EFFECTS OF EXERCISE INTENSITY ON RECOVERY OF THE AUTONOMIC NERVOUS SYSTEM IMMEDIATELY AFTER EXERCISE AND DURING SLEEP, AND ON SUBJECTIVE AND OBJECTIVE SLEEP QUALITY

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ABSTRACT

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The exercise can be considered as a one form of stress, because it induces marked changes in the autonomic regulation of heart rate (HR). During acute exercise, parasympathetic activity decreases and sympathetic activity increases. Parasympathetic activity declines a lot or even disappears, when the exercise intensity is high. After acute exercise cessation, fast changes occur in the cardiac function within few minutes: parasympathetic tone increases, whereas sympathetic activity decreases. However, how fast the vagal reactivation is, and how long autonomic regulation of HRV is disturbed after different exercises is not well known. In addition, exercise has beneficial effects on health. It may improve the sleep duration and quality, especially the amount of slow way sleep. The purpose of the present study was to find out how an acute exercise affects the recovery of the ANS. More specifically, how different exercise intensities affect recovery of heart rate variability (HRV) immediately after exercise and during sleep. In addition, the second purpose of the present study was to investigate how exercise intensity affects the subjective and the objective sleep quality during the nocturnal sleep.

Sixteen healthy moderately active men aged 36 years participated in the study. The subjects performed moderate intensity (60 % of VO_{2max}) and high intensity (75% of VO_{2max}) exercises in a laboratory. The autonomic regulation of HRV and subjective and objective sleep quality were measured during the night before and after exercise sessions. Also biomarkers of stress from salivary samples ware measured after both nights at home. In addition, acute effects of exercise on HRV and biomarkers of stress from salivary samples were measured in the laboratory right after exercise sessions. HRV was measured with Alive heart monitor and analysed with the Firstbeat HEALTH computer software. Body movements of the subjects were detected with the Actiwatch activity monitoring system and analysed with Actiwatch activity & sleep analysis 5 – software.

During the first minute of recovery, high frequency power (HFP) was significantly higher after moderate intensity exercise compared to the values during exercise ($p<0,01$). During the second minute of recovery HFP was first time significantly higher after high intensity exercise compared to the values during exercise, ($p<0,01$). Moreover, HFP was lower after high intensity exercise than after moderate intensity exercise during the immediate recovery period (1.minute: 1,6 ± 0,9 vs. 3,6 ± 1,4 ln[ms²] ($p<0,01$), 2. minute: 2,1 ± 0,8 vs. 4,5 ± 1,2 ln[ms²], 5.-10. minute: 2,8 ± 1,3 vs. 6,1 ± 1,4 ln[ms²]). During the first four hours of sleep, HR was significantly higher (57 ± 8 vs. 52 ± 6, bpm, ($p<0,001$)) and root mean square of successive RRIs (RMSSD) significantly lower (51,0 ± 25,6 vs. 64,8 ± 34,0 ms, $p<0,05$) after high intensity exercise compared to the control. Actual sleep time (7:29 ± 0:40 vs. 6:56 ± 0:41 h:min ($p<0,01$)) and subjective sleep quality (71,3 ± 18,7 vs. 53,0 ± 23,5, $p<0,05$) was higher after high intensity exercise compared to the control.

It seems that vagal reactivation is quite fast after exercise and it depends on exercise intensity. In addition, the results suggest that the high intensity exercise disturb the cardiac autonomic regulation only during early sleep. According to results, the high intensity exercise may improve the sleep quality. These results suggest that the sleep after high intensity exercise is enough for overall recovery and enough to restore homeostasis.

Key words: Autonomic nervous system, parasympathetic activity, sleep quality

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1 INTRODUCTION

The autonomic nervous system (ANS), especially its parasympathetic (PNS) branch, plays a key role in maintaining homeostasis by preparing the body to react to both external and internal signals. Homeostasis is considered the internal state in which dynamic feedback and regulation processes maintain visceral functions within a functional range. There are various types of stress such as mental, physical, and exercise related to overtraining stress, which can disrupt the homeostasis by depressing parasympathetic tone, when internal needs are subjugated in response to external challenges. (Porges 1992, Porges 1995.) An acute stress response increases heart rate, salivary cortisol and immunoglobulin A (IgA) levels. These responses indicate that stress affects autonomic cardiovascular, neuroendocrine and immune responses. (Benhamn 2007, Cacioppo et al. 1994, Dickerson & Kemeny 2004.) Due to these responses, salivary cortisol and IgA levels can be used as biomarkers of stress. In addition, heart rate variability (HRV) is a non-invasive method that can be used to study changes in the ANS activity (Hautala et al. 2009). Parasympathetic tone is used as a novel index of stress, and therefore accurate monitoring of PNS state will provide a mechanism to assess stress (Porges 1992).

Sleep is an important part of our life: it is time for both physical and psychological rest. During normal sleep parasympathetic tone dominates, when heart rate (HR) is slow and blood pressure declines (Trinder et al. 2001). In addition, the demand for energy declines, the function of the immune and endocrine systems refreshes and key cellular components are restored during the sleep. Most importantly, sleep is necessary for learning, memory and synaptic plasticity. (Halson 2008, Mignot 2008.) If sleep is disrupted, many health problems may occur: Sleep loss is associated with an increased risk of myocardial infarction and cardiovascular diseases, impaired glucose metabolism, type 2 diabetes, fatal accidents and infectious diseases. (Åkerstedt & Nilsson 2003, Irwin et al. 2006). Therefore, it is important that the duration of sleep is long enough and sleep quality is satisfying.

However, constant hurry, stressful jobs and poor lifestyle choices negatively affect sleep and its quality thus impairing recovery. Stress is one of the highest risk factors for impaired sleep. Acute stress may decrease parasympathetic modulation and increase sympathetic activity during sleep (Hall et al. 2004, Brosschot et al. 2007.) This kind of autonomic modulation of sleep is associated with poorer sleep maintenance and lower delta activity as well as longer sleep onset time. Therefore, decreased parasympathetic activity disturbs sleep and may induce insomnia (Hall et al. 2004, Morin et al. 2003). Because sleep is the most important restorative period in healthy people, disrupted sleep is associated with poorer rest and recovery (Brosschot et al. 2007).

Exercise does not only improve quality of life, it provides both physical and mental health benefits. Although exercise may be one of the stress factors, by initiating a stress response and delaying the recovery of HRV, exercise might also enhance sleep quantity and quality. Especially, exercise increases the time spent in slow wave sleep, which is considered to be the most restorative period of sleep. Exercise may promote sleep by reducing anxiety and depression, or by increasing the energy demand during the day, which is then restored during the night (restoration theory). In addition, exercise can improve sleep by increasing core temperature and body dehydration (thermogenic effects), or by advancing the circadian phase-shifting effects. Because of these explanations, the beneficial effect of exercise on sleep has generally been presumed, however, research results are conflicting and more research is needed. (Driver & Taylor 2000, Horne et al. 1983, Youngstedt 2005.) The type, intensity and duration of exercise have to be considered in terms of health outcomes. The American College of Sports Medicine and the American Heart Association have updated guidelines to improve and maintain the health for all healthy adults at the aged of 18-65 years: moderate intensity physical aerobic activity should be performed five times per week for 30 minutes at a time or alternatively vigorous intensity aerobic physical activity should be performed three times per week for 20 minutes at a time. (Haskell et al. 2007.)

The purpose of this study was to find out how an acute exercise, which was performed in accordance with the updated guidelines of American College of Sports Medicine and the American Heart Association, affected the recovery of the ANS. The most important

target of interest was to study how different exercise intensities affect recovery of HRV immediately after exercise and during sleep. In addition, the present study was designed to investigate how exercise intensity affects the subjective and objective sleep quality during the following nocturnal sleep. Exercise related stress response was measured with salivary cortisol and IgA samples, which were taken after the exercise and in the next morning. HRV measurements were used to assess the recovery of ANS, and sleep quality was measured by detecting body movements during sleep and with self-report questionnaires.

2 AUTONOMIC NERVOUS SYSTEM AS INDICATOR OF HOMEOSTASIS

The autonomic nervous system (ANS) is a part of the nervous system, which participates in the control of the most visceral functions such as gastrointestinal motility and secretion, urinary bladder emptying, body temperature, arterial pressure and many other activities. These kinds of activities are partly or entirely controlled by the autonomic nervous system, and the effects are very rapid and intensive. Different centers, located in the spinal cord, brain stem and hypothalamus and also in some parts of the cerebral cortex, activate ANS. Furthermore, visceral organs send subconscious sensory signals, which enter the autonomic ganglia, the hypothalamus or the brain stem, and then return to the organ affecting its function. This loop is termed the subconscious visceral feedback loop. The efferent autonomic nerve signals travels to target organs via sympathetic (SNS) and parasympathetic nervous systems (PNS). (Guyton & Hall 2006, 748.) The afferent feedback from organs also has an important role in regulating ANS function, especially parasympathetic tone. This is crucial to homeostasis and physiological stability. PNS and SNS are reciprocally innervated and their effects on a specific organ are antagonistic. This coordination ensures that the body reacts to both internal and external demands and to maintain homeostasis. (Porges 1992.)

The sympathetic nervous system. The sympathetic nervous system consists of two chains of ganglia, which lie parallel to the vertebral column, two prevertebral ganglia (the celiac ganglion and hypogastric plexus) and nerves, which extend from ganglia to the target organs, such as eye, heart, bronchi, pylorus, adrenal medulla and trigone (figure 1). Consequently signals from the cord passes through preganglionic neurons, the ganglion and postganglionic neurons to the target organ. The neurotransmitter in preganglionic nerve endings is acetylcholine, but most of the postganglionic neurons secrete norepinephrine. (Guyton & Hall 2006, 748-749.) SNS among other things activates metabolic output, accelerates heart rate, inhibits intestinal movements, constricts blood vessels and increases blood pressure. These kinds of actions prepare the individual for external challenges and optimize the relationship between organism and

the environment. Moreover, SNS is activated by changes in the external environment via somatic afferent fiber impulses. Therefore, SNS is called an ergotropic system. (Porges 1992.)

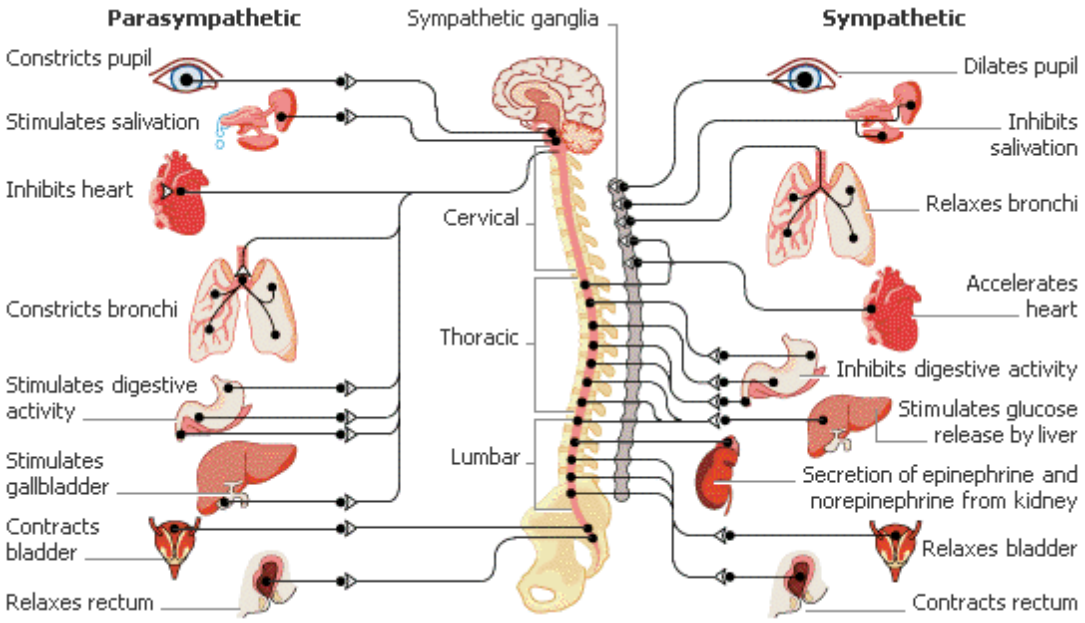


FIGURE 1. The target organs and functions of autonomic nervous system (http://drericchan.wordpress.com/2007/12/02/hydration-its-all-about-the-stress-or-lack-of-it/ 13.5.2009).

The Parasympathetic nervous system. Most of the parasympathetic nerve fibers (75 %) are in the vagus nerves, which pass through the entire thoracic and abdominal regions of the body. Via two vagus nerves, parasympathetic fibers reach the heart, lungs, stomach, liver, pancreas, and kidneys (figure 1). PNS also has pre- and postganglionic neurons, but some of the preganglionic neurons travel uninterrupted into the wall of the target organ, where the postganglionic neurons are located. Thus, some of the postganglionic neurons are very short. The neurotransmitter in the parasympathetic nerve endings is acetylcholine. (Guyton & Hall 2006, 750.) The main purpose of PNS is to promote anabolic activities regarding the restoration and conservation of body energy reserves, and to activate the recovery of vital organs. Among other things, the heart rate is

slowing, peristaltic movement is increasing and the pupils are contracting under the influence of the parasympathetic tone. PNS is activated by internal changes in the viscera. This activation ensures that internal functions are optimised to meet the internal demands, if there are no external challenges. Thus, PNS is called a trophotropic system. (Porges 1992.)

2.1 Autonomic regulation of heart rate

Heart rate (HR) is continuously modulated by different competing influences, which make HR dynamic and responsive. Heart rate variability (HRV) describes these variations of both heart rate and R-R intervals (ventricular depolarisations). HRV is due to the interplay of the sympathetic and parasympathetic nervous systems, which cause that HR is responding to plenty of stimuli such as the actions of ventilation, blood pressure control, thermoregulation and the renin-angiotensin system. The sympathetic nervous system increases HR, by innervating the Sino-Atrial (SA) and the Atrio-Ventricular (AV) nodes as well as the atria and the ventricles of the heart. The regulation is neural but also hormonal. Norepinephrine (NE) released from postganglionic fibres and epinephrine secreted from adrenal medulla act as neurotransmitters. The parasympathetic nervous system decreases HR by innervating the SA and AV nodes and the atrial myocardium. Neurotransmitter acetycholine (ACh) is released due to vagal stimuli. (Winsley 2002.)

The parasympathetic impulses more rapidly affect HR than sympathetic impulse (1 s versus 25 s), and also an increase in HR after withdrawal of vagal tone occurs faster than a decrease in HR after sympathetic impulses are stopped (2-5 s versus over 25 s) (Berntson et al 1997, Winsley 2002). This phenomenon is due to the different mechanisms of responding to the stimulus. The parasympathetic impulses provoke very fast activity of acetylcholinease, which removes ACh from synapse, whereas the sympathetic impulses evoke a sluggish intracellular secondary messenger system response after the initial binding of NE to the receptor sites. Also, the clearance methods of NE compared to ACh are different. Therefore, during parasympathetic

dominance, impulses slowing HR are transient and there is a high degree of HRV. Also due to longer lasting clearance methods of NE, HR remains elevated for a longer period of time and shows less variation during sympathetic dominance. (Winsley 2002.)

The factors that cause sympathetic nervous system activation and increases in HR are stimuli from thermoregulation, the renin-angiotensin system and atrial stretch receptors, whereas variations in vagal tone (and HR) are mainly due to ventilation as well as baro- and chemoreceptors, which also affect HR via sympathetic efferent impulses. Thermoregulation is a result of activation of the hypothalamus to decrease or increase body temperature, which increases either adrenergic or cholinergic sympathetic tone for vasoconstriction or vasodilatation and also affects HR. The renin-angiotensin system increases HR via angiotensin II receptors located in the cardiovascular control centre of the brain. The activation of receptors increases HR to maintain blood pressure. The atrial stretch receptors respond to increased venous return, which distend the atria, when HR increases. (Winsley 2002.) (Figure 2).

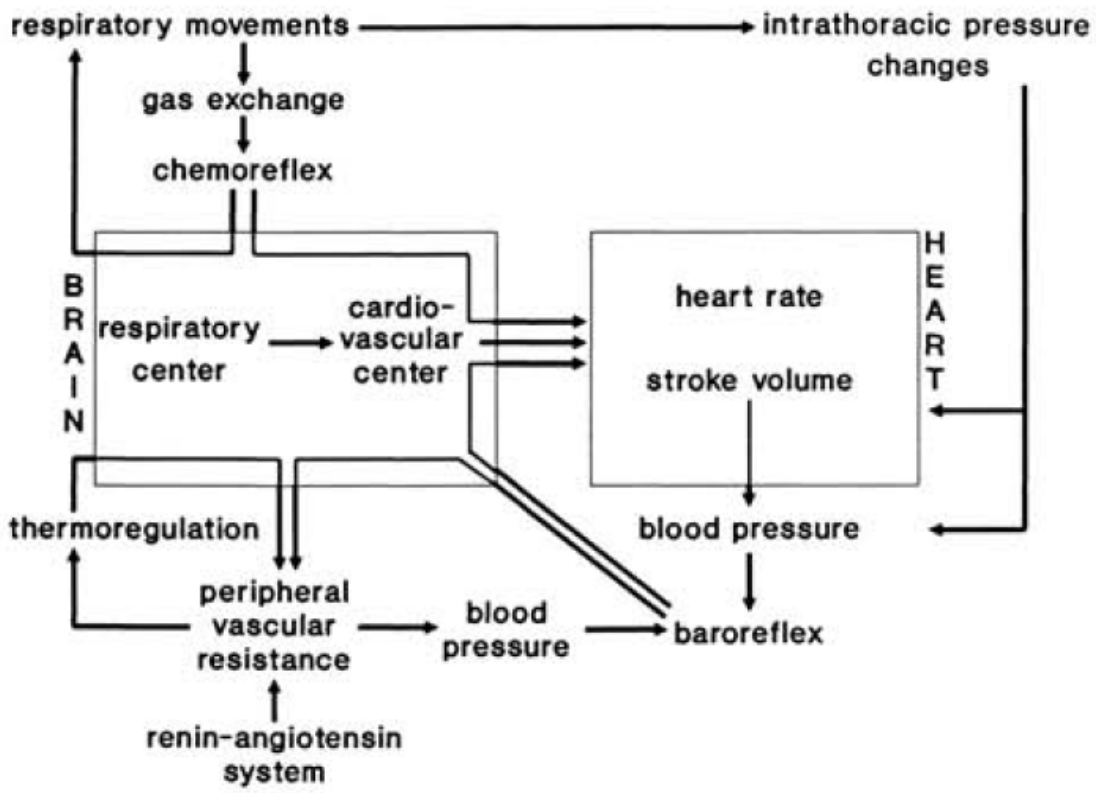


FIGURE 2. The factors affecting autonomic regulation of HR (van Ravenswaaij-Arts et al. 1993).

Ventilation affects parasympathetic tone. Inspiration activates airway stretch receptors to reduce parasympathetic tone, when HR increases towards the intrinsic HR, whereas during expiration, vagal tone is established again and HR slows again. This phenomenon is called respiratory sinus arrhythmia and mainly affects HR. Baroreceptors located in the aortic arch and the carotid sinus react to increased blood pressure and send afferent outflow to brain, when vagal tone increases and HR decreases. Blood pressure modulation of the heart is not only mediated by vagal impulses but also by sympathetic impulses. Chemoreceptors respond to hypoxia, hypercapnia or acidemia. Receptors located in the carotid bodies decline HR, whereas receptors in the aortic bodies increase HR. (Winsley 2002.) (Figure 2).

2.2 Stress response and measures for stress

There are many different ways to define a stress. Thus, there are various types of stress such as mental, physical and exercise-related overtraining stress. Many studies have demonstrated that increased heart rate, increased cortisol levels and increased salivary IgA levels are characteristic of an acute stress response, suggesting that stress affects autonomic cardiovascular, neuroendocrine and immune responses. (Benhamn 2007, Cacioppo et al. 1994, Dickerson & Kemeny 2004.) Alterations in the autonomic nervous system functions are involved in the physiological expression of stress. The purpose of the autonomic nervous system is to maintain homeostasis and react to both internal and external demands. Homeostasis reflects the dynamic feedback and regulation processes of visceral functions to maintain internal states within a functional range, and is mainly regulated by PNS. From this point of view, stress can be defined as the autonomic state, when homeostasis is disrupted and internal needs are subjugated in response to external challenges. According to this model, the parasympathetic tone is depressed to respond to the external needs, and stress may occur. In addition, stress responses and stress vulnerability may be indexed even if there is no shift in the SNS tone. In addition, people, who have problems with homeostasis may, have the greatest stress vulnerability. (Porges 1992, Porges 1995.)

2.2.1 Heart rate variability

Heart rate variability (HRV) as a non-invasive technique that can be used to study changes in autonomic nervous system activity (Hautala et al. 2009). Parasympathetic tone has been considered as a novel index of stress vulnerability and reactivity, thus an accurate monitoring of the PNS state will provide a mechanism to assess stress (Porges 1992). During acute stress, the parasympathetic modulation of HR is depressed and HR increases, but during chronic stress, vagal tone is tonically depressed. In turn, during recovery the parasympathetic tone is predominant, HR is slow and there is a high degree of HRV. The high degree of HRV illustrates the efficiency of neural feedback mechanisms, because the variability is a result of dynamic feedback mechanisms, which are controlled mainly by PSN. Furthermore, the higher the variance of HRV is, the better the body´s ability to react to external signals, and the more flexible is the behaviour. (Porges 1992, Porges 1995.) Low vagal tone is associated with high work stress and high emotional self perceived stress (Dissman et al. 2000, Vrijkotte et al. 2000).

2.2.2 Immunoglobulin A

The major class of the immunoglobulins in mucosal secretion is Immunoglobulin A (IgA) (Benhamn et al. 2009). Plasma cells in the submucosa synthesize IgA, which is then transported across the epithelial cells by the polymeric Ig receptor. IgA has three different functions: 1) the prevention of antigens and microbes from adhering to and penetrating the epithelium, 2) the interference of intracellular pathogens replication during transcytosis across epithelial cells, 3) binding of the antigens on the lamina propria and thereby the facilitation of the antigens excretion across the epithelium back to lumen. (Allgrove et al. 2008). Therefore, salivary IgA is an important part of the first line of defence against micro-organisms that cause, for example, upper respiratory tract infections. The concentration of IgA in saliva correlates relatively closely with resistance to viruses. (Allgrove et al. 2008, Benhamn 2007, Benhamn et al. 2009.)

Salivary IgA concentration has its own circadian rhythm; concentration of salivary IgA is higher in the morning than in the evening hours (Dimitriou et al. 2002).

Salivary IgA can be used as a biomarker of stress, because of its characteristics. Salivary samples are non-invasive and can be taken easily and frequently compared to blood. Furthermore, salivary IgA is biologically relevant as a functional immune end point and its biological half-life is 3 to 6 days, so it is relatively stable. Also the quantitative methods for salivary IgA level are fast and simple such as a radio-immunoassay or enzyme-linked immunosorbent assay. (Ng et al. 1999.) Acute stress increases salivary IgA concentration, but chronic stressors have shown to decrease IgA level. (Benhamn 2007, Benhamn et al. 2009.) Benhanm (2007) has demonstrated that IgA level increases rapidly as a reaction to acute mental stress. The concentration of IgA started to increase when the task instructions were told and continued to increase during mental task. Benhanm (2007), for example, has shown that these changes in salivary IgA concentration were short lived, and high levels of IgA returned to baseline in few minutes.

2.2.3 Salivary cortisol

Cortisol is one of the steroid hormones, more specifically glucocorticoid, which is synthesized from the steroid cholesterol and is secreted from the cortex of the adrenal gland. Secretion of cortisol is regulated by the hypothalamic-pituitary axis, where adrenocorticotrophid hormone (ACTH) and corticotropin-releasing hormone (CRH) have a major role: ACTH stimulates the release of cortisol and the production of CRH in the hypothalamus stimulates the production of ACTH. This regulation system is a negative feedback mechanism, thus increased amounts of cortisol inhibit the synthesis of ACTH and CRH. (Bartels et al. 2003.) When cortisol has been secreted into circulation, it binds either corticosteroid-binding globulin (CBG) (~ 80 %) or albumin (~14 %), and only 6 % of cortisol exists as a biologically active unbound form in plasma. Only active free cortisol can diffuse through the cell membrane and affects its target, whereas plasma protein bound cortisol is prevented to access the target. (Guyton

& Hall 2006, 954, Bartels et al. 2003, Lewis et al. 2005.) Cortisol affects carbohydrate (increases blood glucose concentration by stimulating gluconeogenesis and decreasing glucose utilization from cells), protein and fat metabolism (promotes mobilization of amino acid from muscle tissue and mobilization of fatty acid from adipose tissue), but it also has a function in stress and inflammation resistance (Guyton & Hall 2006, 953-954).

Cortisol secretion has its diurnal rhythm; over 24 hours secretion occurs as 10-15 well-defined pulses. The secretory activity is strongest in the morning, decreases during the day and is at its lowest during the night, but after few hours of sleeping, the secretory activity suddenly increases very quickly. (Bartels et al. 2003, Stone et al. 2001.) Right after awakening, cortisol response is extraordinarily intense: in healthy adults cortisol concentration has been observed to increase about 50 % or even 160 % during the 30 minutes after awakening (Pruessner et al. 1997, Glow et al. 2004). This clear diurnal rhythm follows the sleep-wake cycle and light-dark cycle, which is mainly due to stimulation of cells in the subraciasmatic nucleus, which react to light-dark cycle (Bartels et al. 2003, Stone et al. 2001).

Cortisol levels have been shown to respond to almost any type of stress including both physical and psychological stress (Guyton & Hall 2006, 952-953). During an acute stress state, the hypothalamic-pituitary-adrenal (HPA) axis is activated and plasma cortisol level increases (Dickerson & Kemeny 2004). During a chronic stress state, however, cortisol levels decline, which suggest a dysregulation of the HPA axis functioning during chronic stress (Pruessner et al. 1999). Because the secretion of cortisol changes when one is exposed to stress, changed cortisol levels can be used as a marker of stress. Elevated evening cortisol, elevated but also suppressed 24-h cortisol, elevated cortisol during the night and in the morning can all be a signal of stress (Powell et al. 2002). Especially cortisol awakening response (CAR) is widely used as an indicator of stress nowadays (Clow et al. 2004). Cortisol awakening response denotes the time period immediately after awakening (30-60 min), when cortisol level increases remarkably. This increase might be due to increased energy demands during the transition from sleep to wakefulness. (Clow et al. 2004, Pruessner et al. 1997.) Cortisol

awakening response is a part of the natural circadian cycle of cortisol secretion (Clow et al. 2004), but when cortisol levels are increased compared to repeatedly measured fixed time awakening cortisol levels, it seems to indicate an acute stress response (Pruessner et al. 1999). Cortisol can be measured from plasma, urine and saliva samples and the results are highly comparable between these different techniques. As an alternative choice, salivary cortisol measurements have many advantages: they are easy to take, non-invasive, fast, painless, and stress free. (King & Hegadoren 2002, Putignano et al. 2001.) However, there is a wide range of individual variation in diurnal cortisol levels in salivary samples (Ice et al. 2004, Stone et al. 2001).

3 SLEEP AS RESTITUTION

Sleep is considered a state, in which consciousness is reduced and movements of skeletal muscle and metabolism are decreased. Sleep is an important part of our lives: humans have a tendency to sleep about 7-8 hours per day. (Zisapel 2007.) Although there is no clear consensus as to why humans sleep and why humans require sleep. According to the recent research results, there are three different theories about importance of sleep. The first theory emphasizes that the abnormal regulation of metabolic processes is due to sleep deficit, and that is why sleep is essential in reducing energy demand and refreshing function of the immune and endocrine systems. (Halson 2008, Mignot 2008.) According to this model, effectiveness of human functions and eating peaks at certain time intervals. At another time it is advantageous to sleep and reduce the energy expenditure, of which distribution in the brain is almost 30 % of daily total body energy expenditure. During sleep, however, brain energy expenditure declines, and energy is saved for other processes. This model cannot explain the increase in energy expenditure during rapid eye movement sleep. (Mignot 2008.)

The second model highlights that sleep is necessary for learning, memory and synaptic plasticity: during sleep synaptic downscaling occurs, which eliminates unnecessary connections and leaves only the strongest connections intact. Then, energy and space requirements are reduced, which provides the possibility to maintain crucial learned circuits. (Mignot 2008.)

According to the third theory, the purpose of sleep is to restore key cellular components, which are used up during wakefulness, such as the components of cholesterol, intracellular transport, endo- and exocytosis. (Mignot 2008.) Obviously one can say that sleep is important for both physical and psychological well-being. Although sleep is a complex experience for the sleeper, quality and quantity of sleep are affected by many factors, and there are lots of variation in sleep patterns and need between individuals. (Savis 1994).

3.1 Sleep stages

Sleep can be divided in different stages in which physiological processes in the body are different according to each stage. Sleep stages can be distinguished from each other by using electroencephalography (EEG) patterns combined with electro-oculography (EOG) and electromyography (EMG) patterns. Mainly sleep is divided in two basic stages: non-rapid eye movement (non-REM) sleep and rapid eye movement (REM) sleep. (Savis 1994.) Human sleep consists of 5-6 cycles of 90 minutes, when these different sleep stages (S1-S4 and REM- sleep) are alternating (figure 3). During the first 90-min periods the portion of non-REM sleep is longer and during the last 90-min periods the portion of REM sleep is higher. (Zisapel 2007).

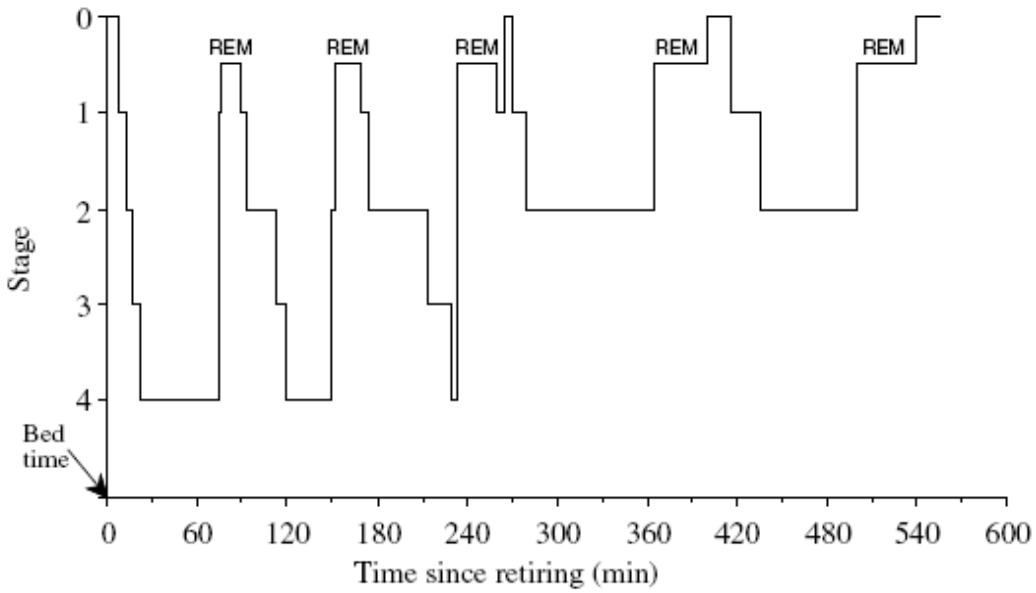


FIGURE 3. Different sleep stages (S1-S4 and REM-sleep) alternate during the night. (Åkersted & Nilsson 2003.)

3.1.1 Non-REM sleep

Non-REM sleep is characterized by slow electrical fluctuations in the brain (Zisapel 2007) and the frequency of fluctuations can be used to indicate sleep intensity or depth.

Non-REM sleep can be subdivided in four stages: S1, S2, S3 and S4. During S1, the transition from wakefulness to sleep occurs, and respiration deepens, responsiveness to outside stimuli reduces, thoughts are drifting (Savis 1994.), muscle activity is still quite high, and there may be some eye movements. The brain waves decelerate to 6-8 Hz and have low amplitude. (Åkersted & Nilsson 2003.) In healthy sleepers, stage 1 is usually short, lasting for only few minutes. Stage 1 is followed by stage 2, when brain waves become slower (4-8 Hz), but some sleep spindles at frequency of 14-16 Hz might occur. Muscle tonus declines and cognitive processes are short and fragmented. About 50 % of total sleep time is spent at this stage, and it lasts 30 minutes in healthy sleepers. This stage does not have great functional importance, but it provides basic recovery. (Savis 1994, Åkersted & Nilsson 2003.)

Stages 3 and 4 are called as delta sleep, because the physiological processes in these stages are very similar. Delta sleep is also called slow wave sleep (SWS), because brain waves have very slow frequencies (0,5 - 4 Hz) and high amplitudes. Delta sleep is the deepest stage of sleep and about 15-20 % of total sleep time is spent at this stage. (Savis 1994.) This stage is considered to be essential for body restitution: growth hormone secretion and cell division peak and cortisol secretion suppresses during delta sleep (Savis 1994, Åkersted & Nilsson 2003). Metabolism declines while breathing, heart rate and cerebral blood flow slow down (Åkersted & Nilsson 2003).

3.2.2 REM sleep

REM sleep differs a lot from non-REM sleep. Eye movements and dreams are typical in REM sleep, as is a virtual absence of muscle tonus in antigravity muscles. An awake brain is also typical in REM sleep, especially the hippocampus, amygdala and occipital areas are active (Savis 1994, Åkersted & Nilsson 2003). Increased cerebral blood flow, brain temperature and brain protein synthesis are also associated with the awake brain. (Savis 1994.) In addition, heart rate, respiratory rate, blood pressure and body temperature are almost the same as during wakefulness. However, body temperature regulation is inhibited, whereupon the sleeper would not respond to ambient temperature changes. (Åkersted & Nilsson 2003.) REM sleep is associated with

learning, memory and synaptic plasticity, and 20-25 % of total sleep time is spent in this sleep stage.

3.2 Sleep regulation

Sleep is a neurochemical process in which sleep-promoting and arousal centers are affecting each other. There are plenty of parallel arousal centers in the brain, which are located mainly in the brainstem and hypothalamus. (Zisapel 2007.) Neurons of these centers are monoaminergic, such as cholinergic, noradrenergic and serotoninergic neurons and are capable of increasing wakefulness (Saper 2005). Although complete alertness and cortical activation are needed for coordinated activity of these neuron networks (Zisapel 2007), compared to the arousal system sleep-promoting centers are few, and the main component of the sleep-promoting system is the ventrolateral preoptic nucleus (VLPO) in the hypothalamus. Neurons in this center are gamma-aminobutyric acid (GABA) ergic and also produce inhibitory neuropeptide galanin. (Mignot 2008, Saper et al. 2005.)

The VLPO neurons have connections with arousal neurons and during the sleep they inhibit the arousal systems. During wakefulness arousal neurons and neurotransmitters such as acetylcholine, noradrenaline and serotonin in turn inhibit the activity of the VLPO neurons. These inhibitory processes create a flip-flop switch, where the transition from wakefulness to sleep, and sleep to wakefulness, is very fast. This process ensures that the gradual transition occurs and intermediate stages do not occur. (Chou et al. 2002, Saper 2005, Zisapel 2007.) There are two types of sleep regulation, which affect and drive these inhibitory processes: a homeostatic regulation and a circadian regulation (Zisapel 2007). Homeostatic and circadian regulations are considered to be independent systems, but the latest research findings suggest that these sleep regulation systems might be connected (Mignot 2008).

3.2.1 Homeostatic sleep regulation

Homeostatic regulation of sleep maintains the balance between wakefulness and sleep state. Homeostatic processes increase the propensity to sleep during sleep deprivation and decrease this propensity during sleep (Achermann & Borbely 2003): The longer one stays awake, the longer and more intensively one will sleep. (Jones et al. 2008, Zisapel 2007.) The mechanism behind this regulation is not well known, but adenosine might have an important role in promoting sleep (Saper et al. 2005, Zisapel 2007). Amount of adenosine in forebrain has demonstrated an increase during wakefulness and a decrease during sleep (Porkka-Heiskanen et al. 1997, Stecker et al. 2000). Adenosine is also associated with increased EEG activity during SWS and therefore may promote sleep (Porkka-Heiskanen et al. 1997). Accumulation of adenosine during wakefulness might affect the transition from wakefulness to sleep by either inhibiting arousal centers or activating sleep-promoting centers (Arrigoni et al. 2006, Saper et al. 2005, Scammel et al. 2001, Stecker et al. 2000). However, the effects of adenosine on sleep regulation may be limited only to specific basal forebrain areas (Zisapel 2007).

3.2.2 Circadian sleep regulation

Sleep is also regulated by a circadian system (Saper et al. 2005). The main purpose of the circadian rhythms is to coordinate the human body with environmental requirements, such as the light-dark cycle and to maintain synchronization between internal physiology processes like the regulation of body temperature, hormone release, sympathetic activation and metabolism (Laposky et al. 2008). Circadian rhythms promote sleep during the sleep state, which ensures continued sleep even though the homeostatic propensity is decreased toward the end of the sleep cycle (Achermann & Borbely 2003). Susceptibility to wakefulness is built up during the day and reaches the maximum just before sleep. At the same time, homeostatic regulation is approaching its peak. Circadian regulation is based on the function of the suprachiasmatic nucleus, which reacts on the external light-dark cycle, firing rapidly during the light period. This nucleus has connections to hypothalamic subraventricular zone, which is an important part of circadian drive. This zone has indirect connections to the VLPO and some parts

of the arousal centers via dorsomedial hypothalamic nucleus. Via this loop the circadian cycle and body temperature, sleep and wakefulness are maintained. (Saper et al. 2005, Zisapel 2007.)

3.3 Stress, sleep and health

During sleep, vagal activity provides an index of normal homeostatic feedback (Porges 1995). During normal sleep, HR and blood pressure decline, this is modulated by the ANS. During non-REM sleep, the PNS dominates, and the shift from sympathetic dominance to parasympathetic dominance appears to be mainly caused by changes in the PNS activity. Autonomic balance during REM sleep is quite similar to wakefulness. (Trinder et al. 2001.) However, acute stress affects the autonomic modulation of sleep: parasympathetic modulation is decreased during non-REM and REM sleep, and sympathetic activity is increased during non-REM sleep (Hall et al 2004). Also Brosschot et al. (2007) demonstrated that during stress and prolonged worrying, HR is increased and HRV is decreased during sleep. Decreased parasympathetic tone and increased sympathetic tone is associated with poorer sleep maintenance and lower delta activity, as well as, longer sleep onset time, and therefore, decreased parasympathetic activity disturbs sleep and may induce insomnia (Hall et al. 2004, Morin et al. 2003). Disrupted sleep is associated with poorer restitution and recovery, because sleep is the most important restorative period in healthy people (Brosschot et al. 2007).

Nowadays many aspects of a stressful life can affect sleep and impair recovery, which might have an important role as a mediator of stressful events on healthy problems. An increased risk of myocardial infarction and cardiovascular diseases, impaired glucose metabolism, type 2 diabetes and fatal accidents are all associated with sleep loss. (Åkerstedt & Nilsson 2003.) Too-short nocturnal sleep increases the risk of coronary artery disease and coronary artery disease mortality, which might be due to the excessive sympathetic activity (Mallon et al. 2002, Ayas 2003). Nilsson et al. (2004) demonstrated the effect of sleep loss on the risk for diabetes and obesity: the results of the study shows that sleep disturbances and possible elevated resting HR increases the

risk of diabetes. Other possible reasons are alterations in glucose metabolism, such as increased insulin resistance, or alterations in appetite regulation, such as decreased amount of leptin-hormone, or decreased energy expenditure (Knutson 2007). Sleep deprivation has shown to alter physiologic characteristics of immune cells and might, via these alterations, increase the risk of infections. Sleep loss increases the number of inflammation markers, such cytokines in blood and also increases transcription of these markers. (Irwin et al 2006). In addition, Everson (2005) reported that sleep loss alters blood leukocytes and activates antibody production in serum, although the antigen of these antibodies is not present. Sleep deprivation is also associated with mortality both in men and women (Nilsson et al. 2001, Hublin et al. 2007). Too short and too long nocturnal sleep is demonstrated to increase mortality (Hublin et al. 2007).

3.4 Measurements of sleep quality

Quality of sleep can be measured subjectively at home with sleep questionnaires, and also objectively by using actigraphy monitoring system, which monitor and collect data from body movements. Actigraphy is a wristwatch-like little computer, which can continuously collect data for prolonged periods (1 week or longer). Actigraphy is based on an acceleration sensor, which translates physical movements into numeric representations. Some devices use a pietro-electric beam to detect the movements in two or three axes. The detected movements are translated to digital counts accumulated across given epoch intervals and stored in the actigraphy memory. Afterwards data can be downloaded to computer and then analysed. (Sadeh et al. 1995, Sadeh & Acebo 2002.)

The data obtained from actigraphy can be used to determine sleep-wake patterns and circadian rhythms (Anconi-Israel et al. 2003). As a non-invasive method, it is easier to use and less expensive as compared to polysomnography (PSG), the gold standard for recording sleep. Actigraphy is usually compared to PSG in validation studies. (Sadeh et al. 1995, Sadeh et al. 2002, So et al. 2005, Wang et al. 2008). Actigraphy is a valid and reliable method to measure sleep patterns in healthy normal people (Anconi-Israel et al.

2003, Sadeh et al. 1994, Morgenthaler et al. 2007), especially, when major periods of sleep and wake are differentiated. It is also accurate, when sleep duration, sleep efficiency and awakenings after sleep onset are measured, but it cannot differentiate reliably between REM-sleep stages and non-REM-sleep stages. (Thorby et al. 1995.) The epoch by epoch agreement for actigrafically measured sleep was quite high (>0,85) compared to PSG. Correlation was also reasonable (>0,80), when the whole night sleep and sleep efficiency was measured. (Sadeh et al. 1995, Sadeh et al. 2002.) However, the accuracy of actigrahpy is reduced, when wakefulness during the night increased and sleep efficiency decreased (Sadeh et al. 2002, Wang et al. 2008) Also some sleep disorders and disorders that involve in altered motility patters attenuate the validity of actigraph. (Sadeh et al. 1995, Sadeh et al. 2002, Thorby et al. 1995.)

Other methods for sleep-monitoring at home are self-reported questionnaires, which are most commonly used to assess sleep quality and insomnia (Buysse et al. 2008, Moul et al. 2004, Smith & Thinder 2001). Self-reported questionnaires are subjective and retrospective in contrast to PSG and actigraphy (Smith & Thinder 2001), and may not be related to these objective sleep measures (Buysse et al. 2008, Moul et al. 2004). Still questionnaires are the cheapest and easiest way to measure sleep quality, and they may also provide such important information about psychological and behavioural aspects of sleep that can not be measured with objective methods (Smith & Thinder 2001, Moul et al. 2004).

4 EXERCISE AND RECOVERY

It has been demonstrated that exercise has an important role in improving the quality of life and providing both physical and mental health benefits. Exercise is an important part of a healthy lifestyle. The type, intensity and duration of exercise have to be considered in terms of health outcomes. The American College of Sports Medicine and the American Heart Association have updated the following guidelines to improve and maintain the health for all healthy adults aged 18-65 years. (Haskell et al. 2007.)

4.1 Primary exercise recommendations

According to the guidelines of the American College of Sports Medicine and the American Heart Association moderate intensity physical aerobic activity (3-6 METs) should be performed five times per week and 30 minutes at a time. Alternatively, vigorous intensity aerobic physical activity (> 6 METs) should be performed three times per week 20 minutes at a time. These activity levels could be combined for example so that a person walks (moderate intensity) two times per week 30 minutes and jogs 20 minutes two times per week. The moderate exercise can be performed in three 10 or more minute periods during the day. The moderate intensity means that heart rate accelerates significantly, so exercise modes such as brisk walk are included in this intensity. In contrast, during the vigorous intensity exercise heart rate and breathing are fast. Besides these activities, one should also maintain daily low intensity activities such as self care, cooking, casual walking or shopping. Recommendations also contain guidelines for muscular strength and endurance. All adults should perform, at least two times per week, activities which maintain or increase muscular strength or endurance. Physical activity and health have a dose-response relationship. Therefore, one should exceed these minimum guidelines, if one wants more health benefits and a reduced risk of lifestyle diseases: type 2 diabetes, cardiovascular diseases, and chronic disabilities. (Haskell et al. 2007.)

4.2 Effects of exercise on the autonomic nervous system

4.2.1 Acute exercise and ANS

Exercise induces marked changes in autonomic regulation of HR, and it is also one form of stress. During acute exercise, HR accelerates firstly because of the withdrawal of vagal tone. When the intensity of exercise increases, HR accelerates because of the increase in sympathetic tone (Hautala et al. 2009). When exercise intensity reaches 50-60 % VO_{2max}, parasympathetic tone declines a lot or even disappears (Hautala et al. 2009, Kaikkonen et al. 2007, Kaikkonen et al. 2008, Tulppo et al. 1998). After exercise cessation, fast changes occur in the cardiac function within few minutes. During this immediate recovery, parasympathetic tone increases, whereas sympathetic activity decreases. HRV provides a mechanism to study the changes in autonomic activity after exercise. (Hautala et al. 2009.) Especially the root mean square residual (RMS) and the root mean square successive difference of the R-R intervals (RMSSD) can be used to measure the early recovery of vagal tone after exercise (Goldberger et al. 2006). Previous studies have investigated immediate, short-term and long-term recovery of HRV after different aerobic exercises. (Hautala et al. 2009.)

One of the first studies that investigated the effects of exercise on heart rate dynamics during recovery was published by Perini et al. (1989). The authors showed that the recovery of HR to baseline after exercise depends on the exercise intensity. After low intensity exercise, HR dropped to baseline values in five minutes, but after moderate and high intensity exercises, HR was still higher than baseline values after five minutes. (Perini et al. 1989.) More recent studies have investigated the immediate recovery of HRV after exercise. Martinmäki and Rusko (2007) have studied HRV during immediate recovery after low and high intensity exercises. Subjects performed bicycle exercises; low intensity exercise, when intensity was 29 % of maximal power and high intensity exercise, when intensity was 61 % of maximal power. They found that vagal reactivation occurred during the first minute of recovery after low-intensity exercise and during the second minute of recovery after high-intensity exercise. The results also showed that high frequency power (HFP) was higher during the end of the recovery

period after low intensity exercise compared with high intensity exercise. Martinmäki and Rusko (2007) have concluded that vagal reactivation occurs quickly after exercise and that the recovery of autonomic control of HR is intensity dependent.

Kaikkonen et al. (2008) have studied post-exercise HR dynamics immediately after different high-intensity exercises (continuous interventions (21 min) at 80 and 85 % of the velocity of VO_{2max} and interval interventions (3x 7 min) at 85 and 93 % of the velocity of VO_{2max}), but this time the subjects were endurance athletes. In this case, they found that during the immediate recovery total power (TP) and low frequency power (LFP) increased, but HFP did not during the first five minutes. They stated that these results are only partly in agreement with the previous studies. They also found that small changes in the exercise intensity, even at high levels, affect recovery of HRV so that higher intensity causes longer recovery. (Kaikkonen et al. 2008). Kaikkonen et al. (2007) have also studied the HRV dynamics during immediate recovery after different endurance exercises: two low-intensity (3500 m and 7000 m at 50 % of the velocity of VO_{2max}), two moderate-intensity (3500 m and 7000 m at 63 % of the velocity of VO_{2max}), and one high-intensity exercise (3500 m at 74 % of the velocity of VO_{2max}). The results showed that HFP increased during the first minute of recovery after low-intensity exercise. However, after moderate-intensity exercise, an increase in HFP was seen after the fourth minute (shorter duration) and after the fifth minute (longer duration), while after high intensity exercise at the end of the recovery period of 30 min. This reflects fast vagal reactivation. In addition, these results showed that after low-intensity exercise the recovery of HFP, and thereby recovery of parasympathetic tone, is faster than after other exercises. (Kaikkonen et al. 2007.) Kaikkonen et al. (2007) have stated that the intensity of exercise affected significantly the post-exercise HRV level.

In summary, it seems that vagal reactivation is quite fast after exercise, but this depends on exercise intensity (Kaikkonen et al. 2007, Kaikkonen et al. 2008). However, recovery of HRV to pre-exercise values could take much longer. In the study of Martinmäki & Rusko (2007), parasympathetic and sympathetic tone reached the pre-exercise values in 10 minutes. However, Kaikkonen et al. (2008) and Kaikkonen et al.

(2007) have shown that after high-intensity exercises the indicators of parasympathetic tone were not fully recovered during the period of 30 min post-exercise.

Also Bernardi et al. (1997) showed that the recovery of the autonomic nervous system takes more time than just a few minutes after prolonged exercise (46 km run). According to the results, SNS predominated whereas the PNS activity was decreased during the first 30 minutes after exercise. The ANS activity returned to baseline values in 24 hours. (Bernardi et al. 1997.) Also James et al. (2002) investigated the effects of interval exercise (6x800m, speed 1 km/h below maximum speed) on HRV and ANS. They found that HRV was not fully recovered during the first hour after exercise, but after 72 h there were no differences compared to resting values. (James et al. 2002.)

Tercziotti et al. (2001) investigated the effects of light (50 % of anaerobic threshold) and moderate (80 % of anaerobic threshold) intensity exercises on autonomic control of cardiovascular activity during the post-exercise recovery. They found that the autonomic control was altered during the first 15 minutes, which was due to both parasympathetic and sympathetic activity levels. After moderate intensity exercise, HR was slightly raised after 60 min. They also found that recovery of the vagal component was strongly dependent on the intensity of exercise. (Tercziotti et al. 2001.) Mourot et al. (2004) have found similar results: during early recovery autonomic activity did not resume to resting values during the first 60 minutes, and this recovery might depend on intensity of exercise. They also found that 24 and 48 hours after the termination of exercise, HRV had returned to baseline values in the supine position, but the presence of tachycardia and reduced total frequency power in the upright position suggested disturbed cardiovascular functions. (Mourot et al. 2004).

Autonomic recovery after exercise in trained athletes was also studied by Seiler et al. (2007). Highly trained runners performed two training sessions with intensity below ventilatory threshold 1 (VT1) (60 min and 120 min) and one training sessions with intensity between VT1 and VT2 (total 60 min: 20 min warm-up, 30 min exercise intensity between VT1 and VT2, 10 min cool-down). In addition highly trained and trained runners performed interval training session above VT2 (total 60 min: 6 x 3 min

96 % VO2 max, 2 min recovery). Three main findings were made: 1) In highly trained subjects, recovery of parasympathetic tone was rapid after running when intensity was below VT1 (5-10 min), whereas recovery was delayed after exercise, when intensity was above VT2 (in 30 min.). This suggests that VT1 may be a key intensity level when autonomic function is perturbed. 2) In highly trained subjects, there was no any further delay in HRV recovery even though intensity was increased. 3) In highly trained subjects the ANS recovery was faster than in trained subjects. These characteristics of highly trained subject may reflect on how highly trained athletes organize the day-to-day variation of training intensity. (Seiler et al. 2007.)

Bunnell et al. (1983) have demonstrated that exhaustive exercise affected HR dynamics so that HR was significantly elevated during the sleep after exercise. Hautala et al. (2001) have examined the effects of prolonged maximal exercise (a 75 km cross-country skiing race) on cardiac autonomic regulation during rest. The results showed that after prolonged vigorous exercise, vagal tone is blunted for several hours, and that the time that is needed for the recovery of parasympathetic tone, is dependent on individual fitness. The vagal outflow did not reach pre-exercise levels until the second day after the race, but then the vagal modulation increased over pre-exercising level. (Hautala et al. 2001.)

According to these studies, HRV recovers slowly as a function of time and is dependent on exercise intensity. Thereby, post-exercise resting levels could be reached in few minutes, if the exercise intensity is low and exercise duration is short, whereas recovery may last even two days when the intensity is high and duration long. (Hautala et al. 2001, Tercziotti et al. 2001, Mourot et al. 2004.)

4.2.2 Exercise training, fitness level and ANS

Aerobic exercise training has been shown to increase parasympathetic tone during the day and during nocturnal sleep (Hautala et al. 2009). Tulppo et al. (2003) have studied the effects of moderate and high intensity aerobic training (8 week) on vagal indexes of

HRV recorded over 24-hour period. The results show that aerobic training, regardless of intensity of exercise, alters autonomic regulation of HR towards parasympathetic dominance (Tulppo et al. 2003). Also Melanson et al. (2001) showed increased resting vagal tone after a 12-week moderate-to-vigorous-intensity endurance exercise program in sedentary men.

Good physical fitness level is also associated with a higher vagal modulation of HR. Studies have reported that parasympathetic tone is much higher in well trained male and female subjects than controls in both laboratory and ambulatory conditions. (Hautala et al. 2009.) However, exercise has also negative effects on HR dynamics. Autonomic imbalance, for example, is also seen in the overtraining syndrome (Hedelin et al. 2000). Hedelin et al. (2000) have expressed that in an overtrained athlete the shift toward increased HRV and a reduced resting HR suggest a cardiac autonomic imbalance with extensive parasympathetic modulation.

4.3 Effects of acute exercise on IgA

The effects of exercise as a stressor on salivary IgA levels have been studied by several researchers. Some of the earlier studies have shown that salivary IgA concentration, secretion rate and IgA to total protein ratio will decrease after high intensity exercises (Mackinnon et al. 1993, Mackinnon & Jenkins 1993, Tharp & Barnes 1990), whereas some studies have not found correlations between exercise and IgA consentration (McDowell et al. 1991). Moreover, salivary IgA secretion has been shown to decrease after high intensity exercise in more recent studies (Engels et al. 2003, Mackinnon et al. 1993, Steerenberg et al. 1997). These results suggest that immune responsiveness is decreased after high intensity exercise adding the risk for upper respiratory track infectious (Steerenberg et al. 1997, Mackinnon et al. 1993, Mackinnon & Jenkins 1993, Tharp & Barnes 1990), but may increase after lower intensity exercises (Allgrove et al. 2008). However the results are still conflicting.

Some recent studies have shown slightly different results. Allgrove et al. (2008) have examined the effects of exercise intensity on concentration and secretion of salivary IgA. The subjects performed three exercises at different intensities (50 % VO_{2max}, 75 % VO_{2max}, an incremental test to exhaustion). Allgrove et al. (2008) found that secretion of salivary IgA increased significantly only after short-duration high-intensity exercise, while concentration of IgA and salivary flow rate were not significantly affected. According to Allgrove et al. (2008) these results did not support the previous studies, where intensity of exercise associated with immune suppression, and they speculated that maybe the intensity or duration of exercise were not strenuous enough in this study. They have also suggested that the results might be associated with changes in activity of sympathetic nervous system (Allgrove et al. 2008).

Davison et al. (2009) have also studied the effects of exercise on IgA levels. They showed that prolonged (2, 5 h at 60 % VO_{2max}) exercise decrease salivary IgA to osmolality ratio, but did not affect IgA concentration or secretion (Davison et al. 2009). Sari-Sarraf et al. (2008) have found that after moderate intensity soccer-specific intermittent exercises the salivary IgA concentration increased immediately after exercise but returned to pre-exercise values 24 h afterwards, whereas, the IgA secretion rate, IgA to osmolality ratio, and IgA to total protein were not affected. They speculated that increase in IgA concentration may result, at least in part, from significant decrease in salivary flow rate. Sari-Saffar et al. (2006) have found earlier that intermittent soccer-specific exercise did not affect IgA variables, but salivary flow rate was suppressed. Thomas et al. (2009) have not traced any differences in IgA concentration after short-term high-intensity cycling exercise in boys aged 15-16 years. According to Shimizu et al. (2007), IgA levels were increased significantly after moderate intensity endurance training in elderly.

According to these studies, effects of exercise on salivary IgA levels are still conflicting. But taken together, it seems that exercise intensity has an important role in affecting the levels of IgA (Steerenberg et al. 1997, Mackinnon et al. 1993, Mackinnon & Jenkins 1993, Tharp & Barnes 1989, Allgrove et al. 2008, Davison et al. 2009, Sari-Sarraf et al. 2008).

4.4 Effects of acute exercise on cortisol

Exercise-induced cortisol secretion has been studied by several researchers, and it seems that acute exercise increases cortisol levels (Elloumi et al. 2003, Karkoulias et al. 2008, Maresh et al. 2006, Rudolph & McAuley 1998; Thomas et al. 2009), and that this increase is related to exercise intensity and duration (Hill et al. 2008, Maresh et al. 2006, Rudolph & McAuley 1998). Moderate to high intensity exercises increase cortisol levels significantly above pre-exercise values, whereas low intensity exercise does not remarkably affect cortisol secretion. It seems that there is an intensity threshold point, when the cortisol concentration starts to increase, and this point is above 60% but below 80 % of VO_{2max}. (Hill et al. 2008, Maresh et al. 2006.) After exposure to acute stress, cortisol levels reach peak values in 30 min, whilst production of ACTH stimulates secretion of cortisol (Lundberg 2005).

4.5 Effects of exercise on sleep

It has been proposed that exercise might enhance the quantity and quality of sleep and especially increase the time spent in slow wave sleep (Driver & Taylor 2000). This idea supports the theories of body restoration and energy conservation: increased daytime activity such as physical exercise results in catabolic processes, which in turn have to be compensated for during the sleep. The higher the energy demands during the day, the longer is the time spent in the most restorative sleep, slow wave sleep. (Taylor et al. 1997). The other theory explaining the relationship between sleep and exercise is the body-heating theory, which proposes that a high and sustained rate of body heating, particularly increased core temperature and body dehydration, enhances the SWS and exercise affects sleep only via body-heating (Horne 1983). The beneficial effect of exercise on sleep has generally been presumed, but research results are conflicting and more research is needed (Driver & Taylor 2000).

4.5.1 Effects of an acute exercise on sleep

The effects of an acute exercise bout on sleep have been studied using both experimental and epidemiological studies and also with objective laboratory studies. The meta-analytical studies have reported small positive effects of acute exercise on sleep. (Driver et al. 2000.) Youngstedt et al. (1997) studied the influence of acute exercise on sleep using quantitative synthesis of literature. The results show that acute exercise has only a small or moderate effect on slow wave sleep (SWS), REM sleep, REM latency and total sleep time. Exercise duration and time of the day when exercise is performed have the most remarkable effects on sleep. The authors highlighted that the present analysis was limited to have a focus only on good sleepers. (Youngstedt et al. 1997.)

Epidemiological studies have reported consistently that acute exercise promotes sleep and this positive impact of exercise has also been reported by some objective laboratory studies (Driver et al. 2000). Bunnel et al. (1983) have investigated the effects of an acute exhaustive exercise (50-70 % VO_{2max} to exhaustion) on the following nocturnal sleep in men and women. The results showed that total SWS and especially stage 4 sleep and also SWS duration before the first REM sleep increased significantly. The latency for the first REM sleep period increased while the duration of the first REM sleep period was reduced. Bunnel et al. (1983) concluded that exhaustive exercise promotes the sleep primarily in the early portion of the night. Dworak et al. (2008) also demonstrated increased SWS after an acute high intensity (85-90% HR_{max} to exhaustion) exercise in children. Results showed reduced stage 2 sleep, increased sleep efficacy and reduced sleep onset latencies. REM sleep was not affected. In conclusion, they stated that high intensity exercise promotes sleep, and these results support the theory of homeostatic regulation of sleep. This was the fact, although moderate intensity (60-70% HR_{max}) exercise did not affect sleep variables. (Dworak et al. 2008.)

Horne et al. (1983) have also indicated increased in SWS after high intensity (80 % VO_{2max}) exercise, but similar effects on sleep were seen after a non-exercise, passive heating condition, where body heating was similar to high intensity exercise. Low

intensity exercise did not affect SWS, but it increased the total sleep time. Based on these results, the authors concluded that high and sustained rate of body heating may enhance sleep depth, and exercise may only have a role as a vehicle for these effects (Horne et al. 1983).

Nevertheless, some of the studies have not supported the idea of increased SWS after exercise. Edinger et al. (1993) showed that acute exhaustive exercise did not significantly affect sleep continuity or architecture during the initial periods of sleep or during the entire sleep time in older people.

Excessive exercise might also have negative effects on sleep. Taylor et al. (1997) reported that during the highest training volumes there were a lot of body movements during the sleep, suggesting sleep disturbances. According to the review article of Driver & Taylor (1996), few studies have reported that intensive exercise (ultramarathon or 20 km in 1,5 hours) may disrupt sleep and induce wakefulness during the night particularly in older men. Driver et al. (1994) showed that after an ultra-triathlon sleep was disrupted: Increased wakefulness, delayed and decreased REM-sleep was seen after the ultra-triathlon, however, duration of SWS was not affected. The authors showed that sleep patterns after a control day (when exercise was not performed), after 15 km run and after 42,2 km run did not differ from each other. (Driver et al. 1994.)

In summary and according to latest reviews (Youngstedt 2005, Driver & Taylor 1996, Driver & Taylor 2000), the effects of acute exercise on sleep are positive but modest and might depend on exercise intensity and duration, the time of the day when exercise is performed, and age, gender, and fitness level of the individual. However, the researchers have usually focused on good sleepers and fit athletes, which might have affected the results. (Youngstedt 2005, Driver & Taylor 1996, Driver & Taylor 2000.)

4. 5.2 Effects endurance training and aerobic fitness level on sleep

It has been demonstrated that regular aerobic exercise has a positive impact on sleep quality and sleep architecture. Taylor et al. (1997) have published a study, where they examined the effect of training volume on slow wave sleep (SWS) proportion during the nocturnal sleep in female competitive swimmers. The results showed that certain sleep variables (sleep onset latency, time awake after sleep onset, total sleep time and rapid eye movement sleep) were the same during the start of the season, during the peak training period and after a pre-competition reduction in training. However, SWS proportion of total sleep was very high during the beginning of the season (26 %) and during the peak training period (31 %), when training loads were high, but significantly reduced during the pre-competition period, when training volume was reduced. Thus the need for restorative sleep is reduced when physical demand is reduced, which suggest that greater physical workloads result in greater need for SWS. The authors also demonstrated that type of exercise might have a big role in the relationship of sleep and exercise: aerobic exercise affects sleep, but anaerobic training may not have similar effects. (Taylor et al. 1997.)

Edinger et al. (1993) have studied the associations between fitness level and sleep in older men. Multivariate and univariate analyses indicated that physically fit men in comparison with sedentary men had more continuous and deeper sleep (shorter sleep onset latencies, less wake time after sleep onset, fewer discrete episodes and sleep stage shifts during the first period of sleep, less sleep in stage 1, a higher sleep efficiency and more slow wave sleep) during the night. The authors concluded that regular exercise improves sleep quality, and differences in sleep quality were associated with subjects´ fitness status. (Edinger et al. 1993.)

Tworoger et al. (2003) have investigated the effects of a yearlong moderate-intensity exercise on subjective sleep quality. Postmenopausal (aged 50-75), overweight or obese sedentary women participated in the study. The subjects performed moderate-intensity exercises in the morning hours (from 10:30 to noon) and in the evenings (from 18:00 to 19:30). The results showed that subjects who exercised more than 225 min/week in the

mornings had less problems with falling asleep than those who exercised less than 180 min/week. On the other hand, subjects who practised exercises during the evening more than 225 minutes/week had more troubles falling asleep than those who exercised less than 180 minutes/week. However, better sleep quality, longer sleep duration, less use of sleep medication were seen in subjects whose maximum oxygen uptake over the year increased more than 10%. These beneficial effects of exercise were not seen in subjects whose maximum oxygen uptake over the year increased only 1 % or less. Tworoger et al. (2003) have concluded that exercise might improve sleep quality, and increased fitness was associated with improvements in sleep but these beneficial effects may be dependent on the amount of exercise and the time of the day when exercise is performed.

Regular exercise is beneficial for humans with sleep disorders. Sherrill et al. (1998) have examined the relationship between regular exercise, physical activity and self-reported sleep disorders among randomly selected adults. The results indicated that regular physical activity at least once per week, participation in a regular exercise program or walking at least 6 blocks per day reduced the risk for sleep disorders that disturb the maintenance of sleep. The authors concluded that regular exercise might be useful in the treatment of patients with sleep disorders. (Sherrill et al. 1998.) King et al. (2008) have also demonstrated that the 12-month moderate-intensity exercise program may have positive but modest effects on measured sleep architecture, and subjective aspects of sleep in older adults with mild to moderate sleep complaints. This exercise program was planned according to current physical activity recommendations. The study further showed that subjects who participated in exercise regularly, had significantly less sleep in stage 1, significantly more sleep in stage 2 and had significantly less awakenings during the first period of the sleep cycle than controls, however, SWS was not affected. These results are parallel to the studies mentioned earlier, despite the effects on SWS. (King et al. 2008.)

Even though there is a lot of information that supports the advantageous effect of physical fitness and regular exercise on sleep, there are also conflicting research results. Paxton et al. (1983) have investigated the effect of aerobic fitness on sleep. They had

two groups of subjects. The first one was a group of athletes, which were measured when they were unfit and also when they were physically fit after their training program. The other one was a group of unfit non-athletic subjects. The study showed that the athletes had higher SWS levels and they slept longer than non-athletes. However, the results did not differ in athletes between the seasons when they were unfit and fit. Paxton et al. (1983) proposed that these differences between athletes and non-athletes may be independent of aerobic fitness level. Also Meintjes et al. (1989) demonstrated that a 12-week physical training programme did not result in any changes in the sleep parameters in spite of increases in maximal oxygen uptake and the point of onset of blood lactate in nine unfit women. Physical fitness associated with increase in lean body mass may facilitate sleep. (Meintjes et al. 1989.) In summary, the endurance training and aerobic fitness level might promote the sleep quality, but the evidence is not compelling (Youngstedt 2005, Driver & Taylor 1996, Driver & Taylor 2000).

4.5.3 Exercise variables affecting sleep

Intensity. Many studies have demonstrated that the intensity of exercise might be a powerful mediator between exercise and sleep; the more vigorous exercise, the more beneficial effects it has on sleep (Bunnel et al. 1983, Dworak et al. 2008, Horne et al. 1983). Although too vigorous exercise with long duration may disrupt sleep, there may be a threshold level in terms of intensity and duration, when sleep is disturbed (Driver & Taylor 1996). Youngstedt et al. (1997) have reported that there are no significant differences between low, moderate and high intensity exercises, when beneficial effects of exercise on sleep are considered. This finding is inconsistent with other studies, but suggests that exercise does not necessarily need to be intense to enhance sleep (Youngstedt 2005).

Volume and Duration. Taylor et al. (1997) have reported that high training volume affects restorative sleep much more than training with lower exercise volume: greater physical workloads results greater need for SWS. Youngstedt et al. (1997) have indicated that the duration of acute exercise, in comparison to other variables, has the

highest effect on sleep, although the authors speculated that this association might result from a clearer description of duration or relevant information in the literature (Youngstedt et al. 1997). Low intensity, but long duration (160 min) exercise has also been shown to increase sleep length but not the depth of the sleep (Horne et al. 1983).

Type. Taylor et al. (1997) have demonstrated that the type of exercise might have a substantial role in the relationship of sleep and exercise: aerobic exercise affects sleep, but anaerobic training may not have similar effects. Trinder et al. (1985) have also concluded that type of training, in which athletes engage, has a substantial effect on sleep. Their study shows that endurance trained athletes spent more time in SWS, had shorter sleep onset latencies and slept longer than power trained athletes (Trinder et al. 1985). Although more recently Singh et al. (1997) have reported that 10-week weight training program increased self reported quality of sleep in older depressed men and women. Improvements were seen in quality of life and depression measurements. However, one could not state, whether the significant improvements in sleep are due to weight lifting or other factors like mood and quality of life. (Singh et al. 1997.)

The time of the day. It has been generally recommended to avoid vigorous exercise 3 hours before bedtime to enhance sleep in insomnia patients (Morin et al. 1999). Youngstedt et al. (1997) have also reported that exercise from 4 to 8 hours before bedtime has the most positive effect on sleep compared to exercise performed more than 8 hours or less than 4 hours before bedtime. Nevertheless, Youngdtedt et al. (1999) have reported that vigorous (65-75 % heart rate reserve for 3 hours) exercise, which ended 30 min before bedtime, did not disturb the sleep. There were no significant differences in subjective and objective sleep variables between the exercise treatment and the control treatment, in which subjects were exposed to bright light for three hours. The authors concluded that their results were inconsistent with general opinion. (Youngdtedt et al. 1999.) O´Connor et al. (1998) have also demonstrated that low and moderate intensity exercise from 90 to 30 min before bedtime did not significantly affect sleep onset latency, the number of awakenings, total sleep time or sleep efficiency, even though the moderate intensity exercise increased the core body temperature before and during sleep. However, the authors speculated that because of the small sample size of the

young healthy moderately active men participating in the study, the results may not be generalized to people with sleep disorders, inactive individuals or females (O´Connor et al. 1998).

5 PURPOSE OF THE STUDY, RESEARCH PROBLEMS AND HYPOTHESES

The purpose of the present study was to find out how an acute exercise session, which was performed in accordance with the updated guidelines of American College of Sports Medicine and the American Heart Association, affects the recovery of ANS. The most important target of interest was to study how different exercise intensities affect recovery of HRV immediately after exercise and during sleep. In addition, the present study was designed to investigate how exercise intensity affects subjective and objective sleep quality during the following nocturnal sleep.

The present research problems and hypotheses are:

1. Do acute exercise sessions at two different intensities (60% and 75% of VO_{2max}) affect immediate recovery of HRV after exercise?

   H1: After high intensity exercise, immediate recovery of HRV and especially recovery of HFP is slower than after moderate intensity exercise. According to Martinmäki & Rusko (2007), Kaikkonen et al. (2007), Seiler et al. (2007), and Kaikkonen et al. (2008), immediate recovery of HRV is slower after high intensity exercise compared to moderate and low intensity exercises.

2. Do acute exercise sessions at two different intensities (60% and 75% of VO_{2max}) affect recovery of HRV during sleep?

   H2: Recovery of HRV and especially parasympathetic tone is blunted for several hours after an exhaustive exercise. The post-exercise recovery of HRV happens slowly as a function of time and is dependent of exercise intensity. Recovery of

parasympathetic tone and HR may last several hours when exercise intensity is high and duration long. (Hautala et al. 2001, Bunnell et al. 1983.)

3. Do acute exercise sessions at two different intensities (60% and 75% of VO_{2max}) have effects on biomarkers of stress, especially on salivary IgA and cortisol concentrations?

   H3: The acute exercise will decrease the salivary IgA concentrations and increase salivary cortisol concentration. Mackinnon et al. (1993), Mackinnon & Jenkins (1993), and Tharp & Barnes (1990) have shown that the high intensity acute exercise decreased the concentration of salivary IgA, while Elloumi et al. (2003), Karkoulias et al. (2008), Maresh et al. (2006) and Rudolph & McAuley (1998) have shown that the acute exercise increases the level of salivary cortisol. According to their studies, moderate to high intensity exercises increase cortisol levels significantly above pre-exercise values (Elloumi et al. 2003, Karkoulias et al. 2008, Maresh et al. 2006, Rudolph & McAuley 1998). Besides there seems to be an intensity threshold point, the time when cortisol concentration starts to increase, which exists above 60% but below 80 % of VO_{2max}. (Hill et al. 2008, Maresh et al. 2006.)

4. Do acute exercise sessions at two different intensities (60% and 75% of VO_{2max}) affect objective and subjective sleep quality?

   H4: Acute exercise promotes the sleep in intensity-dependent manner: when exercise intensity increases the sleep quality improves. The intensity of exercise might be a powerful mediator between exercise and sleep: the more vigorous the exercise, the more beneficial effects it has on sleep (Bunnel et al. 1983, Dworak et al. 2008, Horne et al. 1983).

6 METHODS

6.1 Subjects

Sixteen men at the average age of 36 years volunteered to participate in the study (table 1). All of them were healthy non-smokers with a normal body mass (BMI<25) and they were moderately active. The subjects gave a written informed consent to participate in the study and filled in a health questionnaire before the study. They had the right to withdraw from the study at any time. The study was approved by the Ethics Committee of the University of Jyväskylä.

TABLE 1. Background information of the subjects.

Subjects (n=16)	**mean ± sd**
age	36 ± 4
height (cm)	177 ± 8
body mass (kg)	77,5 ± 10,9
BMI	24,8 ± 2,6
VO_{2max} (ml/kg/min)	48,9 ± 3,9

6.2 Study protocol

In the present study, the autonomic regulation of HRV as well as subjective and objective sleep quality, were measured during the night before and after two different exercise sessions. Also biomarkers of stress from salivary samples were measured after both nights' sleeps at home. In addition, acute effects of exercise on HRV and biomarkers of stress from salivary samples were measured in the laboratory right after the exercise session. The protocol was similar in both exercise sessions, even though the exercise intensity was different (see figure 4).

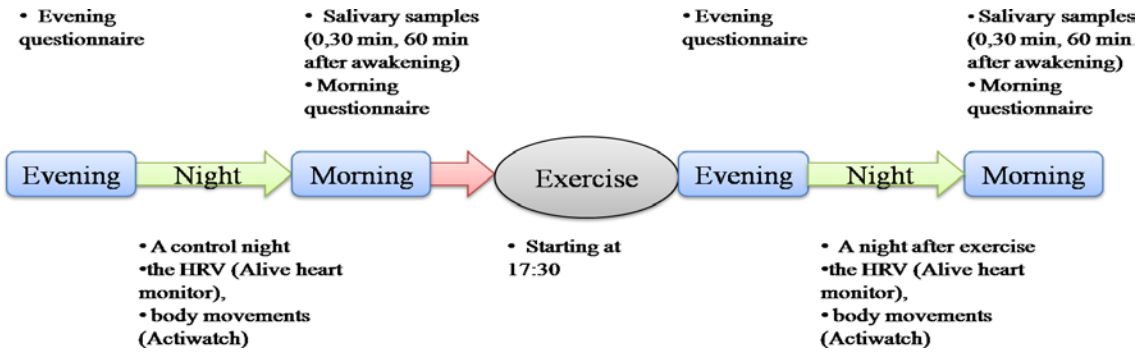


FIGURE 4. Study protocol: The subjects performed two similar exercise sessions with different exercise intensities (60 % of VO_{2max} vs. 75% of VO_{2max}). During the night before and after the laboratory exercise sessions, the autonomic regulation of HRV and sleep quality, were measured.

6.3 Measurements

6.3.1 Graded maximal treadmill test

Before the two exercise sessions, a maximal graded treadmill (Telineryhmä, Kotka, Finland) test was performed to determine individual exercise intensities. The initial test speed of 6 km/h and gradient of 1 % were used in the test. The speed was increased with 1 km/h every 3 min until voluntary exhaustion. In the beginning of the test, the subjects walked, but later they started to run with increasing speed. Breath-by-breath respiratory data (Vmax 229, Sensor Medics, Palo Alto, California, USA) and R-R intervals (RRI) (Suunto T6 wristop computer, Suunto Oy, Vantaa, Finland) were measured during the test. At the end of the each step, fingertip blood samples for lactate analysis were taken. The anaerobic and the respiratory compensation thresholds were determined by blood lactate and respiratory parameters.

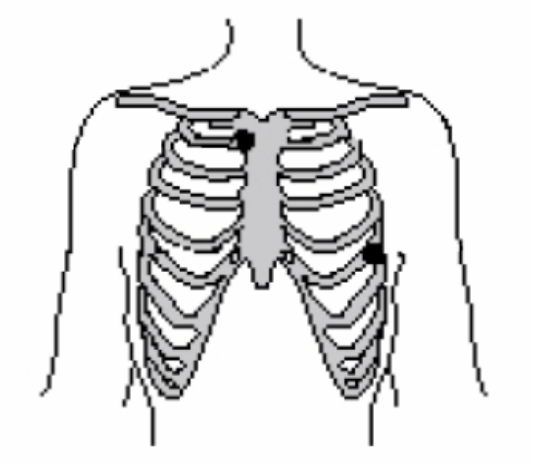
6.3.2 The measurements during the nights at home

During the night before and after the laboratory exercise sessions autonomic regulation of HRV was measured with Alive heart monitor (Alive Technologia, Australia) and

objective sleep quality with the Actiwatch (Cambridge Neurotechnology Ltd, Cambridge, UK, www.camntech.com) at home. The measurements before the laboratory exercises were used as control measurements. The subjects took care of starting the measurements in accordance with verbal and written instructions before they went to bed. The subjects also filled in the questionnaires concerning physical and mental stress and recovery level in the evening before they went to the bed. In addition, right after the subjects woke up in the morning, they filled in a questionnaire about sleep quality and recovery level. Salivary samples were taken immediately, 30 min, and 60 min after awakening. (see figure 4).

Heart rate measurements. Alive heart monitor was used to measure heart rate variables for approximately 8 h during the nights. The device detects R-to-R-peaks from the subject´s ECG signal. Before starting the measurements with Alive heart monitor, two electrodes were placed to the chest; the first one inferior to clavicle right side of the body, and the other one to the axillary line vicinity of sixth costa left side of the body (see figure 5).

FIGURE 5. Locations of electrodes: the first one inferior to clavicle right side of the body, and the other one to the axillary line vicinity of fifth costa left side of the body .



Salivary samples were taken for cortisol and IgA analyses. The subjects gave three samples at home after awakening (immediately after awakening, 30 min and 60 min after awakening). A cotton-chew salivette (Salivette® (Ref 51.1534) Sarstedt , Nümbrecht, Germany) were used to salivary sampling. The subjects chewed salivette

about one minute, which was then placed to plastic tube and stored in a cold place. The samples were frozen in - 80 degrees before the analyses.

Body movement recordings. During the nights, body movements of the subjects were detected with the Actiwatch activity monitoring system (see figure 6). The device records the amount, integration of intensity, and duration of the movement in all directions (maximal sampling frequency = 32 HZ), if the movement is greater than 0.05 g. To avoid gravitational artefacts, the data was low and high –pass filtered with the range of 3-11-Hz. (The Actiwatch Activity Monitoring System: Instructions for Use and Software Manual. Cambridge Neurotechnology). This wristwatch-like computer was placed in the non-dominant wrist just before the bedtime. The epoch interval was set to 0.25 minutes (15 sec.).

FIGURE 6. Actiwatch activity monitoring system.

Self-reported questionnaires. Self-reported questionnaires were used to get information about subjective sentiments. The subjects filled in a questionnaire (morning/-evening questionnaire) concerning subjective sleep quality and recovery level but also physical and mental wellbeing before bedtime and after awakening. They also reported the times when they went to bed and woke up as well as the times when they started and ended the HR recordings.

6.3.3 Exercises

The subjects performed two 30 min exercise sessions in a laboratory at different intensities: moderate intensity (60 % of VO_{2max}) and high intensity (75% of VO_{2max}) exercises. The exercise sessions were performed in counterbalanced order. There was about one week between exercise sessions. The subjects were not permitted to exercise or drink alcohol during the two days before laboratory exercises. The subjects arrived at the laboratory at 5.30 p.m. Firstly, they filled in a questionnaire (pre-exercise questionnaire) concerning physical and mental tiredness and recovery level. That was followed by a body mass measurement and 10 min R-R intervals measurement (RRI) (Alive heart monitor and Suunto t6 wristop computer) at supine rest. Before the measurements with Alive heart monitor, two electrodes were placed to the chest (see figure 4). Before the measurements with Suunto t6 wristop computer, electrodes of the heart rate belt were dampened with water, and the heart belt was tightly placed around the subject´s chest. The heart rate devices were started at the same time.

Before exercise, resting blood and salivary samples were taken. The exercise consisted of a 5-min warm-up and 30-min exercise on the treadmill at the predetermined intensity (60 % or 75% of VO_{2max}). The RPE and RRI were measured during the exercise. Immediately after cessation of running, RRI was measured again for two minutes in a standing position. Subjects were not allowed to speak or move during this period. Blood samples were taken from the fingertip after 2- min recovery period. During the 3-10 min after exercise, the subjects rested in a supine position, and RRI was again measured. After the exercise, the subjects also filled in a questionnaire (post-exercise questionnaire) concerning physical and mental tiredness as well as recovery level, and their body mass were measured. Salivary samples were again taken 15 min and 30 min after the exercise. (see figure 7).

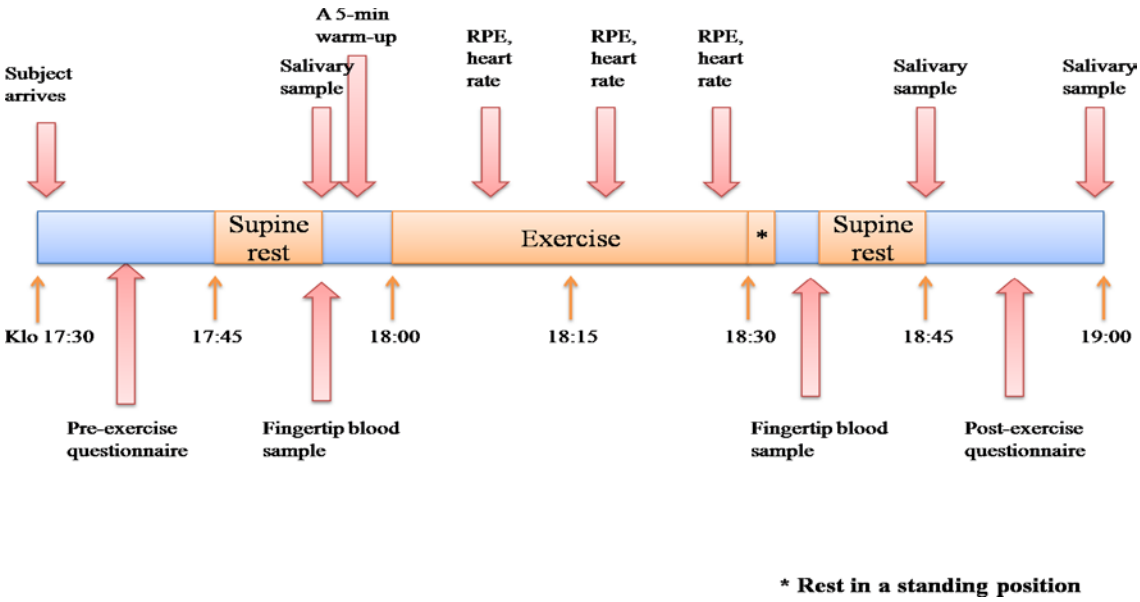


FIGURE 7. The measurements during the exercise sessions.

6.4 Analysis

6.4.1 Heart rate variability analysis

Firstbeat HEALTH (version 3.0.1.0, Firstbeat Technologies Ltd, Jyväskylä, Finland) computer software application was used to analyse the measured R-R intervals. This software analyses different HRV parameters, but also physiological variables describing stress and recovery. From traditional HRV parameters, which the software calculates, HR, root mean square 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) were chosen to this study. HFP, also referred as the respiratory frequency, reflects ventilatory modulation of R-R intervals and is mediated predominately by parasympathetic modulation. So it may provide an index of vagal acivity (Kleiger et al. 2005, Berntson et al. 1997). Also RMSSD describes the vagal activity (Kleiger et al. 2005). LFP is modulated by baroreflexes and chemoreceptor control and has both sympathetic and vagal origin. (Winsley 2002, Berntson et al. 1997.)

Before the analysis, the background information; age, gender, height, weight, smoking and physical activity habits (Jackson et al. 1984), had to be set. According to this information, the software detects subject´s resting and maximal heart rate (ACSM 2001). If the higher maximal heart rate or lower resting heart rate is found during the recording period, the software updates these variables from recorded data. During actual analysis the RRI data was filtered with the artefact detection filter, which corrects falsely detected, missed and premature heartbeats. In order to obtain equidistantly fixed time intervals, the corrected RRI data was re-sampled at the rate of 5 Hz by using linear interpolation. After this preliminary processing, the software calculates HRV variables second by second by using a short-time Fourier Transform method and variables describing respiration rate and oxygen consumption using neural network modeling of data. (Saalasti, 2003). From our statistical analysis data with more than 5% of corrected RRIs were excluded.

MATLAB (MathWorks, version 9.x.x) –program was used to calculate HRV parameters from the analyzed data for the periods of interest. During the laboratory exercises, the mean values for HR and HRV were analyzed for 5 min in supine rest (pre-exercise), first and second recovery minute in standing (post-exercise), and 7 min supine rest (post-exercise) (see figure 7). During the nights, mean values for HR and HRV variables were analyzed for the whole sleep period, 4-hour period starting 30 min after bed time, and hour-by-hour after bed time.

6.4.2 Salivary samples analyses

Cortisol. Cortisol variables from saliva were analysed with Immulite 1000 analyser, which uses chemiluminometric method. The Immulite 1000 Cortisol was used as a reagent. The analytical sensitivity of the method was 5.5 nmol/l, intra-assay precision was 11,5 % at 16,3 ± 1,9 nmol/l, and the total precision was 9,8 % at 12,2 ± 1,1 nmol/l. Absolute cortisol values were analysed from the salivary samples, which were taken in the laboratory and at home in the morning. In addition, from morning salivary samples awakening responses were analysed. The awakening responses were measured with

areas under the cortisol curve (calculated from the awakening to 60 minutes after). These awakening responses were analysed with respect to ground ($AUC_{G0\text{-}30\ min}$, $AUC_{G0\text{-}60\ min}$), with respect to increase after awakening ($AUC_{I0\text{-}30\ min}$, $AUC_{I0\text{-}60\ min}$), and with respect to change after awakening ($AUC_{C0\text{-}30\ min}$ and $AUC_{C0\text{-}60\ min}$). These calculations were made according to Pruessner et al (2003) recommendations.

IgA. IgA variables from saliva were analysed with Konelab XTi 20 analyser, which uses spectrofotometric method. For IgA salivary samples, the method was modified from serum IgA analysis. Konelab IgA Kit no 981669 was used as a reagent. The determination limit, the lowest concentration that can be measured quantitatively, was 0,020 g/l. Intra-assay precision was 2,8 % at 1,14 ± 0,03 g/l and total precision was 8,2 % at 1,14 ± 0,09 g/l. Salivary total protein concentration was analysed with Konelab XTi 20 analyser and Konelab U/CSF Protein Kit no. 981843 was used as a reagent. Detection limit was 0,015 g/l, intra-assay precision 2,0 % at 0,19 ± 0,004 g/l and total precision 3,0 % at 0,19 ± 0,006 g/l. IgA to total protein ratio were analyzed both from laboratory and morning samples.

6.4.3 Movement analysis

The movement analysis were made with Actiwatch activity & sleep analysis 5 -software (version 5.32), which analyses several of the parameters describing sleep by using scoring algorithm. For the analysis, the times when subjects went to bed and woke up, had to be set beforehand. From different sleep parameters, we chose actual sleep time (the amount of sleep), sleep efficiency (the percentage of time spent asleep whilst in bed) and fragmentation index (indicator of restlessness; the addition of percentage of the time spent moving and the percentage of immobility phases of one minute) to this study. (The Actiwatch Activity Monitoring System: Instructions for Use and Software Manual. Cambridge Neurotechnology).

6.4.4 Subjective sleep quality, recovery, and tiredness

In all questionnaires concerning subjective sleep quality, recovery, as well as physical and mental tiredness (evening/morning questionnaire, and pre/post-exercise questionnaire) visual analogue scale (VAS) with the scale 0-100 was used. When sleep quality was asked, the scale was 0=very poor sleep quality, 100=excellent sleep quality, whereas the scale was 0=not at all, 100=very much, when recovery, physical and mental tiredness was asked

6.5 Statistical analysis

Non-parametric Friedman´s two-way analysis of variance (related samples) and Wilcoxon signed rank test (two related samples) were used to compare non-parametric variables between the different conditions. For the parametric variables, comparisons between the different conditions were made with repeated measures ANOVA. The level for statistical significance was determined as $p<0,05$. The star symbols were used to illustrate statistical significance in the figures and tables (*** = $p<0,001$, ** = $p<0,01$, * = $p<0,05$). In the text, results are presented as mean ± SD.

7 RESULTS

7.1 Comparison of exercise modes

After both exercise sessions blood lactate was significantly elevated compared to the pre-exercise values (moderate intensity (MOD): 1,4 ± 0,5 vs. 2,1 ± 0,6 mmol/l, p<0,01, and high intensity (HI): 1,3 ± 0,6 vs. 4,8 ± 1,7 mmol/l, p<0.01). Blood lactate was significantly higher after HI exercise compared to the MOD exercise (2,1 ± 0,6 vs. 4,8 ± 1,7 mmol/l, p<0.01).

The subjective ratings differed after different exercise sessions. RPE was significantly higher after HI exercise compared to the MOD exercise (12,6 ± 1,9 vs. 15,7 ± 1,3, p<0.01). In addition, physical tiredness was significantly increased after HI exercise compared to pre-exercise condition (38,6 ± 18,1 vs. 59,4 ± 19,8, p<0.01), but the MOD exercise did not affect physical tiredness. Furthermore, mental tiredness was significantly lower after MOD exercise compared to the pre-exercise values (36,4 ± 23,9 vs. 24,9 ± 16,7, p<0,05), but the HI exercise did not significantly reduce mental tiredness.

7.2 Acute effects of exercise

7.2.1 HR and HRV

Pre-exercise and exercise. At pre-exercise rest, HFP and LFP did not differ between the MOD and the HI exercises. There were minimal but significant differences in HR and TP values between conditions during pre-exercise rest (p<0,05). During exercises, HR was higher compared to the resting values (p<0,001). In addition, HR was higher during HI exercise than during MOD exercise (173 ± 11 vs. 140 ± 8 bpm, p<0,001) (figure 8). During both exercises, HFP, LFP and TP were significantly reduced as compared to the resting values (p<0,001). In addition, HFP, LFP and TP were significantly lower during

HI exercise than during MOD exercise (HFP: 0,4 ± 0,8 vs. 1,9 ± 1,1 ln[ms²], p<0,01; LFP: 0,0 ± 1,2 vs. 2,5 ± 1,0 ln[ms²], p<0,001; TP: 1,0 ± 0,8 vs. 3,0 ± 0,9 ln[ms²], p<0,001) (figures 9, 10, 11).

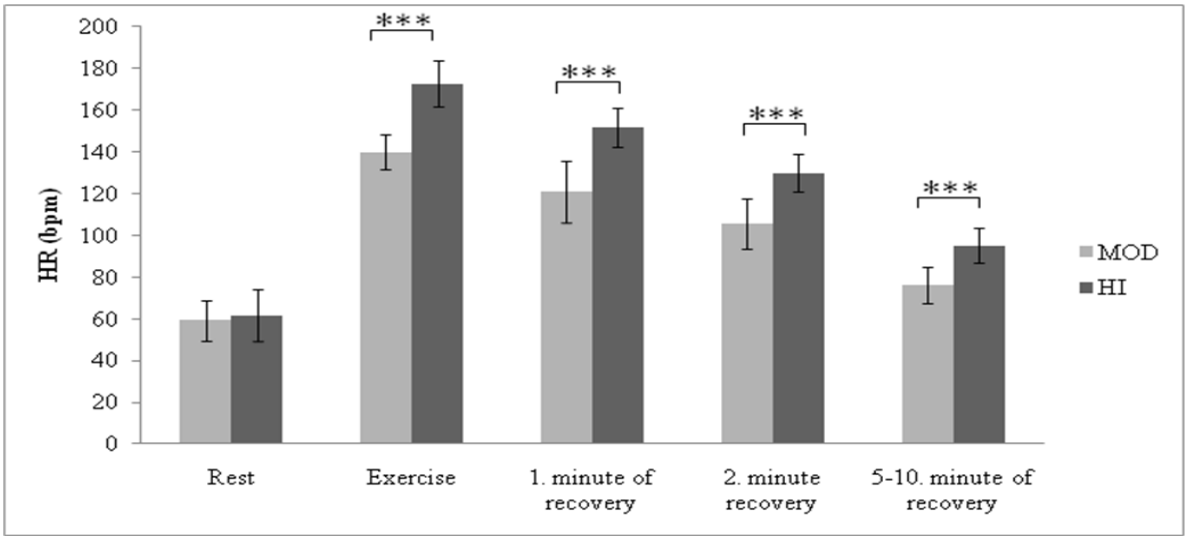


FIGURE 8. HR immediately after MOD and HI exercises. HR was significantly higher after HI exercise vs. MOD exercise (*** = p<0,001). In addition, HR was significantly different within exercise sessions in all time points after both exercises, (p<0,001).

The first minute of recovery. During the first minute of recovery in standing position, HR was higher compared to the resting values after both exercises, but reduced as compared to the values during exercise (p<0,001) (figure 8). In addition, HR was higher after HI exercise than after MOD exercise (152 ± 10 vs. 121 ± 15 bpm, p<0,001) (figure 8). During the first recovery minute in standing position, HFP, LFP and TP were reduced as compared to the resting values after both exercises (p<0,001). Furthermore, after MOD exercise, HFP, LFP and TP were higher than the values during exercise (HFP, LFP: p<0,01, TP: p<0,001). However, after HI exercise, HFP was not increased during the first minute of recovery as compared to the values during exercise. After HI exercise, LFP and TP were higher than the values during exercise (LFP: p<0,001, TP: p<0,01). In addition, HFP, LFP and TP were lower after HI exercise as compared to the MOD exercise during the first recovery minute (HFP: 1,6 ± 0,9 vs. 3,6 ± 1,4 ln[ms²],

p<0,01; LFP: 2,1 ± 1,1 vs. 4,4 ± 1,1 ln[ms²] p<0,001; TP: 2,6 ± 0,9 vs. 4,8 ± 1,2 ln[ms²], p<0,001) (figures 9, 10, 11).

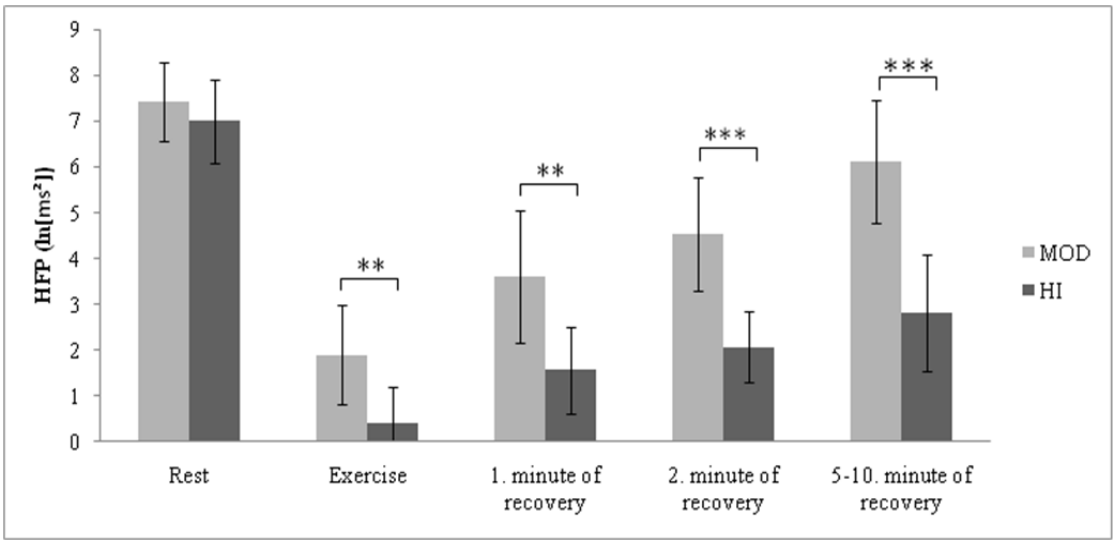


FIGURE 9. HFP immediately after MOD and HI exercises. HFP was significantly lower after HI exercise vs. MOD exercise (*** = p<0,001, **= p<0,01).

The second minute of recovery. During the second minute of recovery in standing position, HR was increased after both exercise sessions as compared to the resting values, but reduced as compared to the values during exercise and the first recovery minute (p<0,001)(figure 8). In addition, HR was higher after HI exercise than after MOD exercise (130 ± 9 vs. 106 ± 12 bpm, p<0,001) (figure 8). Furthermore, after both exercise sessions HFP, LFP and TP were lower than the resting values during the second recovery minute (HI: p<0,001, MOD: HFP: p<0,001, LFP, TP: p<0,01), but higher than the values during exercise (HFP: HI: p<0,01, MOD: p<0,001, LFP: p<0,01, TP: p<0,001). However, HFP did not differ between the first and the second recovery minute after both exercises. LFP after both exercises was increased during the second minute of recovery as compared to values during the first minute of recovery (HI: p<0,01, MOD: p<0,05). During the second minute of recovery, TP was increased after HI exercise compared to the values during the first minute of recovery (p<0,05), but it remained unchanged after MOD exercise. In addition, HFP, LFP and TP were lower after HI exercise than after MOD exercise during the second minute of recovery (HFP:

2,1 ± 0,8 vs. 4,5 ± 1,2 ln[ms²]; LFP: 3,2 ± 1,1 vs. 6,5 ± 1,0 ln[ms²]; TP: 3,5 ± 0,9 vs. 5,9 ± 1,0 ln[ms²], p<0,001) (figures 9, 10, 11).

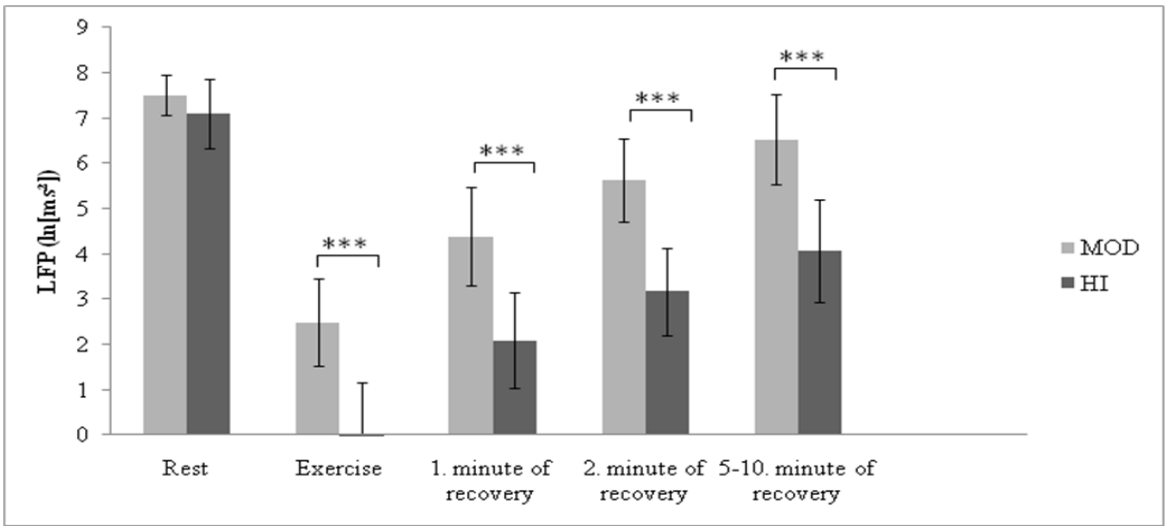


FIGURE 10. LFP immediately after MOD and HI exercises. LFP was significantly lower after HI exercise vs. MOD exercise (*** = p<0,001).

Post-exercise supine rest. During the 5 minute period at supine rest, HR was higher than the pre-exercise resting values after both exercises, but lower than the values during exercise and during the first and the second minutes of recovery (p<0,001) (figure 8). In addition, HR was higher after HI exercise compared to the MOD exercise (95 ± 9 vs. 76 ± 9 bpm, p<0,001) (figure 8). During the 5 minutes rest, HFP, LFP and TP were lower after HI exercises than the resting values (p<0,001), but higher than the values during exercise (HFP: p<0,01, LFP, TP: p<0,001) (figures 9, 10, 11). However, HFP during the 5 minute period of supine rest after HI exercise did not differ compared to the values during the first and the second minute of recovery. During the 5 minutes rest, LFP and TP were higher after HI exercise than the values during the first minute of recovery (LFP: p<0,01, TP: p<0,05), but there were no differences in LFP and TP values between the second recovery minute and the 5 minutes period of supine recovery.

After MOD exercises, during the 5 minutes period of supine recovery, HFP, LFP and TP were not different compared to the pre-exercise resting values, but were higher than

the values during exercise (p<0,001) and the first (HFP: p<0,01, LFP: p<0,05, TP: p<0,01) minutes of recovery (figures 9, 10, 11). HFP was also increased compared to the values during the second minute of recovery (HFP: p<0,05), whereas LFP and TP did not differ from values during second minute of recovery. In addition, HFP, LFP and TP were lower after HI exercise than after MOD exercise during the 5 minute recovery period in supine position (HFP: 2,8 ± 1,3 vs. 6,1 ± 1,4 ln[ms²]; LFP: 4,1 ± 1,1 vs. 6,5 ± 1,0 ln[ms²]; TP: 4,4 ± 1,1 vs. 7,1 ± 1,1 ln[ms²], p<0,001).

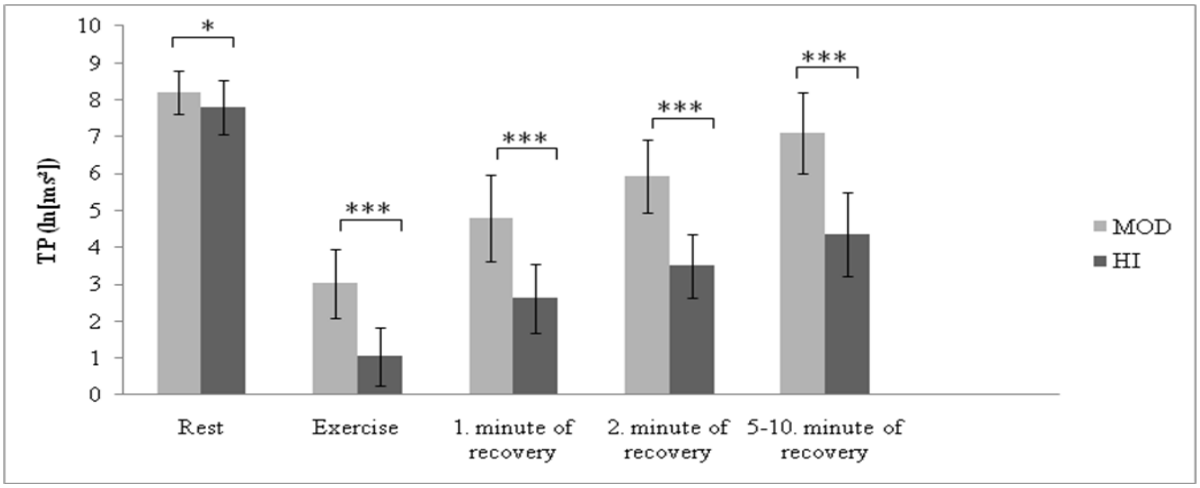


FIGURE 11. TP immediately after MOD and HI exercises. LFP was significantly lower after HI exercise vs. MOD exercise, (*** = p<0,001, * = p<0,05).

7.2.2 Biomarkers of stress in saliva

Cortisol. Before exercises, absolute cortisol concentrations did not differ between the exercises. Cortisol concentration was not affected 15 min and 30 min after MOD exercise, but 15 min after HI exercise, cortisol concentration was higher compared to the pre-exercise value (p<0,05) (table 3). However, there were no differences in cortisol concentration 30 min after HI exercise compared to the pre-exercise value. In addition, absolute cortisol concentration was higher 15 min and 30 min after HI exercise than after MOD exercise (C15: 11,9 ± 8,7 vs. 4,4 ± 3,5 nmol/l, p<0,01; C30: 11,9 ± 9,9 vs. 3,5 ± 2,2 nmol/l, p<0,05) (table 3).

IgA. Before exercises, IgA to protein ratio did not differ between the exercises. It decreased 15 min and 30 min after both exercises compared to the pre-exercise value ($p<0,01$) (table 3). In addition, IgA to protein ratio was lower 15 min after HI exercise compared to the value 30 min after HI exercise ($p<0,05$), whereas there was no difference in IgA to protein ratio between the values after MOD exercise. Moreover, 15 min and 30 min after HI exercise IgA to protein ratio was lower compared to the values after MOD exercise (IP15: 0,12 ± 0,02 vs. 0,16 ± 0,05, IP30: 0,14 ± 0,03 vs. 0,17 ± 0,06, $p<0,05$) (table 3).

TABLE 3. Acute effects of exercise on biomarkers of stress in saliva (mean ± SD). There were significance differences in cortisol values and IgA values between the HI exercise and the MOD exercise (** = $p<0,01$, * = $p<0,05$). There was significance difference in cortisol value and IgA values compared to the pre-exercise values (## = $p<0,01$, # = $p<0,05$).

Cortisol (nmol/l)	**MOD**	***P***	**HI**	***P***
pre-exercise	4,3 ± 3,0		4,0 ± 2,4	
15 min after	4,4 ± 3,5		11,9 ± 8,7	**, #
30 min after	3,5 ± 2,2		11,9 ± 9,9	*
IgA to protein ratio				
pre-exercise	0,23 ± 0,07		0,21 ± 0,07	
15 min after	0,16 ± 0,05	##	0,12 ± 0,02	*, ##
30 min after	0,17 ± 0,06	##	0,14 ± 0,03	*, ##

7.3 Effects of exercise on autonomic regulation of HRV during sleep

Whole sleep. When HRV variables were analyzed for the whole sleeping period, HR did not differ between the control nights. After both exercises, HR was higher compared to the control night (MOD: 51 ± 7 vs. 50 ± 5 bpm, $p<0,05$, HI: 56 ± 8 vs. 52 ± 7 bpm, $p<0,05$). In addition, HR was higher after HI exercise than after MOD exercise (56 ± 8 vs. 51 ± 7 bpm, $p<0,01$). RMSSD did not differ between the control nights. RMSSD was not affected after MOD exercise, but after HI exercise RMSSD was significantly reduced compared to control ($p<0,05$). There was also significant difference in RMSSD

during sleep after HI and MOD exercise (52,0 ± 23,0 vs. 64,1 ± 21,6 ms, p<0,05). HFP, LFP, and TP did not differ during control nights or nights after exercises.

4-hour period. During the first four hours of the sleep, HR, RMSSD, HFP, LFP and TP were not different between the two control nights (table 4). However, HR was higher after both exercise sessions (MOD: 53 ± 6 vs. 50 ± 5 bpm, p<0,05, HI: 57 ± 8 vs. 52 ± 6 bpm, p<0,001) than during control nights. In addition, HR was higher after HI exercise compared to the MOD exercise during the 4-hour period of sleep (57 ± 8 vs. 53 ± 6 bpm, p<0,05) (table 4). During the 4-hour period, RMSSD was lower after HI exercise compared to the control night (51,0 ± 25,6 vs. 64,8 ± 34,0 ms, p<0,05), but there was no difference in RMSSD after MOD exercise compared to the control night. In addition, there was no difference between the HI and the MOD exercises in RMSSD during the 4-hour periods of sleep. HFP after exercises did not differ from HFP during control nights at the 4-hour periods. LFP did not differ between the 4-hour periods after exercise sessions and control nights. However, LFP was decreased after HI exercise compared to the MOD exercise (7,8 ± 0,8 vs. 8,1 ± 0,9 ln[ms²], p<0,05). The MOD exercise did not affect TP during the first 4 hours of sleep. However, after HI exercise TP was significantly lower compared to the control values (8,4 ± 0,8 vs. 8,7 ± 0,7 ln[ms²], p<0,05) and the MOD intensity exercise (8,4 ± 0,8 vs. 8,7 ± 0,8 ln[ms²], p<0,01) (table 4).

TABLE 4. HR, RMSSD, HFP, LFP and TP during the first four hours of the sleep (average ± SD). There were significant difference between the exercises in HR (* = p<0,05), LFP (* = p<0,05) and TP (** = p<0,01). In addition, HR was significantly higher after both exercise compared to the control (### = p<0,001, # = p<0,05) and RMSSD and TP were significantly lower compared to the control values after HI exercise (# = p<0,05).

	MOD	***P***	**HI**	***P***
HR (bpm)				
Control	50 ± 5		52 ± 6	
Exercise	53 ± 6	#	57 ± 8	*,###
RMSSD (ms)				
Control	70,3 ± 32,8		64,8 ± 34,0	
Exercise	64,5 ± 35,2		51,0 ± 25,6	#
HFP ln[ms²]				
Control	7,9 ± 0,9		7,7 ± 0,9	
Exercise	7,8 ± 0,9		7,3 ± 1,0	
LFP ln[ms²]				
Control	8,1 ± 0,6		8,0 ± 0,6	
Exercise	8,1 ± 0,8		7,8 ± 0,8	*
TP ln[ms²]				
Control	8,8 ± 0,7		8,7 ± 0,7	
Exercise	8,7 ± 0,8		8,4 ± 0,8	**,#

Hour-by-hour of early sleep. When HRV variables were analyzed hour by hour during the 4-hour period each night, there were no differences in HR, RMSSD, HFP, LFP and TP between the control nights. During the first hour, HR was higher after HI exercise compared to the control night (63 ± 10 vs. 57 ± 8 bpm, p<0,05) and to the MOD exercise (63 ± 10 vs. 56 ± 9 bpm, p<0,05) (figure 12). HR was not increased after MOD exercise compared to the control night. In addition, RMSSD was lower after HI exercise compared to the control (41,3 ± 21,0 vs. 55,37 ± 17,4 ms, p<0,05) and to the MOD exercise (41,3 ± 21,0 vs. 53,6 ± 25,4 ms, p<0,05) (figure 13). Furthermore, HFP was lower after HI exercise compared to the MOD exercise (7,3 ± 7,0 vs. 7,8 ± 7,5 ln[ms²], p<0,05). However, there were no differences in HFP between the control and the

exercise nights. During the first hour, TP was lower after MOD exercise than after control (8,7 ± 7,6 vs. 8,8 ± 7,6 ln[ms²], p<0,05), but did not differ during the night after MOD exercise compared to the control night. There were no differences in TP between the exercise nights.

During the second hour, HR was higher after both exercises compared to the control values, p<0,05. In addition, HR was higher after HI exercise compared to the MOD exercise (60 ± 9 vs. 54 ± 8 bpm, p<0,05) (figure 12). Furthermore, RMSSD was lower after HI exercise compared to the control (45,5 ± 26,7 vs. 57,2 ± 22,6 ms, p<0,05) (figure 13) and also lower compared to the MOD exercise, but the difference was not significant (p=0,05). RMSSD was not decreased after MOD exercise compared to the control night.

During the third hour of sleep, HR was higher after HI exercise compared to the control night (58 ± 8 vs. 53 ± 7 bpm, p<0,05) and to the MOD exercise (58 ± 8 vs. 53 ± 6 bpm, p<0,05) (figure 12). HR was not significantly increased after MOD exercise compared to the control night. During the third hour of sleep, RMSSD was lower compared to the control, but the difference was not significant (p=0,05) (figure 13).

During the fourth hour of sleep, HR was higher after HI exercise compared to the control night (57 ± 8 vs. 52 ± 7 bpm, p<0,05) and to the MOD exercise (57 ± 8 vs. 51 ± 6 bpm, p<0,05) (figure 12). HR was not increased after MOD exercise compared to the control night. During the fourth hour of sleep, there were no differences in RMSSD between the nights. During the second, third and fourth hours of sleep there were no differences in HFP, LFP and TP between the control and the exercise nights.

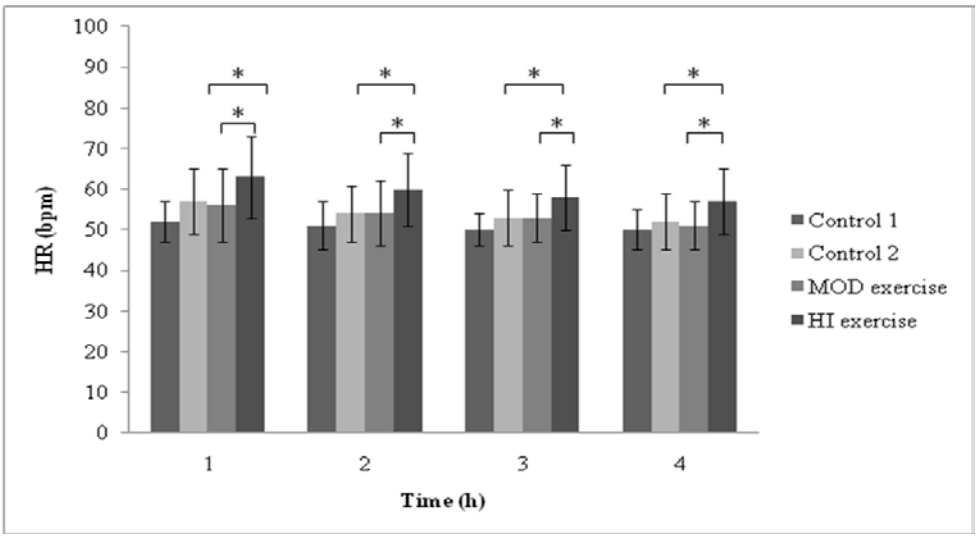


FIGURE 12. HR hour-by-hour of early sleep. After HI exercise, HR was significantly higher compared to the control and to the MOD exercise during the first hours of sleep (* = p<0,05).

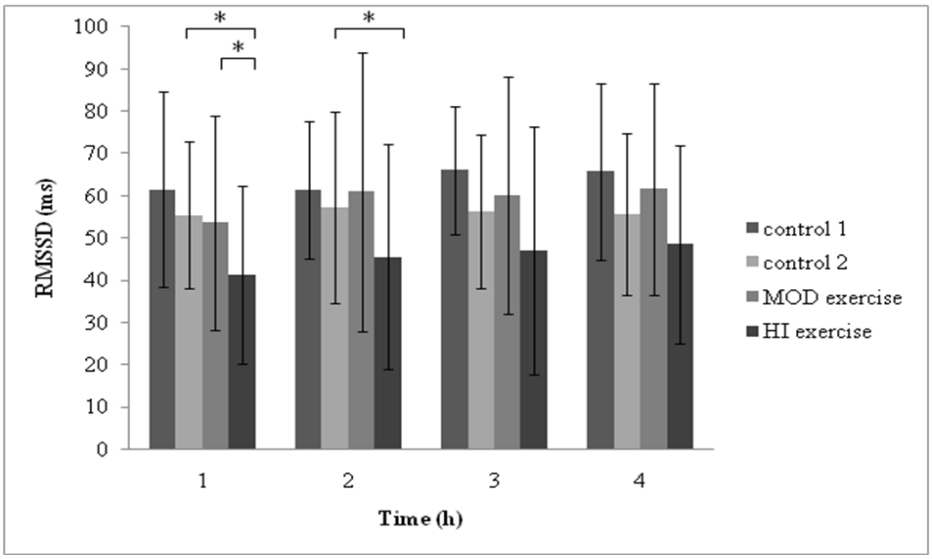


FIGURE 13. RMSSD hour-by-hour of early sleep. During the first hour, RMSSD was significantly lower compared to the control (* = p<0,05) and to the MOD exercise (* = p<0,05). During the second hour, RMSSD was significantly lower compared to the control (* = p<0,05).

7.4 Effects of exercise on morning responses of stress biomarkers in saliva

Cortisol. There were no differences in absolute cortisol concentrations between the control mornings. After MOD exercise, absolute cortisol concentrations immediately, 30 min and 60 min after awakening did not differ from the control values. However, after HI exercise, absolute cortisol concentration was lower 30 min after awakening compared to the control ($p<0,05$) (figure 14), but not different immediately and 60 min after awakening compared to the control. In addition, absolute cortisol concentration was lower 30 min and 60 min after awakening compared to the corresponding values after MOD exercise (C30: 15,4 ± 6,3 vs. 21,3 ± 7,1 nmol/l, C60: 13,2 ± 7,3 vs. 19,6 ± 5,2 nmol/l, $p<0,01$) (figure 14).

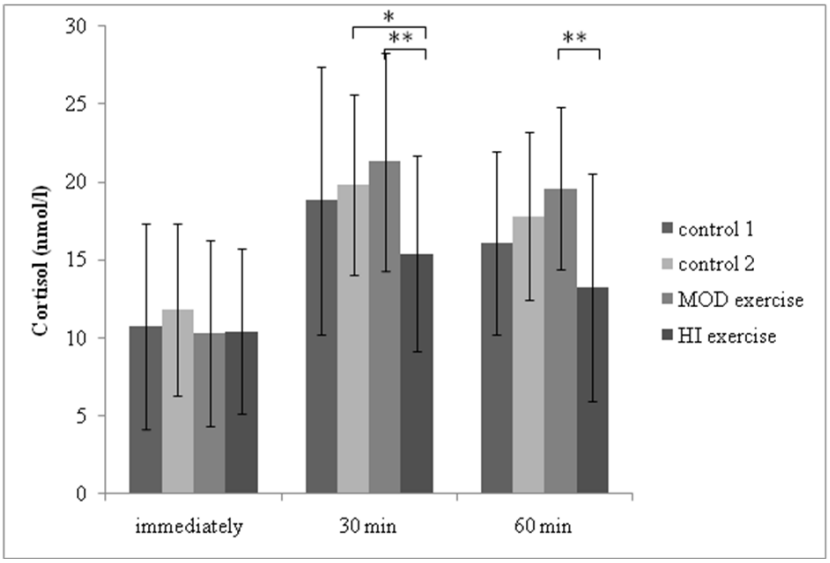


FIGURE 14. Absolute cortisol values immediately, 30 min and 60 min after awakening. After HI exercise, 30 min after awakening, cortisol concentration was significantly lower compared to the control (* = $p<0,05$). In addition, after HI exercise, 30 min and 60 min after awakening, cortisol concentrations were significantly lower compared to the MOD exercise (* = $p<0,05$).

After HI exercise, $AUC_{G0\text{-}30\ min}$ and $AUC_{G0\text{-}60\ min}$ were lower than after MOD exercise ($AUC_{G0\text{-}30\ min}$: 13,2 ± 5,9 vs. 16,3 ± 7,0, $p<0,01$, $AUC_{G0\text{-}30\ min}$: 27,4 ± 11,1 vs. 35,3 ±

11,4, p<0,001). In addition, $AUC_{I0\text{-}30\ min}$ and $AUC_{C0\text{-}30\ min}$, were lower than after MOD exercise ($AUC_{I0\text{-}30\ min}$: 3,3 ± 3,0 vs. 4,9 ± 3,1, p<0,05, $AUC_{C0\text{-}30\ min}$: 3,1 ± 3,3 vs. 4,9 ± 3,1, p<0,05). However, there were no differences in $AUC_{G0\text{-}30\ min}$, $AUC_{G0\text{-}60\ min}$, $AUC_{I0\text{-}30\ min}$, $AUC_{I0\text{-}60\ min}$, $AUC_{C0\text{-}30\ min}$ and $AUC_{C0\text{-}60\ min}$ between the control mornings. In addition, there were no differences in $AUC_{G0\text{-}30\ min}$, $AUC_{G0\text{-}60\ min}$, $AUC_{I0\text{-}30\ min}$, $AUC_{I0\text{-}60\ min}$, $AUC_{C0\text{-}30\ min}$ and $AUC_{C0\text{-}60\ min}$ after both exercises compared to the control mornings.

IgA. There was small but significant difference in IgA to protein ratio immediately after awakening between the control mornings (p<0,01), but there were no differences in IgA to protein ratio between the control mornings at 30 min and 60 min after awakening. IgA to protein ratio was higher after HI exercise immediately after awakening compared to the control (p<0,01) (figure 15), but not different 30 min and 60 min after awakening compared to the control. After MOD exercise, IgA to protein ratio immediately, 30 min and 60 min after awakening did not differ from the control values. In addition, there were no differences in IgA to protein ratio after awakening between the exercises.

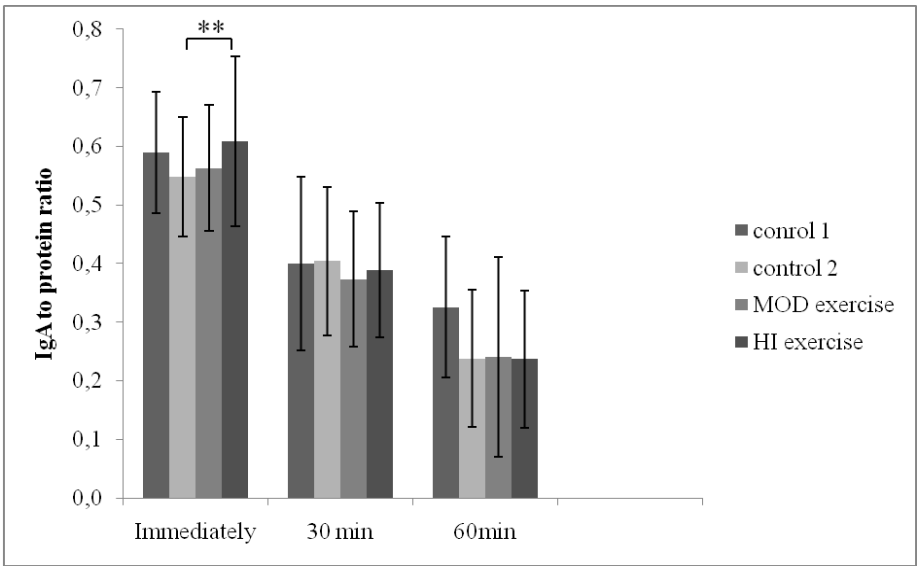


FIGURE 15. IgA to protein ratio immediately, 30 min and 60 min after awakening. IgA to protein ratio was significantly higher after HI exercise immediately after awakening compared to the control (**p<0,01).

Every morning, IgA to protein ratio decreased along with the time after awakening (immediate vs. 30 min: p<0,01, immediate vs. 60 min: p<0,001, 30 min vs. 60 min, p<0,01), but the difference between 30 min and 60 min after awakening was not significant after control night 1 and MOD exercise. The differences in a IgA to protein ratio between different time points were the most significant after HI intensity exercise (p<0,001).

7.5 Effects of exercise on subjective and objective sleep quality

7.5.1 Objective

Actual sleep time did not differ between the control nights. Actual sleep time was significantly longer after HI exercise compared to the control situation (7:29 ± 0:40 vs. 6:56 ± 0:41 h:min, p<0,01), whereas, the MOD exercise did not significantly increase actual sleep time compared to the control night. There were no differences in actual sleep time, when the HI and the MOD exercises were compared. There were no significant differences in sleep efficiency or fragmentation index between the nights.

TABLE 4. Actual sleep time during the nights. Actual sleep time was longer after HI exercise compared to the control night (** = p<0,01).

Actual sleep time	**h:min ± SD**	***P***
control 1	7:02 ± 0:55	
control 2	6:56 ± 0:41	
MOD exercise	7:09 ± 0:44	
HI exercise	7:29 ± 0:40	**

7.5.2 Subjective

Subjective ratings of sleep quality. According to subjective ratings, there were no differences falling asleep between the control nights. It took less time to fall asleep after

MOD exercise compared to the control night (19,1 ± 13,9 vs. 37,0 ± 31,9 min, p<0,05). However, there were no differences falling asleep after HI exercise compared to the control or to the MOD exercise. When subjects were asked, how well they fall asleep compared to the usual falling asleep, the nights did not differ from each other.

Most of the subjects felt that they slept more sufficient after both exercise sessions compared to the control nights (MOD: 75 vs. 31, HI: 63 vs. 38 %, p<0,05). However, sufficiency of sleep did not differ between the exercises. According to subjective ratings, sleep quality did not differ between the control nights. In addition, the MOD exercise did not affect subjective sleep quality compared to the control. However, sleep quality was higher after HI exercise compared to the control night (71,3 ± 18,7 vs. 53,0 ± 23,5, p<0,05) (figure 16). There were no differences in sleep quality between the HI and the MOD exercises. When subjects were asked, how well they sleep compared to the usual sleep, the control nights did not differ from each other. After both exercises, number of subjects, who felt that they slept worse than usual, was lower compared to the control nights (MOD: 31 vs. 88 %, p<0,01, HI: 19 vs. 69 %, p<0,05). There were no differences in how well subjects slept compared to the usual sleep between the HI and the MOD exercises.

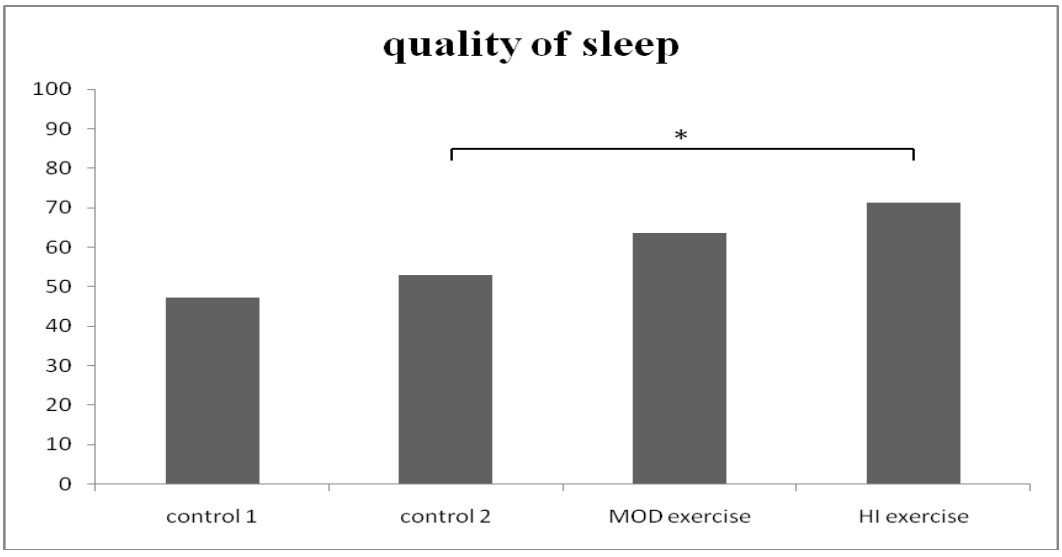


FIGURE 16. Subjective sleep quality after different conditions. Sleep quality was significantly higher after HI exercise compared to the control night (* = p<0,05).

Subjective ratings of physical and mental recovery and stress level. There were no differences between the evening and the morning subjective ratings concerning overall recovery, physical and mental recovery, stress or relaxation level. Tiredness was decreased between the evening and the morning value after HI exercise (64,4 ± 15,0 vs. 44,7 ±26,1 mm, $p<0,05$). There were no differences in subjective ratings concerning mental recovery and stress level in the evening after both exercises. In the evening after HI exercise, physical recovery was lower compared to the control evening (57,7 ± 16,0 vs. 70,9 ± 10,9 mm, $p<0,05$) and to the evening after MOD exercise (57,7 ± 16,0 vs. 67,4 ± 12,5 mm, $p<0,05$). Also overall recovery was lower in the evening after HI exercise than the control evening (14 ± 2 vs. 15 ± 1, $p<0,05$). Relaxation did not differ in the evenings after exercises compared to the control values, but after HI exercise relaxation was higher than after MOD exercise (64,9 ± 13,5 vs. 56,5 ± 17,1 mm, $p<0,05$). In addition, after HI exercise change between the evening and the morning relaxation was significantly higher compared to the control night (7,8 ± 19,8 vs. -3,6 ± 26,2 mm, $p<0,05$). In the evening after HI exercise, tiredness was higher compared to the control evening (64,4 ± 15,0 vs. 50,5 ± 16,8 mm, $p<0,01$) and to the evening after MOD exercise (64,4 ± 15,0 vs. 47,6 ± 15,0 mm, $p<0,05$). The MOD exercise did not affect these subjective ratings.

8 DISCUSSION

The main purpose of the present study was to find out how an acute exercise session affects the recovery of ANS. More specifically, we wanted to study how different exercise intensities affect recovery of HRV immediately after exercise and during sleep. In addition, the present study was designed to investigate how exercise intensity affects subjective and objective sleep quality during the following nocturnal sleep.

The main findings of the present study revealed that HFP started to increase during the first recovery minute after moderate intensity exercise, but during the second minute after high intensity exercise. In addition, HRV returned to baseline values in 10 minutes after moderate intensity exercise, but remained lower during this immediate recovery period after high intensity exercise. Moreover, HRV was lower after high intensity exercise than moderate intensity exercise during the immediate recovery period. This same trend was also seen during sleep. HRV was not fully recovered to resting values after high intensity exercise during the first hours of sleep. In addition, actual sleep time was longer and subjective sleep quality better after high intensity exercise.

8.1 Comparison of exercise modes

The blood lactate and RPE acted as expected: blood lactate and RPE were higher after both exercises as compared to the control, and also higher after high intensity exercise compared to the moderate intensity exercise. Furthermore, physical tiredness was increased after high intensity exercise, but not after moderate intensity exercise, whereas mental tiredness was decreased after moderate intensity exercise, but not after high intensity exercise. In addition, just before the bedtime subjective physical recovery and overall recovery were lower and subjective tiredness was higher after high intensity exercise compared to the control. These results indicate that the exercises were performed according to research plan, and the high intensity exercise was really higher intensity exercise than the moderate intensity exercise.

8.2 Immediate recovery of heart rate variability

During the recent years, the recovery of autonomic nervous system, and especially, the recovery of HRV, has been under research but still the findings are anything but clear. Especially, how fast the vagal reactivation occurs after different exercises is under question. Our hypothesis was that after high intensity exercise, the immediate recovery of HRV, and especially, recovery of HFP is slower than after moderate intensity exercise (Martinmäki & Rusko 2007, Kaikkonen et al. 2007, Seiler et al. 2007, Kaikkonen et al. 2008). The findings of the present study support the hypothesis and are consistent with the previous studies.

According to Tulppo et al. (1998), HFP almost disappears when exercise intensity reaches 50-60 % of VO_{2max}. The present study supports that finding, because at the end of both exercises (moderate intensity = 60 % of VO_{2max}, high intensity = 75 % of VO_{2max}), there was only little HRV left. In the present study, during the first minute of recovery after moderate intensity exercise HRV variables started to increase towards resting values. However, after high intensity exercise HPF did not differ significantly from the respective values during exercise, even though LFP and TP started to increase. HFP started to increase during the second recovery minute after high intensity exercise. In addition, HRV was significantly lower after high intensity exercise compared to the moderate intensity exercise during the whole immediate recovery period. Moreover, HRV did not reach the pre-exercise values during the 10 minutes immediate recovery after high intensity exercise, whereas HRV returned to the baseline values in ten minutes after moderate intensity exercise. These findings are consistent with Martinmäki & Rusko (2007), Kaikkonen et al. (2007) and Kaikkonen et al. (2008), although the protocol and methods were slightly different. In addition, the findings of the present study are parallel with the studies of Terziotti et al. (2001) and Seiler et al. (2007), who state that the recovery of HRV is delayed after high intensity exercise compared to the low and to the moderate intensity exercises.

In the study of Martinmäki & Rusko (2007), HFP increased similarly to our study: HFP started to increase during the first minute of recovery after low intensity (29 % maximal

power) and during the second minute of recovery after high intensity (61 % of maximal power) exercise. In their study, 10 minutes bicycle exercise and short-time Fourier transform method were used. However, according to their study, HRV returned to baseline values in few minutes after both exercises, whereas in the present study recovery of HRV took longer after high intensity exercise. The difference might be due to the different exercise mode, and partly because of the overall loads of the exercises were different.

In the study of Kaikkonen et al. (2007), where different intensity treadmill exercises (two low-intensity: 3500 m and 7000 m at 50 % of the velocity of VO_{2max}, two moderate-intensity: 3500 m and 7000 m at 63 % of the velocity of VO_{2max}, one high-intensity exercise: 3500 m at 74 % of the velocity of VO_{2max}) and short-time Fourier transform method were used, an increase in HFP was seen after the fourth recovery minute after moderate intensity exercise and at the end of 30 minutes recovery period after high intensity exercise. Why was the recovery of HRV slower in their study as compared to our study, even though the protocol was almost the same? One possible explanation could be that in spite of similarities between exercises the overall loads of the exercises were different. On the other hand, the subjects in the study of Kaikkonen et al. (2007) were women. In another study of Kaikkonen et al. (2008), the exercise intensities were higher, the subjects were male athletes and the short-time Fourier transform method was used. Also in their study, the recovery of HRV was slower than in our study, which can be explained by higher exercise intensities. According to these previous studies and the present study, the vagal reactivation is quite fast after exercise, and the increased intensity of the exercise results in slower recovery of HRV.

As shown above, fast changes in the cardiac function occur within few minutes after exercise. During this immediate recovery, parasympathetic activity increases, which is related to different factors: According to Miles et al. (1984), the fast changes in cardiac output, function and contractility after exercise are associated with reduction in HR. In addition, the loss of central command and activation of baroreflexes after exercise is related to the gradual reduction of HR (O´Leary 1996). These changes confirm the

gradual increase in the vagal activity, as well as, withdrawal of the sympathetic activity after exercise (Arai et al. 1989, Oida et al. 1997).

8.3 Heart rate variability recovery during the sleep

The recovery of HRV during sleep after exercise has not been extensively studied. Especially, it would be interesting to see, how long it takes for the HRV to return to baseline values after different exercises, and whether that recovery need so much time that cardiovascular function is still disrupted during sleep. Our hypothesis was that after high intensity exercise, recovery of HRV and, especially, parasympathetic tone is blunted for several hours and the effects of exercise are seen during sleep. According to Mourot et al. (2004), Hautala et al. (2001) and Bunnel et al. (1983), the recovery of parasympathetic tone and HR may last several hours when the exercise intensity is high and duration long.

The findings of the present study are partly parallel with these previous studies. We found that during the whole night, HR was significantly higher after high intensity exercise compared to the control and to the moderate exercise, and RMSSD significantly lower compared to the control. In addition, the same trend was seen in HR and RMSSD, when HRV variables were analysed for 4-hour-periof of early sleep, but then also TP was significantly lower compared to the control night after control day and to the moderate exercise day. More specially, when HRV variables were analysed hour by hour during the 4-hour period of early sleep, HR was significantly higher during each sleeping hour after high intensity exercise compared to the control and to the moderate intensity exercise. RMSSD was lower after high intensity exercise compared to the control and to the moderate intensity exercise during the first hour and lower compared to the control during the second hour of sleep. Although, the other HRV variables were not significantly affected during the sleep after high intensity exercise, higher HR and lower RMSSD, might suggest that autonomic nervous system function is still disrupted during sleep, especially during early sleep. Mourot et al. (2004) did similar conclusions in their study, in which the long-term effects of constant and

interval exercises on HRV were measured. They stated that persistent tachycardia and reduced TP values 24 and 48 h after exercises, indicated that cardiovascular functions were still disturbed (Mourot et al. 2004). Also Hautala et al. (2001) studied the long-term changes in cardiac autonomic regulation after prolonged exercise. They found that high frequency spectral component was lower during the twenty-four hours after prolonged exercise compared to the control situation. In addition, Bunnel et al. (1983) found that HR was significantly higher during the sleep after exhaustive exercise compared to the control. Even though, in these previous studies the exercise intensity or duration was higher than in the present study, the results are in agreement. It seems that 30 min high intensity exercise at 18:00 is enough to disturb the regulation of HRV during sleep, especially during the first hours of sleep.

The disturbed recovery of HRV during sleep might result from the higher sympathetic drive, which is needed to maintain sufficient blood flow. According to Halliwill et al. (1996), systolic and diastolic blood pressure decrease significantly after exhaustive exercise, partly because of reduced sensitivity of arterial blood vessels to sympathetic stimulation. On the other hand, the left ventricular systolic function and the cardiac filling patterns may be altered after exhaustive exercise (Douglas et al. 1987, Whyte et al. 1999). Higher sympathetic drive may be necessary to compensate reduced cardiac function and to maintain effective cardiac output, blood pressure and blood flow (Hautala et al. 2001). When hemodynamics is recovered to normal, sympathetic activity decreases and vagal dominance returns. However, in the present study blood pressure and cardiac function were not measured. Due to that, the causes of disturbed recovery of HRV during sleep can only be speculated.

8.4 Biomarkers of stress

IgA to protein ratio and cortisol concentration were measured to find out how stressful the exercises were. According to literature, cortisol concentration will increase after exercise, if exercise intensity is above 60% of VO_{2max} (Hill et al. 2008, Maresh et al. 2006). The present study supports these previous studies. 15 min after high intensity

exercise, where the intensity was 75 % VO_{2max}, cortisol concentration was significantly higher compared to the pre-exercise values, whereas after moderate intensity exercise, where the intensity was 60 % VO_{2max}, cortisol concentration was not affected. It returned to the baseline in 30 min after high intensity exercise. The present finding and previous results suggest that the acute exercise increases cortisol level (Elloumi et al. 2003, Karkoulias et al. 2008, Maresh et al. 2006, Rudolph & McAuley 1998; Thomas et al 2009), and that this increase is related to the exercise intensity (Hill et al. 2008, Maresh et al. 2006, Rudolph & McAuley 1998).

The effects of exercise on cortisol values were also seen in the absolute cortisol concentration 30 min after awakening. Cortisol concentration 30 min after awakening was lower after high intensity exercise compared to the control and to the moderate intensity exercise. However, there were no differences in cortisol awakening responses, which were measured with the areas under the cortisol curve calculated from the awakening to 60 minutes after. According to the literature, cortisol secretion is the strongest in the morning (Bartels et al. 2003), and cortisol concentration has been observed to increase about 50-160 % during 30 min after awakening (Pruessner et al. 1997, Glow et al. 2004). This cortisol awakening response is a part of the natural circadian cycle of cortisol secretion (Clow et al. 2004). However, according Pruessner et al. (1999) elevated awakening cortisol level compared to the repeatedly measured fixed time awakening cortisol level indicates the acute stress response. In view of previous literature, the results of present study may suggest that the high intensity exercise is enough to disturb the cardiac autonomic regulation during early sleep, but not stressfull enough to increase cortisol concentration after awakening. In addition, there were no differences in the subjective ratings concerning stress level between the situations. This notion together with the lower cortisol concentration 30 min after awakening after high intensity exercise may suggest that the sleep after high intensity exercise had been sufficient enough for the overall recovery and sterling enough to restore the homeostasis.

In the present study, IgA to protein ratio was decreased after both exercises, and the decrease were higher after high intensity exercise compared to the moderate intensity

exercise. These findings support the hypothesis, that the acute exercise session decreases salivary IgA level. Even though in the previous studies the protocol has been slightly different, the findings of the present study are parallel with those previous studies (Mackinnon et al. 1993, Mackinnon & Jenkins 1993, Tharp & Barnes 1990). However, some previous studies have shown slightly different results. According to Allgove et al. (2008), the moderate and the high intensity exercise will increase salivary IgA secretion. Also Sari-Sarraf et al. (2008) stated that the moderate intensity exercise increase salivary IgA concentration. In the study of Allgove et al. (2008), salivary flow rate was not affected, whereas in the study of Sari-Sarraf et al. (2008) salivary flow rate was significantly reduced. This reduction may explain the mentioned findings (Sari-Sarraf et al. 2008). In the present study, IgA to protein ratio was chosen to describe IgA concentration, because salivary flow rate was not measured. The total amount of salivary protein depends on salivary flow rate, so when IgA concentration is compared to the total protein concentration, the differences in salivary flow rate was taken in considerations. However, the present result cannot be compared to the study of Allgove et al. (2008), because they measured IgA secretion.

In the view of most previous findings and the results of present study, the moderate to high intensity exercise is enough to decrease salivary IgA level, and the amount of decrease depends on exercise intensity. This decrease may decrease the immune responsiveness to viruses (Steerenberg et al. 1997, Mackinnon et al. 1993, Mackinnon & Jenkins 1993, Tharp & Barnes 1990). However, the effects of acute physiological stress as exercise may be different compared to an acute mental stress. Benhanm (2007) has indicated that IgA levels increased rapidly as a reaction to the acute mental stress and continued to increase during mental task. However, these changes in salivary IgA concentration were short lived, and the high level of IgA returned baseline in few minutes. (Benhamn 2007).

In the morning after the high intensity exercise immediately after awakening IgA to protein ratio was significantly higher compared to the control morning. However, there were no differences in other time points after awakening after high intensity exercise compared to the control. According to the literature, the concentration of salivary IgA is

higher in the morning than the evening hours (Dimitriou et al. 2002). However, it is not clear how IgA level is affected in the morning after exercise, when compared to the repeatedly measured fixed time awakening IgA level. In the view of the present results, one could suggest that the high intensity exercise is not stressful enough to affect the morning IgA level.

8.5 Objective and subjective sleep quality

It is widely accepted that exercise has many positive effects on health, and due to that recent research interest has directed to how exercise affects sleep quality. The beneficial effect of exercise on sleep has generally been presumed, but research results are conflicting. Our hypothesis was that the acute exercise promotes the sleep in intensity-dependent manner; when the intensity of exercise increases the quality of sleep improves. According to Bunnel et al. (1983), Dworak et al. (2008) and Horne et al. (1983), the more vigorous the exercise is, the more beneficial are its effects on sleep as compared to lower intensity exercises.

The present study partly supports these previous studies. According to the present findings, actual sleep time was significantly higher after high intensity exercise compared to the control. In addition, subjective sufficiency of sleep was higher after both exercises compared to the control, and subjective sleep quality higher after high intensity exercise compared to the control. Moreover, after both exercises, the percentage of subjects, who felt that they slept worse than usual, was lower compared to the control nights. Although, only the actual sleep time from objective variables of sleep quality was affected after high intensity exercise, the result together with subjective sleep quality suggest that, especially, the high intensity exercise promotes the quality of sleep. In addition, subjective tiredness was significantly reduced during the night after high intensity exercise, which also supports the suggestion that high intensity exercise may promote the sleep, and therefore promote the recovery.

In previous studies, where the effects of high intensity exercise were clearer, the exercise intensity was higher (Bunnel et al. 1983, Dworak et al. 2008) and the duration longer (Horne et al. 1983). Hence, if the intensity or the duration of exercise was higher in the present study, the other objective sleep variables would possibly be affected. On the other hand, in these previous studies polysomnographic measurements were used, whereas in the present study, actigraphy was used. Actigraphy should be valid method to measure sleep quality (Sadeh ym. 1994, Morgenthaler 2007). However, for example Youngstedt et al. (1999), who used actigraphy, did not found any differences in objective sleep quality between the night after exercise and the control night, whereas in studies, which had used polysomnograghic measurements, the high intensity exercise had promoted sleep quality (Bunnel et al. 1983, Dworak et al. 2008 and Horne et al. 1983). According to the present and previous findings, exercise, especially the high intensity exercise, may have a positive effect on sleep quality.

How exercise promotes the sleep, is still under questions. Exercise may promote the sleep by reducing anxiety and stress. Sleep problems and disrupted sleep have been demonstrated to associate with acute stress, (Hall et al. 2004, Morin et al. 2003) anxiety and depression (Eller 2006). In turn, physical activity has been shown to have a positive impact on mental well-being by reducing anxiety and depression (Fox 1999, Fox et al. 2007) and by enhancing recovery from work stress (Rook et al. 2006). Therefore, exercise might promote the sleep by reducing anxiety and depression (Youngstedt 2005). In the present study, subjective stress level and mental recovery was not affected after the exercises, thus increased quality of sleep after acute high intensity exercise may not be related to the reduction of stress.

On the other hand, sleep is considered to be essential in reducing energy demand, refreshing the function of the immune and the endocrine systems, and in restoring key cellular components, which are used up during wakefulness (Halson 2008, Mignot 2008). Previous studies suggest that exercise increases energy demand of the day, and the higher the exercise intensity the higher the energy demand. In addition, sleep duration and amount of SWS will increase, if daily energy demands are increased. Therefore, sleep quality may improve after exercise because of the increased SWS.

(Driver & Taylor 2000.) Many studies have supported this idea, because the results have shown that high intensity exercise has a higher effect on sleep than low or moderate intensity exercise (Bunnel et al. 1983, Drowak et al. 2007). As mentioned before, the present results are parallel to those previous studies. However, we can not conclude that our study support the hypothesis of homeostatic sleep regulation, because we did not measure energy expenditure or amount of SWS.

It has been indicated that falling body temperature is essential for sleep initiation (Atkinson & Davenne 2007). Furthermore, additional body temperature elevation before bedtime activates heat-loss, when increased peripheral skin blood flow and sweating decrease the body´s core temperature (Bach et al. 2002, Driver & Taylor 2000, Horne et al. 1983, Youngstedt 2005). Horne et al. (1983) have revealed that exercise might enhance sleep via body temperature elevation, which is in a linear relationship with increasing workload (% VO_{2max}). Thermoregulatory responses are initiated by hypothalamic structures, where sleep-promoting systems are also located, which might explain the relationship between the heat-loss and the sleep mechanisms (Bach et al. 2002, Driver & Taylor 2000). The preoptic area in the hypothalamus contains a high density of thermoreceptors (Bach et al. 2002) and local warming of this pre-optic area might control non-REM sleep (Nakao et al. 1995) and activate heat loss responses (Alam et al. 1996, Atkinson & Davenne 2007). In addition, Methippara et al. (2003) concluded that sleep induction is enhanced by warm sensitive neurons of the preoptic area via its inhibitory effect on many arousal structures. However, we can not conclude that our study support the hypothesis that body-heating after exercise facilitates sleep, because we did not measure body temperature or amount of SWS.

8.6 Study limitations

From a statistical point of few, the number of subjects was unfortunately low, which weakens the statistical power of study. Due to that, standard deviations affected a lot to which results reached statistical significance. In addition, subjects slept at home and started the devices according to the verbal and the written instructions. Maybe because

of that some of the R-R interval measurements did not succeed, and number of subjects reduced even more. This could be prevented with the night measurements in the laboratory. However, the purpose was to create as natural situations as possible, hence the home measurements were the primary choice. Furthermore, the method which was used to measure objective sleep quality, actigraphy, is not the golden standard method, and even though it should be valid and reliable method (Sadeh ym. 1994, Morgenthaler 2007), it may affect the results. This method was chosen, because the purpose was to measure the quality of the sleep at home. In addition, bedtime was not controlled; the bedtime varied between the different conditions, but especially between the different subjects. Moreover, physical activity, eating and drinking could not be fully controlled. However, the subjects got the instructions to follow their usual circadian rhythm, not to exercise, drink alcohol or smoke cigarettes two days before the exercises. All subjects were moderately active, which may affect the results. If the subjects were non-active or slightly active, the results concerning both recovery of autonomic activity and sleep quality could be different.

8.7 Conclusions

According to the present study, HFP started to increase during the first minute of recovery after moderate intensity exercise, but during the second minute of recovery after high intensity exercise. This result suggests that after exercise vagal reactivation is quite fast and intensity dependent. In addition, during the first hours of sleep, HR was higher and RMSSD lower after high intensity exercise compared to the control. Moreover, cortisol concentration was higher and IgA to protein ratio lower immediately after high intensity exercise, but cortisol concentration and IgA to protein ratio were not remarkably affected after awakening in the morning after the high intensity exercise. According to these results, it seems that the high intensity exercise is enough to disturb the cardiac autonomic regulation during early sleep, but not stressful enough to affect biomarkers of stress in saliva after awakening. In addition, according to the results, high intensity exercise promotes the sleep. Together these results suggest that the sleep after

high intensity exercise had been sufficient enough to overall recovery and sterling enough to restore the homeostasis

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