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Effects of resistance exercise and obesity level on ghrelin and cortisol in men

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ABSTRACT

Resistance exercise (RE) is increasingly recommended by health organizations as a weight management tool. The purpose of this study was to examine the effects of an acute high-volume, whole-body RE protocol on the glucoregulatory and ghrelin response in sedentary obese and lean men. Five World Health Organization (WHO) class 1 obese (body mass index [BMI], 30.00-34.99) (age, 21.6 ± 2.5 years; height, 176.3 ± 3.7 cm; body mass, 97.8 ± 8.58 kg; body fat, 34.7% ± 2.95%), 5 WHO 2 (BMI, 35-39.99)/WHO 3 (BMI, ≥40) obese (age, 20.0 ± 1.4 years; height, 177.7 ± 5.15 cm; body mass, 120.8 ± 10.49 kg; body fat, 40.5% ± 5.82 %), and 9 lean men (age, 20.1 ± 2.1 years; height, 177.8 ± 8.7 cm; body mass, 71.7 ± 5.8 kg; body fat, 14.7% ± 3.54 %) completed an acute RE testing protocol (6 exercises, 3 sets of 10 repetitions at 85%-95% 10-repetition maximum with 120- and 90-second rest periods); and blood samples were collected pre-, mid-, and immediately postexercise and during recovery (+50, +70, and +110). Resistance exercise produced differences over time in cortisol, insulin, and glucose. Group differences were observed for ghrelin, with the WHO class 2/3 group having significantly greater ghrelin levels than the lean group (d = 0.28, P = .009) and the WHO class 1 group (d = 0.39, P = .002). Higher ghrelin was significantly associated with lower cortisol only in obese individuals. In addition, higher growth hormone was associated with lower ghrelin in lean individuals. Results suggest that glucoregulatory homeostasis is altered with increasing levels of obesity and that these alterations may mediate the response of cortisol and ghrelin in response to RE.

1. Introduction

Resistance exercise (RE) has increasingly become a mode of exercise recommended for weight management and weight loss in obese individuals [1]. Substantial evidence exists that RE can improve body composition, lipolysis, and muscle growth, which may reduce metabolic risk factors such as obesity, dyslipidemia, and type 2 diabetes mellitus [2].

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Currently, recommended obesity treatments focus on lifestyle modifications to include healthy diet and increased physical activity [3]. However, these recommendations often lead to food cravings and increased appetite. Research on the association of exercise with food intake suggests that exercise has an impact on not only energy expenditure but also regulation of appetite and postexercise energy balance [4].

Energy homeostasis and appetite are strongly influenced by the gut hormone ghrelin, which is the only known appetite-stimulating hormone in humans [5]. This 28–amino acid peptide is produced primarily by neuroendocrine cells in the stomach and has effects in the arcuate nucleus of the hypothalamus as well as the pituitary somatotrophs [6-8]. Ghrelin's principal physiological actions are stimulating food intake, blood glucose, and growth hormone (GH) secretion [9,10]. By promoting food intake, ghrelin also decreases energy consumption and increases body weight [11]. Research has extensively investigated the role of ghrelin in energy balance and adiposity [12-15]. Ghrelin levels are low in simple obesity and are negatively correlated to percentage body fat, but increase with weight loss [16-18]. Resistance exercise may be an innovative strategy for suppressing ghrelin and decreasing appetite during weight loss and subsequent weight maintenance.

The effects of acute exercise bouts on total plasma ghrelin have produced varied results. Most studies have investigated acute aerobic exercise in normal-weight individuals and have demonstrated either no change in ghrelin levels [19-21] or an increase [22]. Few aerobic exercise studies have been conducted in obese individuals; and the results were similar to those conducted in normal-weight individuals, showing either no increase [23] or, in one case, an increase [24]. Those studies conducted on RE have demonstrated either no change [25] or reduced total ghrelin [1,26,27]. Interestingly, of the 4 investigations on acute RE, none was performed with obese individuals. It is therefore unclear as to what effect acute RE has on ghrelin and whether obesity influences ghrelin response to exercise. In addition, it remains unclear if severity of obesity has an effect on total ghrelin. Given the limited literature on obesity level and ghrelin response to exercise and the association between GH, ghrelin, and cortisol, we hypothesize that an RE protocol would demonstrate increases in total ghrelin and cortisol in lean and obese men with more limited increases in obese individuals.

2. Methods

2.1. Study participants

The study included 19 healthy male sedentary volunteers who were free of existing acute or chronic illness, or known cardiovascular, endocrine, or metabolic disease; were not taking any medications or dietary supplements; were nonsmokers; and had not had weight loss greater than 5.0 lb in the past 3 months. Because of menstrual cycle phase causing differing GH responses to exercise, women were excluded from the current study [28-30]. Using the standards established by the World Health Organization (WHO) to classify obesity status, the participants were designated as lean if their body mass index (BMI) was less than 25 kg/m^2, obese WHO 1 classification if their BMI was between 30.0 and 34.9 kg/m^2, and WHO 2/3 classification if their BMI was greater than 35.0 kg/m^2 (Table 1). A preparticipation health history and physical activity questionnaire were used to screen all volunteers for inclusion in this study. The University of Connecticut Institutional Review Board approved all procedures, and all subjects gave informed written consent. All subjects were untrained (having not participated in a resistance training protocol for 6 months or greater) and not currently participating in any structured exercise program for more than 30 minutes 2 times per week.

2.2. Body composition analysis

Participants were weighed on an electronic scale with weight recorded to the nearest 0.1 kg, and height was measured with a standard stadiometer. Fat-free mass, fat mass, and percentage body fat were determined using dual-energy x-ray absorptiometry (GE Lunar Prodigy Advance, Madison, WI).

2.3. Study design

2.3.1. Study controls

At the acute RE protocol visit, the participants entered the laboratory after a 12-hour overnight fast. All participants performed exercise tests at the same time points between 6:30 AM and 11:00 AM (24-hour clock) after a 12-hour fast to account for circadian rhythms of hormone secretion. They had been instructed to abstain from drinking alcohol or ingesting high doses of caffeine (>2 cups per day or >150 mg) and strenuous exercise for 24 hours before the testing protocol. This was done to ensure no confounding effect on normal diurnal or exercise-induced hormonal responses. In addition, subjects were instructed to drink 0.5 L of water the night before and 0.5 L of water the morning of the experimental trials to ensure adequate hydration. Adequate hydration (urine specific gravity ≤1.020) was confirmed before the experimental protocol via urine refractometry.

Table 1 – Subject characteristics

	Lean (n = 9)	WHO 1 (n = 5)	WHO 2/3 (n = 5)
Age (y)	20.1 ± 2.1	21.6 ± 2.5	20.0 ± 1.4
Body mass (kg)	71.7 ± 5.8	97.8 ± 8.58	120.8 ± 10.49
Height (cm)	177.8 ± 8.7	176.3 ± 3.7	177.7 ± 5.15
BMI (kg/m^2)	22.8 ± 1.36*	32.3 ± 1.61*	39.4 ± 2.79*
Fat body mass (kg)	10.1 ± 2.34*	32.3 ± 3.35*	47.3 ± 10.05*
Lean body mass (kg)	59.0 ± 6.0†	61.0 ± 7.86	68.7 ± 4.8†
Percentage body fat (%)	14.7 ± 3.54*	34.7 ± 2.95*	40.5 ± 5.82*

Data are mean ± SD. Subject characteristics by group (lean vs WHO 1 vs WHO 2/3).
* Significantly different ($P < .05$) means between LN, WHO 1, and WHO 2/3 groups.
† Significantly different ($P < .05$) means between LN and WHO 2/3 groups.

2.3.2. *Familiarization and 10 repetition maximum testing*

All participants completed 5 exercise protocol visits and repeated the same RE protocol and warmup at each visit in an effort to familiarize, remove any novelty effects, and establish a 10-repetition maximum (10RM) before the acute resistance exercise testing (ARET) protocol. The RE and ARET protocols were identical and consisted of squat, bench press, leg curls, dumbbell rows, dumbbell shoulder press, and dumbbell step-up exercises, which were performed in the order listed. Squat and bench press exercises had a 120-second rest period between sets (including from warm-up to the first set). The leg curl, dumbbell row, dumbbell shoulder press, and dumbbell step-up exercises had a 90-second rest period between sets and between exercises.

For the 10RM test, participants performed 8 to 10 repetitions at approximately 50% of their estimated 10RM to warm up. The participants then performed progressive 10RM sets with increased loads until the research team determined the 10RM load. No more than 3 to 4 maximum trials were used for each participant and each exercise. Trials were separated by 2 to 3 minutes of rest. In visit 5, participants performed the RE with the weight that would be used during the ARET. Specifically, subjects performed 3 sets of each exercise at 85% to 95% of their previously determined 10RM.

2.3.3. *Acute resistance exercise test*

Participants performed ARET at least 1 week after the fifth visit. At the ARET visit, the participants entered the laboratory after a 12-hour overnight fast; and all exercise tests were performed between 6:30 AM and 11:00 AM (24-hour clock). Participants performed a warm-up on a cycle ergometer followed by a dynamic warm-up (bodyweight lunges, butt kicks, high knees, and squatting movements). Afterward, the participants performed the ARET protocol (6 exercises, 3 sets of 10 repetitions at 85%-95% 10RM with 120- and 90-second rest periods described above); and blood samples were collected pre-, mid-, and immediately postexercise and during recovery (+50, +70 and +110).

2.3.4. *Blood draws and processing*

Before commencing experimental trials, a trained phlebotomist inserted an indwelling Teflon cannula into a superficial forearm vein of the participants after they had sat comfortably for 15 minutes. The cannula was kept patent with a 10% heparin-saline solution. Blood samples were collected in a seated position at 6 time points: 20 minutes after sitting quietly and 15 minutes before the onset of exercise (PRE); halfway through the protocol immediately after the last set of the leg curl (MID); immediately post the ARET (IP); and +50, +70, and +110 minutes post the ARET. During the blood draws, MID and IP participants were seated in a wheel chair. After the exercise protocol, participants remained seated quietly for the duration of the recovery period and were not allowed to fall asleep. Time points were chosen to correspond to differences identified in prior studies [31,32]. Before each blood draw, 3 mL of blood was drawn and discarded to avoid inadvertent dilution of the blood sample. For each blood draw, 10 mL of blood was collected and transferred into appropriate tubes and centrifuged at 1500*g* for 15 minutes at –4°C. Resulting serum and plasma were aliquoted, flash frozen in liquid nitrogen, and immediately stored at –80°C until analysis.

2.3.5. *Biochemical analyses*

Serum samples were analyzed in duplicate for insulin (Calbiotech, Silver Springs, CA) GH, and cortisol (Diagnostic Systems Laboratory, Webster, TX) via commercially available enzyme linked immunosorbent assays. Coefficient of variation for these assays was 2.2%, 6.9%, and 4.5%, respectively. All samples were analyzed in one assay to avoid interassay variation. Plasma ghrelin concentrations were measure via radioimmunoassay (ALPCO Diagnostics, Salem, NH). The intraassay coefficient of variation averaged less than 5%, and all samples were analyzed within the same assay. The plasma glucose levels were measured via glucose oxidase reagent (Pointe Scientific, Canton, MI).

2.4. *Statistical analyses*

All data were assessed for violations of normality and homogeneity of variance. For those variables that violated assumptions, data were log transformed to comply with assumptions before analysis. A mixed repeated-measures analysis of variance (ANOVA) (group × time) was performed for each hormonal response. When significant main effects or interaction effects were observed, Sidak-adjusted pairwise comparisons were used to examine the significant mean differences. Area under the curve (AUC) was computed for each hormone and analyzed using a one-way ANOVA. Given that the AUC quantifies the curvilinear nature of the hormonal response to the exercise stimulus throughout the timeline of the protocol, the AUC was used as a linear measure of hormonal response; and correlations were examined separately within the lean and obese groups (n = 9, n = 10). Pearson product-moment correlation coefficients were used to investigate possible significant associations between total ghrelin and other hormones. Data are given as the mean ± SEM, and statistical significance was assumed for $P \leq .05$. Effect sizes were reported using the η^2 statistic, which reflects the portion of variance accounted for by that effect [33]. Conventions for describing η^2 effect sizes are as follows: small = .01; medium = .06, and large = .14 [34]. All analyses were performed using SPSS statistical software package (version 17.0; Chicago, IL).

3. Results

3.1. *Participant characteristics*

Anthropometric measurements and dual-energy x-ray absorptiometry analysis were used to compare groups of subjects on body composition variables. As expected, significant differences were found between groups for body composition variables (multivariate ANOVA $F = 9.24$; $df = 14, 22$; $P < .001$; $\eta = .86$). Significant differences between group means are indicated in Table 1. In addition, differences in weight and volume by group were analyzed. No significant differences between groups were identified for 10RM weights

for each of the exercises performed (η^2 range = .04-.11). Total volume lifted (weight lifted in kilograms × number of repetitions × sets) was computed and also submitted for analysis. Again, no differences were found between groups for total volume (F = .755; *df* = 2, 18; *P* = .49; η^2 = .09). No differences were found between groups for volume for any individual exercise (η^2 range = .02-.14). Overall, these results indicate that the total work performed between groups was similar.

3.2. *Total ghrelin*

Our goal was to determine whether changes in total ghrelin levels induced by a single bout of high-volume RE differed between obese young adult men and age-matched controls of normal body weight. Total ghrelin levels during exercise did not differ from those at rest. However, the main effect of group was significant (F = 10.101; *df* = 2, 16; *P* = .001; η^2 = .56). Sidak pairwise comparisons indicate that the WHO 2/3 group had significantly greater ghrelin levels than the lean group (*P* = .009) and the WHO 1 group (*P* = .002; Fig. 1). These results suggest that obesity level may demonstrate differing total ghrelin response during acute RE.

3.3. *Cortisol*

Fig. 2 shows the time course of cortisol in all 3 groups. At rest, no differences existed between the groups, although a significant time by group interaction was observed (F = 2.94; *df* = 8, 64; *P* = .007; η^2 = .27). Resistance exercise produced a significant increase over time in cortisol across all groups. Significant differences were found between WHO 1 and WHO 2/3 at time point +70 (*P* < .05) and between WHO 1/LN and WHO 2/3 at time point +110. In addition, the preexercise time points for the lean and WHO 1 groups differed from all other time points (*P* < .05). However, the preexercise time point in the WHO 2/3 obese group differed from immediate postexercise and +50 only.

3.4. *Glucose*

Considerable evidence suggests that ghrelin stimulates blood glucose, which in turn would increase overall insulin levels [7,35]. Resistance exercise did increase glucose in all groups, albeit with differing effects in group over time (F = 2.63; *df* = 5, 80; *P* = .03; η^2 = .14). Across groups, glucose concentrations at midexercise were significantly greater than preexercise and +50, +70, or +110 postexercise concentrations (*P* < 05). There were no differences between groups at any time point. Mean concentrations are presented in Fig. 3.

3.5. *Insulin*

Insulin levels were different between the lean and obese groups at rest and increased during the RE protocol, with different effects by group over time. (F = 2.46; *df* = 10, 80; *P* = .013; η^2 = .24). Insulin levels were significantly different at midexercise when compared with levels at preexercise, postexercise, and +70 and +110 time points (*P* < .05 for all comparisons). The 3 groups differed at the pre and mid time points (Fig. 4). The WHO 1 and WHO 2/3 groups had significantly greater preexercise insulin than the LN group (*P* < .05), and the WHO 2/3 group had significantly greater insulin peak midexercise compared with the LN group (*P* = .01).

3.6. *AUC analysis*

The means and standard deviations of AUCs for ghrelin, insulin, and cortisol for lean, WHO 1 and WHO 2/3 groups are presented in Table 2. One-way ANOVAs for each variable are described below.

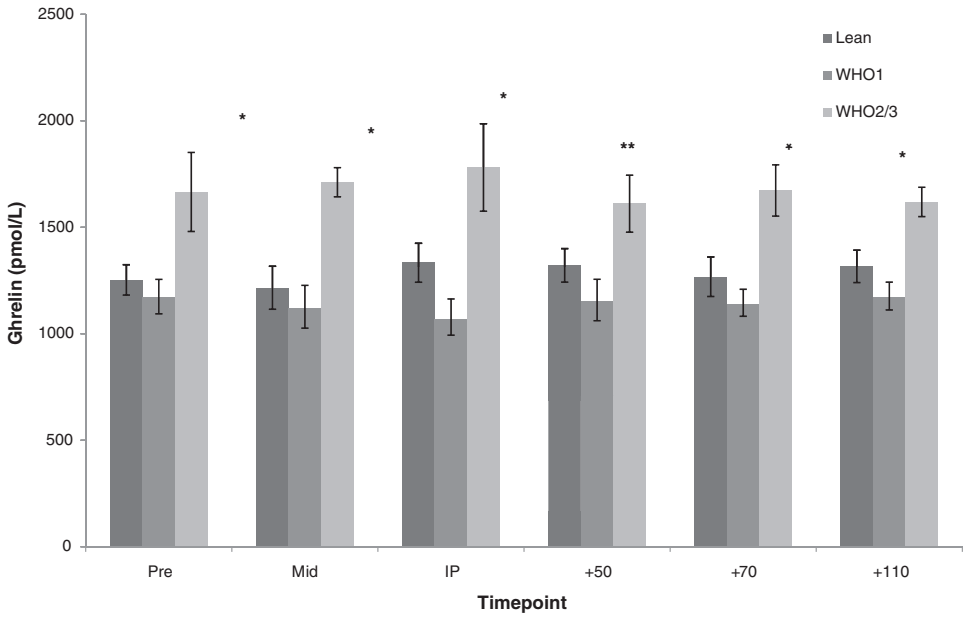

Fig. 1 – Ghrelin response by group (lean vs WHO 1 vs WHO 2/3). Note: *P* < .05. *Significantly different from lean and WHO 1 groups. **Significantly different from WHO 1 group.

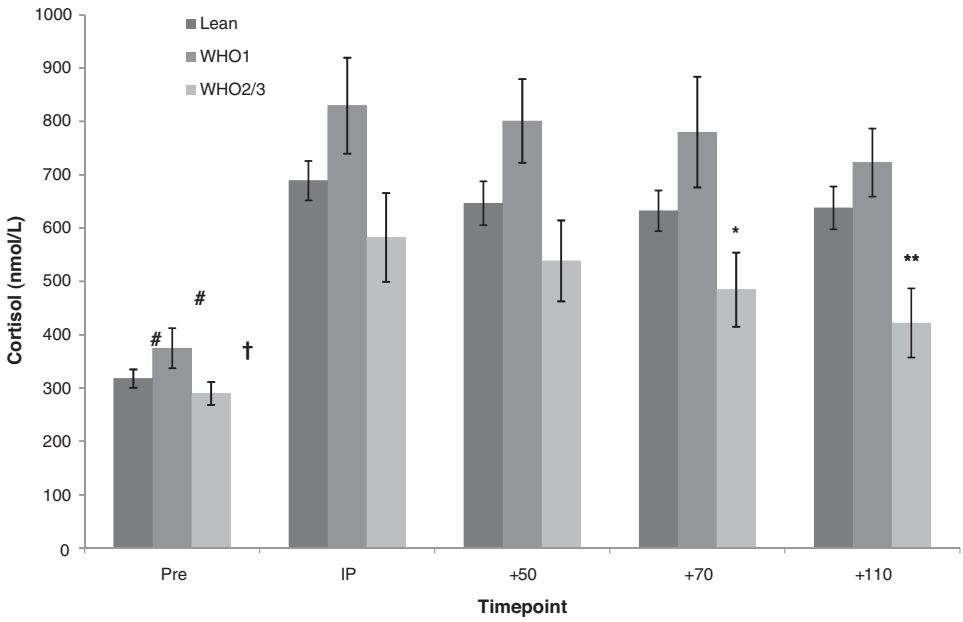

Fig. 2 – Cortisol response by group (lean vs WHO 1 vs WHO 2/3). Note: P < 05. #Significantly different from corresponding pre value. *Significantly different from WHO 1 group. ##Significantly different (P < 05) from WHO 1 and lean groups.

3.6.1. *Ghrelin*

The between-groups effect was significant and replicated the mixed ANOVA main effect for group (F = .780; *df* = 1,17; *P* = .389; η^2 = .044). Sidak-adjusted comparisons examined differences in AUC between groups and indicated significantly greater ghrelin AUC in the WHO 2/3 group than in the LN group (*P* = .008) and the WHO 1 group (*P* = .001).

3.6.2. *Insulin*

There were no significant differences between groups in Insulin AUC (F = 1.85; *df* = 2, 18; *P* = .19).

3.6.3. *Cortisol*

Analysis of cortisol AUC indicated a significant effect of group (F = 3.91; *df* = 2, 18; *P* = .041; η^2 = .329). The WHO 1 group had

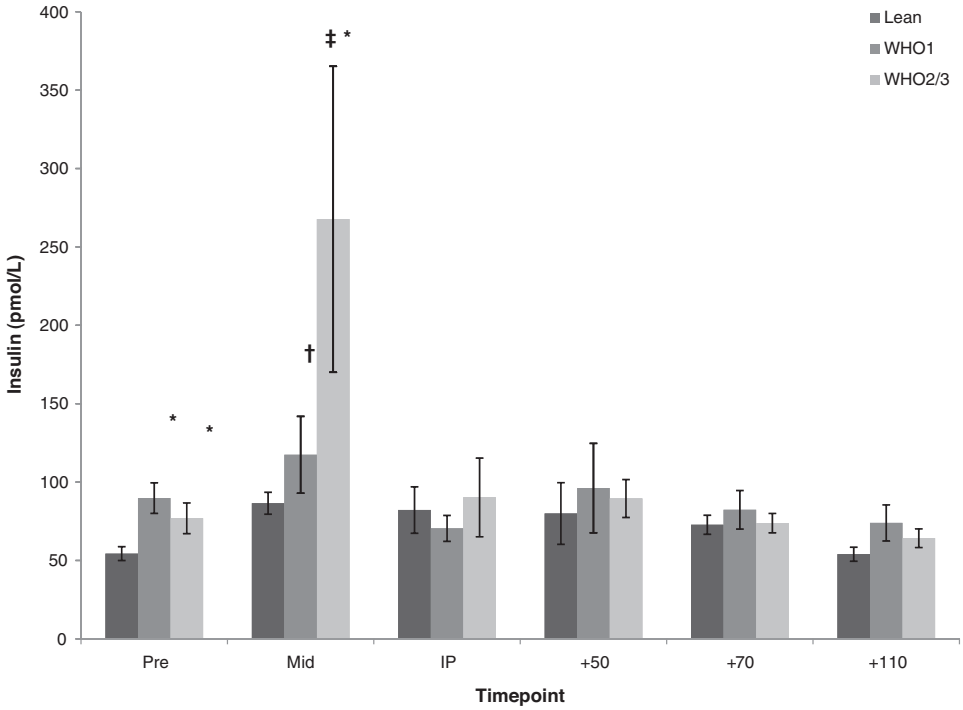

Fig. 3 – Glucose response by group (lean vs WHO 1 vs WHO 2/3). Note: P < 05. #Significantly different from preexercise value.

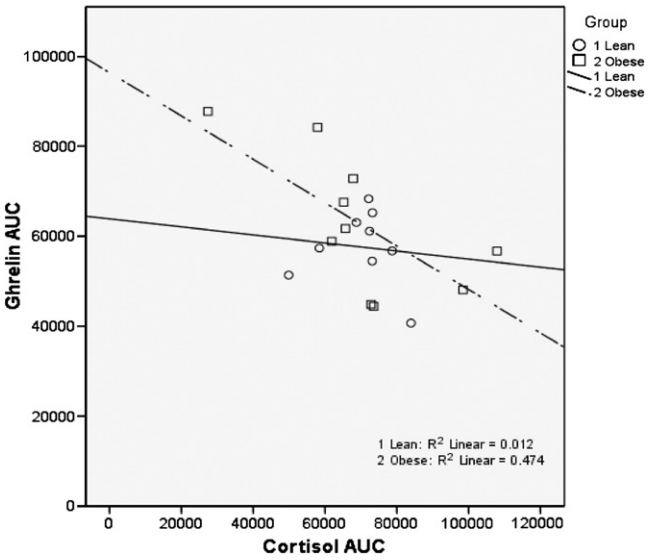

Fig. 4 – Insulin response by group (lean vs WHO 1 vs WHO 2/3). Note: P < .05, #Significantly different from pre value. *Significantly different from corresponding lean value.

significantly greater AUC than the WHO 2/3 group ($P = .038$). No differences were found between the LN and WHO 1 groups.

3.7. *Correlations*

To understand the extent to which the measured parameters were related, we compared total ghrelin to the glucoregulatory hormones and GH using AUCs examined separately in lean vs obese men. Growth hormone concentrations were reported in a previous analysis of these data [36] and are included here for comparative purposes. In lean individuals, ghrelin was inversely associated with GH ($r = -.76$, $r^2 = .58$, $P = .018$). In addition, in lean subjects, the association of cortisol to ghrelin was not significant ($r = -0.11$, $r^2 = .01$, $P = .78$). In obese subjects, there was no significant correlation of ghrelin and GH ($r = 0.02$, $r^2 = .00$, $P = .97$); however, ghrelin to cortisol was inversely associated ($r = -.69$, $r^2 = .47$, $P = .028$). These correlations demonstrate that alterations in adiposity are associated with differential associations between glucoregulatory variables.

Table 2 – Plasma glucose responses to RE

	Glucose (mmol/L)			
	Lean	Obese	Obesity subgroups	
			WHO 1	WHO 2/3
PRE	4.61 ± 0.41	5.04 ± 0.91	4.85 ± 1.17	5.24 ± 0.62
MID	4.95 ± 0.70 *	5.64 ± 0.96 *	5.27 ± 1.27 *	6.02 ± 0.37 *
IP	4.93 ± 1.10	5.43 ± 1.10	5.09 ± 1.64	5.64 ± 0.64
+50	4.83 ± 1.14	5.18 ± 1.0	5.03 ± 1.28	5.32 ± 0.75
+70	4.51 ± 0.65	5.35 ± 0.80	5.20 ± 1.30	5.29 ± 0.41
+110	4.09 ± 1.06	5.05 ± 1.02	4.98 ± 1.41	5.12 ± 0.61

Data are mean ± SD.
* Significant ($P < 05$) difference from corresponding preexercise value.

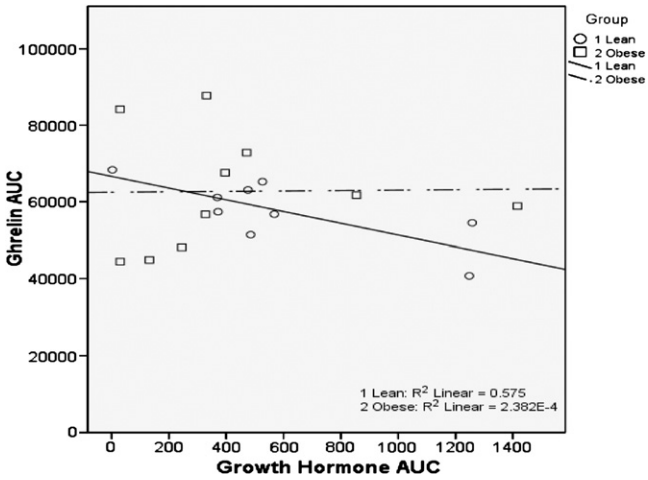

Fig. 5 – Linear correlations between total ghrelin (AUC) vs cortisol (AUC) in lean (n = 9) and obese (n = 9) young adult men.

Fig. 5 depicts the linear associations reported above in lean vs obese men between total ghrelin AUC and cortisol AUC, and Fig. 6 depicts the linear associations reported above in lean vs obese men between total ghrelin AUC and GH AUC.

4. Discussion

In the present study, we evaluated whether a single bout of high-intensity RE would alter levels of total ghrelin, cortisol, glucose, and insulin differently in lean and in WHO 1 and WHO 2/3 obese male subjects. Contrary to our hypothesis, ghrelin did not increase in response to RE. Although several studies have investigated aerobic exercise, few studies have used RE as a method to assess total ghrelin response.

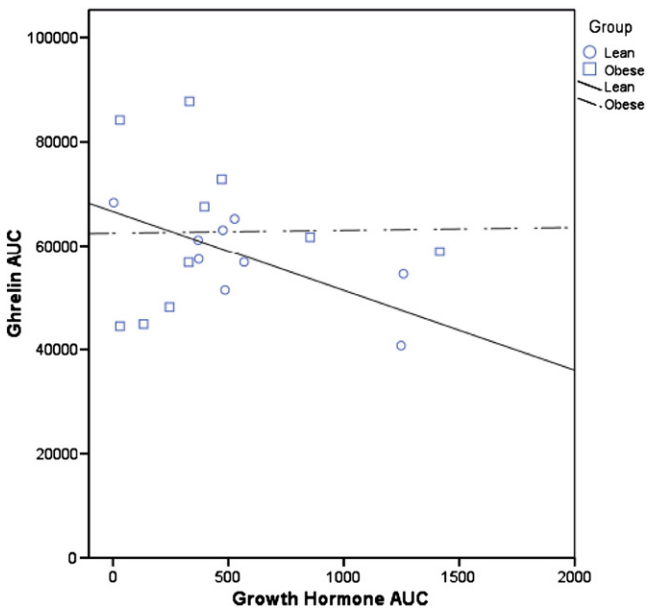

Fig. 6 – Linear correlations between total ghrelin (AUC) vs GH (AUC) in lean (n = 9) and obese (n = 9) young adult men.

Espelund et al [17] noted that the extent of obesity may be moderating whether significant differences in ghrelin levels are found between lean and obese subjects. Our findings support this hypothesis in that only WHO 2/3 obese men had significantly greater concentrations of ghrelin when compared with lean and WHO 1 obese groups. Future research on associations between ghrelin and cortisol and on alterations in ghrelin concentrations in obese subjects should be careful in specifying the extent of obesity in the study subjects.

In this study, the exercises were chosen not only for their ability to produce the desired hormonal response, but also because they represent upper body and lower body exercises commonly used in normal resistance training routines. Several investigators have noted the importance of including both upper and lower (small and large muscle group) body exercises in a GH-stimulating acute protocol [37,38]. Both small and large muscle group exercises were chosen for the RE to properly stimulate GH [37,38]. Previous research indicates that loads that fall below this level would not adequately stimulate GH response [39]. Although some studies have provided evidence that ghrelin levels are suppressed following moderate-intensity acute RE [40], most evidence has been examined following short-term (<4 weeks) exercise; and these studies have demonstrated no change in plasma ghrelin levels during or after exercise, regardless of the exercise intensity and type [11,19-21,23,41-45].

Aerobic exercise in both fed and fasted state has been reported not to alter ghrelin concentrations [42]. However, concentric RE has shown reductions in overall ghrelin values in untrained men [25]. Overall, the current findings suggest no acute response to exercise stimuli. However, ghrelin results for the 3 groups suggest that the increased adiposity has altered the homeostatic endocrine responses. Based on prior research on ghrelin in response to exercise, the WHO 1 and lean groups exhibit ghrelin responses consistent with lean male college students [27] This finding supports the contention that the WHO 1 obese group has simple obesity, or an excess energy state without endocrine dysregulation. However, the WHO 2/3 group has elevated ghrelin that is believed to be due to the upregulation of insulin in response to physiological adaptations resulting from increased, sustained adiposity.

The level of obesity and BMI has been shown to be an important indicator in hormonal response. If obese and lean individuals in the present study had been examined without consideration of level of obesity, it is likely that differences in ghrelin would not have emerged. This may account for divergent findings in ghrelin response in obese individuals in prior literature. This distinction was also noted by Espelund et al [17] who concluded that mildly overweight individuals seemed to have similar ghrelin concentrations as lean participants. Our findings by BMI are consistent with those in other studies where subjects had mean BMI greater than 35 kg/m^2 [46].

Although prior research by Scacchi et al [47] concluded that insulin is not related to GH secretion in obese subjects, more recent findings point to insulin as a mechanism underlying the inhibitory effects of obesity on GH secretion [10,29,38,39,48]. Both glucose and insulin increased significantly from preexercise time points in all groups by the mid time point. These changes are consistent with prior RE research [48,49]. In response to exercise, insulin concentrations increased because of elevated concentrations of glucose and presumably amino acids and free fatty acids [50]. In addition, insulin was not correlated with ghrelin in the lean or obese groups. This finding is consistent with the other studies that have only found this correlation in type 2 diabetes mellitus patients with adjustment for BMI [8,51].

Peripherally, cortisol increases lipolysis in adipose tissue cells and increases protein degradation and, alternately, decreases protein synthesis in muscle cells, causing a release of lipids and amino acids into circulation [38]. The acute exercise response of cortisol has been shown to be significantly influenced by the acute program variables selected [30]. The acute program variables dictate the magnitude of the hormonal response RE can elicit [51,52]. Acute RE program variables include the (1) choice of exercises, (2) amount of resistance used (load), (3) volume (total number of sets, repetitions, and load), (4) the sequence of exercises performed, and (5) rest intervals between sets and exercises [38,51,53]. Protocols that use higher number of sets and lower rest periods have been shown to elicit significantly larger cortisol responses than those with lower sets and longer rest periods [38]. Our results show changes in cortisol concentration from preexercise values. Interestingly, the WHO 2/3 group exhibited significantly blunted cortisol in comparison with the lean and WHO 1 groups. This overall lower cortisol response could be due to chronically high cortisol concentrations and blunted stress responses that the literature has reported in obese individuals [54].

It is important to point out an important limitation of the present results. We measured total ghrelin levels only; and as such, we report the resulting levels of both acylated and des-acyl ghrelin. Because acylated ghrelin accounts for less than 10% of total circulating ghrelin and performs most of the functions of ghrelin, our measurement will likely include molecules possessing ghrelin-like immunoreactivity [55]. Most RE protocols examining ghrelin response have measured total ghrelin; therefore, to allow comparison with other studies, we decided to measure total ghrelin in this sample. Future research should seek to measure total ghrelin, acylated ghrelin, and des-acyl ghrelin to note the exact nature of disruptions in homeostasis in obese populations. In addition, further research examining acylated and des-acyl ghrelin should also explore the effects of exercise-induced stress and different obesity statuses on the activity of ghrelin-O-acyltransferase, which may serve a role in reducing concentrations of acyl ghrelin [56]. An additional limitation of this study is the lack of measurement of leptin. Obese individuals have chronically high levels of fasting leptin, which has been shown to blunt the hypothalamic-pituitary-adrenal axis response of obese individuals [57].

Future studies should examine the effects of longer exercise interventions and replicate these findings in obese women and older adults who may be at risk for comorbidities due to their obesity. In addition, most prior investigations have been done in overweight or WHO 1 obese individuals. Future research should carefully consider the level of obesity, and investigations should recruit and classify subjects by specific BMI to increase understanding of homeostatic

dysregulation that occurs with increasing obesity. Further investigations are also needed to fully understand the relationship between cortisol and ghrelin.

In conclusion, during a high-volume RE protocol, obese and lean individuals exhibited differing ghrelin responses depending on level of obesity. These findings may not be clear when only examining a general obese group. By dividing the obese population by degree of obesity, clear distinctions in ghrelin concentrations and cortisol response to an RE protocol become apparent.

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Conflict of Interest

The authors report no conflicts of interest.

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