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Beyond cognition and sleep: Stop the domino effect

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Abstract

Sleep problems, particularly sleep deprivation and fragmentations, are common hazards in modern lifestyles and may be an unavoidable occupational drawback. Different metabolic or hereditary causes may induce sleep problems. Cognitive functions and closely related metacognition, which depend significantly on decision-making, are essential for learning and problem-solving. When impaired for any reason, the quality of life deteriorates, and the person may become dependent on others in severe cases of dysfunction. Sleep and cognition/metacognition are related, and an insult to one function may lead to the other domain's collapse. The present review highlights the significant causes and conditions of sleep problems, cognitive and metacognitive dysfunctions, the possible mechanisms, interactions, and the potential tools or agents that may improve them.

Keywords: sleep, cognition, metacognition, obesity, probiotics

1. Background

Poor sleep is a common health hazard in modern societies, and it impairs the rhythmic expression of clock genes responsible for circadian rhythm regulation for the central clock in the central suprachiasmatic nucleus (SCN) and the peripheral clocks throughout the body (1). Moreover, sleep deprivation is associated with impaired cognition, metacognition (2), stress coping, and affective disorder (3). These cases are summarized in Fig. 1.

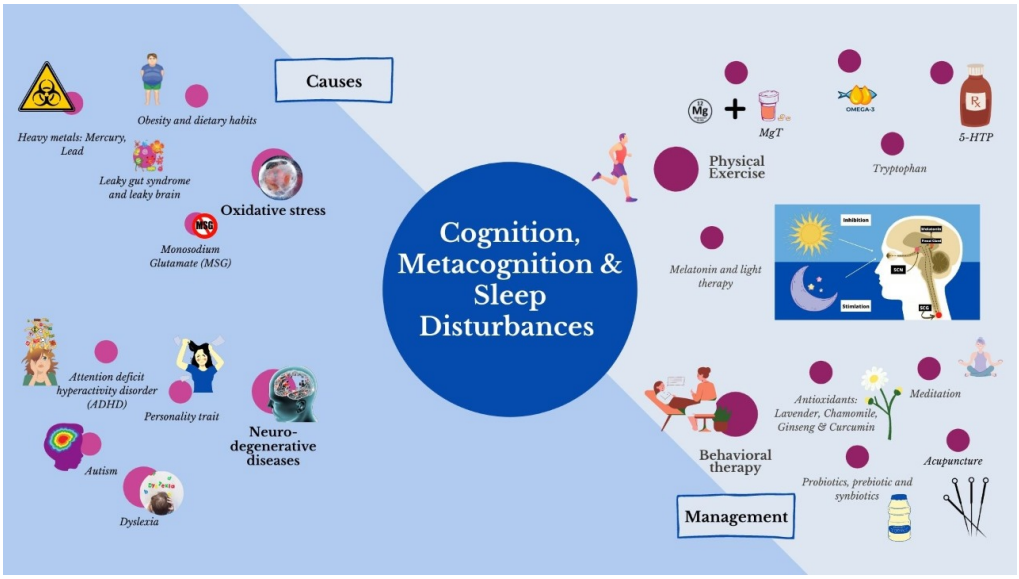

Fig. 1. Causes and management of sleep disturbances

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During sleep, there are ongoing processing and running activity for different cognitive domains like consolidation of motivationally relevant information, which depends on sleep spindles and slow-waves oscillatory activity (4), dopamine system activation (5), decision-making, and conditioned learning (6).

According to Caputo et al. (7), learning and cognitive memory mechanisms play a vital role in developing and maintaining anxiety since exposure to cues related to aversive situations induces high arousal and anticipatory anxiety. Memory becomes unstable following reactivation, and modification is then possible via reconsolidation and extinction, and inhibition of these processes results in attenuation of contextual cues on the anticipatory anxiety (8).

Circadian rhythm refers to all the physiological and behavioral activities repeated in a cyclic manner around 24 hours. The periodicity patterns depend on external factors or cues called zeitgebers, such as exposure to light or dark and temperature changes. The circadian phase determines multiple aspects of sleep physiology, including total sleep duration, the phasic alternation between rapid eye movement (REM) sleep and non-rapid eye movement sleep (NREM), sleep continuity, and spindle activity (9).

Accordingly, the circadian phase affects wakefulness and sleep duration, reflected in synaptic plasticity markers (10). Daily stress, hyper-arousal, and pre-sleep cognitive activities significantly affect sleep latency and insomnia (11).

Melatonin and cortisol levels are hormonal markers of circadian phases. Melatonin is the primary regulator of sleep-wake timing (12). In contrast, cortisol is a stress hormone strongly linked to anxiety and depression and plays a role in arousal (13). Sleep effects on cognition are not related to age. However, aging makes the person more susceptible to the hazards of sleep deprivation and interruption of circadian rhythm and cognition (14, 15). Accordingly, maintaining normal sleep could be a protective or therapeutic tool for certain cognitive disorders (16).

Metacognition is a higher level of managing and controlling cognition; it is firmly related to decision-making and problem-solving with neural systems located in the prefrontal cortex (17). Metacognition is a powerful academic tool that can lift academic achievement and learning if used properly. It represents our driving and control of thinking through two significant steps: metacognitive knowledge and regulation (18). Metacognitive knowledge is what we all know about our thinking, which approach is the best and differentiates between what we know and what we understand (19). The second step for metacognition is metacognitive regulation, which involves the actions and procedures we perform to learn (20).

The direct relationship between metacognition and sleep deprivation is not well studied relative to cognitive functions. Baranski (21) reported that sleep deprivation affects cognition and metacognition with more stress on cognitive functions. In another study by Aidman et al. (2), Using 40 hours of sleep deprivation in 13 Australian Army male volunteers aged 20 - 30 years, tested metacognition functions many times. It showed that fatigue-inducing states affected metacognition rather than cognitive mechanisms.

The present review highlights the significant causes and conditions of sleep problems, cognitive and metacognitive dysfunctions, the possible mechanisms, interactions, and the potential tools or agents that may improve them.

The methodology used for the present review involved electronic searches on PubMed and googled scholar between 1985 and 2021 using the keywords; sleep, cognition, metacognition, obesity, dietary habits, leaky gut syndrome, leaky brain, heavy metals, MSG, exercise, Omega-3, magnesium, probiotics, oxidative stress, antioxidants, herbs, acupuncture.'. We excluded articles in languages other than English and unrelated ones, assessed two hundred eighteen articles for eligibility, and included them in the review.

2. Common causes of impaired sleep and metacognition

2.1. Obesity and dietary habits

Cultural dietary habits are essential determinants for weight gain, sleep quality, and duration consequences. Moreover, in a cross-sectional self–reported questionnaire on African American 1837 adults (75% females), with a mean age of 48.2 + 13.7 years and a mean BMI of 32 + 7.5 Kg/m2, Wu et al. (22) demonstrated that decreasing sleep was related to increasing weight and Body Mass Index (BMI). This creates a vicious pathological circuit of weight gain and sleep abnormality.

Ding et al. (23) revealed an association between high consumption of sugars and sleep deprivation with modified leptin hormone profiles. They explained that shift work affected the hypothalamic-pituitary-adrenal axis and triggered circadian disturbances causing hyperglycemia and excessive fat deposition. Leptin and ghrelin are essential for controlling hunger and sleep, and both hormones are reciprocally regulated in sleep deprivation with higher production of ghrelin and lower production of leptin, resulting in disruption of energy balance (24, 25).

Metacognitive dysfunction is well described with eating disorders like anorexia nervosa (26). By evaluating 44 adults, Quattropani et al. (2016) illustrated that emotional and affective disorders were commonly associated with obesity.

On the molecular level of the neurons, repeated sleep disruption produces oxidative stress, neuronal injury, and loss, especially among the neurons involved in arousals like hypothalamic orexinergic neurons and the locus ceruleus noradrenergic neurons (27).

Obstructive Sleep Apnea (OSA) interrupts or pauses breathing cycles during sleep with airway obstruction. It is a common disorder among obese, related to numerous health

issues (28). Sleep duration and quality are essential to maintain cognitive performance homeostasis (29). It is common in obese individuals with decreased sleep duration or sleep quality, usually with dysfunction of cognitive processes (14, 30), especially consolidation of memories (31) through synaptic remodeling (32). Holloway et al. (32) studied 16 male subjects randomly assigned in a crossover design. They used a high-fat (75%) diet compared to a standard diet (23% fat) for five days. After a 2-week washout period, subjects consumed the opposite diet. They showed that a seven-day high-fat diet reduced attention and reaction time with depression and impaired retrieval speed associated with neuroinflammation. Besides, after studying 4- year cognitive change in 6183 older women, Okereke et al. (33) demonstrated that high intake of saturated fats in young adults impaired memory and cognitive function, causing neurological disorders like dementia and Alzheimer's disease in mid and later life.

Fats increase oxidative stress and change apoptosis. Free radicals cause the progression and development of cognitive dysfunction via interfering with synaptic transmission, mitochondrial function, neuroinflammation, and axonal transport, with neuronal loss in dementia disease (34).

2.2. Monosodium glutamate (MSG)

Monosodium l-glutamate (MSG), a sodium salt of l-glutamate, is commonly used as a taste-enhancing additive. Glutamate, an excitatory neurotransmitter, is a part of its molecule (35). MSG produces excitatory and inhibitory responses according to dose, route, and intake duration (36). Onaolapo et al. (36) compared the effect of food–added MSG on neurobehavior, serum biochemical measures, cerebral cortex, liver, and renal morphology in mice fed a standard diet and high-fat diet for eight weeks.

Glutamate transport across the BBB is well-regulated to protect against glutamate-induced excitotoxicity (37). Prolonged dietary consumption of glutamate in the form of MSG in 64 adult male rats increased glutamate levels in the brain in the long run and impaired hippocampal function (38). This produced forebrain activation in various areas, including the insular cortex, basal ganglia, limbic system, and hypothalamus (39). In studying ten adult rats, MSG prolonged Rapid Eye Movement (REM) sleep duration and its episode frequency (40) and was associated with snoring and sleep breathing disorders in non-obese subjects (41).

MSG causes hyperactive and inattentive behavior (35). Akataobi. (35) demonstrated that MSG affected neonate and adult rats after six weeks of study similarly. Memory impairment with MSG is either a result of interference with glutamate synthesis in the hippocampus or inhibition of the cholinergic system (42).

The effect of MSG on cognition has also been linked to its attenuating effect on the cyclic - AMPK level in the hippocampus (43). Hippocampal AMPK protects neurons and attenuates the damage by b-amyloid and glutamate excitotoxicity (44).

2.3. Leaky gut syndrome and leaky brain

There is a complex bidirectional interaction between the gut microbiome and the brain. This microbiome involves trillions of human microorganisms, including bacteria, fungi, viruses, and protozoa. It has the most significant and vulnerable surface to prevent a leak (penetration) of some food components, environmental factors, and others. Disruption of this barrier causes leaky gut and brain, resulting in neurological diseases such as Alzheimer's, autism spectrum disease, stress, and Parkinsonism (45).

Leaky gut (intestinal hyperpermeability) triggers include physical or psychological stress, nutritional deficiencies, food allergy, food irritants such as casein or gluten, food additives, intestinal dysbiosis, infections, autoimmune diseases, toxins, and NSAIDs.

When significant bacterial or food particles penetrate the intestine, they release inflammatory and immune mediators, initiating more inflammatory and allergic responses, with more intestinal permeability and changes in CNS functions, including mood and behavior (46). In a review by Julio-Pieper et al. (46), they reported that about half of clinical and animal model studies showed intestinal barrier damage in schizophrenia, autism spectrum disorders, and neurodegenerative diseases.

2.4. Oxidative stress

After intracerebroventricular injection of radiolabeled arachidonate into mouse models of Alzheimer's disease, Furman et al. (47) revealed increased biomarkers of oxidative stress in many neurodegenerative diseases. Redox imbalance can cause a leaky gut and leaky brain via immune cells modulating oxidative stress. Ischemia may initiate disruption of the blood GIT and blood-brain barrier and occurs via reperfusion and production of reactive oxygen or free radical formation. This is antagonized by antioxidants (48).

In humans, excessive energy, especially high-fat food, causes oxidative stress and impairs cognitive function. Oxidative stress is more likely to develop in the brain due to the diversity of reactive species to modulate heterogeneous signaling pathways (49). Cognitive dysfunction is linked to low-grade inflammatory stress, inducing cell-mediated immunity and oxidative stress.

2.5. Heavy metals

Mercury

Mercury (Hg) exposure induces aversive memory injury and recognition memory deficits (50). A review by Bjørklund et al. (50) revealed neurological symptoms in dental workers who are occupationally exposed to chronic low levels of metallic Hg. In dentists and dental personnel, memory, neurobehavioral, cognitive, and attention disturbances are more common. Long-standing exposure to HgCl2 impairs memory and induces anxiety (51). Mercury accumulation has

been shown in the pineal gland, which participates in circadian function through melatonin and serotonin (52).

Hg can cause Alzheimer's disease (53), and the high penetrability of Hg into the brain alters sleep patterns by dysregulating the extracellular concentrations of glutamate, acetylcholine, and dopamine. These neurotransmission changes are reflected in non-REM sleep, REM sleep, awakening, and decreased night-time melatonin levels (54).

After intoxicating 20 rats for 45 days, Teixeira et al. (51) showed that $HgCl_2$ accumulates in the hippocampus and cortex regions with a higher affinity for the cortex. Furthermore, Mercury inhibits serotonin binding to brain receptors (53). The 5-HTergic system in the prefrontal cortex and basal ganglia plays a significant role in neuroprotection and cognitive regulation (55).

Mercury exposure in the prenatal period impairs the dopaminergic and glutamatergic system, impairing learning and memory (56) with decreased IQ scores and other attention and spatial tests memory (57).

Methyl mercury, which has sufficient permeation to the brain, causes Minamata disease, producing damage and neurological manifestations of the disease (58).

Additionally, there is an association between mercury toxicity and autistic spectrum disorder (ASD) diagnosis. Of 91 studies from 1999 to 2016, 74% suggested a direct and indirect relationship between Hg and autistic spectrum disorder (59).

Lead

Lead (Pb) is another heavy metal that can penetrate the CNS and negatively affect metacognition and sleep. Lead precipitation in front-hippocampal circuits impairs the acquisition, consolidation, and recall of memories, especially emotional memories, which causes emotional and behavioral dysregulation frequently seen in Pb-exposed children (60). Moreover, lowered intellectual scores, learning and memory scores, visual memory scores, verbal memory scores, and inadequate sleep with inadequate performance during the day have been recorded with lead exposure (61). In a cross-sectional study, Mohammadyan et al. (61) measured the occupational exposure of 40 soldering workers to lead fumes through their blood lead levels and sleep quality and recording of digestive disorders. Impaired sleep quality and gastrointestinal disorders were prevalent.

According to Kalinchuk et al. (62), nitric oxide (NO) donors induce sleep through adenosine production. This means that reduced NO production causes sleep deprivation. Pb changes the activity and expression of neuronal nitric oxide synthase (nNOS) and brains endothelial nitric oxide synthase (eNOS) as it simulates Ca^{+2} and blocks its binding sites for NOS, reducing cerebral NO levels.

In addition to its effect on sleep, the reduced NO production impairs long-term potentiation (LTP), forming the molecular base for learning and memory (63). Lead exposure causes hippocampal damage by denaturating myelin and neuronal nuclear irregularities (64). Allen et al. (65) described memory and metacognition dysfunction using a high–resolution multi-parameter mapping technique in 48 healthy individuals. Furthermore, Lead is a non-competitive N-Methyl-D-aspartate receptor (NMDAR) antagonist. NMDA-Rs are ligand-gated receptors stimulated by glutamate and are vital players during neural development, neuronal plasticity, learning and memory, and LTP (66). Rocha et al. (66) summarized clinical and preclinical studies with various research techniques. They showed that low lead levels decreased cognitive functions and produced maladaptive behavior in human and animal models.

It is worth noting that lead exposure is a risk factor and a common association with Attention Deficit Hyperactive Disorder (ADHD) (67).

2.6. Personality trait

Lack of impulsivity affects sleep behavior, and McGowan and Coogan's (68) study showed that individuals with impulsivity suffered from shorter sleep duration, less efficient sleep, delayed sleep timing, and greater diurnal arousal. Social Anxiety Disorder (SAD) is a common finding in modern societies. It is believed that the way of thinking may affect the severity of this disorder. In clinical and experimental samples included in cross-sectional investigations, Gkika et al. (69) showed that negative beliefs and the dangerousness of thoughts were positively and significantly correlated with SAD.

Using metacognition models, McEvoy (70) stated that Meta-Cognitive Therapy (MCT) was associated with a better outcome in the anxiety and depression symptoms than the usual treatment.

Ronfeldt et al. (71) investigated the effects of third-wave cognitive constructs (mindfulness, psychological inflexibility, and meta-cognitions) on a person's psychological status, stating that mindfulness was inversely proportional to anxiety.

Additionally, after completing an online questionnaire at baseline and one year on 76% of 2291 participants from universities, Sun et al. (72) showed that negative meta-cognitions contributed to developing anxiety and paranoia.

Anxiety and depression symptoms are common in people with physical health conditions, with increased anxiety symptoms in cardiac and cancer patients (73) and diabetes (74). Sleep deprivation is considered a factor that affects mental health; Pires et al. (75) observed that significant anxiogenesis resulted from lack of sleep.

Interestingly, most sleepwalking or talking children also had an anxiety disorder (76). Sleep problems have several adverse health outcomes. Gould et al. (77) study on 109 adults aged 66-92 years revealed that the Geriatric Anxiety Scale (GAS) affective and somatic sub-scales were significantly associated with global sleep quality, suggesting that personalized treatment improved specific anxiety –symptom

domains or vice versa. A study by Nadorff et al. (78) showed that Generalized Anxiety Disorder (GAD) was significantly correlated with alarming dream frequency. Moreover, sleep loss affects brain regions, which are essential for decision-making, such as the prefrontal cortex.

Short and Weber (79) described an association between time of sleep and risk-taking, while Benard et al. (80) revealed that the proportion of suicides among patients with bipolar disorders (BD) was higher than in the general population, with more circadian rhythms and sleep disturbances compared to healthy control.

2.7. Autism

Autism spectrum disorder (ASD) has severe social communication impairments and restricted and repetitive behavior and interests (81). Grainger et al. (82) suggested that the metacognitive ability to express our mindset depended on the same processes that we could anticipate others' mindset ("mindreading"), which was impaired in ASD. They found that people with ASD had mindreading problems and could not precisely monitor their memory details and components. However, they showed that some people with ASD had superior metacognitive abilities relative to neurotypical people.

Furthermore, Griffin et al. (83) reported that adults with ASD had high levels of alexithymia, "a difficulty identifying and labeling one's emotional states". Autism is usually associated with a learning disability and lower levels of IQ, impacting all learning and severely impairing adaptive behavior compared to deficits in overall or general intelligence (84).

Adults with ASD hardly develop independent life with moderate mental capabilities (85). The metacognitive impairment in an ASD can be explained by understanding the representational nature of belief, even their own beliefs. They also have more difficulty understanding desire than neurotypical individuals (86). Additionally, individuals with ASD struggle with 'strategic' aspects of social learning, selecting the relevant and be-imitated information during imitation tasks (87).

Sleep problems are common lifelong comorbidity in ASD and can increase latency, sleep fragmentation, and circadian phase disorders (88, 89). This results from neurotransmitter abnormalities or other commonly associated comorbidities like epilepsy (90).

Sleep difficulties lead to negative consequences during the daytime, like physical aggression, irritability, inattention, hyperactivity (91), impaired cognitive performance, and quality of life (88).

2.8. Dyslexia

Dyslexic readers' abilities are lower than typically developing readers in all forms of self-reported metacognitive knowledge and reading motivation, with lower performance than typically developing readers. However, dyslexic children have normal vocabulary consolidation abilities and can recall novel words like normal children (92). Children with dyslexia have slow-wave sleep with a longer duration and higher frequency of sleep spindles, explaining the affected vocabulary consolidation (93). Carotenuto et al. (94) found that children with dyslexia showed higher rates of Sleep Disturbances Scale for Children (SDSC) pathological scores in the total SDSC score, higher rates in the number of times they woke up, nocturnal hyperkinesis, and snoring.

A recent study by Huang et al. (2020) (95) revealed that salivary melatonin in dyslexic children was less than the average children, and the rhythm of day low/night high disappeared.

2.9. Attention Deficit Hyperactivity Disorder (ADHD)

Attention deficit hyperactivity disorder (ADHD) is associated with academic retardation, the dysfunctional social interaction that results in a poor quality of life. ADHD in adulthood suffers from inattention, hyperactivity, impulsivity, and low academic achievement (96). ADHD management includes cognitive-behavioral therapy (CBT) and stimulant drug treatment with methylphenidate (97).

ADHD negatively impacts different aspects of cognition, including attention, executive functioning, memory, and learning. Objective psychometric tests may provide information about the individual's cognitive efficiency, whereas subjective self-reports might indicate success in individual goal achievement (98)

Gregory et al. (99) study on 2232 twin children associated ADHD with low sleep quality. Caregivers of children with ADHD develop sleeping problems due to their effort to care for them (100). Their children also develop sleep abnormalities resulting in dysfunction (101). Fortunately, poor sleep remits over time in ADHD, which may reassure parents and children with ADHD (99).

2.10. Neurodegenerative diseases

Alzheimer's disease (AD), the commonest dementia worldwide, is associated with sleep deprivation. In 18 longitudinal studies that included 246.786 subjects at baseline and 25.847 dementia cases after an average of 9.49 years of follow-up, patients with insomnia, sleep-disordered breathing, or other sleep disturbances were found to have higher risks of developing AD (102).

A post-mortem study by Lim et al. (103) on 45 older adults with a mean age at death of 89.2 years demonstrated that repeated sleep interruption caused a decreased number of hippocampal neurons in AD than normal controls.

Sleep abnormalities in Parkinson's Disease (PD) are common non-motor symptoms significantly impacting patients' quality of life (104) and precede any motor or cognitive manifestations (105). Abbott and Videnovic (106) hypnotized that circadian and sleep changes may strike the neurodegenerative process in PD that increases the sleep and

circadian abnormalities in a vicious circle pattern with more neurodegeneration and sleep disorders (107)

Sleep problem management may be a potential means to slow disease progression from the early stages of PD (108). Moreover, in PD, there is a dysfunction of circadian rhythm markers such as cortisol, melatonin, C-type natriuretic peptide amino-terminal (NT-proCNP), and Tumor necrosis factor-alpha (TNF-α), and assessment of these markers can be considered as markers for onset and stage of PD (109).

3. Management of dysfunctional metacognition and sleep disorders

3.1. Physical Exercise

A healthy diet and lifestyle play a crucial role in delaying aging health issues and maintaining an acceptable cognitive function level, especially in vulnerable seniors (110). A higher level of total daily physical activity is associated with a reduced risk for neurodegenerative diseases (111).

Regular exercise may play an essential role in the upregulation of brain-derived neurotrophic factor (BDNF) and upregulates the production of several neurotransmitters, like serotonin, associated with mood enhancement and reduced depressive symptoms (112).

Physical exercise positively impacts sleep in both normal conditions and the presence of sleep disorders; it enhances sleep quality and decreases the time to fall asleep (113). Moreover, physical exercise has a restorative property on the brain's prefrontal lobe, positively impacting sleep quality (114).

These exercise-induced actions may indirectly affect cognition as sleep improves neurotransmitters and neurotrophic systems such as; norepinephrine, serotonin, endorphins, BNDF (113), and melatonin (115). The systematic review and meta-analysis reported by Banno et al. (115) included nine studies with a total of 557 participants.

There is a positive correlation between physical exercise and academic performance with higher cognitive and metacognitive skills (116), and this is linked to the remapping of the brain as a part of brain plasticity that is improved and upgraded by physical exercise(117). Physical exercise performance may affect the brain through factors released into circulation with physical exercise.

The bone hormone Osteocalcin level is upregulated by physical exercise, and it is essential in the construction of bones and the regulation of blood sugar (118). Moreover, Osteocalcin affects sleep length, diurnal rhythm, brain signaling, and metacognition. It stimulates the secretion of serotonin, which influences mood and cognition.

The Osteocalcin is composed of a protein gel matrix made from Vitamin D repositories in the body. Therefore, individuals with Vitamin D deficiency are obese and have sleep problems because it is correlated to Osteocalcin's function and its impacts on sleep and body mass(119).

3.2. Omega-3

Omega-3 polyunsaturated fatty acids (PUFA n3) are neuroprotective agents because of their anti-inflammatory and anti-apoptotic actions. Furthermore, Omega-3 regulates the function of growth factors that influence synaptic plasticity and function (120). Omega-3 regulates membrane fluidity and gene expression (121). Low dietary Omega-3 causes impaired glutamate and monoamine synaptic function (122), contributing to depression, cognitive decline, or dementia, especially for AD. These effects of Omega-3 deficiency are related to manipulating the endocannabinoid and inflammatory molecules that result in microglia engulfment of hippocampal synapses. That explains the neuroprotective effect of Omega-3 and its attenuating action for cognitive decline in the elderly (119).

Oily fish is necessary for normal serotonin production and sleep control. Additionally, in 395 healthy children aged 7-9 years, 16 weeks of Omega-3 supplementation elevated the Omega-3 concentration of red blood cells and decreased the severity of obstructive sleep apnea with better sleep patterns (123). Oily fish also provide vitamin D that regulates the sleep-wake cycle. In a trial involving 677 people consuming oily fish, Del Brutto et al. (124) showed a link between dietary fish consumption and sleeping in a population where caught fish formed the primary dietary protein source.

Another study by Komori (125) treating depressed individuals with phosphatidylserine and O3PUFAs showed normalized salivary cortisol (circadian and basal secretion) in responders compared to non-responders. Chronic insomnia is associated with spikes in the release of cortisol (126). In support of the previous studies; Alzoubi et al.'s (127) eight weeks study on rats demonstrated the protective and antioxidant effects of Omega-3 on long and short memory in sleep deprivation.

3.3. Melatonin and light therapy

Dysfunction of the timekeeping system causes Circadian rhythm sleep-wake disorder (CRSWD), leading to the loss of synchrony and harmony between endogenous components of the circadian system and the external ques (128). It may be a primary disorder like circadian phase disorder or secondary to abnormal melatonin secretion in psychiatric or neurological diseases (129).

Various drugs are introduced for sleep disorders, like chloral hydrate, barbiturates, benzodiazepines, modafinil, antidepressants, and anxiolytics. However, these medications have unavoidable side effects, like daytime sleepiness, cognitive dysfunction, and dependency.

It is crucial to consider synchronizing the circadian components with the given therapy, for example, considering the time of melatonin or light therapy for circadian rhythm sleep disorders (128).

The wavelength determines the light therapy efficiency, the strength of the applied light, the time of application, and whether the person was previously exposed to light or not (130). The same light intensity may delay the sleep phase of the circadian cycle if administered before the core body temperature minimum or advance it if administered after it. For the same reasons, exogenous melatonin administration should also be timed by circadian phase for non-24 hours sleep-wake phase disorder. In blind individuals, Tasimelteon (a selective dual melatonin receptor agonist) showed promising results (128).

The pineal gland secretes melatonin into the blood exclusively in the dark following biological time. It is tolerated and has a lower risk of dependence than other sleep medications (131). Melatonin therapy for chronic insomnia is a safe and effective method to improve sleep onset, latency, duration, and quality. Exogenous melatonin stimulates the naturally secreted melatonin, binds to melatonin receptors, and triggers the target signaling pathways; hence, it treats insomnia (132). The pineal gland secretes melatonin into the blood exclusively in the dark following biological time. It is tolerated and has a lower risk of dependence than other sleep medications (131). Melatonin therapy in chronic insomnia is a safe and effective method to improve sleep onset, latency, duration, and quality (133); (134). Exogenous melatonin stimulates the naturally secreted melatonin, binds to melatonin receptors, and triggers the target signaling pathways; hence it treats insomnia (132).

Furthermore, melatonin ameliorates the complications caused by sleep-breathing disorders. The hypoxia-induced hyperglycemia in experimental studies for sleep apnea included 36 mice injected with IP melatonin for 21 days (135). It inhibits the expression of inflammatory cytokines (Tumor necrosis factor-alpha, Interleukin-6, and Cyclooxygenase-2) and fibrotic markers (PC1 and TGF-beta) (136).

On the other hand, melatonin may improve hypersomnolence's central disorder as it alters sleep architecture in narcolepsy, a disorder of circadian rhythm and REM sleep deficit. Changes in REM patterns in narcolepsy patients are like those seen in patients and animal models with the pineal gland removed. Additionally, melatonin's exogenous doses significantly increase REM sleep time in normal cohorts and patients with a central hypersomnolence disorder (132). Melatonin also relieves shift workers' sleepiness (137).

3.4. Tryptophan

A diet rich in Tryptophan and antioxidants improves affective and cognitive domains (138). Tryptophan plays a crucial role in protein synthesis. It is a precursor of biologically active compounds like serotonin, melatonin, quinolinic acid, kynurenic acid, tryptamine, and also coenzymes essential for electron transfer reaction (redox balance of metabolism), such as nicotinamide adenine dinucleotide (NAD^+) (139).

A deficiency in Tryptophan, caused by malnutrition, may affect the central and peripheral serotoninergic pathways, although different nutrition-derived hormonal molecules may rescue some of this deficiency (140). Serotonergic dysfunction has been related to panic, depression, aggression, and suicidality symptoms. Because the serotonin system is involved in various psychiatric disorders and is also involved in the regulation of satiety, it can be important in the pathophysiology of eating disorders such as anorexia nervosa.

Tryptophan is used to treat various disorders but has been withdrawn in most countries. During the treatment of tryptophan preparations, undesirable symptoms include various pulmonary, cutaneous, and neurologic symptoms, eosinophilia-myalgia syndrome, and disease-related muscle pain. Various diseases and disorders are linked with Tryptophan and its metabolites. Increased metabolism of Tryptophan, or undesirable effects of low Tryptophan, such as decreased absorption or intake and signs of depression and neurovegetative complaints, has been observed in different pathology types (141,142).

Murr et al. (143) measured serum concentrations of free Tryptophan and CRP in 1196 patients with coronary artery disease. Lower serum tryptophan levels in patients undergoing coronary angiography were predictive for higher total, cardiovascular, and non-cardiovascular mortalities. Thus, the increased risk in patients with major depression for developing cardiovascular disease, the inadequate response to treatment, and increased morbidity and mortality could relate to more significant disturbances of tryptophan metabolism (144).

The biosynthesis of the most important neurotransmitters for mood stability is serotonin, dopamine, epinephrine, norepinephrine, and nitric oxide (NO), which is achieved by enzymes tryptophan 5-hydroxylase, phenylalanine 4-hydroxylase, tyrosine 3- hydroxylase, nitric oxide synthase, and all these enzymes require BH4 as a cofactor (145).

BH4 is a strong reductant and therefore undergoes oxidation readily (146). Consequently, antioxidants prolong the life span of BH4 and contribute to increasing the activity of the BH4-dependent enzymes (147), and the biosynthesis of the mentioned neurotransmitters increases; this may explain why foods rich in antioxidants are considered as mood enhancers and improve cognitive abilities.

Tryptophan can be used for the management of sleep problems. It is interesting to note that dietary Tryptophan produces therapeutic effects through melatonin. Tryptophan treatment's crucial feature is that it does not directly reduce cognitive ability (148).

Mood and cognition are closely related in older persons (149) and linked to serotonin's biochemistry (150). Classical antidepressants like selective serotonin reuptake inhibitors (SSRIs) increase serotonin levels in the brain leading to enhanced postsynaptic neuronal activity (138).

A meta-analysis of several clinical trials observed a

precognitive effect of antidepressants in patients with major depressive disorder. Accordingly, antidepressants significantly affect psychomotor speed and delayed recall (150). A potential role of tryptophan depletion in cognitive ability has already been suggested and is further strengthened in patients with Alzheimer's dementia and Huntington's disease (151).

3.5. 5-hydroxytryptophan (5-HTP)

The density of serotonergic fibers increases in the hippocampus and decreases in the thalamic paraventricular nucleus (PVN) due to brain serotonin depletion. In contrast, the serotonin precursor 5-hydroxytryptophan (5-HTP) administration could rescue these defects following the reestablishment of brain 5-HT signaling (152).

A study that included 4-week-old mice considered that serotonergic systems affected sleep, and the combined GABA and 5-HTP had synergistic effects on sleep duration and quality (153).

Meloni et al. (154) revealed that scores of the Beck Depression Inventory (BDI-II) and the Hamilton Depression Rating Scale (HDRS21) showed substantially more significant improvement with 5- HTP. Supplementation for night terrors examined the influence of L-5-hydroxytryptophan on sleep terrors. More than half-night terror episodes have been observed in over 93% of children within a month. These results confirm that arousal levels might be positively influenced by treatment with L-5-hydroxytryptophan, resulting in reduced sleep terror behaviors in children (155). The trial of these authors included 45 children aged 3.2-10.6 years.

3.6. Magnesium and magnesium l-threonate (MgT)

Magnesium (Mg^{+2}) is a cofactor for many enzymes, and many organs need it to maintain proper function (156). Furthermore, Mg^{+2} is critical for numerous cellular processes, including enzymatic reactions, ion channel functions, metabolic cycles, cellular signaling, and biomolecules' stability, such as RNA, DNA, and proteins (157).

Mg^{+2} is essential for regulating the structural and functional synapses and synaptic plasticity (158). The intracellular concentration of Mg^{+2} positively correlates to synaptic branches' arborization (156).

Besides, Mg^{+2} regulates NMDA receptors (NMDAR) block, which is essential in controlling long-term potentiation and synaptic plasticity (156).

Several studies indicated that synaptic connections in the hippocampus decline during aging, with the degree of loss of synapses correlating with memory function impairment. The reduction seems to be specific to certain hippocampal subregions. Furthermore, in aging rodents, the dentate gyrus (DG) is the most affected area in the loss of synapses (159). Altering the hippocampus's synaptic efficacy is an initial event in cognitive disorders such as AD (160).

Magnesium deficiency and depression are linked in experimental and clinical studies (161, 162). However, prospective cohort studies failed to find an association between magnesium status and later risk of depression (163). Some intervention studies suggested a beneficial role of magnesium supplementation in treating depression (164), while others have not (165).

Moreover, Lai et al. (166) investigated children's cognitive functions and correlated the results to maternal blood zinc and magnesium levels during pregnancy. They revealed that deficiency of minerals during pregnancy was linked to the cognitive dysfunction of their children. A low Mg^{+2} intake has been associated with poor-quality sleep and inflammatory stress (161).

Magnesium l-threonate (MgT) consists of a magnesium ion and threonate, which exists physiologically within the brain. MgT significantly increases magnesium ion levels within the CSF (156) and improves memory recall and spatial memory (156).

Besides, MgT prevented reducing glutamatergic synaptic transmission under AD-like pathological conditions characterized by excitotoxicity (167). A study of the hypoxic zebrafish model assessed the effects of magnesium on cognitive functions. It showed that pre-treatment with MgT upregulated glutamate transporter EAAT4, improved neuronal survival, and maintained learning following hypoxia induction (168).

3.7. Probiotics, prebiotics, and synbiotics

The gut microbiome helps digestion, the immune system, and mental health (169). Enteroendocrine cells transmit signals from GIT to the brain via bacterial metabolites, hormones such as serotonin, and vagal afferent fibers (170). Roberts et al. (171) explained the role of the gut microbiome as a dynamic factor in the etiology of Alzheimer's disease by demonstrating metabolites from microbiota in the cerebrospinal fluid of Alzheimer's patients.

Chong et al. (172) did a randomized, double-blind, and placebo-controlled multicenter trial on 63 healthy elders over 65 years to study the effects of 12 weeks of probiotics on cognitive functions and mood. They demonstrated that probiotics promote mental flexibility and relieve stress in healthy older adults. This can encourage probiotic use in a healthy diet in adults.

Probiotics supply billions of beneficial bacteria directly and are derived from fermented food. Prebiotics are mainly fibers derived from vegetables, fruits, and cereals. They help the growth and multiplication of beneficial bacteria. Synbiotics are combinations of probiotics and prebiotics. These act by producing neurotransmitters such as gamma-aminobutyric acid (GABA), dopamine, acetylcholine, serotonin, and neurochemicals such as BDNF. All these modulate cognition and mood (173).

In addition, intestinal dysbiosis causes inflammation via the

microbiota-gut-brain axis. Five studies with an aggregated sample of 297 individuals (174) showed that probiotics benefit cognitive function in Alzheimer's disease and mild cognitive impairment subjects by decreasing inflammatory and oxidative markers.

Li et al. (175) clarified that probiotics, prebiotics, and synbiotics are very beneficial therapeutically in improving cognitive function and behavioral and psychological symptoms in patients with dementia.

3.8. Antioxidants

Although oxidative stress is critical in the pathogenesis of many cognitive diseases, some studies revealed that antioxidant therapy is clinically ineffective in these disorders.

Certain precautions are required to be effective clinically. Firstly, dose adjustment is required. Higher doses of antioxidants may be harmful (176). Secondly, some antioxidants have low solubility and absorption with rapid metabolism, so they require new delivery systems. Thirdly, advanced delayed damage cannot be reversed by antioxidants (177); thus, they should be given at earlier stages of the disease.

3.9. Herbs

Lavender

Aromatherapy is a method based on plant-derived essential oils. In 39 elders, a week's daily smell of lavender oil improved cognition and sleep disorders (178). Rafii et al. (179) showed the anxiolytic and sleep-promoting effects in burn patients with lavender and chamomile oil during massage.

Lavender (Lavandula angustifolia) essential oil (EO) is used in emotional disorders and consists primarily of linalool and linalyl acetate (180) and belongs to Lamiaceae's family (181). Linalool is a monoterpene with antioxidant, anti-inflammatory, and anti-convulsant activities, protects neurons from toxicity, and attenuates cognitive and affective disorders in the transgenic model of AD (182)

The anxiolytic action of lavender essential oil depends on its serotonin and glutamate-like characteristics (180) involved in the mechanisms for anxiety, learning, and reconsolidation of memory (183). However, lavender, coriander, and linalool's neuroprotective effects against Aβ- induced neurotoxicity in vitro cellular models are still unknown (7).

Coelho et al.'s (184) study demonstrated the inhibitory effect of vaporized lavender on the conditioned contextual fear memory without affecting the consolidation of tone fear memory, indicating lavender's potential use to manipulate the cognitive aspect of anxiety. Although memory updating depends on reconsolidation, the former process is a consequence of the latter, and the inhibition of memory updating does not necessarily imply impairment of the primary memory (185).

On the other hand, lavender oil reduces daytime sleepiness by improving sleep quality, thus improving mental functions (186), as shown in EEG changes in response to its antianxiety action (181). The Power Spectral Density of alpha and theta waves in EEG showed an increase in response to lavender inhalation, thus is associated with mood enhancement and calming effect (181, 187).

Chamomile

Chamomile is a common anxiolytic, sleep-inducing herb (188). Chang and Chen (189) studied the chamomile effect in postnatal women and showed that consumption of chamomile tea attenuated depression and sleep problems. However, these effects were noticed only for two weeks. On the other hand, Moss et al. (190) demonstrated that chamomile aroma impaired alertness and cognitive performance.

Ginseng

Ginseng has a therapeutic effect on improving cognitive function as learning and memory in neuroinflammatory diseases such as cerebral ischemia, stroke, traumatic brain injury, Parkinsonism, and Alzheimer's disease. It has anti-inflammatory and regenerative effects and improves cell-cell communication via the induction of neurogenesis and angiogenesis (191).

Curcumin

Preparations of curcumin with high bioavailability improve working cognitive function, memory, learning, and mood in healthy older individuals (192). Clinical results of Katherine et al. (193) showed improvement in hippocampal function, suggesting relief of cognitive decline in some populations.

Multiple systemic and central mechanisms are involved via various nutrients, including flavonoids (194).

3.10. Behavioral therapy

Cognitive Behavioral Therapy for insomnia (CBT-i) improved multiple aspects of sleep, like sleep latency, efficiency, and insomnia severity (195, 196).

Sweetman et al.'s (197) trial included 145 participants treated for six months and compared the clinical data (symptoms, signs, investigations, outcome) using Continuous Positive Airway Pressure (CPAP)-which is considered a treatment for sleep apnea- with or without Cognitive and Behavioral Therapy for insomnia (CBT-i) before initiating CPAP treatment. They showed better compliance with CPAP use in the CBT-i group and developed attenuated global insomnia, cognitive enhancement, and a positive effect on sleep impairment six months later.

Despite the supportive data on the benefits of CBT on sleep disorders, the effect on metacognition and cognition is still unclear; meta-cognition and cognition decrease in Meta-Cognitive Therapy (MCT) and Cognitive-Behavioral Therapy (CBT). Specifically, in a study on 74 patients, meta-cognition was more affected in MCT than CBT, but cognition did not show marked alternation (198).

3.11. Meditation

Yoga promotes the mind and self-regulation's meta-cognitive capacity, such as potentiating attention and memory, improving cognitive functioning scores, and sensory awareness. In mild cognitive impairment, patients who received the mindfulness-based stress reduction program showed better neuronal circuits of the cingulate cortex, medial prefrontal cortex, and left hippocampus (199).

Aging is linked with neural structure, function, and cognitive performance; they naturally decline over time. The study by Gard et al. (200) found that Yoga and meditation decreased the fall-off rate for fluid intelligence.

Afonso et al. (201) revealed that 21 elderly women yoga practitioners showed a significantly greater Cortical Thickness (CT) in a left prefrontal lobe cluster. The brain's Default Mode Network (DMN) has three operational modules: two occipital-parietal-temporal subnets and one frontal subnet. Moreover, Fingelkurts et al. (202) showed an enhancement of the frontal DMN module via body scan with repeated yoga meditation, focus on breath, and mantra repetition.

Gard et al. (200) stated that Yoga improved memory, concentration, attention, speed, and accuracy in math computations with less emotional lability, excitability, and aggressiveness. Furthermore, Yoga increased students' self-compassion, emotion regulation skills, and non-judgmental self-reflection. Additionally, Pozuelos et al. (203) showed that a relatively short mindfulness practice period significantly changed brain dynamics related to the internal monitoring of response conflicts and errors.

Furthermore, Xiao et al. (204) showed that 121 perimenopausal women involved in meditation training showed significant improvement in their scores on the Self-Rating Anxiety Scale (SAS) and Pittsburgh Sleep Questionnaire (PSQ). Varghese et al. (205) studied meditation's effect on patients with Type 2 Diabetes Mellitus (T2DM) and revealed a significant improvement in the PSQ score, sleep quality, duration, and function during daytime activities.

Another study on the impacts of Integrated Yoga (IY) Intervention on Sleep Quality among professional caregivers of older adults with AD showed significant sleep quality improvement after one month than baseline (206).

3.12. Acupuncture

Acupuncture is considered complementary medicine, usually used as an additional treatment. It has been reported to improve performance in several tests (Insomnia Severity Index, Sleep Efficiency, Total Sleep Time) (207) and the Hamilton Depression Scale (HAMD) (208).

Acupuncture also improved persistent sleep disturbance in mild Traumatic Brain Injury (mTBI) and Posttraumatic Stress Disorder (PTSD) (209) and reduced insomnia after stroke (210). Wang et al. (211) studied the effect of acupuncture with nimodipine versus nimodipine alone and presented the best scores in the Montreal Cognitive Assessment (MoCA) score in the acupuncture and the drug combination compared to acupuncture or drug alone.

Moreover, electro-acupuncture and body acupuncture combined with cognitive function training increased patients' Mini-Mental State Examination (MMSE) scores compared to the cognitive function training alone (212). Patients had better scores in the Mini-Mental State Examination (MMSE) and Montreal Cognitive Assessment (MoCA) compared to western medications (213).

The present review presented the common cause and comorbidities associated with sleep, cognition, and metacognition dysfunction. Some of these conditions are initiated by affecting either sleep or cognitive functions; others simultaneously hit both domains (sleep and cognition). Primary therapeutic tools to restore sleep and cognitive function have been presented as investigated by previous studies. We recommend further clinical and experimental studies to clarify more data on the missing parts of the molecular mechanism of action for some of the presented drugs and procedures, to evaluate the combination of these factors to assess if there is any synergistic effect, and to check these factors concerning broader and diverse populations (age or sex-based).

Conflict of interest

The authors declare that they have no conflicts of interest.

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