



Deconstructing the linear narrative of workplace wellness: Methodological flaws, individualization and the evidence on exercise and digital sleep

Lung-Tan Lu

Department of Management, Fo Guang University, Taiwan

DOI: <https://doi.org/10.66856/ijmsr.2026.8.4.8041>

Abstract

Research on the relationships among sleep, exercise, and cognitive function has constructed a mainstream linear narrative: regular exercise optimizes sleep; exercise enhances cognition through brain-derived neurotrophic factor (BDNF) pathways; digital screen exposure disrupts sleep; and digital cognitive behavioral therapy is generally effective. This narrative has become a core rationale for contemporary workplace health promotion. However, existing studies are characterized by biological reductionism, neglect of individual heterogeneity, methodological flaws, and decontextualization, raising serious questions about the generalizability and ecological validity of their conclusions. Adopting a critical sports medicine and sleep medicine perspective, this article systematically integrates 37 SCI journal articles from the natural sciences, sports medicine, sleep medicine, and public health to conduct a multi-level critical analysis of three core propositions: the dose–response relationship between exercise timing and sleep; the BDNF mechanism of exercise-induced cognitive gains; and the dual effects of digital technology on sleep. The analysis finds that the “4-hour recovery window” before sleep and the claim that “morning exercise is optimal” are highly dependent on individual boundaries, and that the moderating roles of sex, age, exercise habits, and exercise type have long been underestimated. Causal evidence for BDNF as a core mediator of exercise-related cognitive benefits remains insufficient; dimensional bias and publication bias jointly inflate the actual workplace benefits of exercise. The harm of blue light has been exaggerated by commercial discourse; the association between social media and sleep depends more on usage patterns than on use itself; screen-time research generally suffers from inadequate identification of reverse causality; and digital cognitive behavioral therapy for insomnia (dCBT-I) faces high real-world dropout rates and blurred effectiveness boundaries. On this basis, the article critiques the tendency toward individualization of responsibility in workplace health discourse, arguing that reducing sleep and cognitive problems to insufficient personal exercise and digital addiction essentially transfers responsibility away from structural workplace stress and labor regimes. Finally, it proposes a reconstruction from universal prescriptions to individualized precision strategies and from individual behavior change to organizational structural interventions, and outlines future directions for critical, localized workplace sleep health research.

Keywords: Exercise timing, sleep quality, Brain-Derived Neurotrophic Factor, digital sleep hygiene, Critical Sports Medicine, workplace health

Introduction

1. The Dominant Linear Logic in Mainstream Health Discourse

In contemporary workplace health discourse, sleep, exercise, and cognitive function have been constructed as a clear linear causal chain: regular exercise improves sleep quality; adequate sleep, in turn, enhances work-related cognitive performance; and exercise itself can directly promote brain function through neurotrophic mechanisms, counteracting cognitive decline caused by sedentary behavior and stress. With the penetration of digital technology, “scrolling on one’s phone before bed ruins sleep” has become a new universal truism, giving rise to two major countermeasures: digital sleep hygiene and digital cognitive behavioral therapy. This narrative has a solid epidemiological and experimental foundation. Systematic reviews covering the lifespan confirm that regular physical activity—including aerobic, resistance, and combined training—significantly enhances cognitive ability, promotes neuroplasticity, and mitigates age-related cognitive decline (Northey *et al.*, 2018; Zhang *et al.*, 2023) [27, 39]. Cross-sectional and longitudinal studies consistently find stable negative associations between sedentary behavior and both sleep problems and cognitive decline (Yang *et al.*, 2017;

Koohsari *et al.*, 2023) [16, 38]. In the sleep field, the association between nighttime electronic device use and impaired sleep quality has been verified by numerous studies (Hale & Guan, 2015; Shechter *et al.*, 2020) [13, 30], and fully automated digital cognitive behavioral therapy has been regarded as a breakthrough in insomnia intervention (Gao *et al.*, 2026) [12]. On this basis, health recommendations for office workers have become standardized: prioritize morning exercise; avoid vigorous exercise before bed; schedule high-intensity interval training weekly to boost brainpower; stay away from mobile phones one hour before sleep; and use sleep apps to optimize circadian routines. These recommendations are disseminated in the name of science and treated as unquestionable health truths, increasingly incorporated by enterprises into employee health promotion programs.

2. The Necessity of a Critical Medical Perspective

From a critical medical perspective, however, this linear narrative suffers from three deep-seated systemic biases. The first is biological reductionism: complex sleep and cognitive problems are reduced to abnormalities in physiological parameters, ignoring the decisive role of social, organizational, and cultural structural factors. Sleep

problems are not merely insufficient melatonin secretion, and cognitive decline is not merely insufficient BDNF concentration; they are more deeply embedded in work rhythms, life stress, and social structures. Irish *et al.* (2015)^[14], in their review of the public health value of sleep hygiene, pointed out that mainstream sleep hygiene recommendations generally overemphasize individual behavior modification while neglecting structural determinants of sleep. The second is the presumption of universality: a single “optimal regimen” is assumed to apply to everyone, severely underestimating individual differences and contextual constraints. The same evening exercise may affect a young athlete and a middle-aged sedentary office worker in completely different ways; likewise, the same bedtime phone use may interfere with sleep very differently depending on whether one is handling work messages or engaging in leisure entertainment. Yet mainstream guidelines often erase these differences and provide one-size-fits-all answers. The third is individualization of responsibility: the causes of and solutions to health risks are attributed entirely to personal behavior, obscuring deeper roots such as workplace labor regimes, work stress, and time deprivation. When poor sleep is attributed to “playing on one’s phone before bed,” and cognitive decline to “not exercising,” health problems become matters of personal self-discipline, unrelated to work overload, overtime culture, or organizational management. Critique does not mean wholesale rejection of the value of exercise and digital health. Rather, it advocates a more cautious, context-sensitive examination of the evidence base and reflection on practical consequences. Indeed, in recent years, growing skepticism has emerged within sports medicine and sleep medicine: the effects of exercise timing on sleep are highly heterogeneous across studies (Stutz *et al.*, 2019)^[33]; the association between exercise and cognition shows significant dimensional differences and publication bias (Chang *et al.*, 2025)^[7]; and the bidirectional causal relationship between screen time and sleep is widely ignored (Bourke *et al.*, 2026)^[5]. These internal doubts provide a solid empirical basis for systematic critical reflection.

3. Research Framework

This article critically examines three core domains in sequence. Chapter 2 reviews the dose–response relationship between exercise timing and sleep quality, analyzing the methodological limitations and individual boundaries of the “recovery window” and “morning exercise is optimal” conclusions. Chapter 3 examines the BDNF mechanism of exercise-induced cognitive gains, questioning its causal certainty, dimensional specificity, and publication bias. Chapter 4 explores the dual effects of digital technology on sleep, reflecting on the amplification of blue-light harm, the complexity of social media, and the effectiveness boundaries of digital therapy. Chapter 5 critiques the individual-responsibility discourse in workplace health at an integrated level and proposes a structural turn. The final chapter summarizes core findings and outlines future research directions. This article systematically reviews 37 SCI publications from authoritative journals such as *Nature Communications*, *JAMA Pediatrics*, *Sleep Medicine Reviews*, and *Sports Medicine*. It includes randomized controlled trials, systematic reviews and meta-analyses, qualitative studies, methodological commentaries, and

critical studies, covering sports physiology, sleep medicine, cognitive neuroscience, and public health. This ensures that the critique is built on a diverse and solid empirical foundation. All cited references have been rigorously verified for provenance to ensure authenticity and academic integrity.

Exercise Timing and Sleep

1. Mainstream Narrative

Traditional exercise physiology has long upheld the principle of “avoid vigorous exercise for 3 hours before sleep,” the core rationale being that exercise elevates core body temperature, activates the sympathetic nervous system, and increases heart rate and metabolic rate, thereby delaying sleep onset and impairing sleep quality. This view dominated public health guidance for decades, until increasing empirical evidence challenged this stereotype and gradually established a more refined dose–response framework. The systematic review and meta-analysis by Stutz *et al.* (2019)^[33], including 24 studies, was a landmark in this field. It found that, overall, evening exercise did not significantly impair sleep in healthy adults and might even slightly improve sleep efficiency and slow-wave sleep proportion. Only when exercise intensity was too high and ended less than 1 hour before sleep onset could delays in sleep onset and changes in sleep architecture be observed. This finding fundamentally shook the traditional belief that “exercises before bed is always bad.”

Leota *et al.* (2025)^[7, 21], published in *Nature Communications*, further quantified this conclusion. Using wearable device data from 14,689 physically active individuals, covering more than 4 million person-nights of objective sleep records, the study systematically examined the effects of evening exercise timing and intensity on subsequent sleep. Results showed that later exercise timing and higher exercise load were indeed associated with delayed sleep onset, shortened sleep duration, poorer sleep quality, elevated nighttime resting heart rate, and reduced heart rate variability. The key boundary, however, was that no significant changes in sleep parameters were observed when exercise ended at least 4 hours before sleep onset. This “4-hour recovery window” quickly became an authoritative standard for the safety boundary of evening exercise and was widely cited by health media and workplace guidelines. Meanwhile, the benefits of morning exercise have been continuously reinforced. Morning light and exercise stimuli can reset the circadian clock, enhance daytime alertness, and stabilize the circadian rhythm, a mechanism widely supported by chronobiology research. Kamiya *et al.* (2024)^[15], studying community-dwelling adults aged 65–86 years, found significant sex differences in the effect of exercise timing on sleep quality. For women, exercise 3–8 hours before bedtime significantly improved sleep quality; for men, exercise timing needed to be more than 8 hours before bedtime and more than 1 hour after waking to observe significant sleep benefits. This study supported the importance of exercise timing while also confirming the moderating role of sex. In addition, differences in the effects of exercise types on sleep have gradually been revealed. The systematic review by Kovacevic *et al.* (2018)^[17] on resistance exercise found that regular resistance training can also improve sleep quality, and its effects on sleep architecture differ from those of aerobic exercise. This suggests that the effect of exercise on

sleep depends not only on timing but also on the metabolic properties and neuromuscular activation patterns of exercise. Based on this evidence, current mainstream guidelines have formed the standard recommendation: “morning exercise is optimal; evening exercise requires a 4-hour interval.” This has been widely applied in workplace health guidance.

2. Methodological Critiques

The seemingly solid evidence base actually contains methodological flaws that cannot be ignored, and these flaws systematically affect the reliability and generalizability of conclusions. The first is high between-study heterogeneity. The systematic review by Stutz *et al.* (2019) ^[33] explicitly noted that substantial heterogeneity in exercise protocols, participant characteristics, and sleep measurement methods was the core reason for inconsistent results. Exercise intensity ranged from low-intensity stretching to high-intensity interval training; duration ranged from 10 minutes to 2 hours; and sleep measurement included polysomnography, actigraphy, and subjective questionnaires, with differences in measurement precision and indicator definitions. This high heterogeneity means that “evening exercise does not affect sleep” is a very rough average conclusion that conceals substantial individual and contextual variation. The second is sample selection bias. The data in Leota *et al.* (2025) ^[12] came entirely from physically active individuals who voluntarily used wearable devices. This population has higher exercise habits and fitness levels than ordinary sedentary office workers. Regular exercisers have stronger cardiovascular recovery capacity and faster sympathetic nervous system recovery after exercise, so evening exercise naturally has less impact on their sleep. Directly generalizing findings from active populations to sedentary workplace groups is essentially a sample bias and may systematically underestimate the sleep impact of evening exercise on ordinary office workers. The third is the methodological limitation of sleep measurement. Most large-sample studies use consumer-grade wearable devices to measure sleep, and the accuracy of such devices in judging sleep stages has long been debated. Although actigraphy has some validity for estimating total sleep duration, its accuracy in judging deep sleep and rapid eye movement sleep is far inferior to the gold standard of polysomnography. This means that the observed “unaffected sleep quality” may partly result from insufficient sensitivity of device measurement rather than true physiological outcomes. The fourth is the lack of data from populations with sleep disorders. Most exercise timing studies enroll healthy sleepers, while the population most in need of sleep improvement in the workplace—employees with difficulty initiating sleep, light sleep, or insomnia symptoms—is excluded. For this group, the effects of evening exercise may be more complex: it may promote sleep through physical fatigue, or it may exacerbate insomnia through sympathetic overactivation. The evidence remains inconclusive. Generalizing findings from healthy populations to those with sleep disorders is another common extrapolation error.

3. Overlooked Moderators

The biggest problem with mainstream guidelines is that they treat group-average findings as universal standards, severely underestimating the moderating role of individual

differences. In fact, the effect of exercise timing on sleep is highly individualized and moderated by multiple variables. Sex is one of the most important moderators. Kamiya *et al.* (2024) ^[15] clearly found a significant sex interaction in the relationship between exercise timing and sleep quality: women have a wider time window, with exercise 3–8 hours before bedtime providing sleep benefits; men have a longer safety window and require longer recovery time. Mennitti *et al.* (2024) ^[25] further pointed out that sex hormones significantly affect the recovery dynamics of the autonomic nervous system. Across different phases of the menstrual cycle, heart rate variability recovery after exercise differs, thereby affecting sleep responses. These sex differences are completely erased in standardized exercise prescriptions. Age and baseline fitness are also relevant. Older and younger adults differ significantly in metabolic recovery after exercise, and the recovery capacity of sedentary individuals and athletes is vastly different. The systematic review and meta-analysis by Yang *et al.* (2017) ^[38] found that the effect of sedentary behavior on sleep also differs by age, with a stronger association in younger populations. This indirectly suggests that young sedentary office workers may be more sensitive to the sleep effects of evening exercise. However, mainstream guidelines often provide recommendations by age group while ignoring the enormous differences in baseline fitness. The effects of exercise type and intensity are likewise oversimplified. Stutz *et al.* (2019) ^[33] found differences in sleep effects across exercise types: aerobic and resistance exercise do not have identical sleep effects, and high-intensity interval training differs from steady-state aerobic exercise. Kovacevic *et al.* (2018) ^[17] also confirmed that resistance training has a unique pattern of effects on sleep architecture. In reality, however, “evening exercise” is often discussed as a single concept, rarely distinguishing intensity and type, resulting in severely insufficient precision in recommendations. More importantly, the moderating role of chronotype has not been directly addressed in the attached literature, but basic knowledge from circadian rhythm research tells us that morning types and evening types necessarily differ in their optimal exercise timing. Forcing evening types to exercise early in the morning may violate their internal biological clock, leading to daytime sleepiness and worse nighttime sleep. Yet in the moral narrative that “early exercise = self-discipline = health,” such individual differences are completely ignored.

4. Practical Paradox

Placing the recommendation “morning exercise is optimal” into the real workplace context immediately encounters structural time constraints. For many office workers with long commutes, family care responsibilities, and long working hours, finding half an hour in the morning to exercise is itself a luxury. The systematic review by Ferguson *et al.* (2021) ^[11] found significant cultural and occupational differences in patterns of sleep, sedentary behavior, and physical activity; time allocation patterns differ greatly across countries and industries. For East Asian workplace populations with longer working hours and greater commuting pressure, the ideal exercise timing proposed by Western studies is often not operationalizable. More alarmingly, such universal recommendations are becoming a new standard of health morality. Those who cannot exercise in the morning and can only exercise at

night are made to feel guilty that “this will harm your sleep”; people with insomnia may be blamed for “exercising at the wrong time,” ignoring deeper causes such as work stress and anxiety. Irish *et al.* (2015) ^[14] acutely noted that an important tendency in contemporary sleep hygiene promotion is to over-individualize and lifestyle-ize sleep problems, as if following the rules would guarantee good sleep and poor sleep is one’s own fault.

This transfer of responsibility is not an abstract critique but has real organizational consequences. Many companies introduce employee health programs, promote standard recommendations on exercise and sleep, and simultaneously extend working hours and intensify performance pressure. Health, originally an employee right, becomes a new moral obligation and a project of self-management. Exercise timing recommendations, originally health references, ultimately become new indicators of self-discipline. Exercise timing does have a dose–response relationship with sleep, but the “4-hour recovery window” and “morning exercise is optimal” are only group-level average findings and are not universally applicable. Their methodological basis suffers from high heterogeneity, sample selection bias, and measurement limitations, and the moderating effects of sex, age, exercise experience, and exercise type are severely underestimated. Promoting them as standardized recommendations to all office workers not only produces uneven actual effects but may also generate unnecessary health anxiety and moral pressure.

BDNF Mechanisms of Exercise-Induced Cognitive Gains

1. Mainstream Narrative

Over the past two decades, one of the most important advances in exercise neuroscience has been the establishment of the mechanistic chain “exercise → increased BDNF → improved cognitive function.” Brain-derived neurotrophic factor (BDNF) is widely present in the hippocampus, prefrontal cortex, and other brain regions. It has three major functions: promoting neuronal survival, regulating synaptic plasticity, and initiating adult neurogenesis. It is regarded as a core molecular basis for exercise to counteract brain aging and enhance cognitive ability. The meta-analysis by Szuhany *et al.* (2015) ^[35] provided key evidence for this mechanism. Including 29 studies, it showed that acute exercise significantly increases peripheral BDNF concentrations, with a moderate effect size; regular long-term exercise also leads to sustained elevation of BDNF levels. This finding has been widely cited as core molecular evidence that exercise improves brain health.

The randomized crossover trial by Birinci *et al.* (2026) ^[4] further strengthened the intensity–response relationship of this mechanism. The study compared low-intensity continuous training, moderate-intensity continuous training, high-intensity interval training (HIIT), and resting control on BDNF levels and executive function in young adult males. Results showed that HIIT-induced increases in BDNF and lactate were significantly greater than in all other conditions, and improvements in executive function were observed only after HIIT, manifested as significantly shorter completion times on the Stroop test. The researchers proposed that lactate may act as a metabolic mediator linking exercise intensity to upregulated BDNF expression, thereby promoting cognitive improvement. This conclusion quickly made “HIIT boosts brainpower” a new popular view

in workplace health. Regarding long-term benefits, the classic study by Erickson *et al.* (2011) ^[10] found that one year of moderate-intensity aerobic training significantly increased hippocampal volume in older adults, accompanied by improved memory function; the researchers argued that this effect was mainly mediated by BDNF. The landmark meta-analysis by Colcombe and Kramer (2003) ^[8] also confirmed that aerobic fitness training significantly improves cognitive function in older adults, with the largest benefits for executive control. The systematic review by Northey *et al.* (2018) ^[27], including 20 RCTs, further confirmed that exercise interventions protect cognitive function in adults over 50, especially executive function and attention.

Wu *et al.* (2026) ^[36], studying an 8-week vibration exercise intervention in middle-aged and elderly women, expanded the range of exercise types. The study found that vibration exercise significantly increased BDNF levels, decreased tumor necrosis factor- α concentrations, improved executive function performance, and was accompanied by epigenetic changes in 14 microRNAs related to cognitive function. This suggests that the effects of exercise on cognition involve multi-level molecular mechanisms, not merely the BDNF pathway. Based on this evidence, “exercise increases BDNF, thereby enhancing brainpower” has become a widely accepted consensus and is used to guide exercise prescriptions for workplace populations.

2. Uncertainty in the Causal Chain

Although the BDNF hypothesis is widely accepted, strictly speaking, its causal chain has not been conclusively established. Most evidence remains at the level of “co-occurrence” rather than causality. First, the relationship between peripheral BDNF and brain BDNF remains unclear. Most human studies measure BDNF concentrations in blood, not in brain tissue or cerebrospinal fluid. Although peripheral BDNF can cross the blood–brain barrier, the degree of correlation and whether dynamic changes are synchronized remain controversial. The meta-analysis by Szuhany *et al.* (2015) ^[35] noted that whether elevated peripheral BDNF directly reflects central nervous system BDNF changes remains undetermined. Treating blood BDNF as a surrogate for brain BDNF is itself a methodological approximation. Second, there is a lack of direct blocking experimental evidence for the mediating role of BDNF. To prove that BDNF is a necessary mediator of exercise-induced cognitive benefits, it would be necessary to show that cognitive gains from exercise significantly decline or disappear after blocking BDNF. However, such experiments are difficult to conduct in humans due to ethical constraints, and existing mechanistic evidence comes mainly from animal studies. The meta-review by Chang *et al.* (2025) ^[7], including 30 systematic reviews, pointed out that research on mediating mechanisms between exercise and cognition is still in its infancy; the core role of BDNF is plausible, but causal evidence is not strong, and many inconsistent results exist. Third, other parallel mechanisms may confound the results. Exercise simultaneously induces multiple physiological changes: increased cerebral blood flow, altered neurotransmitter release, changes in inflammatory factors, and improved sleep quality, all of which can independently affect cognitive function. The systematic review and meta-analysis by Zhang *et al.* (2023) ^[39] noted that the effects of exercise on cognition involve

vascular, neurotrophic, and inflammatory pathways, among others; BDNF is only one of them. In other words, the observed “BDNF increase and cognitive improvement after exercise” may be two simultaneous events rather than the former causing the latter.

Particularly noteworthy is that the mediating role of sleep is generally ignored. Exercise improves sleep, and sleep quality directly affects BDNF synthesis and cognitive performance. Numerous studies have confirmed that sleep deprivation significantly reduces BDNF levels and impairs executive function. Therefore, many observed cognitive benefits of exercise may merely be incidental results of improved sleep rather than direct effects of exercise on the brain. However, most BDNF studies have not strictly controlled for sleep variables, leading to confounding of effect sources. As for the hypothesis that “lactate mediates BDNF elevation,” even greater caution is needed. Birinci *et al.* (2026)^[4] observed only simultaneous increases in lactate and BDNF, which cannot prove causality. During exercise, multiple metabolites and hormones, as well as neurotransmitters, change simultaneously, making it difficult to isolate lactate as a driving factor. This hypothesis remains speculative and far from established.

3. Dimensional Bias and Publication Bias in Cognitive Benefits

Another important problem in exercise–cognition research is the selective bias in cognitive outcome measures. Most existing studies use executive function tests (Stroop, Flanker, working memory tasks, etc.), which are most sensitive to acute exercise and most likely to show significant effects. However, this does not mean that all cognitive dimensions benefit from exercise. The meta-analysis by Chang *et al.* (2012)^[6], including 111 studies, found that acute exercise had a significant overall effect on cognitive performance, but with large differences across cognitive domains: the largest effect was on executive function, followed by information processing speed, with a smaller effect on memory. The meta-regression by Lambourne and Tomporowski (2010)^[18, 19] also confirmed that the relationship between exercise-induced arousal and cognitive performance depends on task type, with completely different response patterns for simple and complex tasks.

The abilities most central to office workers’ daily work—complex reasoning, strategic thinking, creative ideation, and prolonged deep concentration—are precisely the dimensions least studied and with the least clear benefits. Existing evidence can at most show that exercise can improve reaction speed on simple cognitive tasks; whether it can improve productivity in complex work still lacks direct evidence. More importantly, publication bias exists. The meta-review by Chang *et al.* (2025)^[7] explicitly pointed out that significant publication bias exists in exercise–cognition research; positive results are significantly more likely to be published than negative results, leading to systematic overestimation of effect sizes in meta-analyses. This is especially pronounced for the effects of acute exercise on cognition. Many studies that did not observe significant effects were not published, creating the illusion that “exercise always improves cognition.” Lambourne *et al.* (2010)^[18, 19] also found an inverted U-shaped dose–response relationship between exercise and cognition: moderate arousal improves cognitive performance, while excessive

arousal impairs it. For office workers already in a state of cognitive fatigue, forcing high-intensity exercise to “boost the brain” may be counterproductive. Yet this point is completely obscured in the popular narrative that “exercise boosts brainpower.”

4. Evidence Boundaries and Reverse Causality in Long-Term Benefits

In addition to controversies over acute effects, the long-term protective effects of exercise on cognitive function also face evidentiary challenges. First, there is the problem of reverse causality in observational studies. Cross-sectional studies find that people who exercise more have better cognitive function, but this may well be because people with better cognitive function are more able and willing to maintain regular exercise, rather than exercise causing better cognition. The systematic review of RCTs by Xu *et al.* (2023)^[37] confirmed the positive effects of exercise interventions on cognition in older adults, but the effect sizes were smaller than those in observational studies, indirectly suggesting that observational results are indeed confounded by reverse causality. Second, the clinical significance of long-term interventions is unclear. Randomized controlled trials show that long-term exercise can indeed produce statistically significant improvements in cognitive indicators, but statistical significance does not equal clinical meaningfulness. Northey *et al.* (2018)^[27] found that the cognitive benefits of exercise interventions are mainly reflected in specific cognitive test scores; whether they translate into improvements in actual daily functioning or reduce future dementia risk still lacks long-term follow-up evidence. Third, there is the issue of effect maintenance. Most exercise intervention studies last 12–24 weeks, with few long-term follow-ups exceeding 1 year. Cognitive aging is a decades-long process, and short-term exercise interventions of several months are unlikely to produce substantial changes in cognitive reserve. Current evidence can at most demonstrate that short-term exercise can produce functional fluctuations in performance; it cannot confirm protection against long-term neurodegeneration. The positive effects of exercise on cognitive function have biological plausibility and preliminary empirical support, but the causal chain of BDNF as a core mediator has not been conclusively established. The validity of peripheral markers and the specificity of mediating mechanisms remain controversial. Existing evidence has obvious cognitive dimensional bias and publication bias, systematically overestimating the magnitude of exercise benefits for work-related cognitive abilities. Packaging HIIT as a “workplace brainpower booster” not only exaggerates the strength of evidence but also ignores individual baseline differences and the moderating role of fatigue states.

Digital Technology and Sleep

1. Mainstream Narrative: Blue Light Harm and Digital Sleep Hygiene

In the digital age, “scrolling on one’s phone before bed causes poor sleep” has become almost a universal consensus, with the core mechanism attributed to blue light’s inhibition of melatonin. Mobile phones, tablets, and computer LED screens emit high-energy short-wavelength blue light, with peak wavelengths at 460–480 nm. This band is directly captured by intrinsically photosensitive retinal

ganglion cells and transmitted to the suprachiasmatic nucleus, thereby inhibiting pineal melatonin synthesis, delaying sleepiness, and disrupting the circadian clock. The systematic review and meta-analysis by Shechter *et al.* (2020) ^[30], including 12 studies, provided evidence for the effects of blue-light interventions. The study found that reducing nighttime short-wavelength blue-light exposure did slightly but significantly improve sleep quality and shorten sleep onset latency. Siraji *et al.* (2023) ^[32] also verified the association between light exposure behaviors and sleep, finding that increased mobile phone use before bed was associated with circadian phase delay, reduced sleep quality, and increased memory and attention problems. Based on this, standard sleep hygiene recommendations are: avoid electronic devices one hour before bed; if unavoidable, enable blue-light filter mode and reduce brightness.

In addition to the physiological effects of blue light, the psychological interference of social media has also been widely discussed. The infinite scrolling mechanism of platforms continuously provides novel stimuli, driving the dopamine reward system and keeping the brain highly alert, making it difficult to enter a pre-sleep relaxed state; fear of missing out, social comparison, and emotional fluctuations further increase the sleep burden. The systematic review by Behera *et al.* (2025) ^[3] covering 21 countries, confirmed that daily social media use is associated with stress, anxiety, depression, and poor sleep quality. The systematic review and meta-analysis by Ahmed *et al.* (2024) ^[2] further verified the negative association between social media use and sleep quality, with problematic use showing significantly larger effect sizes than general use. At the same time, digital technology is also regarded as a new tool for sleep intervention. Fully automated digital cognitive behavioral therapy for insomnia (dCBT-I) makes traditional cognitive behavioral therapy for insomnia online and automated, and is viewed as a low-cost solution to the shortage of professional therapists. The meta-analysis by Gao *et al.* (2026) ^[12], including 15 RCTs, showed that fully automated dCBT-I was associated with significant reductions in insomnia severity and consistent improvements in sleep initiation and sleep efficiency.

2. Contextualizing Blue Light Harm and Effect-Size Inflation

Although the mechanism by which blue light inhibits melatonin is clear, the actual impact of blue-light exposure in the real world has been severely exaggerated. There are three levels of bias here. First, there is a gap between experimental doses and real-world exposure. Most studies confirming significant blue-light effects use high-brightness, short-distance, long-duration laboratory exposure conditions, far exceeding the actual doses of daily mobile phone use. Hale and Guan (2015) ^[13], in their systematic review of screen time and sleep in children and adolescents, pointed out that laboratory blue-light exposure intensity is much higher than in real-life scenarios, so the degree of melatonin inhibition is also exaggerated. The blue-light exposure intensity of daily electronic devices is actually far lower than outdoor natural light, and its inhibition of melatonin is quite limited; only in dark environments with high brightness and prolonged use can measurable physiological effects occur. Second, the effectiveness of blue-light filtering has been commercially exaggerated. Numerous blue-light glasses and phone night modes are

packaged as panaceas for sleep problems. However, the meta-analysis by Shechter *et al.* (2020) ^[30] showed that the effect size of blue-light interventions on sleep improvement is actually not large, and results vary greatly across studies. In other words, blue-light filtering helps, but far less than commercial claims suggest. Many people believe that turning on night mode allows them to use their phones without concern, and consequently use them longer, resulting in a greater overall sleep impact. Third, content matters more than the light itself. The same bedtime phone use—reading a boring document versus watching short videos—has vastly different effects on sleep. Driller *et al.* (2019) ^[9] found that the impact of nighttime electronic devices on sleep largely comes from the arousing content and immersive use rather than blue-light exposure alone. Exciting games, emotional social media content, and cliffhanger series have far greater arousal effects on cognition and emotion than the physiological effects of blue light. Yet mainstream discourse always simplifies the problem to “blue light,” as if filtering blue light solves everything.

3. Social Media and Sleep:

The relationship between social media and sleep is likewise oversimplified. The truism that “using social media causes poor sleep” obscures the complexity of this relationship. First, it is usage patterns, not use itself, that determine the impact. The meta-analysis by Ahmed *et al.* (2024) ^[2] clearly pointed out that only problematic and addictive social media use is strongly associated with decreased sleep quality; normal social and instrumental use has little effect on sleep. The scoping review by Ndubisi *et al.* (2026) ^[26] also confirmed that the association between social media and sleep is highly dependent on use intensity and mode, with very limited negative effects for light and moderate use. Treating “scrolling on one’s phone” as universally bad ignores the necessity and complexity of social connection in the digital age. Second, there is the methodological flaw of reverse causality. This is the most serious defect in screen-time research. Most cross-sectional studies can only demonstrate correlation, not causal direction: does phone use cause poor sleep, or do people with poor sleep use their phones more? The longitudinal scoping review by Sullivan *et al.* (2026) ^[34] found that bidirectional associations do exist: poor sleep quality leads to more screen use the next day, and more screen use further impairs sleep, forming a vicious cycle. However, the effect sizes in the two directions are not equal; the predictive power of sleep on screen use is actually not inferior to that of screen use on sleep. The within-person design study by Bourke *et al.* (2026) ^[5] in JAMA Pediatrics is even more illustrative. The study included 25 studies and 4,562 participants and used within-person analysis to examine the association between daily screen use and sleep. Results found that the correlation coefficient between screen use and delayed bedtime was only 0.079, and the correlation with subjective sleep quality was only −0.092. Although statistically significant, the effect sizes were extremely small, and the actual clinical significance is very limited. This means that the impact of screen use on sleep is actually far smaller than imagined; it is only one of many influencing factors, not the sole cause.

4. Effectiveness Boundaries and Real-World Limitations of dCBT-I

Digital cognitive behavioral therapy is regarded as a star technology for sleep intervention, but its real-world value is

also questionable. First, efficacy is inferior to face-to-face treatment. The systematic review and network meta-analysis by Simon *et al.* (2023) ^[31] compared on-site, digital, and other forms of cognitive behavioral therapy for insomnia. Results found that digital CBT-I was superior to waiting list but significantly inferior to face-to-face treatment. Lu *et al.* (2023) also confirmed that for insomnia patients, the overall efficacy of dCBT-I is inferior to medication and face-to-face psychotherapy. Second, effects are limited in comorbid conditions. The systematic review and meta-analysis by Lee *et al.* (2023) found that dCBT-I can simultaneously improve insomnia and depression/anxiety symptoms, but for patients with moderate-to-severe depression and anxiety, the effects decline significantly. Lin *et al.* (2023) also verified this point: insomnia patients with comorbid depressive symptoms respond worse to dCBT-I. Workplace populations often have sleep problems accompanied by work stress and anxiety, and this group may respond less well to dCBT-I than patients with simple insomnia. Third, real-world dropout rates are extremely high. Completion rates in clinical trials are usually high because of strict inclusion/exclusion criteria and incentive mechanisms. The real-world randomized controlled trial by Schuffelen *et al.* (2023) ^[29] found that dCBT-I was less effective in a heterogeneous real clinical sample than in clinical trials, and dropout rates were higher. For busy office workers, spending time completing online course modules every day is itself another time pressure. Fourth, there is a risk of data-driven health anxiety. With the spread of digital sleep tools, the excessive pursuit of perfect sleep data may paradoxically worsen sleep problems through anxiety. Clinical observations and related studies have widely noted the tendency for sleep tracking to cause anxiety. Data, originally a tool, ultimately become a new source of stress. The impact of digital technology on sleep is bidirectional and complex. The negative effects of blue light and social media are real but severely exaggerated by popular discourse and commercial marketing. Screen-time research generally suffers from inadequate identification of reverse causality and inflated effect sizes. Digital cognitive behavioral therapy provides a new intervention pathway, but its effectiveness boundaries, dropout problems, and real-world applicability need to be fully acknowledged. Simplifying sleep problems to “phone addiction” not only misdirects intervention but also obscures deeper structural causes.

Individual Responsibility Discourse in Workplace Health and the Structural Turn

1. Common Bias: Biological Reductionism and Responsibility Transfer

Integrating the critiques of the three domains above reveals a common paradigmatic bias in current workplace sleep health discourse: individualization of responsibility under the dominance of biological reductionism. Whether focusing on exercise timing, BDNF-mediated cognitive enhancements, or digital sleep hygiene, mainstream literature predominantly adopts a physiological reductionist paradigm. This approach invariably reduces complex systemic issues to individual biological anomalies and prescribes behavioral modification as the sole remedy. Poor sleep is attributed to improper exercise scheduling, cognitive decline to insufficient BDNF levels, and insomnia to pre-sleep screen time. Consequently, this framework

constructs a reductionist narrative: all problems stem from personal lifestyle choices, and all solutions demand individual compliance.

In fact, however, the sleep and cognitive problems of workplace populations are fundamentally structural problems first. The systematic review and meta-analysis by Yang *et al.* (2017) ^[38] clearly confirmed a stable association between sedentary behavior and sleep problems, and sedentary behavior is underpinned by the structural nature of office work. Koohsari *et al.* (2023) ^[16] further verified the negative association between sedentary behavior and sleep quality. When work itself requires sitting for more than eight hours a day, and overtime and commuting compress rest time, requiring employees to solve health problems through personal self-discipline is essentially an unrealistic moral demand. Irish *et al.* (2015) ^[14], in their critical review of sleep hygiene, pointed out a fundamental problem in mainstream sleep hygiene promotion: it assumes that everyone has equal ability to control their sleep time and sleep environment, but in reality, work schedules, shift work, caregiving responsibilities, and economic conditions structurally limit individual choices. Attributing sleep problems to personal bad habits not only ignores structural constraints but may also cause additional health discrimination against disadvantaged groups. More alarmingly, this individualization of responsibility is being institutionalized by companies. Through employee health programs, health wristbands, exercise challenges, and other mechanisms, companies package health as an obligation of employee self-management. When employees develop health problems, the reason is that they are not self-disciplined and do not value health, unrelated to work intensity or organizational environment. Exercise, sleep, and digital self-discipline, originally health issues, ultimately become a new labor ethic.

2. Mismatch between Evidence Level and Recommendation Strength

Another critical issue highlighted in this review is the profound mismatch between empirical evidence levels and the strength of public health recommendations. The findings reveal a stark hierarchy of scientific validation across core propositions. On the one hand, robust, high-level evidence supports established claims, such as the overall benefits of regular exercise for sleep and cognition, the disruptive impact of heavy pre-sleep screen use, and the efficacy of digital cognitive behavioral therapy for insomnia (dCBT-I). On the other hand, several widely promoted guidelines rest on considerably weaker empirical foundations. While moderate evidence supports dose-response relationships in exercise timing, acute exercise-induced BDNF elevations, and melatonin suppression by blue light, many prescriptive rules—including the universal applicability of the “4-hour recovery window,” BDNF as the primary mediator of cognitive gains, the universal superiority of morning workouts, and the clinical efficacy of blue-light blocking tools—rely on speculative or weak evidence that falls short of justifying their strong policy and commercial promotion. However, in public health communication and workplace health guidelines, these conclusions of different evidence levels are treated as equally certain facts and recommended with equally strong language. Weak evidence is packaged as scientific consensus, and conclusions full of conditions and boundaries are simplified into easy-to-follow slogans. For

example, “HIIT boosts brainpower,” “no exercise within 4 hours before bed,” and “blue light ruins sleep” all have preliminary support but are far from conclusive, yet they become absolute truths in dissemination. This mismatch between evidence and recommendation not only misleads the public but also damages the credibility of science. When people find that following the recommendations does not produce the expected results, they may become skeptical of health advice and even question genuinely valuable public health information.

3. Lack of Localization and Ecological Validity

The vast majority of existing studies come from European and American populations, and direct application to Chinese workplaces has obvious ecological validity problems. The systematic review by Ferguson *et al.* (2021) ^[11] found significant annual, seasonal, and cultural differences in patterns of sleep, sedentary behavior, and physical activity, with large differences in population characteristics across countries. Chinese workplaces have a unique labor ecology: longer working hours, more prevalent overtime culture, more obvious presenteeism, a stronger midday nap culture, and different social media usage habits. For example, Western studies rarely consider the impact of midday rest. Chinese society generally has a habit of midday napping, and the effects of midday exercise on afternoon cognitive performance and nighttime sleep are completely different from Western patterns without midday naps. Another example is that Chinese workplaces rely heavily on instant messaging for work communication; after-hours work messages intrude on sleep far more seriously than recreational social media, but research on this is very scarce. More importantly, there are class differences. Employees at different job levels and income levels differ vastly in their control over work time and workspace. Frontline employees may not even be able to leave work on time, let alone maintain regular exercise and digital self-discipline. Universal health recommendations are essentially premised on middle-class living conditions and may ultimately exacerbate health inequalities.

4. Reconstruction Directions

Based on the above critique, the sleep–exercise–cognition health framework for workplace populations needs to achieve three fundamental shifts. First, from universal prescriptions to precise individualization, abandon one-size-fits-all standard recommendations and instead provide stratified plans based on sex, age, baseline fitness, and job characteristics. For example: adjust exercise timing according to work schedules rather than insisting on morning exercise; choose exercise intensity according to cognitive fatigue level to avoid excessive exercise worsening fatigue; adjust digital use habits according to individual sleep patterns rather than mechanically enforcing one hour of disconnection before bed. Emphasize individual subjective feelings over standard data and encourage self-awareness rather than blind adherence to data. Second, from individual behavior change to organizational structural intervention, truly effective workplace health promotion cannot only require employees to change themselves; it must address root problems at the organizational level. This includes: reasonably controlling working hours and overtime; establishing boundaries for after-hours work messages; providing flexible work arrangements; optimizing

meeting efficiency; setting mandatory work breaks; and improving the office environment. The benefits of these structural measures for sleep and cognitive health are far greater than exhorting employees to exercise more and play on their phones less. Third, from digital worship to rational use, objectively view the dual nature of digital technology, neither demonizing mobile phones nor fetishizing digital therapy. Digital tools can serve as aids but cannot replace professional assessment; sleep data can serve as reference but cannot become a source of health anxiety. At the same time, data privacy protection mechanisms should be established, and companies should be prohibited from excessively collecting employee biometric data under the guise of health.

Conclusions

1. Critical Findings

Through a systematic critique of three major domains—exercise timing, BDNF cognitive mechanisms, and digital sleep hygiene—this article draws the following core conclusions. First, mainstream conclusions generally suffer from a presumption of universality, and individual heterogeneity is severely underestimated. The “4-hour recovery window,” “morning exercise is optimal,” and “HIIT boosts brainpower” are all group-average findings; sex, age, fitness, and job characteristics significantly moderate the direction and magnitude of effects. Treating average findings as universal truths not only dilutes effectiveness but may also produce negative consequences (OpenAI, 2026) ^[28]. Second, causal relationships are generally overdetermined, and methodological flaws and publication bias systematically inflate benefits. The core mediating role of BDNF and the sleep damage caused by screen time both suffer from insufficient causal evidence, reverse causality confounding, and exaggerated effect sizes. Academic research and public communication both tend to reinforce certainty and downplay uncertainty, constructing an overly simplified scientific narrative. Third, health discourse is undergoing individualization of responsibility, and structural roots are systematically obscured. Workplace sleep and cognitive problems are essentially products of labor regimes and work stress, but current health discourse completely transforms them into individual lifestyle issues. Exercise and digital self-discipline become new moral obligations, and companies transfer their own health responsibilities through health promotion.

Future workplace sleep health research needs to achieve breakthroughs in three directions. First, conduct more critical, methodology-oriented research. Pay more attention to the impact of reverse causality, confounding variables, and publication bias on conclusions; adopt more within-person designs and qualitative designs to clarify causal direction; report more negative results and marginal effects to avoid selective presentation of evidence. Strengthen in-depth research on exercise mechanisms to clarify the exact roles of molecular markers such as BDNF. Second, promote localization and ecological validity research. Conduct original research on the work patterns, cultural characteristics, and lifestyle habits of Chinese workplaces to verify the applicability of Western conclusions; include participants from different occupations and social classes and address health inequality issues; conduct intervention studies in real work scenarios rather than only laboratory environments. Third, expand research perspectives at the

structural and policy levels. Move beyond a purely individual behavior research framework and explore more deeply the impact of work systems, organizational management, and labor policies on sleep and cognition; evaluate the health benefits of organizational-level interventions; and promote the inclusion of health perspectives in labor policy design (Lu, 2026). Exercise is undoubtedly beneficial for sleep and cognition, and digital technology does bring new health risks and tools. But health is never a purely personal choice; it is deeply embedded in work, social, and institutional structures. The purpose of critique is never to deny the value of exercise and digital health, but to puncture the myth that “as long as you are self-disciplined, you will be healthy,” to see the structural constraints behind individual choices, and to see the boundaries and conditions behind scientific conclusions. For sedentary workplace populations, the true path to health is never to learn to control one’s body more precisely, but to have working conditions and a social environment in which one can sleep well, exercise well, and live well.

References

1. AbuQeis I, Teeti A, Titi, A. The impact of exercise on cognitive function and brain health across the lifespan: A systematic review. *ibrain*,2026;12(2):190–200. <https://doi.org/10.1002/ibra.70024>
1. Ahmed O, Walsh EI, Dawel A, Alateeq K, Espinoza Oyarce DA, Cherbuin N, *et al.* Social media use, mental health and sleep: A systematic review with meta-analyses. *Journal of Affective Disorders*,2024;367:701–712. <https://doi.org/10.1016/j.jad.2024.08.193>
2. Behera N, Khuntia S, Pandey K, Shankar S. Impact of social media use on physical, mental, social, and emotional health, sleep quality, body image, and mood: Evidence from 21 countries—A systematic literature review with narrative synthesis. *International Journal of Behavioral Medicine*. Advance online publication, 2025. <https://doi.org/10.1007/s12529-025-10411-9>
3. Birinci YZ, Pancar S, Şimşek H, Soylu Y, Burac DG, Ionescu A, *et al.* High-intensity interval training differentially modulates acute BDNF and cognitive responses in young adult males: A randomized crossover trial. *Scientific Reports*,2026;16:26869. <https://doi.org/10.1038/s41598-026-56739-4>
4. Bourke M, Maddren CI, Sippel F, Thomas G. Within-person association between daily screen use and sleep in youth: A systematic review and meta-analysis. *JAMA Pediatrics*,2026;180(5):500–509. <https://doi.org/10.1001/jamapediatrics.2025.6490>
5. Chang YK, Labban JD, Gapin JJ, Etnier JL. The effects of acute exercise on cognitive performance: A meta-analysis. *Brain Research*,2012;1453:87–101. <https://doi.org/10.1016/j.brainres.2012.02.068>
6. Chang YK, Ren FF, LRH, Ai JY, Kao SC, Etnier JL, *et al.* Effects of acute exercise on cognitive function: A meta-review of 30 systematic reviews with meta-analyses. *Psychological Bulletin*,2025;151(2):240–259. <https://doi.org/10.1037/bul0000460>
7. Colcombe S, Kramer AF. Fitness effects on the cognitive function of older adults: A meta-analytic study. *Psychological Science*,2003;14(2):125–130. <https://doi.org/10.1111/1467-9280.t01-1-01430>
8. Driller M, Uiga L. The influence of night-time electronic device use on subsequent sleep and propensity to be physically active the following day. *Chronobiology International*,2019;36(5):717–724. <https://doi.org/10.1080/07420528.2019.1588287>
9. Erickson KI, Voss MW, Prakash RS, Basak C, Szabo A, Chaddock L, *et al.* Exercise training increases size of hippocampus and improves memory. *Proceedings of the National Academy of Sciences of the United States of America*,2011;108(7):3017–3022. <https://doi.org/10.1073/pnas.1015950108>
10. Ferguson T, Curtis R, Frayse F, Lagiseti R, Northcott C, Virgara R, *et al.* Annual, seasonal, cultural and vacation patterns in sleep, sedentary behaviour and physical activity: A systematic review and meta-analysis. *BMC Public Health*,2021;21:1384. <https://doi.org/10.1186/s12889-021-11298-3>
11. Gao K, Zhao Y, Zhang X, Su K, Jia J, Xu Y, *et al.* Efficacy of fully automated digital cognitive behavioral therapy for insomnia in adults: A systematic review and meta-analysis. *Sleep and Breathing*,2026;30(3):177. <https://doi.org/10.1007/s11325-026-03723-x>
12. Hale L, Guan S. Screen time and sleep among school-aged children and adolescents: A systematic literature review. *Sleep Medicine Reviews*,2015;21:50–58. <https://doi.org/10.1016/j.smrv.2014.07.007>
13. Irish LA, Kline CE, Gunn HE, Buysse DJ, Hall MH. The role of sleep hygiene in promoting public health: A review of empirical evidence. *Sleep Medicine Reviews*,2015;22:23–36. <https://doi.org/10.1016/j.smrv.2014.10.001>
14. Kamiya K, Hayashi E, Saito M, Nukui Y, Nakayama S, Kanazawa T, *et al.* Effects of intensity, frequency, and time window of exercise on sleep quality among community-dwelling adults aged 65–86 years. *Sleep Medicine*,2024;119:173–178. <https://doi.org/10.1016/j.sleep.2024.04.034>
15. Koohsari MJ, Yasunaga A, McCormack GR, Ishii K., Liao Y, Oka K, *et al.* Sedentary behaviour and sleep quality. *Scientific Reports*,2023;13:1180. <https://doi.org/10.1038/s41598-023-27882-z>
16. Kovacevic A, Mavros Y, Heisz JJ, Fiatarone Singh MA. The effect of resistance exercise on sleep: A systematic review of randomized controlled trials. *Sleep Medicine Reviews*,2018;39:52–68. <https://doi.org/10.1016/j.smrv.2017.07.002>
17. Lambourne K, Tomporowski P. The effect of exercise-induced arousal on cognitive task performance: A meta-regression analysis. *Brain Research*,2010;1341:12–24. <https://doi.org/10.1016/j.brainres.2010.03.091>
18. Lambourne K, Audiffren M, Tomporowski PD. Effects of acute exercise on sensory and executive processing tasks. *Medicine & Science in Sports & Exercise*,2010;42(7):1396–1402. <https://doi.org/10.1249/MSS.0b013e3181cbee11>
19. Lee S, Oh JW, Park KM, Lee S, Lee E. Digital cognitive behavioral therapy for insomnia on depression and anxiety: A systematic review and meta-analysis. *npj Digital Medicine*,2023;6:52. <https://doi.org/10.1038/s41746-023-00800-3>
20. Leota J, Presby DM, Le F, Czeisler MÉ, Mascaro L, Capodilupo ER, *et al.* Dose-response relationship between evening exercise and sleep. *Nature Communications*,2025;16:3297. <https://doi.org/10.1038/s41467-025-58271-x>

21. Lin W, Li N, Yang L, Zhang Y. The efficacy of digital cognitive behavioral therapy for insomnia and depression: A systematic review and meta-analysis of randomized controlled trials. *PeerJ*,2023;11:e16137. <https://doi.org/10.7717/peerj.16137>
22. Lu L. The history of Western holistic health development. AkiNik Publications, 2026.
23. Lu M, Zhang Y, Zhang J, Huang S, Huang F, Wang T, *et al.* Comparative effectiveness of digital cognitive behavioral therapy vs medication therapy among patients with insomnia. *JAMA Network Open*,2023;6(4):e237597. <https://doi.org/10.1001/jamanetworkopen.2023.7597>
24. Mennitti C, Farina G, Imperatore A, De Fonzo, G Gentile A, La Civita E, How does physical activity modulate hormone responses? *Biomolecules*,2024;14(11):1418. <https://doi.org/10.3390/biom14111418>
25. Ndubisi A, Agyapong-Opoku F, Agyapong B. Social media use and sleep quality in adolescents and young adults: A scoping review of reviews. *Children*, 2026;13(1):51. <https://doi.org/10.3390/children13010051>
26. Northey JM, Cherbuin N, Pumpa KL, Smee DJ, Rattray B. Exercise interventions for cognitive function in adults older than 50: A systematic review with meta-analysis. *British Journal of Sports Medicine*,2018;52(3):154–160. <https://doi.org/10.1136/bjsports-2016-096587>
27. OpenAI. *ChatGPT (GPT-5.6)* [Large language model]. <https://chatgpt.com>, 2026.
28. Schuffelen J, Maurer LF, Lorenz N, Rötger A, Pietrowsky R, Giesemann A. *Et al.* The clinical effects of digital cognitive behavioral therapy for insomnia in a heterogenous study sample: Results from a randomized controlled trial. *Sleep*,2023;46(11): zsad184. <https://doi.org/10.1093/sleep/zsad184>
29. Shechter A, Quispe KA, Mizhquiri Barbecho JS, Slater C, Falzon L Interventions to reduce short-wavelength (“blue”) light exposure at night and their effects on sleep: A systematic review and meta-analysis. *SLEEP Advances*,2020;1(1): zpaa002. <https://doi.org/10.1093/sleepadvances/zpaa002>
30. Simon L, Steinmetz L, Feige B, Benz F, Spiegelhalter K, Baumeister H. Comparative efficacy of onsite, digital, and other settings for cognitive behavioral therapy for insomnia: A systematic review and network meta-analysis. *Scientific Reports*,2023;13:1929. <https://doi.org/10.1038/s41598-023-28853-0>
31. Siraji MA, Spitschan M, Kalavally V, Haque S. Light exposure behaviors predict mood, memory and sleep quality. *Scientific Reports*,2023;13:2425. <https://doi.org/10.1038/s41598-023-39636-y>
32. Stutz J, Eiholzer R, Spengler CM. Effects of evening exercise on sleep in healthy participants: A systematic review and meta-analysis. *Sports Medicine*,2019;49(2):269–287. <https://doi.org/10.1007/s40279-018-1015-0>
33. Sullivan EC, Dubicka B, Kirsop H, Henderson LM. The longitudinal effects of social media on sleep among youth: A scoping review. *Computers in Human Behavior Reports*,2026;21:100908. <https://doi.org/10.1016/j.chbr.2025.100908>
34. Szuhany KL, Bugatti M, Otto MW. A meta-analytic review of the effects of exercise on brain-derived neurotrophic factor. *Journal of Psychiatric Research*,2015;60:56–64. <https://doi.org/10.1016/j.jpsychires.2014.10.003>
35. Wu J, Gao D, Leng L, Mai Y, Huang C, Hu M, *et al.* Eight-week Flexi-bar vibration exercise enhances cognitive function via altering the circulating levels of BDNF, TNF- α , and microRNAs in middle-aged and elderly women. *European Journal of Applied Physiology*,2026;126(2):1109–1125. <https://doi.org/10.1007/s00421-025-05992-y>
36. Xu L, Gu H, Cai X, Zhang Y, Hou X, Yu J, *et al.* The effects of exercise for cognitive function in older adults: A systematic review and meta-analysis of randomized controlled trials. *International Journal of Environmental Research and Public Health*,2023;20(2): 1088. <https://doi.org/10.3390/ijerph20021088>
37. Yang Y, Shin JC, Li D, An R. Sedentary behavior and sleep problems: A systematic review and meta-analysis. *International Journal of Behavioral Medicine*,2017;24(4):481–492. <https://doi.org/10.1007/s12529-016-9609-0>
38. Zhang M, Jia J, Yang Y, Zhang L, Wang X. Effects of exercise interventions on cognitive functions in healthy populations: A systematic review and meta-analysis. *Ageing Research Reviews*,2023;92:102116. <https://doi.org/10.1016/j.arr.2023.102116>