

## **THE RELATION BETWEEN PHYSICAL EXERCISE AND SLEEP PHYSIOLOGY IN NON- CLINICAL INDIVIDUALS**

A review

Bachelor Degree Project in Cognitive Neuroscience

15 ECTS

Spring term 2012

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**The Relation Between Physical Exercise and Sleep Physiology in Non-Clinical  
Individuals**

Submitted by Karl-Johan Svensson to the University of Skövde as a final year project towards the degree of B.Sc. in the School of Humanities and Informatics. The project has been supervised by Pilleriin Sikka.

**2012-08-13**

I hereby certify that all material in this final year project which is not my own work has been identified and that no work is included for which a degree has already been conferred on me.

Signature: \_\_\_\_\_

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### Abstract

This essay reviews scientific literature regarding the possible effect of exercise on sleep physiology in non-clinical individuals. Exercise and sleep as research phenomena in the literature is examined, documented experimental evidence and theories on the possible mechanisms of a sleep-exercise relationship is reviewed, and suggestions for future research is made. Exercise and sleep are wide-ranging phenomena with a multitude of variables and aspects, making them difficult to research. Although exercise having historically and popularly being favorably associated with sleep, the objective scientific evidence for such a relationship is conflicting. This possibly due to studies using differing methods of various quality and focusing on different aspects of the object of research. There are several theories regarding how an exercise-sleep connection could function, none has yet to be substantiated by existing evidence.

Keywords: exercise, fitness, physical activity, sleep

## Introduction

Today's society places a lot of demands on its citizens, social as well as work-related, and a lot of people feel that there isn't enough time to meet all the demands. As a result of this situation sleep-time is often compromised, with negative personal consequences related to sleep deficits (Strine & Chapman, 2005). There is constant ongoing research into how or if we can make our time asleep more efficient in regards to mental/physical restoration. Pharmaceuticals are the most common form of sleeping aids, especially in clinical populations (Soares Passos et al., 2011), but are also associated with several negative side effects such as accidents in acute usage and mortality and dependence in chronic usage (Youngstedt, 2003). This makes possible cognitive and behavioral sleep-promoting techniques an attractive alternative. Among non-clinical individuals problems related to sleep is very common in the elderly population (Edinger et al., 1993) and in women during pregnancy (Borodulin et al., 2010). These individuals can be extra sensitive to the possible adverse effects of pharmaceutical sleep aids due to their state of physical stress/decomposition (Borodulin et al., 2010; Reid et al., 2010) and this constitutes further compelling arguments for the pursuit of non-pharmaceutical alternatives.

There are a number of more-or-less popular non-pharmaceutical sleep-enhancing alternatives like light therapy, sleep hygiene education (information on how to optimize sleep environment and behavior), relaxation therapy and cognitive behavioral therapy, but these can be costly and/or relatively difficult to administer (Youngstedt, 2005). This makes exercise an attractive alternative as it is often inexpensive, available to most people, relatively safe and has the added bonus of promoting general health. A scientifically identified causal link between exercise and sleep would therefore have important implications on sleep-enhancing strategies and treatments.

Historically exercise has been closely associated with positive influence on sleep (Youngstedt et al., 2003; Youngstedt, 2005) as sleep has commonly been perceived as restitution for physical labor (Atkinson & Davenne, 2007). This appears to still be the case as research indicates that exercise is overwhelmingly referred to as the number one sleep enhancer in large population surveys (Singh, Clements & Fiatarone, 1997, Youngstedt et al., 2003). It is also a widespread belief within the scientific community, and according to Atkinson & Davenne (2007) and Youngstedt, O'Connor & Dishman (1997) exercise is commonly prescribed as a sleep aid by sleep experts and primary care physicians. It is further officially considered a viable non-pharmacological sleep improvement by the American Sleep Disorders Association (Driver & Taylor, 2000). Scientific studies in the field have been far from unanimous however, presenting conflicting results (Driver & Taylor, 2000; Montgomery & Dennis, 2004; Youngstedt et al., 1997) which could be an indication of an existing or non-existent relationship between the two variables, but also a reflection of the wide variety and qualitative inconsistency between methods and study designs (Youngstedt et al., 2003). The most obvious difficulty in comparing studies lies in the difference of subjective reports (such as sleep diaries, subjective scoring etc.) and more objective measurements (such as polysomnography, actigraphy etc.).

The aim of this review is to investigate what the literature says about the physiological aspect of a possible relationship between exercise and sleep, so focus will be primarily on objective measures and physical theories, but it will also touch upon any relevant non-physical aspects. The first part of the thesis will look closer at exercise, sleep and non-clinical individuals as research phenomena. The second part will look at the existing scientific evidence and highlight some of the problems inherent in the research field. The third part will investigate theories about the mechanism behind a possible exercise-sleep

connection. A discussion summarizing the research and theories will follow and a conclusion regarding the available evidence and suggestions for future research will finish the thesis.

### Exercise

While physical activity is generally defined as any kind of movement of skeletal muscles, exercise is a subvariety characterized by its structured organization of activity and purpose towards enhancing the practitioner's physical fitness ("Exercise," *Encyclopædia Britannica*, 2012). As a variable in research "exercise" is a broad and somewhat imprecise term due to the many subvariables in which it can differ. A common way to categorize different aspects of exercise in review literature of the sleep-exercise research field is dividing it into *acute* exercise and *chronic* exercise (Driver & Taylor, 2000; Youngstedt, 2005), "acute" meaning single instance and "chronic" meaning repetitive or habitual. There is however a multitude of other aspects that are worthy of equal attention when performing or evaluating research on exercise.

Apart from chronic and acute there are other exercise-type-variables such as the *intensity* variable, which indicates the load put on the individual performing the exercise task. Episode *duration* is also worth knowing about as it will surely affect the performance. Two further ways to categorize exercise-types is by the *aerobic* vs. *anaerobic* dichotomy, "aerobic" meaning endurance-like training in the form of running, cycling, swimming etc. and "anaerobic" meaning exhaustive training of short duration like weight-lifting. It is common knowledge that these types affect the human body in different ways, and therefore presumably also a possible exercise-sleep relationship.

There is also the question of the individual performing the exercise task. Even in healthy subjects there is still need to control for task-related fitness; there are probably different baseline- and capacity-values for different individuals due to degrees of fitness,

previous exposure to differing exercise-types/life-styles and body-mass. A large variability within subjects on any of these aspects might cloud any potential research-findings.

Several studies also mention the *environment* in which the exercise is carried out, and the *time-of-day* as important research-aspects. There have been previous associations between temperature and sleep physiology (Horne & Staff, 1983), and lighting-conditions during waking hours are frequently mentioned as a significant influence on sleep (Driver & Taylor, 2000; Helena de Castro Toledo Guimaraes, Bizari Coin de Carvalho, Yanaguibashi & Fernandes do Prado, 2008; Van Someren, Lijzenga, Mirmiran & Swaab, 1997). The question of *when* exercise is performed is also mentioned as a possibly important variable (Borodulin et al., 2010; Dworak et al., 2008; Torsvall, Åkerstedt & Lindbeck, 1984), the consensus being that exercise too close to bed-time might have sleep-disruptive effects. However, a study of the effects of long-term moderate aerobic exercise on sleep in 19 individuals with chronic primary insomnia by Soares Passos et al. (2011) showed that the significant effects noted did not vary between morning and late afternoon exercise conditions, and according to Loprinzi & Cardinal (2011) there is no conclusive evidence that late-night exercise impairs sleep quality.

Then there is the matter of measuring exercise. *Self-reports* on perceived level of exertion are used in some of the literature (Borodulin et al., 2010; Edinger et al., 1993; Youngstedt et al., 2003), monitoring *heart-rate* is also relatively common (Edinger et al., 1993; Dworak et al., 2008; Reid et al., 2010), as is *actigraphy* (a movement-sensor calculating gross motor activity) (Loprinzi & Cardinal, 2011; Robillard, Rogers, Lambert, Prince & Carrier, 2011; Youngstedt et al., 2003). For anaerobic activities such as weight-lifting simple *physical capacity checks* have also been used (Singh et al., 1997). According to Horne (1981) the more preferable method though is the measurement of *maximal oxygen uptake* ( $VO_{2max}$ ) since it apparently is reliable, objective and relatively easily quantifiable.

This method appeared about as much as some earlier mentioned methods in the literature (Bunnell, Bevier & Horvath, 1983; Horne & Staff, 1983; Van Someren et al., 1997).

As indicated above, there is lack of consensus regarding method and study-design within the field and unfortunately most studies do not control for *all* (or even a substantial amount) of the above mentioned potentially crucial aspects of exercise, making comparisons of results extremely difficult; a point also commonly concluded in previous reviews (Horne, 1981; Montgomery & Dennis, 2004; Youngstedt et al., 1997).

### Sleep

If the function of sleep and what triggers it would be scientifically mapped out and established, it is plausible that the question of a possible exercise-sleep connection could be more easily answered. This is far from the case however, and the literature mentions various (and to some extent inter-related) sleep related theories like the previously mentioned influence of temperature (Horne & Staff, 1983), circadian rhythm (Brand et al., 2010; Van Someren et al., 1997; Youngstedt, 2005) as well as the aspect of physical restoration or energy conservation (Atkinson & Davenne, 2007; Driver & Taylor, 2000; Dworak et al., 2008; Horne, 1981). These theories certainly deal with the *physiology* of sleep in terms of the brain and neural functioning, but most studies are limited to investigating the sleep physiology via polysomnography (PSG) recordings of sleep stages, which are commonly divided into non-REM sleep stages N1, N2, N3, N2 and REM (rapid eye-movement sleep).

If there is a significant influence of exercise on sleep, then there is the question of how to interpret results as positive or negative. The most commonly referenced PSG-measured aspects of sleep in the literature are sleep onset latency (SOL), stage 2 (N2) sleep, slow wave sleep (SWS), REM, REM latency (REM-L), total sleep time (TST) and wakefulness after sleep onset (WASO) (Youngstedt et al., 1997). In a PSG recording a host

of variables are monitored, but these are the ones most commonly reported when significant results are noted in the reviewed exercise-sleep research. The amount of SWS has often been perceived as an indicator of sleep quality due to it being the “deepest” form of sleep (Dworak et al., 2008; Youngstedt, 2005), but this has been questioned (Atkinson & Davenne, 2007; Horne, 1981; Youngstedt, 2005 ). Driver & Taylor (2000) sees subjective sleep-perception, WASO and TST as valid quality markers and Youngstedt (2005) argues that REM sleep “can be considered the deepest of sleep by some arousal criteria, and there is also little compelling evidence indicating that SWS is associated with better outcomes (e.g., in terms of daytime sleepiness) than REM sleep” (Youngstedt, 2005, p. 360). This makes the interpretation of PSG-based sleep physiology measurements difficult; what is a detrimental effect and what is an enhancement? The discussion regarding possible quality-indicators of sleep mentioned above is primarily found in earlier reviews of the field, and most of the individual studies reviewed do not make explicit claims regarding their results and quality-aspects of sleep.

As in the case of measuring exercise, the measurement of sleep in the literature is not exclusive to one specific method. PSG recordings were the most common objective form of measurement (Bunnell et al., 1983; Dworak et al., 2008; Edinger et al., 1993; Horne & Staff, 1983; Robillard et al., 2011; Soares Passos et al., 2011; Torsvall et al., 1984), actigraphy was also used in two instances (Van Someren et al., 1997; Youngstedt et al., 2003), but subjective measurements in the form of self-reports are also just as common (Borodulin et al., 2010; Brand et al., 2010; Helena de Castro Toledo Guimaraes, 2008; Loprinzi & Cardinal, 2011; Reid et al., 2010; Sherrill, Kotchou & Quan, 1998; Singh et al., 1997; Soares Passos et al., 2011; Torsvall et al., 1984; Youngstedt et al., 2003), either as the exclusive method or in addition to objective measurements. Subjective reports are problematic though as evidence has suggested that self-perceived sleep-architecture do not correspond with PSG measurements (Soares Passos et al., 2011; Youngstedt, 2003), and are

by nature not applicable to the focus of this review. Worth noting however is that subjective reports are the norm when diagnosing problems of insomnia (Soares Passos et al., 2011), as well as evaluation treatment effectiveness (Reid et al, 2010). Even if one were to establish a positive relationship between exercise and sleep, any impact on the population in general would plausibly be significantly dampened if people could not subjectively perceive an actual enhancement.

Differing theories regarding the function and governing of sleep, conflicting views on how to interpret PSG results, and the use of different measurement-techniques only adds to the study-comparison difficulties previously mentioned in the exercise section.

#### Non-clinical individuals

This thesis focuses mainly on exercise-sleep research generalizable to the majority of the population. Therefore its focus is on non-clinical individuals as the available literature mentions psychological conditions such as clinical anxiety and depression, along with more directly associated sleep related disturbances such as clinical insomnia, as having the effect of providing non-representative variability to findings (Soares Passos et al., 2011; Youngstedt, 2005). However, a problem with this approach is the so-called “ceiling effect”; normal/good sleepers (i.e. non-clinical individuals) may already be close to the “best” possible sleep-level, leaving little room for improvement, hence providing small measurement-readings that can be hard for scientists to distinguish from just “noise”-readings (Youngstedt, 2003). Individuals with compromised sleep naturally have more room for improvement and the effects of successful sleep aids should at least theoretically be more dramatic in that kind of population (Youngstedt, 2003).

## Documented effects of exercise on sleep

The issues of differing samples, methodologies and study design described in the previous sections makes it incredibly difficult to compare studies regarding the possible effect of exercise on sleep. This was already addressed in Horne's extensive review from 1981, where he was unable to reach any consensus apart from indications that a few electroencephalogram (EEG) readings suggested a temporary increase of SWS as a result of acute exercise in sedentary individuals, and a higher SWS-baseline in fit individuals. This has proven to be the most common result *when* a significant sleep-exercise correlation has been noted (Bunnell et al., 1983; Driver & Taylor, 2000; Dworak et al., 2008; Edinger et al., 1993; Horne & Staff, 1983; Robillard et al., 2011). But according to an extensive meta-analysis of acute exercise studies by Youngstedt et al. (1997) mean SWS values are only significantly modified by a mere 1,4 minute. Further evidence is indeed modest and mean SWS increases are generally within the 3-13 minute spectrum (Dworak et al., 2008; Edinger et al., 1993; Horne & Staff, 1983). A notable exception is a study on exhaustive exercise on sleep by Bunnell et al. (1983) where PSG recordings on the night after the exercise condition showed a mean increase in SWS by 24 minutes in the female participants. Participating males showed a more "common" 5,7 minute SWS increase. Worth noting is that the study consisted of a mere 5 women and 4 men, bringing into question problems regarding generalizability of small sample sizes, something that will be discussed later in this section. The majority of these SWS-results come from studies on acute exercise, but slightly differing results are stated in the study of acute exercise on older men by Edinger et al. (1993). This study presented PSG recordings of 12 aerobically fit and 12 sedentary older men during a baseline condition as well as after an acute exercise condition. No effect was noted for the acute exercise challenge, but a comparison of the baseline recordings between the sample groups

showed that the aerobically fit individuals spent a mean 12 minutes longer in SWS than the sedentary individuals. This could be an indication that chronic exercise affects SWS as well.

REM sleep is also commonly affected when a significant PSG-result is reported, usually as a mean REM decrease of 2-6 minutes (Bunnell et al., 1983; Driver & Taylor, 2000; Torsvall et al., 1984, Youngstedt et al., 1997) and a mean REM-L increase of approximately 10 minutes (Bunnell et al., 1983; Driver & Taylor, 2000; Youngstedt et al., 1997). In positive exercise-sleep correlations, SOL is also frequently reported as decreased (Horne & Staff, 1983; Edinger et al., 1993; Montgomery & Dennis, 2004), as well as an increase in TST (Driver & Taylor, 2000; Horne & Staff, 1983; Montgomery & Dennis, 2004; Youngstedt et al., 1997), both variable-changes in the same small-to-modest mean levels as SWS/REM/REM-L. Changes in other NREM stages and WASO are reported, but less frequently. There are also studies reporting conflicting results though (Youngstedt et al., 2003; Edinger et al., 1993), and most reviews concludes that the PSG-based evidence of a potential relation between exercise and sleep is hardly compelling (Driver & Taylor, 2000; Montgomery & Dennis, 2004; Youngstedt, 2003, 2005), mentioning differing or poor methods, experimental designs and uncontrolled confounding variables as potential sources for distortion of results, or even plausible causes for the existence *of* results (Youngstedt et al., 1997). Moreover there is a noticeable lack of objective PSG-based studies when it comes to investigating the phenomenon of chronic exercise. Instead there are several studies based on subjective reports, and the more empirical ones tend to rely on actigraphy (Van Someren et al., 1997; Youngstedt et al., 2003), which resolution is generally limited to measuring motor-rest/activity fragmentation, not specific sleep variables. This is possibly related to matters of funding as experimental PSG-studies of chronic exercise tend to be longitudinal in nature and therefore demanding of staff, facilities and equipment in ways that studies of acute exercise generally are not. Physical changes in the human body and brain are often slow and

progressive so it is a valid assumption that the possible effects of exercise on sleep physiology might be more pronounced in well-designed objective studies of chronic exercise (Horne, 1981; Youngstedt et al., 2003). The previously mentioned study by Edinger et al. (1993) could be evidence of this; no effects were noted regarding acute exercise on sleep, but PSG-comparison of the groups of aerobically fit and sedentary individuals showed that the fit sample had less SOL, less WASO, less stage 1 sleep and more SWS than the sedentary sample.

Apart from the previously mentioned experimental issues like varying exercise types, individual baseline-differences, methodological differences, result interpretations and ceiling effects, there are other aspects that could possibly be an influence on the research evidence. As mentioned earlier, the literature indicates that exercise has a significant effect on depression (Soares Passos et al., 2011; Youngstedt, 2005), but since normal mood fluctuations is not controlled for in a majority of the reviewed literature it is possible that the modest sleep-exercise correlations that has been presented might be a result of equally modest mood affect. Youngstedt (2005) mentions that the documented reductions of REM-sleep might be a sign of this as the phenomenon has also been linked to regressing depression. Also related to this type of reasoning are general quality-of-life improvements, Brand et al. (2010) mentioning active individuals' exercise habits as possible routine-stabilizers, and in a sleep-exercise study of elderly women Helena de Castro Toledo Guimaraes et al. (2008) mentions the increase of an exercise-related social life as a possible influence.

Personal traits and habits might also influence results; people leading physically active lifestyles might have personality traits that attract them to the sort of challenges of strenuous character that exercise provides (Brand et al., 2010; Horne, 1981; Youngstedt, 2005), and this might also extend to sleep architecture. Individuals with a higher tolerance for

stress might prioritize physical activity in various life situations when other less stress-tolerant individuals might not (Youngstedt et al., 2003). It is also likely that people who exercise might also actively try to control their health by reducing or avoiding caffeine and alcohol, which have been associated with affecting sleep negatively (Driver & Taylor, 2000). A lot of studies in the field are of a cross-sectional design (Borodulin et al., 2010; Brand et al., 2010; Helena de Castro Toledo Guimaraes et al., 2008; Loprinzi & Cardinal, 2011), which makes it harder to control for confounding variables like these and ones mentioned in previous sections, and since the majority of studies are not of a strict experimental nature there are also problems of determining causality of any significant results. Better sleep might influence a more active lifestyle and not the other way around (Driver & Taylor, 2000; Edinger et al., 1993; Youngstedt et al., 2003), or there might be a more reciprocal relationship in play where sleep influences the degree of activity and the physical benefits of exercise influences sleep (Borodulin et al., 2010).

The literature also mentions further environmental problems, apart from the possible temperature/light-exposure confoundings mentioned in the exercise section, which may affect results. The artificial environment of a sleep-lab study may by its nature not produce the same results as would occur under more normal conditions as it might be stressful for the individual being examined, a point stressed by Youngstedt et al. (2003) as a possible reason why self-reported surveys often indicates a stronger positive effect of exercise on sleep, as they represent evaluations of the effect on sleep in the participants' home environment as opposed to a possibly sleep-inhibiting experimental environment. On the other hand, sleep-studies conducted in the subjects' home environment might have a hard time controlling for extraneous factors such as noise and temperature; the studies by Edinger et al. (1993) and Torsvall et al. (1984) being good examples, where PSG-recordings were made in the participants' home environment during nights in relation to the exercise

conditions. Edinger et al. (1993) specifically mentions this as a possibly confounding influence on the results.

Problems related to research samples are also common within the field, with many labs recruiting experimental subjects from college populations (a common practice in many scientific fields), which are likely to result in a certain age, health and socio-economic bias, all aspects which can contain or mask various confounding variables that might have an impact on sleep. Age being a particularly sensitive sleep-affecting variable as insomnia frequencies differ between ages; e.g. a more compromised sleeping pattern reported in the elderly (Edinger et al., 1993). Gender bias is also a problem, with a majority of studies having been conducted on men, which may influence the results according to Driver & Taylor (2000) because some research-results indicate that women's sleep might be more susceptible to the influence of exercise. Small sample sizes are also an occurring problem, some studies having only between 6-10 subjects (Bunnell et al., 1983; Horne & Staff, 1983; Torsvall et al., 1984; Van Someren et al., 1997), making it harder to control for unwanted confounding variables.

The small exercise-sleep results (a matter of minutes) available coupled with the possibility of the above mentioned research-related problems, makes it hard to decipher whether they're a product of any exercise-sleep correlation or unwanted background variables, and can influence the generalizability of results to the general population.

#### Theories on exercise-sleep mechanisms

Evidence of an exercise-sleep relationship remains controversial and the physiology of the mechanisms behind a possible correlation is equally unclear. There are a number of theories regarding how such a relationship might function, but a real consensus regarding one definite candidate cannot be found within the literature. Most of the reviewed

research studies do not make specific claims regarding the possible mechanisms behind their results.

*The body restitution and energy conservation theory*

The oldest theory regarding the influence of exercise on sleep was referred to in the introduction and is that of bodily restitution and energy conservation. The popular notion is that intensive physical activity results in sleep enhancement as a way of restoring physical constitution and energy sources. Horne (1981) mentions that one reason for this is a historical association of SWS as one instance where the production of growth hormone (hGH) is facilitated in the human brain. However, further studies have indicated that physical fatigue is often temporary in nature and does not seem to be accompanied by feelings of sleepiness (Horne, 1981; Youngstedt, 2005). In the 1981 study "Exercise and sleep behaviour: A questionnaire approach" by Porter & Horne (as cited in Horne, 1981) 51 "good sleepers" engaged in a week of differing levels of physical activities and an increase in physical activity-levels did not correlate with reports of increased sleepiness; tiredness as a result of exercise was reported as only temporary and did not necessitate sleep. According to Horne & Staff (1983) sleep is actually a state of decreased protein synthesis in the body as a result of the cessation of feeding during sleep. It is stated that if bodily restitution is the function of sleep then there should be more focus on physical bodily sleep readings (Horne, 1981) than EEG readings, since these are limited to cortical neural activity (Dworak et al., 2008). Further reviews concludes that the available evidence makes this theory less than compelling (Driver & Taylor, 2000; Youngstedt, 2005), and none of the individual studies that has been reviewed mentions this theory as a plausible mechanism.

*The thermoregulation theory*

A more popular theory is the one concerning the thermoregulatory properties of exercise. The human body has an endogenously modulated predictable flow of core body temperature fluctuations over the course of a 24 hour period; a so called circadian rhythm (Gilbert, van den Heuvel, Ferguson & Dawson, 2004). Circadian rhythms are believed to be controlled by the suprachiasmatic nuclei (SCN) in the anterior hypothalamus. Before sleep and during sleep onset the core body temperature is down-regulated (lowered) (Youngstedt, 2005) by increased peripheral skin blood flow, causing heat to exit the body (Gilbert et al. 2004). It is hypothesized that this triggers thermosensitive neurons in the pre-optic area/anterior hypothalamus which is neurally connected to the SCN, and through there sends impulses to brain structures known to mediate sleep and wakefulness (Gilbert et al. 2004). Exercise has the capacity to elevate human core body temperature, and the theory is that by disrupting the natural circadian temperature-fluctuations in this way during waking hours it causes the circadian thermal system to compensate for the earlier elevation more aggressively when the natural down-regulation occurs before sleep - i.e., by lowering the temperature more than usual (Gilbert et al. 2004). Since the natural decline in core body temperature has been correlated with sleep, the exercise-induced increase in down-regulation is believed to facilitate sleep even further (Gilbert et al. 2004). Horne (1981) suggests that a more intense down-regulation in sleep-related temperature might be the effect of brain-metabolism and subsequent restitution. Brain metabolism increases dramatically with only moderate temperature changes in the brain (e.g., an increase of a mere 2 °C causes a 20% increase in brain metabolism), and exercise has the capacity to increase brain temperature (Horne, 1981). This increase in metabolism might have the same effect as staying awake for a longer period, and so the proposed sleep-inducing thermal down-regulation is increased to faster enable sleep and thereby metabolic restitution for the brain (Horne, 1981). Some evidence

supporting the thermoregulation theory have shown that passive heating has the same kind of SWS-increasing effects (causing the same kind of core temperature elevation) that exercise supposedly has (Atkinson & Davenne, 2007). In a within-subjects experiment investigating the influence of passive body-heating and exercise on sleep in eight physically fit individuals Horne & Staff (1983) subjected each participant to a high-intensity exercise condition (HI), a low-intensity exercise condition (LI), and a no-exercise passive heating condition (PH). The HI and LI conditions were carried out by treadmill running and determined by  $VO_{2max}$  measurements, and the PH condition was achieved by partial bodily immersion in a tank filled with 42 °C water, and was designed to closely replicate the core body-temperature elevation of the HI condition. PSG-recordings showed that similar increases in SWS took place on nights after the HI condition (SWS > 13,3 min) and the PH condition (SWS > 18 min), but not during the less heat-inducing LI condition, providing support to the theory. The literature also mentions that clinical studies of individuals with disturbed sleep due to insomnia and depression have shown matching disturbances in circadian thermoregulation (Driver & Taylor, 2000). Criticism directed at this theory is usually about its far reaching assumptions and purely correlational nature of the supporting evidence, preventing any conclusions regarding causality to be drawn from the results (Gilbert et al., 2004). It is however the most frequently and explicitly mentioned theory in regard to a plausible mechanism for research results in the reviewed individual studies (e.g., Bunnell et al., 1983; Horne & Staff, 1983; Loprinzi & Cardinal, 2011).

#### *The calcium restoration theory*

Animal studies on epileptic and hypertensive mice have shown that exercise elevates blood-calcium levels, triggering a dopamine (DA) synthesis in the brain (Sutoo & Akiyama, 2003). One suggested mechanism for this is that exercise increases the acidity of the blood, making it dissolve bone calcium throughout the body, and transporting the calcium

to the brain via the blood (Sutoo & Akiyama, 2003). The resulting elevation of DA-levels appears to have a normalizing and restorative effect on brain functions in general in the mice, with cessation of epileptic seizures and blood pressure as a visible result, and longer TST during chemically induced sleep (Sutoo & Akiyama, 2003). If this is applicable to human subjects there is a possibility that the normalizing effects of exercise on general brain function also applies to sleep-moderating aspects, providing a solidification of sleep architecture.

#### *Indirect affect*

Sleep is influenced by multiple factors (Gilbert et al., 2004; Youngstedt et al., 2003) and there are compelling reasons to view possible effects of exercise on sleep as indirect effects, its influence working through mediating factors. The earlier mentioned and well-documented influence of exercise on psychological conditions such as depression and anxiety being a good example (Brand et al., 2010; Driver & Taylor, 2000; Youngstedt, 2005), another being the possibly structuralizing influence on life-style. It was previously hypothesized how exercise might affect the circadian system by thermoregulation, which could arguably be an indirect affect as well as no causal direction could be established. Youngstedt mentions the REM increases documented in his meta-review of acute exercise (Youngstedt et al., 1997) as possible indications of exercise inducing phase-delays in the circadian system, to which this sleep-stage have been associated. There are many ways to affect the SCN (the previously mentioned supposed modulator of the circadian rhythms in the brain) though, and it is believed to be mainly influenced by external visual cues (Van Someren et al., 1997), of which bright light has frequently been identified as a significant cue (Driver & Taylor, 2000; Soares Passos et al., 2011; Van Someren et al., 1997). Lighting conditions is registered by the retina, sending information to the SCN and the pineal gland (Turek & Gillette, 2004). The pineal gland produces larger quantities of melatonin when it is

dark, and this is a further circadian phase-shifting signal used by the SCN to align the circadian rhythm towards a natural day/night 24 h cycle (Turek & Gillette, 2004), a phenomenon which has been correlated with a positive effect on sleep (Van Someren et al., 1997). Since exercise is commonly performed under more well-lit conditions (e.g. outdoors or in a gym) than a lot of sedentary activities it is plausible that the exercise in itself is just a facilitator for the exposure to bright light, which in turn influences sleep via the SCN. In a study mapping levels of physical activity by actigraphy in normal living conditions over time in a group of individuals, concurrent light-sensor recordings indeed correlated increased motor activity with increased light-exposure (Youngstedt et al., 2003). Van Someren et al. states that “increased input into the circadian timing system appears to be a final common path of all suggested mechanisms by which increased physical activity influences circadian rhythms” (Van Someren et al., 1997, p. 152).

### Discussion

There is an interest in non-pharmaceutical sleep facilitators in today's society due to high-stress existence and the possibly negative side-effects of pharmaceutical alternatives (Youngstedt, 2003). Among the popularly perceived non-pharmaceutical sleep enhancers exercise exists as an often cheap, readily available and relatively safe alternative, making it an attractive possibility. This review has looked at the theories and scientific evidence for the possible physiological effect of exercise on sleep in non-clinical individuals.

Exercise is complex as a research phenomenon because the term encompasses a wide variety of activities. There are several subtypes and variables of exercise, the most common division being that of acute and chronic exercise. But there is also intensity, duration and aerobic exercise vs. anaerobic exercise to consider, all aspects impacting the human body in different ways. Individual baseline levels also affect exercise performance as does

exercise-environment (like temperature and lighting conditions), and a further possible influence is the time-of-day during which the exercise is carried out. Different approaches towards these variables, along with several different ways in how to measure exercise performance have fragmented research methods within the field and made it extremely difficult to compare existing studies (Horne, 1981; Montgomery & Dennis, 2004; Youngstedt et al., 1997).

Sleep is an equally complex research phenomenon, the function of which is still controversial in the scientific community. There is also some debate regarding which kind of sleep architecture can be considered “good” sleep, SWS having been the popular indicator (Dworak et al., 2008; Youngstedt, 2005). These interpretation differences coupled with differing sleep-measuring methods like subjective reports, actigraphy and PSG, adds to the study-comparison difficulties. Focusing on results from studies on non-clinical individuals can also be limiting due to possible sleep ceiling-effects (Youngstedt, 2003).

The available PSG-documented evidence of an exercise-sleep correlation in the field is conflicting and when significant results are arrived at the effects are small-to-modest, a matter of minutes generally within the mean 1-13 minute spectra. The most common results are increases in SWS, increases in REM, delayed REM onset, decreases in SOL and increases in TST. Due to the existence of conflicting evidence showing no correlations, and the moderate nature of existing positive correlations it is widely concluded that the available evidence is not very compelling and could be the product of differing or poor methods, experimental designs and uncontrolled confounding variables (Driver & Taylor, 2000; Montgomery & Dennis, 2004; Youngstedt, 2003, 2005; Youngstedt et al., 1997). Not controlling for mood fluctuations and quality-of-life assessments is a possible problem as research has indicated that exercise has an alleviating effect on sleep-affecting psychological conditions such as depression and anxiety (Soares Passos et al., 2011; Youngstedt, 2005).

Cross-sectional studies do not have the possibility of determining causality and might possibly also suffer from personality traits as a confounding variable, as people drawn to physical exercise might have individual traits also affecting their wake-sleep pattern. Possible sleep-affecting environmental confoundings are also an issue; the artificial nature of a sleep-lab and possible disturbances in a subjects' home environment. A lot of experimental sample issues are also present in the literature; recruiting bias (college students), gender bias (studies on men) and small sample sizes. The presence of any of these potential problems certainly invites skepticism towards results and can also negatively affect generalizability to the general population.

There are few theories regarding the physiological mechanisms behind a possible sleep-exercise relationship. The body restitution and energy conservation theory resting on earlier beliefs that SWS has physically restorative properties is now considered unlikely as evidence indicates that sleepiness is separate from physical fatigue (Horne, 1981; Youngstedt, 2005) and that sleep has an inhibiting influence on protein synthesis (Horne, 1981). The thermoregulation theory is more popular and hypothesizes that exercise influences the circadian thermic rhythm in the human body, in which a certain temperature down-regulation is correlated with sleep (Gilbert et al. 2004). The heightened heat levels caused by exercise supposedly stimulates the thermic system, making the sleep-related temperature regulation more pronounced and effective (Gilbert et al. 2004). Evidence supporting this theory consists of studies indicating that individuals with clinical sleep disturbances often have a similarly disturbed circadian thermic rhythm (Driver & Taylor, 2000), and studies indicating that passive heating have the same effect on sleep architecture as some reported effects of exercise (Atkinson & Davenne, 2007; Horne & Staff, 1983). The main problem with this theory is that the supposed evidence is purely correlational and not necessarily supportive of the proposed mechanism in particular. There is also the theory of calcium as a

stabilizer of general brain functioning, thereby possibly also sleep. Animal studies indicates that exercise releases calcium to the brain, causing a DA synthesis that has normalizing qualities in brain-functioning in studied epileptic and hypertensive mice (Sutoo & Akiyama, 2003). It is still unclear if this is applicable to humans however. It is also possible that exercise influences sleep in more indirect ways; through affecting psychological aspects like depression- and anxiety-levels, life-style, and the circadian system via exercise-associated increases in light exposure (Van Someren et al., 1997).

### Conclusion

There are problems coming to far reaching conclusions about the available evidence and theories in the exercise-sleep literature due to all the previously mentioned methodological problems and limitations inherent in much of the research. There is no real consensus in the field regarding the existence or non-existence of a possible effect of exercise on sleep physiology. It is indeed very likely that the small-to-modest existing results are just the product of “noise” due to a wealth of uncontrolled confounding variables. The “indirect affect” approach also seems plausible as normal mood-fluctuations could be considered lighter versions of the clinical conditions related to sleep disturbances.

My suggestions for future research would be to focus on objective studies with a stricter experimental methodology and to develop some sort of basic design-checklist to avoid or call awareness to possible confounding variables. Further efforts in examining the causal merits of the thermoregulatory theory and the applicability of the calcium restoration theory on humans would also be of interest. Another suggestion for future research would be the effects of chronic exercise on sleep, as it is still not sufficiently investigated by objective PSG studies. Brain plasticity is generally not instant so there is no good reason in my opinion to expect instant changes in sleep physiology as a result of exercise.

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