Aerobic training improves exercise-induced lipolysis in SCAT and lipid utilization in overweight men

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De Glisezinski, L., C. Moro, F. Pillard, F. Marion-Latard, I. Harant, M. Meste, M. Berlan, F. Crampes, and D. Rivière. Aerobic training improves exercise-induced lipolysis in SCAT and lipid utilization in overweight men. Am J Physiol Endocrinol Metab 285: E984–E990, 2003; 10.1152/ajpendo.00152.2003.—The aim of this study was to investigate whether endurance training improves lipid mobilization and oxidation in overweight subjects. Eleven young men (25.6 ± 1.4 yr and body mass index 27.7 ± 0.2) performed a 4-mo training program consisting of practicing aerobic exercise 5 days/wk. Before and after the training period, lipid oxidation was explored during a 60-min exercise at 50% of peak O2 consumption by use of indirect calorimetry. Lipid mobilization and antilipolytic α2-adrenoceptor effect were also studied using the microdialysis method in abdominal subcutaneous adipose tissue (SCAT). After training, plasma nonesterified fatty acid (NEFA) levels, at rest and during exercise, were significantly lower than before (P < 0.001). Lipolysis in SCAT was significantly higher after than before training. An antilipolytic α2-adrenoceptor effect in SCAT was underlined during exercise before training and disappeared after. The respiratory exchange ratio was lower after training, i.e., the percentage of lipid oxidation was higher only at rest. The amount of lipid oxidized was higher after training, at rest, and during exercise. Although exercise power was higher after training, the relative intensity was equivalent, as suggested by a similar increase in plasma catecholamine concentrations before and after training. In conclusion, 4-mo training in overweight men improved lipid mobilization through a decrease of antilipolytic α2-adrenoceptor effect in SCAT and lipid oxidation during moderate exercise. Training induced a decrease of blood NEFA, predicting better prevention of obesity.

subcutaneous adipose tissue; microdialysis; catecholamines; α2-adrenoceptor; exercise; lipid oxidation

REGULAR EXERCISE ASSOCIATED WITH DIET has been recommended as an important strategy in the prevention and management of obesity. Endurance training appears to be one of the major factors determining the long-term success of weight loss programs (26, 37). One explanation is that exercise partly counteracts the decline in fat oxidation occurring with weight loss during a hypocaloric diet by maintaining adipose lipolytic responsiveness (25, 36).

In normal-weight subjects exercise training has been found to result in increased fat oxidation (3), but whole body lipolysis during exercise was not significantly enhanced by training (20).

Few studies bear on the effect of exercise on adipose tissue lipolysis in obese subjects, whereas in this population an alteration of catecholamine-stimulated lipolysis has been reported (28, 29). In a previous study, we have shown that 3-mo aerobic training in obese men enhanced the lipolytic β-adrenergic response of adipose tissue and decreased adipocyte antilipolytic activity of α2-adrenoceptors (AR) in vitro (6); moreover, we have shown in vivo that lipolysis at rest was increased during β-adrenergic stimulation after physical training (31). However, we do not know whether, despite better β-adrenergic response at rest, adipose tissue lipolysis was improved during exercise, inducing release of more nonesterified fatty acids (NEFA) after endurance training than before. If NEFA released by adipose tissue are the main factor of success in weight loss, their oxidation particularly by muscle is also important, decreasing their concentration in the blood, and so enhancing lipid oxidation may prevent cardiovascular disease. Energy restriction is known to induce a decline in fat oxidation. On the other hand, low-intensity exercise training in obese women counteracts, at rest, the decline in fat oxidation occurring after hypocaloric diet (25, 36) and increases the contribution of fat oxidation to total energy expenditure during exercise (34). Most studies concerning training and obesity in females concerned postmenopausal women (25, 33); however, with regard to prevention, it seemed more interesting to explore the young population and overweight subjects who are likely to develop obesity.

The purpose of the present investigation was to explore the effect of endurance training, without modification of diet, on mobilization and oxidation of lipids in overweight men during physiological stimulation of the sympathoadrenal system as induced by physical exerci-
Exercise. The lipid mobilization from abdominal subcutaneous adipose tissue (SCAT) was studied using the microdialysis technique (1, 7); moreover, to delineate the importance of the local \( \alpha_2 \)-AR-mediated pathway in SCAT lipolysis, the effect of \( \alpha_2 \)-AR blockade was explored. Lipid oxidation was evaluated by measuring pulmonary gas exchange ratios (respiratory exchange ratios, RER) by indirect calorimetry.

**SUBJECTS AND METHODS**

**Subjects.** Eleven overweight, untrained men (25.6 ± 1.4 yr) participated in the study. The mean body weight was 89.5 ± 1.6 kg (range 78–95 kg) and body mass index (BMI) 27.7 ± 0.2 kg/m² (range 26.7–29.1 kg/m²). The percentage of fat mass was 22.8 ± 0.9%, and the maximal oxygen uptake (VO\(_2\)max), determined during a leg cycling test, was 34.3 ± 1.3 ml·min\(^{-1}\)·kg\(^{-1}\). After the VO\(_2\)max test and during the whole experimental period, the subjects were asked to maintain their usual diet. All were drug free, and their weight had remained stable for ≥3 mo before the beginning of the study.

All subjects had given their written informed consent before the experiments began. The studies were performed according to the Declaration of Helsinki and approved by the Ethics Committee of Toulouse I University Hospital (France).

**Exercise training protocol.** The training program consisted of practicing aerobic exercise (1 h/day), mainly running and cycling, 5 days/wk, for 4 mo under the control of a physical exercise coach. The exercise intensity and duration were increased progressively. The subjects exercised at a target heart rate corresponding to 50–85% of their VO\(_2\)max, the heart rate being monitored with a Polar Accurex Plus Cardiometer (Monitor; La Varenne, St Hilaire, France). Compliance with training sessions was good, as checked by a trainometer (Monitor; La Varenne, St Hilaire, France). Anthropometric assessment: fat mass and fat-free mass remained stable for the whole experimental period, the subjects were asked to maintain their usual diet. All were drug free, and their weight had remained stable for ≥3 mo before the beginning of the study. The evening preceding every trial, the subjects were fasted. The evening preceding every trial, the subjects were asked to eat a standard meal consisting of 3,350 kJ (55% carbohydrate, 30% fat, 15% protein). Experiments were conducted in a ventilated room at a temperature of 19–20°C. At 8:00 AM, two microdialysis probes (Carnegie Medecin, Stockholm, Sweden) of 20 × 0.5 mm and 20,000-molecular-weight cutoff were inserted percutaneously after epidermal anesthesia (200 μl of 1% lidocaine; Astrazeneca, Rueil-Malmaison, France) into the abdominal SCAT at a distance of 10 cm on both sides of the umbilicus. The probes were connected to a microinjection pump (Harvard apparatus, SARL, Les Ulis, France). One probe was perfused with Ringer solution (in mM: 139 sodium, 2.7 potassium, 0.9 calcium, 140.5 chloride) and the second with Ringer plus 0.1 mM phentolamine (\( \alpha_2 \)-AR antagonist). This nonselective \( \alpha_1 \)-/\( \alpha_2 \)-antagonist, having an efficient \( \alpha_2 \)-AR antagonist action in human fat cells in vitro, was the only agent allowed by the Ethics Committees for use in microdialysis assays in humans. The two perfusate solutions were supplemented with ethanol (2 g/l). Ethanol was added to the perfusate to estimate changes occurring in the local SCAT blood flow, as previously described (2, 7). The perfusion was set at a flow rate of 2.5 μl/min. Two 15-min fractions at rest were kept for basal concentration values.

After the rest period the subjects started a bout of exercise on an electromagnetically braked bicycle ergometer (Ergoline, 800s; Ergoline, Hamburg, Germany). VO\(_2\) max was assessed using a graded test conducted on an electromagnetically braked bicycle ergometer (Ergoline, 800s; Ergoline, Germany). VO\(_2\)max was measured using an Oxycon Pro apparatus (Jaeger, Germany). The initial workload was 60 W, and it was increased by 30 W every 3 min until exhaustion. Heart rate was continuously monitored. The highest VO\(_2\) value was considered as VO\(_2\)max, and the workload corresponding to 50% of each subject was calculated. Two criteria assessed that the subjects achieved their true VO\(_2\)max: maximal heart rate measured at exhaustion (188.6 ± 2.6 beats/min) was different from age-predicted maximal heart rate (189 ± 2.8 beats/min), and the maximal RER measured at exhaustion was near 1.1. After the VO\(_2\)max determination, the subjects were randomly assigned to start the experimental protocol during the following week.

**Experimental protocol.** Before and after the training, the subjects were investigated at 8:00 AM. Before the training, the subjects were asked to eat a standard meal consisting of 3,350 kJ (55% carbohydrate, 30% fat, 15% protein). Experiments were conducted in a ventilated room at a temperature of 19–20°C. At 8:00 AM, two microdialysis probes (Carnegie Medecin, Stockholm, Sweden) of 20 × 0.5 mm and 20,000-molecular-weight cutoff were inserted percutaneously after epidermal anesthesia (200 μl of 1% lidocaine; Astrazeneca, Rueil-Malmaison, France) into the abdominal SCAT at a distance of 10 cm on both sides of the umbilicus. The probes were connected to a microinjection pump (Harvard apparatus, SARL, Les Ulis, France). One probe was perfused with Ringer solution (in mM: 139 sodium, 2.7 potassium, 0.9 calcium, 140.5 chloride) and the second with Ringer plus 0.1 mM phentolamine (\( \alpha_2 \)-AR antagonist). This nonselective \( \alpha_1 \)/\( \alpha_2 \)-antagonist, having an efficient \( \alpha_2 \)-AR antagonist action in human fat cells in vitro, was the only agent allowed by the Ethics Committees for use in microdialysis assays in humans. The two perfusate solutions were supplemented with ethanol (2 g/l). Ethanol was added to the perfusate to estimate changes occurring in the local SCAT blood flow, as previously described (2, 7). The perfusion was set at a flow rate of 2.5 μl/min. Two 15-min fractions at rest were kept for basal concentration values.

After the rest period the subjects started a bout of exercise on a leg cycle ergometer (Ergoline, 800s; Ergoline, Germany). VO\(_2\) max was assessed using a graded test conducted on an electromagnetically braked bicycle ergometer (Ergoline, 800s; Ergoline, Germany). VO\(_2\)max was measured using an Oxycon Pro apparatus (Jaeger, Germany). The initial workload was 60 W, and it was increased by 30 W every 3 min until exhaustion. Heart rate was continuously monitored. The highest VO\(_2\) value was considered as VO\(_2\)max, and the workload corresponding to 50% of each subject was calculated. Two criteria assessed that the subjects achieved their true VO\(_2\)max: maximal heart rate measured at exhaustion (188.6 ± 2.6 beats/min) was different from age-predicted maximal heart rate (189 ± 2.8 beats/min), and the maximal RER measured at exhaustion was near 1.1. After the VO\(_2\)max determination, the subjects were randomly assigned to start the experimental protocol during the following week.

**Experimental protocol.** Before and after the training, the subjects were investigated at 8:00 AM after an overnight fast. The evening preceding every trial, the subjects were asked to eat a standard meal consisting of 3,350 kJ (55% carbohydrate, 30% fat, 15% protein). Experiments were conducted in a ventilated room at a temperature of 19–20°C. At
ties were 5.0 and 9.2%, respectively. Ethanol in dialysate and perfusate (5 μl) was determined with an enzymatic method; the intra-assay and interassay variabilities were 3.0 and 4.5%, respectively. Plasma glucose and NEFA were determined with a glucose-oxidase technique (Biotrol kit; Merck-Clevenot, Nogent-s-Marne, France) and an enzymatic procedure (WAKO kit, Unipath, Dardilly, France), respectively. Plasma epinephrine concentrations were measured using RIA kits from Sanofi Diagnostics Pasteur (Marnes la Coquette, France). Plasma epinephrine and norepinephrine were assayed in 1-ml aliquots of plasma by high-performance liquid chromatography using electrochemical (amperometric) detection. The detection limit was 20 pg/sample. Day-to-day variability was 4% and within-run variability 3%.

Statistical analysis. All the values are means ± SE. The responses to exercise were analyzed using analysis of variance (doubly multivariate repeated-measures design), with statistical software: SAS and proc GLM. During exercise, extracellular glyceral concentration response curves, in μmol·l⁻¹·60 min, were calculated as the total integrated changes over baseline values [areas under curves (AUC)] by use of a trapezoidal method. \( P < 0.05 \) was considered statistically significant.

RESULTS

General observations. After 4 mo of training, the weight and percentage of fat mass decreased significantly (87.6 ± 1.6 vs. 89.5 ± 1.6 kg, \( P < 0.05 \), and 21.7 ± 1.1 vs. 22.8 ± 0.9%, \( P < 0.01 \), respectively). \( \dot{V}O_2 \) max values were significantly higher after training (3.53 ± 0.14 vs. 3.11 ± 0.13 l/min, \( P < 0.001 \)). During experiments, exercise power was 88.2 ± 5.8 W before training and 110.0 ± 5.6 W after training (\( P < 0.01 \)). The resting heart rate was 65.8 ± 0.3 before and 58.7 ± 0.3 beats/min after training (\( P < 0.001 \); on the other hand, systolic and diastolic blood pressures stayed unchanged.

Plasma catecholamine concentrations. At rest, catecholamine levels were similar before and after training. During exercise, the plasma norepinephrine concentrations were significantly higher than baseline at the 30th minute (\( P < 0.001 \)) and did not change until the end of exercise either before or after training. After the 60-min recovery period, norepinephrine concentrations decreased but did not reach basal values (Table 1). The plasma epinephrine concentrations increased until the end of exercise (\( P < 0.001 \)) both before and after training. After the 60-min recovery period, the epinephrine concentrations decreased to a value that did not differ from that measured at rest (Table 1). During exercise and during recovery, plasma norepinephrine and epinephrine concentrations were not different before and after training.

Plasma glucose and insulin concentrations. No significant variation of plasma glucose level was observed during the exercise bout or the recovery (Table 1). A significant decrease in plasma insulin concentrations was observed during the exercise (\( P < 0.001 \) before training, \( P < 0.01 \) after training; Table 1). No significant change was noted in plasma glucose concentrations after endurance training, at rest or during exercise. On the other hand, the plasma concentrations of insulin were significantly lower after training than before, at rest (\( P < 0.001 \)), during exercise (\( P < 0.01 \), and after 1-h recovery (\( P < 0.05 \); Table 1).

Plasma NEFA and glycerol levels and dialysate glycerol concentrations. At rest, plasma NEFA concentrations were significantly lower (\( P < 0.001 \)) after training than before (250 ± 25 vs. 494 ± 76 μmol/l). Plasma glycerol concentrations did not differ after endurance training (76 ± 6 vs. 75 ± 6 μmol/l), and the same was also true for the corresponding baseline glycerol concentrations in dialysate in the probe perfused with Ringer solution, i.e., the control probe (54 ± 6 μmol/l and 42 ± 3 μmol/l before and after training, respectively) and in the probe with phentolamine (56 ± 6 μmol/l and 59 ± 10 μmol/l before and after training, respectively). Plasma NEFA concentrations changed weakly along the exercise period, and at the end of exercise NEFA levels were slightly increased compared with baseline values (\( P < 0.05 \), before training). After 15 min of recovery, NEFA concentrations greatly increased (962 ± 123 and 538 ± 59 μmol/l before and after training, respectively) and then decreased but

Table 1. Effect of 60-min exercise and recovery on plasma catecholamines, glucose, and insulin concentrations before and after endurance training

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Exercise</th>
<th>Recovery</th>
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<tr>
<td></td>
<td>30 min</td>
<td>60 min</td>
<td>30 min</td>
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<tr>
<td>Norepinephrine, pg/ml</td>
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<tr>
<td>Before</td>
<td>288 ± 27</td>
<td>1047 ± 78***</td>
<td>1064 ± 96***</td>
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<tr>
<td>After</td>
<td>264 ± 29</td>
<td>919 ± 111***</td>
<td>1059 ± 93***</td>
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<tr>
<td>Epinephrine, pg/ml</td>
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<tr>
<td>Before</td>
<td>27 ± 4</td>
<td>84 ± 11***</td>
<td>112 ± 18***</td>
</tr>
<tr>
<td>After</td>
<td>34 ± 7</td>
<td>76 ± 7***</td>
<td>101 ± 15***</td>
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<tr>
<td>Glucose, mmol/l</td>
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<td></td>
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<tr>
<td>Before</td>
<td>5.2 ± 0.2</td>
<td>4.8 ± 0.2</td>
<td>4.9 ± 0.2</td>
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<tr>
<td>After</td>
<td>4.9 ± 0.1</td>
<td>5.0 ± 0.2</td>
<td>4.9 ± 0.2</td>
</tr>
<tr>
<td>Insulin, μU/ml</td>
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<tr>
<td>Before</td>
<td>5.2 ± 0.4</td>
<td>3.4 ± 0.4***</td>
<td>2.7 ± 0.4***</td>
</tr>
<tr>
<td>After</td>
<td>3.6 ± 0.3***</td>
<td>2.6 ± 0.3**</td>
<td>2.2 ± 0.3**</td>
</tr>
</tbody>
</table>

Values are means ± SE. *P < 0.05, **P < 0.01 and ***P < 0.001 exercise or recovery values vs. rest values; †P < 0.05, ††P < 0.01 and †††P < 0.001 before vs. previous training.

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remained higher than at rest ($P < 0.05$ before training). During all exercise and recovery, plasma NEFA concentrations were significantly lower after than before endurance training ($P < 0.001$; Fig. 1A). Before and after training, plasma glycerol levels increased similarly during the exercise ($P < 0.001$). Glycerol values showed no significant difference between before and after training (Fig. 1B).

During the exercise and before training, the glycerol concentrations in the dialysate increased during the first 15 min in the control probe and continued to increase until the end of exercise. In the probe with phentolamine, the glycerol concentrations in the dialysate were significantly higher than in the control probe throughout the exercise period and remained higher during the 45-min recovery period (Fig. 2A). The calculated AUCs during exercise were $230 \pm 29$ and $228 \pm 33 \mu mol^{-1} \cdot 60 \text{ min}$, respectively ($P < 0.01$). This supports $\alpha$-activity during exercise in SCAT. After training, the exercise-induced increase in dialysate glycerol was significantly higher than before training ($P < 0.05$) in the control probe. In the probe supplemented with phentolamine, the glycerol concentration in dialysate did not differ from that found in the control probe either during exercise or during the recovery period (Fig. 2B). The calculated AUCs during exercise were $152 \pm 21$ and $244 \pm 25 \mu mol^{-1} \cdot 60 \text{ min}$, respectively ($P < 0.01$). Data are expressed as means $\pm$ SE.

Thus $\alpha$-activity in SCAT disappeared after training, which explains the higher lipolysis.

Ethanol outflow-to-inflow ratio in SCAT. Ethanol outflow-to-inflow ratios were expressed as a percentage, i.e., the ethanol concentration in the dialysate divided by the ethanol concentration in the perfusate times 100. At rest, in the control probe, the ethanol ratio was not different before and after training ($77.1 \pm 1.7$ and $78.3 \pm 1.6\%$, respectively); phentolamine did not induce changes in the ethanol ratio ($79.6 \pm 2.3$ and $78.9 \pm 2.3\%$ before and after training, respectively; Fig. 3, A and B). A slight but significant decrease in the ethanol outflow/inflow ratio was observed during the first 15 min of exercise in the control probe as well as in the probe with phentolamine. Before and after training, the addition of phentolamine induced a more prolonged decrease in the ethanol outflow/inflow ratio, which remained significantly lower than before exercise during the whole exercise period.

RER and lipid oxidation. At rest, the RERs were significantly lower ($P < 0.05$) after training than before ($0.75 \pm 0.01$ vs. $0.77 \pm 0.01$). During the exercise, the RER increased during the first 15 min ($0.89 \pm 0.01$ and $0.87 \pm 0.01$, before and after training, respectively) and then decreased during the whole exercise period (Fig. 4). During the 1-h recovery, the RER greatly decreased and reached values lower than those at rest ($0.72 \pm 0.01$ and $0.71 \pm 0.01$, before and after training, respectively). Exercise and recovery data were not significantly different before and after training with respect...
to RER (Fig. 4), but the calculated fat oxidation during 1-h exercise was higher after training than before (27.2 ± 1.8 vs. 22.3 ± 1.7 g lipids/h, P < 0.05). On the other hand, calculated fat oxidation during the 1-h recovery was not different before and after training (8.94 ± 0.47 and 8.69 ± 0.38 g lipids/h, respectively).

**DISCUSSION**

The present study demonstrates that 4 mo of endurance training, without diet modification, increases SCAT lipolysis after a 1-h aerobic exercise bout by decreasing SCAT α2-AR antilipolytic activity in overweight men. On the other hand, for the same relative exercise intensity whole body lipid oxidation increased after training, but the percentage of lipid utilization did not change.

Plasma NEFA concentrations were greatly lowered after exercise training. However, although diet was not modified, loss of fat mass was noticed; this could have contributed slightly to reduced NEFA release. However, NEFA level depends not only on their mobilization from adipose tissue but especially on their utilization by muscle during exercise.

The exercise-induced increase in lipolysis is promoted by increased catecholamine levels and lowered insulinemia. It has been shown that the hormonal response to exercise is determined by relative, and not by absolute, intensity (14). Therefore, in the present study, in both experiments, exercise was performed at the same percentage of V̇O₂ max, and the exercise-induced response of catecholamines (epinephrine and norepinephrine) was similar before and after training. Consequently, the increased exercise-induced lipolysis after training was not associated with different plasma catecholamine concentrations.

The catecholamines control lipolysis by a dual action, stimulation via β-AR and inhibition via α2-AR. The resulting effect of catecholamines is determined by the relative contributions of the two pathways (22). As demonstrated previously in vivo, perfusing an α-AR antagonist (phentolamine) in situ in SCAT by microdialysis probes increases exercise-induced lipolysis in untrained subjects (8, 30); this demonstrates that, during exercise in sedentary men, antilipolytic α2-AR are activated. This antilipolytic effect is more pronounced in obese subjects (29). In our study, the α2-adrenergic responsiveness found in overweight subjects before training might contribute to a lower lipid mobilization. However, only abdominal SCAT was investigated, and we cannot say whether these data are representative of other adipose tissue sites. For example, an in vitro study with isolated abdominal or gluteal adipose cells in obese women showed an α2-AR affinity and density that differed between abdominal and gluteal subcutaneous fat depots (4). It is also necessary to take sexual dimorphism into account. Indeed, our study was restricted to male subjects, and Hellström et al. (15) have shown a different response of α-adrenergic receptors between nonobese men and women: in nonobese women α2-AR activity was not expressed. However, in a previous study (32), we showed that, in obese women, α2-adrenergic receptors are involved in the regulation of lipolysis in SCAT during exercise.

Our in vivo results in overweight men appear to support results of in vitro longitudinal studies (6) in which 12 wk of training elicited a decrease in the α2-antilipolytic action of epinephrine in vitro on fat cells from SCAT of obese men. Moreover, when interpreting the present results, we must consider the differences in fat mass between the two experiments. The higher fat mass before training suggests a higher adipocyte volume, and this could be a reason for the higher α2-AR activity, as it was shown that the α2-AR activity depends on fat cell size (24). Likewise, we found in another experiment (29), that α2-AR activity...
was more striking in untrained obese subjects with higher adipocyte volume than in untrained nonobese subjects, and Hellström et al. have shown that, after weight loss during very low-calorie diet, α2-AR sensitivity decreased (16). However, when obese subjects were trained for 3 mo, although their weight did not change, the antilipolytic effect of α2-AR in vitro was decreased (6); this illustrates the effect of training on AR activity.

Moreover, as already shown (21), plasma insulin concentrations were lower after training, and this could also have contributed to the increased exercise-induced lipolysis. However, plasma insulin levels decreased subsequently to the improved insulin sensitivity (18, 21). In contrast, when α2-AR were blocked, the exercise-induced rise of extracellular glycerol was not different before and after training, which supports an antilipolytic α2-AR activity rather than an insulin antilipolytic effect in SCAT. Then, after only 4 mo, aerobic training led to the disappearance of antilipolytic α2-AR activity (longitudinal effect), as seen in a transversal experiment comparing exercise-trained with sedentary men (8).

The concentration of glycerol in the extracellular space is not determined solely by the rate of lipolysis in adipocytes; it is also influenced by the local blood flow. Pharmacological studies have shown that local blood flow modifies glycerol levels in adipose tissue, i.e., that vasoconstriction increases and vasodilatation decreases extracellular glycerol concentration in adipose tissue (9). During exercise, the increase in extracellular glycerol concentration could be due to changes in blood flow in the adipose tissue. The measurement of ethanol escape through the dialysis probe is a validated nonquantitative method to estimate the changes in vasomotricity in AT (11). In agreement with some authors (15, 30), the stability of the ethanol outflow/inflow ratio found during the exercise bouts indicated that vasomotricity did not change during exercise. Consequently, exercise-induced changes in extracellular glycerol concentration are not influenced by local blood flow changes and consequently reflect changes in local lipolysis in SCAT.

The decreased plasma NEFA after training can be explained by increased lipid oxidation. Regular physical activity is known to improve NEFA utilization in normal-weight subjects (13) mainly during moderate-intensity exercise (3). On the other hand, in obese subjects, lipid oxidation capacity is impaired (5) subsequent to reduction of carnitine palmityltransferase and citrate synthase activity (19). This partly induces high plasma NEFA concentrations. Despite better lipid availability in this population, Ezell et al. (10) did not find differences in lipid oxidation during exercise between obese and nonobese subjects. Few studies have assessed physical exercise effects on lipid oxidation in this population; most experiments associate hypocaloric diet (with great weight loss) with exercise training. In these conditions, exercise training prevents the fall in fat oxidation resulting from diet-induced weight loss in obese men (25). Van Aggel-Leijssen et al. (35)

studied the effect of 3-mo training alone at different intensities (40% and 70% of VO2 max) in obese men. Unlike in our study, these authors showed that lipid oxidation was unchanged at rest but that RER was lower during a 50% VO2 max exercise, especially after training at 40% of VO2 max. The same data were found in women with upper-body obesity; in lower-body obesity, exercise training did not change lipid utilization (34). In our study, the energy supply during exercise was not shifted toward lipids (equivalent exercise RER before and after training), but the VO2 being increased after training (increased exercise workload) and the amount of oxidized NEFA were higher. The method (indirect calorimetry) that we used did not reveal the origin of oxidized NEFA, but it would seem that the nonplasma NEFA were more oxidized after training (17, 27). Even if endurance training did not raise circulating NEFA utilization during an exercise bout, we have shown that the proportion of lipid being oxidized was increased in the 1-h recovery period (23). Moreover, we can assume that, after exercise, intramuscular triglyceride storage was supplied from plasma NEFA, also contributing to the decrease in plasma circulating lipids.

In summary, the present study demonstrates that 4-mo endurance training in overweight subjects improves lipolysis in SCAT during exercise through decreased α2-AR activity. The NEFA level was also very low at rest and remained decreased throughout the exercise and recovery periods. These data can be explained by an increase in the percentage of lipids oxidized at rest and a greater amount of NEFA oxidation during physical activity. This study underlines the effects of training on the α2-adrenergic pathway sensitivity, a determining event for the regulation of human SCAT lipolysis. It indicates the importance of physical training for the regulation of fat mass and for better lipid utilization in obesity prevention.

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