

# Effects of vigorous late-night exercise on sleep quality and cardiac autonomic activity

TERO MYLLYMÄKI<sup>1</sup>, HEIKKI KYRÖLÄINEN<sup>1</sup>, KATRI SAVOLAINEN<sup>2</sup>, LAURA HOKKA<sup>1</sup>, RIIKKA JAKONEN<sup>2</sup>, TANJA JUUTI<sup>1</sup>, KAISU MARTINMÄKI<sup>1</sup>, JUKKA KAARTINEN<sup>2</sup>, MARJA-LIISA KINNUNEN<sup>1,2</sup> and HEIKKI RUSKO<sup>1</sup>

<sup>1</sup>Department of Biology of Physical Activity and <sup>2</sup>Department of Psychology, University of Jyväskylä, Jyväskylä, Finland

Accepted in revised form 30 May 2010; received 26 March 2010

**SUMMARY** Sleep is the most important period for recovery from daily load. Regular physical activity enhances overall sleep quality, but the effects of acute exercise on sleep are not well defined. In sleep hygiene recommendations, intensive exercising is not suggested within the last 3 h before bed time, but this recommendation has not been adequately tested experimentally. Therefore, the effects of vigorous late-night exercise on sleep were examined by measuring polysomnographic, actigraphic and subjective sleep quality, as well as cardiac autonomic activity. Eleven (seven men, four women) physically fit young adults ( $VO_{2max}$   $54 \pm 8$  mL·kg<sup>-1</sup>·min<sup>-1</sup>, age  $26 \pm 3$  years) were monitored in a sleep laboratory twice in a counterbalanced order: (1) after vigorous late-night exercise; and (2) after a control day without exercise. The incremental cycle ergometer exercise until voluntary exhaustion started at  $21:00 \pm 00:28$  hours, lasted for  $35 \pm 3$  min, and ended  $2:13 \pm 00:19$  hours before bed time. The proportion of non-rapid eye movement sleep was greater after the exercise day than the control day ( $P < 0.01$ ), while no differences were seen in actigraphic or subjective sleep quality. During the whole sleep, no differences were found in heart rate (HR) variability, whereas HR was higher after the exercise day than the control day ( $54 \pm 7$  versus  $51 \pm 7$ ,  $P < 0.01$ ), and especially during the first three sleeping hours. The results indicate that vigorous late-night exercise does not disturb sleep quality. However, it may have effects on cardiac autonomic control of heart during the first sleeping hours.

**KEYWORDS** actigraphy, heart rate variability, polysomnography, recovery, subjective sleep quality

## INTRODUCTION

The importance of adequate sleep for psychophysiological wellbeing is emerged when sleep is disturbed. Sleeping problems and sleep loss are increasingly common in modern societies, and may acutely cause several negative reversible effects such as daytime sleepiness, and in the long run more severe consequences associated with illnesses such as cardiovascular diseases, type-2 diabetes, obesity, clinical depression, and even all-cause mortality (e.g. Sigurdson and Ayas, 2007).

*Correspondence:* Tero Myllymäki, Department of Psychology, P.O Box 35, 40014, University of Jyväskylä, Finland. Tel.: +3581 4260 4409; fax: +3581 4260 2841; e-mail: tero.myllymaki@jyu.fi

Moreover, the economic burden of insomnia is high due to work absence and reduced productivity (Daley *et al.*, 2009).

Sleep quality is perceived as good when it is easy to fall into sleep and wake up, and when sleep is continuous and long enough (e.g. Åkerstedt *et al.*, 1997). Thus, both subjective and objective measures of sleep are needed to analyse the perception and the architecture of the sleep, respectively. Especially the proportion of slow-wave sleep (SWS), which is considered physiologically as the most restorative sleep stage, seems to be important for ease of awakening and recovery sleep after sleep loss (Åkerstedt *et al.*, 1997, 2009).

Regular physical activity has consistently been associated with better sleep in survey studies (Driver and Taylor, 2000; Youngstedt and Kline, 2006), thus offered as one tool for

better sleep in general sleep hygiene recommendations (e.g. American Academy of Sleep Medicine., 2001). Modest positive effects on sleep have also been reported after acute exercise (Youngstedt *et al.*, 1997). Especially light- and moderate-intensity exercise early in the evening has been perceived to have positive effects on sleep (Vuori *et al.*, 1988). The mechanisms behind the beneficial effects have been suggested to be related to the energy conservation, tissue restitution and temperature downregulation theories of sleep (Driver and Taylor, 2000; Dworak *et al.*, 2007). The mostly accepted hypothesis is that exercise-induced body heating may activate both temperature downregulation and sleep through the anterior hypothalamus-preoptic area in the brain (McGinty and Szymusiak, 1990). Modern neuroscientific theories suggest that brain energy metabolism and specific neurotransmitter systems may play a crucial role in homeostatic sleep regulation; e.g. high-intensity exercise increased sleep-promoting substance adenosine in rats (Dworak *et al.*, 2007). In addition, exercise may ease anxiety and stress (Petruzzello *et al.*, 1991).

However, vigorous late-night exercise is mentioned by the American Academy of Sleep Medicine. (2001) as one possible practice that may produce increased arousal and lead to inadequate sleep hygiene. This sustained physiological activation during sleep after a day including strenuous physical activity has been reported already by Hauri (1968), although no negative effects on sleep quality were observed. The previous studies concerning late-night exercise and sleep have mostly used actigraphic or subjective methods. These studies have indicated that exercising before bedtime may not necessarily disturb sleep (e.g. O'Connor *et al.*, 1998; Youngstedt *et al.*, 1999). A recent study using polysomnography showed that a 30-min high-intensity exercise 3–4 h before bed time increased SWS and decreased stage-2 sleep compared with baseline night in children, while no respective changes were seen after moderate-intensity exercise (Dworak *et al.*, 2008). Moreover, no changes were observed in mean heart rate (HR) during sleep between the three nights. In addition, a study with sedentary subjects showed that 1 h cycling at moderate intensity completed 2 h before bed time resulted in two-three times shorter sleep onset latency than exercising in the morning or early evening (Kobayashi *et al.*, 1999).

The clear cardiovascular changes that occur during exercise are mainly related to altered autonomic nervous system (ANS) function. During exercise, HR elevates due to withdrawal of vagal activity and increased sympathetic activity. After cessation of exercise, HR recovers towards preexercise values due to sympathetic withdrawal and vagal re-activation (Arai *et al.*, 1989). The changes in ANS activity can be assessed non-invasively by using HR variability (HRV) measurements (Task Force., 1996). Recovery of HR and HRV after exercise may take from minutes up to 24 h, depending on exercise intensity and training background (Furlan *et al.*, 1993; Seiler *et al.*, 2007). Recently, there has been a growing interest to use HR and HRV measurements, also during sleep, for evaluating exercise-induced disturbance of body homeostasis, training

status and recovery from daily load (e.g. Hautala *et al.*, 2009; Hynynen *et al.*, 2010).

The above literature review shows that exercising may lead to increased arousal, which may also have effects on sleep. However, the effects of vigorous late-night exercise on sleep quality and nocturnal cardiac autonomic activity have not been studied sufficiently. Therefore, the purpose of the present study was to test the assumption that late-night exercise disturbs sleep and nocturnal cardiac autonomic activity. Polysomnography, actigraphy, subjective assessments and cardiac autonomic activity-based methods were used to obtain a comprehensive view of the topic. To our knowledge, this was the first study utilizing all these methods at the same time to study the effects of late-night exercise on sleep.

## MATERIALS AND METHODS

### Subjects

Eleven young adults (seven men, four women, aged  $26 \pm 3$  years) participated in the study. Prior to the measurements, the study design was carefully explained to the subjects, and a written informed consent was obtained. The study followed the declaration of Helsinki, and was approved by the ethical committee of the University of Jyväskylä. All subjects were healthy, and did not use any regular or temporary medication during the measurements. They also had a normal sleeping profile based on the Basic Nordic Sleep Questionnaire (Partinen and Gislason, 1995).

### Study design and protocol

The subjects underwent two study days in a counterbalanced order separated by approximately 1 week. The subjects were monitored in a sleep laboratory: (1) after a day including late-night exercise; and (2) after a control day without exercise. During the study period, the subjects were asked to follow their normal sleep–wake cycle, and to use a similar schedule in both measurements. This was controlled by 24-h HR measurement (Suunto Memory Belt, Suunto Oy, Vantaa, Finland) and a diary of activities preceding the sleep laboratory measurements. These home measurements were also planned to minimize the ‘first night effect’, although a counterbalanced design was used.

During the exercise day, an incremental cycle ergometer (Monark Ergomedic 839E, Monark Exercise AB, Vansbro, Sweden) exercise that was designed to end 2 h before going to bed was performed, starting approximately 21:00 hours. The exercise lasted for  $35 \pm 3$  min, and consisted of a 5-min warm-up, 3-min stages of increasing power, and a 3–5 min cool-down. The starting power output was 25 W for females and 100 W for males, and the power increased 25 W after each stage. The subjects were encouraged to continue cycling until voluntary exhaustion or inability to maintain reasonable cycling pace ( $> 60$  rpm). During the exercise, HR was measured continuously with a HR monitor (Suunto t6, Suunto Oy, Vantaa, Finland), and gas exchange with a gas exchange

device (Sensor Medics Vmax 229, Sensor Medics, Yorba Linda, CA, USA). Rating of perceived exertion was asked after each stage accompanied with blood lactate measurements from fingertips. Blood lactate was analysed using BIOSEN C\_line (EKF-diagnostics GmbH, Barleben/Magdeburg, Germany) enzymatic-amperometric device. Maximal oxygen uptake ( $VO_{2max}$ ) was determined as the highest 60-s value during the exercise.

## Measurements

### *Polysomnography*

The subjects slept in a separate, dark and quiet room with similar temperature and humidity for each measurement. Before going to bed, 10 monopolar surface electrodes were attached to the subjects' skull and facial area for the measurement of electroencephalography (EEG), electrooculography (EOG) and electromyography (EMG). The EEG electrodes were placed in internationally determined locations C3, C4 and Fp1. Two electrodes were attached around the eyes for EOG and two on musculus digastric for EMG. The reference electrodes for EOG and C4 were left mastoid, and for C3 and Fp1 right mastoid. The skull electrodes were attached using SLE collodion adhesive. In the facial area, self-attached ABMU electrodes were used. In all electrodes ABRALYT 2000 electrode gel was used to provide adequate flow of electricity. The signal was collected using an amplifier (Brain Vision Quickamp, Brain Products GmbH, München, Germany), A/D-converter and recorder (Brain Vision Recorder Brain Products GmbH, München, Germany).

The data were filtered for 0.5–30 Hz using band-pass filter when analysed. The sleep stage scoring was done by using visual inspection and 30-s window (Rechtschaffen and Kales, 1968) by two researchers separately, and if there were differences in scoring between them, it was corrected together.

The analysed stages included the periods of wakefulness and body movements, stage-1, stage-2, SWS including stages 3 and 4, and rapid eye movement (REM) sleep. Moreover, sleep stages 1–4 constituted non-REM sleep, and total sleep time was the sum of all sleep stages. Sleep onset latency (SOL) was determined in two ways: (1) as time from bed time until the first period of stage 1 lasting longer than a minute (SOL–S1); and (2) from bed time until the first period of any other than stage 1 lasting longer than a minute (SOL–S2).

### *Actigraphy*

The movements of the non-dominant wrist were recorded with an Actiwatch activity-monitoring system<sup>®</sup> (Cambridge Neurotechnology, Cambridge, UK). Actiwatch measures activity by means of a piezo-electric accelerometer, and records the amount, intensity and duration of movement in all directions, and is stored in the memory as activity counts. The sampling frequency was 32 Hz and the epoch of activity counts was 15 s. All movement that is greater than 0.05 g was measured, and

values outside the range of 3–11 Hz were filtered to eliminate gravitational artefacts. Actiwatch Activity & Sleep Analysis 5 software (version 5.32) was used to analyse actigraphic sleep quality. Four actigraphic variables were used in the analyses: fragmentation index (an indicator of restlessness); actual sleep time (the amount of sleep); sleep efficiency (percentage of time spent asleep); and total activity score (the number of activity counts).

### *Subjective sleep quality*

Subjective sleep quality and physical tiredness were assessed using a questionnaire. Subjective sleep quality was assessed in the morning after awakening using a question 'How did you sleep last night?' with the scale 1–5 (1 = well, 5 = poorly). Tiredness was asked in the evening before going to bed and in the morning by a question 'how tired do you feel yourself physically right now?' with the scale 1–10 (1 = not tired at all, 10 = very tired).

### *Cardiac autonomic activity*

ECG R-peak-to-R-peak intervals (RRI) were recorded during sleep using an Alive Heart Monitor (Alive Technologies Pty, Australia) that detects the R-peaks of the electrocardiogram (ECG) with an accuracy of 3 ms.

The RRI data were analysed with Firstbeat HEALTH (version 2.2, Firstbeat Technologies, Jyväskylä, Finland) software application that includes an artefact detection filter, and uses a short-time Fourier Transform method and neural network modelling of the data in the analysis (Saalasti, 2003). Data with more than 5% of corrected RRIs were excluded from our analysis.

The software computes the traditional HRV variables. In this study the analysed variables included HR, root mean square of successive RRIs (RMSSD), high- and low-frequency power (HFP = 0.15–0.40 Hz; LFP = 0.04–0.15 Hz), and total power (TP = 0.04–0.40 Hz). These are generally accepted as indices of cardiac autonomic activity; RMSSD and HFP reflect vagal activity, while LFP reflects both vagal and sympathetic activity (Task Force., 1996).

The software also computes variables describing physiological states (e.g. stress, relaxation and exercise) based on both HR and HRV. The new variables used were relaxation time and percentage. When computing these variables, the software uses HR, HRV, respiration rate calculated from peak frequency of HFP, and neural network modelling (Saalasti, 2003). During relaxation state, vagal activity is dominating, HR is close to individual resting HR, and HRV is individually great and regular. Relaxation time is the duration of all relaxation states during sleep and relaxation percentage is the percentage of relaxation time from the total sleep time.

## Statistical analysis

Natural logarithm transformation was used with frequency domain variables of HRV. All data are expressed as

mean  $\pm$  SD. SPSS for Windows 15.0 software (SPSS, Chicago, USA) was used for statistical analysis. Statistical analyses were made for variables of the whole sleep with non-parametric Wilcoxon signed ranks test. When analysing nocturnal cardiac autonomic activity hour-by-hour after both days, ANOVA for repeated measures and Bonferroni-corrected paired sample *t*-tests were used. The level for statistical significance was set to  $P < 0.05$ .

## RESULTS

### Overview of exercise

The incremental cycle ergometer exercise started at 21:00  $\pm$  00:28 hours, lasted for 35  $\pm$  3 min, and ended 2:13  $\pm$  00:19 hours before bed time. The exercise intensity was light (50–63% of HR<sub>max</sub>) for 12  $\pm$  3 min, moderate (64–76% of HR<sub>max</sub>) for 9  $\pm$  3 min, vigorous (77–93% of HR<sub>max</sub>) for 12  $\pm$  2 min, and very hard (94–100% of HR<sub>max</sub>) for

2  $\pm$  2 min. The blood lactate level was 11.7  $\pm$  2.4 mmol·L<sup>-1</sup> immediately after the exercise. The subjects' VO<sub>2max</sub> during the exercise was 54  $\pm$  8 mL·kg<sup>-1</sup>·min<sup>-1</sup> (men: 57  $\pm$  7 mL·kg<sup>-1</sup>·min<sup>-1</sup>; women: 49  $\pm$  7 mL·kg<sup>-1</sup>·min<sup>-1</sup>).

### Polysomnographic, actigraphic and subjective sleep quality

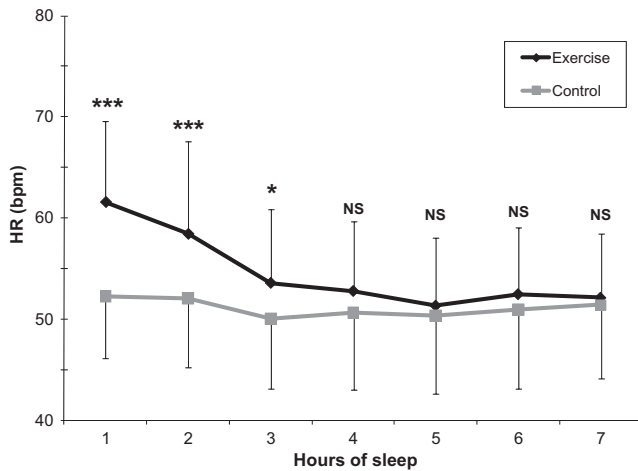
The overview of sleep after both days is expressed in Table 1. Bed and awakening times did not differ between the days. In polysomnographic variables, the only difference was the greater proportion of non-REM sleep after exercise day than control day (73.7  $\pm$  4.6% versus 69.3  $\pm$  4.1%,  $P < 0.01$ ). No differences were observed in actigraphic or subjective sleep quality between the days.

### Cardiac autonomic activity

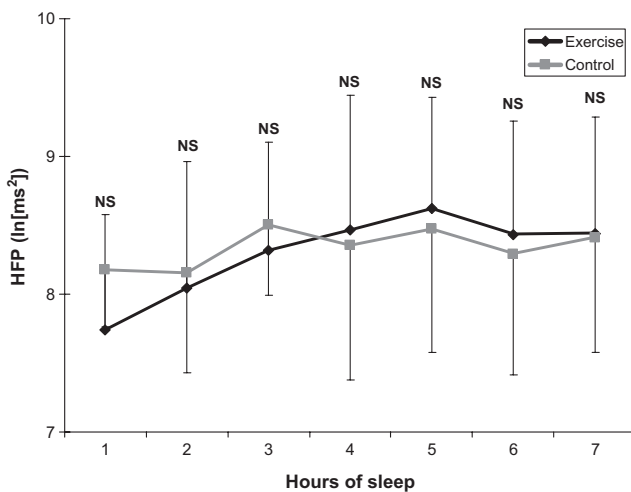
During the whole sleep, HR was higher after exercise day than control day (54  $\pm$  7 bpm versus 51  $\pm$  7 bpm,  $P < 0.01$ ).

**Table 1** Means, standard deviations and statistically significant differences of different variables between sleep after exercise day and control day. All variables are calculated for the whole night's sleep period. Statistical significance is expressed as  $P < 0.05^*$ ,  $P < 0.01^{**}$

	<i>Exercise day</i>	<i>Control day</i>	<i>Sign</i>
	<i>Mean <math>\pm</math> SD</i>	<i>Mean <math>\pm</math> SD</i>	
<b>Polysomnography</b>			
Time in bed (min)	480.6 $\pm$ 30.9	486.0 $\pm$ 43.8	0.248
Sleep onset latency-1 (min)	8.0 $\pm$ 5.7	14.2 $\pm$ 12.4	0.109
Sleep onset latency-2 (min)	18.7 $\pm$ 7.3	28.6 $\pm$ 19.0	0.068
Total sleep time (min)	441.5 $\pm$ 36.7	437.7 $\pm$ 45.1	0.722
Wake/movement (min)	39.2 $\pm$ 22.7	48.3 $\pm$ 30.5	0.248
Stage 1 sleep (min)	39.9 $\pm$ 26.8	47.7 $\pm$ 40.7	0.722
Stage 2 sleep (min)	211.8 $\pm$ 54.1	205.3 $\pm$ 42.9	0.594
Slow wave sleep (min)	92.9 $\pm$ 46.2	83.4 $\pm$ 41.8	0.155
Non-REM sleep (min)	344.6 $\pm$ 40.0	336.4 $\pm$ 34.5	0.79
REM sleep (min)	88.0 $\pm$ 24.9	101.3 $\pm$ 37.0	0.155
Total sleep time (%)	91.8 $\pm$ 4.6	90.1 $\pm$ 6.1	0.182
Wake/movement (%)	8.2 $\pm$ 4.6	9.9 $\pm$ 6.1	0.202
Stage 1 sleep (%)	8.3 $\pm$ 5.9	9.8 $\pm$ 8.2	0.79
Stage 2 sleep (%)	44.1 $\pm$ 10.6	42.2 $\pm$ 8.8	0.075
Slow wave sleep (%)	19.3 $\pm$ 9.8	17.2 $\pm$ 8.4	0.109
Non-REM sleep (%)	73.7 $\pm$ 4.6	69.3 $\pm$ 4.1	0.003**
REM sleep (%)	18.3 $\pm$ 4.4	20.8 $\pm$ 7.4	0.075
<b>Actigraphy</b>			
Fragmentation index	21.6 $\pm$ 10.8	27.7 $\pm$ 11.1	0.285
Actual sleep time (min)	442 $\pm$ 34	445 $\pm$ 42	0.386
Sleep efficiency (%)	93.2 $\pm$ 3.5	92.7 $\pm$ 3.5	0.575
Total activity score	5718 $\pm$ 3586	5366 $\pm$ 2794	0.721
<b>Subjective sleep quality</b>			
Sleep quality (1–5)	3.2 $\pm$ 1.2	3.0 $\pm$ 1.2	0.637
Tiredness at bed time (1–10)	5.7 $\pm$ 2.0	4.9 $\pm$ 1.9	0.201
Tiredness after awakening (1–10)	3.7 $\pm$ 2.1	4.6 $\pm$ 1.9	0.108
<b>Cardiac autonomic activity</b>			
Heart rate (bpm)	54.5 $\pm$ 6.8	51.4 $\pm$ 6.9	0.003**
High-frequency power (ln [ms <sup>2</sup> ])	8.4 $\pm$ 0.8	8.4 $\pm$ 0.7	0.534
Low-frequency power (ln [ms <sup>2</sup> ])	8.8 $\pm$ 0.6	8.8 $\pm$ 0.7	0.859
Total power (ln [ms <sup>2</sup> ])	9.4 $\pm$ 0.6	9.3 $\pm$ 0.7	0.594
RMSSD (ms)	83.9 $\pm$ 33.1	85.1 $\pm$ 31.0	0.859
Relaxation time (min)	341.3 $\pm$ 85.6	411.4 $\pm$ 66.1	0.010*
Relaxation time (%)	71.0 $\pm$ 17.6	84.7 $\pm$ 13.8	0.004**



**Figure 1.** Heart rate (HR) hour-by-hour during sleep after exercise and control days. Asterisks are used to express differences between sleeping hours of the days (\*\* $P < 0.001$ , \* $P < 0.05$ ).



**Figure 2.** High-frequency power (HFP) hour-by-hour during sleep after exercise and control days.

Nocturnal HRV (RMSSD, HFP, LFP, TP) did not differ between the days. Relaxation time ( $341 \pm 86$  min versus  $411 \pm 66$  min,  $P < 0.05$ ) and percentage ( $71 \pm 18\%$  versus  $85 \pm 14\%$ ,  $P < 0.01$ ) were lower after exercise day than control day.

Using hour-by-hour comparisons, an interaction between the day (exercise versus control) and the sleeping hours ( $P < 0.001$ ) was found in HR. HR was higher during the first three sleeping hours after the exercise day than after control day ( $P < 0.001$ – $0.05$ ). No interaction between the day (exercise versus control) and sleeping hours was found when analysing HFP and LFP hour-by-hour, but there was a main effect in sleeping hours ( $P < 0.01$ ). HR and HFP hour-by-hour are illustrated in Figs 1 and 2.

## DISCUSSION

The present study evaluated the effects of vigorous late-night exercise on four different aspects of sleep (polysomnography,

actigraphy, subjective sleep quality and cardiac autonomic activity). The main finding was that late-night exercise did not disturb polysomnographic, actigraphic or subjective sleep quality. This is in contrast to the general sleep hygiene recommendations (e.g. American Academy of Sleep Medicine., 2001). However, exercise resulted in elevated HR during the first three sleeping hours compared with the control day, but it did not affect HRV during sleep. The finding of increased HR was expected, but similar HRV during sleep after both days was somewhat surprising.

### Polysomnographic, actigraphic and subjective sleep quality

Polysomnographic analysis revealed that the proportion of non-REM sleep was greater after the exercise day than control day. This seemed to be due to a non-significantly smaller proportion of wakefulness, and a greater proportion of stage 2 and SWS after the exercise day (Table 1). SWS has been suggested to represent the daily process of restitution, and it is associated with increased growth hormone and decreased cortisol secretion (Åkerstedt and Nilsson, 2003). In most studies dealing with exercise and sleep, the exercise has been performed at least 4 h before bed time. Those studies have reported increased SWS after both high- and moderate-intensity exercises compared with rest day, but also no effect has been observed (Driver and Taylor, 2000; Youngstedt *et al.*, 1997, 2000). Recently, a 30-min high-intensity exercise 3–4 h before bed time increased SWS and decreased stage 2 sleep in children, while no effects were seen after moderate-intensity exercise (Dworak *et al.*, 2008). Thus, the present results seem to be consistent with earlier scientific findings, although the present exercise was performed 2–2.5 h before bed time.

Some indications of shortened SOL after exercise day were observed (Table 1), and therefore the alerting influences of vigorous exercise may not have hindered falling into sleep. Earlier studies have suggested that when moderate-intensity exercise is performed close to bed time, increased body temperature may be more important in providing sleep than the sleep-disturbing influences on the body that physical activity may cause (e.g. O'Connor *et al.*, 1998). Furthermore, an increased SOL has been found when exercising less than 4 h before bed time (Youngstedt *et al.*, 1997), but also no effect (actigraphic measure; Youngstedt *et al.*, 1999) and decreased SOL in children after high-intensity exercise (Dworak *et al.*, 2008) have been reported. Moreover, shortened SOL has been reported after moderate late-night exercise when compared with exercise in the morning and early evening in sedentary subjects (Kobayashi *et al.*, 1999).

In the present study, any decrements in actigraphic or subjective sleep quality after late-night exercise were not found. Previously, a 3-h cycling exercise at 65–75% of HR reserve ending 30 min before bed time did not disturb sleep when measured by actigraphy and subjective estimates (Youngstedt *et al.*, 1999). Therefore, our results from vigorous exercise are parallel with the previous findings with actigraphy.

Earlier, 1-h moderate-intensity exercise shortly before bed time increased nocturnal core body temperature, but did not disturb subjective sleep quality (O'Connor *et al.*, 1998). Moreover, a moderate-intensity exercise performed early in the evening did not affect subjective sleep quality (Youngstedt *et al.*, 2000). Positive effects of late-night exercise on subjective sleep and reduced sleepiness during the subsequent daytime have been reported by Yoshida *et al.* (1998). Thus, our results extend the previous findings that even vigorous-intensity exercise close to bed time does not disturb subjective sleep quality.

### Cardiac autonomic activity

Measuring cardiac autonomic activity by HR- and HRV-based analysis methods have gained remarkable interest lately in the assessment of ANS activity and recovery from daily load due to their non-invasive nature. In this study, a late-night exercise caused higher HR during sleep after exercise day than control day due to elevated HR during the first three sleeping hours. Previous studies concerning daytime exercises have reported elevated HR during sleep after a strenuous exercise day (Hauri, 1968), maximal exercise test (Bunnell *et al.*, 1983) and prolonged exercise (Mischler *et al.*, 2003). However, HR remained unchanged after daytime 30 min cycling exercise at 75% of  $VO_{2max}$  (O'Connor *et al.*, 1993). Similarly to Hauri's study (1968), physiological arousal decreased towards the base values after exercise in the present study, and the most remarkable effects were seen during initial sleep, although no differences were observed in sleep quality between the days.

Unfortunately, information of nocturnal HRV after an acute exercise is very limited, as previous studies have mostly examined HRV within a short time frame after exercise. Those studies have shown that HRV decreases during exercise, and recovery of HRV is dependent on type, intensity and duration of exercise (e.g. Kaikkonen *et al.*, 2008, 2010). High-intensity exercises have reduced HRV for 30–60 min after exercise in trained and highly trained subjects (James *et al.*, 2002; Kaikkonen *et al.*, 2008; Seiler *et al.*, 2007), while HR has seemed to stay elevated for a longer time (Seiler *et al.*, 2007). Surprisingly, no differences in nocturnal HRV between the days were found despite differences in HR in the present study. Other recent findings of nocturnal HRV after exercise indicate that both moderate and heavy endurance exercise at daytime affect both HR and HRV during sleep with a dose–response manner (Hynynen *et al.*, 2010).

Both circadian system and sleep *per se* affect vagal control of HR so that the circadian system downregulates ANS activity as the sleep onset approaches by increasing vagal activity rather than decreasing sympathetic activity (Burgess *et al.*, 1997). One possible explanation for the results is the fact that HR is under both vagal and sympathetic control, but variables of HRV reflect mainly vagal activity as indicated by blockade studies (e.g. Martinmäki *et al.*, 2006). Thus, the elevated HR during early sleep after exercise may be a result of a

non-significant decrease in vagal and increase in sympathetic activity of the ANS. Unchanged HRV may also be related to saturation of HRV at low HR levels (Goldberger *et al.*, 2001). When HR is low, a probable decrease in vagal activity after exercise may not be reflected in changes in HRV between the days. Still there were some indications of lower HRV, suggesting lower vagal activity during the early phase of sleep on both days (Fig. 2).

Other possible explanations for the results are the various physiological changes that occur due to exercise (e.g. increase of thermogenic hormones, elevated body temperature, increased ventilation and blood flow, and replenishment of energy stores), which all may contribute to the excess post-exercise oxygen consumption that is dependent on intensity and duration of exercise (Borsheim and Bahr, 2003). Exercise shortly before bed time has been shown to increase nocturnal body temperature (O'Connor *et al.*, 1998), which may explain increased HR during the first sleeping hours independent of autonomic control. Therefore, the present observations may be mainly related to increased whole body metabolism or temperature after exercise.

The calculation of relaxation variables is based on individual HR and HRV. The significant increase in HR and non-significant decrease in HRV during the first sleeping hours (see both figures) most probably explain the smaller relaxation time and percentage after the exercise day. Thus, HR may have greater impact than HRV on the calculation of these variables. However, also the individually high vagal activation may be important for enhanced recovery, so both can be taken into account. HR and HRV information *per se* have no temporal aspect – rather they express the level of cardiac autonomic activity. It takes time for cardiac autonomic activity to recover after exercise, pointing out the importance of time for adequate recovery from daily load. Also in the allostatic models of stress and recovery, time is considered as one of the key aspects in recovery of physiological systems after stress response (McEwen, 1998). Chronically elevated HR and blood pressure produce wear and tear on the cardiovascular system that can result in cardiovascular disorders (McEwen, 1998). When summarized, these results may indicate delayed recovery of the physiological systems and suggest a need for a longer time for adequate recovery during sleep after an exercise.

### Limitations

This study has some limitations to be taken into account. Body temperature, hormones or other cellular compounds such as adenosine were not measured. Therefore, the mechanisms concerning sleep quality after exercise can only be speculated. Moreover, the laboratory environment surely differs from sleeping at home; the subjects were young and fit without any sleep disorders, so-called 'good-sleepers'; and the number of subjects was relatively small, which all may limit the generalizability of the results. In addition, these physically fit subjects may have been less prone to sleep-disturbing effects of late-night exercise than less active people.

## Conclusions and implications

This study showed that vigorous late-night exercise does not disturb polysomnographic, actigraphic or subjective sleep quality, although it has effects on HR, especially during the first sleeping hours. The present methods provide various information about sleep and recovery. Sleep EEG seems to indicate brain functioning rather than body restoration (Dworak *et al.*, 2007, 2008); actigraphs give movement-related information of sleep (e.g. Ancoli-Israel, 2003); subjective sleep quality expresses perception of sleep (Åkerstedt *et al.*, 1997); and HR and HRV reflect cardiac autonomic activity (Task Force, 1996). It seems that recovery during sleep is a complicated phenomenon where various physiological and psychological aspects should be acknowledged – and probably also measured. In practice, performing this kind of exercise prior to bed time should not have negative effects on sleep in fit young individuals. In conclusion, vigorous late-night exercise procedure did not disturb sleep quality in young healthy adults, but it elevated HR and probably shortened the time for enhanced recovery during sleep.

## ACKNOWLEDGEMENTS

This work was supported by Tekes and companies: Nokia, Suunto, Mawell, Firstbeat Technologies, Vivago, Varma, Mehiläinen, Solaris, and JTO School of Management.

## REFERENCES

- Åkerstedt, T. and Nilsson, P. M. Sleep as restitution: an introduction. *J. Int. Med.*, 2003, 254: 6–12.
- Åkerstedt, T., Hume, K., Minors, D. and Waterhouse, J. Good sleep – its timing and physiological sleep characteristics. *J. Sleep Res.*, 1997, 6: 221–229.
- Åkerstedt, T., Kecklund, G., Ingre, M., Lekander, M. and Axelsson, J. Sleep homeostasis during repeated sleep restriction and recovery: support from EEG dynamics. *Sleep*, 2009, 32: 217–222.
- American Academy of Sleep Medicine. *International Classification of Sleep Disorders, Revised: Diagnostic and Coding Manual*. American Academy of Sleep Medicine, Chicago, 2001.
- Ancoli-Israel, S. The role of actigraphy in the study of sleep and circadian rhythms. *Sleep*, 2003, 26: 342–392.
- Arai, Y., Saul, J. P., Albrecht, P. *et al.* Modulation of cardiac autonomic activity during and immediately after exercise. *Am. J. Physiol. Heart Circ. Physiol.*, 1989, 256: H132–H141.
- Borsheim, E. and Bahr, R. Effects of exercise intensity, duration and mode on post-exercise oxygen consumption. *Review. Sports Med.*, 2003, 33: 1037–1060.
- Bunnell, D. E., Bevier, W. C. and Horvath, S. M. Nocturnal sleep, cardiovascular function, and adrenal activity following maximum-capacity exercise. *Electroencephalogr. Clin. Neurophysiol.*, 1983, 56: 186–189.
- Burgess, H. J., Trinder, J., Kim, Y. and Luke, D. Sleep and circadian influences on cardiac autonomic nervous system activity. *Am. J. Physiol. Heart Circ. Physiol.*, 1997, 273: H1761–H1768.
- Daley, M., Morin, C. M., LeBlanc, M., Gregoire, J. P. and Savard, J. The economic burden of insomnia: direct and indirect costs for individuals with insomnia syndrome, insomnia symptoms, and good sleepers. *Sleep*, 2009, 32: 55–64.
- Driver, H. S. and Taylor, S. R. Exercise and sleep. *Sleep Med. Rev.*, 2000, 4: 387–402.
- Dworak, M., Diel, P., Voss, S., Hollmann, W. and Struder, H. K. Intense exercise increases adenosine concentrations in rat brain: implications for a homeostatic sleep drive. *Neuroscience*, 2007, 150: 789–795.
- Dworak, M., Wiater, A., Alfer, D., Stephan, E., Hollmann, W. and Struder, H. K. Increased slow wave sleep and reduced stage 2 sleep in children depending on exercise intensity. *Sleep Med.*, 2008, 9: 266–272.
- Furlan, R., Piazza, S., Dell’Orto, S. *et al.* Early and late effects of exercise and athletic training on neural mechanisms controlling heart rate. *Cardiovasc. Res.*, 1993, 27: 482–488.
- Goldberger, J. J., Challapalli, S., Tung, R., Parker, M. A. and Kadish, A. H. Relationship of heart rate variability to parasympathetic effect. *Circulation*, 2001, 103: 1977–1983.
- Hauri, P. Effects of evening activity on early night sleep. *Psychophysiology*, 1968, 4: 267–277.
- Hautala, A. J., Kiviniemi, A. M. and Tulppo, M. P. Individual responses to aerobic exercise: the role of the autonomic nervous system. *Neurosci. Biobehav. Rev.*, 2009, 33: 107–115.
- Hynynen, E., Vesterinen, V., Nummela, A. and Rusko, H. Effects of moderate and heavy endurance exercise on nocturnal HRV. *Int. J. Sports Med.*, 2010, 31: 428–432.
- James, D. V. B., Barnes, A. J., Lopes, P. and Wood, D. M. Heart rate variability: response following a single bout of interval training. *Int. J. Sports Med.*, 2002, 23: 247–251.
- Kaikkonen, P., Rusko, H. and Martinmäki, K. Post-exercise heart rate variability of endurance athletes after different high-intensity exercise interventions. *Scand. J. Med. Sci. Sports*, 2008, 18: 511–519.
- Kaikkonen, P., Hynynen, E., Mann, T., Rusko, H. and Nummela, A. Can HRV be used to evaluate training load in constant load exercises? *Eur. J. Appl. Physiol.*, 2010, 108: 435–442.
- Kobayashi, T., Yoshida, H., Ishikawa, T. and Arakawa, K. Effects of the late evening exercise on sleep onset process. *Sleep Res. Online*, 1999, 2(suppl. 1): 233.
- Martinmäki, K., Rusko, H., Kooistra, L., Kettunen, J. and Saalasti, S. Intraindividual validation of heart rate variability indexes to measure vagal effects on hearts. *Am. J. Physiol. Heart Circ. Physiol.*, 2006, 290: H640–H647.
- McEwen, B. S. Protective and damaging effects of stress mediators. *N. Engl. J. Med.*, 1998, 338: 171–179.
- McGinty, D. and Szymusiak, R. Keeping cool: a hypothesis about the mechanisms and functions of slow-wave sleep. *Trends Neurosci.*, 1990, 13: 480–487.
- Mischler, I., Vermorel, M., Montaurier, C. *et al.* Prolonged daytime exercise repeated over 4 days increases sleeping heart rate and metabolic rate. *Can. J. Appl. Physiol.*, 2003, 28: 616–629.
- O’Connor, P. J., Crowley, M. A., Gardner, A. W. and Skinner, J. S. Influence of training on sleeping heart rate following daytime exercise. *Eur. J. Appl. Physiol. Occup. Physiol.*, 1993, 67: 39–42.
- O’Connor, P. J., Breus, M. J. and Youngstedt, S. D. Exercise-induced increase in core temperature does not disrupt a behavioral measure of sleep. *Physiol. Behav.*, 1998, 64: 213–217.
- Partinen, M. and Gislason, T. Basic Nordic Sleep Questionnaire (BNSQ): a quantitated measure of subjective sleep complaints. *J. Sleep Res.*, 1995, 4: 150–155.
- Petruzzello, S. J., Landers, D. M., Hatfield, B. D., Kubitz, K. A. and Salazar, W. A meta-analysis on the anxiety-reducing effects of acute and chronic exercise: outcomes and mechanisms. *Sports Med.*, 1991, 11: 143–182.
- Rechtschaffen, A. and Kales, A. (Eds) *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subject*. Public Health Service, US Government Printing Office, Washington DC, 1968: 1–13.

- Saalasti, S. Neural networks for heart rate time series analysis. Academic dissertation. Jyväskylä Studies in Computing 33, University of Jyväskylä, Jyväskylä, 2003.
- Seiler, S., Haugen, O. and Kuffel, E. Autonomic recovery after exercise in trained athletes: intensity and duration effects. *Med. Sci. Sports Exerc.*, 2007, 39: 1366–1373.
- Sigurdson, K. and Ayas, N. T. The public health and safety consequences of sleep disorders. *Can. J. Physiol. Pharmacol.*, 2007, 85: 179–183.
- Task Force. Heart rate variability: standards of measurement, physiological interpretation and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *Circulation*, 1996, 93: 1043–1065.
- Vuori, I., Urponen, H., Hasan, J. and Partinen, M. Epidemiology of exercise effects on sleep. *Acta Physiol. Scand. Suppl.*, 1988, 574: 3–7.
- Yoshida, H., Ishikawa, T., Shiraiishi, F. and Kobayashi, T. Effects of the timing of exercise on the night sleep. *Psychiatry Clin. Neurosci.*, 1998, 52: 139–140.
- Youngstedt, S. D. and Kline, C. E. Epidemiology of exercise and sleep. *Sleep Biol. Rhythms*, 2006, 4: 215–221.
- Youngstedt, S. D., O'Connor, P. J. and Dishman, R. K. The effects of acute exercise on sleep: a quantitative synthesis. *Sleep*, 1997, 20: 203–214.
- Youngstedt, S. D., Kripke, D. F. and Elliott, J. A. Is sleep disturbed by vigorous late-night exercise? *Med. Sci. Sports Exerc.*, 1999, 31: 864–869.
- Youngstedt, S. D., O'Connor, P. J., Crabbe, J. B. and Dishman, R. K. The influence of acute exercise on sleep following high caffeine intake. *Physiol. Behav.*, 2000, 68: 563–570.