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Effect of Sleep Quality in Amyloid Plague Accumulation in Alzheimers

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Abstract: This study examined the effect of sleep quality in amyloid plague accumulation in alzheimers disease. The study mentioned that Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by cognitive decline, memory impairment, and the accumulation of amyloid-beta (Aβ) plaques in the brain. In carrying out this research, the following subheads were explored: concept of sleep, concept of sleep quality, concept of amyliod plague accumulation and concept of alzheimer's disease. The need for sleep quality as mentioned in the study included: for improved mood, for healthy heart and to regulated blood sugar. Similarly, the study mentioned the effects of sleep quality in amyloid plague accumulation in Alzheimer to include: impaired glymphatic clearance of amyloid-beta, increased tau protein accumulation and APOE-ε4 /sleep disruptions in alzheimer's risk. Furthermore, the study mentioned cognitive behavioral therapy for insomnia (CBT-I), melatonin supplementation and physical activity/ exercise how to improve sleep quality. The study concluded that emerging evidence highlights the crucial role of sleep quality in regulating amyloid-beta accumulation and the progression of Alzheimer's disease. One of the recommendations made was that public health initiatives should raise awareness about the importance of sleep in brain health.

Keywords: Sleep Quality, Amyloid Plague Accumulation and Alzheimers Disease.

Introduction

Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by cognitive decline, memory impairment, and the accumulation of amyloid-beta (Aβ) plaques in the brain. While the exact causes of AD remain elusive, growing evidence suggests that sleep quality plays a crucial role in the regulation of amyloid clearance (Astara, Tsimpolis, Kalafatakis, Vavougios, … and Lappas, 2024). Poor sleep has been linked to increased Aβ accumulation, which may contribute to the onset and progression of Alzheimer's disease. Understanding the relationship between sleep disturbances and amyloid pathology could offer new insights into preventive strategies for AD.

The human brain relies on sleep to clear metabolic waste, including toxic proteins such as amyloid-beta. During deep sleep, the glymphatic system—a waste clearance pathway in the central nervous system—becomes highly active, facilitating the removal of Aβ deposits (Kylkilahti, Berends, Ramos & Lundgaard, 2021). Studies indicate that individuals with chronic sleep deprivation or fragmented sleep exhibit higher levels of amyloid plaques, particularly in the hippocampus and prefrontal cortex, regions essential for memory and cognition (Cordone,

Annarumma, Rossini & De Gennaro, 2019). This suggests that disrupted sleep may accelerate neurodegeneration by impairing the brain's natural cleansing processes.

In addition to its role in waste clearance, sleep influences neuroinflammation, another key factor in AD progression. Sleep disturbances trigger an increase in pro-inflammatory cytokines, which can exacerbate neuronal damage and promote amyloid aggregation (Sadeghmousavi, Eskian, Rahmani and Rezaei, 2020). Furthermore, disrupted sleep cycles can alter the balance of neurotransmitters, such as acetylcholine and glutamate, which are vital for cognitive function. These findings highlight the bidirectional relationship between poor sleep and AD pathology, where sleep disturbances not only contribute to amyloid accumulation but are also worsened by neurodegeneration.

Research on sleep and AD suggests that improving sleep quality may serve as an early intervention strategy to slow disease progression. Behavioral modifications, such as maintaining a consistent sleep schedule, reducing exposure to blue light before bedtime, and managing stress, have been shown to enhance sleep efficiency and reduce amyloid burden. Additionally, pharmacological interventions targeting sleep disorders, such as insomnia or sleep apnea, may provide therapeutic benefits in preventing Aβ accumulation. Addressing sleep disturbances in midlife could be a crucial step in reducing AD risk later in life.

Despite these insights, several questions remain regarding the precise mechanisms through which sleep influences amyloid dynamics. While studies in animal models and human imaging research have provided compelling evidence, longitudinal studies are needed to establish causality and identify the most effective sleep interventions (Musiek & Holtzman, 2016). Additionally, genetic factors and lifestyle choices may interact with sleep patterns, further complicating our understanding of their impact on AD pathology. Future research should focus on developing personalized sleep-based strategies for Alzheimer's prevention.

Emerging evidence underscores the critical role of sleep quality in regulating amyloid-beta accumulation and, consequently, Alzheimer's disease progression. Poor sleep may not only increase the risk of AD but also accelerate cognitive decline through impaired waste clearance, neuroinflammation, and neurotransmitter imbalances. By prioritizing sleep health and developing targeted interventions, researchers and clinicians may uncover new avenues for AD prevention and treatment. Understanding the intricate link between sleep and neurodegeneration is essential in the fight against one of the most prevalent neurological disorders of the 21st century.

Concept of Sleep

Sleep is an extremely complicated process that consists of more than simply closing one's eyelids and counting sheep. It is an active state of unconsciousness produced by the body where the brain is in a relative state of rest and is reactive primarily to internal stimulus. The exact purpose of sleep has not been fully elucidated. Brinkman, Reddy and Sharma, (2023) Several prominent theories have explored the brain and attempt to identify a purpose for why we sleep, which includes the Inactivity theory, Energy conservation theory, Restoration theory, and the Brain plasticity theory.

Sleep is a normal body process that allows your body and brain to rest. At first glance, sleep is deceptively simple. For most people, it's just a matter of getting comfortable, closing your eyes and drifting into slumber. But despite how simple it seems, sleep is one of the most complex and mysterious body processes known to science. Sleep can be defined as "an active state of unconsciousness produced by the body where the brain is in a relative state of rest and is reactive primarily to internal stimulus.

Sleep is an important part of your daily routine—you spend about one-third of your time doing it. Quality sleep—and getting enough of it at the right times—is as essential to survival as food and water. Without sleep, you can't form or maintain the pathways in your brain that let you

learn and create new memories. Lack of sleep makes it harder to concentrate and respond quickly. According to Tubbs and Grandner, (2019) sleep is a naturally recurring and reversible biobehavioral state characterized by relative immobility, perceptual disengagement, and subdued consciousness.

Sleep is a state of reduced mental and physical activity in which consciousness is altered and certain sensory activity is inhibited. During sleep, there is a marked decrease in muscle activity and interactions with the surrounding environment. While sleep differs from wakefulness in terms of the ability to react to stimuli, it still involves active brain patterns, making it more reactive than a coma or disorders of consciousness.

Concept of Sleep Quality

Quality of sleep is defined as the ease of falling asleep and staying asleep each day, with poor quality of sleep known as insomnia. It is typically assessed by the time taken to fall asleep and the number of awakenings during the night, either through self-reporting or objective monitoring equipment. Study by Ellis and Ratnasingam (2018). The concept of sleep quality refers to the ease with which one is able to fall asleep and stay asleep each day. People who cannot fall asleep for hours after going to bed or who find themselves waking up repeatedly throughout the night are said to have a poor quality of sleep, also known as insomnia.

Nelson, Davis and Corbett, (2022). Defined sleep quality as an individual's self-satisfaction with all aspects of the sleep experience. Sleep quality has four attributes: sleep efficiency, sleep latency, sleep duration, and wake after sleep onset. Antecedents include physiological (e.g., age, circadian rhythm, body mass index, NREM, REM), psychological (e.g., stress, anxiety, depression), and environmental factors (e.g., room temperature, television/device use), and family/social commitments. Good sleep quality has positive effects such as feeling rested, normal reflexes, and positive relationships. Poor sleep quality consequences include fatigue, irritability, daytime dysfunction, slowed responses, and increased caffeine/alcohol intake.

Sleep quality is seen as one’s satisfaction of the sleep experience, integrating aspects of sleep initiation, sleep maintenance, sleep quantity, and refreshment upon awakening. Kline, (2013). Sleep quality is a vital construct to clinicians and researchers due to the high prevalence of disturbed sleep and insomnia, and the clear relevance of sleep quality to optimal health and functioning. The quality of your sleep ensures that you get the essential physical, mental, and emotional benefits you need from your slumber. Sleep quality is an important component of sleep health along with sleep duration, sleep satisfaction, and sleep consistency (regularity).

Several studies have examined correlations between sleep quality and how the individual feels immediately on waking and during the day. The results indicate that sleep quality is associated with ease of waking, tiredness, sense of balance and coordination, clear-headedness, how rested, restored and refreshed one feels, and mood and physical feelings on waking. During the day, feelings of tiredness predicted poorer sleep quality and alertness predicted better sleep quality. (Harvey, Stinson, Whitaker, Moskovitz and Virk, 2022).

Concept of Amyliod Plague Accumulation

Amyloid plaques are aggregates of misfolded proteins that form in the spaces between nerve cells. These abnormally configured proteins are thought to play a central role in Alzheimer's disease. The amyloid plaques first develop in the areas of the brain concerned with memory and other cognitive functions. According to Robertson, (2023) Amyloid plaques form when pieces of protein called beta-amyloid aggregate. The beta-amyloid is produced when a much larger protein referred to as the amyloid precurosr protein (APP) is broken down.

Amyloid plaques (also known as neuritic plaques, amyloid beta plaques or senile plaques) are extracellular deposits of amyloid beta (Aβ) protein that present mainly in the grey matter of the brain. Ballard, Gauthier, Corbett, Brayne, Aarsland and Jones, (2011). Some plaques occur in the brain as a result of aging, but large numbers of plaques and neurofibrillary tangles are

characteristic features of Alzheimer's disease. The plaques are highly variable in shape and size; in tissue sections immunostained for Aβ, they comprise a log-normal size distribution curve, with an average plaque area of 400-450 square micrometers (μm2). The smallest plaques (less than 200 μm2), which often consist of diffuse deposits of Aβ,

Amyloid plaques are compact, spherical extracellular deposits consisting of a small (4kDa) protein called the amyloid β-peptide (Aβ). Andrei, Vlassenko, Mintun, Xiong, Sheline, Goate, Phi, Tammie, Benzinger and Morris, (2012) Asserted that, Amyloid-beta (Aβ) accumulation was evaluated with two PIB PET scans about 2.5 years apart in 146 cognitively normal adults. Seventeen of 21 participants with initially elevated Aβ deposition demonstrated subsequent Aβ plaque growth (approximately 8.0% per year) and none reverted to a state of no Aβ deposits.

Study by Strauss, Pan and Sachpekidis, (2025). The most common cause of dementia is Alzheimer's disease (AD), which is a chronic neurodegenerative disease resulting from the deposition of amyloid plaques in the brain. Amyloid plaques block cell-to-cell signaling at synapses, a process which is essential for storing memories, processing thoughts and emotions, and planning. Hypothesis of Alzheimer disease (AD) posits that the accumulation of amyloidbeta (Aβ) peptide, typically aggregated in brain parenchyma as senile plaques is a primary pathogenic feature of AD. However, candidate “disease-modifying” drugs that target Aβ have yet to demonstrate efficacy in persons with symptomatic AD, even when it appears that the treatment may succeed in reducing cerebral Aβ burden.

Concept of Alzheimer’s Disease

Alzheimer’s disease is a condition that affects the brain. Symptoms are mild at first and become more severe over time. It is named after Dr. Alois Alzheimer, who first described the condition in 1906. Common symptoms of Alzheimer’s disease include memory loss, language problems, and impulsive or unpredictable behavior. One of the underlying biological changes of the condition is the presence of plaques and tangles in the brain. Another feature is a loss of connection between the nerve cells, or neurons, in the brain.

Alzheimer's disease is a type of dementia that affects memory, thinking and behavior. Symptoms eventually grow severe enough to interfere with daily tasks. Alzheimer's disease is a brain disorder that slowly destroys memory and thinking skills, and eventually, the ability to carry out the simplest tasks. In most people with Alzheimer's, symptoms first appear later in life. According to WHO (2023). Alzheimer's disease (AD) is a neurodegenerative disease that usually starts slowly and progressively worsens. It is the cause of 60–70% of cases of dementia.

Alzheimer's is a progressive disease, where dementia symptoms gradually worsen over a number of years. In its early stages, memory loss is mild, but with late-stage Alzheimer's, individuals lose the ability to carry on a conversation and respond to their environment. On average, a person with Alzheimer's lives four to eight years after diagnosis but can live as long as 20 years, depending on other factors. The most common early symptom is difficulty in remembering recent events. As the disease advances, symptoms can include problems with language, disorientation (including easily getting lost), mood swings, loss of motivation, self-neglect, and behavioral issue

Alzheimer's disease is the most common cause of dementia. Alzheimer's disease is the biological process that begins with the appearance of a buildup of proteins in the form of amyloid plaques and neurofibrillary tangles in the brain. This causes brain cells to die over time and the brain to shrink. About 6.9 million people in the United States age 65 and older live with Alzheimer's disease. Among them, more than 70% are age 75 and older. Of the more than 55 million people in the world with dementia, 60% to 70% are estimated to have Alzheimer's disease.

The roles of Sleep Quality in Health Promotion

Like eating nutritious food, drinking water, and exercising regularly, getting quality sleep is an important component of overall health. Although the exact reasons humans need to sleep remain

unknown, sleep experts agree there are numerous benefits to consistently getting a full night's rest. Most adults should get at least seven hours of sleep each night. While sleeping, the body performs a number of repairing and maintaining processes that affect nearly every part of the body. As a result, a good night's sleep, or a lack of sleep, can impact the body both mentally and physically.

- **Improved Mood**

Sleep restores the body and improves energy levels, so waking up well-rested can have a positive impact on an individual's mood. Cirelli, (2022). In contrast, people who get inadequate sleep are at higher risk of experiencing mental distress. Blackwelder, Hoskins & Huber, (2021) A chronic lack of sleep can lead to anxiety, depression, and irritability. However, developing a consistent sleep routine often resolves these symptoms.

- **Healthy Heart**

Quality sleep promotes cardiac health. During sleep, heart rate slows down, and blood pressure decreases. NCDC (2021). This means that during sleep, the heart and vascular system are able to rest. However, insufficient sleep is a risk factor for unwanted cardiovascular events. Lack of sleep causes blood pressure to remain high for an extended period of time, increasing the risk of heart disease, heart attack, and heart failure.

- **Regulated Blood Sugar**

Sleep impacts the body's relationship with the hormone insulin, which helps blood sugar, or glucose, enter the body's cells. The cells then use glucose as energy. Sleeping seven hours or more each night helps ensure blood sugar is regulated in the body. CDC (2020). Adults who get less than seven hours of sleep at night are at increased risk for Type 2 diabetes. Without enough sleep, the body's resistance to insulin increases because cells are not able to use insulin appropriately, which leads to too much sugar in the bloodstream.

- **Improved Mental Function**

Sleep is believed to help with memory and cognitive thinking. Brain plasticity theory, a major theory on why humans sleep, posits that sleep is necessary so the brain can grow, reorganize, restructure, and make new neural connections. Puderbaugh and Emmady, (2022). These connections in the brain help individuals learn new information and form memories during sleep. In other words, a good night's sleep can lead to better problem-solving and decision-making skills. A lack of sleep can have a negative impact on the ability to think clearly, form memories, learn well, and function optimally during the day. The ability to think quickly slows down after only a week of insufficient sleep

- **Restored Immune System**

Restorative theories of sleep suggest that sleep restores and repairs the body, making people feel refreshed in the morning. During sleep, the body produces growth hormones necessary for development in children and adolescents. These growth hormones also repair tissues and cells in people of all ages. The body also produces cytokines during sleep, which support the immune system in fighting infections. Inadequate sleep can impact the body's immune response to infection. Chronic sleep loss can make individuals more susceptible to common infections, such as a cold, while insufficient sleep over time can lead to a greater risk for immunodeficiency.

- **Stress Relief**

Getting appropriate sleep each night can help manage stress. When people wake up refreshed, they avoid the stressors that come with functioning while sleep-deprived, such as poor performance, difficulty thinking clearly, and lack of energy. Quality sleep can also reduce anxiety, depression, and other mental health strains related to stress.

➢ Maintaining Healthy Weight

Quality sleep, in addition to exercise, stress management, and healthy eating choices, is an important part. CDC (2021). of maintaining a healthy weight. During sleep, the body naturally produces more of an appetite suppressor, called leptin, while reducing production of the appetite stimulant ghrelin. On nights of too-little sleep, however, production of ghrelin increases and leptin decreases. As a result, a lack of sleep can lead to a greater feeling of hunger. (CDC, 2021).

➢ Athletic Performance

Sleep is a key element of athletic recovery and the body's production of growth hormones is highest during sleep. These growth hormones are necessary for the repair of tissue and likely contribute to muscle growth. Most athletes require eight hours of sleep each night for restoration and to avoid overtraining and improve their performance. Without sleep, athletes are at risk for lowered performance, fatigue, and changes in mood. Performing with less sleep also heightens the risk for injury. Watson, (2017). The potential for injury rises even more when an athlete's sleep time decreases and time spent training increases.

Effects of Sleep Quality in Amyloid Plague Accumulation in Alzheimer

Sleep quality plays a crucial role in brain health, particularly in the clearance of amyloid-beta (Aβ), a protein associated with Alzheimer's disease (AD). Poor sleep disrupts the brain's ability to remove toxic waste, leading to the accumulation of amyloid plaques—a key hallmark of AD. Research indicates that sleeps disturbances, including insomnia and sleeps fragmentation; accelerate neurodegeneration by impairing Aβ clearance and increasing tau protein accumulation. Understanding the effects of sleep quality on amyloid plaque buildup is essential for developing preventative strategies and therapeutic interventions to mitigate Alzheimer's progression.

✓ Impaired Glymphatic Clearance of Amyloid-Beta

The glymphatic system is a waste-clearance pathway in the brain, most active during deep sleep. Poor sleep reduces its efficiency, leading to amyloid-beta (Aβ) buildup—a hallmark of Alzheimer's. Research shows that chronic sleep disturbances impair cerebrospinal fluid flow, reducing Aβ clearance. Deep sleep disruption, especially of slow-wave sleep (SWS), accelerates neurodegeneration by allowing amyloid plaques to accumulate in critical areas such as the hippocampus and prefrontal cortex. Addressing sleep quality through interventions like cognitive-behavioral therapy and lifestyle changes may help slow Alzheimer's progression (Ding, 2024).

✓ Increased Tau Protein Accumulation

Beyond amyloid-beta, tau protein accumulation is a major driver of Alzheimer's disease. Sleep fragmentation and reduced deep sleep lead to increased tau phosphorylation, which contributes to neurofibrillary tangles. Research indicates that poor sleep quality raises tau levels in the cerebrospinal fluid, making neurons more vulnerable to degeneration. According to Decandia (2024), chronic sleep loss accelerates the spread of tau pathology across brain regions, worsening cognitive decline. Sleep interventions, such as melatonin supplements and behavioral therapy, may reduce tau accumulation and improve long-term neurological health.

✓ APOE-ε4 and Sleep Disruptions in Alzheimer's Risk

The APOE-ε4 allele is the strongest genetic risk factor for late-onset Alzheimer's disease. APOE-ε4 carriers experience greater sleep fragmentation, reduced slow-wave sleep, and higher amyloid deposition. Sleep disturbances may interact with APOE-ε4 by increasing neuroinflammation and impairing Aβ clearance. Longitudinal studies confirm that APOE-ε4 carriers with chronic sleep problems show faster cognitive decline than non-carriers. Personalized strategies, such as lifestyle modifications and circadian rhythm therapies, may mitigate sleep-related Alzheimer's risks in genetically susceptible individuals.

✓ **Sleep Deprivation Increases Beta-Amyloid Production**

Amyloid-beta (Aβ) levels fluctuate with the sleep-wake cycle. Sleep deprivation disrupts this cycle, leading to an increase in Aβ production and accumulation. PET imaging studies show that a single night of sleep deprivation results in significantly elevated Aβ levels in brain regions susceptible to Alzheimer's. Chronic sleep disruption exacerbates this effect, accelerating Aβ plaque formation. Emerging research highlights the potential of interventions like sleep-promoting medications and lifestyle changes (e.g., exercise, consistent bedtime) to reduce the impact of sleep loss on Aβ production (Pathmanathan, 2025).

✓ **Disrupted Circadian Rhythms and Amyloid Buildup**

Circadian rhythm disturbances are common in aging and Alzheimer's disease. The circadian system regulates sleep-wake cycles and influences the clearance of amyloid-beta (Aβ). Disruptions in circadian alignment—such as irregular sleep patterns, exposure to artificial light at night, or shift work—have been linked to increased Aβ accumulation. Melatonin, a natural sleep regulator, declines with age, further impairing Aβ clearance. Restoring circadian balance through interventions like bright light therapy and sleep scheduling may reduce Alzheimer's risk and improve cognitive function.

✓ **Sleep Deficits Reduce the Efficacy of Alzheimer's Treatments**

Anti-amyloid therapies, including aducanumab and donanemab, have shown potential in reducing amyloid plaque burden. However, poor sleep quality diminishes the effectiveness of these treatments. Sleep disturbances contribute to persistent neuroinflammation, impairing the brain's ability to clear amyloid plaques. Additionally, sleep disruption may lead to blood-brain barrier dysfunction, reducing drug delivery to affected brain regions. Clinical research suggests that optimizing sleep quality through non-pharmacological interventions and complementary sleep-promoting medications may enhance the effectiveness of Alzheimer's treatments (Ingala et al., 2020).

How to Improve Sleep Quality

Quality sleep is essential for overall health, cognitive function, and disease prevention. Poor sleep is linked to increased stress, impaired memory, and a higher risk of neurodegenerative disorders like Alzheimer's. Improving sleep quality involves lifestyle modifications, behavioral therapies, and medical interventions that regulate the sleep cycle and enhance restorative rest. Research highlights strategies such as cognitive behavioral therapy, melatonin supplementation, physical activity, and mindfulness to promote better sleep. Understanding and implementing these methods can help improve well-being, support brain health, and reduce the long-term risks associated with chronic sleep disturbances.

✓ **Cognitive Behavioral Therapy for Insomnia (CBT-I)**

Cognitive Behavioral Therapy for Insomnia (CBT-I) is a non-pharmacological intervention aimed at improving sleep patterns through behavioral changes. CBT-I is effective in treating chronic insomnia, reducing sleep onset latency, and improving sleep maintenance. It targets maladaptive thoughts about sleep and promotes good sleep hygiene. Studies have also linked CBT-I to improved cognitive function in older adults, making it a valuable tool for Alzheimer's prevention (Uquillas, 2024). Digital and in-person CBT-I programs have shown promising results in improving sleep quality.

✓ **Melatonin Supplementation**

Melatonin, a hormone that regulates the sleep-wake cycle, declines with age, leading to sleep disturbances. Supplementing with melatonin has been shown to improve sleep onset and duration, particularly in older adults. Clinical trials have demonstrated that melatonin not only enhances sleep efficiency but also exhibits neuroprotective properties, potentially reducing Alzheimer's risk. It helps regulate circadian rhythms and may mitigate the negative effects of

artificial light exposure at night. Melatonin supplementation should be carefully timed to avoid daytime drowsiness and optimize its effectiveness.

✓ **Physical Activity and Exercise**

Regular physical activity has been extensively studied for its positive effects on sleep quality. Exercise promotes deeper slow-wave sleep, reduces sleep latency, and decreases nighttime awakenings. Aerobic activities, such as walking, cycling, and swimming, have been particularly effective in improving sleep duration and efficiency. Individuals who engage in consistent physical activity report fewer sleep disturbances and lower rates of sleep apnea. Additionally, exercise plays a crucial role in reducing stress and anxiety, both of which are linked to poor sleep quality.

✓ **Bright Light Therapy for Circadian Rhythm Regulation**

Circadian rhythm disruptions contribute to poor sleep quality, especially in individuals with neurodegenerative disorders. Bright light therapy (exposure to bright artificial light in the morning) has been found to reset the body's internal clock, improving sleep onset and duration. Light therapy enhances alertness during the day and promotes deeper sleep at night. It is particularly beneficial for individuals with delayed sleep phase syndrome or shift work disorder. Consistent exposure to natural sunlight in the morning can also have similar beneficial effects.

✓ **Mindfulness and Meditation for Sleep Improvement**

Mindfulness-based stress reduction (MBSR) and meditation have been found to improve sleep quality by reducing stress and promoting relaxation. A study by Black & Slavich (2021) demonstrates that individuals who practice mindfulness report shorter sleep onset latency, fewer nighttime awakenings, and greater overall sleep satisfaction. Meditation reduces hyperarousal, a major contributor to insomnia, and enhances parasympathetic nervous system activity, which is crucial for sleep. Mindfulness interventions have been effectively used to treat both primary and secondary insomnia, making them a valuable, non-invasive sleep aid.

Conclusion

Emerging evidence highlights the crucial role of sleep quality in regulating amyloid-beta accumulation and the progression of Alzheimer's disease. Poor sleep increases the risk of AD by impairing waste clearance, triggering neuroinflammation, and disrupting neurotransmitter balance. Deep sleep enhances the brain's ability to remove toxic proteins, while sleep disturbances accelerate cognitive decline. Improving sleep through behavioral and medical interventions may serve as an effective strategy to slow AD progression. Although further research is needed, prioritizing sleep health can offer promising avenues for prevention and treatment. Understanding this connection is essential in combating Alzheimer's and preserving cognitive function.

Recommendations

1. Public health initiatives should raise awareness about the importance of sleep in brain health. Encouraging regular sleep schedules, reducing blue light exposure before bedtime, and managing stress can improve sleep quality, potentially reducing amyloid-beta accumulation and Alzheimer's risk.
2. Healthcare providers should integrate sleep assessments into routine check-ups, especially for individuals at risk of Alzheimer's. Early diagnosis and treatment of sleep disorders like insomnia and sleep apnea can help mitigate amyloid buildup and slow cognitive decline.
3. More longitudinal studies should be conducted to establish causal links between sleep quality and Alzheimer's progression. Investigating pharmacological and non-pharmacological interventions, such as cognitive-behavioral therapy for insomnia, may lead to new preventive and therapeutic strategies for AD.

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