Effects of 10 Days of Endurance Exercise Training on the Suppression of Whole Body and Regional Lipolysis by Insulin*

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ABSTRACT
The aim of this study was to evaluate in premenopausal women (10 sedentary obese women) the effects of 10 days of exercise on the suppression of whole body and regional lipolysis by insulin. Lipolysis was determined using $^{2}$H$_{5}$-glycerol infusion and microdialysis of sc adipose tissue during a two-stage hyperinsulinemic-euglycemic clamp [10 (LO) and 20 (MO) mU/min]. Microdialysis probes were positioned in abdominal and femoral sc adipose tissue to monitor interstitial glycerol and blood flow. Basal plasma glycerol was 86.7 ± 17.0 and 100.3 ± 19.8 μmol/L before and after training, respectively ($P < 0.05$). Plasma glycerol was suppressed to a greater extent after [to 47 ± 5% (LO) and 42 ± 5% (MO) of basal] than before [to 62 ± 8% (LO) and 55 ± 8% (MO) of basal] training. The rate of appearance of glycerol was suppressed to 49 ± 7% and 40 ± 5% of basal during LO and to 38 ± 5% and 30 ± 4% of basal during MO ($P < 0.05$) before and after training in each of these tissues. The results indicate that the antilipolytic response to insulin can be improved through endurance exercise training. Intraabdominal adipose tissue or skeletal muscle may be the site of improved antilipolytic response to insulin after training, as improvement was not evident in abdominal or femoral sc adipose tissue. (J Clin Endocrinol Metab 85: 1498–1504, 2000)

INSULIN-STIMULATED glucose uptake into both skeletal muscle and adipose tissue is enhanced in the trained, compared to the sedentary, state (1). There are also many studies of improved glucoregulatory control after endurance exercise training (2–4). However, there are no in vivo studies in humans designed for the specific purpose of determining the effect of endurance training on the antilipolytic action of insulin. Release of fatty acids from intraabdominal fat stores has been implicated in causing hepatic and peripheral insulin resistance (5). We have previously reported that obese women have an impaired response to the antilipolytic action of insulin compared to nonobese premenopausal women (6). Jensen (7) demonstrated that premenopausal women have impaired suppression of lipolysis in abdominal fat relative to men. Habitual endurance exercise training has been shown to prevent the accumulation of intraabdominal fat (8, 9) and to independently enhance the glucoregulatory action of insulin (2), but it is not known whether training also enhances the antilipolytic effect of insulin. An enhanced antilipolytic response in the intraabdominal fat depot would reduce free fatty acid release into the portal circulation, thereby reducing glucoregulatory insulin resistance.

Simultaneous assessment of regional lipolysis in sc adipose tissue by microdialysis and whole body lipolysis by stable labeled isotope kinetics allows for determination of the relative importance of sc vs. other depots of adipose tissue (presumably intraabdominal or skeletal muscle) in the antilipolytic response to insulin. In this context, the specific aim of this study was to evaluate the effects of 10 days of exercise on the suppression by insulin of whole body [glycerol rate of appearance (Ra)] and regional (microdialysis of sc adipose tissue) lipolysis during a hyperinsulinemic-euglycemic clamp in obese premenopausal women.

Subjects and Methods

Subjects
Ten obese premenopausal women [>28% body fat; maximal oxygen uptake (VO$_{2}$ max), <35 mL/kg/min] participated in the study after giving their informed consent according to the institutional review board at Washington University School of Medicine. The volunteers were not suffering from any chronic or acute illnesses, and none of the subjects had a prior history of cardiovascular disease. Three of the subjects were glucose intolerant as determined by a oral glucose tolerance test performed as a screening for participation in this study. Subjects reported that they were weight stable for the 6 months preceding the investigation. All subjects were eumenorrheic, in that all had monthly menses for the 12 months preceding the investigation.

Protocol
Subjects were studied before and after 10 days of endurance training. Whole body lipolysis was evaluated by $^{3}$H$_{5}$-glycerol kinetics in the basal state and during two low dose insulin infusions. Regional lipolysis was evaluated simultaneously by placing microdialysis probes in sc adipose tissue in the abdominal (two probes) and femoral (two probes) regions.
Endurance training

The endurance training program consisted of 1 h/day (10 days of 14 consecutive days) of exercise on a motorized treadmill, cycle ergometer, stair-climbing ergometer, and/or rowing ergometer. Twenty-five minutes of the exercise session were performed at a constant workload set to elicit approximately 70% VO\textsubscript{2}\text{max}. The final 30 min of the exercise session was designed to consist of five 5-min intervals at an exercise intensity set to elicit 100% VO\textsubscript{2}\text{max} (the actual intensity maintained by subjects was 90% VO\textsubscript{2}\text{max}), with 2.5 min of rest between intervals. This exercise training program has been previously demonstrated to result in significant improvements in VO\textsubscript{2}\text{max} in the absence of measurable changes in body composition. The effects of exercise training are therefore investigated without the confounding factor of changes in body composition.

Body composition and fat distribution

Fat and fat-free masses were measured by dual energy x-ray absorptiometry (QDR-1000/w, Hologic, Inc., Wallingford, MA) before and after endurance training, as described previously using version 5.64 of the enhanced whole body analysis program (10). Regional adiposity was also evaluated anthropometrically. Circumference measurements were made at the waist, hip, and midthigh. The anatomical landmarks used to locate the circumference sites in a reproducible manner were those described in the Anthropometric Standardization Reference Manual (11).

VO\textsubscript{2}\text{max}

VO\textsubscript{2}\text{max} was determined on a motorized treadmill. Subjects walked at 3 mph for 3 min, and speed was then increased by 1.0 mph every 3 min to 5 mph. The speed was then held constant, and grade was increased by 2% every minute until exhaustion. The fractional oxygen and carbon dioxide content of expired air were measured continuously, and oxygen uptake was calculated for each 30-s interval using an automated, open circuit system (8). At least two of the following criteria were met to establish that VO\textsubscript{2}\text{max} had been attained: a plateau in VO\textsubscript{2} despite an increase in treadmill speed or grade, attainment of age-predicted maximal heart rate, and a respiratory exchange ratio greater than 1.10.

Hyperinsulinemic-euglycemic clamp and glycerol kinetics

The hyperinsulinemic-euglycemic clamp studies were performed as described previously (12) at the Washington University General Clinical Research Center after an overnight fast. Although the time of the clamp procedure was randomized with respect to the menstrual cycle for each subject, subjects were studied at the same point in the menstrual cycle before and after 10 days of exercise. This was accomplished by delaying the beginning of exercise training for 16 days after the initial (pretraining) hyperinsulinemic-euglycemic clamp. In the trained state, subjects were studied 16–24 h after their last exercise bout to study individuals in the habitual state of exercise training. Subjects were instructed to consume at least 250 g carbohydrate/day for the 3 days before the clamp procedure. Three baseline venous blood samples were obtained at 5-min intervals to determine plasma substrate and hormone concentrations and background isotope enrichments. A primed (1.5 μmol/kg, constant (−0.1 μmol/kg/min) infusion of \textsuperscript{2}H\textsubscript{5}-glycerol (99%; Tracer Technologies, Newton, MA) was started and continued for 270 min (90 min basal period, 90 min at each insulin infusion rate). The actual isotope delivery rate was determined by measuring the enrichment of the isotope in the infused fat. The 90-min basal period was followed by two sequential 90-min primed, constant insulin infusions. The insulin was delivered at rates of 10 and 20 mU/m\textsuperscript{2}min. It was anticipated that these infusion rates would result in plasma insulin concentrations of approximately 15 and 35 μU/mL. These levels were chosen because the insulin concentration for a half-maximal suppression of lipolysis has been shown to be approximately 35 μU/mL in obese subjects (13).

The rate of glucose disposal was estimated from the steady state glucose infusion rate over the final 30 min of each insulin infusion. It is recognized that this may have resulted in an underestimate of the actual glucose disposal rate, as hepatic glucose production may not have been completely suppressed at the low insulin concentrations. Furthermore, the insulin infusion rates were expected to result in plasma insulin concentrations in the low, flat portion of the sigmoidal dose-response curve for the effect of insulin on glucose disposal (i.e., very small changes in glucose disposal could be expected). The glucoregulatory data were therefore not the focus of these investigations.

Blood samples were obtained at 10-min intervals during the final 30 min of each of the 90-min stages for the determination of plasma substrate and hormone concentrations as well as isotope enrichments. Plasma samples were stored at −80 C and subsequently analyzed for free fatty acids (NEFA kit, Wako Chemicals, Dallas, TX), glycerol (14), insulin (15), and epinephrine and norepinephrine (16). Interassay coefficients of variation are 8% for glycerol, 14% for insulin, 4% for free fatty acids, and 11% for catecholamines in the concentration range of samples in this study.

\textsuperscript{2}H\textsubscript{5}-glycerol analysis

The analysis of \textsuperscript{2}H\textsubscript{5}-glycerol was performed, as we have previously described (6), using a modification of the negative ion chemical ionization gas chromatography-mass spectrometry method described by Gilker et al. (17). The Ra of glycerol over the last 30 min of each stage was calculated using the nonsteady state equations of Steele (18), assuming a volume of distribution for glycerol of 270 mL/kg (19). The coefficient of variation for Ra was 7.1 ± 2.2% as determined from day to day reproducibility of enrichment determinations (n = 6).

Assessment of regional lipolysis by microdialysis

The microdialysis probes (DL-3, Biosanautical Systems, Inc., West Lafayette, IN) consisted of inlet tubing (30 cm) and outlet tubing (15 cm) separated by a polyacrylonitrile dialysis membrane (3 cm in length; id, 0.25 mm; od, 0.35 mm). Probes were sterilized using ethylene oxide gas and were soaked for a minimum of 16 h overnight in 5% ethanol while perfused with Ringer’s solution to remove the glycerol coating that was placed on the dialysis membrane during the manufacturing process. The absence of measurable glycerol on the microdialysis membrane was verified by the absence of glycerol in the dialysate during perfusion of the microdialysis probes with Ringer’s solution in vitro before insertion into the research subjects.

Microdialysis probes were inserted under sterile technique and local anesthesia (0.1 mL 1% lidocaine without epinephrine) into the abdominal (two probes, bilaterally – 3 cm lateral to the umbilicus) and femoral (two probes, – 3 cm apart, midthigh) sc adipose tissue in each subject. This was accomplished by inserting a 14-gauge catheter through the skin, advancing it through the sc adipose tissue parallel to the skin, and then exiting through the skin. The needle was withdrawn, the microdialysis probe was threaded through the catheter, and the catheter was removed, leaving the membrane portion of the probe embedded in the adipose tissue.

Microdialysis probes were perfused (Harvard infusion pump, model 22, Harvard Apparatus, South Natick, MA) at 2.0 μL/min with Ringer’s solution containing 2.5 mmol/L glucose and 5 mmol/L ethanol for 60 min. No samples were collected during this time to allow for equilibration of the microdialysis system and to allow the initial trauma of probe insertion to subside (20, 21). Glucose (2.5 mmol/L) was added to the perfusate to minimize the drainage of glucose from the interstitial fluid. No significant net loss or gain of perfusate fluid occurred over the dialysis membrane during the microdialysis process under the conditions of this experiment, as verified by weighing of dialysate samples. The inclusion of ethanol in the perfusate permitted the detection of changes in blood flow in the region surrounding the probe (22, 23). Ethanol at the concentration used (5 mmol/L) has been shown to have no effect on lipolysis in sc adipose tissue (23). The ethanol data are presented as the outflow/inflow ratio [ethanol\textsubscript{dialysate}/ethanol\textsubscript{perfusate}], which is inversely related to blood flow in the region surrounding the microdialysis probe (21). The ongoing dialysate was collected in 15-min fractions (30 μL). The dialysate samples were stored at 4 C and analyzed within 48 h for glycerol (14) and ethanol (21). We have previously determined the relative recovery for glycerol over this type of dialysis membrane to be approximately 60% in group of premenopausal women (6). The range of recoveries, however, can be from 50–70% at the perfusion flow rate and membrane characteristics employed, and can vary with changes in adiposity (24, 25).
**Statistics**

Differences were analyzed by one-way repeated measures ANOVA. If significance was obtained, the Newman-Keuls post-hoc analysis was used to identify group differences. The effects of insulin infusion on blood or dialysate parameters as well as differences between pre- and posttraining VO₂max were evaluated using two-tailed paired Student’s t tests. All data are expressed as the mean ± se. The level of significance was set at P < 0.05.

**Results**

**Subject characteristics**

Subject characteristics with respect to age, VO₂max, body composition, and fat distribution are presented in Table 1. Body weight and indexes of fatness were unchanged after training, whereas VO₂max (milliliters per kg/min) was increased 6.4% (P < 0.05) after training.

**Hormone and free fatty acid concentrations**

There were no significant differences between pre- and posttraining in basal serum free fatty acid, epinephrine, norepinephrine, and insulin concentrations (Table 2). No changes from basal were measured in norepinephrine and epinephrine concentrations. Insulin concentrations increased to a similar extent upon insulin infusion before and after training. Free fatty acid concentrations were not suppressed to a greater extent than the low dose during the moderate dose insulin infusion after, compared to before, training.

**Glycerol concentrations and kinetics**

**Basal.** In the basal state, plasma glycerol concentrations were higher after training (100.3 ± 19.8 μmol/L) than before training (86.7 ± 17.0 μmol/L; P < 0.05). Basal abdominal and femoral dialysate glycerol concentrations were not different after, compared to before, training.

Glycerol Ra values were 2.70 ± 0.36 and 2.69 ± 0.30 μmol/kg BW·min under basal conditions before and after training, respectively (P = NS). Body weight and fat mass did not change with training; glycerol Ra values per unit fat mass were therefore also similar before and after training.

**Insulin infusion.** Plasma and dialysate glycerol concentrations were significantly reduced both before and after training in response to the first insulin infusion (Fig. 1). The only further significant reduction in glycerol concentration during the second insulin infusion before training occurred in the femoral dialysate. After training, glycerol Ra and glycerol concentrations in plasma and dialysates were reduced during the moderate dose compared to the low dose insulin infusion.

In relative terms, the reductions in plasma glycerol were

### Table 1. Subject characteristics

<table>
<thead>
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<th>Posttraining</th>
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<tr>
<td>n</td>
<td>10</td>
<td>10</td>
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<tr>
<td>Wt (kg)</td>
<td>69.4 ± 4.6</td>
<td>69.7 ± 4.7</td>
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<td>Body fat (%)</td>
<td>32.6 ± 3.2</td>
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<td>Fat mass (kg)</td>
<td>23.7 ± 4.0</td>
<td>23.6 ± 4.0</td>
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<tr>
<td>FFM (kg)</td>
<td>45.7 ± 1.9</td>
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<tr>
<td>VO₂max (L/min)</td>
<td>2.06 ± 0.12</td>
<td>2.19 ± 0.14 *</td>
</tr>
<tr>
<td>VO₂max (mL/kg·min)</td>
<td>31.2 ± 2.4</td>
<td>32.4 ± 2.6 *</td>
</tr>
</tbody>
</table>

FFM, Fat-free mass.

* Significantly different from pretraining (P < 0.05).

### Table 2. Plasma insulin, catecholamine, and serum free fatty acid concentrations

<table>
<thead>
<tr>
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<th>Pretraining</th>
<th>Posttraining</th>
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</thead>
<tbody>
<tr>
<td>Insulin (μU/mL)</td>
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<td></td>
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<tr>
<td>Baseline</td>
<td>7.2 ± 2.1</td>
<td>7.4 ± 2.2</td>
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<tr>
<td>Low dose</td>
<td>19.7 ± 1.5</td>
<td>18.9 ± 1.4</td>
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<tr>
<td>Moderate dose</td>
<td>31.1 ± 1.9</td>
<td>34.2 ± 3.3</td>
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<tr>
<td>Free fatty acid (μmol/L)</td>
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<tr>
<td>Baseline</td>
<td>645.2 ± 50.1</td>
<td>651.0 ± 56.4</td>
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<tr>
<td>Low dose</td>
<td>204.5 ± 61.7</td>
<td>191.4 ± 53.5</td>
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<tr>
<td>Moderate dose</td>
<td>146.4 ± 62.5</td>
<td>100.3 ± 30.9</td>
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<tr>
<td>Epinephrine (pg/mL)</td>
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<tr>
<td>Baseline</td>
<td>28.9 ± 4.6</td>
<td>27.6 ± 3.7</td>
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<tr>
<td>Low dose</td>
<td>28.6 ± 3.9</td>
<td>28.9 ± 5.5</td>
</tr>
<tr>
<td>Moderate dose</td>
<td>29.9 ± 5.1</td>
<td>31.0 ± 4.7</td>
</tr>
<tr>
<td>Norepinephrine (pg/mL)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>209.8 ± 18.2</td>
<td>205.1 ± 18.9</td>
</tr>
<tr>
<td>Low dose</td>
<td>199.4 ± 13.7</td>
<td>207.8 ± 19.7</td>
</tr>
<tr>
<td>Moderate dose</td>
<td>206.2 ± 17.8</td>
<td>204.5 ± 16.0</td>
</tr>
</tbody>
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* Different from basal (P < 0.05).
** Different from pretraining (P < 0.05).
greater after training compared to before training during both stages of insulin infusion (Fig. 2). Glycerol Ra was reduced to a greater extent after training compared to before training only during the moderate dose insulin infusion. There was no greater reduction in dialysate glycerol concentration after training compared to before training in either abdominal or femoral sites.

**Nutritive blood flow**

The basal ethanol outflow/inflow ratios from probes in abdominal sc adipose tissue were 0.537 ± 0.028 and decreased to 0.507 ± 0.033 (P < 0.01) during the low dose insulin infusion. The outflow/inflow ratios were further reduced to 0.475 ± 0.032 (P < 0.01) during the second insulin infusion. Basal ethanol outflow/inflow ratios from probes in femoral sc adipose tissue were 0.561 ± 0.028, were reduced to 0.490 ± 0.028 (P < 0.01) during the low dose insulin infusion, and were further reduced to 0.440 ± 0.031 during the moderate dose insulin infusion (P < 0.01). These data indicate that nutritive blood flow per tissue volume was increased upon insulin infusion.

Although, as noted above, ethanol outflow/inflow ratio decreased (increase in adipose tissue nutritive blood flow) in response to insulin infusion before endurance training, similar decreases in ethanol outflow/inflow ratio were recorded after training. Ethanol outflow/inflow ratios from probes placed in abdominal adipose tissue decreased from 0.564 ± 0.074 before insulin infusion (P = NS compared to pretraining) to 0.485 ± 0.044 (P < 0.01) and 0.459 ± 0.041 (P < 0.01) during the low and moderate dose insulin infusions, respectively. Ethanol outflow/inflow ratios from probes placed in femoral adipose tissue decreased from 0.585 ± 0.041 before insulin infusion (P = NS compared to pretraining) to 0.499 ± 0.036 (P < 0.01) and 0.447 ± 0.028 (P < 0.01) during the low and moderate dose insulin infusions, respectively.

**Glucose disposal**

During the first insulin infusion, glucose disposal rates were lower after training (1.69 ± 0.28 mg/kg/min) than before training (1.98 ± 0.32 mg/kg/min; P < 0.05). During the second insulin infusion, glucose disposal rates tended to be higher after training (4.05 ± 0.72 mg/kg/min) than before training (3.76 ± 0.69 mg/kg/min; P = 0.1).

**Discussion**

We have in this study demonstrated that endurance training significantly enhances the suppression of whole body lipolysis by insulin. This effect was not seen in abdominal or femoral sc adipose tissue. These findings demonstrate that adipose tissue other than sc adipose tissue, i.e. intraabdominal or IM (between or within muscle fibers) triglyceride stores, are the major sites of adipose tissue responsible for the improvement in antilipolytic response to insulin. We have previously reported no difference in the antilipolytic re-

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**Fig. 2.** Plasma glycerol, glycerol Ra, and dialysate glycerol from microdialysis probes placed in abdominal and femoral sc adipose tissue before (■) and after (□) 2 weeks of endurance training during a low dose (10 mU/m^2·min) and moderate dose (20 mU/m^2·min) insulin infusion. Data are the mean ± se (n = 10) and are expressed as a percentage of basal. ***, Different from pretraining (P < 0.05). †, Different from low dose (P < 0.05).
response to insulin infusion in trained compared to sedentary individuals (6); however, the current study is the first to our knowledge to report the effects of endurance training on the whole body (glycerol kinetics) and regional (microdialysis) antilipolytic responses to insulin infusion in a prospective manner.

Regional differences in the antilipolytic effect of insulin before training

With respect to regional differences in response to the antilipolytic action of insulin, the data from the present study support our previous findings (26, 27) that femoral sc adipose tissue is more resistant to the effects of insulin than is abdominal sc adipose tissue. Femoral adipose tissue was more resistant to the antilipolytic action of insulin than was abdominal adipose tissue, which was more resistant than the whole body antilipolytic response to insulin, both before and after training. These findings coupled with previous observations that femoral adipocytes of women have a metabolic profile that favors storage to a greater degree than abdominal adipocytes (28) indicate that triglyceride turnover is higher in femoral than abdominal sc adipocytes. Our reports of a decreased antilipolytic action of insulin in femoral compared to abdominal sc fat depots are not consistent with in vitro findings of Bolinder et al. (29) and Arner et al. (30) or in vivo findings of Jensen et al. (7). These researchers report equal or greater antilipolytic response in femoral than abdominal adipocytes.

Effect of training on basal glycerol Ra and dialysate glycerol concentrations

Basal glycerol Ra was unchanged after training, although fasting glycerol concentrations were elevated. Basal glycerol Ra and fasting plasma glycerol concentrations were more than 50% higher in the trained group than in the sedentary groups in our previous cross-sectional study of trained and sedentary women (6). Although the elevated fasting plasma glycerol concentrations may (31) or may not (32) be due to an acute effect of the recent exercise bout (16–24 h before testing), the elevated glycerol Ra appears to be due to a cumulative training effect, which cannot be duplicated with only 2 weeks of endurance training. In our previous study of premenopausal women, differences were also found between trained and sedentary individuals in dialysate glycerol concentrations from probes placed in abdominal sc adipose tissue under basal conditions. These differences were proportional to differences in Ra per kg fat mass. It was therefore suggested that the slightly higher lipolytic rate in sc adipose tissue of trained compared to sedentary nonobese individuals may have accounted for the increased basal whole body lipolysis in the trained group (glycerol Ra). This effect of training on basal glycerol release from sc adipose tissue also appears to be an effect of an extended period of endurance training and was not seen in the present study after 2 weeks of endurance training.

Effect of training on the antilipolytic response to insulin: site of action

Although there is limited information regarding the effects of exercise training on the antilipolytic response to insulin, Suda et al. (33) reported an increased suppression of lipolysis by insulin in the epidydimal fat pad of exercise-trained rats. We have now demonstrated an increased suppression of lipolysis by insulin at the whole body level, but not in sc adipose tissue, in exercise-trained humans. The negative results of our previous study (6) may have been due to the small sample size (n = 6) and the cross-sectional study design. However, trained and sedentary premenopausal women in the previous study were nonobese and were therefore relatively insulin sensitive compared to the current group of premenopausal women, which was comprised of mildly to morbidly obese women. A training effect with regard to the antilipolytic action of insulin may be more predominant, and therefore easier to detect, in insulin-resistant individuals.

Although intraabdominal fat mass was not determined before and after training in the present study, it is unlikely that intraabdominal fat mass changed significantly after only 2 weeks of training in the absence of changes in body composition. Furthermore, the hormonal milieu (epinephrine, norepinephrine, insulin) was not different after, compared to before, training. If intraabdominal adipose tissue were the site of increased suppression of lipolysis after training, increased insulin action must have been the mechanism. It has been shown that resistance to the antilipolytic action of insulin in vitro is more pronounced in adipocytes from the omental adipose depot than in those from sc adipose depots (34). The antilipolytic response to insulin in intraabdominal adipose depots may therefore have been enhanced to a greater extent than in sc adipose depots after successive days of exercise.

Another candidate site for training effects on the antilipolytic response to insulin would be triglyceride stores within or between muscle fibers. Studies of glycerol release in men using arteriovenous differences over the limb (leg) have been inconclusive (3, 4, 35). Dela et al. showed both no change in (4, 35) as well as an increased response to (3) the antilipolytic action of a maximal dose of insulin after 10 weeks of a one-legged exercise training program. It is possible that there is a response to training in skeletal muscle that does not occur in adipose tissue. There is, in fact, evidence that different phosphodiesterase subtypes regulate antilipolysis in skeletal muscle and adipose tissue (23).

Microdialysis probes were not placed in skeletal muscle or intraabdominal fat of the subjects in the present study. Whether im or intraabdominal triglyceride stores were the site of increased antilipolytic insulin action after training must therefore be addressed in future studies; investigation of the latter is clearly limited due to the inaccessibility of intraabdominal fat.

Potential limitations

In the present investigations, the dialysate glycerol concentration was not a direct measure of the interstitial glycerol concentration due to incomplete equilibration (recovery) of
glycerol over the dialysis membrane. Changes in nutritive blood flow could potentially result in changes in dialysate glycerol concentration independent of changes in the lipolytic state of the fat cells (23, 36, 37). Microdialysis was used in the present study to monitor both lipolysis and nutritive blood flow in sc adipose tissue. Ethanol outflow/inflow ratio was reduced (increased nutritive blood flow) in both adipose tissue depots in response to insulin infusion. Although there were no significant differences in ethanol outflow/inflow ratios under basal and insulin-stimulated conditions, the mean outflow/inflow ratio in the abdominal site appeared to be higher at baseline and was reduced in response to insulin infusion to a greater extent after, compared to before, training. This would indicate that the nutritive blood flow response to insulin in the abdominal adipose tissue depot might have been greater after, compared to before, training, thus resulting in greater removal of glycerol from the interstitium. The percent decline in the abdominal dialysate glycerol concentration after training may therefore have been exaggerated. Consequently, the data for the percent decline in dialysate glycerol concentration before and after training are probably more similar than they appear. Alterations in sc adipose tissue nutritive blood flow, therefore, did not change the main conclusion regarding dialysate glycerol results, e.g. there were no differences in the antilipolytic response to insulin in sc adipose tissue after, compared to before, endurance training.

Summary

Before training, the greatest antilipolytic effect of insulin was seen in the plasma glycerol concentrations, whereas the poorest antilipolytic response to insulin was seen in the femoral sc adipose tissue glycerol levels. Thus, the order of the antilipolytic response (determined by dialysate glycerol concentrations) to insulin was plasma > abdominal sc adipose tissue > femoral sc adipose tissue. There was no effect of training on this order of sensitivity, although there were increases in the magnitude of the whole body antilipolytic response. During insulin infusion, there was a greater relative suppression of whole body lipolysis after, compared to before, endurance training. However, there was no effect of training on the relative degree of suppression of regional sc adipose tissue lipolysis. Basal lipolysis per kg fat mass was not affected by 2 weeks of endurance training. These data indicate that improvements in the antilipolytic action of insulin can be made in the absence of significant improvements in the sc adipose tissue response to insulin. Fat depots responding to training may be intraabdominal fat, although an improvement in the antilipolytic action of insulin on fat in muscle (i.e. between or within muscle fibers) could not be ruled out. These findings demonstrate that obese women can improve the antilipolytic response to insulin through endurance training.

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