases in the preprandial period and decreases in the postprandial period, since ghrelin is the only known hormone to have orexigenic effects (11, 45).

The effect of acute aerobic exercise, before weight loss, on plasma total ghrelin concentrations and appetite has been studied extensively, but the findings are equivocal. Most studies show total ghrelin does not change during exercise (9, 12, 35, 43, 44, 46) or during the postexercise recovery period (3, 9, 22, 30–31, 35), whereas other studies show increases during exercise (10, 15) or during the postexercise recovery period (23, 32), and yet others show decreases during the postexercise recovery period (48, 50) or during the postprandial period after an acute aerobic exercise bout (18, 34). All of these studies were associated with variable alterations in appetite. These studies are limited by the fact that total ghrelin was measured. In humans, there are two endogenous isoforms of ghrelin, including acylated and des-acylated ghrelin (together equal to total ghrelin), with des-acylated ghrelin constituting the majority of total ghrelin in circulation (5). Acylation of ghrelin is required for ghrelin to bind to the growth hormone-secretagog receptor (5), and, in humans, only acylated ghrelin has pituitary and pancreatic endocrine activity (5, 6). Thus acylated ghrelin is thought to have a greater impact on appetite. Given that des-acylated ghrelin represents such a great percentage of total ghrelin concentrations, it is possible that changes in total ghrelin mask changes in acylated ghrelin, as demonstrated by Shiiya et al. (46). Thus it is important that acylated ghrelin be measured when associations between acylated ghrelin and appetite are made.

The majority of studies have examined the impact of exercise on fasting or postprandial acylated ghrelin concentrations and appetite responses in normal-weight (NW) individuals. One study reported acylated ghrelin concentrations increased during running, but not walking, with no change in energy intake at a subsequent meal (32). Another study reported acylated ghrelin concentrations or appetite were not altered during aerobic exercise (26). In contrast, most studies showed that acylated ghrelin concentrations and perceived hunger were reduced during exercise in the postprandial period (4) and preprandial period (2, 4, 7, 8, 14, 24, 25, 52, 53), but no significant change in acylated ghrelin concentrations or appetite were observed during a subsequent postexercise meal (8, 25, 27, 52). These studies used NW individuals, but there is evidence that adiposity may influence acylated ghrelin concentrations. For instance, one study reported higher fasting acylated ghrelin concentrations in obese (Ob) compared with NW men and women (42). However, this response is not consistent between studies, since another study showed no difference in fasting acylated ghrelin concentrations between NW or Ob men (51), whereas another study reported lower acylated ghrelin concentrations in overweight adolescents compared with NW adolescents (33). The combined effect of exercise and feeding on acylated ghrelin concentrations and perceived hunger or fullness has not been studied extensively in Ob individuals. The two studies available utilized a short-term (4–5 days) training model and assessed acylated ghrelin and appetite 18–36 h after the last exercise bout. One study reported that, in overweight/Ob adults, short-term aerobic ex-
exercise training, with or without energy replacement, blunted the postprandial decline in acylated ghrelin concentrations in women but not in men (19). To our knowledge, only one study has examined the potential differential effect of adiposity (NW vs. Ob) on acylated ghrelin concentrations and perceived hunger and fullness in response to exercise and feeding. In this study, although fasting and postprandial acylated ghrelin concentrations increased and perceived fullness decreased after 5 days of aerobic exercise training in NW and Ob adolescents, these changes were of greater magnitude in NW compared with Ob adolescents (33). However, no study has determined how adiposity (NW vs. Ob) influences fasting or postprandial acylated ghrelin concentrations, perceived hunger, and fullness in adults in response to a single bout of aerobic exercise before feeding.

The purpose of this study was to determine the impact of a single aerobic exercise session performed the night before a standardized mixed meal on fasting and postprandial acylated ghrelin concentrations, perceived hunger, and fullness in NW and Ob adults. We hypothesized that Ob individuals, compared with NW individuals, would have blunted acylated ghrelin concentrations and alterations in perceived hunger and fullness after a single bout of aerobic exercise.

METHODS

Participants. This study was approved by the University of Missouri Health Sciences Institutional Review Board. All participants provided written, informed consent before participating. All participants were screened to determine eligibility, and this included the assessment of height, weight, body composition via the BOD POD, completion of physical activity, diet history, and medical history questionnaires, and peak O2 uptake (\(\dot{V}O_2\) peak) during a stress test. All participants fasted for 3 h before testing. Skintight clothing and a swim cap were worn by all participants during testing. Jewelry and eyeglasses were removed before testing. Body composition was estimated using the Siri equation from the computer program.

Exercise testing and free-living physical activity. The stress test/aerobic fitness test was conducted on a motor-driven treadmill (Quinton Q-Stress TM55, Cardiac Science, Bothell, WA). The electrical activity of the heart was assessed with a 12-lead electrocardiograph (ECG). Oxygen consumption was measured using indirect calorimetry (ParvoMedics TrueOne 2400 metabolic cart, Salt Lake City, UT). The exercise test consisted of 2-min stages that progressively increased in intensity. During stage 1, the treadmill velocity and grade were set at 2.5 miles/h at 0% grade. Every 2 min thereafter, the speed was increased by 0.5 miles/h until 3.5 miles/h was reached. After the maximum speed was reached, then the grade was increased 2% every 2 min thereafter until the participant could no longer continue. The highest oxygen consumption value recorded was used as the \(\dot{V}O_2\) peak.

During day 3 of both trials, the participants wore a pedometer (Walk4life, Oswego, IL). The pedometer was worn on the hip during the day, including the acute exercise bout. During the acute exercise session, the participants performed 1 h of brisk treadmill walking at a moderate intensity (55–60% of \(\dot{V}O_2\) peak). During exercise, indirect calorimetry was used to assess oxygen consumption, and the treadmill speed and grade was adjusted throughout the exercise session to ensure that the oxygen consumption was within the target range.

Test meal. The test meal was given on the morning of day 4 between 0700 and 0900, 12 h after the acute exercise session on the evening of day 3. The participants consumed the test meal within 15 min. The test meal (600 calories, 45% carbohydrate, 40% fat, and 15% protein) consisted of scrambled eggs, English muffin with margarine, and 10 fl. oz. of an artificially flavored sweetened drink.

Perceived hunger and fullness. Perceived hunger (appetite) and perceived fullness (satiety) were assessed with a 100-mm linear visual analog scale (VAS) at baseline, 5 min after the meal, and every 30 min after the meal for 4 h. The question used to assess hunger was, “

How hungry do you feel?”

and the question used to assess fullness was, “

How full do you feel?”

The participants marked a single vertical line through the horizontal line of each scale between the extremes (i.e., “not at all hungry” to “as hungry as I have ever felt,” and “not at all full” to “as full as I have ever felt”) to indicate their perceived hunger and fullness at that time. Prior research has shown the VAS is a valid and reliable scale used to assess perceived hunger and fullness (16, 40).

Blood collection and analysis. On the morning of the mixed meal test, a catheter was placed into the antecubital vein of the forearm of the participant. Next, two baseline venous blood samples were taken, and then the participants were given the meal. Postprandial blood sampling began once the test meal was completely ingested, and blood samples after the meal were taken 5, 10, 15, 20, 30, 40, 50, 60, 75, 90, 120, 150, 180, 210, and 240 min after ingestion of the meal. At each time point, ~3 ml of blood was collected. Half (1.5 ml) of the blood was put into serum separator tubes and was allowed to clot at room temperature before spinning. The other half was put into chilled EDTA tubes pretreated with 15 \(\mu\)l of Pefabloc SC added (DSM Nutritional Products, Branch Pentapharm, Switzerland), as suggested by the manufacturer, to preserve acylated ghrelin. Samples were separated by centrifugation at 3,000 RPM for 15 min at 4°C and frozen at −80°C until analysis. Plasma hormone concentrations of acylated ghrelin were determined using a MILLIPLEX magnetic bead-based quantitative multiplex immunoassay with the MAGPIX instrumentation (Millipore, Billerica, MA). The intra-assay coefficient of variability (CV) was 4.62%, and the interassay CV was 11.84%.

Dietary records. The participants self-selected the foods they ate during both trials, except for the test meal. Diet was controlled by

J Appl Physiol • doi:10.1152/japplphysiol.00515.2013 • www.jappl.org

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having the participants keep written dietary records with the exact timing of all meals, snacks, and beverages consumed during days 1–3 of each trial. During the second trial performed, a copy of the dietary record from the first trial was given to the participant, and they were asked to precisely replicate the record during the subsequent trial. The dietary macronutrient composition was determined using the Nutrition Data System for Research software (NDSR, University of Minnesota, Minneapolis, MN).

Calculations and statistical analysis. Postprandial acylated ghrelin, perceived hunger, and perceived fullness responses between conditions were quantified and compared using the incremental area under the curve (iAUC), which was calculated using the trapezoidal method (41). There is no universal way to quantify postprandial responses, but we believe the iAUC is the more appropriate measure for all outcomes. The iAUC corrects for changes in baseline values (even small, nonsignificant changes are corrected) and better represents the change from baseline. Thus only iAUC data is presented. The food records for the 3 days before each testing day were pooled together, and the average was used for the statistical analysis. The IBM SPSS statistical software version 20 (IBM, Armonk, NY) was used to perform the statistical analysis. The participant characteristics and the metabolic data during exercise between NW and Ob groups were compared using an independent samples t-test. Daily caloric intake and step counts between each trial, within each group, were compared using a paired samples t-test. A mixed-model repeated-measures ANOVA with Bonferroni adjusted post hoc tests were used to examine differences in fasting and postprandial acylated ghrelin, perceived hunger, and perceived fullness between trials and groups. Statistical significance was set at $P \leq 0.05$. In addition, we performed several secondary analyses. There is some evidence that sex may influence the effect of prior exercise on acylated ghrelin responses (19). We performed a separate analysis to examine any potential effect of sex with a mixed-model repeated-measures analysis of covariance (ANCOVA), with sex as the covariate. However, we did not find any effect of sex on any variable (data not shown). In addition, another secondary analysis we performed included a mixed-model repeated-measures ANCOVA using baseline adiposity (body fat percent) as a covariate for all variables. All values are means ± SD, unless otherwise denoted.

RESULTS

Participant characteristics. The participant characteristics are listed in Table 1. Fourteen NW (six women, eight men) and 14 Ob (eight women, six men) individuals completed this study. The age, height, diastolic blood pressure, and maximal oxygen consumption of the NW and Ob participants were not significantly different, whereas all other variables were significantly greater ($P \leq 0.02$) in the Ob group compared with the NW group. Physical activity levels (step counts) were significantly greater on day 3 during the EX trial compared with the NoEX trial for both NW (EX: 12,270 ± 2,963 steps; NoEX: 6,538 ± 2,914 steps; $P < 0.01$) and Ob (EX: 13,316 ± 1,985 steps; NoEX: 5,262 ± 2,386 steps; $P < 0.01$), but there were no significant group differences.

Exercise. During the exercise bout, the mean speed and grade were 3.42 ± 0.31 miles/h and 5.05 ± 1.73% grade in the NW group and 3.17 ± 0.27 miles/h and 3.50 ± 1.64% grade in the Ob group. The gross energy expenditure (calories), mean oxygen consumption (ml·kg fat-free mass$^{-1}$·min$^{-1}$), intensity (% of $V_{O2peak}$), and mean heart rate during the 1-h exercise bout are listed in Table 2.

Diet. During days 1–3 of each trial, daily caloric intake in the NW group was not different between trials (EX: 1,824 ± 639 cal; NoEX: 1,908 ± 401 cal). Similarly, caloric intake was not different between trials in the Ob group (EX: 1,992 ± 586 cal; NoEX: 1,941 ± 506 cal). Caloric intake was not different between groups.

Fasting and postprandial acylated ghrelin responses. The acylated ghrelin responses are illustrated in Fig. 1. Fasting acylated ghrelin values were 18% lower ($P = 0.03$) during the EX trial (93 ± 9 pg/ml) compared with the NoEX trial (114 ± 17 pg/ml) in the NW group, whereas in the Ob group, fasting acylated ghrelin was not significantly different between trials (EX: 75 ± 16 pg/ml; NoEX: 75 ± 17 pg/ml). Fasting acylated ghrelin was not significantly different between Ob and NW individuals, but there was a trend for fasting acylated ghrelin to be lower in Ob individuals (EX trial: $P = 0.16$; NoEX trial: $P = 0.06$). The 4-h iAUC was 39% ($P = 0.02$) lower during the NoEX trial (−8,618 ± 1,196 pg/ml per 4 h) compared with the EX trial (−5,250 ± 1,490 pg/ml per 4 h) in the NW group (indicating a greater decline in acylated ghrelin concentrations from the baseline value during the NoEX trial), whereas there was no significant difference between trials in the Ob group (EX: −4,539 ± 1,561; NoEX: −5,359 ± 1,852). The 4-h iAUC was not significantly different between groups. In a secondary ANCOVA analysis, when we adjusted for adiposity using body fat percent as a covariate, the fasting and iAUC acylated ghrelin responses were not significantly different between trials ($P > 0.05$). This finding further suggests that the differential effects of prior exercise on acylated ghrelin in NW and Ob individuals are due to differences in adiposity.

Fasting and postprandial hunger responses. Perceived hunger responses are presented in Fig. 2. Fasting perceived hunger values were not significantly different between trials within each group or between groups (NW EX: 65 ± 6 mm; NW NoEX: 63 ± 6 mm; Ob EX: 52 ± 7 mm; Ob NoEX: 49 ± 7

Table 2. Energy expenditure, $VO_2$, and heart rate during acute exercise bout

<table>
<thead>
<tr>
<th></th>
<th>Normal-Weight Subjects ($n = 14$)</th>
<th>Obese Subjects ($n = 14$)</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calories expended</td>
<td>430 ± 116</td>
<td>519 ± 148</td>
<td>0.09</td>
</tr>
<tr>
<td>$VO_2$, ml · kg $FFM^{-1}$ · min$^{-1}$</td>
<td>27.7 ± 4.6</td>
<td>28.3 ± 3.7</td>
<td>0.73</td>
</tr>
<tr>
<td>Intensity ($VO_2$ as %peak)</td>
<td>56 ± 8</td>
<td>58 ± 5</td>
<td>0.39</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>141 ± 18</td>
<td>143 ± 12</td>
<td>0.68</td>
</tr>
</tbody>
</table>

Values are means ± SD. $VO_2$, oxygen consumption.
The 4-h hunger iAUC was not significantly different between trials within each group or between groups (NW EX: \(-773 \pm 198 \text{ mm/4 h}\); NW NoEX: \(-539 \pm 141 \text{ mm/4 h}\); Ob EX: \(-499 \pm 151 \text{ mm/4 h}\); Ob NoEX: \(-598 \pm 176 \text{ mm/4 h}\)). These perceived hunger responses were not altered when adjusted for adiposity.

**Fasting and postprandial fullness responses.** Perceived fullness responses are presented in Fig. 3. Fasting perceived fullness values were not significantly different between trials within each group or between groups (NW EX: 11 \pm 3 mm; NW NoEX: 9 \pm 3 mm; Ob EX: 21 \pm 6 mm; Ob NoEX: 20 \pm 6 mm). The 4-h fullness iAUC was attenuated by 46\% (\(P = 0.05\)) during the EX trial (409 \pm 114 mm/4 h) compared with the NoEX trial (750 \pm 168 mm/4 h) in the Ob group (indicating fullness increased less in response to the meal during the EX trial), whereas there were no significant differences in fullness iAUC values between trials in the NW group (EX: 910 \pm 155 mm/4 h; NoEX: 893 \pm 123 mm/4 h). In a secondary ANCOVA analysis, it was revealed that the fullness iAUC response was dependent on adiposity, suggesting that

![Graph 1: Acylated ghrelin responses in normal-weight and obese individuals. Values are means ± SE *Significant difference between trials within normal-weight group (\(P < 0.05\)).](image1)

![Graph 2: Hunger responses in normal-weight and obese individuals. Values are means ± SE.](image2)
the differential effects of prior exercise on fullness in NW and Ob individuals are due to differences in adiposity.

**DISCUSSION**

This is the first study to examine whether adiposity (NW vs. Ob) influences the effect of acute aerobic exercise and feeding on acylated ghrelin concentrations, perceived hunger, and perceived fullness in adults. The main findings of this study are that prior aerobic exercise 1) suppresses fasting acylated ghrelin concentrations and attenuates the change in acylated ghrelin (iAUC) in response to feeding in NW, but not Ob, individuals, and 2) suppresses fed-state perceived fullness in Ob, but not NW, individuals.

Prior research examining the effect of exercise on acylated ghrelin concentrations used a NW population, and these studies had the participants exercise in the morning either immediately before (2, 4, 7, 8, 14, 24–25, 27, 32, 52, 53) or after feeding (4) and showed that, during exercise, acylated ghrelin concentrations decreased (2, 4, 7, 8, 14, 24–25, 52, 53), with one study reporting an increase (32). In response to postexercise feeding, the acylated ghrelin response was not altered (2, 7, 25, 27) or lower (8, 52), indicating an attenuated decline in postprandial acylated ghrelin concentrations. Due to differences in study designs, particularly the timing of exercise, it is difficult to compare our findings to prior work. However, our data extends on these findings and suggests that night exercise reduces fasting acylated ghrelin concentrations the next morning in NW, but not Ob, individuals.

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The mechanism responsible for the reduced fasting acylated ghrelin concentrations in NW individuals is not completely understood, since we have not directly assessed this in the present study. However, a couple of mechanisms have been proposed. The gut enzyme ghrelin O-acyl transferase (GOAT) acylates ghrelin, giving it full biological activity (55). Exercise reduces gastric blood flow in humans (38, 39), and a study in rats showed that gut ischemia reduced ghrelin levels (54); thus it is possible that the exercise-induced suppression of gastric blood flow reduced GOAT activity, thus reducing fasting acylated ghrelin concentrations in NW individuals, whereas this process was impaired in Ob individuals. It is also possible that reduced fasting acylated ghrelin concentrations are mediated via altered vagal efferent nerve activity. Research has shown that increases in circulating ghrelin are mediated by...
increased vagal stimulation (47); thus it is possible that prior exercise reduced vagal stimulation, ultimately reducing fasting acylated ghrelin concentrations in NW individuals (46), whereas this process was impaired in Ob individuals. However, these mechanisms are only speculative and warrant further study.

In the present investigation, we observed no change in fasting or postprandial perceived hunger and fullness responses in NW individuals in response to exercise, although the appetite hormone acylated ghrelin was suppressed (moved in a direction expected to suppress appetite). This finding suggests that NW individuals will not compensate for the energy deficit created by exercise, at least in the short term. This finding is corroborated by most (2, 14, 20, 25, 27, 32, 52) but not all (35) research that has shown that prior aerobic exercise in NW or overweight individuals, compared with no exercise, does not significantly increase ad libitum absolute energy intake at a postexercise buffet meal and results in a lower relative energy intake (energy intake minus the net energy expenditure of exercise) (14, 20, 25, 32). However, in Ob individuals, we found that, despite no changes in acylated ghrelin in response to exercise, the standardized meal resulted in less fullness, which could potentially lead to a compensatory increase in energy intake. However, we believe this is unlikely, since Kissileff et al. (28) found that, in Ob women, moderate-intensity exercise, compared with no exercise, significantly increased perceived hunger but did not alter perceived fullness or ad libitum energy intake in overweight/Ob women. Similarly, George et al. (17) reported that prior exercise did not alter ad libitum energy intake in overweight women. Although prior exercise may alter perceived fullness in response to feeding in Ob individuals, the available evidence does not suggest that acute exercise increases ad libitum energy intake in Ob individuals. However, given the limited amount of research examining the impact of acute exercise on ad libitum energy intake in Ob individuals, more research is warranted in this area.

We observed a disconnect between the hormonal control of appetite (acylated ghrelin) and psychological perceived hunger and fullness ratings, and this has been documented before (8, 25, 52). These findings highlight the complexity of appetite control. In addition to acylated ghrelin, other factors play a role in hunger and fullness responses to exercise and feeding, including other hormones (PYY, PP, NPY, cholecystokinin, GLP-1) in addition to environmental and psychological factors that were not measured in this study. We recently reported that acute aerobic exercise the night before a breakfast meal (a study design identical to this study design) did not alter fasting or postprandial GLP-1 concentrations in NW or Ob individuals (21). This finding suggests that alterations in GLP-1 does not explain the altered fullness we observed in Ob individuals and that perhaps PYY, PP, NPY, or cholecystokinin are responsible. Further research is warranted to identify the hormone(s) that may play a role in the reduction in fullness with prior exercise in Ob individuals.

This study is not perfect and has some limitations that should be considered when the results are interpreted. First, our study population consisted of Caucasian young adults; thus extrapolation of our findings to other populations of different ages or races is difficult. Second, we gave our subjects a standardized breakfast meal and did not assess ad libitum energy intake; thus the translation of our findings to the real world where people eat ad libitum is limited. However, it is not completely uncommon for people to eat standardized mixed meals of a fixed size (i.e., at a restaurant when a given amount of food is served). We used a fixed-size meal because we wanted to compare the effects of adiposity on acylated ghrelin, perceived hunger, and perceived fullness while controlling for the confounding effect that meal size could have on acylated ghrelin, perceived hunger, and perceived fullness responses. Last, the participants in the present study exercised after dinner, and appetite was assessed at a breakfast meal the next day. Thus extrapolation of these findings to situations when exercise is performed at another time of day is limited.

In summary, acylated ghrelin and perceived fullness responses are differentially altered by acute aerobic exercise in NW and Ob individuals. Exercise performed the night before a standardized mixed meal suppresses fasting acylated ghrelin concentrations and attenuates the change in postprandial acylated ghrelin in NW, but not Ob, individuals. In addition, prior exercise had no effect on perceived hunger or fullness in NW individuals but reduced the perceived fullness response to a standardized mixed meal in Ob individuals. Taken together, the disconnect between acylated ghrelin and perceived hunger and fullness in the present study suggests factors other than acylated ghrelin influence the effect of exercise and feeding on appetite in NW and Ob individuals.

ACKNOWLEDGMENTS

We thank Monica L. Kearney for help with some of the data collection and Dr. Tom Thomas for assistance with the study design.

GRANTS

This study was funded by a University of Missouri Research Council Grant (J. A. Kanaley) and by a University of Missouri Institute for Clinical and Translational Science Pilot Grant (T. D. Heden).

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


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