**Prolonged Daytime Exercise Repeated Over 4 Days Increases Sleeping Heart Rate and Metabolic Rate**

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**Catalogue Data**


**Key words:** lipid oxidation, catecholamines, room calorimeter, endurance trial  
**Mots-clés:** l’oxydation des lipides, catécholamines, chambre calorimétrique, épreuve d’endurance

**Abstract/Résumé**

The aim of this study was to determine the influence of prolonged exercise repeated for 4 days on sleeping heart rate (SHR) and metabolic rate (SMR). Eleven young untrained men exercised at moderate intensity 5 hrs daily for 4 days, alternately on a cycle ergometer (57.0 ± 1.3% VO₂max) and a treadmill (64.7 ± 1.6% VO₂max). They spent the night prior to the exercise period (control, C) and the 4 nights following exercise days (N1 to N4) in room calorimeters for the measurement of SHR, SMR, and respiratory quotient (RQ) from midnight until 6 a.m. Every morning, before the exercise bouts, plasma-free epinephrine (E) and norepinephrine (NE) levels were measured. After exercise, all SHR values were significantly higher than at C level (52 ± 1 bpm, p < 0.001) and the highest value was observed on N2 (61 ± 2 bpm). SMR increased by 11.2 ± 1.5% from C to N1, p < 0.001, and then plateaued up to N4, whereas RQ decreased from C (0.833 ± 0.009) to N2 (0.798 ± 0.005) and then plateaued. Plasma NE levels were higher the morning after each day of exercise and

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peaked on N2, whereas no significant variations were found for E. Variations of SHR between C and N2, and N3 and N4 were correlated with changes of SMR. No significant relationships were found between morning plasma NE, and either SMR or SHR variations. To conclude, prolonged exercise repeated for 4 days was associated with increases in SHR and SMR during the night following each day of exercise concomitantly with an enhanced lipid oxidation. The sustained stimulation of the sympathetic nervous system may be partly responsible for these effects.

L'objectif de cette étude est de déterminer, chez 11 jeunes hommes non-entraînés, l’influence d’un exercice prolongé et répété durant 4 jours sur la fréquence cardiaque (FC) et la dépense énergétique (DE) durant les nuits suivant les jours d’exercice. Cinq heures d’exercice par jour sont effectués durant 4 jours à une intensité modérée sur ergocycle (57,0 ±1,3% VO₂max) et tapis roulant (64,7 ± 1,6% VO₂max) alternativement. Les sujets passent la nuit précédant la période d’exercice (Contrôle, C) et les 4 nuits suivant chaque journée d’exercice (N1 à N4) en chambre calorimétrique afin de mesurer, entre minuit et 6 hr du matin, la FC et la DE ainsi que le quotient respiratoire (QR). Les concentrations plasmatiques d’adrénaline (AD) et de noradrénaline (NORAD) libres sont mesurées chaque matin avant les exercices. Durant la période d’exercice, toutes les valeurs de FC sont supérieures à celle obtenue en C (52 ±1 bpm, p < 0,001), et la valeur la plus élevée est observée en N2 (61 ± 2 bpm). DE augmente de 11,2 ±1,5% de C à N1, p < 0,001, puis se maintient en plateau jusqu’à N4. QR diminue de C (0,833 ± 0,009) à N2 (0,798 ± 0,005), puis reste stable. Les concentrations plasmatiques de NORAD, qui sont plus élevées le matin après chaque jour d’exercice, sont maximales en N2, alors qu’aucune différence significative n’est observée pour AD. Les variations de FC et de DE entre C et N2 ainsi qu’entre N3 et N4 sont corrélées avec les modifications de DE. En revanche, aucune relation significative n’existe entre les concentrations matinales de NORAD et les variations de FC ou de DE. En conclusion, l’exercice prolongé et répété durant 4 jours est associé à une augmentation de FC et DE durant la nuit suivant chacun des 4 jours d’exercice ainsi qu’à une augmentation de l’oxydation des lipides. La stimulation prolongée du système nerveux sympathique pourrait être en partie responsable de ces effets.

Introduction

The effect of exercise on total daily energy expenditure occurs not only during physical activity but also persists during the postexercise recovery period. Consequently, exercising enhances total daily energy expenditure due to increased energy expenditure (EE) linked to the physical activity itself, but also to an increased resting metabolic rate (RMR) after the end of exercise (Westerterp et al., 1994). During the recovery period from exercise, this acute effect on RMR, called “postexercise oxygen consumption” (EPOC), decreases exponentially toward the resting level (Gaesser et al., 1984). However, the duration of EPOC is still contested. It depends probably on intensity and duration of exercise (Poehlman, 1989) and physical fitness of subjects (Short and Sedlock, 1997), and may continue up to 24 hrs after a submaximal exercise of long duration (Maehlum et al., 1986).

The prolonged increase in RMR was found to affect sleeping metabolic rate (SMR) or heart rate (HR), an indicator of RMR, during the night following exercise; this was true whatever the intensity of cycling exercise (Goldberg et al., 1990), or after a 61/2-hr walk (Bonnet, 1980), or after 3 bouts of afternoon walking at 50 to
70% \( \dot{V}_{\text{O}_2} \text{max} \) (Bunnel et al., 1985). However, SMR as measured by whole-body indirect calorimetry was not significantly increased either after a 3-hr moderate-intensity exercise (Bielinski et al., 1985) or after a short, high-intensity exercise (Melanson et al., 2002). In addition, the impact of exercise on subsequent sleeping heart rate (SHR) was more pronounced in untrained than in trained subjects (Walker et al., 1978). Moreover, the effects may depend on the interval of time between the end of exercise and the beginning of sleep. This could explain why O’Connor et al. (1993) failed to show any significant elevated night HR following 30 min of daytime cycling at 75% \( \dot{V}_{\text{O}_2} \text{max} \), before or after training, because the duration of interval between the end of exercise and sleep differed greatly between subjects.

Only one study has examined the effect of daily exercise repeated over several days on night HR (Roussel and Buguet, 1982). Six hours of walking at 35% \( \dot{V}_{\text{O}_2} \text{max} \) during 6 consecutive days induced a night HR elevation of about 10% after each day of exercise compared to the control night. The authors suggested that the increase in nocturnal HR may result from an increase in catecholamine secretion. Indeed, since catecholamines are known to mobilise substrates (Pequignot et al., 1980) and stimulate EE through \( \beta \)-adrenoreceptors (Galbo, 1983), they may contribute to the restorative processes observed after prolonged exercise and participate in the increase in RMR and HR. Although the measurements of plasma catecholamines were lacking in the study conducted by Roussel and Buguet (1982), the hypothesis of persistent sympathetic stimulation during the nights following prolonged exercise repeated over several days is consistent with the results that we observed during a 6-day Nordic ski race. Thus, we have demonstrated that this type of exercise induced a sustained catecholamine release and sympathetic activation which was illustrated by a progressive free and conjugated NE and epinephrine (E) accumulation during the race, and which also plateaued on the 4th day (Fellmann et al., 1992).

In this context, the aim of our work was to study the impact of a 5-hr bout of moderate exercise repeated during 4 days, and simulating our previous field study (Fellmann et al., 1992), on sleeping HR (SHR) and SMR during the night following each day of exercise, and to examine the role of sympathetic nervous system stimulation on these possible changes.

**Material and Methods**

**SUBJECTS**

Eleven young men (age 25.0 \( \pm \) 0.6 years), who were recreationally active in that they exercised 2 to 5 hours per week, volunteered for this study. All were non-smokers. The study received approval from the National Committee of Human Protection in Biomedical Research. The subjects were informed of potential risks involved with the procedure, and all gave their written consent.

Four days before beginning exercises, the subjects undertook an infra-maximal incremental test in 3-min stages on a treadmill (Super 2500, Gymrol, Roche La Molière, France). For the 5-min warm-up, the speed was 7.9 \( \pm \) 0.1 km·h\(^{-1}\). Every 3 minutes the speed was increased by 1.5 km·h\(^{-1}\) until heart rate was near 80% of the theoretical maximal value. On the same day, 2 hours later, maximal oxygen uptake (\( \dot{V}_{\text{O}_2} \text{max} \)) was determined on a cycle ergometer (Ergomeca, La Bayette,
France). The workload was $99.6 \pm 6.1$ W for the 5-min warm-up. Every 3 minutes the load was increased by 30 W until exhaustion. The criteria for determining whether $V\text{O}_2\text{max}$ was reached were a respiratory quotient (RQ) of at least 1.1 and a HR close to the age-predicted maximal value. During these tests an electrocardiogram was recorded continuously (Schiller, Villiers/Marne, France). For both tests, the volume of expired air was collected into Douglas bags for 30 s at the end of each stage and was measured with a Tissot spirometer. CO$_2$ and O$_2$ fractions in the expired air were measured by CO$_2$ and O$_2$ analyzers (CPX I D, MedGraphics, St Paul, MN). The results of these tests were used to adapt the exercise intensity on the cycle ergometer and treadmill during the exercise procedure.

**PROCEDURE**

Subjects were studied in pairs since the laboratory was equipped with two room calorimeters. The study was composed of three periods:

*Adaptation Night.* Five days before beginning the exercises, the subjects spent one evening and night (from 7 p.m. to 6:30 a.m.) in two large open-circuit room calorimeters so they could adapt to their new environment.

*Control Period (C).* The day before the exercise period, subjects entered the room calorimeters at 7 p.m. They spent the rest of the day there watching television, reading, etc. They had dinner at 7:30 p.m. and went to bed at 10 p.m. Heart rate, energy expenditure, and respiratory quotient were measured from 8 p.m. to 6 a.m. The next morning they left the room calorimeter at 6:30 a.m. in a fasted state; they voided the bladder and lay supine on a bed at moderate temperature ($20–22^\circ$C) for 1 hour. At 8 a.m. a venous blood sample was drawn in order to determine catecholamine concentration. The subjects were then scanned by dual-energy X-ray absorptiometry (DEXA) using Hologic QDR 4500 (Hologic Inc., Bedford, MA), and body composition was analysed using the Whole Body version 5.55 software. Due to methodological considerations, DEXA measurements were only taken on 9 subjects. In addition, urine was collected over the 24-hr control period for the measurement of urinary nitrogen (Nu).

*Exercise Period.* The experimental period consisted of 4 similar days during which exercise was performed in the laboratory at moderate temperature (20 to 25 °C) (Figure 1). Each day between 9:15 a.m. and 5:30 p.m. the subjects exercised for 5 hours alternately on an ergocycle and a treadmill, i.e., three 50-min

![Figure 1](data:image/png;base64,iVBORw0KGgoAAAANSUhEUgAAA...)

**Figure 1.** Timing of exercise days (Ex = exercise session).
bouts in the morning and three 50-min bouts in the afternoon, with a 15-min rest period between two bouts. A 2-hr 15-min break was allowed for lunch. The cycle load and treadmill speed, calculated from the data obtained during the previous tests, corresponded to a moderate exercise intensity: 57.0 ± 1.3% $\dot{V}O_2$max (i.e., 121.1 ± 4.9 W) for cycling, and 64.7 ± 1.6% $\dot{V}O_2$max (i.e., 8.2 ± 0.3 km·h$^{-1}$) for running. Food and beverages were allowed ad libitum but were carefully measured. Throughout the procedure, no alcohol or caffeine was consumed.

After the last exercise bout, the subjects showered and went immediately into their respective room calorimeters at 7 p.m. The timing of the rest of the day and night (N1 to N4) was similar to that of Control. At the end of N4, body composition was assessed using the same technique as on C. As for Control, urine was collected over 24-hr periods.

MEASUREMENTS

**Diet.** During the experimental period, food and beverages were freely available during the 4-day exercise period. Individual foods and beverages offered, as well as leftovers, were weighed on a precision scale (±0.1g; LB 3000, Bioblock Scientific, Illkirch, France) to determine food consumption. Energy and nutrient intake (carbohydrate, lipid, and protein percentages) were then assessed from dietary records using Geni software with French (Regal) and German (Souci) tables for food composition (Micro-6, Villers les Nancy, France).

**Energy Expenditure.** From 8 p.m. to 6 a.m., EE was determined by whole-body indirect calorimetry using two large open-circuit room calorimeters for continuous measuring of oxygen consumption and carbon dioxide production (Morio et al., 1997). EE was calculated over periods of 5 min using Brouwer’s equation (Brouwer, 1965):

$$EE \ (kJ) = \ [16.18 \times O_2 \ (l\ STPD) + 5.0 \times CO_2 \ (l\ STPD)] \times 0.99$$

The coefficient of 0.99 was used to take into account the oxidative catabolism of protein because urinary nitrogen excretion during the night was not measured (McLean, 1986). The validation and accuracy of measurements were controlled regularly, as described previously (Vermorel et al., 1995). Sleeping metabolic rate (SMR) was also determined from midnight to 6 a.m. while the subjects were sleeping. Data corresponding to the awake periods that were characterized by a steep increase in HR (>10 bpm) and EE for more than 2 minutes were removed from the pool. In contrast, the steep increases in HR for less than 2 sec, usually observed during REM sleep, were not taken into account and the corresponding data were retained for SMR calculation. Upon awakening, subjects were queried about their sleep, and their responses were compared to the data obtained by HR and EE recordings. Between two nights, the room calorimeters remained closed so that gas concentrations could be kept high and measurements could begin quickly after the subjects returned to the chambers.

During exercise periods, EE was determined from $\dot{V}O_2$ and $\dot{V}CO_2$ measurements. At the beginning and end of each exercise period, the volume of expired air was collected into Douglas bags for 30 s and was measured using a Tissot spirometer. $CO_2$ and $O_2$ fractions in the expired air were measured by $CO_2$ and $O_2$ analyzers (CPX 1 D, MedGraphics, St Paul, MN).
Heart Rate. During the day (from 6:30 a.m. to 7 p.m.) heart rate was recorded every 15 s with a commercially available device (Sport Tester PE 4000 Polar Electro, Kempele, Finland). In the room calorimeters, from 7 p.m. to 6:30 a.m., HR was recorded continuously minute-by-minute using telemetry (Life Scope 6, Nihon Kohden Corp., Tokyo, Japan). Sleeping heart rate was analyzed during the sleep period from midnight to 6 a.m.

Quantity of Lipid Oxidized During the Night. Respiratory quotient (RQ) was calculated as the ratio of CO$_2$ production to O$_2$ consumption. The amount of lipids oxidized from midnight to 6 a.m. was estimated from O$_2$ consumption, CO$_2$ production, and Nu using the equations of indirect calorimetry described by Ferrannini (1988). Even if protein oxidation has an increasing role in energy production during the exercise periods, it can be hypothesized that Nu was proportional to EE.

Catecholamine Assay. Venous blood samples (10 ml) were collected into chilled heparinized tubes (Vacutainer®). They were immediately centrifuged 3,000 g at 4 °C for 15 min. Plasma was then frozen and stored at −80 °C for further analysis. Plasma-free norepinephrine (NE) and epinephrine (E) concentrations were determined by high-performance liquid chromatography assay with electrochemical detection (HPLC-ED) (Sagnol et al., 1990).

**Results**

**MEASUREMENTS**

All the subjects completed all exercise sessions.

Body Composition. The physical characteristics of the subjects are presented in Table 1. There were great variations in body weight (BW) and body composition, with the percentage of fat mass ranging from 11 to 23%. Mean V. O$_2$ max of subjects was 47.3 ± 1.0 ml·kg$^{-1}$·min$^{-1}$ BW.

Body weight did not vary significantly between C and N4 (74.7 ± 2.0 kg vs. 74.9 ± 1.8 kg, $p = 0.68$). No significant difference in fat-free mass (FFM) was found between C and N4 (63.2 ± 1.8 kg vs. 63.5 ± 1.8 kg, $p = 0.33$), whereas fat mass (FM) decreased significantly, from 14.7 ± 1.1% to 14.1 ± 1.0%, $p = 0.03$.

Food Intake. Daily energy intake did not vary significantly during the 4 days of the exercise period, averaging 17.55 MJ. In all, 53.9 ± 0.4% of energy was supplied by carbohydrates, 14.8 ± 0.4% by protein, and 31.3 ± 0.2% by lipids. Energy intake increased significantly at dinner, however, from 4.36 ± 0.12 MJ on C, to 6.41 ± 0.21 MJ on N1; 7.16 ± 0.34 MJ on N2; 6.46 ± 0.21 MJ on N3; and 6.41 ± 0.16 MJ on N4 ($p < 0.0001$). Carbohydrate, protein, and lipid intake increased at dinner from C to the exercise periods, i.e., from 137.6 ± 4.5 g to 204.8 ± 5.6 g for
Table 1  Physical Characteristics of Subjects (N = 11)

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Height (cm)</th>
<th>Body weight (kg)</th>
<th>FFM (kg)</th>
<th>FM (%)</th>
<th>$\dot{V}O_2$max (ml·kg⁻¹·min⁻¹ BW)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ± SEM</td>
<td></td>
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</tr>
<tr>
<td>25.0 ±0.6</td>
<td>177.9 ±1.7</td>
<td>74.7 ±2.0</td>
<td>63.2 ±1.8</td>
<td>14.7 ±1.1</td>
<td>47.3 ±1.0</td>
</tr>
<tr>
<td>Range</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>22.0–29.0</td>
<td>169.0–189.0</td>
<td>62.3–83.6</td>
<td>55.8–71.0</td>
<td>10.5–22.6</td>
<td>41.1–52.7</td>
</tr>
</tbody>
</table>

Note: FFM = fat-free mass; FM = fat mass; $\dot{V}O_2$max = maximal oxygen consumption.

Figure 2. Time course of heart rate (HR) during the night, from 5:30 p.m. to 8 a.m., before (C) and during the exercise period from N1 to N4 (mean values and SEM).

protein; from 45.6 ±0.8 g to 61.5 ±0.9 g for carbohydrate; and from 34.0 ±0.9 g to 57.4 ±1.5 g for lipids, on average.

Heart Rate. The time course of HR during control and exercise days, from 5:30 p.m. at the end of the last exercise session to 8 a.m. just before blood was drawn, is shown in Figure 2. Between 5:30 and 8 p.m., HR decreased during exercise days from 141 ±2 bpm to 83 ±2 bpm, on average. The highest values were observed on N1, the lowest on N4; the greatest difference between N1 and N4 (19 bpm) was found at 5:40 p.m. Between 8:30 p.m. and 8 a.m., HR still decreased from, on average, 83 ±1 bpm to 60 ±1 bpm for exercise days, and from 69 ±3 bpm to 52 ±2 bpm for C. Heart rate was significantly higher during N1 to N4 than
Table 2  Measurements (mean ± SEM) During Sleep and During the Exercise Period (N1–N4)

<table>
<thead>
<tr>
<th></th>
<th>SMR  (kJ·min⁻¹)</th>
<th>SHR (bpm)</th>
<th>RQ</th>
<th>Lipid oxidized (g)</th>
<th>Glucose oxidized (g)</th>
<th>E (pmol·L⁻¹)</th>
<th>NE (pmol·L⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>4.99 ± 0.10</td>
<td>52 ± 1</td>
<td>0.833 ± 0.009</td>
<td>20.6 ± 1.5</td>
<td>45.1 ± 3.6</td>
<td>164 ± 55</td>
<td>1805 ± 27</td>
</tr>
<tr>
<td>N1</td>
<td>5.54 * ± 0.10</td>
<td>59 * ± 1</td>
<td>0.825 ± 0.008</td>
<td>25.9 * ± 1.6</td>
<td>47.3 * ± 3.3</td>
<td>240 ± 55</td>
<td>2260 * ± 22</td>
</tr>
<tr>
<td>N2</td>
<td>5.61 * ± 0.11</td>
<td>61 * ± 1</td>
<td>0.798 *+ ± 0.005</td>
<td>30.4 *+ ± 1.5</td>
<td>35.4 *+ ± 2.4</td>
<td>191 ± 22</td>
<td>2982 *+ ± 20</td>
</tr>
<tr>
<td>N3</td>
<td>5.60 * ± 0.08</td>
<td>59 *† ± 1</td>
<td>0.795 *+ ± 0.008</td>
<td>30.4 *+ ± 1.7</td>
<td>35.1 *+ ± 2.8</td>
<td>158 ± 22</td>
<td>2231 *† ± 17</td>
</tr>
<tr>
<td>N4</td>
<td>5.72 *+ ± 0.10</td>
<td>58 *†‡ ± 1</td>
<td>0.803 *+ ± 0.009</td>
<td>30.1 *+ ± 2.0</td>
<td>38.6 *+ ± 3.3</td>
<td>186 ± 22</td>
<td>2290 *† ± 195</td>
</tr>
</tbody>
</table>

Note: Metabolic rate (SMR), heart rate (SHR), and respiratory quotient (RQ) during sleep from midnight to 6 a.m., and morning plasma-free epinephrine (E) and norepinephrine (NE) concentrations before (C) and during the exercise period N1 to N4.

Values significantly different: From C, *p ≤ 0.05; From N1, †p ≤ 0.04; From N2, ‡p ≤ 0.05 (paired t-test).

during C. In addition, from midnight to 6 a.m., mean SHR was also higher during N1 to N4 than during C: 7 ± 1 bpm higher on N1; 9 ± 1 bpm higher on N2; 7 ± 1 bpm higher on N3; and 6 ± 1 bpm higher on N4, compared to C (p < 0.001). Heart rate was slightly but significantly higher during N2 than during N3 and N4 (Table 2).

Figure 3 illustrates the time course of SHR during each night between midnight and 6 a.m. SHR decreased in this period during N1 to N3, p < 0.0001, and tended to decrease during C with a minimal value observed at 4:45 a.m. (50 ± 1 bpm, p = 0.1). In addition, the difference in SHR between C and N1 to N4 was higher from midnight to 2 a.m. (10 bpm on average at midnight) than later on (7 bpm on average at 6 a.m.). During the first part of the night (midnight to 2 a.m.) of the exercise period, SHR exhibited the highest value on N2 and the lowest value on N4. The values obtained on N1 and N3 were not significantly different. For the subsequent sleep period, 2 to 6 a.m., the SHR values did not differ significantly between the N1-to-N4 period.

Energy Expenditure and Sleeping Metabolic Rate. From 7 p.m. to 6:30 a.m., energy expenditure decreased significantly from 7.85 ± 0.36 to 4.85 ± 0.17 kJ·min⁻¹ for C, and from 9.39 ± 0.27 to 5.40 ± 0.14 kJ·min⁻¹ for exercise periods N1 to N4, on average.
From midnight to 6 a.m., mean SMR values were 11.8 ± 1.2% higher, on average, during N1 to N4 than during C (Table 2), with the highest increase obtained on N4 (+13.5 ± 0.6%, p < 0.0001). The kinetics of SMR during the four nights are shown in Figure 4. During all the nights, SMR decreased continuously from midnight to 6 a.m. and was always higher during N1 to N4 than during C.

RQ and Lipid Oxidation. RQ decreased significantly from C to N2, by 0.035 ± 0.007 unit, p < 0.001, and then plateaued from N2 to N4 (Table 2). The quantity of oxidized lipids increased significantly from C to N1 by 30.5 ± 9.4%, p = 0.005, and from N1 to N4 by another 20.4 ± 6.3%, p ≤ 0.05, that is, 55.3 ± 11.8% from C to N2–N4. In contrast, glucose oxidation decreased significantly from C to N2–N3 (−17.9 ± 7.5% on average, p = 0.03). The contributions of glucose and lipid oxidation to SMR amounted to 39.3 ± 3.1 and 45.6 ± 3.3%, respectively, during the Control night, and to 30.4 ± 2.1% and 54.2 ± 2.6%, respectively, during N1 to N4.

Plasma-Free Catecholamines. Plasma norepinephrine (NE) levels on the morning after each exercise day were significantly higher than the value obtained on C, with a peak observed on N2 (+69 ± 14%, see Table 2), whereas no significant differences were found for epinephrine (E) concentrations between C and the exercise period (ANOVA: p = 0.31).

CORRELATIONS

No relationships were found between variation (in percent) of either SMR or SHR and morning plasma NE between C and the exercise period N1 to N4. The percents of variation of SHR and SMR were positively correlated between C and N2 (p = 0.04; r² = 0.38) and between N3 and N4 (p = 0.02; r² = 0.46).
Discussion

This study demonstrated that prolonged exercise repeated over 4 days induced an increase in both heart rate and metabolic rate during the postexercise nights, with a concomitant shift toward lipid oxidation.

As the activity was not standardised from 8 p.m. until the subjects fell asleep, and may have differed according to subjects and nights, we focused on the prolonged effects of exercise only during the sleep period. The most important finding was that SMR (+11.8 ± 1.2% on average) was elevated during the nights following each day of prolonged exercise. The elevation of night EE was not due to a change of fat-free mass (FFM), which is a major determinant of basal metabolic rate (Westerterp, 1992), because no changes of FFM as measured by DEXA were detected after the 4 days of exercise. In the present study the elevation of SMR may be interpreted as due to the exercises performed earlier in the day.

It is now recognized that the increased oxygen consumption which occurs during exercise declines slowly after the cessation of exercise. This EPOC may depend on the magnitude and duration of exercise (Maehlum et al., 1986). In the present study, the prolonged (5 hrs daily) and moderate-intensity exercises (57–65% $\dot{V}O_2$max) that were imposed may have been sufficient to induce an elevation of metabolic rate which persisted during the night. The mean elevated SHR (Table 2), a valid indicator of metabolic rate, supports this hypothesis. These results are consistent with those reported by Roussel and Buguet (1982), who found an increase in SHR of 10.2% as compared to Control during the night following each exercise day (6 hrs walking at 35% $\dot{V}O_2$max) repeated over 6 days. This increase was slightly lower than that observed in our study, +13.8% on average for the 4
nights. Moreover, it is noteworthy that the progressive decline in SHR observed in C, with a minimal value obtained at 4:45 a.m. reflecting the HR endogenous circadian rhythm (Degaute et al., 1991), was maintained during the nights following the exercise days but at a higher level.

Three main mechanisms could be evoked to explain the increase in SHR and SMR over the nights following the exercise days: (a) the effect of elevated body temperature; (b) an increased thermic effect of food (TEF); and (c) a persistent sympathetic stimulation.

First, the elevation of body temperature (Q_{10} effect) during and after exercise may contribute to the increase in metabolic rate during the night. However, Roussel and Buguet (1982) have ruled out the effect of postexercise nocturnal hyperthermia on HR increase because they failed to find an increase in body temperature in all their subjects after a 6-hr march.

Second, the TEF on energy expenditure during the night must also be considered. Indeed, energy intake was greater from N1 to N4 as compared to C. In addition, several researchers showed that prior exercise potentiated TEF (Pi-Sunyer and Segal, 1992; Poehlman, 1989). However, the increase in EE due to TEF decreases rapidly during the first 4 hours following a meal (Pi-Sunyer and Segal, 1992). Therefore it is difficult to estimate the contribution of TEF to the increase in EE between midnight (4\ 1/2 hrs after dinner) and 6 a.m.

Finally, and in agreement with previous reports (Buguet et al., 1980; Roussel and Buguet, 1982), the higher SHR and SMR observed during the nights of N1 to N4 as compared to C may be due to the sympathetic nervous system (SNS) stimulation induced by the exercise sessions. Therefore plasma-free NE concentrations, which are known to induce tachycardia (Bulbring and Tomita, 1987) and stimulate cellular energy production processes (Brehm, 1988), were in fact elevated at the end of each night of the exercise period. Since free NE is the reflection of instantaneous SNS activation and not a delayed indicator (Sagnol et al., 1990), the increased free NE level observed at the end of each night was probably due to an increased SNS activity during exercise which may have persisted throughout the night. Moreover, it is conceivable that the difference between SHR during the Control night and Nights N1 to N4, which was higher at the beginning than at the end of the night, was due to a decrease in catecholamine concentrations during the nights.

As compared to C, the lower nighttime RQ over the exercise periods indicates an enhanced contribution of lipid oxidation to energy production and a decrease in glucose oxidation (Table 2). These alterations were observed from the first night for lipids and from the second night for carbohydrates, indicating a fast metabolic adaptation of the tissues. Furthermore, the increased fat oxidation agrees with the significant loss of fat mass (0.5 \pm 0.2 kg, \( p = 0.01 \)) at the end of the exercise protocol. This shift toward lipid oxidation supports the hypothesis of a possible role of NE in stimulating fatty acid mobilization and oxidation (Snitker et al., 1998) during the night. Thus the persistence of a high NE level during recovery from exercise may contribute to the restoration of glycogen (Sagnol et al., 1989).

This costly restorative process has been suggested as being one of the slow components of EPOC (Bielinski et al., 1985; Borsheim et al., 1998; Brehm, 1988). The high plasma NE level may also have enhanced the triglyceride/fatty acid cy-
clinging rate (Borsheim et al., 1994), which is known to be a costly regulatory process (Wolfe et al., 1990). As suggested by Bahr et al. (1990), the energy cost of the stimulation of this cycle may account for the elevation of postexercise EE.

Although morning NE levels were always higher during the exercise period than after C, the repetition of exercise days may have attenuated sympathetic stimulation. The NE plasma levels and SHR during N3 and N4 were indeed lower as compared to N2, although exercise was of the same magnitude and duration and ended at the same time of day. Such adaptation processes have been described previously: the catecholamine response to a prolonged cycle test was blunted following short-term training (90 to 120 min per day for 3 days; Helyar et al., 1997). A possible increase in NE turnover could be also suggested; however, at present there are no data on such events. The HR decrease may also be related to the plasma volume expansion usually observed after such endurance events (Fellmann, 1992; Green et al., 1991). The increase in plasma volume may generate a substantial filling of the left ventricle, which through the Starling effect induced a greater stroke volume. Consequently, the same cardiac output and oxygen consumption could be maintained with a lower HR. This last phenomenon could explain the dissociation between SHR and SMR trends in the present study.

To conclude, the 4-day prolonged daytime exercise of moderate intensity increased SHR and SMR values during the night following each day of exercise. The increase in plasma catecholamines, which were induced by the exercise bouts and persisted throughout the night, may have triggered in part the increase in EE and favoured lipid oxidation. These results also point to the need to take into account the increase in energy expenditure during the recovery period, especially during the night following such events, in order to evaluate the total energy requirements.

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