THE ASSOCIATION BETWEEN SLEEP AND PHYSICAL ACTIVITY IN HYPERTENSIVE INDIVIDUALS

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ABSTRACT

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Physical activity and sleep are fundamental factors for health and quality of life for humans. Being physically active decreases the risk for several chronic conditions and getting sufficient sleep can positively impact. Nonetheless, sleep has often been neglected in the society nowadays and sleep disturbances have increased. The lack of sleep is a risk factor for development of hypertension which is the most common but preventable risk factor associated with mortality. Patients with hypertension generally have poorer sleeping patterns that can cause other health problems. Physical activity is considered a non-pharmacological treatment for sleep disturbances and it could help improve poor sleeping habits in hypertensive patients. The aim of the study was to investigate the association between sleep and physical activity in hypertensive individuals. This study used a cross-sectional design. The participants were 45 hypertensive individuals (51.5 ± 8.8 years) with average blood pressure of 135/14 mmHg. Portable polysomnography device SOMNO HD™ was used to record objective sleep and Pittsburgh Sleep Quality Index questionnaire was used to assess subjective sleep quality. Acute physical activity was recorded with Firstbeat Bodyguard 2 device and regular physical activity was determined based on the questionnaire. Cardiorespiratory fitness was tested on the treadmill according to the USAFSAM protocol. The data were analysed with the IBM SPSS Statistics 21 – software. Shapiro-Wilk test, one-way ANOVA and Kruskal-Wallis test were used as the analysing methods. The effect size were calculated with partial eta squared ($\eta_p{}^2$) and partial epsilon squared ($\varepsilon_p{}^2$). Participants were split into 3 groups based on their regular physical activity level, acute physical activity and cardiorespiratory fitness. There were no statistically significant differences between any of the groups in objective or subjective sleep parameters. The main finding of the study was that physical activity is not associated with sleep patterns in hypertensive individuals. These findings are not in accordance with current literature, however, the effects of physical activity on sleep is small in most studies. Association between physical activity and sleep requires more randomized controlled trials with exercise interventions in this clinical population.

Key words: physical activity, sleep, hypertension, sleep quality, exercise, cardiorespiratory fitness

ABBREVIATIONS

BP	Blood pressure
CRF	Cardiorespiratory fitness
CVD	Cardiovascular diseases
EEG	Electroencephalography
EMG	Electromyography
EOG	Electrooculography
GH	Growth hormone
HR	Heart rate
NREM	Non-rapid eye movement
OSA	Obstructive sleep apnoea
PA	Physical activity
PSQI	Pittsburgh Sleep Quality Index
PSG	Polysomnography
REM	Rapid eye movement
SE	Sleep efficiency
SOL	Sleep onset latency
SWS	Slow-wave sleep
S1	Stage 1
S2	Stage 2
S3	Stage 3
SNS	Sympathetic nervous system
TIB	Time in bed
TST	Total sleep time
WASO	Wakefulness after sleep onset

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1. INTRODUCTION

It has become common knowledge that physical activity improves quality of life and prevents several chronic conditions. Being physically active is essential to improve health and reverse the effects of sedentary lifestyle, which has become more prominent in the modern world. Some benefits occur shortly after an exercise session, while long-term effects can be seen by habitually engaging in physical activity throughout the lifespan (Physical Activity Guidelines for Americans 2nd edition 2018).

Another important but often neglected base for good health is sufficient sleep. We live in a fast-paced world and neglect the importance of sleep. Sleep is often considered unessential, however, there are important detrimental effects associated with poor sleep patterns. The lack of sleep has been shown to be a risk factor for the development of many chronic diseases, hypertension being one of them (Lo et al. 2018).

Hypertension is one of the most common yet preventable cardiovascular risk factors (Physical Activity Guidelines for Americans 2nd edition 2018) that has been greatly associated with mortality (Fernandez-Mendoza et al. 2012). According to the World Health Organization (WHO 2019), hypertension is diagnosed if the reading of systolic blood pressure on two days of measurements is above 140 mmHg and/or the diastolic blood pressure readings on both days is over 90 mmHg. When left untreated it can lead to many adverse health outcomes. It has been generally known that physical activity has a beneficial effect on the health parameters in this population, but less attention has been focused on sleep. It has been proposed by literature that hypertensive patients are more likely to have sleep disorders and poor sleeping patterns (Han et al. 2019; Tiede et al. 2015). This can lead to feeling less energized throughout the day which can, in turn, lead to a more sedentary lifestyle. It has been widely accepted that poor sleep quality affects activity levels, but is the opposite also relevant? Can low activity levels affect sleep quality?

In the past decades, several studies have investigated this question and found that in general, physical exercise has a small effect on sleep (Banno et al. 2018) but this has rarely been studied in hypertensive individuals. There is a growing consensus that physical activity benefits sleep in this population (Dolezal et al. 2017) but more research is needed.

If exercise would be proven beneficial for sleep quality in hypertension, this could provide an incentive for changing lifestyle of this population. If these participants slept better, this could mean that they would improve their behaviour by choosing healthier habits and improving quality of life which is especially important in the elderly population.

The aim of this master thesis is to assess the association between physical activity and sleep in people with diagnosed hypertension. Firstly, the effect of habitual physical activity is assessed. Then the relationship between acute physical activity and sleep is investigated. Lastly, the correlation between cardiorespiratory fitness and sleep is inspected. Results can benefit people with high blood pressure to develop effective strategies on how to improve their sleep with exercise and increase the quality of life.

2. PHYSICAL ACTIVITY

It is common knowledge that physical activity (PA) is beneficial for the majority of people and it can affect several health aspects of individuals. In this chapter, the role and recommendations of physical activity is explained.

PA is any type of movement produced within a body by contraction of skeletal muscles that increases energy expenditure above a basal level (Physical Activity Guidelines for Americans 2nd edition 2018) and includes activities in day-to-day living. Exercise on the other hand, is a structured and planned form of physical activity that is defined by frequency, duration and intensity. PA is generally known as a health-enhancing behaviour since it influences almost every body system, and is a key determinant in the promotion and maintenance of human health. Decreasing sedentary behaviour and increasing physical exercise has tremendous benefits for everyone, regardless of age, health status or race. Being active is one of the most important and cost-effective actions that people can take to improve their health (Physical Activity Guidelines for Americans 2nd edition 2018).

The number of preventable diseases is increasing in the world and regular PA favourably influences seven out of ten the most common chronic diseases. Yet nearly 80 % of adults are not meeting guidelines for aerobic and muscle-strengthening exercise, and only half of the individuals meet aerobic guidelines. Physical inactivity is therefore one of the leading risk factors for development of chronic diseases such as hypertension (Physical Activity Guidelines for Americans 2nd edition 2018). In other words, sedentary behaviour, which is any waking behaviour characterized by low level of energy expenditure, less than or equal to 1.5 metabolic equivalents (MET), has been associated with increased risk of premature mortality and cardio metabolic risk biomarkers in adults (Physical Activity Guidelines for Americans 2nd edition 2018).

2.1. Guidelines for adults

For substantial health benefits, adults should engage in at least 150 to 300 minutes of moderate- or 75 to 150 minutes of vigorous-intensity aerobic exercise per week (Physical Activity Guidelines for Americans 2nd edition 2018). Preferably, it is advised to spread aerobic activity throughout the week, and it can be split throughout

the day. The literature suggests that additional exercise beyond PA guidelines for adults results in more benefits. Equally as important as aerobic exercise are muscle strengthening activities, which also result in improved health parameters. Muscle-strengthening activities or resistance training involves producing force with the major muscle groups and should be performed at least 2 days per week to provide optimal benefits (Physical Activity Guidelines for Americans 2nd edition 2018). Nonetheless, if adults cannot reach those recommendations, they are advised to decrease sitting time and move more throughout the day (Physical Activity Guidelines for Americans 2nd edition 2018).

2.2. Physical activity assessment

The goal of PA assessment is to identify an optimal exercise dose for individuals and investigate possible correlations of exercise variables to health parameters (Ainsworth et al. 2015). When assessing, it is important to identify frequency, duration, intensity and type of activity preformed (Ainsworth et al. 2015).

PA assessment with self-reported tools have been in use for nearly 50 years. Indirect measures of PA include questionnaires, detailed diaries and PA logs (Ainsworth et al. 2015). Self-reported measured present low burden to the respondent and are cost-effective at the same time (Ainsworth et al. 2015). Questionnaires are most frequently used among subjective PA assessment tools and are classified into three groups: global questionnaires, short-term questionnaires and quantitative history recall questionnaires (Ainsworth et al. 2015).

The most important limitation with use of subjective measured is that assessed PA is related to the accuracy of recall and reporting bias. Therefore, individuals tend to overestimate or underestimate PA (Ainsworth et al. 2015). It has been shown that self-reported questionnaires were accurate when describing high intensity PA but not when reporting low-to-moderate intensity (Ainsworth et al. 2015). Moreover, attempts to reconcile subjective measures with direct measures have shown that subjective error is not systematic but random (Ainsworth et al. 2015). Thus, objective PA assessment tools are thought to validate subjective reporting and reduce human error in reporting bias and PA recall.

Direct methods of measuring PA include motion sensors such as accelerometers, pedometers, heart rate (HR) monitors and multiple-sensor devices (Ainsworth et al. 2015). These tools can give information about intensity, volume, duration, distance and energy expenditure and can tell more accurately physiological or mechanical parameters that correspond to PA (Ainsworth et al. 2015). Although direct measure devices are considered more accurate, there is still no gold-standard wearable monitor as the choice depends on a variety of factors (Ainsworth et al. 2015).

2.3. Physical activity in hypertensive patients

Increased physical activity is considered a first-line intervention for prevention of hypertension, and a treatment strategy for patients with stage one or stage two hypertension (Ghadieh & Saab 2015). Aerobic exercise is almost completely free of secondary effects when not contraindicated (Ghadieh & Saab 2015) and it should be advised for all individuals. It has been shown that one bout of aerobic exercise can lower office and ambulatory blood pressure in hypertensive individuals (Boutcher & Boutcher 2016). Similarly, regular participation in aerobic exercise improves blood pressure and promotes general health (Boutcher & Boutcher 2016). Exercise produces anti-inflammatory action through the sympathetic nervous system and hypothalamic-pituitary-adrenal axis. Thus, PA has direct effects on blood pressure (Ghadieh & Saab 2015). Moderate- and higher-intensity aerobic exercise (up to 70% of maximal oxygen consumption) produce similar hypotensive effect (Boutcher & Boutcher 2016). It has also been found that intermittent aerobic and anaerobic exercise that involves mini-bouts of high-intensity and rest periods of light-intensity exercise, also significantly reduce blood pressure. This high-intensity intermittent exercise typically results in greater aerobic fitness with less training volume and it is a time-efficient strategy to produce adaptations and blood pressure benefits. High-intensity exercise affects endothelial function, arterial stiffness, insulin resistance and mitochondrial biogenesis (Boutcher & Boutcher 2016). Literature on resistance training is scarce, but some studies found that even one bout of resistance training reduces daytime ambulatory blood pressure in hypertensive adults (Boutcher & Boutcher 2016). Clinical review by Ghadieh and Saab (2015) shows that dynamic exercise training can lower systolic and

diastolic BP, whereas regular dynamic resistance exercise failed to show changes in office systolic blood pressure in one meta-analysis (Boutcher & Boutcher 2016).

3. SLEEP

Sleep is a rest state that humans need to recuperate normally and is considered to be one of the main factors contributing to well-being and health (Chiang and Kang 2012). In the following chapter methods for sleep measurement, sleep structure and sleep recommendations are discussed.

3.1. Measurement of sleep structure

Sleep is an active physiologic state that can be measured by many methods, but it is challenging to interpret results on sleep as positive or negative. There are a number of methods that appear in the literature. For example, sleep parameters can be measured via sleep logs and questionnaires. Methods like actigraphy, accelerometers and polysomnography (PSG) can assess sleep objectively (Natteru and Bollu 2018). Sleep technology is used to evaluate and manage sleeping disorders, prevent and reduce morbidity of sleep disorders and improve sleep quality, daytime performance and quality of life (Chiang and Kang 2012).

PSG is considered to be the gold standard for sleep technology (Parmeggiani and Velluti, 2005). PSG is a method developed for physiological description of sleep (Hirshkowitz 2016). The word was derived from Greek and Latin where word “poly” means many, “somnus” refers to sleep and “graphein” stands for write (Natteru and Bollu 2018). The device records multiple parameters of sleep (Vaugh and Giallanza 2008) and the most commonly used variables in the literature are sleep onset latency (SOL), stage 2 (S2) sleep, slow-wave sleep (SWS), rapid eye movement (REM) sleep, REM latency, total sleep time (TST) and wakefulness after sleep onset (WASO) (Youngstedt, O´Connor and Dishman 1997).

3.2. Sleep structure

Normal sleep of an adult starts briefly with stage 1 (S1), followed by stages 2, 3 and REM sleep. Non-rapid eye movement (NREM) sleep, which consists of stages 1-3, accounts for 75-80% of total sleep time (Casale, Brugnoli & Giradi 2013). The

sequence of stages is characterized as a sleep cycle (Parmeggiani and Velluti 2005) typically lasting 80-120 minutes (Yaremchuk and Wardrop 2011). A person usually goes through 3-4 cycles per night, depending on the duration of sleep (Yaremchuk and Wardrop 2011). During the first part of the night, there is more SWS and in the second half of the night, there is a greater percentage of REM sleep (Parmeggiani and Velluti 2005). The amount of SWS is inversely proportional to the amount of time spent awake, increasing with sleep restriction and decreasing with naps (Yaremchuk and Wardrop 2011).

Identification of sleep stages is based on the different characteristics of electroencephalographic (EEG), electrooculographic (EOG) and electromyographic (EMG) levels during each epoch. Epochs in PSG consist of 30-second intervals. The sleep stages correspond to the depth of sleep where sleep in stage 3 (S3) is deeper than in S1 (Parmeggiani and Velluti 2005). In order for PSG recordings to be applicable in science, there was a need for standardization. Nowadays, Manual for the scoring of Sleep and Associated events is used worldwide, published by American Academy of Sleep medicine published in 2017 (Natteru and Bollu 2018).

3.2.1. Stage 1

S1 is described as a state of transition from wakefulness to sleep but most individuals that are awakened at this stage report that they were still awake (Yaremchuk & Wardrop 2011). EEG rhythm starts to slow down from alpha activity (8 - 13 Hz) to slower low voltage mixed frequency pattern of theta waves (Parmeggiani and Velluti 2005; Yaremchuk & Wardrop 2011) and sharp vertex waves can also be seen (Casale, Brugnoli & Giradi 2013). EOG transitions from eye blinks to slow asynchronous movements, mainly horizontal (Yaremchuk & Wardrop 2011). EMG detects low-voltage tonic activity, interrupted by abrupt muscular contractions of the extremities, which may wake the person (Parmeggiani & Velluti 2005). According to Yaremchuk and Wardrop (2011), S1 represents less than 5% of adult's sleep time.

3.2.2. Stage 2

S2 is considered to be the real onset of sleep (Parmeggiani & Velluti 2005) and it begins approximately 10-12 minutes after the S1 (Casale, Brugnoli & Giradi 2013). The arousal threshold is higher than in S1, so it is more difficult to wake up an individual (Yaremchuk & Wardrop 2011). S2 is characterized by sleep spindles, which are short rhythmic waves of 12-14 Hz with 20-30 μV amplitude and duration of >0.5 seconds, theta activity and K complexes. K complexes are a rapid high-voltage negative wave followed by positive components that are slower and higher in amplitude (Natteru & Bollu 2018). At the beginning of the S2, slow eye movements can be visible, but they disappear later. During this stage, tonic muscle activity is attenuated (Parmeggiani & Velluti 2005). S2 sleep accounts for 45-55% of total adult sleep time and is the primary component of NREM sleep (Yaremchuk & Wardrop 2011).

3.2.3. Stage 3

S3, which is the deepest form of sleep, starts 30-60 minutes after the S2 (Casale, Brugnoli & Giradi 2013) and has the highest arousal threshold (Yaremchuk & Wardrop 2011). It is characterized by scarce mental activity and increased parasympathetic activity (Parmeggiani & Velluti 2005). S3 is often characterized as SWS or delta sleep, due to a predominance of delta waves with frequency ranging from 0,5 - 2 Hz (Yaremchuk & Wardrop 2011). However, K complexes and spindles can still be seen (Parmeggiani & Velluti 2005). There is no activity in EOG and no modifications in EMG with respect to S2, since very low-amplitude tonic activity can persist (Parmeggiani & Velluti 2005). S3 accounts for 20-25 % in young adults, but the percentage diminishes with age (Yaremchuk & Wardrop 2011).

3.2.4. REM sleep

In REM sleep, EEG is characterized by a low amplitude fast pattern in beta frequency that is mixed with small amounts of theta rhythms (Casale, Brugnoli & Giradi 2013). During this stage rapid eye movements appear. They can be horizontal, vertical,

oblique, binocularly symmetrical, isolated or appear in bursts (Parmeggiani & Velluti 2005). Suppression of muscle tone is visible in EMG recording. There are exceptional twitches that happen in facial muscles and distal extremities. In this stage, we must distinguish between tonic events (muscular atonia and EEG), which persist throughout the stage, and phasic events, which appear at random (REMs or twitches) (Parmeggiani & Velluti 2005). During REM sleep the body is almost paralyzed but the brain is very active since dreaming is common during this stage (Yaremchuk & Wardrop 2011). REM sleep accounts for 20-25% of total sleep time (Yaremchuk & Wardrop 2011; Casale, Brugnoli & Giradi 2013).

3.3. Other sleep parameters

From PSG data, other sleep parameters than sleep staging can be also calculated. Time in bed (TIB) refers to the duration of time from when participant went to bed to the final awakening. Some people fall asleep earlier and for some it might take longer. This interval, from deciding to go to sleep ("lights out") to the first epoch of sleep is called sleep onset latency. The time of actual sleep recorded during PSG is termed as total sleep time or sleep duration. Because there is a high variance in TST, TIB and SOL, sleep efficiency (SE) is different among individuals. SE is the ratio of total sleep time to time in bed (i.e., TST/TIB x 100). During the night, people wake up many times. The amount of time they spend awake, after they have already fallen asleep, is categorized by wake up after sleep onset (WASO) (Pandi-Perumal, Spence & BaHammam 2014).

3.4. Sleep quality

Some authors argue that the most commonly used parameter for defining sleep quality is SWS because it is supposed to be the deepest form of sleep (Dworak et al. 2008). This thinking has been questioned by other scientists (Youngstedt 2005). With this in mind, the interpretation of PSG parameters remains difficult.

On the other hand, sleep quality is largely subjective and sleep laboratory measures cannot always correlate with perceived sleep quality (Buysse et al. 1989). Therefore,

many studies use standardized Pittsburgh Sleep Quality Index (PSQI) as a quantitative measure of subjective sleep quality that quickly identifies poor and good sleepers. The questionnaire assesses sleep quality during the previous month asking 19 self-rated questions and 5 questions by the bedpartner (or roommate). The latter are not tabulated into scoring of PSQI and are only used for clinical information. Questions are then grouped into seven component scores that are summed to yield a global PSQI score. On a level 0-21, higher scores indicate worse sleep quality (Buysse et al. 1989).

3.5. Sleep disturbances

Sleep is one of the most important parameters when it comes to health and wellbeing. Nonetheless, many people neglect sleep as it is commonly thought of as a passive state. Thus, sleep related problems and diseases have increased in the recent years. It has become a public health problem since 15-30% of the adult population complains of frequent sleep quality disturbances (Buysse et al. 1989) and at least 10% of the population has insomnia which is comorbid with number of diseases (Akerstedt et al. 2016). 30-40% of the US population is estimated to have problems with falling asleep or daytime sleepiness (Hossain & Shapiro 2002), and approximately one third of adults do not meet the guidelines of at least 7 hours of sleep per night (Kakinami et al. 2017; Watson et al. 2015). Problems with sleep quality are common already in young adults since they affect up to 40% of people (Morbidity and mortality weekly report, 2011), but they often go undiagnosed. Inadequate or disturbed sleep is related to many negative health outcomes (Loprinzi & Cardinal. 2011), such as impaired cognitive performance, mood, glucose metabolism, appetite regulation and immune function (Dolezal et al. 2017). Moreover, even poor sleep quality affects self-rated health, obesity, mental health and mortality (Kakinami et al. 2017). It has been debatable in the past how to determine good and bad sleep. According to Ohayon et al (2017), good sleep quality is characterized as shorter sleep latencies, fewer awakenings and reduced wake after sleep onset and compromising these parameters can result in bad sleep quality. Furthermore, Kakinami and colleagues (2017) found that poor sleep quality is also defined as lower SE, more sleep disturbances, use of sleep medication and daytime disfunction (Kakinami et al. 2017).

Individuals cope with sleeping problems in various ways. Pharmacological treatments that are used as self-help remedies for poor sleep show only short-term efficacy (Buman & King. 2010), and overuse can lead to tolerance or dependence (Kakinami et al. 2017). This is why low-cost non-pharmacological treatments for sleep problems are needed. Common treatment that is used often is cognitive behaviour therapy, but other non-pharmacological treatments include sleep hygiene, stimulus control, muscle relaxation etc. Non-pharmacological treatments act slower but do not result in any adverse effects (Buman & King, 2010). For these treatments to be effective and attractive for individuals, they need to be highly adoptable, accessible and low cost. One of the most potent treatments that meets these criteria is exercise (Buman & King 2010). Exercise is recommended throughout the lifetime and can rarely result in negative outcomes, whereas other sleep-enhancing remedies, such as sleeping pills, are not recommended for long-term use (Youngstedt 2005).

Exercise is a simple and inexpensive way to promote sleep. Increased PA and reduced sedentary behaviour could be considered as a recommended non-pharmacological treatment since exercise was positively associated with sleep quality and quantity in many observational studies (Dolezal et al. 2017), with better subjective sleep (King et al. 2008; Yang et al. 2012) and sleep latency in experimental studies (Yang et al. 2012). It is also generally believed among individuals that regular exercise can enhance sleep, affect other health parameters simultaneously, and enhance overall quality of life (Buman & King 2010).

3.6. Sleep characteristics in hypertension

Hypertensive patients are more prone to cardiovascular diseases (CVD), since the risk of them increases as blood pressure rises above 115/75 mmHg (Pescatello et al 2004). Being that adequate sleep prevents adverse cardiovascular outcomes, it is extremely important that hypertensive patients get enough sleep (Lo et al. 2018). Moreover, adequate sleep is also important for prevention of the disease, since sleep disturbances and short sleep duration have been linked to development of hypertension (Lo et al. 2018; Fernandez-Mendoza et al. 2010; Fung et al. 2011; Li et al. 2018; Mirjat et al. 2020; Meng, Yheng & Hui 2013). Insufficient sleep increases BP and HR because of increased (sympathetic nervous system) SNS activity after the night when sleep was

restricted (Gangwisch 2014). If this happens often, it can lead to structural adaptations and can cause a risk for hypertension.

Not only insufficient sleep, also sleep disorders, especially sleep deprivation and obstructive sleep apnoea (OSA), have also been linked to the development of hypertension (Mirjat et al. 2020). OSA, which is complete or partial collapse of narrowed pharynx (Khan 2006), is an independent risk factor for the development of hypertension (Toth & Sica 2010). Sleep loss is considered a pathophysiological mechanism since it activates SNS and promotes inflammation. This condition is characterized by the activation of the hypothalamus-pituitarity-adrenal axis and SNS, which is susceptible to hypertension (Mirjat et al. 2020). Moreover, because of heightened sympathetic drive during sleep, which is caused by OSA, there is absence of nocturnal dipping pattern in blood pressure (BP), which causes hypertension (Toth & Sica 2010). In the normal sleeping pattern, a significant drop in the mean BP and HR is visible, because autonomic tone exhibits parasympathetic dominance. BP values in general drop by 10-20% during sleep, which is recalled as dipping phenomenon. In the hypertensive patients, however, the dipping phenomenon might be diminished (Culebras 2013).

Not only is the lack of good quality sleep is risk factor for development of hypertension, but patients with elevated BP have also reported poor subjective sleep quality when compared to controls (Tiede et al 2015). Study by Batal and colleagues (2010) addresses poor sleep quality in 29 out of 40 patients with pulmonary hypertension, assessed by PSQI. Hypertensive patients are more likely to have difficulty sleeping, complain about unstable sleep and have trouble waking up in the morning. Moreover, epidemiologic data suggests that prevalence of insomnia is higher with hypertensive individuals than the general population (Hayes, Anstead & Phillips 2009). Not having a good night's rest can affect quality of life, especially in hypertensive individuals that are in general more fatigued. That is why physical activity is advocated as an effective intervention for the treatment of disordered sleeping in a variety of conditions, such as cardiovascular disease, type 2 diabetes etc. (Dolezal et al 2017).

4. PHYSICAL ACTIVITY AND SLEEP

Physical activity is known to affect every body system, including the brain, which could possibly improve cognitive function, reduce anxiety, depression and improve sleep (Sharma et al. 2006). Experimental evidence shows that PA increases the central blood flow and makes alterations in neurotransmitter and amino acid transport through the blood-brain barrier (Hollman et al. 1994). The acute benefits os PA on the brain consist of reduced anxiety, improved sleep and cognitive function. Others, such as improvements in deep sleep and long-term anxiety, are seen with regular chronic PA (Physical Activity Guidelines for Americans 2nd edition 2018). According to the Physical Activity guidelines for Americans (2018), habitual chronic exercise in adults resulted in improved sleep outcomes, such as increased sleep efficiency, sleep quality, deep sleep, reduced daytime sleepiness and decreased frequency of use of medication to aid sleep. Guidelines point to a dose-response pattern where greater volumes of moderate-to-vigorous PA are associated with greater effects on sleep (Physical Activity Guidelines for Americans 2nd edition 2018). However, it should be emphasized that people with already optimal sleeping habits have little room for improvement and those with greater initial impairment in sleep have greater room for improvement.

4.1. Mechanisms of effects of physical activity on sleep

The function of sleep and its trigger is not yet scientifically established; therefore, it is hard to answer how PA affects sleeping patterns. Favourable effects on sleep can be explained by multiple pathways including circadian rhythm, metabolic, immune, thermoregulatory, vascular, mood and endocrine effects (Chennaoui et al. 2015).

Figure 1 presents the possible effects that PA can have on sleep. Acute physical activity affects body core temperature, endocrine system and metabolism and inhibits autonomic nervous system. Regular physical activity on the other hand additionally affects inflammation factors and mood. Moreover. there is a probable link between regular physical activity and circadian rhythm. Regular and acute physical activity therefore indirectly affects sleep parameters through previously mentioned factors.

Increased sleep time and enhanced SWS can thus have a positive effect on mood and might increase the chances for exercise participation.

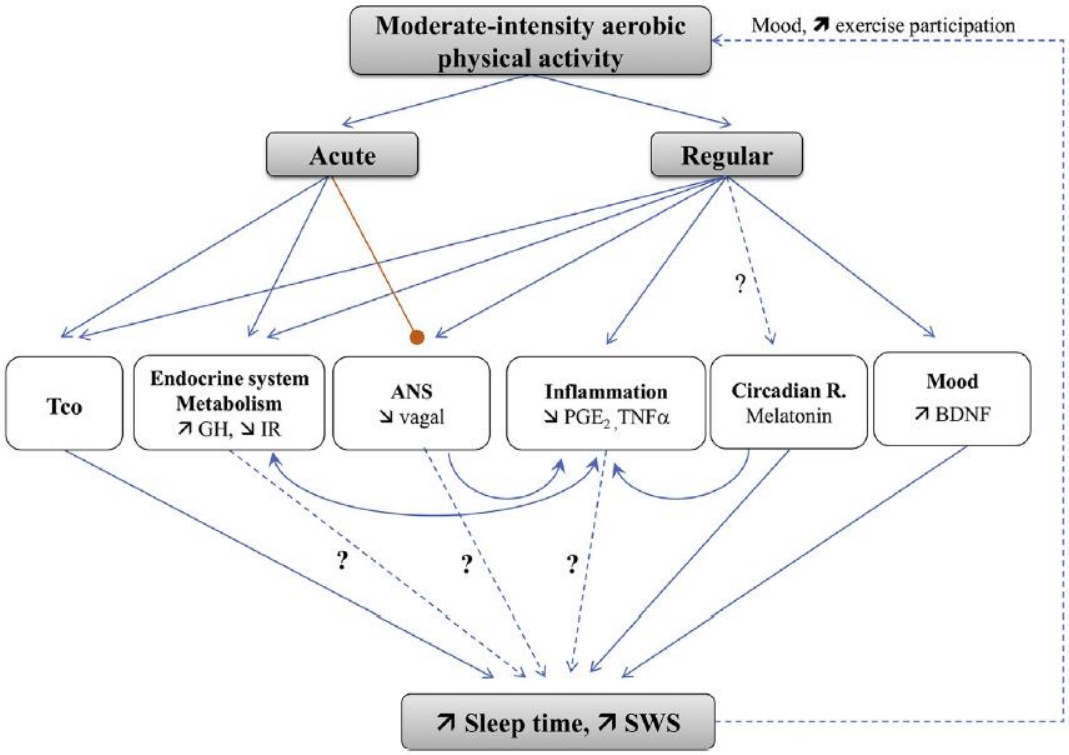

FIGURE 1. Possible effect of acute or regular moderate intensity physical activity on sleep. ANS = autonomic nervous system, BDNF = brain derived neurotrophic factor, Circadian R. = circadian rhythm, GH = growth hormone, IR = insulin resistance, PGE_2 = prostaglandin E_2, SWS = slow wave sleep, Tco = body core temperature, TNF-α = tumor necrosis factor alpha. (Chennaoui et al, 2015).

In general, the literature mentions various sleep related theories, but several studies suggest there are two advanced rationales explaining the phenomena.

Firstly, physical activity leads to physiological changes that are favourable to homeostatic sleep regulation when SWS is used as a marker (Driver and Taylor 2000). Secondly, acute exercise is thought to stabilize the circadian system and reduce daytime sleepiness (Youngstedt 2005). Other hypotheses have been placed, for example, that sleep serves as an energy conservation function, body tissue restitution function and temperature down-regulation function. This is why exercise could have a potent effect on sleep. It is a stimulus that elicits depletion of energy stores, tissue breakdown and elevation of body temperature (Youngstedt 2005). There are numerous moderators of exercise-sleep relationship with conflicting evidence. The role of

exercise might be affected by a complex set of physiological and psychological factors (Buman and King 2010).

4.1.1. Anxiety reduction

Anxiety reduction seems to be the most plausible mechanism according to Youngstedt (2005). Disrupted sleep is often a hallmark of anxiety, and chronic insomnia is associated with increased psychological arousal, since hyper arousal is seen in insomniacs (Buman &King 2010). There is sufficient evidence that acute and chronic exercise reduces anxiety, thus it is plausible that exercise stimuli reduce anxiety which can promote sleep (Youngstedt 2005; Buman & King 2010).

4.1.2. Thermogenetic effect

One of the most commonly tested hypotheses for sleep function is that the preoptic area of anterior hypothalamus is linked with sleep and temperature down-regulation (Youngstedt 2005; Driver & Taylor 2000). Evidence suggests that the trigger for evening sleep onset is a decline in the body temperature by increased skin blood flow. Sleep onset is thus associated with peripheral heat dissipation (through vasodilation and sweating) and a reduction of metabolic rate and temperature during sleep (Driver & Taylor 2000; Loprinzi & Cardinal 2011). Exercise is a factor that raises body temperature more readily than other stimuli and can thus activate heat-loss (Driver & Taylor 2000). Studies found that even passive temperature elevation in sauna could activate temperature down-regulation, which is associated with deeper forms of sleep (Buman & King 2010). It is debatable whether SWS is associated with better sleep outcomes than REM but if that is the case, exercise might promote SWS through increased body temperature (Youngstedt 2005).

In a study conducted by Horne and Moore (1985), they compared the effect of exercise with and without additional body cooling on sleep parameters. Six female participants ran at 75% of their maximal aerobic capacity on two separate occasions (hot and cold conditions). After the run in the hot conditions, SWS was increased significantly and REM was decreased. Cooling condition showed no difference compared to baseline.

Authors concluded that body-heating effects during running might promote SWS sleep and cooling could eliminate any potential SWS increase (Horne and Moore 1985).

4.1.3. Body restoration and energy observation

Theory suggests that anabolic activity during sleep is improved when preceded by high catabolic activity during wakefulness (Driver & Taylor 2000). Since exercise is a stress that readily depletes energy stores and increases energy expenditure it should facilitate sleep and longer sleep duration in order to recover (Driver & Taylor 2000; Erlacher, Erlacher & Schredl 2015). Evidence to support this hypothesis are studies that show increased SWS after a session of acute exercise. Moreover, subjective sleep complaints (efficiency, quality and duration) often improve in response to amounts of chronic and acute exercises (Buman & King 2010).

4.1.4. Circardian phase-shifting effect

The circadian system regulates sleep-wake cycle and is regulated by our central biological clock, the suprachiasmatic nucleus (Buman & King 2010). Nucleus is affected by endogenous (body temperature, melatonin) and exogenous (bright light, meal timing, exercise) cues, and thus synchronizes activity, consumption and rest to the circadian cycles (Buman & King 2010; Gangwisch 2009). Time cues from the earth's 24-hour rotation affect the circadian system to promote a specific temporal and environmental niche (Johnson et al. 2003). The magnitude and direction of circadian rhythm phase shifts is described by the phase-response curve. The cues are the most fundamental chronobiological tool to ease circadian misalignment.

When cues are not synchronized, disrupted sleep can occur. It has been shown that light is one of the strongest synchronizers of circadian rhythm and when humans are exposed to artificial light in the evenings it can affect phase-shifting (Buman & King 2010; Gangwisch 2009). According to Buman and King (2010), exercise can also mediate phase shifts, especially in the modern society where individuals rely on modern conveniences to minimize physical activity (Gangwisch 2009). In a recent study, Youngstedt, Elliott and Kripke (2019) confirmed phase-response-curves were established for moderate exercise.

4.1.5. Cytokine concentration effects

Exercise elevates proinflammatory cytokines interleukin-1, IL-6 and tumor necrosis factor-alpha (TNF-α) after acute exercise. It has been shown in animals that IL-1 and TNF increase SWS. Vigorous exercise has resulted in great increases in plasma concentration of IL-1 and IL-6 and TNF in some studies, while moderate exercise increased the concentration of these cytokines only to moderate values. However, the latter promotes drowsiness, whereas a higher concentration of cytokines is associated with increased night-time wakefulness. This is consistent with studies that show that ultra-endurance activity can increase night-time wakefulness. According to studies, it is plausible that elevated cytokines may affect sleep directly or through restorative thermoregulation (Buman & King 2010).

4.1.6. Brain neurochemistry

Brain neuropeptides and neurotransmitters can affect sleep cycles directly and indirectly. Firstly, directive pathways suggest for role of orexin, catecholamines and serotonin. Orexin has a central role in the sleep-wake cycle and its deficiency in dogs and mice can cause narcolepsy. It is an important neuropeptide for energy homeostasis (spontaneous PA, energy expenditure and feeding behaviour). Another factor that could contribute to fatigue and thus affect sleep is the interaction of catecholamines and serotonin. However, it is unlikely that a single peptide or neurotransmitter accounts for sleep improvements through exercise (Buman & King 2010).

Secondly, exercise can indirectly affect sleep because it changes factors that interfere with sleep and thus improves sleep quality and duration. To illustrate, exercise improves functional capacity, controls weight, improves health-related quality of life and reduces medication. These parameters often disrupt sleep and changing those would affect sleep in an indirect matter. Moreover, exercise often changes behaviours (reduced smoking, substance use), which could also affect sleep. In summary, a number of pathways are plausible, but it is most likely that effects are a result of multiple mechanisms (Buman & King 2010).

4.2. Effect of exercise on sleep

There is no consensus whether or to what extent PA affects sleep patterns. Studies show conflicting evidence whether PA is favourably associated with physiological sleep function (Brand et al. 2010). It is difficult to compare studies due to different training modalities, volumes and methods used for assessment. What is more, it is challenging to determine the effect of exercise on healthy people, because their sleep is usually close to optimal, leaving little room for improvement (Hague et al. 2003). Epidemiologic studies usually show favourable associations (Youngstedt & Kline 2006; Youngstedt 2005; Buman & King 2010), however, the level of evidence cannot be as strong as with experimental research.

Review by Buman & King (2010) found out that aerobic and resistance exercise at level of national recommendations appears to be sufficient to improve subjective sleep quality. Additional exercise above guidelines may benefit sleep further. It was emphasized that older adults are an important target population for intervention given decline in sleep quality with age. Exercise up to 4-8 hours before bedtime may be optimal, yet exercise any time of the day is also beneficial (Buman & King 2010).

4.2.1. Epidemiologic studies

Epidemiologic studies have an advantage over experimental studies being they can include larger sample sizes. Youngstedt and Kline (2006) and Buman and King (2010) wrote in their reviews that epidemiologic studies consistently report significant positive associations between self-reported exercise and subjective sleep patterns. In a review by Youngstedt (2005), it was found out that many epidemiologic studies indicate a positive association between exercise and sleep, and suggest that PA is one of the most effective behaviours to promote good night sleep. In a more recent review, it was concluded that exercise significantly improves apnoea-hypopnea index, overall sleep quality, sleep latency and subjective sleep quality (Kelley and Kelley 2017). According to the “Sleep in America” poll by the National Sleep Foundation (2003) older participants who reported to exercise more than once per week had better overall sleep, less difficulty falling or staying asleep and less daytime sleepiness compared to participants who exercised less than one time per week. The latter had more complaints

on almost every index of sleep. In addition, Wennman, Kronholm and Partonen (2014) suggest that higher leisure-time physical activity was correlated with better sleep. When people are given an open question about habits that help them fall asleep or practices to promote quality of sleep, responders state physical activity as the most common (Youngstedt & Kline 2006). For example, every third respondent in an epidemiological survey by Urponen, Vuori, Hasan and Partinen (1998) felt that PA had a positive impact on sleep. Authors emphasized that PA is one of the most commonly reported behavioural factors that promote sleep. Moreover, they found that temporary lack of exercise seemed to impair quality of sleep. Many epidemiological studies support the association between lower quantiles of exercise with insomnia (Youngstedt & Kline 2006). Regression analysis from a study in Japan (Kim et al. 2000) showed insomnia odds ratio of 1.3 associated with no habitual exercise. This is comparable to being unemployed and unable to cope with stress. Moreover, Inoue and colleagues (2013) concluded that any type of physical activity, at work walking or intentional exercise, was associated with lower prevalence of insomnia.

Notwithstanding, other studies show no effect of PA on sleep, regardless of intensity (Kakinami et al. 2017). One study observed college students who were normal sleepers and failed to support epidemiologic data regarding exercise value on sleep. They found no correlation between PA and sleep parameters (Youngstedt et al. 2003). Results of the study are consistent with evidence that show only modest effect of exercise on sleep.

4.2.1.1 Subjective and objective assessment of physical activity and sleep

Many epidemiologic studies use subjective perception of PA and quality of sleep. Kakinami and colleagues (2017) warn that subjective measures of PA usually overestimate PA, nevertheless they are still considered clinically useful and well-validated tool. For instance, the following study compared subjective and objective measures of sleep and PA. 56 adolescent vocational school students participated in the study and wore accelerometers, slept with EEG device and were also given questionnaires regarding sleep and PA. It was concluded that subjective levels of PA are good predictor for self-reported sleep, and objective PA has a higher influence on objective sleep. Nevertheless, objective levels of PA are less predictive of favourable

sleep patterns in general (Lang et al. 2013). It is plausible that the association between sleep and PA has less to do with behavioural patterns than individual self-perception of being physically active (Lang et al. 2013).

On the other hand, some studies show that objective measures of PA are associated with better subjective sleep patterns. To illustrate, in a study by Loprinzi and Cardinal (2011) 3081 participants wore actigraph for 7 days and after that they were asked questions about their sleeping habits. Participants in their study who met national guidelines regarding PA (at least 150 minutes of moderate-intensity exercise or 75 of vigorous exercise per week) were less likely to feel sleepy during the day, have leg cramps during sleeping and had less difficulty concentrating, compared to participants being less active. Authors summarized that objectively measured PA was positively related with general productivity sleeping related parameters. Similarly, in a study by Erlacher, Erlacher and Schredl (2015) they showed significant beneficial effects of objective measure of exercise on self-rated sleep among adults with chronic sleep problems. About 50% of their participants said that a 6-week PA intervention had an effect on sleep improvement. The number of steps was related to improvement in PSQI (average of 3.1 point) score, which suggests that more steps benefit sleep quality.

4.2.1.2. Limiting factors of epidemiologic studies

In spite of positive associations between sleep and exercise in epidemiologic studies it is important to note that the effect of exercise on sleep is modest compared to depression and stress (Youngstedt & Kline, 2006). In general, the majority of prior epidemiologic research has applied supportive view that acute and chronic exercise promote sleep. Regardless of the positive associations, epidemiologic studies cannot be considered as conclusive due to many limiting factors. Firstly, these large sample studies cannot control confounding factors that could affect sleep parameters such as nutrition, daylight exposure, tobacco use, mental health etc. (Youngstedt 2005). Secondly, epidemiologic studies usually rely on self-reported measure of PA and sleep which has unknown validity. Finally, the most important issue when analysing epidemiological studies, is that causality cannot be inferred from associations. Thus, less sleep could be a cause of less exercise and not vice versa (Youngstedt & Kline 2006).

4.2.2. Experimental studies

Experimental studies, in contrast to epidemiological, show a smaller effect of exercise on sleep (Youngstedt & Kline 2006). Driver and Taylor (2000) reviewed two meta-analyses, and they showed small effects of acute exercise on sleep. Effects generally resulted in increased TST (ES 0.31-0.41), prolonged REM latency (0.29-0.52), decreased REM sleep (0.14-0.49) and increased SWS (0.22). All of the effects were modest in size, however their subjects consisted of good sleepers. Similarly, a study on adolescents suggests that the amount of exercise predicts slow wave sleep and decreases REM-sleep (Brand et al. 2010). Adolescents who exercised more had longer sleep time, higher SE, more stage shifts, less S2 sleep and light sleep, increased stage 4 and SWS and decreased REM-sleep. Moreover, more weekly exercise was correlated to better subjective sleep (Brand et al. 2010). A more recent systematic review and meta-analysis reports that in middle aged women, low- to moderate-level of PA, with intervention lasting from 12-16 weeks, had a positive effect on subjective sleep quality (Rubio-Arias et al. 2017).

4.2.2.1. Intervention studies

A considerable body of experimental literature exists on good sleepers and this could impose a potential limitation. In many studies done on healthy subjects, exercise interventions did not correlate with better sleeping characteristics (Oudegeest-Sander et al. 2013). Because healthy individuals have good sleep habits at baseline it is hard to determine effect of exercise on their sleeping patterns since there is not much room for improvement. One way to assess this phenomenon is to decrease PA of highly active people. One study recruited 16 healthy athletes and assessed their sleep after a full sedentary day. Their SWS decreased for an average of 15.5 min, REM increased by mean of 17.9 min and SOL decreased for 24 min. There was no difference observed between conditions for TST, SE, WASO or foot temperature. Authors concluded that reducing physical activity in sports people has clear physiological consequences that affect depth of sleep. Not exercising can alter sleep pattern, possibly through effects of thermoregulation (Hague et al. 2003).

When studies are done on older individuals that generally have poorer sleeping patterns, the effects of exercise are more visible (Dolezal et al. 2017). An older study showed that a 16-week intervention, consisting of 30-40 min endurance sessions, improved self-rated sleep quality in older adults with moderate sleep complaints. The study concluded that older adults that have moderate sleep complaints can improve subjective sleep quality by engaging in a moderate-intensity exercise program (King et al. 1997). A more recent study supported this conclusion, and stated that acute exercise promotes sleep quality (Dolezal et al. 2017). Jose et al (2016) completed an exercise intervention and assessed the influence of different types of exercise (aerobic, anaerobic and resistance training and control group) on sleep quality of hypertensive elderly subjects. It was concluded that both types of exercise improved sleep quality by reducing sleep fragmentation index, increasing the percentage of minutes motionless and increasing sleep efficiency.

4.2.2.2. Limitations

There are several possible explanations why some experimental studies fail to show an association in contrast to epidemiologic studies. When sleep is being assessed by subjective measure, psychological rather than physiological parameters are being assessed. Secondly, if participants are good sleepers at the baseline, a ceiling effect can be observed. That means that there is little room for improvement (Youngstedt et al. 2003). Another limitation to experimental studies include small sample sizes that do not provide strong statistical evidence (Youngstedt & Kline, 2006). Finally, it is difficult to draw conclusions from studies and generalize the effects of exercise on sleep, because studies often differ regarding exercise variables (type, duration, intensity and time of day).

4.2.3. Acute exercise

Comparing regular versus acute exercise, the first shows more promising effects regarding its effect on sleep. In acute exercise, the association is not well defined (Driver & Taylor 2000; Youngstedt & Kline 2006), however small benefits on sleep are seen (Kredlow et al. 2015).

After exercise, the feeling of fatigue may be perceived as sleepiness and interpreted as facilitating sleep (Driver & Taylor 2000). Thus, some studies suggest that positive effects of exercise on sleep happen only when exercise is conducted close enough to bedtime to stimulate a thermoregulatory response (Driver & Taylor 2000). Other mechanisms behind the beneficial effects of acute exercise could be related to energy conservation and tissue restitution (Driver & Taylor 2000).

Most previous research that studied the effect of acute exercise session on sleep parameters showed low to moderate effects. A study that assessed the effect of exhaustive exercise on men and women found out that changes in sleep architecture happen primarily in the early proportion of the night. Individuals increased SWS and REM latency in the first cycle, decreased first REM duration and had moderate increase in stage 4 and total SWS time (Bunnell, Bevier and Horvath 1983). In the same fashion, other studies showed increased SWS, REM sleep latency (Flausino et al. 2011), decreased REM sleep (Driver & Taylor 2000) and increased TST, which increases SE (Youngstedt, O´Connor & Dishman 1997; Kredlow et al. 2015). Research done on physically fit adults showed that the proportion of NREM sleep in general was greater after exercise day (Myllymäki et al. 2011). Similarly, in newer research done by Dworak and colleagues (2008) acute exercise 3-4 hours before bedtime elevated SWS, decreased S2, increased SE and decrease SOL in children.

Results from a meta-analysis in 2015 showed that acute exercise had small beneficial effects on TST, SOL and SWS. The latter change was more significant when participants did cycling rather than running (Kredlow et al. 2015). Because exercise also was shown to reduce sleep disturbances, SE was better. In general, there were no significant changes observed in REM latency, stages 2-4 or number of awakenings, yet exercise affected REM duration in a way that was associated with shorter REM sleep (Kredlow et al. 2015).

Another way exercise can be implemented into the daily life is walking. This low-intensity PA is accessible to everyone and often underrated. It can be measured by commercial devices that track the number of steps participants make in a day. Daily steps have thus become a meaningful metric how people can measure their activity and track progress. To this date, it still remains unknown whether daily steps are related to improved sleep quality. On the other hand, a recent study suggests that there

is an association between daily steps and sleep. Sullivan, Robinson and Lachman (2019) report that women who took more steps reported better sleep quality than those who were less active. Moreover, when the analysis was done for within person comparison, it showed that when participants were more active than average, they also reported better sleep quality and duration (Sullivan, Robinson and Lachman 2019). When sleep and physical activity were measured by actigraphy, a positive correlation was found between activity count and increased TST (Kishida & Elavsky 2016). On the contrary, Youngstedt et al (2003) found no evidence of within-person associations between physical activity and sleep in healthy and active adults.

4.2.4. Regular exercise

Many chronic exercise studies do not show compelling evidence about exercise promoting sleep; however, a lot of them used good sleepers as subjects with little room for improvement. Therefore, if participants are good sleepers at baseline, the effect of exercise is small to modest (Youngstedt 2005). In contrast, studies done on individuals with sleeping problems or older individuals show significant improvement in self-reported sleep parameters due to exercise (Youngstedt 2005). Kredlow and others (2015) show a large and significant effect size of regular exercise on sleep quality in a sample of individuals with sleep complaints. Likewise, in an experimental study by Erlacher, Erlacher and Schredl (2015) number of steps and physical activity were significantly related to the improvement of subjective sleep quality in adults with chronic sleep complaints. It was reported in many studies that participation in an exercise training program had moderately positive effects on subjective and objective sleep quality in middle-aged and older adults with sleep problems (Yang et al. 2012; King et al. 2008). Participants who exercised had significantly better global PSQI, reduced sleep latency and medication use (Yang et al. 2012). It has to be noted that the exercise intervention needs to be of sufficient duration to allow for changes in sleep patterns, however, people should be cautious of overtraining since this can lead to increased fatigue and disrupted sleep (Driver & Taylor 2000).

Higher levels of PA are associated with better health outcomes and lowering level of sedentary behaviour can have positive effects on sleep. In the recent years, general PA level, and not only structured exercise, has been recognized as increasingly important.

For example, decreasing sitting time can have positive effects on health status and quality of life. In a recent study that involved 658 participants, authors wanted to assess whether PA and sedentary behaviour is associated with sleep quality and quantity in younger adults. They found that each additional hour of TV and computer use per day was associated with 17% and 13% increase in the odds of poor sleep quality. After adjusting for PA, the association remained significant with odds ratio of 1.15. On the contrary, sedentary behaviour was not associated with sleep quantity. They concluded that more sedentary activity was associated to poorer sleep quality but sleep was not associated with PA (Kakinami et al. 2017).

Data from many studies show sleep-promoting effects of chronic exercise or improved fitness on sleep. Large beneficial effect from exercise was seen in increased sleep quality and efficiency (Kredlow et al. 2015), because TST and amount of SWS increased (Driver & Taylor 2000). Exercise also has small-to-medium effects on SOL and significantly moderate-to-strong effects on all subscales of PSQI (Kredlow et al. 2015).

On the other hand, some studies did not find difference in sleeping patterns after exercise intervention (Harp 2015). However, there was improved sleep quality seen in overweight and obese individuals. This is likely due to the decrease in body fat percentage from participating in regular exercise (Harp 2015). Studies suggest that the exercise programme (chronic PA to affect sleep) should be of sufficient duration, more than 8 weeks, to induce positive effects on sleep (Driver & Taylor 2000).

4.3. Exercise variables affecting sleep

There is a wide choice of exercise and PA parameters that could affect sleep available in the literature. It is therefore hard to draw conclusions, because studies do not use standardized exercise protocols to examine effect on sleep. According to Youngstedt, O´Connor and Dishman (1997), exercise duration and time of day were the most consistent moderator variables on sleep.

4.3.1. Mode

Mode refers to type of exercise. Most experimental studies used aerobic exercise as their physical activity intervention to investigate its effect on sleep. It is plausible that aerobic exercise affects sleep more than other modes of exercise, since Driver and Taylor (2000) found that endurance athletes had the highest and power-training group the lowest level of SWS. However, less experimental research is done on resistance training. One mechanism that could contribute to better sleep after a resistance session is stimulation of growth hormone (GH) secretion, which is pronounced during SWS (Buman & King 2010). Two randomized control trials used high intensity progressive resistance training and found favourable effects for sleep quality. Results for lower intensity were modest which could suggest a dose-response pattern (Buman & King 2010). Two older studies showed that weight-lifting exercise intervention was effective in improving subjects' sleep quality and overall quality of life (Singh, Clements & Fiatarone 1997), and that resistance training increased SWS (Browman 1980). In accordance with these results, a study from 2015 shows that resistance training promotes sleep (Erlacher, Erlacher & Schredl 2015). Not only resistance or cardiovascular training, but also other exercise interventions, such as walking, yoga, Tai Chi, Baduanjin, worksite exercise and mind-body, show promise as effective modalities to improve sleep (Erlacher, Erlacher & Schredl 2015; Dolezal et al. 2017). Likewise, a recent review suggests that all forms of exercise, moderate-intensity aerobic, resistance training, mind-body exercise, produce better sleep quality measured by PSQI or wrist actigraphy (Dolezal et al. 2017).

4.3.2. Volume, frequency and duration

It is possible that many studies have not found an effect of exercise on sleep because their volume was too low considering their participants' baseline level of PA. To illustrate, sedentary people have more general benefits after joining PA programs since their overall fitness is low and room for improvement is high. Study by Sherrill, Kotchou and Quan (1998) showed that even low-intensity activity, such as walking is significantly associated with better sleep patterns and less sleep complaints. However, if already fit subjects join the program, they will need higher stimulus to produce

adaptations and improvements. Same thinking can be applied to sleep. A meta-analysis by Youngstedt, O´Connor and Dishman (1997) showed that exercise volume based on national PA guidelines acutely improves SWS, REM, TST and stage 2. Similarly, Diver and Taylor (2000) state that exercise lasting more than one hour daily results in the most reliable effects for increased TST, REM latency and decreased REM sleep. It was found that duration is more consistent for moderating sleep variables on acute effect of exercise than other factors such as fitness, time of day etc. Kredlow et al. (2015) report that longer exercise duration increases the magnitude of beneficial effects on sleep for TST, SWS, sleep onset latency and stage 4. In another case, when exercise was conducted for a shorter amount of time, for example less than 1 hour, negligible effects were seen on sleep (Youngstedt, O´Connor & DIshman 1997).

Epidemiologic studies often correlate higher frequency and/or volume to better sleeping patterns. In the poll by National Sleep Foundation (2003) responders who exercised more than 3 times per week had better sleep results than those who exercised only 1-2 per week, which favours dose-response hypothesis. In addition, many clinical trials that exceeded the recommended values showed greater effects on sleep. This suggests a dose-response effect of exercise on sleep, however it is not known if this is due to the increased duration, intensity or both (Buman & King 2010). On the contrary, a recent meta-analysis failed to show significant effects of higher exercise frequency per week on any sleep outcomes (Kredlow et al. 2015)

A number of questions regarding the effect of exercise intensity on sleep remain to be addressed since studies show inconclusive results (Driver & Taylor 2000). Some researchers argued that intensity was not associated with sleep changes (Erlacher, Erlacher & Schred 2014; Kredlow et al. 2015), while others reported that vigorous PA tends to predict good sleep better than moderate PA (Lang et al. 2013). According to their study, the optimal exercise intensity for a favourable sleeping pattern is vigorous PA because it produces greater positive effects than moderate. Likewise, Dolezal and colleagues (2017) found that individuals with greater perceived exhaustion during exercise had better effects on objective sleep parameters.

It is difficult to examine only the effect of exercise intensity on sleep since there are other covariates included. Nevertheless, some activity is always better than nothing and Driver and Taylor (2000) state in their review that high and moderate intensity to

exhaustion both resulted in increased SWS compared to rest. In general, long duration and intensity can represent a dose response on sleep, so more PA is always encouraged (Driver & Taylor 2000; National Sleep Foundation, 2003).

4.3.3. Timing

There are controversial findings regarding late night PA, but the American Academy of Sleep Medicine (AASM) indicates that vigorous late-night exercise increases arousal and disturb sleep (American Academy of Sleep Medicine 2001). A study by Bulckaert and colleagues (2011) found out that mild PA up to 1 hour before sleep decreases parasympathetic dominance, and can thus have a negative influence on sleep quality. There are many mechanisms that could contribute to the phenomena; however, they should be studied more in depth. A study by Dolezal and others (2017) suggests that working earlier in the day is more beneficial for sleep parameters, and Sayk and colleagues (2015) found that the timing of exercise is important when assessing sleep quality. Similarly, Fairbrother et al (2014) found that early morning exercise, in comparison to afternoon or evening session, might be the most beneficial for sleep quality and improvement of nocturnal blood pressure in prehypertensive individuals. Additionally, a study on postmenopausal women found out that only exercise before dinner is beneficial for sleep parameters, whereas participants that exercised later in the evening had more trouble falling sleep (Tworoger 2003).

However, in spite of common beliefs, most studies have impaired to find that late night exercise impairs sleep quality (Flausino et al. 2011). For example, Myllymaki et al (2011) found that late-night exercise does not influence sleep quality, but it can affect cardiac autonomic control of the HR at the beginning of sleep. Alley et al (2015) investigated how 30-minute of resistance training during different times of the day (7 AM, 1 PM and 7PM) affects sleep architecture and nocturnal blood pressure in college-aged subjects compared to control days (without exercise). All exercise conditions resulted in less time awake after sleep onset compared to the control day, with the 7PM session resulting in the most significant decrease compared to the control. Comparing results from different exercise times in the day, there were no differences in sleep architecture or nocturnal BP. Authors concluded that resistance training at any time of the day might improve sleep quality and nocturnal BP compared to non-exercise day.

In the same fashion, Youngstedt and Kline (2006) emphasize that experimental studies failed to support the assumption that late-night exercise impairs sleep. These findings seem to contradict long-standing sleep hygiene tips that advise not to exercise before bedtime. It is thus recommended to exercise without focusing on the timing, as long as planned PA is not at the expense of sleep duration (Chennaoui et al 2015). Likewise, according to the guidelines (Physical Activity Guidelines for Americans 2nd edition 2018) benefits of exercise on sleep are similar for exercise performed more than 8 hours before bedtime, 3-8 hours before or less than 3 hours before bedtime.

4.3.4. Fitness status

Cardiorespiratory fitness (CRF) provides an objective surrogate measure of change in habitual physical activity exposure (Dishman et al. 2015). Buman & King (2010) reported that there is an association between CRF and sleep quality when participants are unfit and sedentary. On the contrary, the sleep improvements are limited by a ceiling effect in fit subjects. Dolezal and colleagues (2017) comment in their review that improving cardiorespiratory fitness could be a useful intervention for improving sleep. Mota and Vale (2009) examined the association between sleeping quality and CRF in adolescent girls and found a significant correlation. Likewise, other studies show a correlation between higher CRF and sleep quality (Lee & Lin 2007; Shapiro et al. 1984).

Driver & Taylor (2010) report improved TST, SOL and time awake when physical fitness was improved. In a cross sectional study by Strand and colleagues (2013) they found an inverse relationship between CRF and symptoms of insomnia, independent of age, BMI, cardiovascular risk, alcohol and depression and anxiety.

When comparing athletes with controls, they tend to have better sleeping patterns than unfit population (Brand et al. 2010; Buman & King, 2010). One study on older men compared the sleep patterns of fit and sedentary subjects without sleep complaints. Results showed that fit subjects had shorter onset latencies, less wake time after onset, less stage 1, higher sleep efficiency and more SWS. Authors concluded that exercise and fitness might have a significant effect on sleep of older man (Edinger et al. 1993).

The limitation of the former study is not manipulating the exercise parameters. Thus, it is possible that fit subjects exercise more because they sleep better and not vice versa.

Nonetheless, Dishman and colleagues (2015) carried out a longitudinal study where they were tracking changes in CRF and its association to sleep complaints. They found out that each minute of decline in treadmill endurance, which is equivalent to decline in CRF of 0.5 MET, between ages 51 and 56 increased the odds of sleep complaints by 1.7% in men and 1.3% in women. Similarly, intervention studies in sedentary subjects showed that as participants increased their CRF levels, they concurrently increased sleep quality. In a study done on overweight/obese sedentary women, those who increased their VO_{2max} more had longer sleep duration, were less frequently falling asleep during activities and used less sleep medication (Tworoger et al. 2003).

On the other hand, one older study done on young women fails to demonstrate positive effects of increased CRF on sleep. The authors suggest that increased fitness might only facilitate sleep when there is a concurrent increase in lean body mass, which did not happen in their study (Meintjes, Driver & Shapiro 1989). Similarly, many studies on fit subjects do not show strong associations; nonetheless this can be due to the ceiling effect (Oudegeest-Sander et al. 2013; Buman & King 2010). Physically fit individuals tend to be good sleepers in general so there is little room for improvement (Buman & King 2010). Seven longitudinal studies were done on good sleepers using objective sleep parameters, such as PSG. Participation in an exercise programme that improved fitness resulted in improved sleep quality in some studies (longer sleep duration, shorter SOL and higher levels of SWS), but remained the same in others. Thus, there is still no clear evidence if improved fitness per se facilitate better sleep in good sleepers at baseline (Driver & Taylor 2000).

5. PURPOSE OF THE STUDY

The aim of this master thesis is to evaluate the association between physical activity and sleep characteristics in hypertensive individuals.

Research question 1: "Does regular PA promote sleep in hypertensive individuals?"

Hypothesis 1: Regular PA has positive effects on subjective sleep quality measured with Pittsburgh Sleep Quality Index.

Studies based on questionnaire data suggest that people who are more physically active have less complaints regarding sleep and better PSQI score (Buman & King 2010; "Sleep in America" poll by the National Sleep Foundation 2003; Yang et al. 2010). Furthermore, aerobic and resistance exercise at levels that meet the national recommendations are sufficient to improve subjective sleep quality (Buman & King 2010). In a meta-analysis by Kredlow et al. (2015) and a review by Chennaoui et al. (2015) authors concluded that regular exercise had significant beneficial effects on all subscales of PSQI. In middle-aged women, low to moderate levels of physical activity had positive effects on subjective sleep quality (Rubio-Arias et al. 2017).

Hypothesis 2: Regular PA has positive effects on objective sleep quality parameters; sleep duration, REM duration, REM %, NREM duration, NREM %, SOL duration, sleep efficiency, S1 duration, S1 %, S2 duration, S2 %, S3 duration, S3 % and number of awakenings that last more than three minutes.

The literature suggests that PA has, in general, small effects on sleep parameters in the general population (Buman & King 2010). A meta-analysis by Kredlow et al (2015) reveals that regular exercise has small beneficial effects on TST and SE, small-to-medium beneficial effects on SOL, and moderate beneficial effects on sleep quality. In an older meta-analysis, it was concluded that chronic exercise increases SWS, TST, decreases REM sleep, SOL and WASO in good sleepers (Kubitz et al. 1996).

Research question 2: "Does acute physical activity based on the number of daily steps promote sleep in hypertensive individuals?"

Hypothesis 3: The number of steps is positively associated with subjective sleep quality measured with Pittsburgh Sleep Quality Index.

Erlacher and colleagues (2015) found that the number of steps per day and the duration of PA are associated with improved subjective sleep. A recent review (Banno et al. 2018) reports that regular exercise was beneficial for sleep quality measured by PSQI, for the participants who were diagnosed with insomnia. In the same manner, Kelley and Kelley (2017) conclude that exercise significantly improves subjective sleep quality. Sullivan, Robinson and Lachman (2019) report that monthly and daily physical activity were associated with positive subjective sleep outcomes.

Hypothesis 4: The number of steps is positively associated with objective sleep parameters; sleep duration, REM duration, REM %, NREM duration, NREM %, SOL duration, sleep efficiency, S1 duration, S1 %, S2 duration, S2 %, S3 duration, S3 % and number of awakenings that last more than three minutes.

The effects of exercise on sleep in healthy individuals are small to medium. According to the meta-analysis and systematic reviews, acute exercise has small beneficial effect on TST, SOL, SE, S1 duration and SWS. Exercise also prolongs REM latency, decreases REM duration (Kredlow et al. 2015; Driver & Taylor 2000; Youngstedt, O´Connor & Dishman 1997; Kubitz et al. 1996) and reduces the severity of insomnia (Banno et al. 2018). Likewise, Myllymäki and colleagues (2011) have found that exercise increases the proportion of the NREM sleep. In a recent study, Kashida and Elavsky (2016) report positive association between PA and increased TST.

Research question 3: "Is higher CRF associated with better sleep characteristics in hypertensive individuals?"

Hypothesis 5: Higher CRF is associated with better subjective sleep quality measured with Pittsburgh Sleep Quality Index.

Increased fitness was associated with better subjective sleep in postmenopausal women (Tworoger et al. 2003). In a younger population, adolescent girls who were classified as fit were twice as likely to report better sleep quality (Mota & Vale 2009). Similarly, female college students with lower levels of CRF reported worse sleep quality measured by PSQI (Lee & Lin 2007). Cross-sectional analysis in a study by Strand and others (2013) showed that there was an inverse relationship between CRF and insomnia symptoms. Similarly, Dishman and colleagues (2015) found that the odds of sleep complaints increased by 1.7% in men and 1.3% in women for each minute of decline in a treadmill performance.

Hypothesis 6: "Higher CRF is associated with better objective sleep parameters; sleep duration, REM duration, REM %, NREM duration, NREM %, SOL duration, sleep efficiency, S1 duration, S1 %, S2 duration, S2 %, S3 duration, S3 % and number of awakenings that last more than three minutes.

Fit subjects had shorter SOL, less WASO, less stage 1 sleep, higher SE and more SWS than sedentary subjects (Edinger et al. 1993). Driver & Taylor (2010) wrote that when physical fitness is improved, it improves TST, SOL and time awake during the night. Shapiro and colleagues (1984) found that increases in fitness among eight army recruits decreased SOL, WASO and improved SE.

6. METHODS

This Master Thesis was part of a bigger project "HealthBeat - Heart rate variability analytics to support behavioural interventions for chronic disease prevention and management". The project was a collaboration between the University of Jyväskylä, Central Finland Health Care District and Firstbeat Technologies. Data collection was conducted between September 2018 and March 2020. The purpose of the project was to assess individuals that had type 2 diabetes and/or increased risk of cardiovascular conditions to examine their stress reactions and physical fitness. Thus, it would be possible to develop a computerized assessment of physical activity, fitness, recovery and stress.

6.1. Subjects

Participants were recruited by online and local advertising and targeting personnel that worked in health care centres to inform their patients about the study. In total, 76 subjects were recruited for the HealthBeat project and have gone through participation screening. Before the participation, each subject gave written consent to complete the study.

The inclusion criteria were 1) age between 18 and 64 years old, 2) BMI under 40kg/m^2, 3) diagnosed arterial hypertension, and 4) sufficient physical fitness to safely perform exercise tests.

The following exclusion criteria for the study were applied 1) specific medications (tricyclic antidepressants, insulin, serotonin and noradrenaline reuptake inhibitors and β-blockers), 2) hypertension-mediated organ damage, 3) valvular heart disease, 4) continuous atrial fibrillation, 5) secondary hypertension, 6) ischaemic heart disease, 7) pacemaker, 8) severe heart disease, 9) cardiac bifurcation, 10) balloon dilated or bypassed coronary artery disease, 11) left bundle branch block, 12) diabetic neuropathy, retinopathy or nephropathy, 13) heart failure, 14) chronic obstructive pulmonary disease, 15) cerebrovascular disease, 16) unstable asthma, 17) cancer, 18) anaemia, 19) uncontrolled thyroid disorder, 20) any type of psychiatric illness, 21) physical function deficit, 22) pregnancy or breastfeeding, 23) substance abuse, and 24)

obstructive sleep apnoea that requires continuous positive airway pressure and 25) chronic neurological disease (multiple sclerosis, Alzheimer's, Parkinson's).

Of the 76 subjects, 12 were excluded due to not having hypertension, two were excluded after health screening, three subjects withdrew before the cardiopulmonary exercise test, one subject was excluded after the cardiopulmonary test, 10 had asthma and 4 subjects were excluded due to smoking.

In total, 45 hypertensive participants (36 female, 9 male) were included in the study and their age ranged from 34 to 64 years (M= 51.5, SD = 8.8). Mean (±SD) systolic blood pressure was 135 mmHg (14) and diastolic blood pressure was 84 mmHg (7). Subject characteristics are shown in TABLE 1. The subjects were classified into three groups three times based on their amount of PA weekly, the number of steps and CRF result.

TABLE 1. Subject characteristics.

Age (years)	Height (cm)	BMI (kg/m2)	Systolic BB (mmHg)	Diastolic BB (mmHg)
51.5 ± 8.8	167.7 ± 8.9	27.7 ± 3.8	135 ± 14	84 ± 6

PSQI (global)	Sleep duration (min)	SWS %	weekly PA (min)	Daily steps	CRF (ml/min/kg BM)
5.8 ± 3.0	414.1 ± 44.6	17 ± 6	177 ± 124	4890 ± 2589	32 ± 6.6

6.2. Study design

Each participant went through six visits in one month. On the first day, subjects underwent health screening and examination by a general physician. Next, blood samples were taken in a fasted state. To examine their physical fitness, subjects performed a cardiopulmonary exercise test and self-paced 30-min walk test. Approximately a week after the physical fitness tests, nocturnal sleep structure was assessed. Finally, on the last visit, subjects underwent a psychological stress test. For the thesis, the data was collected from visits one, three, and five.

6.2.1. Health screening

Participants were examined by a general physician and a nurse during the first visit. Firstly, they reported on their medical history (oral anamnesis and preliminary questionnaire), and later their clinical status was assessed. Resting blood pressure (Maxi Stabil 3, Welch Allyn GmbH & Co. KG, Jungingen, Germany) was taken. Physical examination and health screening primarily focused on assessing signs and symptoms of individuals that would show cardiovascular, metabolic or renal diseases. Subjects were also asked about their current level of PA and desired exercise intensity.

6.2.2. Physical activity

Regular PA was assessed based on a series of questions covering leisure-time PA. Participants were asked questions regarding frequency, intensity and duration of exercise. Subjective habitual PA volume was calculated as average frequency (times per week) and duration (min). Total PA volume was expressed as the sum score of minutes per week. Physical activity intensity was defined based on the question "Is your physical activity during leisure time about as tiring as". Walking and alternatively walking and jogging was coded as moderate exercise and jogging and running were coded as vigorous exercise. Participants that reported more than 300 min of moderate exercise intensity or more than 150 min of vigorous exercise intensity per week were classified as high exercisers. Subjects that completed between 150 - 300 min of moderate and 60-150 min of vigorous exercise per week were classified as average exercises and participants with less than 150 min of moderate exercise per week were classified as low exercisers (Physical Activity Guidelines for Americans 2nd edition, 2018). Thus, 12 participants were classified as low, 20 participants as average and 13 participants as high exercisers.

Objective PA was based on the number of steps the participants walked per day. Subjects wore digital device (Firstbeat Bodyguard 2) for 3 consecutive days, which recorded the number of steps per day. The step count of the day when polysomnography measurement was done was used for the analysis. Participants were divided into 3 groups based on tertiles. Low step count participants performed less

than 3477 steps per day, average step count participants performed between 3478 and 5988 steps and high step count participants performed more than 5989 steps daily.

6.2.3. Cardiorespiratory fitness

To measure cardiorespiratory fitness of the participants, an exercise test on the treadmill (JUOKSUMATTO OJK-1, Telineyhtymä, Kotka, Finland) was done following the USAFSAM protocol (Wolthuis et al. 1977). Participants were told to abstain from eating and drinking stimulative drinks (coffee, tea, cola) for at least two hours prior to the test and to avoid strenuous physical activity and alcohol for at least 36 hours before the test. The bioimpedance device (InBody770, InBody Co., Ltd., Seoul, South Korea) was used to measure weight and body composition. After 5-minute rest in a supine position arterial pressure from the left brachial artery was measured (SunTech Tango M2, SunTech Medical, Inc., Morrisville, USA).

After 5 min standing rest, subjects started walking on the treadmill for 3 minutes at 3.2 km/h with 0% incline. After the first 3 minutes, the walking speed was increased to 5.3 km/h, and it stayed at the same level throughout the test. The incline was increased by 5% every 3 minutes until volitional task failure. Each participant rested for 5 minutes after the test, 1 minute in a standing position and 4 minutes in a supine position.

The determination of VO_2peak was the highest value of a 30-second average VO_2 interval (American Thoracic Society & American College of Chest Physicians 2003). Value for VO_2 was examined as body-mass adjusted to avoid effects of between-group differences (Lolli et al. 2017). Participants were divided into three groups based on the ACSM classification of VO_2max results according to age and gender. 10 participants had poor and fair VO_2max result, 12 participants had good VO_2max max result and 23 participants achieved excellent VO_2max result.

6.2.4. Subjective sleep quality

Data for subjective sleep quality was collected from the Pittsburgh Sleep Quality Index (PSQI) questionnaire (Buysse et al. 1989). It is a self-rated questionnaire to assess sleep quality and disturbances over a 1-month interval. PSQI consists of 19 individual questions that are categorized into seven different components: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication and daytime dysfunction. Each of the components is weighted equally on a 0-3 scale and in the end, they are summed together to yield a global PSQI score which ranges from 0-21. A higher score indicates poor sleep quality (Buysse et al. 1989).

6.2.5. Objective sleep quality

Sleep structure was assessed with portable polysomnography device SOMNO HD™ (Germany). The sleep measurement was conducted following the AASM guidelines. Electroencephalography (EEG) electrodes were placed based on the international 10-20 system to frontal (F3, F4), central (C3, C4), occipital (O1, O2) position. Mastoid electrodes (M1, M2) were placed as a reference electrode. Two electrodes (E1, E2) were placed around the eyes to record EOG. To record EMG, 3 electrodes were placed on the chin (EMG1, EMG2, EMG3). Electrocardiogram (ECG) was recorded with two ECG electrodes. The data from electrodes was recorded at a sampling rate of 512 Hz for EEG, EMG and ECG. The start of the measurement was set manually from each participant when they went to sleep and stopped the next day when they woke up. Sleep stages were scored according to the AASM guidelines (American Academy of Sleep Medicine 2001).

6.3. Statistical analysis

Participants were divided into three groups three times, based on the level of physical activity, the number of steps per day and the result of CRF test. Initially, descriptive statistics for each dependent variable was measured based on three group classifications. Before testing for differences in sleep parameters, differences in

subject characteristics between groups were tested with one-way ANOVA. To investigate significant differences between groups for a specific subject characteristic, Tukey HSD post hoc test was used.

For each of the groups, the normality of the distribution of dependent variables was tested with the Shapiro-Wilk test and boxplots were examined to search for potential outliers. One-way ANOVA was conducted to compare groups for the PSQI index. Partial eta squared was used to calculate the effect size. Because of the outliers, small sample size and non-normal distribution of objective sleep variables, non-parametric Kruskal-Wallis test were used to look for differences. Partial epsilon squared was used to calculate the effect size.

All statistical analysis was done with Statistical Package for the Social Sciences (IBM SPSS, version 21, Chicago, IL, USA). The results are presented as mean ± standard deviation. The null hypothesis was set at the 95% confidence interval.

7. RESULTS

The results are presented for each research question separately since the classification into groups was based on different physical activity characteristics.

7.1. Sleep and regular physical activity

Participants were classified into three groups based on their reported level of physical activity. There were no statistically significant differences between groups for subject characteristics.

There were no statistically significant differences between group means of PSQI in different levels of PA, low exercisers (M = 6.4 ± 3.4), average exercisers (M = 5.9 ± 3) and high exercisers (M = 5.1 ± 2.6), as determinded by one-way ANOVA (F(2,42) = 0.64, p = 0.53, $\eta_p{}^2$= 0.029). Figure 1 shows participants' PSQI index based on their level of PA.

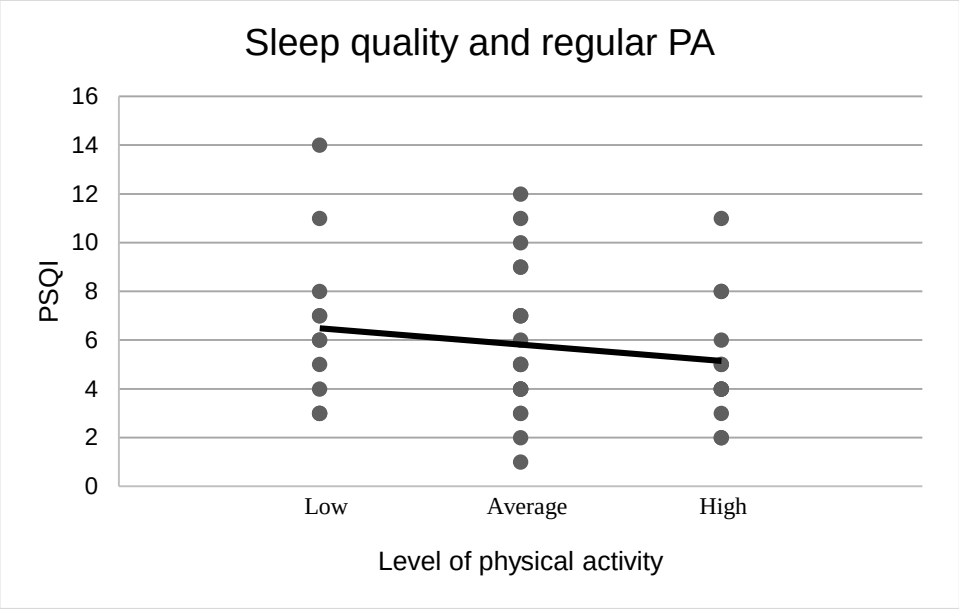

FIGURE 1. Reported subjective sleep quality assessed by PSQI between different groups based on the weekly level of physical activity reported by the questionnaire.

Kruskal-Wallis test was conducted to examine the differences in sleep characteristics according to the amount of the participants' level of PA. No significant differences in sleep duration (χ^2= 0.43, p = 0.81), REM duration (χ^2= 2.59, p =0.27), REM % (χ^2=

3.07, p = 0.22), NREM duration (χ^2= 0.77, p = 0.68), NREM % (χ^2= 0.52, p = 0.77), SOL (χ^2= 2.4, p = 0.30), sleep efficiency (χ^2= 0.52, p = 0.77), S1 duration (χ^2= 0.24, p = 0.89), S1 % (χ^2= 0.23, p =0.89), S2 duration (χ^2= 2.43, p = 0.3), S2 % (χ^2= 2.65, p =0.27), S3 duration (χ^2= 3.22, p =0.2), S3 % (χ^2= 2.89, p =0.24), and wake number (χ^2= 0.2, p = 0.91) were found among three categories of the participants (low exercisers, average exercisers, high exercisers). Table 2 shows sleep parameters assesed by PSG based on different levels of PA. The effect size was calculated with partial epsilon squared and all the effect sizes are very small.

TABLE 2. Descriptive statistics of sleep characteristis for low (n = 12), average (n = 20) and high (n = 13) exercisers).

	Groups			ε^2
	low exercisers (M ± SD)	average exercisers (M ± SD)	high exercisers (M ± SD)	
sleep duration (min)	442.0 ± 25.6	409.6 ± 45.5	413.7 ± 44.6	0.01
REM (min)	96.6 ± 26.3	85.1 ± 27.3	86.5 ± 27.3	0.06
REM (% of TST)	20 ± 6	18 ± 5	16 ± 5	0.07
NREM (min)	325.4 ± 38.1	324.5 ± 34.2	334.2 ± 44.4	0.02
NREM %	68 ± 7	69 ± 5	69 ± 7	0.01
SOL (min)	21.0 ± 17.3	16.7 ± 9.5	12.8 ± 8	0.05
SE (% of TST)	88 ± 5	87 ± 6	86 ± 9	0.01
STAGE 1 (min)	19.7 ± 12.0	18.9 ± 14.8	21.2 ± 16.1	0.01
STAGE 1 (% of TST)	4 ± 3	4 ± 3	4 ± 3	0.01
STAGE 2 (min)	224.5 ± 37.7	217.8 ± 42.5	239.3 ± 61.0	0.06
STAGE 2 (% of TST)	47 ± 7	46 ± 8	50 ± 12	0.06
STAGE 3 (min)	81.2 ± 27.0	87.9 ± 23.8	73.8 ± 30.3	0.07
STAGE 3 (% of TST)	17 ± 27	19 ± 6	16 ± 7	0.07
number of awakenings	3 ± 3	3 ± 6	3 ± 3	0.01

REM, rapid eye movement; NREM, non-rapid eye movement; SOL, sleep onset latency; SE, sleep efficiency; STAGE 1, stage 1 of NREM sleep; STAGE 2, stage 2 of NREM sleep; STAGE 3, stage 3 of NREM sleep; TST, total sleep time.

7.2. Sleep and acute physical activity

Participants were divided into three equal groups based on the number of daily steps. Tertiles were calculated by SPSS. Cut off point for the low step count group was 3477 steps (M = 2288 ± 226) and for the medium step count group the cut-off point was at 5988 steps (M = 4468 ± 171). High step count group maximum value was 10734 (M = 7914 ± 418). There were no observed differences between groups for subject characteristics.

There were no statistically significant differences between group means of PSQI based on the daily number of steps (low step count (M = 5.9 ± 3.4), medium step count (M = 6 ± 3.3) and high step count (M = 5.5 ± 2.4)) as determined by one-way ANOVA (F(2,42) =0.09, p = 0.91, $\eta_p{}^2$= 0.004). Figure 2 shows participants' PSQI index based on the number of steps they took in a day.

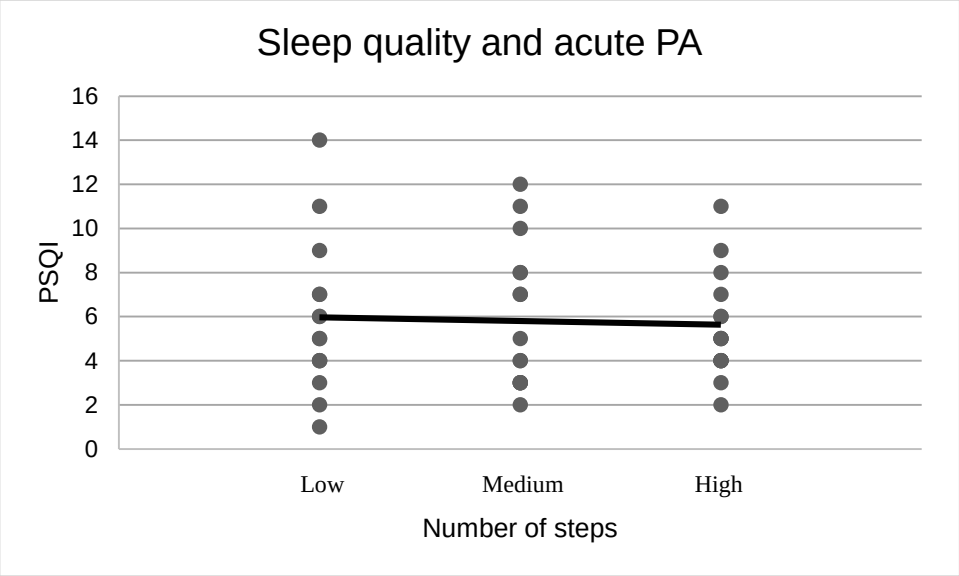

FIGURE 2. Reported subjective sleep quality assessed by PSQI between different groups based on acute physical activity measures as steps taken in a day.

Kruskal-Wallis test was conducted to examine the differences in sleep characteristics according to the different number of steps. No significant differences in duration (χ^2= 3.23, p = 0.2), REM duration (χ^2= 4.09, p =0.13), REM % (χ^2= 2.28, p = 0.43), NREM duration (χ^2= 1.05, p = 0.59), NREM % (χ^2= 1.71, p = 0.43), SOL (χ^2= 0.80, p = 0.67), sleep efficiency (χ^2= 2.54, p = 0.28), S1 duration (χ^2= 0.07, p = 0.96), S1 % (χ^2= 0.02, p = 0.99), S2 duration (χ^2= 0.71, p = 0.7), S2 % (χ^2= 0.21, p = 0.9),

S3 duration (χ^2= 2.05, p = 0.36), S3 % (χ^2= 1.83, p = 0.4), and wake number (χ^2= 2.07, p = 0.36) were found among three categories of the participants (low step count, average step count, high step count). Table 3 shows sleep parameters mesured by PSG based on how many steps participants took during the day. The effect sizes were calculated with partial epsilon squared and all the effect sizes are very small.

TABLE 3. Descriptive statistics of sleep characteristis for low step count (n = 15), medium step count (n = 15) and high step count (n = 15).

	Groups			
	low step count (M ± SD)	medium step count (M ± SD)	high step count (M ± SD)	ε^2
sleep duration (min)	417.0 ± 32.7	428.8 ± 47.4	395.7 ± 48.2	0.07
REM (min)	84.2 ± 22.8	97.8 ± 30.4	77.6 ± 25.7	0.09
REM (% of TST)	18 ± 5	20 ± 7	17 ± 5	0.05
NREM (min)	336.6 ± 33.8	331.0 ± 43.1	318.1 ± 36.4	0.02
NREM %	71 ± 7	67 ± 6	68 ± 6	0.04
SOL (min)	14.1 ± 8.8	18.8 ± 16.0	17.3 ± 10.0	0.02
SE (% of TST)	89 ± 5	87 ± 7	85 ± 7	0.06
STAGE 1 (min)	17.7 ± 10.7	20.4 ± 14.6	21.2 ± 17.3	0.00
STAGE 1 (% of TST)	4 ± 2	4 ± 3	4 ± 3	0.00
STAGE 2 (min)	227.0 ± 39.6	230.9 ± 59.2	219.4 ± 43.3	0.02
STAGE 2 (% of TST)	48 ± 9	47 ± 10	47 ± 8	0.01
STAGE 3 (min)	88.9 ± 27.2	79.7 ± 26.7	77.4 ± 26.7	0.05
STAGE 3 (% of TST)	19 ± 5	17 ± 6	17 ± 6	0.04
number of awakenings	2 ± 3	2 ± 2	5 ± 7	0.05

REM, rapid eye movement; NREM, non-rapid eye movement; SOL, sleep onset latency; SE, sleep efficiency; STAGE 1, stage 1 of NREM sleep; STAGE 2, stage 2 of NREM sleep; STAGE 3, stage 3 of NREM sleep; TST, total sleep time.

7.3. Sleep and cardiorespiratory fitness

Participants were classified into three groups based on their CRF; poor and fair CRF (M = 33.3 ± 1.7 ml/min/kg or males and M = 24 ± 1.2 ml/min/kg for females), good CRF (M = 38.7 ± 1.2 ml/min/kg for males and M = 29.6 ± 1.5 ml/min/kg for females) and excellent CRF (M = 42.1 ± 1.5 ml/min/kg for males and M = 33.2 ± 1.3 ml/min/kg for females). There were no statistical significant differences between groups in age, height and BP. There were statistical significant differences between groups in BMI as determinded by one-way ANOVA ($F(2,42)=9.6$, $p< 0.001$). A Tukey post hoc test revealed that the BMI was statistically lower in group with excellent CRF (26.1 ± 0.5, $p = 0.00$) and good CRF (27.8 ± 1.3, $p < 0.001$) compared to the group with poor and fair CRF (31.5 ± 1.1 ml/min/kg). There were no statistically significant differences between the excellent and good group ($p = 0.307$).

There were no statistically significant differences between group means of PSQI in different levels of CRF, poor and fair (M = 5.4 ± 2.5), good (M = 5.3 ± 5.6) and excellent (M = 6.3 ± 3.4), as determinded by one-way ANOVA ($F(2,42) =0.55$, $p = 0.58$, $\eta_p{}^2= 0.026$). Figure 3 shows participants' PSQI index based on their CRF.

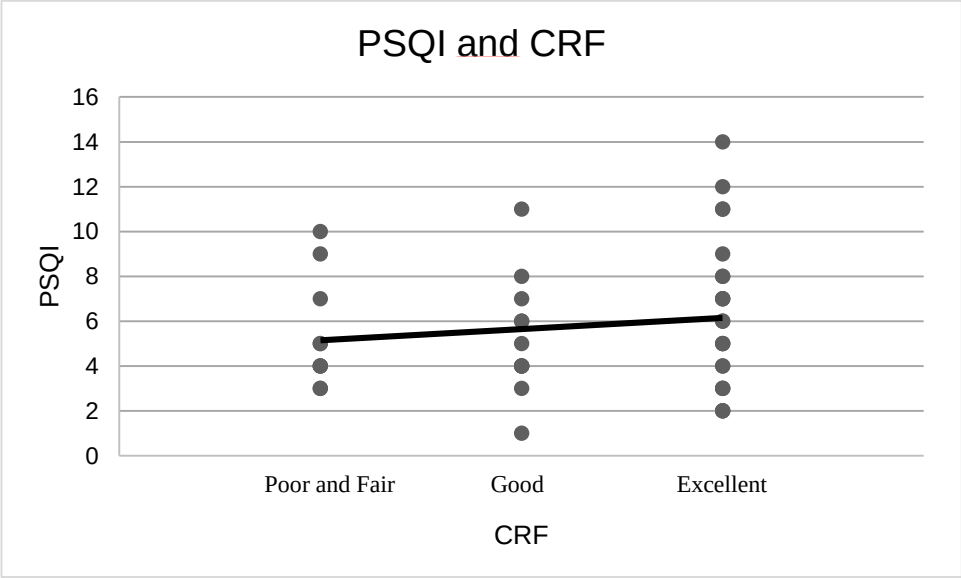

FIGURE 3. Reported subjective sleep quality assessed by PSQI between different groups based on the CRF.

Kruskal-Wallis test was conducted to examine the differences in sleep characteristics according to the different CRF of the individuals. No significant differences in sleep duration ($\chi^2= 2.65$, $p = 0.27$), REM duration ($\chi^2= 2.02$, $p =0.34$), REM % ($\chi^2= 1.76$,

p = 0.41), NREM duration (χ^2= 0.72, p = 0.7), NREM % (χ^2= 0.18, p = 0.91), SOL (χ^2= 4.41, p = 0.13), sleep efficiency (χ^2= 2.29, p = 0.32), S1 duration (χ^2= 0.5, p = 0.78), S1 % (χ^2= 0.73, p = 0.69), S2 duration (χ^2= 2.46, p = 0.29), S2 % (χ^2= 1.43, p = 0.49), S3 duration (χ^2= 1.96, p = 0.38), S3 % (χ^2= 1.85, p = 0.4), and wake number (χ^2= 1.32, p = 0.52) were found among three categories of the participants (poor and fair CRF, good CRF and excellent CRF). Table 4 shows sleep parameters assessed by PSG based on participants' CRF levels. The effect size was calculated with partial epsilon squared and all the effect sizes are very small.

TABLE 4. Descriptive statistics of sleep characteristics for poor and fair CRF (n=10), good CRF (n = 12) and excellent CRF (n = 23).

	Groups			
	poor and fair CRF (M ± SD)	good CRF (M ± SD)	excellent CRF (M ± SD)	ε^2
sleep duration (min)	417.0 ± 45.0	430.0 ± 31.9	404.5 ± 49.1	0.06
REM (min)	88.8 ± 28.6	94.9 ± 25.3	81.2 ± 27.5	0.05
REM (% of TST)	19 ± 6	20 ± 5	17 ± 6	0.04
NREM (min)	328.3 ± 46.2	335.1 ± 22.6	323.3 ± 40.9	0.02
NREM %	70 ± 8	69 ± 4	68 ± 7	0.00
SOL (min)	17.5 ± 9.8	11.1 ± 7.2	19.3 ± 13.9	0.09
SE (% of TST)	89 ± 6	88 ± 6	85 ± 7	0.05
STAGE 1 (min)	20.4 ± 11.8	18.1 ± 13.1	20.3 ± 16.1	0.01
STAGE 1 (% of TST)	4 ± 3	4 ± 3	4 ± 3	0.02
STAGE 2 (min)	225.0 ± 48.8	243.6 ± 27.7	216.8 ± 53.4	0.06
STAGE 2 (% of TST)	48 ± 9	50 ± 6	46 ± 10	0.03
STAGE 3 (min)	82.9 ± 15.2	73.4 ± 31.0	86.1 ±28.2	0.05
STAGE 3 (% of TST)	18 ± 4	15 ± 6	18 ± 7	0.04
number of awakenings	2 ± 2	3 ± 3	4 ± 5	0,03

REM, rapid eye movement; NREM, non-rapid eye movement; SOL, sleep onset latency; SE, sleep efficiency; STAGE 1, stage 1 of NREM sleep; STAGE 2, stage 2 of NREM sleep; STAGE 3, stage 3 of NREM sleep; TST, total sleep time.

8. DISCUSSION

The present study addressed the question whether physical activity and sleep are related in hypertensive patients. Based on the previous findings, it was hypothesized that higher subjective and objective physical activity would result in better subjective and objective sleep parameters. In general, our results demonstrated that low PA was not associated with poor sleep characteristics. We showed that subjective and objective sleep quality was not related to different levels of habitual physical activity, acute PA measured by daily steps and the level of CRF.

8.1. Sleep characteristics and physical activity in the sample

The analysis of subject characteristics was interesting because the subjects were in general poor sleepers, as suggested by the mean score of PSQI being 5.8. Buysse and colleagues (1989) suggested that a total PSQI score of 5 or more defines poor sleepers, with a sensitivity of 89.6% and a specificity of 86.5%. A similar pattern of subjective sleep in hypertensive patients was obtained in a study by Tiede and colleagues (2015). More than 50% of hypertensive individuals reported poor subjective sleep quality, compared to the healthy controls. Moreover, the participants on average reported 6.9 hours of sleep per night, which is slightly less than the recommended 7 hours for adults of the National Sleep Foundation (Hirshkowitz et al. 2015). These results tie well with previous studies wherein authors report that sleep disturbances and short sleep duration have been linked to the development of hypertension (Lo et al. 2018; Fernandez-Mendoza et al. 2010; Fung et al. 2011; Li et al. 2018; Mirjat et al. 2020; Meng, Yheng & Hui 2013). Moreover, altered sleeping patterns, such as poor sleep quality and shorter sleep duration, have been found in participants with already diagnosed disease (Han et al. 2019). Our study adds to the literature that hypertensive patients have altered sleeping patterns compared to the general population. The relationship could be explained by various mechanisms. For example, increased BP could be associated with heightened SNS which alters sleep duration. Moreover, short sleep duration can disrupt circadian rhythm and autonomic balance (Han et al. 2019). Some hypotheses even go beyond what is happening during the night. Sleep deprivation is a stressful condition that increases salty cravings during the day and supresses renal salt fluid

excretion (Han et al. 2019). Thus, the participants are even at greater risk of developing or worsening their hypertension. The average percentage of participants' SWS sleep in our study was 17% which is less than the average 20-25% and this could indicate less restorative sleep. Since all of the subjects have been diagnosed with hypertension, it is plausible that this is one of the factors that could affect their sleep quality. The participants have reported being on various medications and the mean BP of the sample remains within the reference ranges (Wermelt & Schukert 2017). Since the low quality sleep can have a negative impact on the quality of life, it is extremely important to address this issue in hypertensive patients, since they are more prone to the CVD (Mirjat et al. 2020).

Another way to combat the negative effects of hypertension is to implement more PA into the life of individuals. Ghadieh and Saab (2015) emphasize that increased PA is considered a first-line intervention as a treatment strategy for the patients with stage one or two hypertension. Moreover, authors in Physical Activity Guidelines Advisory Committee Scientific report (2018) suggest that aerobic and dynamic resistance exercise might be equally effective in reducing blood pressure. Adults with hypertension are encouraged to engage in 90 minutes or more of moderate intensity or 45 minutes of vigorous intensity aerobic exercise per week (Physical Activity Guidelines Advisory Committee Scientific report 2018). The average PA of patients in this sample was more than 150 minutes per week, which is in line with the national recommendations in the Physical Activity Guidelines for Americans (2018) for the general population and guidelines from Pescatello et al (2004) that advise 30 minutes or more of moderate-intensity of PA per day, most days of the week. Looking at these results it could be speculated that this sample was more active than the general hypertensive population. One possible explanation is that these participants liked to exercise and decided to take part in the study. On the other hand, there might have been other hypertensive individuals who were not as fit or liked to exercise and that is why they decided not to participate in the study. Thus selection criteria could provide a biased sample of already fit individuals. It is possible to assume that adults who do not like exercise and are sedentary would not decide to participate in this kind of study. Stating that, it is not surprising that more than half of the participants had excellent cardiorespiratory fitness, according to the ACSM standards (American College of Sports Medicine, 2018), adjusted for age and gender.

Nonetheless, even though the participants reported sufficient levels of PA and physical fitness, the objective assessment did not show similar results. The mean number of daily steps in the sample was 4890 steps per day, which is considered as a sedentary lifestyle according to Tudor-Locke and Basset (2004). In a newer study by Tudor-Locke and colleagues (2011), it is stated that many government and professional organizations worldwide recommend 10000 steps per day for healthy adults (Tudor-Locket et al. 2011). Similarly, Physical Activity Guidelines for Americans (2018) distinguish between low (under 7000 steps), moderate (7000 steps) and high (10 000 steps) PA. Looking at our sample, only 17.8 % of participants exceeded the 7000 steps and out of those, only two participants reached 10 000 steps. From this data we could assume that, either participants over-estimate their subjective level of PA, or they complete their exercise session without accumulating a high number of steps, for example biking or swimming. Furthermore, the literature suggests that pedometer and step count results provide inexact index of exercise volume as the quality of the steps usually cannot be determined (Garber et al. 2011). Guidelines for hypertensive population regarding the step count are not clear, however in some studies participants were also encouraged to increase the number of steps to 10 000. This significantly improved their BP (Gu et al. 2020). All things considered, increasing the number of steps to improve the habitual level of physical activity should be one of the primary goals since it can positively affect blood pressure and other quality of life parameters.

8.2. Regular physical activity and sleep

Global PSQI index was not statistically different between the three groups of exercisers. A trend of decreased index can be seen between low exercisers and high exercisers ($M = 1 \pm 3$) but the differences are not significant, and the effect size is very small. This is not consistent to the previous studies since many of them show an association between habitual PA and self-assessed sleep (Kellez & Kelley 2017; Rubio-Arias et al. 2017; Chennaoui et al. 2015; Kredlow et al. 2015; Sullivan et al. 2019). For example, Youngstedt & Kine (2006) and Buman & King (2010) thoroughly examined this relationship in their reviews and reported that epidemiologic studies consistently report significant positive associations between self-reported exercise and subjective sleep patterns. A recent systematic review and meta-analysis showed that exercise significantly lowered PSQI index for a mean of 2.87. Nonetheless, the quality

of evidence was reported to be low by the authors (Banno et al. 2018). According to many questionnaires and cross-sectional studies, many participants report that being physically active reduces their sleepiness and improves their perception of sleep ("Sleep in America" pool 2003; Urponen et al. 1988; Loprinzi & Cardinal 2011). Similary, Brand and colleagues (2010) investigated if vigorous exercise was related to improved sleep. They compared adolescents (athletes versus control group) and found that according to the sleep logs, athletes had better sleep patterns including better sleep quality, shorter SOL, less WASO and were less tired throughout the day. When an intervention was carried out by King and colleagues (2008) on inactive older people with mild sleep complaints, they found out that the exercise group had more improvements in PSQI and other subjective sleep parameters. Even when participants did only a resistance training intervention, their subjective sleep-quality significantly improved (Singh et al. 1997). Similar benefits were reported in other studies with PA that met the national guidelines (King et al. 2008; King et al. 1997). Buman and King (2010) concluded that aerobic and resistance exercise at the levels of national recommendations appear to be sufficient to improve subjective sleep quality in various populations. In a longitudinal study, where participants were older people, habitual PA was related to a lower prevalence of insomnia and the authors suggest that high-frequency PA may help reduce insomnia (Inoue et al. 2013). When studies were done on older individuals, the most consistent changes occurred in self-rated sleep quality (Buman & King 2010).

On the other hand, some studies are in line with the findings from the present study and do not show an association between self-reported PA and subjective sleep. Kakinami and colleagues (2017) did not find any association between PA intensity and duration to sleep quality or quantity in young adults. A similar conclusion was reached by Harp (2015) who found that a 15-week exercise intervention did not change the sleeping pattern in the majority of the participants. One possible explanation of not finding differences in our study could be attributed to the fact that participants might have overestimated their physical activity levels. On the other hand, the results might have been statistically significant if the sample size was bigger.

Objective sleep parameters, assessed by PSG, were not statistically different between the three groups. There were a few differences observed between different levels of exercise, but all the effect sizes calculated were very small. This suggests that the self-

reported amount of physical activity did not have an effect on objective sleep parameters. This is in contrast to the previous studies, since most of them observed small beneficial effects on sleep quality (Chennaoui et al. 2015). In a meta-analytical review, authors found small beneficial effects on TST, SE, SOL and sleep quality (Kredlow et al. 2015). In the current study, a trend of decreased SOL can also be seen among high exercisers, but the results are non-significant and the effect size is very small. Sleep efficiency and TST decreased with increased level of physical activity but again, the results were non-significant. Review by Driver and Taylor (2000) reports that acute exercise has small-to-moderate effects on increasing SWS, delaying REM latency and reducing REM sleep but our study does not support these findings. On the contrary, high exercisers had non-significant 3.4% lower amount of SWS. King and colleagues (2008) reported increased time in S2, decreased time in S1 and fewer awakenings for people who exercise but this trend was not seen in the current study. In intervention studies, it is suggested that exercise programmes should be of sufficient duration to provide small-to-modest benefits. Buman and King (2010) suggest at least a 16-week intervention to provide beneficial effects on sleep. When studies increased the level of PA above the national recommendations, dose-response effects were seen. Nonetheless, if the participants exercised less than 150 minutes per week, no improvements were seen in sleep structure (Buman & King, 2010). The organizational manner of this study was cross sectional, and this cannot be fully compared to the intervention studies.

On the other hand, some studies do not fully support the hypothesis that increased habitual PA promotes sleep (Youngstedt 2005; Youngstedt et al. 2003). Yang and colleagues (2012) did not find differences in sleep parameters, for example TST and SE, when participants were engaging in difference exercise programmes. Likewise, Oudegeest-Sander and colleagues (2012) did not confirm a positive correlation between an exercise intervention and better sleeping characteristics. Some positive correlations were seen between energy expenditure and SE in young adults, but there were non-significant differences in SOL, TST and WASO in young or older adults.

There are several limitations and explanations why there were no observed sleep differences among different levels of physical activity. Firstly, subjects in the present study were asked about their levels of PA throughout the month so the level of exercise was not objectively assessed. According to the literature, subjective measures of PA

usually overestimate the actual levels of PA (Kakinami et al. 2017). It is plausible to assume that the participants were not as active as they reported, thus no differences were seen among them. Moreover, since the participants were recruited based on their hypertensive status and not based on sleep complaints, there might have been a ceiling effect. If participants are good sleepers at the baseline, that means that exercise will not have a significant effect on their sleeping patterns. Secondly, it is possible that the association between sleep and PA is more pronounced in the previous studies because of the individual's self-perception of being physically active. Thus, more active individuals think they sleep better, but this has nothing to do with the behavioural patterns. What is more, it is possible that people use sleepiness and physical fatigue as synonyms, therefore they might think they are feeling sleepy when in reality they are just experiencing body fatigue. Thirdly, this was merely a cross-sectional study and causality cannot be inferred from epidemiologic associations. To investigate the true effects of habitual PA on sleep, a randomized controlled trial with an exercise intervention should be done. Although current literature suggests a positive relationship between sleep and exercise in observational studies, this could be explained by the fact that participants exercise more because they sleep better and not vice versa. Better sleep has been associated with greater willingness to exercise (Youngstedt 2005), and also better health in general has been associated with better sleep and ability to exercise. Ideally in an exercise intervention study, participants would undergo PSG nights after spending a couple of weeks in an exercise intervention, that would control other factors, such as light-exposure, nutrition, smoking etc. However, these kinds of studies are hard to produce because humans are not machines and we will never be able to absolutely limit the confounding factors. Then again, it would be interesting if the self-reported and objective levels of sleep would improve in the current participants if they would be included in an intervention study. It would seem that an appropriate exercise intervention could help them improve some sleep parameters. Additionally, we must recognize that PA does not only influence sleep but affects almost every organ system in our bodies. It would be interesting to investigate how the exercise intervention would affect their blood pressure and other health parameters.

8.3. Acute physical activity and sleep

The results demonstrate that there are no differences in self-reported sleep between groups who completed more or fewer steps per day. This suggests that the acute PA measured by steps taken does not affect perceived sleep quality. Comparatively to the literature, other studies have in general found positive association between self-reported sleep quality and acute physical activity (Dolezal et al. 2017) and these effects seem to be more pronounced when participants are older subjects or subjects with sleep complaints (Kredlow et al. 2015). A study by Lang and colleagues (2013) found that even higher PA levels in adolescents are related to better sleep quality and less insomnia symptoms. In a study that was also assessing the number of steps and its association to subjective sleep, they showed beneficial effects. Improvements in subjective sleep quality was seen after a combined intervention, therefore the authors concluded that the number of steps is significantly related to subjective sleep (Erlacher et al. 2015). Similar conclusions have been reached in a newer study stating that low-impact PA, like walking, is positively associated with sleep quality (Sullivan et al. 2019).

Nonetheless, there are some studies who failed to show that exercise intensity or duration has any impact on subjective sleep (Mylyymäki et al. 2012; Mylyymäki et al. 2011; Youngstedt et al. 2003). Similarly, we reached the same conclusion and our results fail to support epidemiologic data on the value of acute exercise on sleep. It has to be noted that participants in general did a very low number of steps, therefore their level of PA was very low, even in the "highest step count" group category. It is possible that because of that there were no differences found in subjective sleep quality.

This study also failed to support the previous findings regarding the positive relationship between acute exercise and objective sleep characteristics. Together the present findings suggest that active levels measured by the number of steps do not affect objective sleep parameters. Literature is not consistent when it comes to the objective measures of sleep, but most research still shows small promising effects of exercise on sleep (Driver & Taylor 2000; Youngstedt 2005). For example, older studies show that acute exercise increases SWS, REM latency and decreases REM sleep (Youngstedt 1997; Kubitz et al. 1996). The adverse trend was seen in our study since participants with least steps completed had a higher percentage of sleep time in

S3, which corresponds to SWS. Moreover, REM duration and percentage was higher in the medium step count group, but none of these differences were significant. Newer studies add to the findings that acute exercise influences TST, SOL, SE and S1 sleep (Kredlow et al. 2015; Lang et al. 2015).

On the other hand, there are studies that failed to support changes in sleep characteristics after acute exercise sessions (Edinger et al. 1993). Hauge and colleagues (2003) found differences in SWS, REM and SOL after a full sedentary day, yet no differences were observed for TST, SE, WASO. The latter results are consistent with our study.

This study suffers from several weaknesses. For example, compared to other studies that assessed structured exercise sessions, our research was focused on general PA throughout the day. PA, compared to exercise, is not a planned activity and cannot be measured by intensity, duration and frequency. In most of the previous study designs participants had to complete a full workout that had defined duration and intensity, whereas in the present research we merely tracked how many steps per day participants completed. This measure gives an approximate value of how physically active the participants were but does not define exact exercise parameters. Therefore, some participants might have been more active than it seems from the number of steps. To illustrate, participants might have had a very sedentary day but then completed resistance training in the gym or did indoor cycling which did not affect their step count. We must remember that actual physical activity could not be attributed just to one parameter measure, but it is a combination of many different factors.

Second limitation could be seen when comparing the number of steps between the groups and seeing that mean values are not much different. According to the mean values, two groups did less than 7000 steps per day, which is considered as a sedentary lifestyle. Moreover, even the highest group passed the line just slightly. Remembering that more than 10000 steps per day is advised in most of the guidelines, we could argue that this sample was sedentary and there were not that many differences between groups in the number of steps per se, therefore it would be harder to spot any differences in sleep quality and characteristics. Looking at the results one might suggest that there is a trend of better sleep in more sedentary groups but that would be irrational due to the study design. In this case, we could not possibly control for the

cofounding factors that affect sleep, such as light exposure, sleep hygiene, mental state, etc. Therefore, any sleep differences in groups could not be explained by physical activity alone. It also needs to be noted that exercise might have indirect effect on sleep by altering body composition, metabolic rate, cardiac function etc.

8.4. Cardiorespiratory fitness and sleep

This research rejects the hypothesis that CRF has an effect on subjective sleep quality, since no statistically significant differences were seen among the groups. Some literature supports these findings, as authors did not find correlations between physical fitness and sleep characteristics in young or older adults (Oudegeest-Sander et al. 2012; Meintjes et al. 1989; Paxton et al. 1984). Moreover, there were no differences found in other objective sleep characteristics when comparing individuals with different CRF in this study.

There is conflicting evidence in the literature regarding CRF and sleep quality. Some studies suggest that as CRF increases, so does sleep quality, while others show that the relationship between exercise and sleep remains independent of CRF (Buman & King 2010). For example, it might be that sleep differences occurred because of improved body composition and not CRF, per se (Buman & King 2010), which is supported by other research (Meintjes et al. 1989). Nonetheless, there were differences between 3 groups in BMI and fat mass in our sample, yet we did not find any differences in sleep parameters. Thus, our study doesn't support previous findings regarding body composition.

In spite of that, some studies support the association between CRF and sleep. For example, Edinger and colleagues (1993) and Shapiro et al (1984) observed differences in SOL, WASO, SE and slow waves when comparing fit versus unfit individuals. Poor subjective sleep quality was also associated with lower CRF in all age groups (Lee & Lin 2007; Mota & Vale 2009; Strand et al. 2013; Tworoger et al. 2003), and the symptoms of insomnia increased with low physical fitness (Dolezal et al. 2017). When a longitudinal study was done, it showed that each minute of decline in treadmill endurance increased incidence of sleep in the middle age population (Youngstedt et al. 1997). Similarly, Tiede and colleagues (2015) completed a study on hypertensive

individuals and found that participants who had worse sleep quality also did 100 m shorter distance on the 6-minute walk test. Even though our participants had in general high levels of CRF, this did not seem to affect their subjective or objective sleep parameters.

One limitation of our implementation is that we did not manipulate CRF values. In the case of our study, we merely observed current physical fitness of individuals, but did not follow them for a long time to see how changes in CRF would affect sleep. Even if changes among the groups would be observed, we could not definitely say that these differences are due to physical fitness alone. There are other factors that have not been controlled. For instance, nutrition, drinking, use of medication etc. Moreover, the correlation does not equal a causational relationship. For example, fit subjects could choose a more active lifestyle because they sleep better and feel more energetic and not vice versa.

9. CONCLUSION

The main finding of the study is that regular and acute physical activity is not associated with self-reported or objective sleep. Moreover, cardiorespiratory fitness is not an indicator of better sleep quality and architecture in hypertensive population. These findings are in contrast with the current literature that supports a weak association between exercise and sleep in most populations. It remains difficult to compare studies that investigate the correlation between sleep and physical activity, because different modalities are used to assess both parameters. Furthermore, it is hard to adjust for confounding factors that have greater effect on sleep, such as sleep hygiene and bright light exposure. Equally important thing to note is the ceiling effect, when the population is healthy without sleep complaints. Future research should consider to what extent physical activity affects sleep, compared to other factors more carefully. In addition, larger experimental studies with identical exercise protocols should be done on different populations in order to be comparable. Current variations in methods makes it hard to draw consistent conclusions.

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