Lower Excess Postexercise Oxygen Consumption and Altered Growth Hormone and Cortisol Responses to Exercise in Obese Men

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Context: Obesity is associated with altered patterns of substrate utilization at rest and during exercise.

Objective: The relationship between obesity and fat oxidation during recovery from exercise was examined.

Hypothesis: The postexercise shift toward fat oxidation is blunted in the obese state, reflected by higher respiratory exchange ratio (RER), blunted GH, and increased cortisol values compared with lean controls.

Design: Each subject completed two 160-min protocols (baseline and exercise). During baseline, subjects rested for 160 min; during exercise, they completed 30 min of cycling at ventilatory threshold, followed by 130 min of rest.

Setting: This study was performed at the University of Alberta.

Subjects: Healthy untrained (maximal oxygen consumption, <45 ml/kg/min or <3.35 liter/min) lean (<16% body fat; n = 6) and obese (>25% body fat; n = 7) men, aged 30–39 yr, were studied.

Main Outcome Measures: RER, GH, cortisol, oxygen consumption, heart rate, tympanic temperature, and lactate were obtained during both protocols at matched time intervals and analyzed by repeated measures ANOVA.

Results: During baseline, there were no differences detected between lean and obese groups for any of the measured variables. In contrast, during exercise, peak GH levels were blunted (P < 0.05) and cortisol levels were elevated (P < 0.05) in the obese compared with the lean subjects, but RER values were similar in the two groups. The differences in GH and cortisol persisted during the postexercise period accompanied by higher RER values (P < 0.05) and reduced total oxygen consumption (P < 0.05) in the obese group.

Conclusion: These findings indicate that exercise-induced fat oxidation is diminished in obese men. (J Clin Endocrinol Metab 91: 678–686, 2006)

The recovery period after exercise presents a unique opportunity to investigate substrate oxidation, because the upper limit of fat oxidation can be elicited by exercise. During the recovery period, a shift from carbohydrate (CHO) to fat as a fuel substrate occurs to prevent further depletion of muscle glycogen stores. This is supported by measures of the respiratory exchange ratio (RER; the ratio of carbon dioxide production relative to oxygen consumption) that are often lower after exercise (1, 2). The corresponding observation that oxygen consumption remains elevated above resting levels after exercise, called excess postexercise oxygen consumption (EPOC) (3), probably reflects this shift toward fat utilization, because the oxygen cost of fat catabolism is greater than that of CHO catabolism.

Although some suggest that the obese state is associated with reduced fat oxidation and increased glucose metabolism (4–9), others report an increased reliance on fat use during exercise (10, 11). Both higher (12) and lower (10, 11) RER values have been derived from whole-body calorimetry studies. Direct measurement of fat metabolism has also been used to assess fat oxidation rates, but these results are equally controversial due to difficulties with accurate identification of free fatty acids sources and accounting for reesterification rates. To our knowledge, there are no studies that have compared the postexercise period, which is characterized by enhanced fat oxidation, in lean and obese men. Examining substrate use during this recovery phase would help clarify the influence of excess body fat on fat oxidation.

The mechanisms underlying substrate utilization in response to exercise are complex. CHO and fat metabolism during exercise is influenced by many factors, such as gender, fitness level, nutritional status, physical activity, level of adiposity, altered neurohormonal control over metabolism, and others (4, 6, 13, 14). GH and cortisol are key hormones involved in the pathogenesis of obesity. Compared with nonobese individuals, GH levels at rest may be similar or lower in obese subjects, but in response to exercise are blunted (15–20). Although resting cortisol levels in the obese may be higher compared with those in lean individuals (15, 21), its response to exercise is not well documented. High-intensity, long-duration exercise increases cortisol concentrations; some have documented similar responses in the nonobese and obese subjects (15, 16, 22), whereas others (18) observed a significantly greater cortisol response in the obese compared with the lean subjects. Contradictory findings may be due to differences in prescribed exercise duration and volume.

Abbreviations: CHO, Carbohydrate; EPOC, excess postexercise oxygen consumption; HR, heart rate; RER, respiratory exchange ratio; VO2max, maximal oxygen consumption; VT, ventilatory threshold.

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intensity (2, 22, 23). Although the GH response to exercise in the obese is clear, additional investigations are needed to clarify the cortisol response.

Most exercise studies examining substrate utilization in the obese have collected their data only during the exercise bout, yet it may be during the postexertional period that differences in metabolic fuel selection emerge. Therefore, the purpose of this investigation was to compare the RER, GH, cortisol, and EPOC responses to exercise and during recovery in obese and lean men.

Subjects and Methods

Subjects

Healthy, untrained (<45 ml/kg/min and less than three sessions per week of physical activity) men between the ages of 30 and 40 yr agreed to participate in this study. Seven obese subjects (>25% body fat) (24) and six lean controls (<16% body fat) were studied. The characteristics of the subjects are listed in Table 1. Self-report questionnaires established participants to be nonsmoking, free from diabetes, hypertension, and coronary heart disease, and they were not taking medications known to influence metabolism. All subjects were weight stable (±5 kg) at least 3 months before our study. An institutional ethics review board at University of Alberta approved this study, and all volunteers provided written informed consent before participation.

Procedures

Figure 1 provides an overview of the sequence of tests the volunteers were asked to complete. Eligibility for the study was determined by the measurement of percent body fat (hydrostatic weighing) and aerobic fitness [maximal oxygen consumption (VO2max), graded exercise test].

Percent body fat

Percent body fat was estimated using hydrostatic weighing following established protocols (25). Residual lung volume was determined using the helium dilution technique (26).

Aerobic fitness

VO2max was assessed using a standardized incremental exercise protocol (27) performed on a Monarch 818E cycle ergometer (Monarch, Stockholm, Sweden). Subjects were instructed to avoid vigorous exercise during the baseline protocol (27) performed on a Monarch 818E cycle ergometer (Monarch, Stockholm, Sweden). Subjects were instructed to avoid vigorous exercise during the baseline protocol, subjects rested quietly while measurements of VO2, RER, HR, tympanic temperature, lactate, GH, and cortisol were made and used as reference values. Typically, preexercise hormonal levels are used as baseline values, yet this approach neglects the circadian rhythm of cortisol and may underestimate the cortisol response to exercise; therefore, hormonal profiles were assessed on a separate occasion from the exercise intervention, but at the same time of day, to establish a reference baseline (29).

The exercise protocol included a 30-min (time 0–30) exercise bout on a cycle ergometer set at an intensity level equivalent to the subjects’ individual VT. The power output corresponding to VT was determined from the VO2max test according to the ventilatory equivalent for CO2 (VE/VO2) method (30). An intensity level set at VT was selected because key hormonal responses and possibly a prolonged EPOC are elicited at higher intensity exercise (>60% VO2max) and/or >20 min (2, 22, 31). As well, Frey et al. (31) suggest that the variability in EPOC results may be reduced by setting individual workloads relative to VT.

For both protocols (separated by at least 1 wk) subjects arrived at the laboratory between 1400 and 1500 h after a 4-h fast, and an indwelling catheter was inserted into the antecubital vein. The first blood draw was made at this time (time −30). For both the baseline and exercise protocols, subjects remained seated for 30 min, after which (0 min) were connected to the MedGraphics CRX-D metabolic cart (Minneapolis, MN). During the baseline protocol, subjects were seated for 50 min (time 50), and during the exercise protocol, they completed 30 min of exercise at VT, followed by a 20-min rest. A 10-min break followed (time 50–60), after which measurements for both protocols resumed with the subject lying supine with his head positioned under a canopy for the remainder of the collection period (time 60–130). Tympanic temperature (Thermoscan infrared thermometer, Braun, Kronberg, Germany), HR (Polar heart rate monitor), and blood samples were taken at time −30, 0, 30, 50, 70, 90, 110, and 130. Calibration of the metabolic cart was performed every 20 min.

EPOC

The volume of oxygen consumed above resting levels during the recovery period after exercise denotes the EPOC (3). EPOC magnitude was calculated by summing excess VO2 during the recovery period (the difference between exercise and baseline; time 30–130). The cessation of EPOC was defined as 2 or more consecutive minute values of oxygen consumption at or below the baseline VO2 (32, 33). Based on the literature, our exercise protocol was not anticipated to elicit an EPOC duration greater than 2 h (14, 34, 35).

RER

RER was monitored continuously and was calculated from expired gases. Steady-state RER was obtained by averaging each 5-min interval.

TABLE 1. Characteristics of obese and lean subjects

<table>
<thead>
<tr>
<th></th>
<th>Obese (n = 7)</th>
<th>Lean (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>36.1 ± 3.4</td>
<td>34.5 ± 2.6</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>179.9 ± 3.7</td>
<td>175.1 ± 10.1</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>103.3 ± 11.4*</td>
<td>66.3 ± 7.4</td>
</tr>
<tr>
<td>BMI (kg/m2)</td>
<td>31.9 ± 3.88</td>
<td>21.7 ± 1.7</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>33.5 ± 8.3*</td>
<td>10.2 ± 1.4</td>
</tr>
<tr>
<td>FFM (kg)</td>
<td>69.8 ± 7.2*</td>
<td>56.1 ± 6.1</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>32.2 ± 5.7*</td>
<td>15.4 ± 1</td>
</tr>
<tr>
<td>VO2max (liter/min)</td>
<td>3.2 ± 0.5*</td>
<td>2.7 ± 0.3</td>
</tr>
<tr>
<td>VO2max (ml/kg/min)</td>
<td>31.3 ± 6.1*</td>
<td>41.8 ± 4.4</td>
</tr>
<tr>
<td>VO2max (ml/kg/FFM/min)</td>
<td>46.2 ± 5.7</td>
<td>48.7 ± 4.6</td>
</tr>
<tr>
<td>VO2max at VT (%)</td>
<td>67.4 ± 5.3</td>
<td>68 ± 8.2</td>
</tr>
<tr>
<td>PO at VT (watts)</td>
<td>154 ± 35</td>
<td>155 ± 34</td>
</tr>
</tbody>
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Values are means ± SD. FFM, Fat-free mass; PO, power output. * Significantly different from lean (P < 0.05).
Hormone and lactate levels
A total of eight 3-ml blood samples were collected on the following schedule: time 0, 30, 60, 90, 120, and 180 min. From each blood sample, 0.5 ml was removed to analyze the blood lactate concentration in duplicate using a Phillips PU 8800 UV/VIS spectrophotometer (Pye Unicum Ltd., Cambridge, UK) following established methods (36). The remaining blood (2.5 ml) was allowed to clot at room temperature for 45 min, then was centrifuged (10 min at 2500 rpm), and serum aliquots were stored at −80°C until analyzed. Hormonal assays (GH and cortisol) were performed on serum samples in duplicate in a single assay using commercially prepared RIA kits (GammaCoat [125I], Diasorin, Stillwater, MN). Lower limits of detection for GH and cortisol were 0.4 ng/ml and 0.2 μg/dl, respectively. The intraassay coefficients of variation were 10.5% and 6% for GH and cortisol, respectively (37).

Data analysis
Results are reported as the mean ± sd. Student’s t test was used to compare subject characteristics, including their energy and macronutrient intakes. Average oxygen consumption across the baseline period for the obese and lean men was also compared by Student’s t test and served as the groups’ respective reference values upon which oxygen consumption during the exercise period was assessed.

A two-way ANOVA (obese vs. lean) with one factor repeated measure (time) was used to evaluate GH, cortisol, RER, temperature, and lactate responses between groups during the baseline and exercise periods. If significance was documented, values at single time points were compared by one-way ANOVA. Recovery was defined as the time point at which baseline and exercise values were not statistically different from each other. For all statistical procedures, the significance level was set at P < 0.05.

VO2 and RER data for one lean subject were not analyzed from the baseline period for the obese and lean groups, respectively. On the exercise day, all variables were similar between groups before the start of exercise (time 0). In response to and after exercise, differences between groups were detected for VO2, RER, HR, tympanic temperature, and lactate. Figures 2A and 3A illustrate circulating levels of GH and cortisol, respectively, between obese and lean men during the baseline period. Figure 4, A and B, plots the RER values across the baseline period for the obese and lean groups, respectively.

Exercise protocol
The response to exercise stimulation was compared between obese and lean men. On the exercise day, all variables were similar between groups before the start of exercise (time 30). In response to and after exercise, differences between groups were detected for RER, GH, cortisol, and EPOC. HR, tympanic temperature, and lactate were not different across the exercise session.

Exercise bout. Both groups exercised at the same relative intensities (VT) and similar power outputs (obese, 154 ± 35 W; lean, 155 ± 34 W) during the submaximal exercise bout. The average VO2 during the 30-min exercise load was similar in obese and lean groups (36.4 ± 34 ml/kg FFM/min, respectively). As well, HR (obese, 157 ± 13.6; lean, 154 ± 18.8 beats/min), lactate (obese, 4.6 ± 1.8; lean, 6.0 ± 2.9 μmol/l), temperature (obese, 36.4 ± 0.7; lean, 36.9 ± 1.2°C), and RER (obese, 0.93 ± 0.08; lean, 0.95 ± 0.03) during the exercise load (time 30) were not significantly different between groups.

VO2. In response to exercise, the magnitude of EPOC in the lean group (absolute, 4.2 ± 0.9 l; relative, 76.0 ± 15.0 ml/kg FFM) was 28% greater than that in the obese group (absolute, 3.0 ± 1.3 l; relative, 43.9 ± 20.2 ml/kg FFM; P < 0.05). The duration of EPOC was also significantly longer in the lean
group (46.8 ± 6.7 min) compared with the obese (27.9 ± 14.8 min; Fig. 5).

RER. In response to exercise and during the recovery period, RER in the obese group remained significantly higher than that in the lean group (Fig. 4C). Steady-state RER values revealed significant differences between lean and obese subjects during the following recovery times: time 60–65 (P = 0.03), time 65–70 (P = 0.018), time 70–75 (P = 0.003), time 75–80 (P = 0.003), time 115–120 (P = 0.054), and time 125–130 (P = 0.024). This accounts for 30 of 100 min (30%) of the recovery period.

Hormones. During exercise and throughout the recovery period, the lean group had higher GH concentrations compared with the obese group (P < 0.05). The peak GH responses to exercise were 44.0 ± 22.2 and 13.4 ± 12.2 ng/ml and for the lean and obese subjects, respectively (P < 0.05). Figure 2B illustrates the GH response during exercise. In contrast, cortisol levels in the obese were greater than those in the lean (P < 0.05). Peak concentrations were noted at time 30 and were 25.5 ± 7.7 μg/dl in the obese and 13.1 ± 5.3 μg/dl in the lean (P < 0.05). Figure 3B illustrates the cortisol response for the lean and obese groups during the exercise session.

Baseline vs. exercise: time required for recovery

In response to exercise, significant differences from baseline were detected in GH, cortisol, VO₂, HR, and lactate, which reached peak values at the end of exercise (time 30). Compared with baseline, significantly lower RER values were detected in the lean group only. In the lean group, RER was significantly lower at time 60 through time 130 (with
exception of time 86–95; Fig. 4B). RER in the obese group was not statistically different from the baseline (Fig. 4A).

Figure 5 illustrates the recovery time points for each measured variable and EPOC across the 160-min sampling period. In lean men, heart rate levels returned to baseline before the cessation of EPOC, whereas blood lactate, RER, cortisol, and GH levels returned to baseline between time 90 and time 130. In contrast, blood lactate and GH levels returned to baseline near the end of EPOC, whereas heart rate and cortisol levels remained elevated until time 130.

Discussion

The purpose of this study was to investigate the relationship between obesity and fat oxidation during recovery in a group of lean and obese men completing a standardized bout of exercise. Our hypothesis, that the shift toward fat oxidation in the postexercise period is blunted in the obese state, was confirmed. Findings from our study showed higher RER, blunted GH, increased cortisol values, and smaller EPOC compared with lean controls.

During the baseline condition, there were no detectable differences between the lean and obese groups. Resting RER and concentrations of GH and cortisol were similar between our lean and obese men, agreeing with the findings of others (5, 16, 19). During the exercise condition, only GH and cortisol levels became significantly different between groups, whereas all other variables were similar in their responses to exercise. Compared with our lean subjects, we observed lower GH and higher cortisol levels in the obese subjects, and these have been documented by others (18).

During the postexercise period, the hormonal differences between the lean and obese men persisted, accompanied by a significant drop in RER in the lean, but not the obese, group. In addition, the time for HR, lactate, and EPOC to return to baseline values differed between groups.

A possible interpretation of our findings is that the post-
exercise reduction in fat oxidation and differences in hormonal profiles may be due to insulin resistance, a condition that often exists in obese individuals (10). Our subjects reported themselves to be free from diabetes, but insulin sensitivity was not evaluated. Braun et al. (10) examined insulin resistance in overweight, exercising women and noted that less CHO was used during exercise, probably related to sparing muscle glycogen and possibly low initial muscle glycogen stores (10). Substrate utilization during the postexercise period was not assessed in this study, but is typically characterized by preferential use of fat to spare further depletion of muscle glycogen (2, 38); hence, it may be possible that the lower CHO and higher fat use observed during exercise in the overweight women would continue after ex-

**Fig. 4.** Time plots of mean RER values in obese (n = 7; • and ▼; A) and lean (n = 5; ○ and △; B) subjects during baseline (160 min of rest) and exercise (30 min of rest, 30 min of exercise, 100 min of recovery) conditions. C, Comparison of RER responses in obese and lean subjects during exercise. Error bars (SD) have been omitted. Asterisks denote a significant difference (P < 0.05).
exercise. This remains speculative, but even if some aspect of insulin resistance does explain, at least partially, the observed shift in substrate use during exercise, the obese men in our study did not demonstrate the shift away from CHO toward greater fat use that Braun et al. described (10).

Although differences in RER were detected during a 50-min submaximal (45% VO2max) bout of exercise completed by insulin-sensitive and insulin-resistant women (10), RER values in our lean and obese men remained similar across a 30-min bout of exercise performed at an average of 67–68% VO2max. If insulin resistance helps explain differences in RER at a lower intensity of exercise, it would be reasonable to expect a higher intensity of activity to result in the same divergence in RER. We cannot confirm the absence of insulin resistance in our subjects, yet our RER findings would suggest that this is not contributing to our results.

Although our baseline and exercise conditions were not randomized, the likelihood of our results being distorted is small, because the time period between protocols was sufficient to eliminate any possible residual effect of the testing procedures. As well, before the baseline testing period, subjects were familiarized with the laboratory setting and testing protocols when completing their maximal oxygen consumption test.

The GH response to and after exercise in our obese men was diminished compared with that in the lean controls. The lower GH response to exercise in the obese is well established (9, 15–18), but during the recovery period is less clear. After exercise of sufficient intensity, FFA utilization increases, perhaps due to the dominant lipolytic effects of GH. Pritzlaff et al. (39) reported that the positive relationship between exercise intensity and energy expenditure from fat during the recovery period is due to GH release. Evidence from the study by Gibney et al. (40) also demonstrates the reliance of fat oxidation on adequate GH secretion; fat metabolism at rest, during, and after exhaustive exercise was examined in GH-deficient subjects receiving GH replacement or a placebo. After 3 months of GH withdrawal, markers of fat oxidation (plasma FFA and glycerol) were significantly depressed in response to exercise compared with those in the GH-treated group. Interestingly, the withdrawal of GH also led to increases in total body fat accompanied by a loss of lean body mass.

During the postexercise period, we also documented sig-

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**Fig. 5.** EPOC and recovery time points for HR, lactate, cortisol, GH, and RER in lean (n = 6; A) and obese (n = 7; B) groups. The arrow indicates the time at which differences between baseline and exercise were no longer significant. Baseline VO2 is represented by the dashed line.

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ificantly lower RER values in our lean men accompanied by a higher EPOC, illustrating the shift toward fat oxidation and higher energy expenditure (28%), respectively. The significantly lower GH response in the obese men may have contributed to the RER and EPOC results.

Compared with the lean subjects, our obese men exhibited an increased cortisol response to exercise, which continued into the recovery period. These results agree with the report by Jungmann et al. (18), but two other studies found comparable or no change in cortisol response to exercise (15, 16). Comparing data from these studies becomes difficult, because the subjects varied in age, gender, and metabolic condition (i.e. some were diabetic), which could contribute to the differences in cortisol results. As well, the exercise protocol employed in the latter two studies were of short duration (10 min) and probably inadequate to elicit a cortisol response (23, 29, 41).

Although changes in glucose production and clearance occur during low intensity or short duration exercise, cortisol is unlikely to contribute to these changes. For example, Febbraio et al. (42) found no change in circulating insulin, glucagon, epinephrine, norepinephrine, cortisol, and GH at 40% VO2max. They attributed increased glucose metabolism to IL-6, an acute mediator of glucose homeostasis that is stimulated by skeletal muscle contraction and possibly by low muscle glycogen stores. To date, comparisons of the IL-6 response to exercise in lean and obese men have not been completed, but genetic variants in the IL-6 receptor gene are associated with susceptibility to obesity and its metabolic consequences (43).

In summary, exercise of sufficient intensity and duration leads to a marked shift toward fat oxidation, yet the postexercise GH, cortisol, and RER responses to a standardized bout of exercise in obese men reveal a reduced potential for fat oxidation. In contrast, lean men demonstrated a clear shift toward fat oxidation during recovery from exercise accompanied by a 28% higher EPOC. In combination, these findings suggest that excess body fatlessness facilitates the metabolic response to exercise.

Acknowledgments

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