

Lipid oxidation in overweight men after exercise and food intake

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Abstract

Fat oxidation (FO) is optimized during low- to moderate-intensity exercise in lean and obese subjects, whereas high-intensity exercise induces preferential FO during the recovery period. After food intake during the postexercise period, it is unknown if FO differs according to the intensity exercise in overweight subjects. Fat oxidation was thus evaluated in overweight men after low- and high-intensity exercise during the recovery period before and after food intake as well as during a control session. Ten healthy, sedentary, overweight men (age, 27.9 ± 5.6 years; body mass index, 27.8 ± 1.3 kg m⁻²; maximal oxygen consumption, 37 ± 3.9 mL min⁻¹ kg⁻¹) exercised on a cycloergometer (energy expenditure = 300 kcal) at 35% (E35) or 70% (E70) maximal oxygen consumption or rested (Cont). The subjects were fed 30 minutes after the exercise with 300 kcal (1256 kJ) more energy in the exercise sessions than in the Cont session. Respiratory quotient and FO were calculated by indirect calorimetry. Blood samples were analyzed to measure plasma glycerol, nonesterified fatty acid, glucose, and insulin. During exercise, mean respiratory quotient was lower ($P < .05$) and FO was higher ($P < .01$) in the E35 than in the E70 session (FO [in mg min⁻¹]: E35 = 290 ± 12 , E70 = 256 ± 38 , and Cont = 131 ± 7). Conversely, FO was higher in the E70 than in both the E35 session and the Cont session during the immediate recovery as well as during the postprandial recovery period ($P = .005$ for all; FO from the end of the exercise to the end of the session [in grams]: E70 = 45.7 ± 8.9 , E35 = 38.2 ± 6.8 , and Cont = 36.0 ± 4.3). Blood parameters did not differ between the 3 sessions but changed according to the absorption of the nutrients. In overweight subjects, high-intensity exercise increased FO during the postexercise period even after food intake compared with the low-intensity exercise and the control session.

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Contribution of each author to the manuscript:

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1. Introduction

Recent prospective [1,2] and retrospective [3] studies have observed a linear relationship between body mass index (BMI) and the risk of cardiovascular disease, especially in young men [4]. Waist circumference characterizes overweight and obesity in men and is an independent factor of insulin resistance among youths. Strategies for weight decrease are therefore of particular importance for young overweight men and young obese male patients who are concerned about the android body shape and excess weight.

Because exercise appears to be one of the major factors in long-term success in weight maintenance [5], there is a growing number of studies on the role of physical exercise in the management of obesity and metabolic disorders [6]. Indeed, physical exercise improves both lipid mobilization in adipose tissue [7] and fat oxidation (FO) in overweight men [8].

In lean sedentary subjects, the percentage of FO decreases with exercise intensity, whereas energy expenditure (EE) increases [9]. Fat oxidation is thus optimized during low to moderate exercise (30%–50% maximal oxygen consumption [$\text{VO}_{2\text{max}}$]) [10]. In contrast, high-intensity exercise induces preferential FO during the postexercise period [11–14]. In obese patients, similar results were observed in a 24-hour respiration chamber by Saris and Schrauwen [15]: FO during the 3 hours of exercise at 38% maximal aerobic power output is higher than that observed during 1 hour 30 minutes of exercise at higher intensity (80%/50% maximal aerobic power output), whereas FO during the postexercise period tended to be higher after the high-intensity exercise compared with the low one.

In preceding studies in lean subjects and obese patients, FO has been evaluated during the postexercise period in fasted conditions [16] or in feeding conditions with unspecified timing [15,17]. Furthermore, it has been observed that exercising in the fasted state optimizes FO [8,18–20]. Consequently, it is unknown whether postexercise FO is maintained in the fed condition after low- or high-intensity exercise, in particular in overweight subjects. This is of particular interest because, in real-life conditions, overweight subjects find it difficult to avoid eating after exercise and the recommended exercise protocol in a weight management strategy needs to be optimized.

The present study was therefore conducted to investigate the effect of high- and low-intensity exercise matched for equicaloric EE on FO during immediate postexercise recovery and, above all, during the following postexercise recovery period after food intake. The study was conducted in overweight subjects.

2. Subjects and methods

2.1. Subjects

The participants were 10 young (age = 27.9 ± 5.6 years) sedentary ($\text{VO}_{2\text{max}} = 37 \pm 3.9 \text{ mL min}^{-1} \text{ kg}^{-1}$) men. All

participants were overweight (weight = $89.1 \pm 6.1 \text{ kg}$; BMI = $27.8 \pm 1.3 \text{ kg m}^{-2}$; range, 26.2–29.3); there was an excess of fat mass ($27.4\% \pm 3.6\%$), and the waist circumference was increased (waist circumference = $98 \pm 4 \text{ cm}$). They were recruited by advertisements in local newspapers. The study was performed according to the Declaration of Helsinki and approved by the Ethics Committee of Burgundy. The subjects provided written informed consent before the measurements.

In the 15 days preceding the study, participants had a medical checkup with blood pressure measurements, electrocardiogram, blood analysis (glycemia, lipidemia), and fat/lean mass evaluation using a total-body dual-energy x-ray absorptiometer (QDR 4500; Hologic, Waltham, MA).

Inclusion criteria were overweight (BMI between 25 and 30 kg m^{-2}) with a stable body weight during the 3 months preceding the study, good health, no medication, low physical activity ($<2 \text{ h wk}^{-1}$), and no smoking. Exclusion criteria were cardiovascular disease; diabetes mellitus or other metabolic diseases; fasting hyperglycemia ($>6.1 \text{ mmol L}^{-1}$), hypercholesterolemia ($>5.7 \text{ mmol L}^{-1}$), and hypertriglyceridemia ($>1.6 \text{ mmol L}^{-1}$); $\text{VO}_{2\text{max}}$ greater than $45 \text{ mL min}^{-1} \text{ kg}^{-1}$; eating disorders; dieting or fasting; food snacking ($>5\%$ of total daily energy intake); alcohol consumption ($>10 \text{ g d}^{-1}$); and aversions to the foods offered.

2.2. Experimental sessions

The 3 sessions were held on 3 consecutive weeks (1 session per week) according to a randomized crossover procedure. Two sessions differed by the intensity/duration of the exercise imposed. In these exercise sessions, the duration of the cycling exercise was adjusted to ensure a total EE of 300 kcal (1256 kJ). Thus, on one day, the participants performed an exercise session at 35% $\text{VO}_{2\text{max}}$ (E35), which led to an average duration of exercise of 54 minutes (± 6.1 ; range, 44–62); during the other session, the subjects performed an exercise at 70% $\text{VO}_{2\text{max}}$ (E70), which led to an average exercise duration of 26 minutes (± 2.6 ; range, 21–29). A time-matched control session with no exercise (Cont) was carried out to rule out any chronobiological effect.

2.3. Maximal exercise test and exercise sessions

One week before the study, participants' $\text{VO}_{2\text{max}}$ was evaluated using an incremental exhaustive exercise test conducted on an electromagnetically braked bicycle ergometer (Ergometrics 800, Ergoline; Jaeger, Würzburg, Germany). Gases were analyzed on each respiratory cycle using a computerized ergospirometer (Oxycon Pro, Jaeger). During the test, heart rate was continuously monitored using a heart rate monitor (Ergocard, Jaeger); and blood pressure was regularly measured by an exercise-adapted monitor (Tango Stress Test BP Monitor; Suntech Medical Instruments, Raleigh, NC). The initial workload (60 W) was increased by 30 W every 3 minutes until exhaustion. The exercise was considered maximal when the following usual criteria were achieved [7]: maximal heart rate measured at exhaustion

superior to 90% of the age-predicted maximal heart rate, respiratory quotient (RQ) measured at exhaustion superior to 1.1, plateau in VO_2 , and rate of subjects' cycling declining at the end of the test (less than 50 rpm).

During the 2 experimental exercise sessions (E35 and E70), the exercises were performed at a constant oxygen consumption rate of either 35% or 70% $\text{VO}_{2\text{max}}$ and at a constant workload/pedal rate indicated by the ergometer.

2.4. Energy intake and meal composition

During the 15 days preceding the study, individual habitual energy intake was evaluated by a dietary diary. The participants had to report all the food eaten during 7 consecutive days. All the eaten foods were weighed at home on a digital balance accurate to 1 g (QZ3; Terrailon, Chantou, France); and later on, their energy intake was computed by Bilnut 4.0 software (SCDA Nutrisoft, 37390 Cerelles, France).

During the 2 days before each session, individual energy intake was adjusted to that recorded by the dietary diary (mean energy intake at lunch, 1076 ± 193 kcal [4505 ± 808 kJ]), with 20% energy ingested during breakfast, 40% during lunch, and 40% during dinner. From one subject to the other, the meals were of identical nature but individually adjusted in weight (using digital balances, PS 15; EXA, Gradignan, France) to achieve the same nutrient composition (carbohydrates, $56\% \pm 3\%$; lipids, $29\% \pm 3\%$; and proteins, $15\% \pm 2\%$). For each subject, the meals were always identical. All meals were consumed in the laboratory.

During the control session, individual energy intake provided by the meal offered at lunch was calculated to correspond to the usual daily energy intake. The energy content of the lunch in the E35 and E70 sessions was the same as that in the control session plus 300 kcal because the energy spent in each exercise was 300 kcal (1256 kJ). In the 3 sessions, meals served at lunch were of the same nature and composition, with balanced proportions of nutrients (carbohydrates, $55\% \pm 4\%$; lipids, $29\% \pm 3\%$; and proteins, $16\% \pm 1\%$).

2.5. Chronological and general procedure

The design of the 3 experimental sessions is shown in Fig. 1. After 1 night of sleep spent in a chamber of the laboratory, subjects were woken up and, after bladder voiding (first daily urines were not collected, but all following urines until the end of the session were collected as the total urinary excretion over the entire session and analyzed), weighed (precision, 100 g; KCC150; Mettler-Toledo, Albstadt, Germany). At 7:30 AM, a heparinized catheter was inserted into an antecubital vein for subsequent blood samplings. At 8:00 AM, overnight-fasted subjects sat on a reclining chair for preexercise measurements (initial resting period). The duration of this period varied according to the session (Cont, 135 minutes; E35, 75 minutes; E70, 105 minutes). The subjects then exercised on a bicycle ergometer for approximately 60 and approximately 30 minutes ("Experimental sessions") in the E35 and E70 session, respectively. After a second rest period of 30 minutes (immediate recovery) or a corresponding rest period in the

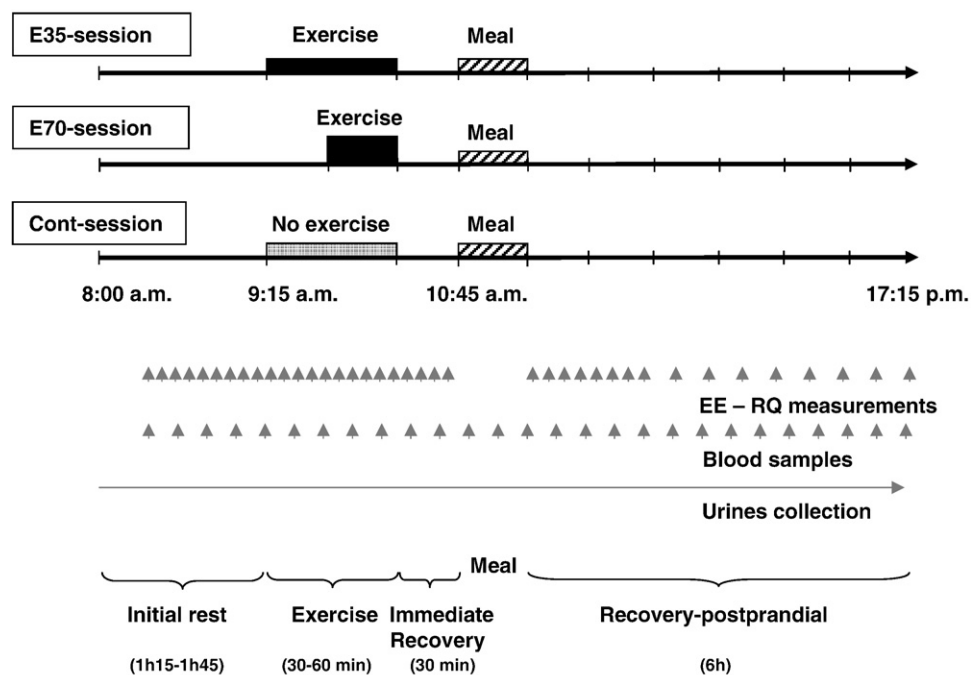


Fig. 1. Design of the experimental protocol in the 3 experimental sessions (Cont, E35, and E70). Each session was randomly assigned. ▲: Energy expenditure and RQ assessments or blood sample withdrawal.

Cont session, subjects were fed. Meals were eaten from 10:45 to 11:15 AM. Subsequently, subjects rested during the following 6 hours (postprandial recovery period).

Throughout each session, subjects sat on a reclining chair close to the bicycle ergometer and were invited to remain calm (subjects could only read or listen to music), except during meal consumption. All measurements were made in a ventilated room to ensure a constantly controlled room gas composition as well as constant ambient temperature and humidity.

2.6. Gas exchange measurements and calculation

Expired gases were collected through an oronasal mask (Hans Rudolph, Kansas City, MO). Gases were collected (Fig. 1) during the last 6 minutes of every successive 15-minute period from the beginning of the initial resting period to the lunch as well as during the first 2 hours after the meal (values during the last 4 minutes before mask removal were averaged for calculation of EE and RQ). Later on (during the last 4 hours of the session), gases were collected during the last 6 minutes of every successive 30-minute period (mean values during the last 4 minutes were used for calculations). Expired gases were measured by an ergospirometer (Oxycon Pro, Jaeger). The analyzers (paramagnetic for O₂, infrared for CO₂) were calibrated at the beginning of each session as well as before and after each exercise period.

From oxygen consumption (V_{O₂}) and carbon dioxide production (V_{CO₂}), EE, RQ, and FO were calculated [21]. After the meal, EE and RQ values corresponded to values not linked to protein oxidation according to urinary nitrogen excretion. Fat oxidation was calculated as FO (in grams per minute) = $1.67 \times (V_{O_2} - V_{CO_2}) - 1.92n$, with n = urinary nitrogen excretion. Urinary nitrogen excretion after the meal was calculated as [(total urinary nitrogen excretion over the entire session)/(session's time)] \times 6. The amount of fatty acids oxidized over time was calculated as FO (in grams) = FO (in grams per minute) \times time (in minutes). Fat oxidation during exercises (E35 and E70) was adjusted with FO during the corresponding period in the Cont session (eg, FO during the E70 exercise session was calculated for the exercise period *plus* an additional period in the resting condition to match the exercise period of the E35 session).

The thermic effect of food (TEF) was calculated in the Cont session as the difference between EE measured during the 6 hours after the meal and EE measured during the hour preceding intake (the latter being extrapolated to 6 hours). Thermic effect of food in the exercise sessions was evaluated from TEF of the control session adjusted to the energy intake in the corresponding session because subjects had eaten 300 kcal (1256 kJ) more energy in exercise sessions than in the Cont session (TEF [exercise sessions] = TEF [Cont session] \times energy intake [exercise sessions]/energy intake [Cont session]). The EE not linked to TEF after the meal intake (ie, during postprandial recovery period) was calculated as the total EE measured during the 6-hour postprandial period

minus the TEF of the corresponding session (similar calculation was done for FO during the postprandial recovery period).

2.7. Blood sampling and biochemical determinations

Three milliliters of blood was drawn every 30 minutes from the initial resting period to the end of the session. The blood, collected on ice, was immediately centrifuged then frozen for further analysis. Glycerol and nonesterified fatty acid (NEFA) in plasma were analyzed by an enzymatic method (Sigma, St Louis, MO, and Wako kit, Unipath, Dardilly, France, respectively). Plasma glucose was determined with a glucose oxidase technique (Biotrol kit; Merck-Clevenot, Nogent-sur-Marne, France). Plasma insulin concentrations were measured using a radioimmunoassay kit from ICN Pharmaceuticals (Orsay, France). Nitrogen in urine was determined by nitrogen elemental analysis (Flash EA1112 analyzer; Thermo-Electron, Waltham, MA) [22].

2.8. Statistical analysis

Data are presented as means \pm SE. A 2-way repeated-measures ANOVA was used to compare RQ and FO between each exercise session and over time. A probability of .05 was used as the 2-sided level of significance for this ANOVA. When a significant main effect of exercise intensity was found, significant pairwise differences for exercise intensity were tested using an ANOVA with repeated measures for the time factor. To control the familywise effect, a Bonferroni correction was applied to fit the controlled level of significance. Statistical analyses were computed using Stata 6.0 software (Stata, College Station, TX).

3. Results

3.1. Energy intake

Energy intake did not differ between the 2 exercise sessions (E35, 1355 ± 134 kcal [5673 ± 561 kJ]; E70, 1322 ± 167 kcal [5535 ± 699 kJ]) but was higher than that offered in the control session (1081 ± 101 kcal [4526 ± 423 kJ], $P < .001$).

3.2. Energy expenditure (Table 1)

During the initial resting period (ie, before exercise), there was no significant difference in EE between the 3 sessions. There was no difference between the 2 exercise sessions with regard to the increase in EE during the exercise. During the immediate recovery period, EE was higher in the E70 than in the E35 session ($P < .01$); both were higher than that during the corresponding period of the control session (E70 vs Cont, $P < .001$; E35 vs Cont, $P < .05$). During the 6 hours of the postprandial recovery period, there was no significant difference between the 3 sessions with regard to calculated EE not linked to TEF as well as total EE.

Table 1

Energy expenditure (in kilocalories) in the 3 sessions (Cont, E35, and E70) according to the initial resting, the exercise, the immediate recovery, and the postprandial recovery periods (TEF)

	Cont	E35	E70
Initial resting (60 min)	88 ± 9	84 ± 7	87 ± 6
Exercise (E35 ~60 min, E70 ~30 min)		314 ± 10	317 ± 10
Immediate recovery (30 min)	43 ± 4	47 ± 4*	56 ± 7 ^{†,‡}
Postprandial-recovery (6 h) (total EE)	628 ± 36	642 ± 37	664 ± 58
Postprandial-recovery (6 h) (total EE minus TEF)	526 ± 53	510 ± 64	535 ± 80

Values are means ± SD.

* $P < .05$ vs Cont session.

[†] $P < .001$ vs Cont session.

[‡] $P < .01$ vs E35 session.

3.3. Respiratory quotient (Fig. 2)

There was no significant difference in RQ during the initial resting period between the 3 sessions. In contrast, mean RQ during exercise was higher in the E70 than in the E35 session ($P < .05$); both were higher than mean RQ measured in the corresponding period of the Cont session (E70 vs Cont, $P < .001$; E35 vs Cont, $P < .01$). During the immediate recovery period, mean RQ was lower in the E70 session than in both the E35 and the Cont sessions ($P < .05$); there was no significant difference between the 2 latter sessions. At any time during the 6 hours of the postprandial recovery period, RQ values were lower in the E70 than in the Cont session ($P = .03$) and moderately lower in the E70 than in the E35 session ($P = .11$).

Considering the 6 hours of the recovery period after the meal, mean RQ values were lower in the E70 compared with Cont ($P = .02$) and E35 sessions (difference did not reach significance, $P = .09$).

3.4. Fat oxidation

During the initial resting period, there was no significant difference in FO between the 3 sessions (Table 2 and Fig. 3). During the bout of exercise, FO (in grams per minute) was

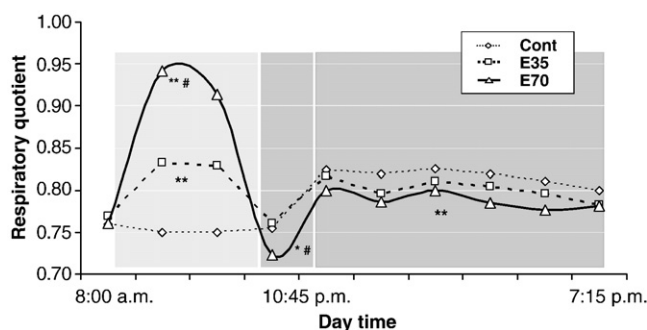


Fig. 2. Respiratory quotient during each session (Cont, E35, and E70). On the x-time axis, from the left to the right, the different periods are represented (initial rest, exercise, immediate recovery, and postprandial recovery). * $P < .05$ and ** $P < .01$ vs control session. # $P < .05$ vs 35% $\text{VO}_{2\text{max}}$.

Table 2

Fat oxidation (in grams) in the 3 sessions (Cont, E35, and E70) according to the initial resting, the exercise, the immediate recovery, and the postprandial recovery periods

	Cont	E35	E70
Initial resting (60 min)	7.4 ± 0.3	6.9 ± 0.5	7.4 ± 0.5
Exercise			
Cont and E35 (~60 min)	7.8 ± 0.8	17.4 ± 0.7 [†]	
Cont and E70 (~30 min)	3.9 ± 0.7		7.7 ± 1.1 ^{†,‡}
Immediate recovery (30 min)	3.6 ± 0.7	3.9 ± 1.3	5.6 ± 1.4 ^{‡,§}
Postprandial-recovery (6 h)	32.4 ± 3.9	34.3 ± 5.7	40.2 ± 7.9 ^{†,‡}
Immediate recovery + postprandial-recovery (6 h 30 min)	35.0 ± 4.3	38.2 ± 6.8 [†]	45.7 ± 8.9 ^{†,‡}

Values are means ± SD. To compare the Cont session to the exercise sessions, FO in the Cont session is expressed during a period paired to the period of each exercise.

* $P < .05$, [†] $P < .01$, and [‡] $P < .001$ vs Cont session.

[§] $P < .05$, ^{||} $P < .01$, and [¶] $P < .001$ vs E35 session.

higher in the E35 than in both the E70 and the Cont sessions (Fig. 3, $P < .01$ for both). During the immediate recovery period, FO was higher in the E70 than in both the E35 and the Cont sessions (Table 2 and Fig. 3, $P < .001$ for both). At any time during the 6 hours of the postprandial recovery period, FO was higher in the E70 compared with the Cont and E35 sessions (Fig. 3, $P = .03$ for both), with no time effect detected.

Considering the 7 hours of the entire recovery period (immediate recovery plus postprandial recovery), cumulative FO was higher in the E70 compared with the Cont and the E35 sessions (Table 2, $P = .005$ for both).

3.5. Blood variables

Plasma glucose, insulin, glycerol, and NEFA did not differ between the 3 sessions whatever the period of

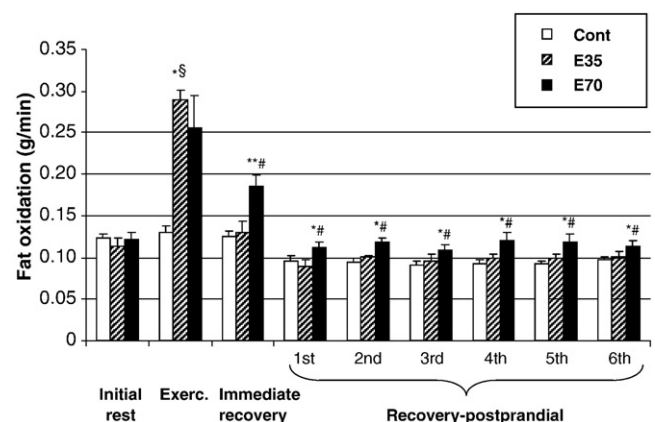


Fig. 3. Fat oxidation (in grams per minute) during each session (Cont, E35, and E70). On the x-time axis, from the left to the right, the different periods are represented (initial rest, exercise, immediate recovery, and postprandial recovery). * $P < .05$ and ** $P < .001$ vs control session. # $P < .001$ vs 35% $\text{VO}_{2\text{max}}$ session. § $P < .01$ vs 70% $\text{VO}_{2\text{max}}$ session.

Table 3

Blood parameters (glucose, insulin, glycerol, and NEFA) in the 3 sessions (Cont, E35, and E70) according to the initial resting, the exercise, the immediate recovery, and the postprandial recovery periods (NEFA)

	Cont	E35	E70
Glucose (mmol L ⁻¹)			
Initial resting	4.4 ± 0.7	5.1 ± 0.6	5.0 ± 0.6
Meal	5.6 ± 1.0 [†]	5.3 ± 1.0	6.4 ± 1.4*
Postprandial-recovery (6th h)	5.0 ± 0.8*	5.0 ± 0.6	5.0 ± 0.8 [‡]
Insulin (mU mL ⁻¹)			
Initial resting	3.6 ± 1.6	3.7 ± 1.8	4.1 ± 1.7
Meal	30.0 ± 23.0 [†]	35.2 ± 28.7 [†]	51.4 ± 40.1 [†]
Postprandial-recovery (6th h)	7.3 ± 3.2 ^{†,§}	9.1 ± 4.3 ^{†,§}	7.3 ± 4.0 ^{†,§}
Glycerol (μmol L ⁻¹)			
Initial resting	93.8 ± 30.9	101.5 ± 49.8	102.4 ± 71.0
Meal	49.5 ± 43.0 [†]	53.1 ± 30.1*	54.4 ± 52.9 [†]
Postprandial-recovery (6th h)	66.8 ± 36.7	62.1 ± 24.8*	107.5 ± 56.0 [§]
NEFA (μmol L ⁻¹)			
Initial resting	0.25 ± 0.09	0.19 ± 0.07	0.19 ± 0.08
Meal	0.19 ± 0.08 [†]	0.20 ± 0.08	0.17 ± 0.10
Postprandial-recovery (6th h)	0.14 ± 0.07 [†]	0.20 ± 0.10	0.20 ± 0.06

Values are means ± SD.

**P* < .05 and [†]*P* < .01 vs initial resting.

[‡]*P* < .05 and [§]*P* < .01 vs meal.

measurements (initial rest, immediate recovery, postprandial recovery) (Table 3). In all sessions, glucose and insulin increased (*P* < .001 for both) and glycerol decreased (*P* < .05) from before to after ingestion of the meal, with values returning to the preprandial values at the end of the postprandial recovery period (ie, during the 6-hour postprandial recovery period).

4. Discussion

The present study conducted in overweight subjects confirms that the ratio of carbohydrate-fat utilization during exercise relied more on carbohydrates than fatty acids when exercise intensity was increased and that the reverse was observed during the subsequent immediate recovery period. Surprisingly, the ingestion of a mixed meal during the postexercise period led to a higher rate of fatty acid oxidation after the high-intensity exercise in comparison with the low-intensity exercise and the control session.

The higher RQ values observed during the higher-intensity exercise in overweight subjects and then the lower RQ values noted during the immediate recovery period after the higher-intensity exercise are in agreement with the results of Saris and Schrauwen [15] in obese patients. Both studies indicate that carbohydrates are the major energy source during high-intensity exercise with compensatory FO during the subsequent postexercise period. The mechanisms leading to the higher FO rate during the

postexercise period could be related, according to others [12,14,16,23,24], to a higher carbohydrate-sparing effect to maintain carbohydrate homeostasis in the fasting state. In normal-body weight subjects, Kuo et al [16] and Henderson et al [25] noted no difference in FO during the postexercise period according to the exercise intensity. The differences between the studies could be due to differences in FO between lean and overweight subjects [26–31].

After ingestion of the mixed meal during the postexercise period, fatty acid oxidation remained higher after the higher-intensity exercise compared with the lower one and the control session (+18% and +5%, respectively). In normal-body weight subjects, several studies reported that ingestion of a meal during the postexercise period did not suppress fatty acid oxidation. Indeed, Krzentowski et al [32] observed that an exercise (3 hours at 50% VO₂max) compared with a control session without exercise increased lipid oxidation for 3 to 7 hours despite a 100-g glucose load in the postexercise period. Likewise, Bielinski et al [33] noted that lipid oxidation in the postexercise period (exercise: 3 hours at 50% VO₂max) was higher than that after a control session without exercise for at least 18 hours, although subjects ingested a mixed meal during the postexercise period. Kiens and Richter [24] then Kimber et al [34] also reported that, during the postexercise period (exercises: 20 minutes at 75% VO₂max then ~90 minutes of alternating 2-minute bouts of 90% and 50% VO₂max), resynthesis of muscle glycogen resulting from exercise-related depletion was a high priority for 18 hours and that lipid oxidation increased during this period despite a high carbohydrate intake. Finally, Folch et al [11] observed that FO was higher during the postexercise postprandial period after the higher-intensity exercise compared with a lower one. In overweight subjects, to our knowledge, FO has never been reported after different exercise intensities then food intake. Our results reveal that high-intensity exercise increases the rate of FO during the postexercise period in comparison with lower-intensity exercise. Thus, the compensatory increase in FO after high-intensity exercise (*a*) was not blunted by ingestion of a mixed meal and (*b*) persisted even after the sixth hour of the postexercise postprandial period.

The compensatory increase in FO following the high-intensity exercise after ingestion of the mixed meal in overweight subjects is of interest because metabolism, which normally turns toward preferential carbohydrate oxidation after the absorption of nutrients [34,35], could have restrained or even abolished the fatty acid oxidation observed during the recovery period. Indeed, it is well known that FO is optimized in the fasted state [18–20]. Moreover, ingesting carbohydrates could have increased insulin secretion that could have limited the lipolytic process and per se the availability of fatty acids for oxidation during the postexercise postprandial period in overweight men [36]. Insulin (like carbohydrates) could also have reduced FO by inhibition of carnitine palmitoyl transferase [37].

Plasma parameters depended principally on ingestion/absorption of the meal in the present observation because glucose and insulin increased whereas glycerol and NEFA changed after food intake to the same extent in both exercise sessions. Similar plasma variations to those in the present experiment have been reported for glucose, insulin, and glycerol after exercise then a high carbohydrate intake [24,34]. Therefore, FO in the E70 session might originate mainly from intramuscular triglycerides and plasma triglycerides [38,39]. The muscle mechanisms leading to the high FO during the postprandial recovery period after high-intensity exercise in overweight subjects remain to be investigated.

Based on the relative use of lipids and carbohydrates as fuels during exercise, numerous studies have led to recommendations of low-intensity exercises in weight management programs for overweight and obese subjects [40,41]. Considering the recovery period, other studies among normal-weight subjects have shown the beneficial effect of high-intensity exercise compared with low-intensity exercise on FO [42–44]. According to these observations and the present results during the recovery period, high-intensity exercise should be considered in the management strategy of overweight subjects. Furthermore, our results indicate that eating a meal even in a large quantity during the recovery period does not suppress FO after high-intensity exercise. This may reinforce the hypothesis, as discussed by Achten and Jeukendrup [45] and as observed by Schneiter et al [46], that eating a meal before or during exercise seems to have a stronger antilipolytic effect than eating the same meal during the postexercise period.

In conclusion, in young overweight male subjects, high-intensity exercise (cycling ~30 minutes at 70% VO_2max) induces higher lipid oxidation after ingestion of a large meal than does low-intensity exercise (cycling ~60 minutes at 35% VO_2max). In clinical practice, this result is of particular interest in the treatment of excessive body weight because (1) the recommended exercise protocol in a weight management strategy needs to be optimized, (2) shorter exercise periods are easier to fit into everyday life, and (3) eating a meal in the postexercise period does not abolish FO induced by high-intensity exercise.

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