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Effects of one night's sleep deprivation on anaerobic performance the following day

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Abstract The purpose of this study was to determine the effect of one night's sleep deprivation on anaerobic performance in the morning and afternoon of the following day. Thirteen healthy males were studied twice in a balanced, randomized design. The experiment consisted of two conditions 1 week apart. In the sleep deprivation condition (SDN) subjects remained awake overnight and in the control condition (reference night, RN) the same subjects slept at home, retiring between 2230 and 2330 hours, as decided individually, and rising at 0500 hours. In both conditions, activity, sleep and diet were monitored by actimetry and daily activity and dietary diaries. Physical performance testing was carried out at 0600 hours and at 1800 hours after the one night of sleep and the one night of sleep deprivation. At each test occasion, subjects were measured for maximal power (P_{\max}), peak power (P_{peak}) and mean power (P_{mean}). Blood lactate concentrations were measured at rest, at the end of the force–velocity (F – V) test, just before and just after the Wingate test and again 5 min later. Oral temperatures were measured every 2 h. In both conditions, the results showed a circadian rhythm in temperature. Analysis of variance revealed a significant (sleep \times time of day of test) interaction effect on P_{peak} , P_{mean} and P_{\max} . These variables improved significantly from morning to afternoon after RN and SDN. The reference night was followed by a greater improvement than the SDN. Up to 24 h of waking, anaerobic power variables were not affected; however, they were impaired after 36 h without sleep. Analysis of variance revealed that blood lactate concentrations were

unaffected by sleep loss, by time of day of testing or by the interaction of the two. In conclusion, sleep deprivation reduced the difference between morning and afternoon in anaerobic power variables. Anaerobic performances were unaffected after 24 h of wakefulness but were impaired after 36 h without sleep.

Keywords Anaerobic performance · Daily variations · Lactate · Sleep deprivation · Temperature

Introduction

Many factors are associated with limiting work performance, one of which is sleep deprivation. Although athletes and coaches believe that adequate sleep is essential for peak performance, there are many situations in which sleep is disturbed prior to an athletic event. An athlete may lose sleep owing to jetlag or anxiety. The effects of sleep deprivation on a person's rating of perceived exertion, mood and cognitive functions are well documented (Bonnet 1980; Martin 1981; Angus et al. 1985; VanHelder and Radomski 1989). However, when it comes to the effect of sleep deprivation on physical or physiological performance, the findings are conflicting and less conclusive (Bulbulian et al. 1996). The reason for the discrepancies may be that various exercise modes, frequencies, intensities and durations have been employed, as well as different evaluation procedures (Symons et al. 1988a). Other factors that may explain the differences among such studies are varying durations of sleep deprivation and subjects' ages. Another factor that is not always taken into account is that the circadian rhythms of recorded variables might persist during sleep deprivation (Angus et al. 1985; Jewett et al. 1999).

The effects of sleep deprivation on cardiovascular changes during aerobic exercise have been studied extensively (VanHelder and Radomski 1989). Few studies, however, have investigated the role of sleep deprivation on performance at short-term exercise involving

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anaerobic metabolism. A few studies of short-term maximal effort have reported how supramaximal performance can be maintained despite sleep deprivation. Symons et al. (1988a) demonstrated no significant change in the isometric strength of flexors and extensors, peak isokinetic torque at $3.14 \text{ rad}\cdot\text{s}^{-1}$, muscular endurance, peak power output, fatigue index or blood lactate after the Wingate anaerobic test. Takeuchi et al. (1985) further showed that 64 h without sleep did not impair isometric hand-grip strength or peak torque for leg extension at 3.14 and $5.22 \text{ rad}\cdot\text{s}^{-1}$. However, sleep deprivation did impair vertical jump and knee extension torque at low velocity ($1.04 \text{ rad}\cdot\text{s}^{-1}$). Conversely, Bulbulian et al. (1996) reported that sleep loss of up to 30 h affected peak torque but had no effect on the fatigue index.

Changes in sleep-wake patterns or the dephasing of the circadian rhythms of various physiological variables could account for substandard athletic performance (Ilmarinen et al. 1980; Johnson 1982). The existence of circadian rhythms in human performance is now well established (Reilly et al. 1997). The major rhythms relevant to an examination of performance are body temperature and the sleep-wake cycle. The possible responses of circadian rhythms to aerobic performance have been studied extensively (Reilly et al. 1997). However, few studies have investigated the time-of-day dependence of responses to short-term exercise involving anaerobic metabolism, and they have produced conflicting results. Comparing Wingate test results, Hill and Smith (1991) and Melhim (1993) found that values were higher in the afternoon than in the morning. But with the same test, Down et al. (1985) and Reilly and Down (1992) reported no circadian fluctuation of these parameters. Just one study has been carried out on diurnal variation in a force-velocity test ($F-V$) (Bernard et al. 1998). The authors noted higher maximal anaerobic power in the afternoon than in the morning. A variety of factors such as type and intensity of exercise, morningness-eveningness chronotype, age, jet-lag, sleep deprivation, and time of day of training can have a bearing on the daily variations of performance levels (Reilly et al. 1997). We also know that such rhythms are reduced or tend to disappear when external indicators of clock time (*zeitgebers*) are removed (Conroy and Mills 1970). Montelpare et al. (1992) showed how circadian rhythms in cardiorespiratory and gas exchange responses to a moderate standardized treadmill walking task are gradually attenuated during a period of sleep deprivation. To our knowledge, the effects of sleep deprivation on anaerobic exercise during cycle ergometry the following morning and afternoon have not been assessed. It was the aim of the present study to confirm that performance in $F-V$ and 30-s Wingate tests has time-of-day effects, and to examine the effects of one sleepless night of any fluctuations.

The problems with designing such studies are: (1) the amount of supervision exercised over the subjects (or lack thereof) (VanHelder and Radomski 1989); and (2) subjects experienced an unequal amount of stress while

participating in the sleep deprivation condition and control condition (Symons et al. 1988a). The present investigation got round these problems by implementing strict control of the amount of rest and activity in both the sleep deprivation and the control conditions. The presupposition was that performances would be altered more in the afternoon than in the morning on the day after one night of sleep deprivation than after the control night.

Methods

Subjects

Thirteen healthy male physical education students [mean (SD) age 22.4 (2.4) years, height 1.8 (0.1) m, body mass 67.7 (6.6) kg] volunteered to take part in the study. Subjects were fully informed of the procedures and signed a consent form before participating, and ethical approval for the investigation was secured from the ethics committee of the CHU Cote de Nacre, Caen, France. They were in good state of fitness. Two weeks before, they had passed their physical test in order to graduate as physical education students at the university. They were non-smokers and took no medication. In order to recruit a homogenous group, all subjects were intermediate type, having been selected on the basis of their chronotypes, using the Horne and Östberg (1976) "morningness-eveningness" questionnaire (score between 42 and 58). Selected subjects also had regular sleeping schedules based on the Bastuji and Jouvet (1985) calendar, completed over a period of 1 month. Mean estimated sleep duration was 7.5 (0.5) h. Subjects rose at 0630 (0030) hours and retired to bed at 2300 (0030) hours.

Experimental design

The experimental protocol was in two parts, A and B. In Part A, each subject slept at home (retiring at any time between 2230 and 2330 hours, as decided individually), rising at 0500 hours to come to the laboratory. That day, all subjects performed two test sessions at 0600 (0600 RN, i.e. 1 h awake) and 1800 hours (1800 RN, i.e. 13 h awake). Instructions about sleep and diet were given to the subjects prior to experimentation. They were required to eat a standard isocaloric meal at 0730, 1200 and 2000 hours taken together. Only water was allowed ad libitum between meals. Just a cool drink of water was taken prior to the 0600 hours test. During the period of investigation subjects were prohibited from taking in food or drink any known stimuli (e.g. caffeine) that would possibly enhance wakefulness, or agents such as alcohol. Subjects were requested to maintain their habitual physical activity throughout the experimental period, and to avoid strenuous activity during the 24 h preceding the test sessions. Compliance with directions relating to pre-test sleep, activity and diet was checked by actimetry and daily activity and dietary diaries. Part B was identical except that no sleep was allowed during the night. Subjects arrived at the laboratory at about 2200 hours and were not allowed to go to sleep at their usual bedtime. During this time, subjects did such things as watch television, read books, play music or work at a computer. They were strictly supervised by two experimenters so as to ensure that no-one dozed off or consumed alcohol or beverages containing caffeine. In this Part B, the test sessions were also performed at 0600 (0600 SDN, i.e. 24 h awake) and 1800 hours (1800 SDN, i.e. 36 h awake). Parts A and B were carried out at least 1 week apart in random order, and subjects followed their normal routines during the week before each part. Spontaneous body movement was assessed continuously by wrist actigraphy (Gachwiler Electronic). Each actigraph contained a piezoelectric transducer sensitive to movements of 0.1 g acceleration. Actimetric devices were worn on the non-dominant arm from 2000 hours the day before the

tests to the end of the experiment. The actigraphs were returned to the laboratory and the data downloaded from the memory into a PC via an Actigraph Interface Unit. Time series were first edited using information listed in the diaries to remove segments during which the monitor was not worn. The variables were sleep period (when the activity level was less than two movements per minute), the activity level during the day (activity counts/1-min epochs) and the immobility index during the night (II, %): percentage of epochs with an activity count = 0, reflecting the fragmentation of sleep. In Part A, subjects fell asleep at 2240 (0015) and they slept for 0625 (0015) hours. The activity level during the day was not statistically different (ANOVA) in the two experimental conditions. All the test sessions took place in similar conditions of temperature and relative humidity [20 (1)°C, 50 (5)%, respectively]. Each test session began with the measurement of body mass on digital scales. The hourly schedule for both conditions is presented in Table 1.

Temperature

In Part B, oral temperatures were recorded with a digital clinical thermometer (accuracy $\pm 0.05^\circ\text{C}$) inserted sublingually for at least 3 min at 0200 hours and every 2 h thereafter. These recordings were always carried out before test sessions and meals, with subjects having rested in a supine position for at least 15 min. In Part A, to avoid disturbing the subjects' sleep, no temperature recordings were made during the night. In order to obtain a recording at 0200 hours, the subjects were asked to wake up themselves during a normal night 1 week after the end of the experiment.

Exercise testing

Maximal power (P_{\max}), peak power (P_{peak}) and mean power (P_{mean}) of the legs were calculated according to the F - V test protocol proposed by Vandewalle et al. (1987) and the Wingate test proposed by Bar-Or (1987). The tests were performed on an Ergomeca cycle ergometer with an electronic rev-counter and recorder fitted onto the wheel.

Force-velocity test

The F - V test involved repetitive short maximal sprints (6 s) against increasing braking forces set before the start of the exercise. Seat height was adjusted for each subject. This height was recorded and kept the same for each subject in each trial. The feet were held in

the pedals with toe-clips. The subject remained in a sitting position during each F - V test and was vigorously encouraged to reach the maximal pedalling rate as quickly as possible. The test began with a braking force of 29.4 N. After 5 min of recovery, the braking force was increased by anything from 14.7 N to 19.6 N depending upon a subject's capability and the same exercise was repeated until subjects were unable to reach a peak velocity higher than 100 $\text{rev}\cdot\text{min}^{-1}$. The subjects generally performed six or seven short all-out sprints. Peak velocity (V) was measured during each sprint for each braking force (F) and used to calculate the F - V relationship for cycling exercises according to the least-squares method. These F - V relationships are linear for peak velocities ranging from 100 to 200–220 $\text{rev}\cdot\text{min}^{-1}$ (Vandewalle et al. 1987). The relationship between force and velocity can be expressed as follows:

$$V = V_0(1 - F/F_0)$$

where V_0 is the intercept with the velocity axis; and F_0 the intercept with the force axis. An estimate of maximal velocity at zero braking force is presented by V_0 . It is assumed that F_0 is the braking force corresponding to zero velocity. Given the linear F - V relationship in cycling, the optimal braking force and the optimal velocity [i.e. the braking force and the pedal velocity corresponding to maximal power (P_{\max})] are equal to 0.5 F_0 (F_{opt}) and 0.5 V_0 (V_{opt}). Therefore, P_{\max} is equal to:

$$P_{\max} = 0.5V_0 \cdot 0.5F_0 = 0.25V_0F_0$$

Wingate test

The Wingate test involved a 30-s maximal sprint against constant resistance. For each subject the load was determined according to body mass using the optimization tables of Bar-Or (1987) (0.087 $\text{kg}\cdot\text{kg}^{-1}$ body mass). Subjects were given vigorous verbal encouragement during the test. Seat height was adjusted to each subject's satisfaction and toe-clips were used to prevent the subject's feet from slipping off the pedals. This height was recorded and kept the same for each subject throughout the trials. P_{peak} is the highest mechanical power elicited during the test. This index was taken as the highest average power over any 3- to 5-s period. P_{mean} is the average power sustained over the entire 30-s period.

For both the F - V and Wingate tests, the characteristics of the cycle ergometer enabled P_{\max} , P_{peak} and P_{mean} to be calculated as follows: (1) for the F - V test P (W) = [F_{opt} (kg) \cdot 9.81 \cdot V_{opt} ($\text{rev}\cdot\text{min}^{-1}$)/60] \cdot 6.12 m = F_{opt} (kg) \cdot V_{opt} ($\text{rev}\cdot\text{min}^{-1}$) and (2) for the Wingate test P (W) = [F (kg) \cdot 9.81 \cdot V ($\text{rev}\cdot\text{min}^{-1}$)/60] \cdot 6.12 m = F (kg) \cdot V ($\text{rev}\cdot\text{min}^{-1}$). In both cases 6.12 m is the distance covered by a point on the rim of the flywheel per pedal revolution.

Table 1 Hourly schedule for both conditions

Time (hours)	Activity
2000	Actimetry Isocaloric dinner
2200	Part A: go to sleep Part B: subjects gather in the laboratory
0500	Part A: awakening of subjects
0600	Test session Beginning of F - V test
0630	End of F - V test
0700	Wingate test
0730–0800	Isocaloric breakfast
0800–1200	Lesson, being seated
1200–1300	Isocaloric lunch
1330–1730	Lesson, being seated
1800	Test session Beginning of F - V test
1830	End of F - V test
1900	Wingate test
2000	Isocaloric dinner

Blood lactate concentrations

Prior to each test session, a hyperemia-inducing cream was applied to the lobe of one ear. Blood samples (10 μl) were collected from this ear lobe at rest, at the end of the F - V test, just before (i.e. 30 min after the end of the F - V test), just after the Wingate test then again 5 min later. The samples were analysed using an entirely enzymatic method to determine lactate concentration using lactate oxidase, which catalyses the oxidation of lactate to pyruvate. The different readings were taken with the Plus LP 20 miniphotometer (by Dr Lange, Germany).

Statistical analysis

The data were analysed using multiple analysis of variance (MANOVA) [2 (sleep) \times 2 (time of day of the test)] with repeated measures on both factors. MANOVA was followed by separated two-way ANOVA analysis (sleep \times time of day of the test) to detect which variable was affected. When appropriate, significant differences among means were tested using Tukey's post-hoc test.

Table 2 Mean (SD) values for body mass, peak power (P_{peak}), mean power (P_{mean}), maximum power (P_{max}), braking force corresponding to zero velocity (F_0) and maximal velocity at zero braking force (V_0) ($n = 13$) at the two times of day after reference night (RN) and after sleep deprivation (SDN)

Variables	After reference night		After sleep deprivation		Statistical differences – ANOVA post hoc analyses			
	0600 RN (1 h awake)	1800 RN (13 h awake)	0600 SDN (24 h awake)	1800 SDN (36 h awake)	0600 RN vs. 1800 RN	0600 SDN vs. 1800 SDN	0600 RN vs. 0600 SDN	1800 RN vs. 1800 SDN
Body mass (kg)	67.8 (6.8)	67.8 (6.6)	67.9 (6.5)	67.6 (6.6)	***			
P_{peak} ($W \cdot kg^{-1}$)	10.2 (0.7)	11.1 (0.7)	10.3 (0.9)	10.7 (0.6)	***	*	ns	*
P_{mean} ($W \cdot kg^{-1}$)	7.9 (0.7)	8.6 (0.7)	7.9 (0.8)	8.3 (0.8)	***	**	ns	*
P_{max} ($W \cdot kg^{-1}$)	14.1 (1.2)	15.2 (1.4)	13.9 (0.9)	14.5 (1.2)	***	***	ns	***
F_0 (kg)	18.1 (1.6)	18.6 (1.5)	18.1 (1.1)	18.1 (1.4)	***			***
V_0 ($rev \cdot min^{-1}$)	208.3 (7.1)	217.7 (8.4)	208.9 (6.5)	213.2 (7.9)	***	*	ns	***

ns $P > 0.05$, * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$

The phase and amplitude of the circadian oscillator (the “body clock”) were estimated by Cosinor analysis of the temperature data. A least-squares regression analysis using Cosinor analysis was employed to determine the best fit of the combined 24-h period cosine function, i.e. $Y(t) = M + A \cos(\omega t + \phi)$ where M is the rhythm-adjusted mean or mesor (midline estimating statistic of rhythm), A is the amplitude (equal to 0.5 of the peak-to-trough variation due to rhythmicity), ϕ is the acrophase (time of the maximal level in circadian rhythm referenced to local 0000 hours) and $\omega = 2\pi/\tau$ the angular speed (in our study, the angular frequency is one cycle per day as τ is equal to 24 h). A Student’s t -test for paired samples was used to compare the temperature phase values of cosine function parameters. A probability level of 0.05 was selected as the criterion for statistical significance. All statistical tests were processed using STATISTICA Software (StatSoft, France).

Results

The MANOVA including all variables led to a significant effect of Sleep (Wilks’s $\Lambda < 0.001$, $P < 0.001$) and time of day (Wilks’s $\Lambda < 0.001$, $P < 0.001$) as well as a significant interaction between the two factors (Wilks’s $\Lambda < 0.001$, $P = 0.001$).

Body mass

Body mass data are given in Table 2. The two-way ANOVA (sleep \times time of day of the test) for body mass indicates no significant interaction, no significant main effect for sleep and no significant main effect for time of day of the test.

Wingate test

The results of the Wingate test variables calculated at the two times of day after RN and SDN are given in Table 2.

Peak power

For P_{peak} , there was a significant interaction ($F_{(1, 12)} = 11.09$; $P = 0.005$) (sleep \times time of day of the test). The post-hoc test revealed that after both RN and SDN, P_{peak} improved significantly from morning to afternoon ($P < 0.001$ for RN and $P = 0.04$ for SDN). The improvement was greater after RN [9.3 (4.5)%] than after SDN [3.9 (5.9)%, $P = 0.008$]. Considering the effect of sleep deprivation, in comparison with the reference night, no significant difference was observed after 24 h of sleep loss ($P > 0.05$), but P_{peak} was significantly lower after 36 h of sleep deprivation ($P = 0.01$).

Mean power

For P_{mean} , a significant ($F_{(1, 12)} = 5.44$; $P = 0.03$) (sleep \times time of day of the test) interaction was found. After RN

and after SDN, the P_{mean} improved significantly between morning and afternoon ($P < 0.001$ for RN and $P = 0.004$ for SDN). The improvement was greater after RN [7.7 (5.4)%] than after SDN [4.5 (3.8)%, $P = 0.03$]. The results also indicate that P_{mean} was not affected by 24 h of sleep loss ($P > 0.05$). However, it was decreased after 36 h of sleep deprivation ($P = 0.03$).

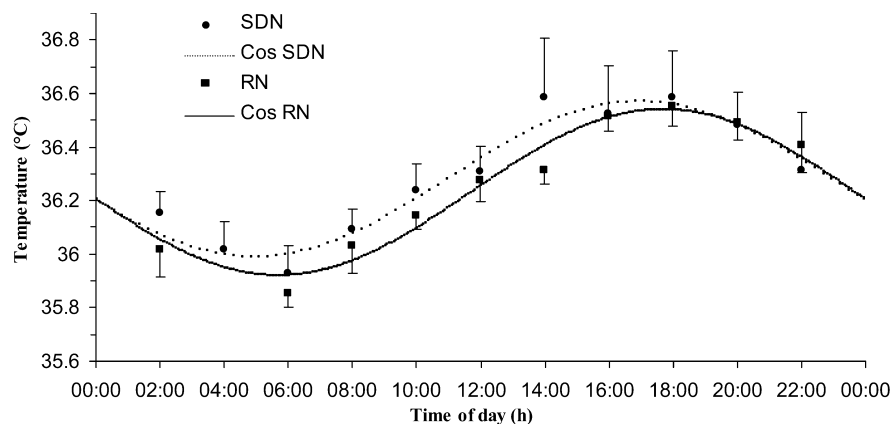
F–V test

The results of the F–V test variables calculated at the two times of day after RN and SDN are given in Table 2. For P_{max} , there was a significant ($F_{(1, 12)} = 17.91$; $P = 0.001$) (sleep \times time of day of the test) interaction. The post-hoc test revealed that after both RN and SDN, P_{max} improved significantly from morning to afternoon ($P < 0.001$ for RN and $P < 0.001$ for SDN). The improvement was greater after RN [8.3 (6.2)%] than after SDN [3.7 (5.7)%, $P = 0.002$]. Sleep deprivation of up to 24 h did not lower P_{max} ($P > 0.05$), but P_{max} was decreased by 36 h of sleeplessness ($P < 0.001$). There was also a significant interaction effect for sleep by time of day of the test ($F_{(1, 12)} = 6.69$; $P = 0.02$) for V_0 . Results of post-hoc tests revealed that V_0 improved significantly from morning to afternoon after RN and SDN ($P < 0.001$ for RN and $P = 0.03$ for SDN). The improvement was higher after RN [4.5 (3.4)%] than after SDN [2.1 (1.4)%, $P = 0.02$]. Sleep deprivation of up to 24 h did not affect V_0 ($P > 0.05$), but V_0 was significantly lower after 36 h of sleep loss ($P < 0.001$). As for F_0 , it was unaffected by sleep, by time of day of the test, or by the interaction between the two.

Table 3 Mean (SD) values of blood lactate ($\text{mmol}\cdot\text{l}^{-1}$) ($n = 13$) at the two times of the day after the reference night (RN) and after sleep deprivation (SDN)

	After reference night		After sleep deprivation	
	0600 RN (1 h awake)	1800 RN (13 h awake)	0600 SDN (24 h awake)	1800 SDN (36 h awake)
Rest	1.24 (0.15)	1.22 (0.10)	1.23 (0.16)	1.22 (0.17)
End F–V test	2.53 (0.38)	2.56 (0.30)	2.52 (0.29)	2.55 (0.46)
Pre Wingate test	1.36 (0.18)	1.35 (0.27)	1.35 (0.13)	1.36 (0.14)
End Wingate test	5.92 (1.47)	6.16 (1.43)	5.93 (1.00)	5.92 (1.20)
At 5 min post Wingate test	11.28 (0.17)	11.32 (0.49)	11.32 (0.25)	11.32 (0.47)

Fig. 1 Circadian rhythm in oral temperature during reference night (RN) and sleep deprivation (SDN) conditions. Mean values (SD) are shown. Best-fit curves between the experiment data and the cosine curve (Cos RN ; $r = 0.98$, $P < 0.001$ and Cos SDN ; $r = 0.97$, $P < 0.001$)



Blood lactate concentrations

Mean values for lactate after RN and SDN at the two times of day are given in Table 3. Regarding lactate analysis, the two-way ANOVA revealed that for all measurements blood lactate concentrations were not affected by sleep loss, by time of day of the test, or by the interaction between the two.

Circadian rhythm of oral temperature

Mean values for oral temperature data at each test time during RN and SDN conditions are shown in Fig. 1. A circadian rhythm of temperature was present in both instances. Cosinor analysis of oral temperature data gave the result shown in Table 4. This table indicates that there was a slight significant phase advance under sleep-deprivation conditions (0044 (SD 0052) hours, $P < 0.02$).

Discussion

This study was designed in such a way that any difference in physical performance between conditions could be attributed to sleep deprivation. Since both conditions (RN and SDN) contained an identical exercise protocol it was possible to isolate the effects of sleep loss when comparisons were made between conditions (Symons et al. 1988a).

In the present study, the morning test was always conducted before the afternoon test. The way round this

Table 4 Cosinor analysis of temperature data during Reference night (RN) and Sleep deprivation (SDN) conditions values are given as the mean (SD) of $n=13$ subjects

Variable	Reference night	Sleep deprivation
Mesor (°C)	36.24 (0.07)	36.29 (0.03)
Amplitude (°C)	0.32 (0.04)	0.30 (0.09)
Acrophase (hours)	1740 (0028)*	1700 (0044)

*Significant difference from acrophase after sleep deprivation

quandary is to duplicate everything, with the order counter-balanced, but this doubles the workload and the inconvenience to subjects. Since two tests were administered on the same day, it is possible that an ordering effect contributed to the findings. It is not assumed, however, that young, fairly fit physical education students would be affected by fatigue after $F-V$ and Wingate tests performed twice with 12 h between each test session. Stamford et al. (1978) found no difference in maximal oxygen uptake ($\dot{V}O_{2max}$) during three tests repeated with as little as 10 min between each test. Melhim (1993) observed that female physical education students were not affected by fatigue after a Wingate test performed four times with 6 h between each test. Moreover, in both conditions the subjects had higher P_{peak} , P_{mean} and P_{max} at 1800 than at 0600 hours. Thus, it is unlikely that there were any residual fatigue effects. Twelve hours was enough to recover from the morning anaerobic tests. Alternatively, it is also unlikely that there was any benefit from a "warm up" effect of morning exercise – 12 h is a long break and cumulative effects of this kind would have been observed in the earlier studies cited above, which were performed closer together.

The effect of one night without sleep (i.e. 24–36 h of sleep deprivation) on the following day's morning and afternoon fluctuations in performance in $F-V$ and 30-s Wingate tests was investigated in the present study. After a reference night, P_{peak} and P_{mean} were higher in the afternoon than in the morning. These results were consistent with previous reports (Hill and Smith 1991; Melhim 1993). Conversely, Down et al. (1985) and Reilly and Down (1992) reported no circadian rhythm. The latter authors suggested that the strong level of motivation required from subjects during the 30-s Wingate test might interfere with the results and minimize the time-of-day effect. The time-of-day effect on P_{max} observed in the present study agrees with the findings of Bernard et al. (1998). During this last-mentioned study, subjects were tested only three times over a 24-h period (at 0900, 1400 and 1800 hours). Even the peak-to-trough (5–7%) observed for maximal power is likely to have been underestimated considering that there was only 9 h between the morning and afternoon recordings and that the earliest session was at 0900 hours, whereas in the literature the times of minimal circadian rhythm values were generally observed between 0500 and 0600 hours. In the present study the two daytime points (0600 and 1800 hours) used were times at which normal circadian rhythms are beginning

to rise (0600 hours) or have passed the normal peak (1800 hours).

After SDN, the time-of-day effect observed for P_{peak} , P_{mean} and P_{max} persisted but with differences between morning and afternoon that were significantly lower than after RN. Previous studies have reported that the circadian rhythm might be attenuated during a period of sleep deprivation. Ahnve et al. (1981) demonstrated that the circadian pattern to the cardiovascular variables is disturbed or obliterated by sleep deprivation, which keeps synchronizers constant. Similarly, Montelpare et al. (1992) showed that circadian rhythms in cardiorespiratory and gas exchange responses to a moderate, standardized treadmill walking task became progressively attenuated during a period of continuous waking. In a recent study, we have shown that during continuous regular physical exercise involving total sleep deprivation over 24 h, isometric maximal strength still varies with time of day (Callard et al. 2000).

In the present study, for any measurements taken after RN and SDN, blood lactate concentrations were unaffected by chronobiological effects. Accordingly the time-of-day effect in power variables was not reflected in the blood lactate responses. The present study demonstrates that $F-V$ exercise elicits statistically significant elevations in blood lactate and that this response occurs at each of the time points chosen for testing. This is consistent with previous studies, which document how the stimulus of $F-V$ exercise is sufficiently potent to cause an increase in blood lactate (Mercier et al. 1989, 1991).

Oral temperature showed a circadian rhythm after both RN and SDN, but sleep loss was associated with a slight phase advance in the rhythm. These results are consistent with a previous report (Meney et al. 1998). The phase advance could indicate an advance of the circadian oscillator (the body clock) or be a mathematical effect due solely to the marked increase of temperature by 1400 hours. The former is possible because an individual is exposed to artificial light throughout a night of sleep deprivation, including the initial part of the circadian rhythm, which is a time of great sensitivity to any form of light (Honma et al. 1987; Czeisler et al. 1989). Boivin et al. (1996) and Minors and Waterhouse (1994) indicated the effectiveness of low-intensity light (as would come from artificial indoor lighting) in achieving shifts of about an hour or so, this being the position in the present investigation, which was conducted close to the winter solstice when sunrise is at about 0800 hours. In both conditions (RN, SDN) oral temperature was higher at 1800 hours than at 0600 hours. Thus, the higher value of P_{max} , P_{peak} , and P_{mean} in the afternoon than in the morning after RN and SDN may be linked to changes in body temperature (Melhim 1993; Bernard et al. 1998). The exact mechanisms are not known but it has been suggested that the higher body temperature may enhance metabolic reactions, increase the extensibility of connective tissue, reduce muscle viscosity and increase the conduction

velocity of action potentials (Shephard 1984). Bergh and Ekblom (1979) have demonstrated how in warming and cooling experiments maximal anaerobic power drops by 5% for every 1°C drop in muscle temperature.

The present study showed that performances of the Wingate and $F-V$ tests were unaffected after subjects had experienced 24 h of sleep deprivation. These results agree in part with one previous report (Mougin et al. 1996) after partial sleep deprivation. A number of studies of short-term maximal efforts have also reported that supramaximal performance can be maintained under sleep deprivation. Takeuchi et al. (1985) showed that a sleep deprivation of 64 h had no effect on the times for the 40-m dash, or isometric strength and peak torque for leg extension at 3.14 and 5.22 rad·s⁻¹. Cunningham et al. (1986) showed that performance of the Wingate test was unaffected after 48 h of sleep deprivation. Symons et al. (1988a, 1988b) found that maximum isometric and isokinetic muscular strength and endurance, peak power output, mean power output, fatigue index, blood lactate after the Wingate test, maximal voluntary contraction (MVC), the rate of force development, and the time required to produce 25%, 50%, 75% and 100% of MVC were not affected by sleep deprivation of at least 60 h. Hill et al. (1994) reported that anaerobic capacity as defined by oxygen deficit was unaffected by one night's sleep loss.

Interestingly, the present study shows that 36 h of sleep deprivation resulted in a decrement of P_{peak} , P_{mean} and P_{max} , which to our knowledge has never been demonstrated before. This decrease might have even been greater if the subjects had slept longer during the reference night. Duration of sleep deprivation (24 h vs. 36 h) did not explain why anaerobic performances were affected by 36 h of sleep deprivation and not by 24 h of sleeplessness because P_{peak} , P_{mean} and P_{max} were higher at 1800 hours (36 h awake) than at 0600 hours (24 h awake). One possible explanation of our results is cumulative fatigue working against the normal circadian rhythm. It was previously suggested that sleep deprivation may cause central or peripheral fatigue in subjects (Takeuchi et al. 1985). Another possible explanation for these results is to consider that they are dependent on the time of day of the recordings and that the acrophase and the amplitude of the rhythm of P_{peak} , P_{mean} and P_{max} has been affected by sleep deprivation. To our knowledge, in previous studies attempting to analyse the same energy system, no recordings have been taken in the late afternoon. Unfortunately our experimental design only picked two times of the day, which is not enough to verify this hypothesis. Having more recordings (i.e. every 4 h) requires recovery from the muscular effort made at each test session, which is not possible in a study of sleep deprivation because it multiplies the workload and the inconvenience to subjects. The decrease in power variables may occur because subjects are less motivated and aroused. Sleep deprivation affects primarily the higher central nervous system cognitive centres (Bonnet 1980), and motivation is a key factor in

the validity of tests of anaerobic power and capacity. The classic observation of Ikai and Steinhaus (1961) showed how maximum voluntary muscular contraction is closely dependent upon the level of arousal in the individual being tested. The impact of diurnal variations in arousal upon muscular performance is also well documented (Shephard 1984). Sleep deprivation both dampens and distorts the normal circadian cycle of arousal, with a decrease of alpha wave activity on the electroencephalogram (Kollar et al. 1966; Akerstedt 1979; Shephard 1984). The lack of effect of 24 h of sleep loss on P_{peak} , P_{mean} and P_{max} may be due to the fact that motivation and arousal are less affected by sleep loss at 0600 hours the following day.

Our results indicate that blood lactate concentrations were unaffected by sleep deprivation for any measurement taken in the morning or afternoon. The duration of the $F-V$ and Wingate tests was probably too short to induce maximal utilization of anaerobic glycolytic potential (Bedu et al. 1991). Accordingly, the decrease in anaerobic performance in the $F-V$ and Wingate tests after 36 h of sleep deprivation is not explainable in terms of blood lactate accumulation.

Finally, the mechanism whereby sleep loss decreases anaerobic performance only after 36 h of sleep deprivation is not clear. Further studies are needed to address the underlying mechanism behind the effect of one night's sleep deprivation on anaerobic performance.

In conclusion, one night's sleep deprivation reduced the difference between morning and afternoon in P_{peak} , P_{mean} and P_{max} values. Sleepiness impaired anaerobic performance at 1800 hours after 36 h of wakefulness. However, morning responses to Wingate and $F-V$ tests were unchanged by sleep loss after 24 h of wakefulness in our experiment or more (up to 60 h) in the literature. Some involvement of circadian rhythm impairment may have to be considered.

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References

- Ahnve S, Theorell T, Akerstedt T, Froberg JE, Halberg F (1981) Circadian variations in cardiovascular parameters during sleep deprivation. A noninvasive study of young healthy men. *Eur J Appl Physiol* 46:9–19
- Akerstedt T (1979) Altered sleep/wake patterns and circadian rhythms. *Acta Physiol Scand Suppl* 469:1–48
- Angus RG, Heslegrave RJ, Myles WS (1985) Effects of prolonged sleep deprivation, with and without chronic physical exercise, on mood and performance. *Psychophysiology* 22:276–282
- Bar-Or O (1987) The Wingate anaerobic test an update on methodology, reliability and validity. *Sports Med* 4:381–394
- Bastuji H, Jouvet M (1985) Intérêt de l'agenda de sommeil pour l'étude des troubles de la vigilance. *Electroencephalogr Clin Neurophysiol* 60:299–305

- Bedu M, Fellmann N, Spielvogel H, Falgaraitte G, Van Praagh E, Coudert J (1991) Force-velocity and 30-s Wingate tests in boys at high and low altitudes. *J Appl Physiol* 70:1031-1037
- Bergh U, Ekblom B (1979) Influence of the muscle temperature on maximal muscle strength and power output in human skeletal muscles. *Acta Physiol Scand* 107:33-37
- Bernard T, Giacomoni M, Gavarry O, Seymat M, Falgairette G (1998) Time-of-day effects in maximal anaerobic leg exercise. *Eur J Appl Physiol* 77:133-138
- Boivin DB, Duffy JF, Kronauer RE, Czeisler CA (1996) Dose-response relationships for resetting of human circadian clock by light. *Nature* 379:540-542
- Bonnet MH (1980) Sleep, performance and mood after the energy-expenditure equivalent of 40 hours of sleep deprivation. *Psychophysiology* 17:56-63
- Bulbulian R, Heaney JH, Leake CN, Sucec AA, Sjöholm NT (1996) The effect of sleep deprivation and exercise load on isokinetic leg strength and endurance. *Eur J Appl Physiol* 73:273-277
- Callard D, Davenne D, Gauthier A, Lagarde D, Van Hoecke J (2000) Circadian rhythms in human muscular efficiency: continuous physical exercise versus continuous rest. A crossover study. *Chronobiol Int* 17:693-704
- Conroy RTWL, Mills JN (1970) Human circadian rhythms. Churchill, London
- Cunningham DA, Noble EG, Paterson D, Pettigrew F, Taylor AW (1986) Combat engineer fatigue in sustained operations. Final contract report to Canadian Department of national Defence, project No. 01SE. 97711-4-8015
- Czeisler CA, Kronauer RE, Allan JS, Duffy JF, Jewett ME, Brown EN, Ronda JM (1989) Bright light induction of strong (type 0) resetting of the human circadian pacemaker. *Science* 244:1328-1333
- Down A, Reilly T, Parry-Billings M (1985) Time of day and performance of the anaerobic test. *J Sports Sci* 3:214
- Hill DW, Smith JC (1991) Circadian rhythm in anaerobic power and capacity. *Can J Sport Sci* 16:30-32
- Hill DW, Borden DO, Darnaby KM, Hendricks DN (1994) Aerobic and anaerobic contributions to exhaustive high-intensity exercise after sleep deprivation. *J Sports Sci* 12:455-461
- Honma K, Honma S, Wada T (1987) Phase-dependent shift of free-running human circadian rhythms in response to a single bright light pulse. *Experientia* 43:1205-1207
- Horne JA, Östberg O (1976) A self assessment questionnaire to determine morningness-eveningness in human circadian rhythms. *Int J Chronobiol* 4:97-110
- Ikai M, Steinhaus AH (1961) Some factors modifying the expression of human strength. *J Appl Physiol* 16:157-163
- Ilmarinen J, Ilmarinen R, Korhonen O, Nurminen M (1980) Circadian variation of physiological functions related to physical work capacity. *Scand J Work Environ Health* 6:112-122
- Jewett ME, Dijk DJ, Kronauer RE, Dinges DF (1999) Dose-response relationship between sleep duration and human psychomotor vigilance and subjective alertness. *Sleep* 22:171-179
- Johnson LC (1982) Sleep deprivation and performance. In: Webb WB (ed) *Biological rhythms, sleep and performance*. Wiley, New York, pp 111-141
- Kollar EJ, Slater GR, Palmer JO, Doctor RF, Mandell AJ (1966) Stress in subjects undergoing sleep deprivation. *Psychosom Med* 28:101-13
- Martin BJ (1981) Effect of sleep deprivation on tolerance of prolonged exercise. *Eur J Appl Physiol* 47:345-354
- Melhim AF (1993) Investigation of circadian rhythms in peak power and mean power of female physical education students. *Int J Sports Med* 14:303-306
- Meney I, Waterhouse J, Atkinson G, Reilly T, Davenne D (1998) The effect of one night's sleep deprivation on temperature, mood, and physical performance in subjects with different amounts of habitual physical activity. *Chronobiol Int* 15:349-363
- Mercier J, Mercier B, Prefaut C (1989) Participation of lactic anaerobic metabolism during short and intense repeated exercises. *C R Seances Soc Biol Fil* 183:60-66
- Mercier J, Mercier B, Prefaut C (1991) Blood lactate increase during the force velocity exercise test. *Int J Sports Med* 12:17-20
- Minors DS, Waterhouse JM (1994) Deriving a "phase response curve" from adjustment to simulated time zone transitions. *J Biol Rhythms* 9:275-282
- Montelpare WJ, Pyley MJ, Shephard RJ (1992) Evaluating the influence of sleep deprivation upon circadian rhythms of exercise metabolism. *Can J Sport Sci* 17:94-97
- Mougin F, Bourdin H, Simon-Rigaud ML, Didier JM, Toubin G, Kantelip JP (1996) Effects of a selective sleep deprivation on subsequent anaerobic performance. *Int J Sports Med* 17:115-119
- Reilly T, Down A (1992) Investigation of circadian rhythms in anaerobic power and capacity of the legs. *J Sports Med Physical Fitness* 32:343-347
- Reilly T, Atkinson G, Waterhouse J (1997) *Biological rhythms and exercise*. Oxford University Press, Oxford
- Shephard RJ (1984) Sleep, biorhythms and human performance. *Sports Med* 1:11-37
- Stamford BA, Rowland R, Moffatt RJ (1978) Effects of severe prior exercise on assessment of maximal oxygen uptake. *J Appl Physiol* 44:559-563
- Symons JD, VanHelder T, Myles WS (1988a) Physical performance and physiological responses following 60 hours of sleep deprivation. *Med Sci Sports Exerc* 20:374-380
- Symons JD, Bell DG, Pope J, VanHelder T, Myles WS (1988b) Electro-mechanical response times and muscle strength after sleep deprivation. *Can J Sport Sci* 13:225-230
- Takeuchi L, Davis GM, Pyley M, Goode R, Shephard RJ (1985) Sleep deprivation, chronic exercise and muscular performance. *Ergonomics* 28:591-601
- Vandewalle H, Pérès G, Monod H (1987) Standard anaerobic exercise tests. *Sports Med* 4:268-289
- VanHelder T, Radomski MW (1989) Sleep deprivation and the effect on exercise performance. *Sports Med* 7:235-247