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Review

The Somnolent Youth-Sleep and the Influence of Exercise: A Narrative Review

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Abstract: Sleep is crucial to the physiological and psychological development of youth. The implications of insufficient sleep on learning and school performance are well documented. However, adequate sleep often goes unmet due to a combination of biological, societal, and environmental factors, especially during adolescence. In the present review, the changes to adolescent sleep patterns, and its consequences on cognition and learning are briefly explored. We then review the empirical literature of the role of exercise in regulating adolescent sleep patterns, and its potential mechanisms.

Keywords: adolescence; children; sleep; exercise

1. Introduction

The function of sleep is multifaceted, and complex. Research on the importance of sleep, and consequences of sleep loss, show its significance on various somatic, cognitive and psychosocial outcomes. Curcio and colleagues [1] aptly define sleep as an active, repetitive and reversible behavior serving several different functions, such as repair and growth, learning or memory consolidation, and restorative processes. Based on this all-pervading definition, sleep alone could have significant implications on school performance and health amongst adolescents (In this review we operationally define “adolescent” as an individual with a chronological age between 10 [2] and 20 [3] years of age). However, evidence shows that adolescents have poor sleep quality and later sleep times, with Asian

adolescents leading in this global trend [4]. Interventions to address this would have significant implications on adolescent quality of life and performance. Addressing these needs may be a novel and easily implementable means to dampen the perennial concerns of rising health care costs [5] and holistic health outcomes [6] at a population level. Although interventions such as light therapy are suggested as a means to cause an advancement in the adolescent circadian rhythm [7] to enable earlier sleep schedules and counter the effects of early school start times [8], such interventions have financial requirements that schools and caregivers may not be able to meet. Pharmacological interventions, on the other hand, may have dose-dependent, unwanted side-effects [9]. The need for a cost-effective and less-problematic means to address the poor sleep quality during adolescence is apparent. Although exercise has been widely promoted to improve a multitude of health outcomes in adolescents [10], its complimentary effects on sleep have not yet been given as much advocacy.

While there has been several reviews on the effects of sleep on academic and cognitive outcomes in adolescents [8,11–13], the need for exercise and physical activity amongst adolescents [10,14–16], and the influence of exercise on sleep amongst adults [17–19], there has not been a review integrating these topics, specifically looking into the impact of sleep loss, and the sleep promoting effects of exercise in adolescents.

2. Background on Sleep

Sleep is classified into several distinct stages [20]. Stages 1–4 are classified as non-rapid eye movement (NREM) sleep, and a fifth stage classified as rapid eye movement (REM) sleep. In healthy individuals, sleep is entered through NREM sleep [21]. These stages provide a numerical indication of the natural progression in which they typically occur from the start of sleep onset, which are paralleled by increasing arousal limits. The NREM sleep stages have distinct brain electrical activity and are also associated with low muscle tone and psychological activity [21]. Conversely, REM sleep is most distinguishable by obvious movements of the eye along with brain electrical activity that have quicker rhythms and lower amplitude, resembling a state of wakefulness [22]. REM sleep is sometimes termed as active or paradoxical sleep due to this resemblance to wakefulness, despite the loss of muscle tone [23].

The organization of human sleep–wake patterns is governed by circadian and homeostatic processes. The circadian processes are governed by the “master clock” in the suprachiasmatic nucleus within the hypothalamus of the brain. This pacemaker provides coordinated oscillations and specific alignments of events (circadian rhythms) over the course of a day (24 h). This process promotes wakefulness and opposes sleep pressure brought about by the homeostatic process [24]. This homeostatic process causes an ever increasing pressure to sleep during periods of wakefulness and dissipates during bouts of sleep [25]. In adolescents, both these processes undergo key changes largely due to puberty and brain maturation exaggerating a delayed sleep phase preference and chronotype [26–28].

3. Adolescent Sleep Requirements and the Role of Sleep on Learning and Cognition

Sleep experts propose that school-aged children obtain 10 to 11 h of sleep while adolescents have recommendations of 8.5 to 9.5 h [29]. These recommendations are largely unmet [4], with most obtaining roughly 7.5 h of sleep per night [30], suggesting a worldwide epidemic of sleep-deprived youth. When sleep loss occurs chronically, sleep debt starts to accrue, and is theorized to cause cumulative waking

deficits [31]. Amongst adults, there is strong evidence showing a linear decrease in neurocognition following partial sleep restriction [32], with restriction of sleep to 6 h per night for two weeks showing performance deficits similar to two nights of total sleep deprivation (SD) [33]. Due to the increased tendency for adolescents to have insufficient sleep, it is likely that even marginal increases in sleep durations would result in improved performance outcomes.

Adolescent Sleep Patterns and Learning Outcomes

The problem of shortened sleep durations in adolescence exists worldwide. However, the issue is more pronounced amongst Asian children and adolescents [4,30]. Worldwide meta-analyses on sleep patterns of children and adolescents consistently show significantly shorter sleep durations and later bedtimes amongst Asian adolescents, along with greater rates of daytime sleepiness, due to academic commitments when compared to their American and European counterparts [4,30]. Although the negative influence academic pressure has on sleep quality is also corroborated in North America, some researchers suggested this to be more prevalent amongst Asian students due to the cultural pressure to excel academically [34–37].

Reductions in optimal sleep durations reduce the necessary overnight brain activity required for complex cognitive tasks [11] including memory acquisition, consolidation and integration [38,39], creativity [40], abstract thinking [1] and academic performance [41]. Real-time brain imaging techniques confirm that sleep plays a crucial role in motor memory learning and consequently in improving motor performance [42]. However, adolescents are exposed to insufficient or poor quality of sleep due to their characteristically late bedtimes and school demands. This consequently causes impaired executive functioning due to the dysfunction of the prefrontal cortex, which is particularly sensitive to sleep loss [43]. Sleep is also known to enhance performance following learning, while disrupted REM and Stage 2 sleep can cause a decline in these performance gains [44,45]. As REM and Stage 2 sleep predominate the final third of nocturnal sleep [21], shortened sleep durations are likely to cause sleep loss in these specific sleep stages and consequently impact cognitive functioning [46].

In an investigation to examine the effects of a single bout of partial SD (5 h in bed) on cognitive functioning in children and adolescents, impairments were noted in aspects of higher cognitive functioning such as verbal creativity and abstract thinking [47]. Additionally, Sadeh *et al.* [48] examined the effects of modest SD and extension on measures of children's neurobehavioral functioning by either extending or restricting sleep by an hour over three nights. The children that extended their sleep by 30 min scored significantly better in their performance measures of the digit forward memory test, sustained visual attention, response inhibition, and motor speed, while deteriorations in performance were present amongst the children that restricted their sleep by the same amount of time. The magnitude of these differences presented by Sadeh and colleagues were equivalent to the loss of two years of cognitive and neurobehavioral development. Collectively, and in contrast to findings on adults [49,50], these findings show that children and adolescents may have the ability to mitigate the effects of brief bouts of partial SD, with these compensatory effects becoming absent during abstract or complex tasks such as memory encoding, working memory and long-term memory consolidation [12].

This impairment of cognitive functioning and the resultant decline in children and adolescents learning capability and academic performance were extensively reviewed in a meta-analysis by

Dewald *et al.* [11]. The analysis showed that sleep duration, quality, and daytime sleepiness had a small but significant effect on school performance, albeit to varying degrees. Daytime sleepiness had the most profound effect on school performance, followed by sleep quality and sleep duration. This finding is in concordance with the results of a study by Anderson and colleagues [51] that show associations in executive functioning and sleepiness rather than sleep durations in adolescents. This highlights that sleep duration is not the dependent factor on cognitive performance. Other investigations [4,11] reveal that daytime sleepiness was a better predictor of certain domains of performance such as mood state and grade point average. The contribution of daytime sleepiness in predicting the multiple aspects of an adolescent academic life is especially of concern as pivotal discoveries on adolescents reveal that biological brain changes may be contributing to the increase in daytime sleepiness independent of sleep durations [27,52]. When this is coupled with short sleep durations, these effects are exacerbated.

4. Issues Surrounding Adolescent Sleep Patterns

4.1. Biological Factors

The circadian processes that modulate sleep regulation in adolescents are altered around puberty, when brain maturation occurs, resulting in a delayed sleep-wake behavior during the second decade of life due to the misalignment of the processes that regulate sleep [53,54]. The average human is intrinsically aligned to daily rhythms of about 24.12 h under experimental conditions [55,56]. However, a lengthening of the circadian period is postulated to occur during adolescence, to an intrinsic circadian rhythm of 24.27 h [57,58]. Apart from this, a delayed circadian phase preference in adolescents is noted in several studies [3,59,60]. A review by Carskadon and Acebo [57] highlights this delay in sleep/wake patterns by comparing the weekday and weekend sleep patterns of adolescents, with differences between weekend bedtime and weekend rise time delay compared with weekday averages estimated to be 1.5 and 3 h, respectively. These differences are reflective of the “catch up” sleep required due to the deficit accumulated during the course of the week. Circadian phase preference, also referred to as chronotype, is a behavioral construct related to the time of day (morningness or eveningness) best suited for waking behavior [54]. The differences in adolescent chronotype are reflected in the timing of sleep, peak of cognitive abilities, personality, psychological and physical dysfunctions and well-being [61]. Studies analyzing the associations between pubertal development and chronotype show that more mature adolescents, especially girls, have a later phase preference [59,60], with the reversal of these changes almost predictive in determining the end of adolescence [3]. This delay in circadian phase is confirmed by delays in salivary melatonin secretion in older adolescents [62].

Another key alteration to the processes that regulate sleep in adolescents is the reduced rate of homeostatic sleep pressure during puberty. This is evident in the reduction of delta and theta electroencephalographic (EEG) power during sleep [63], as well as a reduction of slow wave sleep (SWS) during the NREM sleep across adolescence [52]. In what is probably the most extensive longitudinal study on adolescent sleep patterns, Campbell and Feinberg [27] identified a sharp decline in SWS at the commencement of adolescence, up till the age of 16. It is hypothesized that this decline is potentially due to pervasive changes due to the brain maturation processes [64,65]. The authors suggest that the homeostatic need for sleep is dependent on the duration of wakefulness prior to the sleep period

and the intensity of waking brain activity; the reduction of brain metabolic rates during adolescence would elicit a reduced accumulation of sleep inducing substrates. Resultantly, the homeostatic sleep need accumulates slower during waking amongst adolescents causing them to further delay their bedtimes [52]. These findings were corroborated in a separate study [62], which showed that adolescents have lesser sleep tendency as compared to pre-pubertal children, as assessed by sleep onset latency, after different durations of time awake.

The biological changes in the processes that regulate sleep during adolescence were evident in an analysis of subjective sleep patterns of over 25,000 European adolescents that showed a distinct age-dependent preference for a later behavioral phase marker (chronotype) around puberty, followed by a reversal in the chronotype delay shortly after the age of 20. An identical trend was also present in a parallel study by the same researchers on adolescents living in several separate secluded geographical locations that were sociologically distinct from each other, suggesting that this phase delay was independent of technological, social and cultural factors, and was largely due to biological changes during adolescence [3]. These findings led to the postulation that the reversal of chronotype patterns may in fact be a biological marker indicating the end of adolescence.

4.2. Environmental Factors

Apart from the biological issues that affect adolescent sleep durations, several other influences also play contributory roles. It is well documented that adolescents and children spend a large amount of time on technology [66–69]. A comprehensive review by Cain and Gradisar [70] to ascertain the effects of common forms of technology found that television watching, computer usage and electronic gaming have significant consequences to the sleep of children and adolescents. It is suggested to affect adolescent sleep by displacing good sleep hygiene practices, increasing physiological arousal and anxiety, and delaying circadian rhythms by suppressing melatonin secretion [71–73]. Mobile phone usage prior to sleep is shown to potentially modify sleep architecture [74,75]. Social opportunities and peer group influences are also touted to have an effect on adolescent sleep patterns [76], as the pressure to develop relationships during this phase of life is of particular pertinence [77]. The prevalent use of the Internet and online social media during the night may cause delays to bedtimes and shorten sleep durations [78,79]. Recently, more attention has also been given to the impact of stimulant use, in particular caffeine and energy drinks, on adolescent sleep patterns [80–82]. The current prevalence of the overconsumption of caffeine-containing food products amongst adolescents is high [83]. It is generally appealing to this population due to its ability to help cope with the daytime sleepiness ubiquitous during the second decade of life [84]. Caffeine intake, is known to be associated with sleep difficulties, interruptions in the normal sleep cycles, and longer sleep onset latencies amongst adolescents [85,86], thereby potentially causing a vicious cycle in which the consumption of caffeine prevents the adolescent from sleeping at night, leading to elevated caffeine consumption during the day as a result of the consequent daytime sleepiness.

Another significant influencing factor to the shortening of adolescent sleep on school days is the early school start time. As adolescents are known to have later bedtimes, the need to wake early for school often results in truncated sleep and resultant daytime sleepiness [87]. Over time, this results in chronic sleep debt on weekdays [88]. To compensate for this sleep loss over the course of a school week,

adolescents have the tendency to display catch-up sleep on weekends [88], which is associated with behavioral and emotional problems [89]. Several research groups have investigated the impact of modest delays in school start times on adolescent school performance [90,91]. In a pioneering longitudinal study involving nearly 18,000 North American adolescents, schools attempted to cater to the biological sleep patterns of adolescents by delaying high school start times by slightly more than an hour [92]. Researchers tracked various outcomes of school performance over six years (three years prior and three years following the changes) and reported improvements in punctuality, attendance, alertness, as well as academic performance and graduation rates. These corroborate more findings from another longitudinal study with over 9000 students [93]. In addition to the increase in nocturnal sleep duration and improvements in academic outcomes on standardized tests, a later school-start time was also reported to be associated with reduced substance abuse, depressive symptoms, and risk of being involved in automobile accidents. It is clear that this modification of daily schedules to accommodate the biologically natural propensity for a later sleep-wake schedule, without changing the duration of a school day, allows students to obtain more sleep during school nights, which have direct positive outcomes on several aspects of school performance.

5. Effects of Exercise on Sleep

The notion that participating in exercise improves an individual's sleep quality has persisted well before the development of modern day sleep science [94]. Exercise is noted as a potential non-pharmacological means of improving adult sleep quality by the American Academy of Sleep Medicine [95]. The shared belief is evident in laypeople and sleep experts alike [96]. Epidemiological studies also consistently show that exercise is associated with improved sleep quality [97–100]. Additionally, several studies account that partaking in regular physical activity is associated with a reduced risk of sleep disturbances and better sleep quality in adults [101]. The body of evidence suggests that vigorous exercise increases total sleep times by reducing sleep onset latency (SOL) and waketime after sleep onset (WASO) [18,102,103]. Separately, chronobiological research shows that exercise can facilitate a phase advance in daily circadian entrainment in adult populations [104,105] due to its role as a non-photic time cue [106]. This is of particular relevance to adolescents due to changes toward an evening circadian phase preference as well as a later circadian phase shift caused by the lengthened intrinsic period of the adolescent circadian clock; that is, a longer internal day length [107]. The rest of this section provides a more detailed review of the multifaceted relationship between sleep and exercise amongst adolescents.

The research on the acute effects of exercise on adolescent sleep is not new. Its effect on sleep quality was experimentally highlighted in 1966 on college students [108]. Using three conditions to compare the effects of afternoon, evening, and no exercise routines on sleep architecture, it was reported that the afternoon exercise condition elicited significantly greater proportions of SWS when compared to the no exercise and evening exercise condition. Notably, the evening exercise condition also caused greater sleep disturbances in the manner of wakefulness. These deleterious effects of nighttime exercise were corroborated in a recent, highly-controlled, study on mature adolescents [109], showing no increases in SWS following evening exercise, but a longer sleep onset due to higher states of physiological arousal following intense exercise prior to nighttime sleep, and a thereby significantly shortening of total sleep

time. However, a separate quasi-experimental study on mature adolescents investigating the effects of sport/exercise participation prior to bedtime reported that higher self-perceived exercise exertion before bedtime elicited better sleep efficiency, more SWS, shorter sleep onset times, and shorter waketimes after sleep onset [110]. Closer analysis into these two studies revealed key differences in the inclusion criteria of the participants, with Oda and Shirakawa [109] opting for participants with potentially lower levels of fitness and with irregular exercise routines, while Brand and colleagues [110] recruited individuals that regularly partook in vigorous exercise/sport at least three times a week in the last 12 months. Taken together, these findings provide evidence that the intensity and timing of an acute bout of exercise, as well as the fitness levels of the individual, need to be accounted for as they can cause very different outcomes to adolescent sleep architecture.

In order to assess the mechanisms in which exercise influenced sleep, Dworak *et al.* [111] administered various exercise and SD conditions on rats and found that only highly intense exercise elicited significant global increases in adenosine concentrations in the rat brain, with no changes noted in the moderate exercise condition. In contrast, sleep caused adenosine levels to progressively decline while increasing brain adenosine triphosphate and adenosine diphosphate. In their follow up study, Dworak and colleagues sought to examine the effects of an acute bout high-intensity exercise on adolescent sleep patterns [112]. Polysomnography (PSG) sleep patterns were compared between a moderate exercise (65%–70% at maximal heart rate for 30 min) and high-intensity exercise (three sets at 85%–90% at maximal heart rate for 10 min each) condition on a cycle ergometer in the evening. It was noted that only the high-intensity exercise elicited significant changes in sleep architecture as shown by elevated SWS, reduced stage 2 sleep, increased sleep efficiency and shortened sleep onset latency due to the increased homeostatic sleep pressure accrued due to exercise. These findings were found to be homologous with findings in young adults following intense exercise [113], but contrary to the effects of intense exercise on adults [114]. The findings by Dworak *et al.* [112] are especially salient as it was the first to experimentally report the benefits of exercise on adolescent sleep patterns, with high-intensity exercise being more relevant for this population.

The increase in SWS is characteristic of an increased homeostatic pressure to sleep [115], or potentially, a higher level of fitness [116]. There is evidence that chronic participation in exercise, and consequent fitness improvements, can induce improvements to adolescent sleep patterns. Shapiro and colleagues reported increased SWS sleep following improvements in fitness in a study examining the changing sleep patterns of army recruits during their 18-week basic training [117]. Laboratory PSG and fitness tests to determine maximal oxygen uptake were obtained at the start, middle and end of the military program. The authors noted continued improvements in anaerobic threshold during the monitoring period. However, increases in SWS were most pronounced in the first nine weeks (start to middle), with this proportionate increase in SWS plateauing till the end of the training program. The authors were careful to state that due to the naturalistic nature of the study, it was not possible to truly ascertain if it were the improvements in fitness, or exposure to specific modalities of exercise that resulted in the changes in sleep architecture. These findings should come with a caveat in that the increases in SWS in this study could also have been attributed to a state of partial sleep restriction [118], often experienced in military training programs [119].

The changes to adolescent sleep following participation in long-term exercise/training is also evident in observational studies on European adolescents with high exercise levels, and is a factor in improving

sleep quality. This was highlighted in a series of studies by Brand and colleagues comparing the sleep patterns of adolescents engaged in higher amounts of exercise against those with lower levels of activity [120–123]. The group first highlighted better subjective sleep quality and stability between weekday/weekend sleep schedules amongst male footballers that were known to participate in chronic intense training [122]. In a follow-up study, the authors reported better objective sleep efficiency as a result of shorter durations of waketime after sleep onset and shorter sleep onset latency periods, as measured through PSG, amongst adolescent footballers when compared to non-athletic age-matched controls that took part in significantly lesser durations of vigorous exercise (12.69 vs. 1.56 h per week) [120]. Importantly, these values were seen on a day without exercise training, highlighting the prolonged effects of exercise on adolescent sleep patterns. These potential persisting effects of chronic exercise were also reported in their analysis involving high and low exercisers (8.5 vs. 2 h per week) [121]. In this investigation, high-exercising adolescents were found to have greater proportions of SWS, lesser REM sleep, and better sleep efficiency when compared to relatively inactive adolescents. Similar to the previous study, the PSG sleep measures were obtained on a day without exercise. In a larger scale ($n = 434$) subjective examination on the subjective quality of sleep of high-level adolescent athletes and age-matched controls (17.7 vs. 4.7 h of exercise per week), non-athletic adolescents had lower sleep quality, with males having a greater risk of poor psychological functioning [123]. In their latest study [124], the researchers examined the effects of self-reported, and objectively measured amounts of physical activity on subjective and objective sleep indices in adolescents and young adults. Their data were somewhat equivocal, in that, greater physical activity (self-reported and objective) was associated with better sleep outcomes, despite females reporting to have spent less time in vigorous physical activity but having greater sleep efficiency than their male counterparts. In this study, moderate-to-vigorous physical activity only accounted for 11 min per day, which could explain the ambiguous findings, and highlights the potential need for greater durations and higher intensities of exercise to elicit positive changes in adolescent sleep patterns. In agreement with the previous findings in studies on adolescent athletes by Brand and colleagues, Suppiah and colleagues [125] identified that high-level adolescent athletes who participated in the higher intensity sport of badminton (more time in 61%–90% at maximal heart rate zone) have less light sleep and WASO, and more deep sleep when compared to high-level adolescent athletes who partook in the lower intensity sport of bowling. These comparative studies between low and high exercising adolescents seemingly add weight to the dose dependent relationship between exercise and adolescent sleep changes.

Despite the original hypothesis that adenosine increases only occur following vigorous exercise, similar noticeable changes were observed in adolescents that participated in moderate-intensity morning running for three weeks [126]. In their study, Kalak and colleagues examined the impact of a short-term (three week) morning exercise intervention on adolescent sleep patterns and psychological functioning. Relatively inactive adolescent participants (2.2 h of moderate physical activity per week on average) were randomly assigned to either an exercise group that participated in self-paced running exercise every morning of the five school days per week or, a non-participative control group that remained on the side-lines of the running track when the exercise intervention was conducted. PSG sleep stage readings were conducted prior to the start and immediately after the end of the exercise intervention. After the intervention, the running group reported better sleep quality in that their SWS increased, sleep onset latency decreased, longer REM sleep latency, and increased total sleep time. Improvements in subjective

sleep data were reported in both groups, with the difference being statistically significant in the running group. In contrast to the expected increase in SWS and reduction of REM sleep following acute or chronic exercise [103], a more recent study found no changes in SWS and increased REM sleep following a three month exercise intervention on obese adolescents [127]. In their study, Mendelson and colleagues reported that a 12-week exercise program (between 160 and 180 min per week) consisting of aerobic and resistance training, was effective in improving total sleep time by reducing time awake after sleep onset, without affecting sleep stage characteristics. The lack of change in SWS proportions was attributed to aerobic training that was possibly inadequately intense. However, as the exercise intervention used was mixed, these results could also be attributed to the involvement of resistance training exercise, which has been reported to not alter sleep stage measures in adults [128,129].

While the exact changes to sleep stage characteristics following exercise is still equivocal in adolescents, its ability to reduce sleep onset latency, waketime following sleep onset, improve sleep efficiency and total sleep time is fairly ubiquitous. In contrast to research on adults [19], a minimal intensity threshold may exist in order for exercise to elicit an effect on adolescent sleep stage characteristics. This may be in part due to the slower accumulation of sleep pressure amongst adolescents [63], with only high-intensity exercise resulting in changes to SWS proportions due to depletions in brain energy stores [111].

5.1. Explanations for the Exercise–Sleep Quality Nexus

Several hypotheses explain the link between exercise and sleep quality [111,130–132]. The evidence suggests that exercise plays a role in modifying the homeostatic sleep regulative process (Process S), or acts as a non-photic Zeitgeber (time giver) that can phase shift normal circadian rhythms (Process C). While the evidence that exercise influences sleep is fairly convincing [18,102,103,116,131–133], the mechanisms explaining these changes are still ambiguous.

5.1.1. Body Restitution and Energy Conservation

The earliest theory explaining the changes in sleep patterns is that exercise causes sleep to be deeper and extended for longer durations in order for the tissue restitution [134] and energy conservation [135]. The body restitution theory appears viable due to observations of increased SWS following exercise bouts, which caused a concomitant increase in growth hormone production [136]. This compensatory theory suggests that a highly catabolic activity during the wakefulness would result in an increased anabolic state during the following sleep periods. However, later studies failed to corroborate the findings of Adamson *et al.*, in reporting increases in growth hormone production during sleep following exercise. The energy conservation theory posits that sleep is a state of enforced rest so as to limit activity and energy expenditure. This theory seems to lend support to studies finding longer durations of SWS in physically fit participants and individuals that participated in high volumes of aerobic exercise [112,137–139]. However, studies on ultra-long distance exercise cast doubts on the validity of the theory [114,140,141]. While some researchers acknowledge these theories [138,142], their validity are still largely speculative.

5.1.2. Thermoregulation

Core body temperature disruptions are an alternate hypothesis used to explain the increases in SWS following exercise. This is based upon evidence of a reduction in core body temperature and increased peripheral skin blood flow at sleep onset [143,144]. The hypothesis that exercise influences sleep through thermoregulation originated from evidence showing that the anterior hypothalamus/preoptic area plays a vital role in both sleep and body temperature regulation [145]. It is suggested that exercise accelerates the normal rate of decline of body temperature prior to sleep onset, leading to improved sleep quality and deeper sleep [146]. However, this hypothesis is challenged by Flausino and colleagues [101]; in their study, they examined the influence varying exercise intensities and durations 2–3 h before bedtime on sleep patterns and body temperature amongst good sleepers. The exercise conditions caused significant improvements in sleep efficiency and lowered WASO despite there being no differences in core temperature after exercise. While the thermoregulatory theory is frequently mentioned as the underlying mechanism explaining the exercise-sleep relationship, its inconsistent outcomes casts doubts on its tenability.

5.1.3. Brain Energy Hypothesis—Adenosine

A relatively recent hypothesis suggests that adenosine, a product of cerebral energy consumption and sleep-promoting nucleotide in the central nervous system, may play a role in influencing sleep following exercise. The premise of this theory stemmed from observations of the cerebral contributions and modifications that occur during exercise [147]. Additionally, SD research show that prolonged SD causes a greater homeostatic sleep drive, along with increased levels of extracellular adenosine. The increased homeostatic sleep drive is reflected through an increase of delta power during SWS [148].

5.2. Potential Antagonizing Effects of Overly-Intense Exercise

While exercise seems to have beneficial effects on promoting sleep quality, there is also speculation about the existence of an optimal threshold level of physical training beyond which sleep is disrupted [102,114]. The findings that exercise improves sleep quality are inconsistent with studies by Wong *et al.* [149] and Awad *et al.* [150] that report an antagonizing effect of exercise on sleep. In an attempt to confirm the hypothesis that vigorous intensity exercise would elicit changes to sleep patterns in adults, participants were randomly allocated to treadmill exercises at 45%, 55%, 65% and 75% of their maximal oxygen uptake (VO_{2max}), a no-exercise day in a crossover manner with a two day washout period between testing sessions. Apart from increasing the durations of Stages 1 and 2 sleep after the 65% and 75% exercise condition, no other changes were reported [149]. Wong and colleagues claim that these findings resembled those of Awad *et al.* [150] who found that higher levels of physical activity were associated with increases in light sleep and sleep disruptions. Both groups suggested that exercise was a stressor, which influenced sleep negatively. Studies on ultra-long distance exercise also cast doubts on the validity of the positive effects of exercise on sleep [113,114,140,141]. Driver *et al.* [141] observed sleep disruptions in the form of increased wake-time after sleep onset and no change in SWS following an ultra-triathlon when compared to nights following no-exercise, a 15 km run and a 42 km run. Additionally, Montgomery and colleagues [114] noted decreases in SWS following a 42.2 km marathon.

In a study on aerobically fit males [140], it was demonstrated that excessive exercise training loads potentially caused a somatic stress response due to activation of the hypothalamic-pituitary-adrenal (HPA) axis. Resultantly, the heightened response of the HPA axis from baseline measures was posited to cause sleep disruption in the form of shortened sleep time and SWS [151]. Based on these findings, it is possible that exercise has an inverted-U relationship on sleep characteristics, in which sleep becomes disrupted when exercise intensity and duration exceeds an individual's ability. Similar symptoms of sleep disruptions have been noted in athletes suffering from overtraining syndrome [152]. More research is required to confirm this possibility.

6. Conclusions

Sleep is well established as an integral part of physical growth and cognitive functioning. However, adolescents are known to have a tendency toward poor sleep quality and later sleep times as a result of maturational, academic and social factors. This results in late bedtimes, difficulties during sleep onset, nocturnal awakenings, difficulties waking in the morning and daytime sleepiness. In the absence of pharmacological or behavioral interventions, exercise has potential benefits on promoting sleep quality in this population. However, ambiguity still exists in its underlying mechanisms that affect sleep, and if an ideal exercise load exists that could better facilitate sleep in adolescents. The body of evidence also suggests that exercise has a positive effect on adolescent sleep. There also exists strong evidence of its direct benefits on the cognition of children and adults. It is still unclear if the impact of exercise on learning may be mediated by the impact of exercise on sleep. Considering the neural changes during adolescents that predispose them to higher levels of daytime sleepiness and its effects on academic performance, it would be of interest for future research to understand the specific effects of exercise on cognition, and the degree in which sleep potentially mediates cognition in the relationship. Further highly-controlled experimental research is warranted to definitively demonstrate this causality and to provide a more nuanced understanding of the optimal exercise threshold that can positively impact adolescent sleep.

Author Contributions

HTS made substantial contributions to overall conception, drafting and critically revising the manuscript, and approved of the final version to be published. MC made substantial contributions to drafting and critically revising the manuscript.

Conflicts of Interest

The authors declare no conflict of interest.

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