

Postexercise macronutrient oxidation: a factor dependent on postexercise macronutrient intake¹⁻³

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ABSTRACT

Background: It has been widely shown that exercise increases postexercise fat oxidation and energy expenditure.

Objective: The aim of this study was to investigate the effect of exercise on postexercise substrate oxidation and energy expenditure when the exercise-induced expenditure of energy and macronutrients oxidized is compensated by an equivalent intake immediately after exercise.

Design: Twenty-four-hour energy expenditure (24EE) and macronutrient oxidation of 8 young men were measured in a whole-body indirect calorimeter under the 2 following, randomly assigned conditions: 1) a control session of sedentary activities in the calorimeter for 61 h and 2) a similar session preceded by 60 min of exercise at 50% of maximal oxygen consumption. Immediately after exercising, subjects ingested a milk shake containing the same amount of energy (above resting metabolic rate) expended during exercise and with a food quotient corresponding to the mean exercise respiratory quotient. 24EE and substrate oxidation were compared between conditions on a day-to-day basis (days 1, 2, and 3) and for the 61-h observation period.

Results: There was no difference in 24EE between the 2 conditions. Moreover, the composition of the postexercise fuel mix oxidized, as reflected by the respiratory quotient, was strictly the same under the 2 conditions.

Conclusion: Voluntary postexercise compensations in energy and macronutrient intakes play a major role in the ability of exercise to alter postexercise substrate utilization. *Am J Clin Nutr* 1999;69:927-30.

KEY WORDS Carbohydrate oxidation, glycogen depletion, fat oxidation, respiratory quotient, fat oxidation, substrate utilization, exercise

INTRODUCTION

The finding that endurance-trained individuals have elevated fat utilization in the resting state (1, 2) has raised the possibility that exercise can produce an overall increase in fat oxidation, particularly in the postexercise state. This effect is particularly relevant for obesity-prone individuals who are known to have reduced fat oxidation after weight reduction (3).

Bielinski et al (4) studied energy expenditure and substrate oxidation in the postexercise recovery period using whole-body

indirect calorimetry. They showed that 17 h after a 3-h exercise session at 50% of maximal oxygen consumption ($\dot{V}O_2\text{max}$) there was a 4.7% increase in resting energy expenditure and a significant decrease in respiratory quotient (RQ). Other studies also showed a persisting increase in metabolic rate after exercise bouts of different durations and intensities (5, 6) as well as reduced RQs (5, 7, 8). Conversely, Weststrate et al (7) did not find any effect of exercise on resting metabolic rate (RMR) 12 h after a 90-min exercise bout at $\approx 25\text{--}35\%$ of $\dot{V}O_2\text{max}$. These results may be related to an exercise intensity that was too low, as shown previously (6).

The enhancement in fat oxidation after exercise reflected by the decrease in RQ may be secondary to the glycogen depletion and to the acute negative energy balance induced by the exercise stimulus. It is possible then that the reduced carbohydrate availability promotes a shift from glucose to fat oxidation. To verify this hypothesis, Calles-Escandon et al (9) submitted 21 subjects to one of four 10-d treatments: 1) control, 2) overfeeding, 3) exercise, and 4) overfeeding and exercise. They found that after an exercise session at 50% of maximal oxygen consumption representing 50% of daily RMR, fat oxidation increased at rest independently of a postexercise dietary compensation matching the cost of exercise.

According to the classic concept developed by Randle et al (10), fat metabolism can substantially alter carbohydrate metabolism. However, little evidence has supported this concept in exercise studies. In a recent study, it was shown that fatty acid oxidation is regulated by carbohydrate metabolism via changes in blood insulin concentrations during exercise (11), probably by controlling the rate of fatty acid entry into the mitochondria (12). Thus, during recovery, the exercise-induced glycogen depletion and the decrease in circulating insulin could provoke an increase in fat oxidation by enhancing fatty acid metabolism, perhaps until carbohydrate stores are replenished. Specifically, this may

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TABLE 1

Age, physical characteristics, maximal oxygen consumption ($\dot{V}O_{2\max}$), and resting metabolic rate (RMR) of subjects¹

Characteristic	Value
Age (y)	24.8 ± 2.8
Weight (kg)	74.7 ± 8.2
Height (cm)	173.0 ± 8.0
Percentage fat (%)	16.6 ± 5.3
$\dot{V}O_{2\max}$ (mL·kg ⁻¹ ·min ⁻¹)	53.7 ± 7.1
RMR (MJ/d)	7.2 ± 1.2

¹ $\bar{x} \pm SD$.

mean that the substrate mix oxidized is altered in the postexercise state as long as carbohydrate balance is not restored, which seems concordant with the theory proposed by Flatt (13).

In the present study, we attempted to document this phenomenon by measuring postexercise macronutrient oxidation under conditions in which liquid supplementation compensating for the carbohydrate and lipid oxidized (over the resting rate) was provided immediately after exercise. We submitted 8 volunteers to an exercise session followed by 61 h in a respiratory chamber to measure postexercise energy expenditure and substrate oxidation.

SUBJECTS AND METHODS

Eight men gave their written consent to participate in this study. They were aged 24.8 ± 2.8 y and were considered moderately active (3–5 h aerobic training/wk). To establish their level of physical condition, each subject was first submitted to a maximal oxygen consumption test on a treadmill. In the same morning, body composition was assessed by using the underwater weighing method and RMR was measured by indirect calorimetry. Physical characteristics, RMR, and the maximal oxygen consumption of subjects are presented in **Table 1**.

The experimental protocol consisted of 2 randomly assigned 61-h stays in a whole-body indirect calorimeter, including 1 control session and 1 session preceded by 60 min of treadmill exercise at ≈50% of maximal oxygen consumption. This particular intensity was chosen because it is an exercise stimulus compatible with the fitness of most individuals as well as its documented potential to alter exercise energy metabolism (4, 8).

Experimental sessions were separated by 1–2 wk and subjects maintained constant body weight and composition before both sessions. Each session included 3 d in the chamber. On day 1, the subject entered the chamber at 1600 and went out at 0900 the next morning (day 2) for a 1-h break. At 1000, he entered the chamber for the second day and went out at 0900 the next morning (day 3) for 1 h. At 1000, he entered the chamber for the last day of testing. The session ended the next morning (day 4) at 0700. On day 1, the first 2 meals of the day were eaten in our laboratory to standardize food intake over the 8 h preceding both sessions. During the exercise session, the subject exercised at 1430 and then took a shower at 1530 before entering the chamber at 1600. The experimental protocol and testing procedures were approved by the Ethical Committee of Laval University.

Maximal oxygen consumption and exercise session

$\dot{V}O_{2\max}$ was determined by an incremental treadmill exercise test to the point of exhaustion (14) using an automated open-circuit gas analysis system. Ventilation (type S340A respirometer;

VacuMed, Ventura, CA) and the oxygen (S3-A analyzer; Applied Electrochemistry Sunnyvale, CA) and carbon dioxide (R1 analyzer; Anarad, Santa Barbara, CA) fractions in expired air were measured every minute during the test to determine oxygen consumption and RQ. An electrocardiogram was recorded and supervised throughout the test according to usual safety conditions in this type of testing. The same system was used during the submaximal exercise session to measure energy expenditure and RQ. Measurements were taken for 5 min at the end of each 15-min period.

Body composition

Percentage body fat was estimated from body density (15), which was determined by the underwater weighing technique. The helium-dilution technique was used to determine residual lung volume, which was performed before the hydrostatic weighing measurement (16).

Twenty-four-hour energy expenditure measurement and substrate oxidation

For each session, subjects stayed in the whole-body indirect calorimeter for a total of 61 h, from 1600 on day 1 to 0700 on day 4. As described previously (17), carbohydrate and lipid oxidation were derived from the rate of oxygen consumption and carbon dioxide production (model Magnox 4G and model Uras P analyzers, respectively; Hartmann and Braun, Frankfurt, Germany) on the basis of the assumption that protein oxidation is closely adjusted to protein intake (18). Each morning, subjects were allowed to get out of the chamber for 1 h to shower and to allow for the room to be cleaned and the analyzers recalibrated. Movements in the chamber were measured by a passive infrared movement detector that detects small movements such as the displacement of an arm.

During both chamber sessions, subjects were fed to maintain energy balance by consuming a diet (a typical Canadian menu) for which the energy content corresponded to 1.32 × RMR (food quotient = 0.85) partitioned into 3 meals (17). Immediately after exercise, an additional diet manipulation (carbohydrates and fat) was performed to compensate for the energy expended and macronutrients oxidized during exercise. A milk shake (2%-fat milk, ice cream, sugar, strawberry jelly, and heavy cream), containing the amount of energy above RMR expended during the exercise and having a food quotient corresponding to the mean exercise RQ, was ingested after the subject had taken a shower and had entered the chamber.

Statistical analysis

Results are expressed as means ± SDs. Each variable was compared between conditions on a day-to-day basis (days 1, 2, and 3), as a 24-h overall mean, and if relevant, as a 61-h total by using paired Student's *t* tests. A two-way analysis of variance was also performed on each variable to assess the effect of exercise and time from day 1 to day 3. Statistical analyses were conducted with JMP software (SAS Institute Inc, Cary NC).

RESULTS

The amount of energy expended above the resting value during the 60-min exercise session was 2.6 ± 0.2 MJ and the mean RQ was 0.912 ± 0.019. The milk shake contained this amount of energy and the food quotient matched the mean exercise RQ.

Energy intake was 9.5 ± 1.2 MJ/d (RMR × 1.32), except on day 1 of the exercise session when the milk shake was ingested



TABLE 2Twenty-four-hour energy expenditure (24EE), heart rate, and movement rate measured in control and exercise sessions¹

Day	Control	Exercise
24EE (MJ/d)		
1	8.3 ± 1.1	8.5 ± 1.0
2	8.3 ± 0.8	8.5 ± 0.8
3	8.7 ± 1.0	8.5 ± 0.7
24-h mean	8.4 ± 0.9	8.5 ± 0.7
Total	25.3 ± 2.8	25.5 ± 2.3
Heart rate (beats/min)		
1	61 ± 4	65 ± 4 ²
2	60 ± 3	62 ± 4 ³
3	62 ± 5	63 ± 5
24-h mean	61 ± 3	63 ± 4 ³
Movement (bursts/24 h)		
1	3236 ± 2062	4287 ± 1789
2	2407 ± 812	1821 ± 1237
3	2121 ± 1041	2013 ± 1191
24-h mean	2588 ± 848	2707 ± 802

¹ $\bar{x} \pm \text{SD}$.^{2,3}Significantly different from control: ² $P < 0.01$, ³ $P < 0.05$.

in surplus. Twenty-four-hour energy expenditure (24EE) was not different in the 2 conditions for each separate day or for the entire 61-h mean or total energy expenditure. There was no significant fluctuation in energy expenditure from day 1 to day 3 in either condition. The absence of difference between conditions was not due to a higher rate of movement during the control calorimeter session because spontaneous activity was not different in the 2 sessions. Heart rate measured in the calorimeter was significantly higher after exercise on day 1 and day 2 and as an overall 24-h mean (**Table 2**).

Exercise followed by a postexercise intake compensation did not modify the composition of the fuel mix oxidized. Indeed, the postexercise RQ was not different between the sessions, whether expressed on a day-to-day basis or over the entire observation period. Hence, postexercise fat and carbohydrate oxidation were not different between the 2 sessions (**Table 3**).

DISCUSSION

We investigated the effect of exercise on prolonged postexercise energy expenditure and substrate oxidation when the energy expended and substrates oxidized during exercise were immediately compensated by isoenergetic macronutrient intakes. 24EE was not different between the control and exercise periods for the first 17 h after exercise or for the entire 61-h chamber session. This was despite a significantly higher heart rate in the exercise compared with the control session in the first 2 d of the protocol.

Several studies (4–9) have investigated the excess of oxygen consumption per se or RMR after exercise. However, many discrepancies exist among the results of these different investigations, possibly because of differences in methodology (eg, exercise intensity and duration, method of measurement, and duration of measurement). Another factor that could have influenced the results is feeding subjects before or after exercise. A few studies have examined energy expenditure over a prolonged period after exercise, necessitating the feeding of subjects during the course of the experimental protocol. Bielinski et al (4) compensated for the energy expended during exercise by increasing the energy intake

TABLE 3Substrate oxidation and mean respiratory quotient during control and exercise sessions¹

Day	Control	Exercise
Lipid oxidation (g/d) ²		
1	73.1 ± 31.2	62.7 ± 22.9
2	59.8 ± 18.6	84.1 ± 30.1
3	69.5 ± 32.9	61.7 ± 30.4
24-h mean	82.0 ± 23.2	82.5 ± 18.6
Total	202.4 ± 57.5	208.5 ± 46.1
Carbohydrate oxidation (g/d) ²		
1	135.5 ± 59.6	174.9 ± 42.5
2	249.9 ± 48.8	215.0 ± 101.0
3	226.9 ± 77.1	229.4 ± 76.6
24-h mean	237.6 ± 37.1	247.1 ± 50.9
Total	612.3 ± 86.8	619.3 ± 129.4
24-h respiratory quotient		
1	0.826 ± 0.05	0.850 ± 0.03
2	0.869 ± 0.03	0.836 ± 0.04
3	0.859 ± 0.05	0.865 ± 0.05
24-h mean	0.851 ± 0.02	0.855 ± 0.03

¹ $\bar{x} \pm \text{SD}$.²Values are raw scores for 17 h on day 1, 23 h on day 2, and 21 h on day 3; the 24-h mean represents the mean of the extrapolated 24-h values.

at breakfast 3 h before the exercise treatment. This early compensation would have placed subjects in a positive energy balance before the treatment and probably influenced the glycogen and fat metabolism during and, consequently, after exercise. Other studies have not compensated for the energy cost of exercise and offered isoenergetic diets during the control and exercise sessions (5, 8). The sustained negative energy balance may also have influenced the subsequent energy expenditure measurements.

The increase in postexercise RMR has been attributed to many factors, including the extra energy cost of storing exogenous carbohydrates as glycogen in the liver and muscles (19), the increased protein synthesis (20), hormonal changes (21), and the increase in catecholamine turnover (4). However, these factors do not appear to be of major importance in this study because a dietary compensation prevented the effect of exercise on energy expenditure.


Insulin changes have been reported after a single bout of exercise (4, 21). During exercise, the insulin concentration was shown to vary according to carbohydrate availability (11), which normally decreases during exercise (2, 3), leading to a decrease in circulating insulin (4, 11). It was also shown that glucose availability and insulin both regulate fat oxidation by reducing the mobilization of plasma fatty acids and oxidation of fatty acids from intramuscular triacylglycerol (11) as well as by controlling the rate of entry of long-chain fatty acids into the mitochondria (12). Taken together, these findings suggest that the carbohydrate depletion induced by exercise favors a decrease in circulating insulin after exercise. This is expected to result in an increase in fatty acid availability and entry into the mitochondria to be oxidized. This could explain the increase in fat oxidation after exercise that was reported previously (4, 5, 7, 8).

Some studies have shown that the RQ can be reduced for as long as 24 h after a single exercise bout (4, 5). Our results do not support these findings, probably because of the immediate nutrient compensation after the exercise. We suggest that the immediate compensation for the substrate mix oxidized, espe-

cially carbohydrates, rapidly replenishes glycogen stores and attenuates the shift to fat oxidation that normally follows exercise. This conclusion is reinforced by the fact that carbohydrate oxidation was not different between the conditions even though carbohydrate intake was higher after exercise because of the surplus provided by the milk shake. We assume that this surplus was used to replenish glycogen stores, according to the RQ-FQ concept proposed by Flatt (13).

Calles-Escandon et al (9) reported a reduction in RQ of 0.051 at rest after exercise training even when energy expended during exercise was compensated for by a mixed supplement. They concluded that exercise increases fat oxidation independently of changes in substrate intake and energy balance. However, because the compensation was not matched to the substrate oxidized, it is hazardous to make such conclusions. We showed that when the FQ of the compensation is matched to the mean exercise RQ, the shift to fat oxidation that normally occurs after exercise is prevented. Further investigations are needed to determine the real effect of such compensation on glycogen stores and insulin action. However, we also showed recently that 60 min of aerobic exercise exerts a daily negative energy balance of as much as 1700 kcal (7106 kJ) when associated with a low-fat diet that provides the equivalent of daily energy expenditure without the cost of exercise (22). Taken together with the findings of the present study, this clearly shows that avoidance of energy and substrate compensation after exercise is of major importance to maintaining a negative energy balance and to deriving the beneficial effects of exercise on substrate oxidation, especially in the course of a weight loss program.

The lack of effect of exercise on substrate oxidation and energy expenditure in the present study cannot be attributed to the moderate intensity and duration of the exercise. In fact, foregoing studies have reported a significant effect on metabolic rate (1, 4, 6) and a reduced RQ up to 18 h (1, 4) after a single bout of exercise (30–180 min) at 50% of maximal oxygen consumption. We are confident that the intensity and duration of the treatment were sufficient to produce an effect on investigated variables and that the equal energy and macronutrient compensation tested in this study was the main factor explaining the lack of effect of exercise on postexercise energy expenditure and substrate metabolism.

The main finding of the present study is the absence of postexercise changes in energy expenditure and substrate oxidation when exercise was immediately followed by a snack containing the same amount of energy and nutrient oxidized during the exercise bout. This is probably because of the accelerated replenishment of glycogen stores and recovery of energy balance. 

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