Eur J Appl Physiol (2012) 112:801–809
DOI 10.1007/s00421-011-2034-9

ORIGINAL ARTICLE

Effects of exercise intensity and duration on nocturnal heart rate variability and sleep quality

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Received: 15 February 2011 / Accepted: 29 May 2011 / Published online: 12 June 2011

Abstract Acute physical exercise may affect cardiac autonomic modulation hours or even days during the recovery phase. Although sleep is an essential recovery period, the information on nocturnal autonomic modulation indicated by heart rate variability (HRV) after different exercises is mostly lacking. Therefore, this study investigated the effects of exercise intensity and duration on nocturnal HR, HRV, HR, and HRV-based relaxation, as well as on actigraphic and subjective sleep quality. Fourteen healthy male subjects (age 36 ± 4 years, maximal oxygen uptake 49 ± 4 ml/kg/min) performed five different running exercises on separate occasions starting at 6 p.m. with HR guidance at home. The effect of intensity was studied with 30 min of exercises at intensities corresponding to HR level at 45% (easy), 60% (moderate) and 75% (vigorous) of their maximal oxygen uptake. The effect of duration was studied with 30, 60, and 90 min of moderate exercises. Increased exercise intensity elevated nocturnal HR compared to control day ($p < 0.001$), but it did not affect nocturnal HRV. Nocturnal HR was greater after the day with 90- than 30- or 60-min exercises ($p < 0.01$) or control day ($p < 0.001$). Nocturnal HRV was lower after the 90-min exercise day compared to control day ($p < 0.01$). Neither exercise intensity nor duration had any impact on actigraphic or subjective sleep quality. The results suggest that increased exercise intensity and/or duration cause delayed recovery of nocturnal cardiac autonomic modulation, although long exercise duration was needed to induce changes in nocturnal HRV. Increased exercise intensity or duration does not seem to disrupt sleep quality.

Keywords Exercise · Recovery · Heart rate variability · Actigraphy · Subjective sleep quality

Introduction

Heart rate variability (HRV) which is a non-invasive method to study autonomic nervous system (ANS) activity has increasingly been used to examine training load, disturbance of body's homeostasis, and recovery state after training (e.g., Pichot et al. 2000; Buchheit et al. 2004; Hynynen et al. 2010; James 2002; Kaikkonen et al. 2007, 2008; Martinmäki and Rusko 2008; Seiler et al. 2007). As far as exercise training is concerned, it is associated with better aerobic fitness, overall health (e.g., Haskell et al. 2007), and enhanced ANS function reflected by increased vagal modulation of the heart indicated by greater HRV (e.g., Sandercock et al. 2005; Hautala et al. 2009; Thayer et al. 2010). However, recovery of cardiac autonomic activity during sleep after different acute exercise sessions has not been well investigated despite that sleep is a crucial recovery period. Instead, the studies have mostly focused on the acute effects within a narrow time frame after exercise. These studies have reported that HRV diminishes during exercise, and acute recovery of HRV seems to be associated with type, intensity, and duration of exercise (Kaikkonen et al. 2007, 2008; Martinmäki and Rusko 2008; James

Communicated by Keith Phillip George.

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2002) as well as training background (Seiler et al. 2007). In addition, it has been found that full recovery of autonomic activity may take several hours or even days after an exercise (Furlan et al. 1993; Mourot et al. 2004; Seiler et al. 2007).

According to general sleep hygiene recommendations, exercising is suggested for adequate sleep, but not late in the evening due to possible arousing effects (e.g., American Academy of Sleep Medicine 2001). These arousing effects may cause disturbed sleep, which has been associated with similar physiological reactions that are found during the stress response, e.g., increased cardiovascular activation or cortisol secretion (Åkerstedt 2006). Previous studies on autonomic modulation during sleep after exercise have found increased nocturnal HR and decreased HRV after moderate intensity exercise or marathon run at daytime (Hynynen et al. 2010). However, also negligible effects on nocturnal HRV after extremely long cross-country skiing race (Hautala et al. 2001) or vigorous late-night exercise have been reported (Myllymäki et al. 2011).

Therefore, the purpose of the present study was to examine experimentally the effects of exercise intensity and duration on nocturnal cardiac autonomic activity, actigraphic sleep quality, and subjective sleep quality. Based on the previous literature, it was hypothesized that increased exercise intensity and duration would result in greater disturbance to nocturnal autonomic modulation and sleep variables. To our knowledge, this is the first study exploring systematically the effects of different exercise protocols on nocturnal autonomic modulation and sleep.

Methods

Subjects

Fourteen healthy moderately physically active male subjects aged 35.9 ± 4.3 years (mean ± SD) and with body mass index 24.2 ± 1.7 kg/m^2 participated in the study. Eleven subjects reported that they performed vigorous intensity physical activity at least once a week (on average three times a week), and three subjects performed moderate intensity physical activity two to three times a week. In popularity order, their activities were cycling, walking, strength training, and running. The physical demands of their occupations were easy. The study design was carefully explained to the subjects, and a written informed consent was obtained before the measurements. The study design followed the declaration of Helsinki and was approved by the local ethical committee. The subjects did not use any medication during the measurements and had a normal sleeping profile based on the Basic Nordic Sleep Questionnaire (Partinen and Gislason 1995).

Study design and measurements

Before the experimental exercises, a maximal dynamic exercise test was performed in a laboratory to measure the subjects' maximal oxygen uptake (VO_{2max}) and HR for setting submaximal exercise levels to be utilized during the home exercises. Thereafter, a control sleep was measured after a day without exercise. The 5 days with different exercise sessions at home were separated by at least 48 h from each other, and sleep variables were measured after each exercise session. The subjects were allowed to decide the order of exercises freely. The subjects were not allowed to do any other exercises or drink alcohol within 24 h before any measurement. All exercise sessions started at 6 p.m. and were followed by the measurements during the succeeding night's sleep.

Maximal exercise test

A maximal dynamic exercise test was performed on a treadmill. During the test, the subjects' HR was measured continuously with an HR monitor (Suunto t6, Suunto Oy, Vantaa, Finland) and gas exchange with a gas analyzer (Sensor Medics V_{max} 229, Sensor Medics Corp., Yorba Linda, California, USA). The test started at the speed of 6 km/h, each stage lasted for 3 min including a brief stop for blood lactate (BLa) measurement from fingertips, and the speed was increased 1 km/h after each stage. At the end of each stage, rating of perceived exertion (RPE) using the Borg's scale (6–20) was obtained. The subjects were encouraged to continue running until voluntary exhaustion or inability to maintain the required speed. BLa was later analyzed using BIOSEN C_line enzymatic-amperometric device (EKF-diagnostics GmbH, Barleben/Magdeburg, Germany). Maximal oxygen uptake (VO_{2max}) was determined as the highest 60-s value reached during the test.

Exercise sessions

The five running exercise sessions at home followed the updated recommendations for physical activity of the adults (Haskell et al. 2007). The effect of intensity was studied using 30 min of constant steady-state exercises at HR level corresponding individually to 45% (easy), 60% (moderate) or 75% (vigorous) of VO_{2max}. The effect of duration was studied using 30, 60, and 90 min of moderate intensity exercise sessions. The subjects were instructed to perform each exercise session using individual HR range (e.g., 140–150 bpm) to achieve the planned training effect, and thus an HR monitor was used to control the subjects' HR level when exercising. In addition, the subjects were asked to evaluate RPE immediately after finishing each exercise using a questionnaire.

Sleep measurements

The subjects were instructed to measure ECG R-peak to R-peak intervals (RRI) and actigraphic sleep quality during sleep after each exercise day. RRI data was measured using Alive Heart Monitor (Alive Technologies Pty Ltd, Australia) that detects the R-peaks of ECG with an accuracy of 3 ms. The movements of the non-dominant wrist were recorded with an Actiwatch activity monitoring system® (Cambridge Neurotechnology Ltd, Cambridge, UK), which measures activity by means of a piezo-electric accelerometer. Actiwatch records the amount, intensity, and duration of movement in all directions, and the data are stored in the memory as activity counts. The sampling frequency was 32 Hz and the sampling epoch of activity counts was set to 0.25 min (15 s). All movements greater than 0.05*g* were measured, and values outside the range of 3–11 Hz were filtered to eliminate gravitational artefacts. The subjects were also instructed to evaluate their subjective sleep quality in the morning after awakening using a question "How did you sleep last night?" using 100 mm visual analog acale (0–100) with verbal instructions extremely badly (0) and extremely well (100).

Data analysis

Cardiac autonomic activity

The RRI data were analyzed with Firstbeat HEALTH (version 3.0.1.0, Firstbeat Technologies Ltd, Jyväskylä, Finland) computer software application. The software includes an automatic artifact detection filter and a short-time Fourier transform method and neural network modeling of the data in the analysis (Saalasti 2003). If a measurement included more than 5% of erroneous RRIs, the data were excluded from our analysis. Therefore, 9 out of 84 measurements were excluded from the analysis.

The software computes traditional HRV variables. In addition to HR, the analyzed variables in the present study included root mean square of differences of successive RRIs (RMSSD), high frequency power (HFP, 0.15–0.40 Hz), low frequency power (LFP 0.04–0.15 Hz), and total power (TP, 0.04–0.40 Hz), which are generally accepted as indices of cardiac autonomic activity. Although the heart has its own intrinsic rhythm and it is also affected by hormonal factors and reflexes, the regulation of HR is mostly achieved by the balance between the sympathetic and parasympathetic branch of ANS, the latter dominating usually at rest (e.g., Carter et al. 2003). All HRV, particularly RMSSD and HFP, seems to be mostly under parasympathetic control (Taylor et al. 1998; Martinmäki et al. 2006).

The software computes also variables describing physiological states (e.g., stress, relaxation, and exercise) using neural network modeling based on the information on HR, HRV, and respiration rate calculated from the peak frequency of HFP (Saalasti 2003). The new variable included in the present study was relaxation percentage i.e., the duration of relaxation state during sleep. During the relaxation state, vagal activity dominates, HR is close to individual resting HR (not greater than 10–20% from HR reserve) and HRV is individually great (i.e., close to individual resting level) and regular. As neural network modeling is used by the software, no exact formula for calculating these variables from HR and HRV can be given. However, the present methods have also previously been utilized to study the recovery of ANS function during sleep after exercise (Myllymäki et al. 2011).

Variables of cardiac autonomic activity were analyzed for the 4-h period of sleep starting 30 min after reported bedtime. The 4-h period was used to have a constant time period for each night and subject, and to minimize the effects of different time required to fall asleep. Moreover, especially the early phase of sleep provides an optimal measurement condition for HRV as it is a quiet sleep period containing mostly slow wave sleep, and it is thus minimally affected by external factors or body movements (Brandenberger et al. 2005). The 4-h period has also been used in the previous studies concerning nocturnal cardiac autonomic activity (Pichot et al. 2000; Hautala et al. 2001; Hynynen et al. 2010; Myllymäki et al. 2011).

Sleep quality

Actigraphic sleep quality was analyzed with Actiwatch Activity and Sleep Analysis 5 software (version 5.32, Cambridge Neurotechnology Ltd, Cambridge, UK). 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). Actigraphy has been shown to be a reliable method to objectively measure sleep quality, i.e., amount of sleep and movements during sleep in normal sleepers and its results are highly correlated with polysomnography (Ancoli-Israel 2003). However, also subjective measures are needed to obtain information on perceived sleep quality and sleep satisfaction (Åkerstedt et al. 1994; Kushida et al. 2001). The subjective sleep quality was analyzed from the subjects' responses in a visual analog scale. Visual analog scales are commonly used in sleep studies (e.g., Zisapel and Nir 2003), as they are sensitive to treatment effects and allow usage of parametric testing. In addition, it was possible to analyze sleep quality for a specific night instead of

more often assessed general sleep quality over a longer time period.

Statistical analysis

The R software for statistical computing (version 2.12.2) was used for statistical analysis. To assess the effects of exercise intensity, variables after the days with different intensity exercises and after a control day were compared. To investigate the effects of exercise duration, variables after the days of moderate exercises with different duration, and after control day without exercise were used. The number of subjects varied between 11 and 14 in different exercise sessions due to missing data after strict excluding criteria. Due to missing values, the data were analyzed using mixed models' variance component model with the subject as the random factor. The analysis method takes into account the dependence of the subject's measurements and can utilize data maximally even if some missing values exist. If significant main effect was found, pairwise comparisons were made using Tukey's HSD method with adjusted p values. Data transformation methods were used when needed to get the data normally distributed. In the text, tables and figures, all data are expressed as mean ± SD. The level for statistical significance was set to $p < 0.05$.

Results

Load of different exercise sessions

The subjects' VO_{2max} during the maximal exercise test performed before the home exercises was 49.4 ± 3.8 ml/kg/min. Significant main effect for HR and RPE ($p < 0.001$ for both) was found between different intensity exercises, and both variables ($p < 0.001$) differed between each 30 min of home exercises with varying intensity (see Table 1). There was a significant main effect for RPE ($p < 0.001$), but not for HR after exercises with different duration. HR was at the same level during each moderate intensity exercises with varying duration (see Table 2). RPE was greater in the context of 90-min exercise than 30- or 60-min exercises ($p < 0.001$ for both), but there was no difference in RPE between the 30- and 60-min exercises. The 30-min easy exercise was perceived as easy, both 30- and 60-min moderate exercises as somewhat difficult, 90-min moderate exercise as difficult, and vigorous intensity 30-min exercise as very difficult. Thus, HR level during different exercises showed that an appropriate exercise load was induced, which was confirmed also by the subjects' perceived exertion.

Table 1 Summary of exercise and sleep variables during the days with different intensity exercises and during a control day

	30 min easy	Moderate	Vigorous	Control day
Exercise variables				
HR (bpm) during exercise	113 ± 9	139 ± 7$^{\#\#\#, +++}$	159 ± 8$^{\#\#\#, ¤¤¤}$	–
RPE at the end of exercise	10 ± 2	13 ± 2$^{\#\#\#, +++}$	17 ± 1***, $^{¤¤¤}$	–
Sleep variables				
Bedtime	23:13 ± 0:54	23:24 ± 1:02	23:28 ± 0:59	23:24 ± 0:57
Awakening time	6:50 ± 0:59	6:49 ± 1:09	7:04 ± 1:18	6:59 ± 1:09
Time in bed	7:38 ± 0:51	7:26 ± 1:04	7:35 ± 1:05	7:38 ± 0:46
Actigraphic sleep quality				
Actual sleep time (h:min)	7:04 ± 0:50	6:52 ± 1:02	7:06 ± 1:04	7:03 ± 0:45
Sleep efficiency (%)	92.6 ± 3.5	92.5 ± 2.1	92.8 ± 2.3	92.4 ± 3.0
Activity score (*N* of counts)	5007 ± 2444	5621 ± 2246	5553 ± 1970	5257 ± 2447
Fragmentation index	22.7 ± 11.4	26.2 ± 7.6	23.6 ± 9.5	22.0 ± 6.9
Subjective sleep quality				
Subjective estimate (0–100)	57 ± 15	63 ± 23	54 ± 22	60 ± 22
Cardiac autonomic activity				
HR (bpm)	53 ± 5	54 ± 5***	56 ± 4***	52 ± 5
RMSSD (ms)	72 ± 29	71 ± 38	66 ± 37	75 ± 33
HFP (ln[ms^2])	8.1 ± 0.8	7.8 ± 1.1	7.8 ± 1.0	8.0 ± 0.9
LFP (ln[ms^2])	8.4 ± 0.6	8.2 ± 0.7	8.1 ± 0.8	8.3 ± 0.6
LF/HF-ratio	1.6 ± 0.6	1.9 ± 1.0	1.6 ± 0.7	1.6 ± 0.8
TP (ln[ms^2])	9.0 ± 0.7	8.8 ± 0.9	8.7 ± 0.9	8.9 ± 0.7
Relaxation percentage (%)	79 ± 20	72 ± 29	66 ± 26 ***	90 ± 10

*** $p < 0.001$ compared to control

$^{\#\#\#}$ $p < 0.001$ compared to easy exercise

$^{¤¤¤}$ $p < 0.001$ compared to moderate exercise

$^{+++}$ $p < 0.001$ compared to vigorous exercise

Table 2 Summary of exercise and sleep variables during the days with different exercise duration and during a control day

	30 min	60 min	90 min	Control day
Exercise variables				
HR (bpm) during exercise	139 ± 7	141 ± 7	143 ± 9	–
RPE at the end of exercise	13.2 ± 1.6	13.8 ± 0.8	15.4 ± 1.5$^{\#\#\#,\ ¤¤¤}$	–
Sleep variables				
Bedtime	22:59 ± 1:05	23:20 ± 0:56	23:12 ± 0:59	23:24 ± 0:57
Awakening time	6:49 ± 1:09	6:59 ± 1:02	6:53 ± 1:12	6:59 ± 1:09
Time in bed	7:26 ± 1:04	7:32 ± 0:43	7:42 ± 1:01	7:38 ± 0:46
Actigraphic sleep quality				
Actual sleep time (h:min)	6:52 ± 1:02	7:01 ± 0:42	7:06 ± 1:04	7:03 ± 0:45
Sleep efficiency (%)	92.5 ± 2.1	93.2 ± 2.0	92.2 ± 3.0	92.4 ± 3.0
Activity score (*N* of counts)	5621 ± 2246	5186 ± 2095	5906 ± 2348	5257 ± 2447
Fragmentation index	26.2 ± 7.5	23.0 ± 8.0	27.0 ± 11.2	22.0 ± 6.9
Subjective sleep quality				
Subjective estimate (0–100)	63 ± 23	67 ± 18	60 ± 18	60 ± 22
Cardiac autonomic activity				
HR (bpm)	54 ± 5*	54 ± 6*	59 ± 5***, $^{\#\#,\ ¤¤}$	52 ± 5
RMSSD (ms)	71 ± 38	65 ± 27	56 ± 25**	75 ± 33
HFP (ln[ms^2])	7.8 ± 1.1	7.8 ± 0.9	7.6 ± 0.9*	8.0 ± 0.9
LFP (ln[ms^2])	8.2 ± 0.7	8.2 ± 0.6	8.0 ± 0.6	8.3 ± 0.6
LF/HF-ratio	1.9 ± 1.0	1.8 ± 1.0	2.1 ± 1.1**	1.6 ± 0.8
TP (ln[ms^2])	8.8 ± 0.9	8.7 ± 0.6	8.6 ± 0.7	8.9 ± 0.7
Relaxation percentage (%)	72 ± 29*	72 ± 21*	48 ± 27***, $^{\#\#,\ ¤¤}$	90 ± 10

*** $p < 0.001$, ** $p < 0.01$, and * $p < 0.05$ compared to control, $^{\#\#\#}$ $p < 0.001$ and $^{\#\#}$ $p < 0.01$ compared to 30-min exercise, $^{¤¤¤}$ $p < 0.001$, and $^{¤¤}$ $p < 0.01$ compared to 60-min exercise

Effects of exercise intensity (30-min easy, moderate and vigorous)

Significant main effects for HR ($p < 0.001$) and relaxation percentage ($p < 0.01$) after different intensity exercises were found. Nocturnal HR was higher after the moderate and vigorous exercises than after the control day ($p < 0.001$ for both) (Fig. 1a). No differences in HRV variables were observed between the exercise days or between the exercise days and the control day (Fig. 1b). Relaxation percentage was lower after the vigorous exercise day than after the control day ($p < 0.001$) (Fig. 1c). No significant differences were observed in any variables of actigraphic or subjective sleep quality between the days with different intensity exercises.

Effects of exercise duration (30, 60, and 90 min moderate)

Significant main effect for HR and relaxation percentage ($p < 0.001$ for both), as well as RMSSD and HFP ($p < 0.05$ for both) was found between different exercise durations. Nocturnal HR was higher after each day with different exercise duration than after the control day ($p < 0.001$–0.05) (Fig. 2a). Nocturnal RMSSD and HFP were lower ($p < 0.01$ and $p < 0.05$, respectively) after the day with 90-min exercise than after the control day (Fig. 2b). In addition, nocturnal relaxation percentage was lower after each exercise day than after the control day ($p < 0.001$–0.05) (Fig. 2c). It was also lower after the day with 90- than 60-min or 30-min exercise ($p < 0.01$ for both). Nocturnal HR was higher after the day with 90-min exercise than with 60- or 30-min exercise ($p < 0.01$). No differences were found in nocturnal HRV between the days with different exercise duration. Actigraphic and subjective sleep quality remained unchanged also between the days with different exercise duration, and between the aforementioned days and the control day.

Discussion

The present study investigated the effects of exercise intensity and duration on nocturnal cardiac autonomic modulation, actigraphic sleep quality, and subjective sleep quality. All methods were non-invasive and allowed to study the recovery process and sleep after exercise at home. The subjects were moderately active and healthy males, who performed five different running exercise sessions with HR guidance. The exercise protocol followed physical activity guidelines for the adults, and the exercise sessions were similar to that are routinely performed by individuals in endurance training programs. The results showed that both

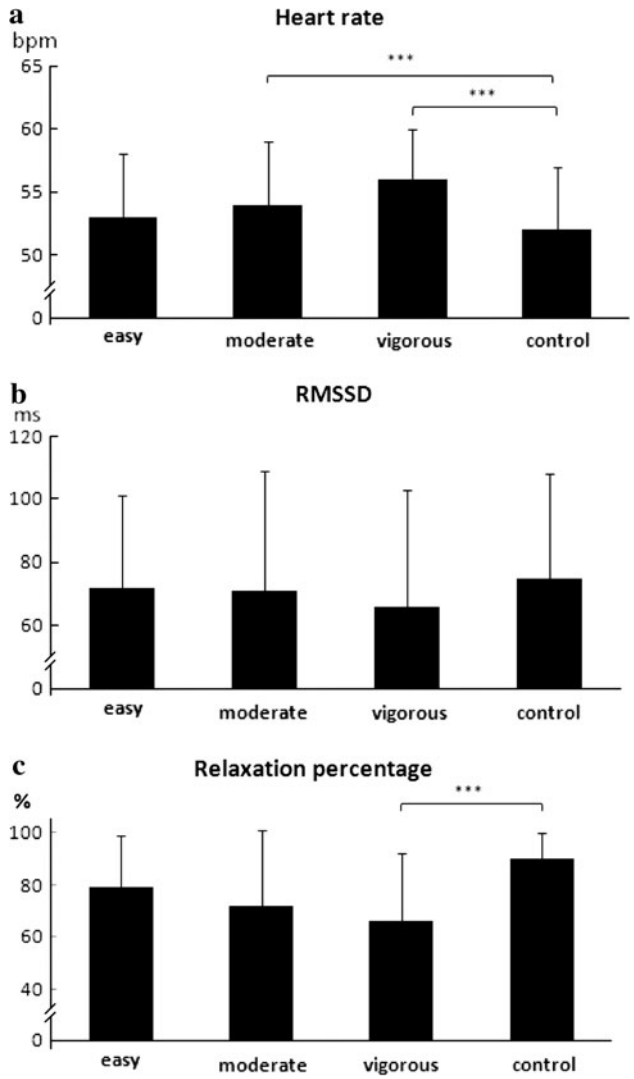

Fig. 1 Nocturnal heart rate (**a**), RMSSD (**b**) and relaxation percentage (**c**) after the days including 30-min exercise sessions with different intensity and after a control day. ***$p < 0.001$, **$p < 0.01$, and *$p < 0.05$

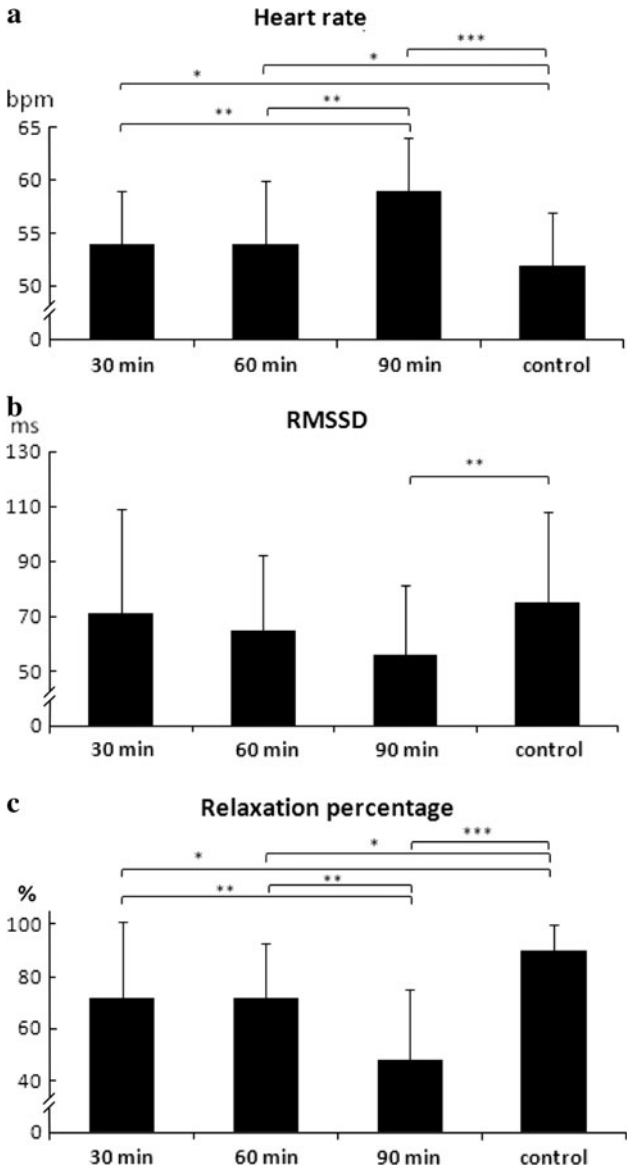

Fig. 2 Nocturnal heart rate (**a**), RMSSD (**b**) and relaxation percentage (**c**) after the days including moderate intensity exercise sessions with different duration and after a control day. ***$p < 0.001$, **$p < 0.01$, and *$p < 0.05$

increased exercise intensity and duration affected cardiac autonomic modulation during sleep after exercise, whereas sleep quality assessed by actigraphy or subjective evaluations was not affected by intensity or duration of exercise.

Effects of exercise intensity and duration on nocturnal cardiac autonomic activity

Increased exercise intensity elevated nocturnal HR and decreased relaxation percentage. However, increased exercise intensity did not affect HRV during sleep (see Fig. 1a–c; Table 1). The findings of the present study are mostly in line with previous studies showing continued physiological activation, i.e., increased HR during sleep after exercise (Hauri 1968; Bunnell et al. 1983, 1985; Roussel and Buquet 1982). Similarly, results showing increased HR, decreased relaxation percentage, and unaffected HRV have been reported after a vigorous 30-min late-night exercise when sleeping in a sleep laboratory (Myllymäki et al. 2011). Another study reported increased HR and decreased HRV during sleep after a 50-min moderate exercise (Hynynen et al. 2010). In addition, a study that investigated nocturnal blood pressure, actigraphic sleep quality, and HR, but not HRV, reported that intensity of exercise sessions (40 or 70% of VO_{2peak}) performed in the morning did not affect nocturnal HR or sleep variables although it caused significantly reduced nocturnal ambulatory blood pressure (Jones et al. 2009).

Studies on short-term recovery have reported that increasing exercise intensity causes slower recovery of vagally mediated HFP during the 30-min phase immediately after exercise (Kaikkonen et al. 2007). The impact of exercise intensity on short-term HRV recovery has also been reported by other studies (Kaikkonen et al. 2008; Martinmäki and Rusko 2008). Recently, short-term HRV recovery was found to be exercise intensity dependent, but surprisingly, significant autonomic disturbance was limited only to a very short (15 min) period even in unfit inactive young subjects after vigorous exercise (Gladwell et al. 2010).

The present result also showed that the longest exercise duration (90 min) with moderate intensity had greater effects on nocturnal HR, HRV, and relaxation percentage than the shorter exercise sessions (30 or 60 min) at the same intensity (see Fig. 2a–c). Previously, greater HR and lower HRV during sleep after both 50-min moderate exercise and 3.5-h marathon run at daytime compared to control sleep have been reported (Hynynen et al. 2010). Moreover, a 75-km cross-country skiing race lasting 4.5 h starting in the morning affected daytime HR and HRV, but not nocturnal HRV in absolute values (Hautala et al. 2001). It should be reminded that extremely long and difficult competitive exercises (such as marathon run or 75-km skiing) are not very common in daily life, and this type of events are often associated with nervous anticipation that may affect results. In addition, it is difficult to compare different exercise protocols performed at different times of the day. In studies of short-term HRV recovery, one study reported that exercise duration had no effect on HRV recovery (Kaikkonen et al. 2007), but in another study increased exercise duration decreased immediate HRV recovery (Kaikkonen et al. 2010).

The disturbed autonomic modulation, i.e., increased HR and decreased HRV, reflects parasympathetic withdrawal and/or sympathetic predominance (Goldberger 1999; Thayer et al. 2010). This may be caused by increased whole body metabolism, and circulating metabolites or catecholamines leading to elevated sympathetic outflow and/or reduced parasympathetic reactivation after exercise (Parekh and Lee 2005; Mourot et al. 2004). In addition, reduced blood pressure after exercise followed by stimulated arterial baroreflex to increase sympathetic activity, as well as changes in plasma volume due to exercise have been presented to possibly play a role (e.g., Mourot et al. 2004). These changes may reflect the effectiveness of exercise training, and some level of disturbance of homeostasis is surely needed to obtain the training effect. On the other hand, chronically disturbed ANS function may be associated with impaired health and possibly to a higher risk for cardiac events such as sudden death (Thayer et al. 2010).

When summarized, the results of the present study seem to support the previous findings that increased exercise intensity or duration affect nocturnal autonomic modulation in a dose–response manner, although long exercise duration was needed to see significant changes in nocturnal HRV. Previously, it has been reported that HRV was more sensitive than HR in assessing disturbance of homeostasis after exercise (Hynynen et al. 2010; Pichot et al. 2000). In the present study, HR seemed to be more consistently associated with the preceding activity than HRV, the latter being affected only after the longest exercise. Therefore, by combining information on both HR and HRV (e.g., by analyzing when HR is close to individual resting HR and when HRV is individually high as in relaxation percentage), more detailed indication on good recovery state may be obtained. Based on the present results, it also seems that exercise may affect cardiac autonomic activity several hours after exercise, as the sleep measurement occurred during a period from about 5 to 9 h after the cessation of exercise.

Effects of exercise intensity and duration on sleep quality

Sleep quality after different exercise sessions was assessed in the present study with non-invasive actigraphs and subjective evaluations. Although measurements of cardiac autonomic modulation during sleep showed continued physiological activation after intensive or long exercise sessions, no differences were observed in actigraphic or subjective sleep quality between the days with varying exercise intensity or duration. The present findings are in line with a study showing unaffected actigraphic or subjective sleep quality after vigorous 30-min late-night exercise (Myllymäki et al. 2011) or a study reporting unaffected actigraphic sleep quality after exercises with different intensities performed in the morning (Jones et al. 2009). Moreover, a study with highly fit cyclists reported that a long 180-min cycling exercise at 70% of maximal capacity ending 30 min before bedtime did not disturb actigraphic or subjective sleep quality (Youngstedt et al. 1999). Similar results were also reported with 60-min exercise at either moderate (60% of maximal capacity) or low intensity at late evening (O'Connor et al. 1998).

Previously, polysomnographic studies have reported improved sleep, i.e., increased total sleep time and slow wave sleep, and delayed and reduced REM-sleep, especially after acute exercise sessions at moderate to high intensity and with sufficient duration (Driver and Taylor 2000). The most consistent results have been obtained with exercise durations of 1 h or more (Driver and Taylor 2000), although half an hour seems to be enough for fit population according to some studies (Horne 1981). Despite that previous studies have reported positive effects of acute exercise on sleep, there also seem to be a threshold level for duration and intensity beyond which sleep is disrupted (Driver and Taylor 2000). It seems that the subjects in the present study did not reach that threshold of sleep disturbance even with

the most difficult exercises of the protocol, and therefore the sleep-promoting effects of exercise may have overcome the possible sleep-disturbing arousing effects of strenuous exercising. Thus, the present results confirm previous findings and suggest that aerobic exercise sessions performed in the early evening following the physical activity guidelines for healthy adults do not disturb actigraphic or subjective sleep quality.

Limitations

This study has some limitations to be taken into account when interpreting the results. The number of subjects was relatively small, which may have affected our findings. Polysomnographic measurements were not used in the present study, and therefore the effects of exercise on sleep stages cannot be discussed. This study was performed in a home environment and therefore several methods that can be easily used to monitor sleep and recovery at home were utilized. However, this can be considered also as an advantage as all the methods were non-invasive and the results were not affected by a laboratory environment. The subjects were responsible for controlling exercise intensity and duration themselves, which may have had some effects on the results. However, based on the measured RPE and HR-values in the context of exercise, the different exercise sessions seemed to be as strenuous for the subjects as expected. As the exercise sessions were performed in a home environment, it was not possible to measure the volume of exercise, which thus was not taken into account in the statistical analysis. Moreover, the subjects followed their normal sleep–wake cycle during the study. Therefore, as it was impossible to design bed and awakening times beforehand for the subjects, the protocol was designed so that all exercise sessions started at a constant time (6 p.m.). The results showed that the realized bed and awakening times were practically constant, and therefore the time from the cessation of exercise to sleep measurement varied approximately an hour (as exercises lasted for 30–90 min). However, the analyzed 4-h period for nocturnal cardiac autonomic activity occurred from about 5–9 h after the exercise sessions and therefore we believe that this is a minor limitation, which did not have notable effect on the results. Finally, the order of exercise sessions was evenly distributed and can thus be assumed to have no systematic effect on the results.

Conclusions and implications

The results of the present study showed that increased exercise intensity elevated nocturnal HR, but it did not affect nocturnal HRV, which was decreased only after the longest exercise in the present study. Although increased exercise intensity and duration caused some disturbance of cardiac autonomic modulation, sleep quality assessed by actigraphs and subjective estimations remained unaffected. Thus, acute exercise sessions performed according to physical activity guidelines during the early evening should not have much sleep disruptive effects among moderately active men. In practice, it seems that 30-min exercise sessions with a wide intensity range can be recommended for moderately active adults when it comes to enhancing health and improving fitness without disturbing sleep. Long exercise sessions may disturb more cardiac autonomic modulation and thus also the possibility of sleep disturbance is greater if the subjects are unaccustomed to that kind of physical activity. Thus, it is advisable to perform long exercise sessions earlier during the day, use slightly lower exercise intensity for improving the body's metabolism and long-term endurance, and/or take care of good sleep the next night for ensuring good recovery.

Acknowledgments This work was supported by Tekes and the following companies: Nokia, Suunto, Mawell, Firstbeat Technologies, Vivago, Varma, Mehiläinen, Solaris, and JTO School of Management, and by a grant from the Finnish Cultural Foundation.

Conflict of interest Heikki Rusko is currently a stockowner of Firstbeat Technologies Ltd., Finland.

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