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Pathway linking nutritional status to cognitive function in older adults with chronic diseases: exploring the mediating role of sleep quality

Abstract

Objective This study aimed to explore the pathway linking nutritional status to cognitive function in older adults with chronic diseases, and whether sleep quality potentially mediates their association.

Methods A cross-sectional study was conducted among 248 older adults with chronic diseases who were admitted in a district tertiary hospital, Shenzhen, China from January to December 2022. The participants completed the sociodemographic and clinical characteristics questionnaire, the Mini Nutritional Assessment Short Form (MNA-SF), the Montreal Cognitive Assessment (MoCA), and the Pittsburgh Sleep Quality Scale (PSQI). Linear regression and the bootstrap method were employed for data analysis.

Results We observed that older adults with chronic diseases showed notable cognitive decline, as indicated by a MoCA score (mean = 23.60, SD = 4.68). The direct effect analysis indicated that older adults with compromised nutritional status were more likely to experience cognitive decline ($\beta = 1.006$, $P < 0.001$). The mediation analysis revealed that sleep quality partially mediated the relationship between nutrition and cognitive function, accounting for 17.59% of the overall effect. Specifically, poor sleep quality may increase the risk of subsequent cognitive decline among older adults with compromised nutritional status.

Conclusions This study identified that the impact of nutritional status on cognitive performance was partially mediated by sleep quality in older adults with chronic diseases. Further supporting the importance of high-quality sleep in maintaining brain health during aging. Monitoring and enhancing nutritional status and sleep quality may contribute to mitigating the progression of cognitive decline.

Keywords Older adults; Cognitive function; Nutrition; Sleep; Mediating role

1.Introduction

Slight cognitive decline is prevalent among older adults, and without appropriate management, it may progress to mild cognitive impairment or even dementia, thereby jeopardizing their ability to perform activities of daily living and social functioning. [1] As a serious impediment to healthy aging, cognitive impairment has been recognized as a considerable public health issue, as well as overwhelming financial burden on health-care systems and families. The surveillance data reported that approximately 75% of the Chinese population aged 60 or above suffered from at least one chronic disease. [2] It is estimated that the prevalence of cognitive impairment in older adults with chronic diseases is up to 35.02%. [3] The risk of cognitive impairment is greatly increased in older adults with chronic diseases due to age-related degenerative changes, long-term disease interference, or deterioration of illness. This increased risk leads to greater susceptibility to a variety of adverse clinical outcomes, creating a vicious cycle that severely constrains the quality of life for this population. [4, 5] Hence, it is crucial to pay specific attention to cognitive impairment in older adults with chronic diseases at the earliest possible stage. Effectively delaying or mitigating cognitive decline may enhance health outcomes in later life, highlighting the urgent need to identify and address potentially modifiable risk factors that can help reduce cognitive impairment.

Over recent decades, the intricate connection between nutritional status and cognitive function has garnered significant attention for both academics and practitioners. Nutritional health is the result of the balance between nutrient intake and requirements, characterized by its multifactorial nature, which is related to physiological functioning, psychological conditions, and social factors. [6] Malnutrition is a significant nutritional health issue in older adults with chronic diseases, often accompanied by compromised physical and mental health, which contributes to a range of adverse clinical outcomes. [7] It is commonly observed that nutritional deficiencies are often associated with lower cognitive performance in older adults, particularly when individuals identified as malnourished face a heightened risk of cognitive impairment. [8] For example, Soest APM et al, [9] based on a cohort study, found that each point increase in nutrient status index, consisting of homocysteine, vitamin D and n-3 PUFAs, was associated with a 50% higher risk of dementia. Furthermore, Kishino Y et al [10] observed that nutritional deficiency increased the behavioral and psychological symptoms of dementia in those with MCI and early-stage Alzheimer's disease, especially emotional disinhibition and verbal aggressiveness. In addition to the aforementioned research, studies have pointed out that the impact of nutrition on brain function is reversible and that nutritional interventions can improve cognitive performance. This suggests that improving nutritional status may be a potentially modifiable factor for delaying cognitive impairment. [11] Given this context, it is indispensable to explore positive factors that may help mitigate the detrimental effects of malnutrition on cognitive functioning.

Sleep is a fundamental physiological function essential to human biology. A good night's sleep facilitates waste clearance in the brain, supports the maintenance of synaptic plasticity, and ensures the normal functioning of the nervous system. [12] Remarkably, sleep disturbance can result in a heightened risk of deterioration in age-related chronic diseases and a greater need for frequent medical visits. [13] Previous studies have demonstrated that sleep disturbance accelerates age-related cognitive decline and the development of dementia, as well as contributes to increased mortality. [14] Wong R et al [15] found a connection between sleep-maintenance insomnia and sleep-medication usage prior to cognitive changes, and a higher incidence of subsequent dementia. Meanwhile, research has revealed that sleep abnormalities are strongly associated with compromised nutritional status. Individuals with compromised nutritional status are more prone to irregular sleep-wake cycle, insomnia, daytime dysfunction, and poor subjective sleep quality. [16, 17] While earlier studies have identified a connection between nutritional status, sleep quality, and cognitive function, the specific mechanisms underlying this relationship have not been adequately reported. Thus, there remains a need to gain a deeper understanding of the potential linkages among them.

Given the existing literature, we hypothesized that various dimensions of sleep quality could mediate the relationship between nutritional status and cognitive function in older adults with chronic diseases. To test this hypothesis, this study aimed to (1) investigate the pathway linking nutritional status to cognitive function in older adults with chronic diseases and (2) examine whether sleep quality and its dimensions mediate this association. Our findings are expected to provide valuable insights into how nutritional status impacts cognitive function. Additionally, clarifying the potential role of sleep quality in the relationship between nutritional status and cognitive function will offer important perspectives for clinical applications aimed at preventing and delaying cognitive impairment.

2. Method

2.1 Research design and participants recruitment

This study utilized a cross-sectional design. Participants were recruited from a district tertiary hospital in Shenzhen, Guangdong Province, China, from January to December 2022. Participants were included if they: (1) were aged $\geq$ 60 years; (2) had confirmed at least one chronic disease that meets the WHO diagnostic criteria; [18] and (3) did not have significant communication barriers. Exclusion criteria were: (1) a diagnosis of Alzheimer's disease or other types of dementia; (2) a history of prior mental disorders; (3) malignancy; and (4) being in the acute or terminal phase of the disease. The researchers performed face-to-face assessments with participants and recorded their responses in detail. The sample size was calculated using the G*Power 3.1.9 [19] based on a medium effect size (effect size of 0.15, alpha of 0.05, power of 0.95) with 11 associated predictors, resulting in a minimum of 178 participants. In order to improve the accuracy and robustness of the mediation model and to account for an anticipated ineligibility rate of 20%, the minimum sample size was expanded to 223. Ultimately, 248 participants were included.

2.2 Ethics approval

This study protocol was approved by the Ethics Committee of Huazhong University of Science and Technology Union Shenzhen Hospital (approval number: KY-2022-040-01). Each participant signed a written consent form prior to assessment, and all data collected were kept strictly confidential.

2.3 Measures

2.3.1 Sociodemographic and clinical characteristics

Data on age, gender, marital status, education, living arrangements, number of chronic diseases, history of smoking, history of alcohol consumption, and Body Mass Index (BMI) were collected using a structured questionnaire specifically developed for this study.

2.3.2 Nutrition

Nutritional status was evaluated using the Mini Nutritional Assessment Short Form (MNA-SF), [20] which consists of 6 items: motility, neurological problems, BMI or calf circumference, food intake, weight loss, and acute diseases or psychological trauma. The total MNA-SF score ranges from 0 to 14 and is classified as follows: well-nourished (≥ 12), at risk of malnutrition (8-11), and malnourished (≤ 7). The MNA-SF has been shown to be a reliable tool for assessing nutritional status in Chinese older adults, with a Cronbach's alpha of 0. 0.771 for its internal consistency. [21] For the statistical analyses in this study, the MNA-SF score was treated as a continuous variable.

2.3.3 Cognitive function

Cognitive function was assessed using the Montreal Cognitive Assessment (MoCA), a widely used tool for cognitive function screening. [22] The MoCA evaluates 7 cognitive domains as follows: visuospatial and implementation abilities, naming, attention, verbal fluency, abstraction, delayed recall, and orientation. The total score of the MoCA ranges from 0 to 30, with a score of 26 or higher indicating normal cognitive function. A higher score suggests better cognitive function. It demonstrated good internal consistency reliability, with a Cronbach's alpha of 0.807, in previous research on screening for mild cognitive impairment in Chinese older adults. [23]

2.3.4 Sleep quality

Sleep quality was examined using the Chinese version of the Pittsburgh Sleep Quality Index (PSQI). The Chinese version of the PSQI demonstrated good internal consistency reliability, with a Cronbach's alpha of 0.842. [24] It comprises 18 self-rated items organized into 7 domains: subjective sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, sleep-medication usage, and daytime dysfunction. Each domain is scored on a scale of 1 to 3, and total score greater than 7 is indicative of poor sleep quality. When the cut-off point is 7, the sensitivity and specificity are 98.3% and 90.2%. [24] A higher PSQI score indicates worse sleep quality.

2.4 Statistical analyses

The data were analyzed using IBM SPSS 25.0. Descriptive statistics were presented as frequencies (percentages) for categorical variables and as means ± standard deviations (SD) for continuous variables. Initially, we utilized Pearson's correlation method to analyze the associations among nutritional status, sleep quality, and cognitive function. Subsequently, after controlling for confounders such as age, gender, and the number of chronic diseases, we performed a three-tiered hierarchical multiple regression analysis using SPSS PROCESS macro (version 3.4, model 4) to explore the mediating pathway of sleep quality between nutritional status and cognitive function.

In the first step, linear regression analysis was used to test the association between nutritional status and cognitive function. In the second step, linear regression analysis was performed again to examine the relationship between nutritional status and sleep quality. In the third step, linear regression analysis was conducted one more time to further explore the relationship between nutritional status and cognitive function, including sleep quality as a mediating variable. Finally, the extent of the mediation effect was examined, and the 95% confidence intervals (CI) for the direct, indirect, and total effects were estimated using the Bootstrap method with 5000 resamples. Each bootstrap sample was generated by randomly selecting participants with replacement, and the mediation effect was estimated for each sample. The 95% CI for the mediation effect was calculated based on the bootstrap distribution, providing robust evidence for the significance of the mediation effect. [25] A statistically significant mediating role was indicated if the 95% CI for the indirect effect excluded 0. All tests were two-sided, and statistical significance was set at $p < 0.05$.

3. Results

3.1 Characteristics of the participants

The characteristics of the 248 participants were displayed in Table 1. The mean age was 71.56 years (SD = 8.59), with a range between 60 and 95 years. 50.4% were men, 12.1% lived alone, and more than half (65.7%) were married. In addition, 66.1% of the participants with less than a high school education. The total score of cognitive function, nutritional status, and sleep quality was 23.60 ± 4.68, 11.57 ± 1.92, and 8.95 ± 3.48, respectively.

3.2 Correlations between nutritional status, sleep quality, and cognitive function

Pearson correlation analysis indicated that nutritional status had a positive correlation with cognitive function ($r=0.528$, $P < 0.001$), and sleep quality was significantly negatively associated with nutritional status ($r=-0.498$, $P < 0.001$) and cognitive function ($r=-0.424$, $P < 0.001$). In addition, cognitive function was significantly negatively correlated with six dimensions of sleep quality. Surprisingly, cognitive function was not associated with sleep-medication usage (Table 2).

3.3 linear regression analysis for the correlation among nutritional status, sleep quality, and cognitive function

To further explore the correlation between nutritional status, sleep quality, and cognitive function, we conducted a three-tiered hierarchical multiple regression analysis. Step 1: the significant impact of nutritional status on cognitive functioning was clarified, with the deeper the impairment of nutritional status in older adults with chronic diseases, the worse the integrity of cognitive function ($\beta=1.006$, $F=19.647$, $P < 0.001$), revealing that the total effect was valid. The interpretative power of the model was 45.3%. Step 2: it was confirmed that nutritional status had a significant influence on sleep quality ($\beta=-0.811$, $F=9.438$, $P < 0.001$). The interpretative power of the model was 28.5%. Step 3: when nutritional status and sleep quality concomitantly predicted cognitive function, the regression coefficient of nutritional status on cognitive function was reduced by 0.177 (β=0.829, F=19.183, P < 0.001). The interpretative power of the model was 47.2%. See table 3 for details.

3.4 Sleep quality is a mediator between nutritional status and cognitive function

The structural associations of sleep quality and its dimensions between nutrition and cognitive function were illustrated in Fig. 1. Sleep quality mediated the relationship between nutrition and cognitive function, with a mediating relative effect value of 17.59%. Among the dimensions of sleep quality, subjective sleep quality had the highest mediating relative effect value (14.71%), followed by daytime dysfunction (12.92%), sleep disturbances (11.83%), and sleep duration (10.04%). Nevertheless, sleep latency and sleep efficiency were not mediators between nutrition and cognitive function. Detailed information was summarized in Table 4.

4.Discussion

Against the background of the accelerating process of global population aging, it is a priority to identify factors that can delay or reverse age-related cognitive decline. Given the potentially modifiable effects of nutritional health on the brain's cognitive functions, as well as the protective impact of high-quality sleep on neurological function, this study examines, for the first time, the pathway linking nutritional status to cognitive function in Chinese older adults with chronic diseases, utilizing sleep quality as a feasible mediator. The findings confirmed that better overall sleep quality may buffer the association between compromised nutritional status and cognitive decline. The most surprising result was that the dimensions of sleep quality, specifically sleep medication usage, sleep latency, and sleep efficiency, did not mediate the link between nutritional status and cognitive function. In other words, the absence of sleep medication usage, shortened sleep latency, or improved sleep efficiency did not prevent cognitive impairment. This finding was not entirely consistent with earlier studies, [14] but there was no essential conflict. This might be due to the following reasons. Firstly, awareness of sleep hygiene among Chinese older adults is limited, with over half of poor sleepers believing that regular sleep medication usage disrupts sleep structure or is ineffective. [26] Additionally, only a small number of participants in this study used sleep medication to address their sleep disorders, which may not have provided enough variability within the sample. Consequently, the survey data were too limited to provide a definitive assessment of the link between sleep medication usage and cognitive function. Thirdly, there was wide inter-individual variability in the correlation of overall sleep duration with sleep latency and sleep efficiency. If sleep latency and sleep efficiency do not negatively affect sleep architecture, self-reported trouble falling asleep and inefficient sleep may not meaningfully reduce an individual's sleep quality, regardless of their expectations.

The study revealed that compromised nutritional status might be a risk factor for cognitive decline in later life, which was in line with the findings of previous research. [9, 27, 28] Growing evidence suggests that nutritional screening factors such as anorexia, weight loss, oral function, BMI, and physical mobility are strongly associated with cognitive function. [9, 27, 29] A plausible explanation is that numerous nutrients play a critical role in maintaining brain energy metabolism and brain tissue integrity, potentially delaying or resisting pathological changes in the brain. [27] On the other hand, some scholars have observed that insufficient energy intake may cause nerve cell damage and central nervous system disorders. [30] In addition, malnutrition can induce neuronal energy deficiency, synaptic dysfunction, and accelerated neuronal apoptosis, which may in turn lead to thinning of the cerebral cortex and the development of neurological diseases. [27, 31] Nutritional deficiencies also promote inflammatory responses in central and peripheral immune cells and interfere with circulating factors that regulate brain function. [28] In contrast to existing research, this study focused on older adults with chronic diseases. These individuals may experience long-term disease interference, which can affect appetite, nutrient intake, and consequently, the rate of cognitive decline. [32] At present, the concept of brain-healthy nutritional interventions in clinical settings is relatively lagging because of insufficient awareness among healthcare professionals and delays in accessing the latest relevant evidence and technology. In this context, considering the findings of this and previous studies, standardizing nutritional screening is essential for older adults with chronic diseases upon hospital admission. It should also be a dynamic assessment over the course of hospitalization for early identification of nutritional risk and the development of an appropriate nutritional management plan. It is worth noting that, although cognitive impairment is a prevalent geriatric syndrome without a curative treatment, some modifiable factors contributing to cognitive impairment can be addressed (e.g., anemia, Vitamin B12 and D deficiency, depression, social isolation, etc.). [1,8] Thus, intervention strategies targeting nutritional issues can be beneficial choice for delaying cognitive decline.

A number of mechanisms underlie the link between nutritional status and cognitive function. We demonstrated that sleep quality could mediate the association between nutritional status and cognitive function in older adults with chronic diseases, with the mediating effect accounting for 17.59% of the overall effect. This finding provides new evidence supporting existing studies that indicate compromised nutritional status may negatively affect subsequent cognitive performance through reduced sleep quality. Several possible mechanisms might explain how compromised nutritional status affects sleep quality. Firstly, malnutrition could lead to cerebral white matter damage and neurodegeneration, thereby impairing the regulatory functions of the brain. [33,34] Secondly, insufficient intake of tryptophan results in reduced melatonin synthesis. [35] Finally, malnutrition-related inflammation may cause white matter hyperintensities (WMHs), which have been found to contribute to insomnia by impairing the integrity of the tracts between the left thalamus and the pars triangularis. [33,36] Furthermore, previous studies have linked poor sleep quality to an increased risk of cognitive impairment. [14, 37] Sleep disturbances lead to decreased clearance of amyloid-β and tau proteins, which serve as pathological markers of cognitive impairment, and induce neuroinflammation. [38, 39] Another possible reason is that older adults with sleep problems are more prone to feelings of helplessness, adversely affecting cognitive function due to a lack of available internal and external resources. [15] Thus, poor sleep quality might increase the risk of subsequent cognitive impairment among older adults with inadequate nutrition.

The majority of participants in this study were at risk of malnutrition. Face-to-face assessments indicated that their nutritional screening scores might be lower than their actual scores. Firstly, energy expenditure increases during the acute episode of chronic diseases, impairing the overall nutritional status of older adults to varying degrees. Secondly, the acute episode of chronic diseases and relevant treatment activities may partially limit physical mobility. Given these factors, our study may overestimate the link between nutritional status and cognitive function while underestimating the mediating role of sleep quality.

The findings of this study provide a hypothetical mechanism for how nutritional status might contribute to cognitive decline. Nevertheless, sleep quality is unlikely to be the only mediator between nutritional status and cognitive function due to the diversity of nutritional screening indicators. The study of cell molecular mechanisms has demonstrated that lifestyle can mediate the role of nutrient-sensing pathways in human cognitive aging. [40] Other nutrition-related parameters, such as depression and perceived stress, may also influence this process and require further in-depth exploration.

Another theoretical implication of this study is the potential to slow or even reverse cognitive decline in later life. We confirmed that sleep quality partially explains the association between nutritional status and cognitive function, indicating that older adults with both reduced sleep quality and compromised nutritional status may exhibit poorer cognitive performance than those with compromised nutritional status alone. In light of these findings, sleep management should be targeted for early prevention and treatment of cognitive decline in older adults with chronic diseases, particularly those at risk of malnutrition.

For individuals suffering from compromised nutritional status combined with reduced sleep quality, it is essential to enhance the standardized management and treatment of nutritional issues. Additionally, a systematic assessment of the severity, phenotype, and nature of sleep disorder should be conducted. Effective self-reporting strategies, such as the PSQI scale, can be adopted for sleep monitoring. Furthermore, sleep hygiene education should be reinforced, and symptoms of sleep disorders should be treated through effective sleep medication and non-pharmacological intervention strategies, such as cognitive behavioral therapy, resistance training, and music interventions. [41, 42]

5. Limitations

The findings offer new insights into the effects of nutritional status on cognitive function, enriching our understanding of the connection between nutritional health and aging. Nevertheless, several limitations of this study must be acknowledged. First, it focused on older adults admitted to a district tertiary hospital, resulting in a sample that may not be representative of the broader population. Second, sleep duration, sleep latency and weight change were self-reported by participants, which may introduce recall bias. Third, the MNA-SF scale used in this study only provides a rough estimate of individuals' nutritional status; future research should incorporate nutrition-related biomarkers for a more comprehensive assessment. Finally, this study examined only one mediating variable, highlighting the need for further exploration of additional factors associated with cognitive function in older adults with chronic diseases.

6. Conclusions

The mediator model in this study established a pathway from nutritional status to cognitive function, partially mediated by sleep quality. The findings indicated that nutritional status not only directly affects cognitive function among older adults with chronic diseases, but also indirectly influences it by affecting sleep quality, underscoring the importance of high-quality sleep in maintaining brain health during aging. Hence, this study may contribute to a better understanding of the mechanisms underlying the development of cognitive impairment. A favorable nutritional status may reduce the risk of sleep disorder and, consequently, cognitive decline. Implementing sleep hygiene strategies within the framework of nutritional management may serve as a cost-effective public health measure to mitigate cognitive decline.

Declaration of Generative AI and AI-assisted technologies in the writing process

There was no use of generative AI or AI-assisted technology in the writing process.

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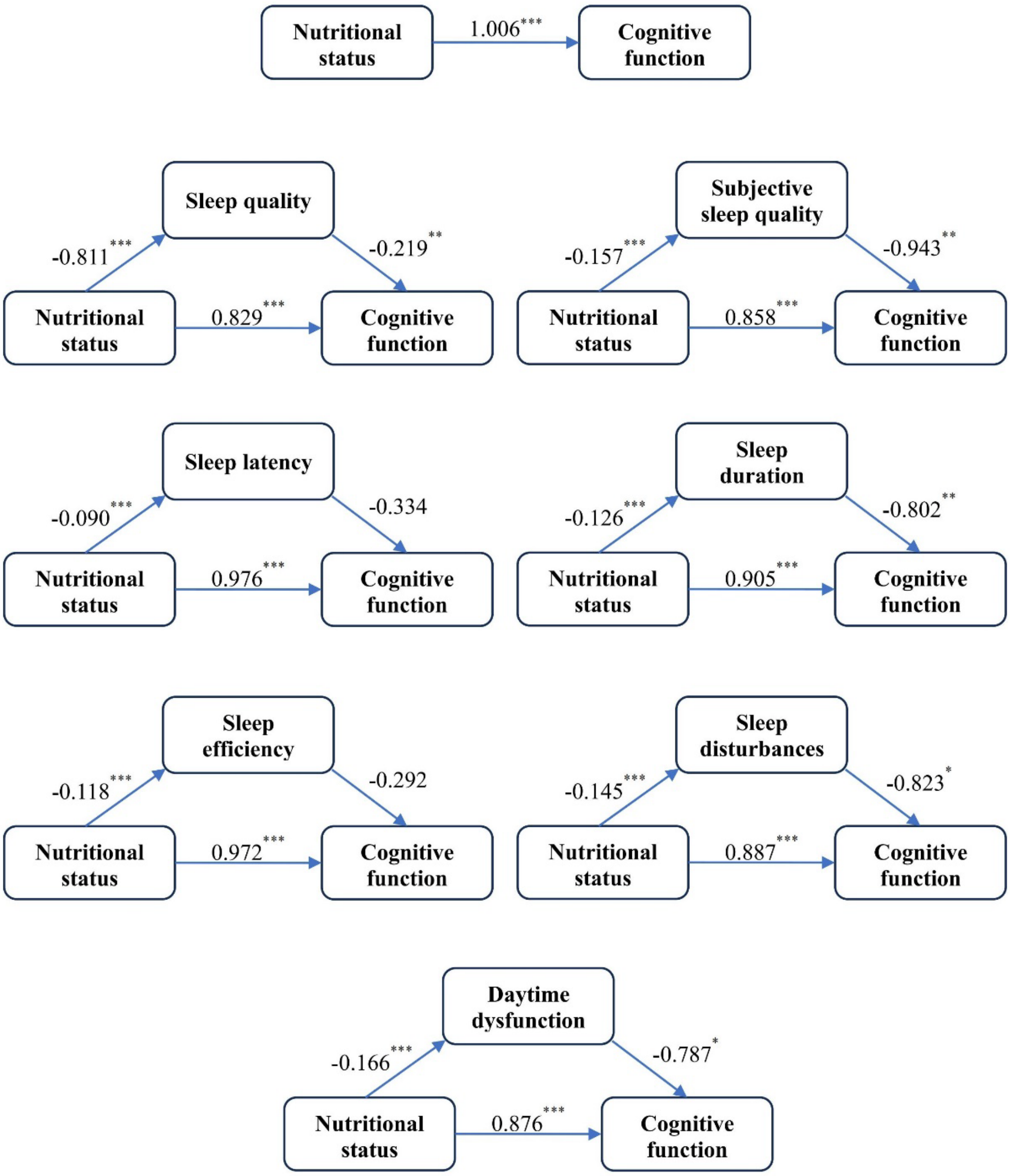

Fig. 1 A model diagram of sleep quality and its six dimensions mediated nutritional status and cognitive function. (Note: ***$p < 0.001$, **$p < 0.01$, *$p < 0.05$.)

Table 1 Characteristics of participants (N=248)

Variables	Mean ± SD or n (%)
Age (years)	71.56 ± 8.59
60-69	108 (43.5)
70-79	89 (35.9)
≥80	51 (10.6)
Gender	
Male	125 (50.4)
Female	123 (49.6)
Marital status	
Married	163 (65.7)
Divorced or widowed	85 (34.3)
Education	
Primary school and below	61 (24.6)
Junior school	103 (41.5)
High school	57 (23.0)
University degree and above	27 (10.9)
Living alone	
Yes	30 (12.1)
No	218 (87.9)
No. of chronic disease	
1	68 (27.4)
2	115 (46.4)
≥3	65 (26.2)
History of smoking	
Yes	87 (35.1)
No	161 (64.9)
History of drinking	
Yes	102 (41.1)
No	146 (58.9)
BMI	23.87 ± 3.21
Cognitive function	23.60 ± 4.68
Nutritional status	11.57 ± 1.92
Sleep quality	8.95 ± 3.48

Table 2 Correlations between nutritional status, sleep quality, and cognitive function

Variables	1	2	3	4	5	6	7	8	9	10
Nutritional status[1]	1.000									
Sleep quality[2]	-0.498***	1.000								
Subjective sleep quality[3]	-0.421***	0.865***	1.000							
Sleep latency[4]	-0.313***	0.644***	0.533***	1.000						
Sleep duration[5]	-0.387***	0.778***	0.665***	0.418***	1.000					
Sleep efficiency[6]	-0.349***	0.762***	0.602***	0.444***	0.592***	1.000				
Sleep disturbances[7]	-0.383***	0.734***	0.549***	0.362***	0.423***	0.438***	1.000			
sleep-medication usage[8]	-0.103	0.258***	0.182**	0.125*	0.061	0.060	0.174**	1.000		
Daytime dysfunction[9]	-0.406***	0.736***	0.566***	0.334***	0.424***	0.390***	0.565***	0.225***	1.000	
Cognitive function[10]	0.528***	-0.424***	-0.368***	-0.229***	-0.350***	-0.252***	-0.342***	-0.113	-0.365***	1.000

Note: ***p < 0.001, **p < 0.01 (two-tailed).

Table 3 Mediating effects of sleep quality between nutritional status and cognitive function pathway model analysis

Regression equation		Global fit index			Significance of regression coefficient	
Outcome variable	Predictor variable	R	R^2	F	β	t
cognitive function	Nutritional status	0.673	0.453	19.647***	1.006	7.376***
sleep quality	Nutritional status	0.534	0.285	9.438***	-0.811	-7.003***
cognitive function	Nutritional status	0.687	0.472	19.183***	0.829	5.618***
	sleep quality				-0.219	-2.900**

Note: ***p < 0.001, **p < 0.01 (two-tailed).

Table 4 Mediating effect values of sleep quality and its dimensions

Path	Effect	Beta	Boot SE	95%CI	Relative effect value (%)
Nutritional status →Sleep quality →Cognitive function	Total effect	1.006	0.136	0.738,1.275	17.59%
	Direct effect	0.829	0.148	0.538,1.120	
	Indirect effect	0.177	0.078	0.031,0.338	
Nutritional status →Subjective sleep quality →Cognitive function	Total effect	1.006	0.136	0.738,1.275	14.71%
	Direct effect	0.858	0.144	0.576,1.141	
	Indirect effect	0.148	0.066	0.029,0.286	
Nutritional status →Sleep latency →Cognitive function	Total effect	1.006	0.136	0.738,1.275	NA
	Direct effect	0.976	0.142	0.697,1.256	
	Indirect effect	0.030	0.040	-0.047,0.113	
Nutritional status →Sleep duration →Cognitive function	Total effect	1.006	0.136	0.738,1.275	10.04%
	Direct effect	0.905	0.140	0.629,1.182	
	Indirect effect	0.101	0.046	0.016,0.197	
Nutritional status →Sleep efficiency →Cognitive function	Total effect	1.006	0.136	0.738,1.275	NA
	Direct effect	0.972	0.141	0.694,1.250	
	Indirect effect	0.034	0.043	-0.052,0.123	
Nutritional status →Sleep disturbances →Cognitive function	Total effect	1.006	0.136	0.738,1.275	11.83%
	Direct effect	0.887	0.144	0.604,1.170	
	Indirect effect	0.119	0.060	0.012,0.247	
Nutritional status →Daytime dysfunction →Cognitive function	Total effect	1.006	0.136	0.738,1.275	12.92%
	Direct effect	0.876	0.144	0.592,1.161	
	Indirect effect	0.130	0.059	0.016,0.247	

Note: Beta, standardized coefficient; CI, confidence interval; SE, standardized error; NA, not applicable.