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CLINICAL REVIEW

Sleep and exercise: A reciprocal issue?

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SUMMARY

Sleep and exercise influence each other through complex, bilateral interactions that involve multiple physiological and psychological pathways. Physical activity is usually considered as beneficial in aiding sleep although this link may be subject to multiple moderating factors such as sex, age, fitness level, sleep quality and the characteristics of the exercise (intensity, duration, time of day, environment). It is therefore vital to improve knowledge in fundamental physiology in order to understand the benefits of exercise on the quantity and quality of sleep in healthy subjects and patients.

Conversely, sleep disturbances could also impair a person's cognitive performance or their capacity for exercise and increase the risk of exercise-induced injuries either during extreme and/or prolonged exercise or during team sports.

This review aims to describe the reciprocal fundamental physiological effects linking sleep and exercise in order to improve the pertinent use of exercise in sleep medicine and prevent sleep disorders in sportsmen.

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Introduction

Over the last decade exercise has been extensively recommended as a major factor for improved health in the general population, in the elderly and in many groups with chronic diseases such as obesity, type 2 diabetes, cardiovascular diseases, depression and even cancer [1,2]. Increasing exercise has been found beneficial for reducing weight, preventing pain, improving mood and enhancing the quality of sleep in patients with insomnia [1,3–5].

Getting sufficient sleep has also been recommended as insufficient sleep has been identified as an associated risk factor for major public health concerns: obesity, type 2 diabetes, cardiovascular diseases, depression and accidents [6–8]. Sleeping 7–8 h has systematically been found to be associated with lower risks of morbidity and mortality.

However it is still difficult to understand exactly how exercise impacts on sleep and vice versa. In particular, very frequent associations have been found between sleep loss and exercise-induced injuries [9,10] suggesting physiopathological interactions between sleep and injuries. Conversely, good sleeping habits and moderate physical activity could be mutually beneficial [11–13] and trigger a virtuous circle that improves fitness, particularly in sleep disorders patients.

The aim of this review is therefore: 1) to understand how exercise affects sleep physiology, via its impact on temperature, cardiac and autonomic function and the endocrine and immune systems; 2) to clarify how the duration of sleep affects exercise (exploring the impact of sleep loss, sleep restriction and sleep extension); 3) to observe the reciprocal influence between sleep disorders (insomnia and sleep apnea) and exercise.

Effects of exercise on sleep physiology (Fig. 1)

Definitions

Physical activity, sport, exercise and physical fitness are terms that lead to confusion. The term physical activity describes any form of movement that results in energy expenditure and includes all the activities in day-to-day living, whether professional,

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Abbreviations

AHI	apnea-hypopnea index	OSA	obstructive sleep apnea syndrome
BDNF	brain-derived neurotrophic factor	PSG	polysomnography
BMI	body mass index	PSQI	Pittsburgh sleep quality index
CPAP	continuous positive air pressure	REM	rapid eye movement
GH	growth hormone	RLS	restless leg syndrome
HR	heart rate	SD	sleep deprivation
HRV	heart rate variability	SMD	standardized mean difference
ICSD-2	international classification of sleep disorders-2nd edition	SNS	sympathetic nervous system
MAE	moderate-intensity aerobic exercise	SOL	sleep onset latency
NO	nitric oxide	SWS	slow wave sleep
NREM	non-rapid eye movement	TST	total sleep time
		VO ₂ max	maximal oxygen consumption
		WASO	wake after sleep onset

domestic or leisure-time activities [14]. Contrary to sport, physical activity is not performed competitively. Exercise is a component of physical activity; it is planned, structured and defined by its frequency, intensity and duration. Physical fitness is the ability to perform physical activity. A recent recommendation from the American College of Sports Medicine and the American Heart Association regarding physical activity and public health in adults advises that, in order to promote and maintain health, moderate-intensity aerobic physical activity for a minimum of 30 min on five days each week, or vigorous-intensity aerobic physical activity for a minimum of 20 min on three days each week should be carried out [15]. Schematically, moderate-intensity activities are those in which the heart rate (HR) and breathing are raised but where it is still possible to speak comfortably; whereas vigorous-intensity activities are those in which the heart rate is higher, breathing is heavier and conversation is harder.

Effects of exercise on sleep architecture

The effects of exercise on sleep are modulated by factors such as individual characteristics and exercise protocol. Individual characteristics include sex, age, fitness level, type of sleeper and body mass index (BMI), whereas exercise protocol includes acute or regular, aerobic or anaerobic, and different characteristics such as intensity, duration, environment (indoor or outdoor, hot or cold environment) and the time of day. These variables have contradictory effects on sleep. Various studies of this topic have concentrated on good and relatively young sleepers (<35 y) [16–19]; the scientific literature on the elderly and poor-sleepers using objective measurements (polysomnography) is poor [17,20]. It is important to keep in mind a possible ceiling and floor effect of exercise on sleep in good sleepers (i.e., little room for improvement in sleep); subjects with sleep disorders would have the greatest potential for improvement. Interestingly, several studies have since focused on these groups [2,11,21,22].

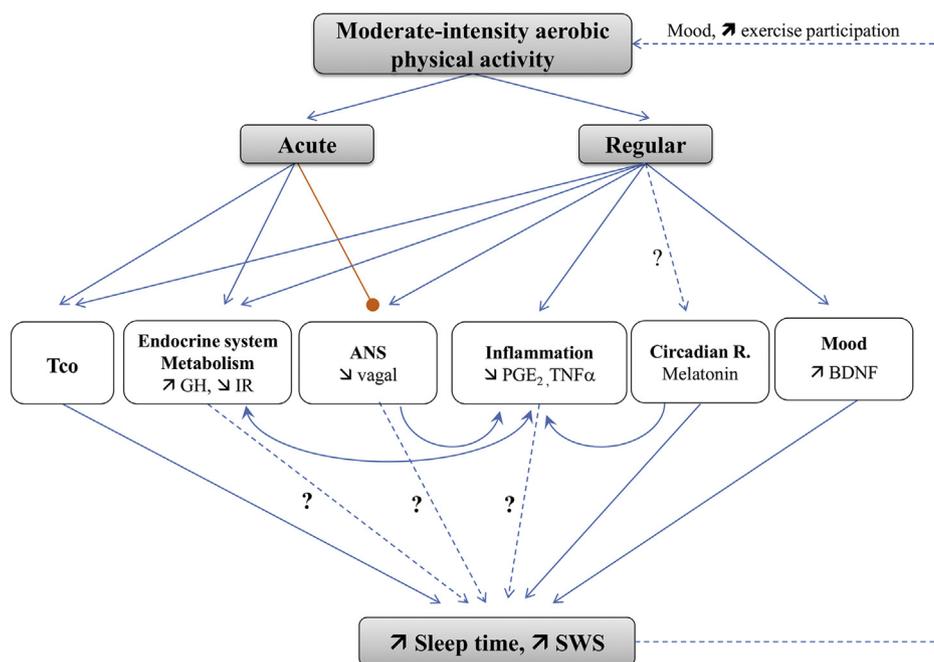


Fig. 1. Possible effects of acute or regular moderate intensity aerobic physical activity on sleep. ANS = autonomic nervous system, BDNF = brain-derived neurotrophic factor, Circadian R. = circadian rhythm, GH = growth hormone, IR = insulin resistance, PGE₂ = prostaglandin E₂, SWS = slow wave sleep, Tco = body core temperature, TNF- α = tumor necrosis factor alpha, —> link, -.-> probable link, —●— inhibits (red). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Acute exercise

More specifically, two meta-analyses reported that the effects of acute exercise on sleep architecture showed a small increase in slow wave sleep (SWS) and rapid eye movement (REM) sleep latency and a decrease in the amount of REM sleep [16,18]. The influence of exercise on improving sleep onset latency (SOL) and decreasing wake after sleep onset (WASO) was found positive when the exercise was performed more than 8 h or less than 4 h before sleep [16,18,19]. However, the studies reporting these deteriorations after exercising in the evening may be biased by the fact they were performed on sedentary women (mean $\dot{V}O_{2\max}$: $32 \text{ ml kg min}^{-1}$) [23], or after an extreme race (30 or 43 km) [24]. In these studies, the stress induced by exercise was very important and sleep was disturbed, probably by the stress. It has also been found that exercising before bedtime may not necessarily disturb sleep (evaluated by actigraphy or questionnaire) [25,26]. Furthermore, recent studies have reported that vigorous or moderate late-night exercise in children and young good sleepers (2–3 h before bedtime) did not affect the SOL or WASO but using objective polysomnographic (PSG) measurements was actually seen to improve SOL [27,28].

In conclusion, these findings seem well established and contradict long-standing “sleep hygiene” tips that advise not to exercise close to bedtime. The National Sleep Foundation has amended its sleep recommendations for good sleepers to encourage exercise without any caveat as to time of day as long as this is not at the expense of sleep duration. Furthermore, once the working day is over, the evening is a moment when exercise can be incorporated into the daily routine as a leisure activity.

Regular exercise

Regular exercise represents an interesting non-pharmacological treatment for poor sleepers [29]. In Kubitz's meta-analysis, regular exercise (called chronic) was commonly found to be associated with increased SWS, total sleep time (TST) and decreased REM sleep, SOL and WASO in good sleepers [16]. A study of fifty-one adolescents (53% female) over a 3-wk period, including 30 min of moderate-intensity exercise every weekday morning confirms these results. It showed an improvement of objective duration and sleep efficiency, SOL was decreased and REM sleep latency increased compared to the control group (without exercise) [30]. Moreover, the adolescents reported an enhancement of subjective sleep quality, mood, and concentration during the day. Other recent studies reported longer TST, shorter WASO and greater sleep efficiency in adolescents with high physical activity levels [31,32]. Over the past decade, a few studies have investigated the effect of regular exercise in older populations and in people with sleep complaints [33–35]. Indeed, in middle-aged and older adults with sleep problems, pooled analyses indicate that exercise training has a moderate beneficial effect on sleep quality, sleep latency and medication usage [34,36,37]. However in the 23 behavioral intervention trials for insomnia reviewed by these authors, just three of them used objective measurements (e.g., PSG). Moreover, a long training program is usually needed to observe any significant effect of exercise on an objective (e.g., PSG study) improvement in the sleep quality of older adults with sleep complaints was observed after a 12-month exercise program [34,36]. However a shorter period of exercise training (i.e., 16 wk of moderate-intensity exercise) only impacted on subjective sleep quality assessed by the Pittsburgh sleep quality index (PSQI) and the global sleep score but not objective sleep [38]. The dose–response effect of exercise training on the subjective sleep quality of postmenopausal women has been confirmed recently [39]. Naylor and Penev also recently observed a similar increase in SWS with only daily social and physical (low intensity) activity over 2 wk in a 65–92-y old group [35].

Basic research in animals may be helpful in understanding the link between exercise and sleep. A period of 8-weeks of chronic aerobic exercise performed at night onset (equivalent to the morning for humans) in old rats significantly decreased sleep fragmentation (35%) and increased EEG delta power (0.5–4 Hz) during non-rapid eye movement (NREM) sleep. The changes in sleep persisted for 2 wk after the end of exercise, indicating the enduring effects of exercise on the central nervous system [40].

Either way, more rigorous methodological studies need to be conducted to better demonstrate the beneficial effects of exercise training on sleep patterns as previous studies have not always controlled the indirect effects of factors such as light exposition, changes of food intake, mood, the effects of antidepressants and circadian rhythms. Moreover, because exercise leads to significant long-term improvements in body composition, basic metabolic rate, cardiac function, glucose control, mood and immune function [18,41], it is actually hard to discriminate the direct and indirect pathways implicated in the exercise-induced changes to sleep.

Effects of exercise on body temperature during sleep

Many studies have reported a clear link between sleep and body temperature [42,43]. Indeed, sleep could be promoted by a decrease in body temperature (0.5–1 °C) [44] whereas an increase in body temperature (1.5–2.5 °C) alters sleep onset [45,46]. This decrease at the level of the brain, in particular the pre-optic area, seems to trigger sleep [47]. So since exercise affects body temperature both during the exercise and recovery, it has been suggested that exercise could influence sleep.

Exercise induces an increase in central, skin and cerebral temperature, related to the intensity and duration of the exercise and the climatic conditions (temperature, humidity) [48,49]. So when the exercise ceases, the body temperature is raised [44] with the same effects on sleep as passively changing the body temperature. When subjects are acutely exposed to heat immediately before sleep, decreases in total sleep time and SWS occur, being often accompanied by synchronic (concomitant) diminution in REM sleep [49].

Conversely, immersion in warm water, inducing a rise in body temperature of 1.5–2.5 °C, decreased the SOL and enhanced the SWS the following night [45,46]. Horne et al. (1985) also compared the effects of a running exercise in the hot condition (by wearing extra clothes) and cold condition (via body cooling); only exercise in the hot condition elicited increases in SWS [50]. Indeed, after the initial passive or exercise-induced hyperthermia, the thermoregulatory mechanism comes into play, decreasing the body temperature via a peripheral heat dissipation mechanism corresponding to peripheral vasodilation. This gradient between distal and proximal skin temperatures seems to be essential for initiating sleep [51,52]. This rapid decline in core body temperature following active or passive heat exposure increases the likelihood of sleep onset and may facilitate entry into the deeper stages of sleep [42,53]. Moreover, regular physical activity (1 h, three times per week), induces a more regular decrease in body temperature and promotes sleep [53].

Effects of exercise on cardiac and autonomic function during sleep

Sleep is characterized by important changes in the circadian rhythm of heart rate and blood pressure induced by declining sympathetic and increasing parasympathetic activity during the night, in particular during SWS [54]. As investigation of cardiac function in humans is straightforward, many studies have assessed the changes in autonomic nervous system activity non-invasively by using heart rate variability (HRV) and HRV is frequently applied to understand autonomic changes during different sleep

stages and the effect of insomnia, sleep disordered breathing during sleep and the daytime (See for review [55]). Sleep disorders and particularly sleep loss induces a well-known constant sympathetic overactivity associated with heart rate, blood pressure and an increased risk of cardiovascular and metabolic diseases [55–57]. During chronic insomnia, patients showed an increase in heart rate and a decrease of HRV, before sleep onset and during stage-2 NREM sleep, but compared to controls, showed no differences during Stage-3 NREM sleep. These results are consistent with the hypothesis that autonomic hyper-arousal is a major pathogenic mechanism in primary insomnia [58]. During exercise, the sympathetic nervous system (SNS) is widely stimulated [59]. Several investigators have examined the changes in cardiac and autonomic function immediately after a single bout of sub-maximal exercise (15–180 min), characterized by an increase in heart rate caused by a decrease in parasympathetic activity and an increase in sympathetic activity [60,61]. These changes in sympathovagal balance immediately post exercise gradually return to pre-exercise values during the following 24 h [61]. A meta-analysis on the effects of exercise on heart rate variability has reported that regular exercise improves vagal modulation, with a resulting decrease in heart rate [62]. Several studies have shown an increased heart rate during sleep after daytime exercise [24,27,63,64]. Similarly, moderate and heavy exercise (marathon) leads to decreased nocturnal global HRV, indicative of a decrease in parasympathetic activity [63]. However, when exercise was taken very late in the day, other studies observed either no change in HRV parameters during sleep or only at the beginning of the night [65,66]. Thus, the elevated heart rate during early sleep seen after exercise may be a result of a non-significant decrease in vagal activity and increased sympathetic activity [27].

Conversely, a period of 2 mo of intensive training has been seen to increase vagal activity and decrease sympathetic activity during sleep in sedentary subjects [59]. This enhancement of vagal modulation after regular exercise is interesting as increased heart rate variability is a predictor of reduced risk of future cardiovascular events [67]. Overtraining (defined as excessive training with insufficient recovery and chronic decrement of performance) induced a contrary effect, i.e., a progressive decrease in parasympathetic activity and an increase in sympathetic activity during sleep in sedentary or trained subjects [59,68] and reduced heart rate variability was seen to predict poor sleep quality in a case-controlled study of chronic fatigue syndrome [69].

Nevertheless, we failed to find evidence that changes in HR and autonomic activity could directly affect or improve sleep quality and quantity. Burgess et al. [70] observed that cardiac sympathetic activity did not significantly change across NREM-REM cycles. Their results also indicate that the effect on cardiac sympathetic activity of the time spent asleep may be greater than the influence of sleep cycles. Recently, Myllamaki et al. [65] observed that neither increased exercise intensity nor duration disturbed sleep quality (actigraphic study) although significant increases in HR and changes in HRV were observed.

Effects of exercise on endocrine function during sleep

The main hormonal axes, which respond to exercise, are the gonadal and somatotrophic axes, the hypothalamo–pituitary–adrenal axis and the SNS axis. The metabolic and hormonal changes during exercise are responsible for important modifications, both to some central neurotransmitters and some immune functions.

During human sleep, the hypothalamo–pituitary–adrenal and SNS axes are down-regulated with the decrease of plasma cortisol, epinephrine and norepinephrine levels but at the same time there

is a marked increase in growth hormone (GH), prolactin, and melatonin [71,72]. A rapid increase in plasma thyroid stimulating hormone is observed in the early evening and at around the beginning of sleep [73]. However at the end of sleep, there is a decrease in thyroid stimulating hormone levels.

Nocturnal GH concentrations following daytime physical activity have been reported to increase [74,75], decrease [76,77] or remain unchanged [75,76].

In the 1995 study by Kern et al. [75], the authors investigated the hypothesis that long-duration exercise of moderate, but not low intensity, during the day changes the typical temporal patterns of GH and cortisol during subsequent nocturnal sleep in tri-athletes. They observed that this exercise did not affect the nocturnal plasma levels of GH or cortisol. However, when the night was divided into two periods, the long-duration exercise of moderate intensity induced a decrease in GH secretion and increased cortisol secretion during the first part of sleep, while in the second part of sleep GH secretion was increased and the increase in cortisol secretion was lower.

More recently Tuckow et al., 2006 [78], investigated the effects of a daytime exercise bout on subsequent overnight GH secretion. They demonstrated that despite the modification in secretory dynamics, no change in 12-h mean or integrated GH concentrations were noted in the control and exercise protocol conditions. So although quantitatively similar total levels of GH were secreted overnight in the two conditions, resistance exercise modified the dynamics of secretion by attenuating burst mass and amplitude but increasing burst frequency.

Exercise can also affect the levels of melatonin; however the results are conflicting due to indirect effects (light exposure, time of day, intensity of exercise, gender and age) [79,80] and have an indirect effect on sleep.

Effects of exercise on metabolic functions during sleep

In the last forty years, there has been great interest in the role of sleep on metabolic and endocrine function in the relationship between sleep loss, obesity and diabetes [81,82]. Yet as Uchida et al. pointed out in their 2012 review, no data are available for the effects of exercise on metabolic functions during sleep [83].

However, growth hormone and cortisol are two hormones that have an impact on glucose regulation. The effect of exercise on GH and cortisol secretion can induce increased glucose utilization during the REM phase of sleep and increased glucose levels in the evening with reduced insulin sensitivity [84]. More research needs to be done on the effects of exercise on metabolic functions during sleep.

Effects of exercise on the immune-inflammation response during sleep

Sleep and the circadian system influence the immune functions [80,85] and there is increasing evidence to suggest bi-directional communication between sleep and the immune system. Investigations of the normal sleep–wake cycle indicates that immune parameters (numbers of undifferentiated naive T cells) and the production of pro-inflammatory cytokines (interleukin-6, tumor necrosis factor alpha, interleukin-12) increase during early nocturnal sleep whereas immune cells (natural killer cells) and the production of anti-inflammatory cytokines (interleukin-10) increase during daytime.

Because sleep loss induces elevated levels of cortisol, decreased testosterone and growth hormone levels [83], these results could explain the potential protective effect of exercise in sleep deprived subjects. Nevertheless, a new study must be conducted in order to

confirm this hypothesis. And new research needs to be done on the anti-inflammatory effects of physical training on immunoinflammatory responses induced by sleep deprivation (SD) and/or sleep restriction.”

Effects of exercise on mood during the night

In addition to physiological changes, it is well known that exercise also improves mood state [86–88], which can also be an important additional factor in improving sleep. Moreover, sleep disorders are associated with an increased risk of anxiety and the development of depression [89]. Physical training is significantly associated with a decrease in anxiety and its physiological indicators and can reduce the prevalence of depression and improve the mental health of large populations [90]. Moreover, regular exercise decreases REM sleep which has a significant anti-depressant effect over time [91]. The anti-depressant effects of exercise have recently been well studied, and the improved mood results from elevated levels of brain-derived neurotrophic factor (BDNF) which is directly attributable to exercise and so indirectly, to improved sleep quality [83,86,87,90]. Such pathway relationships between the acute and chronic effects of exercise and alterations in sleep should be examined in future studies.

To conclude this part, we have shown that many interdependent pathways may explain the impact of exercise on sleep quality. Further research is required to explain how exercise impacts on the quantity of sleep and how these pathways interact to modify SWS and REM sleep.

Effects of sleep on exercise

Introduction

Improving the sleep quality of sportsmen and high-level athletes is important, because it is vital for high levels of mental and

physical performance, general well-being and the recovery process but also for the prevention of exercise-induced diseases [92–95]. Athletes do not usually benefit from optimal sleep conditions and are exposed to circadian disruption (jet lag during international events), sleep habit changes (hotel, two players per room ...), stress and muscular pain due to intense night time exercises as explained previously. Moreover, sleep loss could also alter exercise activity in non-trained subjects and patients via many direct (fatigue, sleepiness) and indirect effects (mood, vigilance ...) (see Fig. 2).

Effects of acute sleep loss on exercise performance

It is consensually admitted that sleep loss may have substantial psychological effects. However, the impact of sleep loss on exercise performance appears to us both inconclusive and unconvincing. Exercise performance depends on physiological, psychological and biomechanical parameters and the impact of sleep deprivation on these is poorly understood at the moment.

Regarding maximal oxygen consumption (VO_{2max}), a determining factor in aerobic effort, some authors have shown SD to be associated with a decrease in VO_{2max} [96–98], others did not find any change of VO_{2max} associated with SD [99–102]. Similarly, oxygen consumption during constant-load efforts seems not to be influenced by SD [97,103–105], while exercise performance during constant-load efforts was decreased in the SD condition [102–104,106]. Some authors have explained the impact of SD on exercise as an enhanced perception of the exertion during exercise known to decrease sub maximal performance [107–109]. These psychological changes during exercise and the many adverse effects of SD on cognitive performance such as increased lapsing, cognitive slowing, memory impairment, a decrease in vigilance and sustained attention, shifts in optimum response capabilities [110] or dysfunctional emotional regulation [111] may influence physical performance especially during sports.

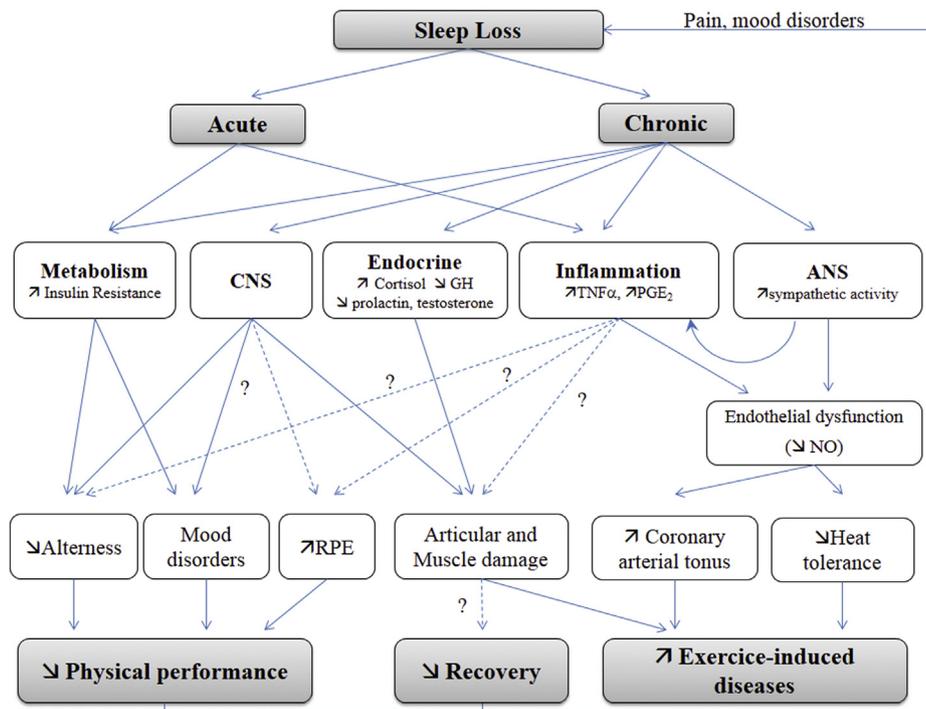


Fig. 2. Possible effects of acute or chronic sleep deprivation/sleep loss on physical performance, muscle recovery and exercise-induced diseases. ANS = autonomic nervous system, BDNF = brain-derived neurotrophic factor, BP = blood pressure, Circadian R. = circadian rhythm, IR = insulin resistance, GH = growth hormone, HR = heart rate, PGE_2 = prostaglandin E_2 , RPE = rating of perceived exertion, $TNF-\alpha$ = tumor necrosis factor alpha, —> link, -.-> probable link.

Based on scarce literature it appears that in both males and females, SD (one night) is not associated with any change in anaerobic performance [112] nor in the contributions of the anaerobic energy systems to high-intensity exercise [101,112]. Maximal strength loss was not observed during either isometric or isokinetic contractions of upper or lower limbs during 60 h of continuous wakefulness [105,113]. However, other studies observed a modest decrease in functional performance of the upper and lower limbs after a complex combination of sleep loss and exercise or after prolonged SD (36–100 h of wakefulness) [114–117]. Moreover SD seems to reduce the difference between morning and afternoon performance assessed with anaerobic power variables [114,118].

It is also hypothesized that acute SD may affect exercise via immuno-inflammatory changes. In particular it was observed that sleep restriction increased pro-inflammatory cytokine, GH, and testosterone concentrations after physical exercise (brief sprints) but did not affect cortisol responses [119]; nor did a 30-h period of SD alter leukocyte trafficking, neutrophil degranulation or secretory immunoglobulin A responses either at rest or after submaximal and strenuous exercise [120]. However, an increase in insulin resistance and a decrease in glucose tolerance, both associated with SD (although partially reversed by physical activity) [121] were also seen as contributing to exhaustion.

Effects of chronic sleep restriction and sleep quality on exercise performance

Short sleep is very prevalent among industrial populations and may affect up to 30% of young adults [122]. However there is, to our knowledge, no literature devoted to the association between chronic sleep restriction and the level of physical exercise in healthy subjects and little is known about the effects of chronic sleep restriction (sleep debt) on exercise and performance.

Similarly, the role of sleep quality on exercise is still an unexplored field of research. We have found only one study in a 14,148 sample of United State Army active, reserve, and National Guard members (83.4% male), in which sleep quality was assessed from two questions validated by the PSQI (0–6). Poor sleepers (5–6) were significantly ($P < .001$) more likely than good sleepers (0–1) to meet aerobic exercise and resistance training recommendations, and pass their army physical fitness test in the last quartile [123].

Effects of sleep loss on exercise-related injuries

Some studies indicate that chronic or acute sleep loss is directly correlated to athletic injuries [121,122]. Other authors defined a specific disease called “fatigue-related injuries”, related to sleeping ≤ 6 h the night before the injury and stated that this reduced amount of sleep is a direct, independent risk factor for injuries during exercise [9]. In particular, sleep deprivation increases the risk of over-strain injuries to the locomotor system [123] that could be linked to the decrease of proprioception and postural control [124,125] and reaction time [104] observed after acute sleep loss.

Recent sleep loss also impairs the functional recovery of muscles following injury. Specifically, 8 h of sleep deprivation acutely down regulated activity of the protein synthesis pathway that repairs muscle damage and triggered contractile function deficits during recovery [95,124]. These findings highlight the role of sleep in the regeneration of damaged muscle tissue. Resistance exercise could be a non-pharmacological strategy to minimize or reverse sleep deprivation-induced muscle damage [92].

Moreover, athletes practicing contact sports who experienced concussions during the previous year reported more symptoms of sleep disturbance and poorer sleep quality than did the controls [125] and subjects with a low sleep quantity the night before the

concussion reported both a greater number greater number of symptoms and more severe symptoms after the concussion [126]. Sleep restriction is a potent contributor to the development of somatic symptoms, particularly in males associating both sleep deprivation and exercise [10] who reported increased pain sensitivity [127]. In particular, sleep problems are associated with an increased risk of chronic pain in the lower back, neck and shoulders [13]. Nevertheless, regular exercise and maintenance of normal body weight may reduce the adverse effects of mild sleep problems on the risk of chronic pain [13]. Taken together, these results suggest the creation of a pernicious circle including sleep loss, injuries, decreased recovery patterns, an increased pain that themselves favor sleep disturbances.

Sleep disturbances are also associated with overtraining during periods of high volume training [94,128,129]. Moreover reduced sleep quality can be associated with higher prevalence of upper respiratory tract infections in overreaching populations [130].

The physiopathological pathways implicated probably associate elevated levels of cortisol and inflammation and the decreased levels of testosterone and growth hormone observed during acute and chronic sleep loss, that may interfere with tissue repair and growth [124,127,131,132].

Moreover, some epidemiological and laboratory studies have suggested that SD could decrease the tolerance to exercise in extreme weather (i.e., heat or cold) [133–138]. So, sleep needs to be preserved before and during exposition to environmental thermal constraints. This question is of importance for subjects who do outdoor sports with their inherent environmental constraints and those who take part in long trail competitions with their additional sleep deprivation constraints [133].

Indeed, among United States high school athletes, heat illness is the third leading cause of death and SD has been identified as one of the risk factors for heat illness and exercise-induced injuries [134]. In particular, SD has been observed in more than 20% of cases of passive heat stroke [135,136]. SD is also considered as a predisposing factor for heat acclimation inability [137] or exertional heatstroke [134,135]. Landis et al. [137] interestingly observed that one night of total SD (24–33 h of wakefulness) alters thermoregulatory responses. This response is characterized by 1) the attenuation of peripheral vasodilatation during passive or active heat exposure [137–139] and 2) a lower sweat rate during moderate intensity exercise [139,140]. The impact of SD was rather strong, even in trained athletes as it has been observed that 28 h of wakefulness were sufficient to alter the benefit of 10 d of exercise-heat acclimation [141]. Conversely, Moore et al. [142] observed that three nights of partial SD (i.e., 2 h of sleep per night) did not alter thermal strain, whole-body sweat rates or performance during exercise in the heat.

The mechanisms that link SD to environmental diseases are not well known and require further study. Adaptation to environmental stress or exercise-induced heat exposure requires intact vasomotricity and sweat gland activity [143]. In particular, during heat exposure, a decrease in heat-induced vasodilatation, a well-known nitric oxide (NO)-dependent endothelial mechanism, leads to decreased tolerance to heat and caloric dissipation through the skin [144]. Many studies have described that, in healthy subjects, an alteration of endothelial NO production occurred after only one [145] or three nights of sleep restriction [146] or after 29 h of continuous wakefulness [56]. This dysfunction could be related to an increase in sympathetic vasoconstrictor activity [147] and/or localized endothelial dysfunction [148] induced by a low-grade inflammatory response to sleep deprivation [56,131], that could persist after two nights of recovery [132]. It has been demonstrated that low-grade inflammation, as observed after upper respiratory or gastrointestinal illness, is sufficient to decrease endothelial-

dependent vasodilatation and tolerance to heat [135] via an alteration of the bio-availability of endothelial-NO [149].

Other authors have suggested that acute sleep loss is also a risk factor for cold injuries (i.e., hypothermia and freezing of the extremities) [150]. Recent work has demonstrated that 29 h of wakefulness alters local response to cold [151] characterized by lower skin temperatures and cutaneous blood flow during cold exposure and recovery. These changes are considered as risk factors for local cold injuries (frostbite) and decreased dexterity during cold exposure [152]. These potential effects of sleep loss need to be taken into account when physical activity is carried out in the cold, in particular for sports needing high dexterity (biathlon, mountaineering ...). The mechanisms involved are probably a combination of increased sympathetic activity and alterations to endothelial NO availability [153] both implicated in cold-induced vasodilatation [153], a frostbite and dexterity protective mechanism.

Protective effects of exercise before and during sleep deprivation

To date, only a few studies have evaluated the extent to which exercise modifies performance in the SD condition and most of these have regarded exercise as an additional stress factor [154,155]. In particular, it has been shown that, in humans, short bouts of exercise may improve the sleepiness and fatigue associated with SD. However, acute exercise is not likely to prevent performance decrements [154]. Similarly, the effect of eight, intense cycling sessions (15 min at 20 km/h against a load of 2.5 kg) during 48 h of continuous wakefulness was seen to be associated with an improvement in complex addition tasks, short-term memory and auditory vigilance [155].

More recently, it was observed in rodents that regular physical exercise improves many aspects of brain function and could even induce neuroprotection. Indeed, 4 wk of physical training has been shown to prevent the impairment of long-term memory associated with sleep deprivation (24-h continuous wakefulness or 96-h REM sleep deprivation) [156,157]. One possible explanation advanced by the authors was that alterations of synaptic plasticity in the hippocampus (CA1 area) [157] and the dentate gyrus [158] were blocked, probably via a decreased level of BDNF [156,158] and an increase in the protein phosphatase calcineurin [158] and oxidative stress [159].

Sleep extension

Recent research has investigated how physical performance may be improved using sleep extension. Mah et al. [12] studied the impact of extended sleep over 5–7 wk on physical performance in young basketball players. Extended sleep contributes to improved athletic performance especially in shooting percentage and sprint times. Cognitive performance (reaction time), mood, fatigue, and vigor were also improved with increased TST. However, even if subjects were well-trained before beginning the study, these results need to be confirmed with traditional controls. Moreover, one week of sleep extension (10 h in bed per day) improved resilience during subsequent sleep restrictions and facilitated task acquisition during recovery, demonstrating that nightly sleep duration exerts long-term (days, weeks) effects [160]. The extent to which sleep restriction, measured objectively, impaired alertness and performance and the rate at which these impairments were subsequently reversed by recovery sleep, varies as a function of the amount of nightly sleep obtained prior to the sleep restriction period. This suggests that the physiological mechanism(s) underlying chronic sleep debt may undergo long-term (days/weeks) accommodative/adaptive changes [161]. Crediting a “sleep bank” may be an protective countermeasure to subsequent sleep deprivation. Thus behavioral interventions designed to increase sleep duration may

be an effective strategy in the treatment of various pathologies [162,163].

Clinical issues

Exercise and insomnia

Insomnia affects one adult in four in most countries [164] and 10% of these are severely affected. Insomnia is consensually defined based on the criteria of the DSM-IV [165] or the international classification of sleep disorders-2nd edition (ICSD-2) [166]. Due to its high prevalence and to the possible emotional impact of performance and competition on sleep, it is understandable that insomnia may affect exercise and performance. Conversely exercise has been proposed as a way to improve sleep in subjects with insomnia. Baron et al. [11] demonstrated that sleep influences next-day aerobic exercise rather than exercise influencing sleep. The relationship between TST and next-day exercise was stronger for those with shorter TST at baseline. These results suggest that improving sleep may encourage exercise participation. There are two sides to this issue that will be discussed in more detail.

Does insomnia affect exercise?

Even if a large consensus exists regarding the fact that its daytime impact is a major issue in the definition of insomnia [165–167], the nature and the magnitude of this impact are still controversial. Some studies have underlined objective impairments in subjects with primary insomnia: a high level of cortisol [168] and a higher heart rate variability [169]. However, these objective data do not suggest a clear link between insomnia and fatigue, irritability or a decrease in daytime functioning. In a review on insomnia and daytime functioning, Riedel and Lichstein [170] proposed that the paucity of objective findings in the literature may be due to the fact that a) attempts at objective verification have focused on variables that are unimpaired rather than areas of actual impairment, or that b) methodological problems, such as between-subject variability, have hidden actual differences between insomniacs and persons without insomnia. Daytime sleepiness has received the most attention, but it is becoming clear that a large number of insomniacs are not sleepy during the day [89,170] except when they have other sleep disorders (such as sleep apnea) or associated sedative treatments [171]. Using a multiple sleep latency test Bonnet and Arand [89] demonstrated that insomniacs were even more alert than good sleepers during daytime.

Consequences of insomnia: relevance for athletic performance

There is no doubt that the question of the impact of insomnia on athletic performance is a crucial issue. Competition is considered by many athletes as stressful enough to impact their sleep the night before an event and they may worry about the influence of poor sleep on their performance. There are few studies devoted to the impact of insomnia on performance in athletes [172]. Performance is a mix of physical and cognitive excellence and the question is to understand how insomnia may affect both aspects. Travel and jet-lag are also disrupting sleep and are frequently experienced by athletes before a competition [173]. The jet-lag disorder is a circadian rhythm sleep disorder that occurs as a consequence of rapid travel through multiple time zones. The traveler may experience excessive sleepiness, fatigue, insomnia, irritability, dysphoric mood, gastrointestinal disturbance, or other symptoms after arrival at the destination. Jet lag symptoms arise from the desynchronization between the body's circadian rhythm, which is synchronous with the location of departure, and the new sleep/wake cycle required at the destination. The body clock desynchronization may affect sleep quality, quantity, sleepiness and performance which appear to be

very sensitive to a global synchronization of physiological circadian variables such as internal temperature, cortisol levels and cardiac and respiratory rhythms which are all synchronized by the clock [174]. It is therefore in the interest of the individual athlete and team to understand the effects of jet lag and the potential adaptation strategies that can be adopted such as light, chronobiotics (e.g., melatonin), exercise, and diet and meal timing [175].

Regarding the impact of insomnia on the physical performance of athletes, several mechanisms may be involved. Insomnia may be associated with SD and it is well documented in this review that sleep deficit has consequences on several physical factors for performance in young athletes [103,109]. Insomnia may also be associated with increased sleep fragmentation, which may also impact negatively on daytime activity, as has been shown in shift workers [176,177]. However we have no knowledge of specific data regarding the performance of athletes with insomnia. Alongside physical performance, it is easy to understand that attention, concentration and memory can also have a major impact in some sporting competitions and that insomnia may alter these cognitive factors of excellence in performance. However it is still unclear exactly how insomnia impacts on next-day cognitive performance. In a recent review examining neurobehavioral impairments in primary insomnia and attempting to identify which cognitive domains were most consistently impaired in this group, the authors found that it was attention tasks, which have a high cognitive load, and working memory tasks that showed performance deficits in insomnia patients [178].

Insomnia may also result from the competition itself. Increased internal temperatures due to high intensity exercise, muscular pain and increased emotions may contribute to disturbed sleep which may have implications for subsequent exercise training.

The interaction between insomnia, mood and anxiety has been extensively described [179] and is very relevant, even in young adults and college students [180]. Insomnia may be an early symptom of depression and anxiety, which may themselves affect performance. Conversely insomnia is a risk factor in the development of stress, anxiety and depression [179].

Finally drug treatments for insomnia may also affect alertness the following day and therefore impact on an athlete's performance. Again there is little literature either on the prevalence of athletes taking hypnotics or on the effects of hypnotics on performance. In a questionnaire survey completed by 1459 high school student athletes in the east of France, 4% stated that they had used doping agents at least once in their life (their main source of supply being peers and health professionals). Thirty-four percent of the sample smoked some tobacco, 66% used alcohol, 19% cannabis, 4% ecstasy, 10% tranquilizers, 9% hypnotics [181].

One study compared cognitive function using the critical flicker fusion test in athletes treated with zolpidem versus placebo [182]. They estimated that the effect of zolpidem was only hypnotic and did not disturb psychomotor and physical performance on the following day in healthy adults. They then suggested that Zolpidem could be used in healthy athletes to adjust their extrinsic sleep disturbances and the resulting psychomotor and physical impairments. This was also found with both zolpidem and zaleplon in a survey of 12 healthy male trekkers who used these treatments to improve their sleep, but who demonstrated no alteration in performance or acute mountain sickness at 3613 m altitude [183]. In a review devoted to the effects of melatonin in sport medicine [175], the authors estimated that melatonin could be useful for treating some sleeping disorders, even though the interactions between sleep, melatonin and exercise had not been studied extensively with trained study participants. It is unknown whether melatonin plays a role in some exercise training-related problems such as amenorrhea and over-training syndrome.

Does exercise influence insomnia?

Alongside cognitive behavioral therapy, exercise is commonly recommended as a treatment for insomnia. How exercise may affect sleep has already been discussed, but we will look here at the literature studying exercise and insomnia from an epidemiological and clinical point of view.

There has been to our knowledge only one study (by Merrill et al.) to look at the epidemiological effects on insomnia of an intensive lifestyle modification program. The program associated sport and stress management, 10 h per week over 4 wk in 2624 adults with insomnia [184]. Insomnia was decreased by 64% in the group. Those who failed to lower their coffee/tea use after four weeks were significantly more likely to have a sleep disorder and be highly emotive. Other studies have concentrated on sleep disorders in general rather than insomnia, using the PSQI. Yang et al. recently reviewed the effects of exercise training on sleep quality in middle-aged and older adults with sleep problems [36]. From six studies involving 305 participants (241 females) and an average training duration of 10–16 wk they found that the participants with an exercise program had a better global PSQI score, with a standardized mean difference (SMD) of 0.47 (95% CI 0.08–0.86) than did the control group. The exercise group also had significantly reduced sleep latency (SMD 0.58, 95% CI 0.08–1.08), and medication use (SMD 0.44, 95% CI 0.14–0.74). However, the groups did not differ significantly in sleep duration, sleep efficiency, sleep disturbance, or daytime functioning.

Clinically, we found relatively few studies devoted specifically to exercise and insomnia. Guilleminault et al. used 22 patients with insomnia to test the effects of moderate-intensity aerobic exercise (MAE) associated with sleep hygiene therapy on their sleep, evaluated by a sleep log and actigraphy. After four weeks of the regimen, they observed a non-significant trend towards an increased total sleep time and a reduced total wake time [185]. Passos et al. tested the effects of three different modalities of acute physical exercise, assessed by polysomnography, on the sleep of 48 patients with chronic primary insomnia [5]. The patients were assigned to four groups: control, MAE, high-intensity aerobic exercise, and moderate-intensity resistance exercise. Significant changes in sleep were only observed in the MAE group, with a 55% reduction in sleep onset latency, a 30% reduction in total wake time, as well as an increase in total sleep time (18%) and sleep efficiency (13%). This MAE program incorporated the following exercises: shoulder press, chest press, vertical traction, leg press, leg curl, leg extension, abdominal crunch, and lower back exercise. Three sets of 10 repetitions at 50% of one-repetition maximum were performed, interspersed with 90-s recovery intervals. The exercise session time was approximately 50 min. Passos et al. [22] also investigated the effects of long-term (6 mo) moderate aerobic exercise on insomnia, in a smaller group of subjects, using polysomnography. They found a significant decrease in sleep onset latency (from 17.1 to 8.7 min) and wake time after sleep onset (from 63.2 to 40.1 min), and a significant increase in sleep efficiency (from 79.8 to 87.2%) following exercise. These effects did not vary between morning and late-afternoon exercise. Sleep quality after aerobic exercise was also assessed in 17 sedentary adults aged ≥ 55 y with insomnia, using the PSQI [186]. Patients were randomized into two groups: 16 wk of aerobic physical activity plus sleep hygiene compared to non-physical activity plus sleep hygiene. The physical activity group improved in sleep quality on the global PSQI, sleep latency, sleep duration, daytime dysfunction, and sleep efficiency PSQI sub-scores compared to the control group.

However the intake of many other sedative antihistaminic or benzodiazepine hypnotic drugs may be associated to side effects such as daytime sleepiness in the morning, confusion, nausea, muscle weakness [162,163]. The prescription of hypnotics in

athletes has therefore to be limited and if possible not proposed for the first time at the eve of competitions.

Exercise and sleep apnea

Sleep apnea affects from 5 to 10% of adults. Males, and those who are overweight or obese are more frequently and severely affected [187]. Sleep apnea is consensually defined based on the criteria of the ICSD-2, which lists non-restorative sleep, sleepiness and intermittent hypoxemia as crucial signs of the disease; this may potentially impact on exercise. Reciprocally it is not at the moment obvious that exercise may have an impact on the sleep apnea symptomatology. However, as it is well recognized that exercise has a powerful impact on weight loss and that weight loss improves sleep apnea [188], it is therefore not easy to clarify the relationship between sleep apnea and exercise independently of weight.

Does sleep apnea affect exercise?

It is widely recognized that sleep apnea does in fact limits exercise due to several associated factors.

From a cardiovascular point of view, obstructive sleep apnea syndrome (OSA) is commonly associated with specific exercise response characteristics, like exaggerated blood pressure [21,189] or delayed heart rate recovery [190,191]. Recently Mansukhani et al. carefully examined the link between OSA severity and exercise testing outcomes independently of BMI and other cardiopulmonary risk factors in a sample of 1204 patients [190]. They concluded that patients with severe OSA (apnea-hypopnea index, AHI ≥ 30) had a significantly lower functional aerobic capacity and heart rate recovery, and a higher post exercise blood pressure than those without OSA (AHI < 5) after accounting for confounding factors (all $p < 0.05$). They concluded that OSA severity was associated with reduced functional aerobic capacity and increased post-exercise blood pressure independently of other confounding variables.

Sleepiness may be one explanation for reduced physical activity in patients with OSA. From a behavioral point of view, sleepiness reduces the need and the time for physical activity. In a survey of 40 patients with obstructive OSA aiming to clarify their exercise capacities and their possible relationships with other findings, a battery of aerobic and anaerobic exercise tests was performed in these patients and 40 control subjects [191]. They showed that the apnea-hypopnea index was a significant independent predictor of aerobic capacity after controlling for a variety of potential confounders including BMI. They also suggested that the lower aerobic exercise capacity of patients with OSA might be due to daily physical activity that is restricted by the OSA itself. Sleepiness also reduced the hypertensive response to exercise [21]. However the lack of exercise itself seems to be predictive of the degree of sleepiness in OSA. In an important survey of 1106 consecutive patients (741 men and 365 women) referred to a sleep clinic for OSA, daytime sleepiness was assessed with the Epworth sleepiness scale and activity evaluated with a quantifiable physical activity questionnaire [192]. Linear regression analysis of the total sample after adjusting for age, BMI, sex, central nervous system medication, and diabetes showed that AHI, depression, and lack of regular exercise were significant predictors of sleepiness. Predictors of mild or moderate sleepiness for both sexes were depression and AHI, whereas predictors of severe sleepiness for men were lack of regular exercise, depression and minimum saturation of oxygen and for women, AHI.

It is also clearly possible that the reduced observed VO_2 peak observed in sleep apnea may explain the reduced activity and fatigue [189,191]. Understanding how continuous positive air

pressure (CPAP) may improve physical exercise in patients treated for OSA may clarify the relation between sleep apnea, fatigue and exercise. Alonso-Fernandez et al. found nasal CPAP reversed the depressed cardiovascular response to exercise in their study of 30 patients with OSA before and after CPAP versus sham CPAP (control) and 15 healthy subjects [193]. CPAP therapy was associated with highly significant improvements in all the indices of left ventricular systolic performance response during exercise, whereas with sham CPAP, all of them remained unchanged. CPAP also allowed an increase in walking distance in 12 subjects with OSA recovering from heart failure [194]. It was observed that during an exercise test, CPAP increased the distance covered (538 ± 78 m versus 479 ± 83 m) when compared to placebo. Ackel d'Elia et al. found nasal CPAP plus exercise to be more efficient for reducing sleepiness in 32 males with OSA than CPAP alone [195]. They showed that a 2-mo exercise program associated with CPAP treatment has a positive impact on subjective daytime sleepiness, quality of life (physical functioning and general health perception), and mood state (tension and fatigue) in OSA patients. It is generally acknowledged that OSA causes inflammation, with elevated levels of C-reactive protein and tumor necrosis factor alpha and interleukin-6 which are associated with sleepiness and fatigue [196]. Physical exercise and CPAP improves inflammatory profiles and possibly excessive daytime sleepiness due to its improvement of inflammatory profiles.

Does exercise impact on sleep apnea?

Here also the relationship with obesity, cardiovascular function, inflammatory state and sleepiness makes it difficult to understand how exercise may play a role in preventing or improving sleep apnea syndrome. However several recent studies have insisted on the beneficial impact of exercise on sleep apnea. It seems that combining both exercise and weight loss with CPAP may provide the most effective treatment for many patients with OSA.

In an epidemiological setting, Awad et al. recorded the exercise habits of 1521 adults of the Wisconsin sleep cohort between 1998 and 2000 [197]. The study demonstrated that exercise was associated with a reduced incidence of sleep-disordered breathing. Controlling for BMI did not explain all the exercise-sleep disordered breathing associations and there remained a non-significant trend in reduced incidence of sleep disordered breathing. This finding suggests that exercise may also affect sleep-disordered breathing via pathways other than weight loss.

From a clinical point of view, two recent studies have shown that exercise may improve the severity of OSA by up to 50% of the AHI, independently of the effect of weight loss [198,199], but this improvement in AHI is not always found to be significant [200]. Kline et al. showed in 42 subjects a significant independent weight loss effect of chronic exercise on AHI. They observed that while only 25% of individuals who completed the exercise-training program experienced treatment success (i.e., post-intervention AHI < 20 and reduction $\geq 50\%$), 63% experienced an AHI reduction $\geq 20\%$. Thus, when evaluated as a stand-alone treatment for OSA, the efficacy of exercise training seems to be lower than with CPAP or multilevel surgery, but similar to other surgical treatments and approximately equivalent to a 10% reduction in body weight. The mechanisms of action remain unclear. Exercise clearly decreases sleepiness and improves daytime functioning in patients with OSA. Kline et al. observed this in 43 sedentary adults with OSA randomized to 12 wk of moderate-intensity aerobic and resistance exercise training or a low-intensity stretching control treatment [199]. Sleepiness and functional impairment due to sleepiness were also improved, in the exercise versus control groups, to a similar degree in terms of effect sizes ($d > 0.5$), though these changes were not significant. More work has certainly to be done on inflammatory pathways in order

to better identify how exercise improves OSA independently of weight reduction.

Along with insomnia and OSA, other sleep disorders such as restless leg syndrome (RLS) or hypersomnia may also have consequences on sleep and consequently on athletic performance. RLS is defined according to the ICSD-2 as [165] an urge to move the limbs that is usually associated with paresthesias or dysesthesias [201] symptoms that start or become worse with rest [202] and who's symptoms are at least partially relieved by physical activity, and [202] who's symptoms worsen in the evening or at night. RLS frequently also has a primary motor symptom that is characterized by the occurrence of periodic leg movements during sleep. To our knowledge only one short letter has been published, stating that in Brazil 13% of 61 marathon runners had RLS compared to a prevalence of 7% in the general population [201]. Several studies have shown that aerobic exercise is efficient in improving RLS in patients with uremic RLS [202,203].

What exercise to recommend to sleep-disorder patients?

As described previously, exercise has been recommended as a non-pharmacological treatment for insomnia [5,22,36,184,185] or sleep-disordered breathing [197]. However, physical activity needs to be progressive in patients suffering from sleep disorders, starting at low intensity and preceded by a physical/cardiovascular check-up. Indeed, growing epidemiological evidence indicates that short duration sleepers and those with sleep disorders are at greater risk of sudden cardiac death [204], coronary heart disease, myocardial infarction, angina, stroke [205] or diabetes [206].

Every heart disease is a risk factor for exercise-induced acute cardiac events, during the exercise itself or the immediate recovery period [207], so we need to be cautious when recommending acute intense exercise to subjects with sleep disorders.

Indeed, short periods of sleep due to restriction or deprivation are a sufficient stress to induce increased blood pressure [208], increased coronary vascular tonus [209], coronary calcifications [210], increased sympathetic activity [211,212], systemic inflammation (interleukin-6, e-selectin) [211,212] and endothelial dysfunction [213,214]. Moreover, Sekine [145] et al. showed in healthy subjects that just one night of sleep restriction (50%) resulted in a decrease of coronary flow velocity reserve, a sign of coronary endothelial dysfunction. An alteration of endothelial function has also been described after three nights of sleep restriction in nurses [146] or just one night of total sleep deprivation [56] in healthy subjects.

Endothelial dysfunction, an early predictive indicator of cardiovascular disease, is characterized by a decrease in endothelial-dependent NO production [215], which plays a protective role for the vascular bed, preventing the abnormal coronary artery vasoconstriction that leads to heart stroke [216]. Endothelial dysfunction is thus directly associated with exercise-induced myocardial ischemia [217]. Indeed, the increased myocardial metabolic demand and tachycardia [217] is only observed during exercise with the respect of an intact endothelial-dependent vasodilatation mechanism.

So, we can hypothesize that patients suffering from sleep disorders are probably at risk for exercise-induced myocardial ischemia or coronary heart failure and acute coronary events. We therefore emphasize the importance of cardiologic medical screening before starting a physical activity program with subjects with poor sleep or sleep disorders [218]. Moreover, physical exercise must be progressive and initially moderate, in order to improve endothelial function [219] and decrease the risk of death [220]. Indeed, aerobic exercise appears to be more beneficial than resistance exercise for improving endothelial function [221]. Moderate

aerobic exercise training is considered as sufficient to decrease oxidative stress, inflammation, insulin resistance and to prevent the risk of heart attack in patients with coronary spastic angina [222]. Based on the study by Kemi et al. in rats, which showed that moderate and high exercise training have the same beneficial effect on endothelial function [223] we strongly recommend moderate aerobic exercise training in subjects suffering from sleep disorders.

Conclusion

Sleep and exercise influence each other through complex, reciprocal interactions including multiple physiological and psychological pathways. Following practical recommendations, moderate aerobic exercise training could be prescribed as a pertinent non-pharmacological treatment of sleep disorders. New fundamental research must be carried out to improve our knowledge of the complex physiological effects and to understand the benefit of exercise in the promotion of sleep in both healthy subjects and patients. Sleep is also often mismanaged in sportsmen, with pejorative consequences on cognitive performance, effort perception and exercise-induced diseases. Better understanding of the physiological responses to sleep loss is needed to enhance physical performance.

Practice points

1. Regular moderate-intensity aerobic exercise is recommended in the treatment or the prevention of sleep disorders.
2. Exercising before bedtime may not necessarily disturb sleep in good sleepers.
3. Beneficial effects of regular exercise on sleep may be explained by multiple pathways with the interaction of circadian rhythm, metabolic, immune, thermoregulatory, vascular, mood and endocrine effects.
4. Except for its psychological patterns, the effects of sleep loss on exercise performance appear inconclusive.
5. Sleep loss must be considered as a risk factor for exercise-related injuries
6. Sleep quantity and quality must be studied in athletes using easy and inexpensive methods such as a sleep questionnaire, actigraphy or ambulatory polysomnography.

Research agenda

In the future, we need to:

1. Assess more precisely how exercise impacts on metabolic functions during sleep.
2. Have more standardized exercise protocols for populations with sleep disorders, insomnia and sleep apnea (with objective measurements).
3. Evaluate the effects of acute and intense exercise in sleep-deprived subjects.
4. Understand the anti-inflammatory effects of physical training on immuno-inflammatory responses induced by sleep deprivation and/or sleep restriction.

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