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Review Article

Review Article

Cognitive benefits of sleep: a narrative review to explore the relevance of glucose regulation

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Abstract

Sleep is essential for maintaining optimal health. Both sleep duration and quality have been linked to various physiological functions and physical and mental health outcomes. Nutrition has been shown to impact sleep parameters, from the nutrient composition of foods, such as tryptophan levels, to the physiological response to foods, such as the glucose response. However, the relationship between glycemic control and sleep, and its impact on next-day benefits, particularly on cognitive performance, remains complex and is not fully understood. This narrative review aims to explore the relationship between glycemia and sleep, and how it may affect cognitive performance the following day. The review includes data from observational and interventional studies, discussing mechanisms of action that may explain the modulating effect of glycemia on sleep and cognition. The evidence suggests that lower postprandial glucose and low variation of nocturnal glucose are associated with better sleep quality and shorter sleep onset latency. Good sleep quality, in turn, is positively associated with cognitive processes such as sustained attention and memory consolidation measured the next day after sleep. Future research opportunities lie in investigating the effects of modulating the glycemic and insulinemic responses through evening meals on sleep quality and next-day cognitive performance. Well-designed clinical trials involving healthy individuals are necessary to establish the effects of these interventions. Controlling glycemic and insulinemic profiles through the evening meal may have significant implications for improving sleep quality and cognitive performance, with potential impact on individual mental health, productivity, and overall well-being.

Key words: sleep; cognitive performance; glycemia; sleep onset latency; sleep quality; attention; memory; glucose response

Statement of Significance

Poor sleep quality has become a widespread syndrome of our times, while the maintenance of cognitive performance is increasingly relevant for all populations and age groups. Therefore, finding ways to improve sleep quality through effective nutritional solutions may have a tremendous impact on individual health and productivity. This narrative review aims at describing the triangular relationship between glycemia, sleep, and cognitive function to better understand the role of the evening meal and nocturnal glycemia on next-day cognitive performance. Considering the well-established role of sleep in memory consolidation and attention, glycemic control before bedtime and its impact on sleep take on newfound importance for next-day cognitive performance, ultimately contributing to overall well-being.

Introduction

Sleep is an essential biological process that plays a vital role in maintaining optimal health and optimizing cognitive function. It is closely linked to various physiological functions including metabolism, immune, hormonal, and cardiovascular systems [1–3]. Sleep serves as a restorative process, allowing the brain to consolidate memories, process information, repair neural connections, and clear waste [4, 5]. Although it is difficult to standardize the number of hours slept by different individuals and populations due to significant interindividual variability in

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sleep duration, it is estimated that humans sleep an average of 7 hours in a 24-hour period [6]. While many studies have generally considered the number of hours of sleep as a primary predictor of well-being outcomes, the quality of sleep such as sleep onset latency (SOL), sleep efficiency (SE), or sleep maintenance [7] has also been shown to be highly important—increasing the robustness of sleep as a predictor of health outcomes [8, 9]. Evidence from observational studies has established a link between primarily poor sleep duration but also quality and the development of metabolic diseases, for example, diabetes, obesity, hypertension, cardiovascular diseases, depression, reduced cognitive function, and negative mood [10–17]. It is important to note that these conditions can also negatively affect sleep, creating a bidirectional relationship.

The American Academy of Sleep Medicine and the Sleep Research Society issued a joint consensus recommending 7–9 hours of total sleep time (TST) with an SE of at least 85% [5]. A healthy sleep is thus one that offers adequate duration, regularity, quality, and the absence of sleep disturbances. Sleep duration is generally measured using objective methods, the gold standard of which is polysomnography, and subjective measures including standardized questionnaires and sleep diaries [18, 19]. Sleep quality is objectively measured in the literature using polysomnography and actigraphy, and subjectively through questionnaires (e.g. Pittsburgh sleep quality index [PSQI]) able to capture information pertaining to quality measurements such as SOL, SE, and sleep maintenance [7].

Research has demonstrated the impact of nutrition on various sleep parameters, namely sleeping time, sleep disturbances, and insomnia [20–22]. For instance, some nutrients including amino acids, vitamins, and minerals (e.g. B3 and B6, glycine, zinc) have been shown to facilitate sleep initiation and enhance sleep quality [23]. Tryptophan stands out as a promising amino acid for promoting sleep, as it serves as a precursor for melatonin and serotonin, both of which play a role in improving sleep quality in humans [24]. Particularly interesting is the role that insulin plays in increasing tryptophan bioavailability to the brain and melatonin synthesis through hypothalamic regulation. Research has shown that tryptophan has to compete with large neutral amino acids (LNAA) for crossing the blood–brain barrier and that tryptophan's availability to the brain is enhanced by the action of insulin which reduces the competition of LNAA at the blood–brain barrier by shunting dietary amino acids to the periphery [24]. Knowing the effect of exogenous glucose sources on the insulinemic response, this raises the question of the effect of dietary glycemia/insulinemia modulation on sleep parameters. Through their impact on the glycemic load (GL), macronutrients like carbohydrates and fibers, along with meal composition (e.g. carbohydrates to protein ratio), have been associated to sleep parameters [25]. However, literature contains divergent findings on the association between dietary carbohydrate intake and sleep quality in different populations, leaving open questions about the glucose response to a meal and its impact on sleep [24, 25]. The body of research around nutrition and sleep increasingly points toward more complex mechanisms by which a mix of foods consumed during the day or closer to bedtime affects our sleep and our restful state the next morning [21]. Research on dietary intervention studies has explored how the acute effects of carbohydrate intake and related glycemic response can influence cognitive performance. Findings suggest that both timing of intake and baseline levels of glycemia may have an impact on cognitive function [26–28]. Therefore, the objective of the present narrative review is to describe the triangular relationship between glycemia, sleep, and cognitive function to better understand the role of the evening meal and nocturnal glycemia on next-day cognitive performance. In this work, we present data from observational and interventional studies and discuss mechanisms of action possibly explaining the modulating effect of nocturnal glycemia on sleep and cognition.

Methods

The source articles were identified mainly using the electronic database MEDLINE (PubMed). In some cases, relevant articles identified outside of the initial MEDLINE search are described. The search focused on human studies with adults 18 years and older, and the search terms were multiple. To explore the studies focusing on glycemia, the most critical input search terms encompassed "glycemi*," "glycaemi*," "blood sugar," and "insulin." In addition, the search terms "sleep" and/or "cognit*" were used as input or output, depending on the relationship to explore. The initial searches included an otherwise healthy population with no insomnia (i.e. irrespective of body mass index classification). Due to the limited number of findings in healthy individuals, the search was extended to prediabetic and diabetic population. The full-text articles published in English that were relevant for this review were selected. To qualify the publications for further evaluation, the titles and abstracts were initially screened according to the search criteria. Studies that did not meet the search criteria were excluded.

Relationship Between Glucose Metabolism and Sleep

Glycemia and sleep appear to be tightly related as increasingly reported in the literature showing positive associations between sleep restriction and insulin resistance. Individuals with poor glucose control tend to have poorer sleep than healthy individuals, and the mechanism driving this relationship is suggested to involve a mutual effect of glycemia on sleep. Just like habitual short sleep duration is associated with an increased risk of diabetes and short sleep restriction can lead to reduced insulin sensitivity, glycemia, and insulinemia as well as glucose variability may affect sleep quality.

The importance of maintaining normal blood glucose rhythm for sleep quality

Glucose metabolism during sleep is closely linked to the circadian rhythm of insulin and glucose levels [29]. Numerous studies have measured endocrine changes overnight, in particular for insulin, glucagon, cortisol, and growth hormone, reporting that 24-hour glucose evolution is tightly regulated by the interplay between these hormones [29]. Normally, blood glucose levels do not fall considerably during the night in order to sustain the overnight fast [29]. In fact, research has shown that glucose tolerance follows a circadian rhythm, with reduced glycaemic control in the evening and night for healthy adults [29]. In other words, glucose levels remain higher at night than they would during the day in an extended fasting condition. While a slightly elevated overnight blood glucose is considered normal in healthy individuals, insulin resistance and nocturnal glucose variability can negatively impact sleep quality [30]. Elevated glucose levels and large glycemic fluctuations overnight are noted in individuals with impaired glucose tolerance and have been linked with poor sleep quality [31–33].

Even within the glucose-intolerant population, poor sleepers have been found to have higher daytime [34–38] and nighttime [31] blood glucose and insulin levels compared to good sleepers [31]. Overall, studies—most of which are in diabetic populations—suggest that higher glucose variability is associated with longer SOL while reducing glucose variability can improve sleep quality [31, 39–42]. One observational trial in 40 nondiabetic individuals monitoring 24-hour glucose levels, physical activity, and sleep for 2 weeks reported that glucose variation was positively correlated with SOL [39]. Studies in nondiabetic adults are limited and although this study used a common activity tracker instead of polysomnography and only showed the association with the standard deviation of recorded glucose levels, the authors' conclusion is supported by other clinical trials in type 1 and type 2 diabetic subjects confirming that glucose variability is negatively associated with sleep quality [31, 40–42]. In addition, late mealtimes which can cause high glucose fluctuations into the night have been shown to result in glucose intolerance overnight and worse sleep quality than routine mealtimes [43, 44]. Meanwhile, intranasal insulin administration before bedtime has demonstrated some benefits on sleep-associated endocrine regulation in elderly individuals and to a lesser extent in younger adults, through hypothalamic–pituitary–adrenal (HPA) stress axis downregulation [45]. Taken together, these findings suggest not only a bilateral association between glycemic control and sleep [46–48] but also a central effect of insulin on sleep regulation. Improving sleep through glucose modulation before sleep is a relevant avenue which is increasingly being investigated in interventions focused on the carbohydrate composition and quality in diets and evening meals. The available research is detailed in the next section.

Multifaceted effects of dietary carbohydrates on sleep quality

The impact of dietary carbohydrates on nocturnal sleep quality has been a topic of great interest. High-carbohydrate meals are often believed to cause feelings of drowsiness and tiredness which begs the question of whether this connection between carbohydrate and sleep is supported by scientific evidence.

Mechanistically, insulin release promotes the uptake of certain amino acids by peripheral tissues, which can favor the transport of tryptophan into the brain [49]. Since high-glycemic index (GI) meals typically cause a surge in insulin, they might be expected to enhance sleep. However, the literature on the effects of high-GI meals and diets on sleep quality is controversial [24]. While some studies suggest a positive relationship between high-GI meals/diets and sleep quality, most studies have observed a rather detrimental effect on sleep [30]. The negative effects may be due to disturbances in carbohydrate metabolism and hormonal balance, namely of cortisol, adrenaline, growth hormone, and glucagon. High-GI diets have been associated with decreased SOL, decreased slow-wave (SW) sleep, and increased rapid eye movement (REM) sleep [50, 51]. This suggests that while they may promote sleep initiation, high-GI meals may reduce the time spent in restorative SW sleep, which is important for learning and memory [52]. In addition, a low GL has shown positive effects on sleep continuity measures including SE [53]. The consumption of high-GL meals can lead to compensatory hyperinsulinemia [54] which may result in reactive hypoglycemia. Nocturnal hypoglycemia was shown to reduce sleep quality and well-being the next day [55–57].

Although further studies are warranted to confirm the direct effect of high-GL evening meals on reactive hypoglycemia in a healthy population, this pathway is hypothesized as a contributor to worsening sleep quality after a high-GL meal [30, 58]. The GL of a carbohydrate-rich mixed meals is determined by both the quantity and quality of carbohydrates and is affected by other factors such as the presence of fiber or other bioactives. A systematic review and meta-analysis suggest that carbohydrate quantity modulation does not have major effects on sleep parameters, except for a slight increase in the restorative SW sleep observed with low-carbohydrate meals [53]. High-carbohydrate intake moderately increases REM sleep and sleep depth, potentially linked to benefits for memory consolidation and certain cognitive processes [50, 51]. As for quality, consuming high-quality carbohydrate foods such as whole wheat and fiber-rich options with a low glycemic load has been emphasized as essential in a recent review for preventing large variations in nocturnal glucose levels that can disrupt sleep [30]. Supporting this, high-fiber intake was positively associated with time spent in SW sleep, and low glycemic index meals with lower prevalence of insomnia [30, 59].

An important differentiating point in elucidating the relationship between postprandial glycemia and sleep quality is the duration of the intervention, as most studies are acute or short term. In a most recent clinical trial testing different nutritional interventions along with exercise on sleep quality in men, the authors confirm that while high-GI meals have been thought to be superior for their effect on sleep initiation, they are not better than low-GI meals when tested for longer periods (4 weeks) [60]. Further long-term sleep studies are required to better understand the value of regulating blood glucose for a healthy sleep quality.

Relationship Between Sleep and Cognition

Significant impact of sleep duration and quality on next-day cognitive performance

Sleep plays a crucial role in maintaining optimal cognitive performance, and the association between sleep duration and cognitive function has been widely investigated. Observational studies have shown that both short and extended sleep duration are inversely associated with cognitive performance of working memory, executive function, and attention in elderly individuals [61], and of working memory and arithmetic in healthy young adults [62]. Despite some inconsistencies in the link between sleep quality and general cognitive performance, sleep quality has consistently been associated with performance on specific cognitive outcomes, namely sustained attention and memory [62–64]. The importance of sleep quality on cognitive performance is corroborated by studies in undergraduate students [65], young and older adults [66] where subjective good sleep quality was correlated to better-sustained attention, emotional memory, visual working memory, as well as memory recall, and verbal fluency. An analysis of cognitive and polysomnographic outcomes in a large sample of 206 healthy men and women, aged 20–84 years, revealed that faster response times are associated with more SW sleep, and reduced accuracy with frequent awakenings or less REM sleep [67]. These findings suggest that SW sleep positively contributes to processing speed and that sleep continuity and REM sleep can support brain function as measured by performance accuracy. In line with these results, studies on university students have shown that sleep quality is also associated with academic performance [68, 69]. Shift work, which disrupts hormonal rhythms, sleep–wake cycles, and metabolic health, is associated with reduced processing speed, working memory, psychomotor vigilance, cognitive control, and visual attention. These effects may partly be due to increased fatigue caused by disrupted sleep patterns [70].

Critical role of sleep duration in performance of attention and memory

Studies on sleep restriction and deprivation have highlighted the critical role of sleep duration in sustaining attention [71–73]. These clinical trials have shown that when individuals are deprived of sleep for 24 hours or more, their ability to maintain sustained attention, also known as vigilance, is significantly affected. This means that their capacity to stay focused and alert over an extended period is compromised. Interestingly, similar findings have been observed when individuals experience partial sleep deprivation or sleep restriction [72, 74, 75]. Sleep schedule shifts can also impair vigilance, as shown in a clinical trial conducted in China [46].

Research on sleep restriction and its impact on memory performance has yielded varying conclusions depending on the type of memory being studied. On the one hand, the effects of sleep restriction on visuospatial working memory and short-term memory are not conclusive despite clear impairments in these domains involving the processing and retention of visual information observed in some studies after sleep deprivation [66, 76–78]. On the other hand, declarative and procedural memory appears to be consistently affected by sleep loss as highlighted by meta-analyses from Newbury et al. in 2021 [79]. Findings emphasize the importance of sleep for memory and learning processes, supported by recent research on the impact of sleep deprivation on memory consolidation and next-day learning [80].

In summary, available research supports the role of sleep in maintaining healthy cognitive function. Adequate sleep is particularly important for sustaining attention, supporting declarative and procedural memory, and, to some extent, enhancing visuospatial working memory. Sleep loss disrupts these cognitive functions by interfering with the neural processes involved in learning and memory consolidation. Prioritizing sufficient sleep is essential for optimizing cognitive performance.

Potential of interventions promoting sleep to enhance next-day cognitive performance

Evidence from intervention studies indicates that solutions aimed at promoting sleep in healthy populations have the potential to enhance cognitive performance [81–85]. Among the approaches used to target both sleep and next-day cognitive performance are interventions aimed at increasing melatonin [86, 87] or tryptophan availability [83]. In the clinical study conducted by Markus et al. [83], evening α-lactalbumin intake, compared to a placebo protein, led to a significant reduction of sleepiness and to improved alertness the next morning. Such effects were explained by the changes in plasma ratio of tryptophan to LNAA and were stronger in subjects with mild sleep complaints [83].

Similarly, a 7-day supplementation of a glycine-rich collagen peptides, compared to a placebo control, was found to reduce the number of awakenings and cognitive performance in athletic males with sleep complaints [85]. While in many studies the effectiveness of a solution on cognition may be mediated by improved sleep, there is mechanistic support for some solutions (e.g. glycine) to directly affect brain function in regions involved in cognition, as discussed by Thomas et al. [85]. The authors also mention previous human research having demonstrated a sleep-independent effect of collagen peptides on cognitive function in older adults [88].

Moreover, supplementation of botanicals like *Mentha spicata* and *Coriandrum sativum* has been shown to be beneficial on both sleep and cognitive functions [81, 84]. These effects are likely to be due to mechanisms of action different than glycemic regulation, such as antioxidant and anticholinesterase activity of bioactive compounds found in these plants [81, 84].

Several clinical studies aiming at enhancing sleep and cognition via nutrition solutions did not observe a significant change in both outcomes [41, 86, 87, 89]. For example, a study involving a 3-month supplementation of Tart Montmorency cherries observed improvements in sustained attention and feelings of alertness among a sample of 50 middle-aged adults, but no significant changes in sleep [87]. This contradictory result is likely due to the use of subjective sleep measures instead of objective ones. A systematic review and meta-analysis has shown that tart cherry can objectively and significantly enhance TST and SE, despite individuals not subjectively perceiving such benefits [90]. The discrepancies noted in the research literature are largely due to the considerable differences in study designs (whether parallel or crossover), the methods used to measure the outcomes (subjective or objective), the characteristics of the study populations (such as age or sleep habits), the nature and duration of the interventions, and the specific cognitive outcomes targeted.

Finally, although not specifically focused on cognitive outcomes, one study showed that high-GI meal was found to improve both sleep duration and SE, as well as physical performance of visual reaction times [91]. Among various measures of physical performance, time to reaction was the only parameter positively influenced by the restorative effects of improved sleep, showing an increase proportional to the sleep improvements observed [91].

Relationship Between Glycemia and Cognition

Importance of glucose regulation to support optimal cognitive performance

Glucose metabolism plays a crucial role in supporting optimal cognitive performance in healthy adults. Disruptions in blood glucose homeostasis, whether acute or chronic, can have detrimental effects on a wide range of cognitive functions. Conditions such as type 2 diabetes and metabolic syndrome, characterized by insulin resistance and resulting in elevated blood sugar levels, are particularly known to impair various cognitive functions [92, 93]. These conditions notably impact learning and memory, but also extend to psychomotor speed, attention, and executive functions.

It is crucial to prioritize healthy glucose regulation to support optimal cognition and mitigate the negative impact of blood glucose disturbances on brain function.

During cognitive tasks, the brain requires a constant supply of glucose as its primary energy source [94, 95]. Studies have shown that cognitive tasks, such as attention, memory, and problem-solving, are associated with increased glucose levels in specific brain regions [96]. Hormones like insulin and glucagon help maintain an adequate supply of glucose to the brain by regulating its uptake from the blood and release from storage sites as required [97]. Glucose intake has been extensively studied in relation to cognitive outcomes, with memory performance being a particularly sensitive measure [98]. Glucose intake immediately before or after a learning task has been shown to improve memory performance, the effect on recall lasting up to 24 hours [99]. In a clinical trial conducted by Herzog et al. [100], the impact of the amount of energy intake during the day on memory performance the following day was investigated under conditions of sleep and total sleep deprivation. To this aim, maltodextrin-enriched standardized meals and drinks were administered to participants equaling either ~50% or ~150% of the estimated daily energy expenditure. The "high-energy" group consumed three meals

enriched with maltodextrin to increase energy content, including dinner at 19:00. Surprisingly, there were no observed differences between the high- and low-energy conditions in terms of learning and recall. This may suggest that supplying more energy at the evening meal does not provide any additional benefit for memory performance. The authors also reported that plasma glucose levels before learning (on the first day) or in the morning before the recall test did not differ between conditions. Unfortunately, glucose measures were not taken during the night. Further research is needed to better understand the relationship between glucose intake, sleep, and memory consolidation. Additionally, investigating glucose levels during the night may provide valuable insights into the mechanisms underlying the effects of glucose on memory.

Impact of carbohydrate timing on attention and memory

Numerous studies have investigated the impact of glycemic index and load on cognitive functions, specifically focusing on memory and attention as measured by standard computerized cognitive tasks. Previous research has highlighted the benefits of maintaining stable blood glucose levels through low glycemic load meals in normoglycemic subjects, particularly in preventing declines in attention and memory during the late postprandial period (75–222 minutes) [101]. In this context, the ingestion of a blueberry beverage has been shown to decrease glucose and insulin concentrations in response to a meal within the first 120 minutes [102]. In addition to the metabolic benefits, blueberry ingestion supported the maintenance of cognitive performance, compared to placebo intake, which was associated to a decline in performance. These effects were observed in tasks assessing delayed recognition memory and executive functions, particularly under conditions of increased cognitive demand [102].

A recent meta-analysis of clinical trials found that low glycemic load breakfasts had a positive effect on episodic memory, but only during the late postprandial period [26]. The timing of the meal and glucose control were identified as significant factors influencing cognitive performance. Individuals with better glucose control enhanced their episodic memory with the consumption of a low glycemic load breakfast, specifically 2 hours or more after the meal. No difference was observed between low and high glycemic load meals in the early and mid-postprandial period, regardless of glucose control status [26]. This suggests that the direct effect of glucose response and GI on cognition may be time-dependent. For example, a study on night shift workers found that higher carbohydrate intake before a vigilance test improved sustained attention when consumed 1 hour before the test, but worsened attention when consumed 2–3 hours before the test [103]. This highlights the importance of considering the timing of meals relative to cognitive measurements. While previous research on the relationship between dietary carbohydrate and cognition has primarily focused on acute interventions during the day, one study found that the GI of the evening meal can have an impact on memory, the following day [104]. The GI effect of a meal persisted even after an overnight fast and consumption of a standardized breakfast. This phenomenon, known as the "second meal cognitive effect," suggests that the glycemic response to a previous meal can influence the glycemic response to the subsequent meal. It is hypothesized that consuming an evening meal with a more consistent glycemic response throughout the night may enhance cognitive performance on the next day compared to meals that result in greater glycemic variability. However, further research is needed to thoroughly understand the relationship between meal timing, glycemic response, and cognitive outcomes. Some insights in this direction may come from the systematic reviews by Sharifi et al. and Senderovich et al. investigating the impact of time-restricted eating and intermittent fasting on cognitive outcomes [105, 106]. While both works conclude that these dietary approaches can have positive effects on cognitive outcomes, it is important to note that available clinical evidence is still limited and sometimes contradictory. Hence, additional research is needed to better understand the optimal time window and potential cognitive benefits, particularly for specific target populations.

Does Nocturnal Glycemia Impact Next-Day Cognitive Performance Through Improving Sleep?

Studies on adults with type 1 and type 2 diabetes have reported low sleep quality in this population [11, 36, 37], as well as impairments in specific cognitive domains [92, 107]. A cross-sectional study involving prediabetic and type 2 diabetic patients found that glucose control was significantly associated with cognition, with diabetes status independently predicting reduced cognitive performance [108]. Additionally, the study identified SE as another independent predictor of decreased cognitive function. These findings highlight the importance of addressing both glucose control and sleep quality in diabetic populations to mitigate cognitive impairments.

Griggs et al. explored the association between rest-activity rhythms and glycemic outcomes in a 6- to 14-day observational study in young adults with type 1 diabetes [109]. The authors observed that stronger rhythm adherence was associated with a lower risk of hyperglycemia, longer TST, less daytime sleepiness, and better executive function. Robust rhythms were also positively associated with the risk of hypoglycemia, an unexpected finding knowing that hypoglycemia is generally inversely associated with cognitive performance. However, this finding may have been partly confounded by the duration of type 1 diabetes in the study population. Indeed, when the authors added the duration of diabetes into the model, the relationship between rhythm robustness and hypoglycemia risk disappeared, and there was a positive association between diabetes duration and time spent in hypoglycemia. Another ambiguous relationship is that of hypoglycemia with cognitive function. Although severe hypoglycemia has been shown to impair next-day cognitive function in type 1 diabetic subjects [110] and in normoglycemic individuals [111], it can be argued from the relevant literature that this depends on the severity and duration/frequency of hypoglycemia. For instance, research indicates that moderate frequent hypoglycemia may in fact support cognitive function through a mechanism of brain adaptations making nutrient delivery and utilization more efficient, especially in times of energy deficits [112]. For this reason, answering the question on whether nocturnal glycemia impacts next-day cognition through sleep requires interventional trials modulating glucose at bedtime and monitoring glycemia throughout the night in normoglycemic individuals to exclude the confounding factors related to glucose control in diabetic individuals. Also, these intervention trials must include longer study periods to better understand the possibility of brain adaptation to chronic moderate hypoglycemia.

Today, and although a parallel may be drawn between the individual relationships of postprandial glycemia and sleep with

cognition, very few studies have investigated the effect of glucose modulation on sleep quality and next-day cognition. Glycemic index/load of meal or diet was studied as a factor affecting cognitive performance with findings generally showing that while high-GL meals appear to enhance sleep and cognition on the short term, they show deteriorating effects on the longer term [103]. Consequently, low-GI meals inducing a slow and steady rise in blood glucose are more likely to be beneficial for cognitive performance [26]. Yet, these findings are based on daytime studies, making it difficult to extrapolate conclusions to bedtime meals and their impact on next-day cognition. Nilsson et al. [113] conducted a crossover study investigating bedtime meals with different glycemic indices and fiber type and content in 15 healthy subjects. The authors reported that meals containing dietary fiber, particularly resistant starch had a regulatory effect on the glycemic response lasting until the next morning breakfast, compared to white bread, a lower-fiber high-GI meal [113]. The next-day effect was attributed to an improvement of glucose tolerance during the night, not necessarily driven by the glycemic index but rather by the fiber type and content of the meal. Unfortunately, Nilsson et al. did not measure sleep parameters or next-day cognitive performance in their study. Lamport et al. did measure cognitive performance in their crossover trial in healthy individuals consuming evening meals with different GIs to measure the impact of glucose modulation on next-day cognition [104]. Memory, psychomotor skill, and executive function were assessed after the evening meal and again after a high-GI breakfast the next day. Results of this study show improved memory after the high-GI breakfast for the high-GI evening meal condition compared to the low-GI evening meal condition. The same authors ran a similar study in 2013 showing again that memory was improved at breakfast after the high-GI evening meal compared to low-GI evening meal although the opposite was observed when tests were run directly after the evening meal [114]. These findings disagree with the daytime studies on cognition and studies on sleep generally concluding that a low-GI meal is more beneficial than a high-GI meal for sleep and cognition. In the case of Lamport et al., no sleep parameters were measured. Therefore, the question still remains whether these interventions affect overnight glycemia in such a way to improve sleep quality and its cognitive consequences. Further research to explore the impact of selected nutrients on nocturnal glycemia, sleep, and consequently the impact on next-day cognition is required. In fact, one study has looked at these three parameters together, although the intervention was not a nutritional modulation of glucose, but rather insulin-induced hypoglycemia. Jauch-Chara et al. investigated whether induced hypoglycemia during early nocturnal sleep affected the sleep-associated consolidation of declarative memories in both diabetic and healthy subjects [56]. Findings revealed that the episode of hypoglycemia increased time spent awake during the first part of nighttime sleep, and resulted in significantly impaired memory retention and mood on the following day [56]. Of interest, despite the increase in self-reported fatigue and reduced memory performance, performance of vigilance and attention was not affected by hypoglycemia [56].

In summary, the implications of glycemia modulation on sleep patterns and next-day cognition are complex and may well depend on the severity of glucose fluctuations caused by the meal and the timing of the pre-bedtime meal. Glycemia modulation may also not affect all cognitive domains equally. Today, there is evidence that pre-bedtime meals can affect sleep and next-day cognition, and it suggests that changes in nighttime glycemia significantly impact selective cognitive domains the next day. However, we cannot fully answer the question to elucidate whether the impact of pre-bedtime meals on cognition is mediated by an effect on sleep.

Role of HPA axis hormones in the triangular relationship between glycemia, nocturnal sleep, and cognitive performances

One mechanism that supports the mediating role of sleep in the effect of glycemia on next-day cognition involves changes in the HPA axis hormones [115]. The HPA axis is the main stress response system in animals and humans. It is a complex mechanism that mediates the response to a stressor by activating a cascade of neuroendocrine pathways leading to regulation of metabolism, immune response, and other physiological functions in response to stress. HPA axis hormones include cortisol, adrenocorticotropic hormone (ACTH), and norepinephrine (NE). These hormones are tightly and bidirectionally linked to sleeping patterns and sleep quality [116]. Indeed, HPA axis hormone levels follow a circadian rhythm such that cortisol levels are low during the night and reach their highest peak upon awakening. Therefore, in normal individuals, cortisol levels increase in wakefulness but also in light sleep and is reduced during SW sleep (non-REM stage 3), or deep sleep (N3) [117, 118]. Sleep stages, sleep quantity, and quality have shown a significant relationship with plasma HPA axis hormone levels as well. In fact, disturbed sleep or shorter sleep time in normal individuals is characterized by an increase in cortisol levels compared to normal sleep, suggesting an activation of the HPA axis [119, 120]. While deep sleep has an inhibitory effect on the HPA axis and hormones release (namely cortisol, ACTH, and NE), its activation leads to sleep disturbances and interruptions [121]. This was shown in several human experiments where corticotropin-releasing hormone and ACTH were administered to test the excitatory effect of the HPA axis on sleep quality. Findings show decreased SE and reduction in non-REM and SW sleep, with stronger effects observed in middle-aged individuals compared to young counterparts [122]. Perhaps this explains the rise in insomnia prevalence in modern society [116], keeping in mind the inter-individual variability in sensitivity/reactivity to stress [123], one of many factors being age [122].

The HPA axis plays an instrumental role in the maintenance of basal homeostasis. In fact, glucocorticoid receptors are ubiquitous in our cells, impacting basic physiological systems with direct consequences on metabolism, immunity, and brain function. The balance in HPA axis hormones is therefore crucial to maintain homeostasis in these systems, and any HPA axis dysregulation has proven to be deleterious to health. For instance, adequate concentrations of cortisol are important for healthy cognitive function while excessive or sustained levels of cortisol have shown to negatively impact these functions, in particular hippocampal-dependent memory (e.g. declarative memory), and executive function evidenced by lower performance on tests measuring attention, abstract thinking, and cognitive flexibility [124–126]. Nutrient metabolism is also associated with HPA axis regulation as excess cortisol was shown to increase gluconeogenesis [127, 128], insulin resistance, and reduce glucose uptake by muscle. High levels of cortisol have repeatedly been associated with increased deposition of visceral fat and the metabolic syndrome [129].

This relationship between the HPA axis and glucose metabolism is bidirectional. Just like a dysregulation of the axis leads to impaired metabolic health, hyperinsulinemia and diabetes

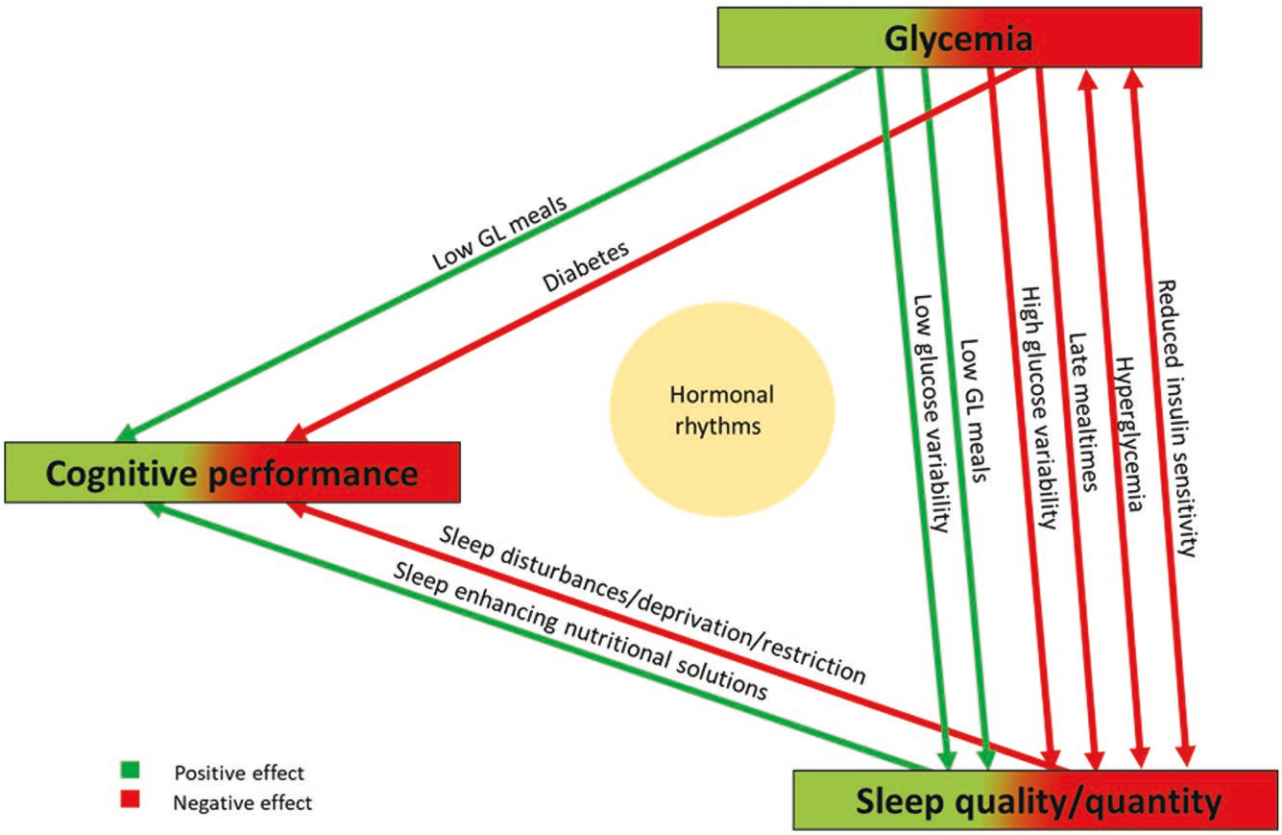

Figure 1. Triangular relationship between glycemia, nocturnal sleep, and cognitive performances. GL, glycemic load.

have been shown to drive an increased activation of the HPA axis, consequently increasing cortisol levels, leading to functional hypercortisolism [129]. Acute rises in blood glucose and insulin associated with the intake of a carbohydrate meal stimulate cortisol production. In addition, postprandial glucose and insulin responses are higher after an evening meal compared to the same meal consumed during the daytime [130]. Therefore, it can be hypothesized that insulin-stimulating meals consumed before bedtime may activate the HPA axis, causing disruption in hormonal rhythms, specifically rising cortisol levels overnight, affecting sleep and consequently next-day cognition.

Gaps and Opportunities for Future Research

The reviewed evidence shows that lower postprandial glucose and low variation of nocturnal glucose are associated with better sleep quality and shorter SOL. In turn, sleep quality is positively associated with cognitive processes. More specifically, numerous studies show a positive impact of good sleep quality on sustained attention and memory. These studies support the key role of sleep on memory consolidation and attention.

In parallel, improving sleep through nutritional interventions has demonstrated next-day cognitive benefits although findings of supportive studies are not confirmed across the literature. This may be due to differences in study design, population, and types of interventions. The gaps in the literature present multiple research opportunities to test the impact of modulating the glycemic/insulinemic potential of the evening meal on the overnight profile of blood glucose, insulin, and HPA axis hormones, as well as the effect of the meal on sleep quality and next-day cognitive performance. Important outstanding questions to be answered include how pre-bedtime glucose modulation and overnight glucose profile affect cognition after a full-night sleep and whether this effect is dependent on the sleep-enhancing impact of pre-bedtime meals. To this aim, interventional studies carefully manipulating the energy density, macronutrient composition, and timing of the evening meal are warranted. Additionally, incorporating polysomnography, continuous glucose monitoring, and next-day neuropsychological testing will provide valuable insights. By carefully evaluating these factors, a deeper understanding of the three-way interaction between glycemia, sleep, and cognition—illustrated in Figure 1—can be attained, leading to the development of optimal dietary strategies for enhancing cognitive function through optimizing sleep quality. Besides carbohydrate quantity and quality, other ingredients in a meal can be considered for their effect on postprandial glycemia and insulinemia. For instance, the protein and fat composition of a meal has a significant impact on the postprandial glucose response [131]. Similarly, the type of protein, fiber, and plant extracts [132–134] included in a meal is important in modulating postprandial glycemic and insulinemic responses. Glucose-modulating solutions can be useful additions to evening meals given their proven efficacy on glucose control, and more particularly postprandial glucose response.

Conclusion

The present review explored the role of sleep in enhancing cognitive functions and its connection to glucose metabolism. More specifically, we have attempted to understand if a modulation of glucose response to the evening meal can influence sleep quality and consequently next-day cognition.

In summary, there is extensive research supporting the pivotal role of sleep on cognition, and an equally rich body of research on the modulating the effect of the glycemic load on cognition. The cognitive domains that have been most extensively investigated are memory and attention. However, the mediating role of sleep in the relationship between glycemia modulation and next-day cognition is yet to be fully understood. In fact, confounding factors such as meal patterns and the metabolic state of individuals add to the complexity of this relationship. Nonetheless, it has become increasingly clear that large excursions in glucose and insulin levels overnight carry deleterious effects on sleep quality and that the evening meal may be key to controlling glycemic and insulinemic profiles overnight and into the next morning. Given the established importance of sleep for memory consolidation and improved attention, glycemic control pre-bedtime and its impact on sleep take on a newfound importance for next-day cognition.

Poor sleep quality is a widespread syndrome of our times, while the maintenance of cognitive performance is increasingly relevant for all populations and age groups. Therefore, finding ways to improve sleep quality through effective nutritional solutions may have a tremendous impact on individual cognitive health, productivity, quality of life, and finally societal health.

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