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Exploring Causal Pathways to Sleep Quality in Young Adults Using a Multimodal Data-Driven Causal Discovery Analysis

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Purpose: Poor sleep quality is prevalent across the population and may significantly impact both physical and mental health. However, our understanding of the complex mechanisms underlying poor sleep quality is still incomplete, particularly regarding the various contributing factors. To address this, we utilized a data-driven causal discovery analysis (CDA) approach to explore causal pathways of sleep quality.

Patients and Methods: We relied on a large sample of healthy young adults from the Human Connectome Project (HCP; n = 1206 [54% female, 56% unmarried/non-cohabiting]) to explore causal pathways of sleep quality. We first used exploratory factor analysis to cluster 122 broad phenotypic variables into 21 factors and computed the functional connectivity of 13 resting-state brain networks. Then, using Greedy Fast Causal Inference (GFCI), we simultaneously integrated the obtained phenotypic factors, brain network connectivity, and sleep quality into the causal discovery analysis and ultimately constructed a causal model.

Results: The model proposes a hierarchical structure with causal effects propagating through complex interactions across multiple domains, ultimately linked to changes in sleep quality. Our causal model identified three phenotypic factors (negative affect, somaticism, and delay discounting) as directly linked to sleep quality. In addition, we examined causal models of sleep quality across gender (male and female) and relationship status (unmarried/non-cohabiting and married/cohabiting) and found some demographic-specific pathways.

Conclusion: Our data-driven model reveals complex mechanisms by which factors from different domains influence sleep quality and highlights several key factors that influence sleep quality, which may have important implications for the development of sleep theories and the improvement of sleep quality.

Keywords: sleep quality, causal discovery analysis, resting-state fMRI, negative affect, somaticism, delay discounting

Introduction

Sleep is a critical human function that profoundly correlates with physical health, mood, cognitive functions, social life, and quality of work.[1–4] Adequate sleep enhances attention, memory, positive mood, and work performance, while also reducing disease risk.[3,5] Conversely, poor sleep quality is associated with fatigue, daytime dysfunction, impaired immunity, and increased risks for cardiovascular, metabolic, and mental health disorders, such as anxiety and depression.[2–4,6,7] Despite its importance, sleep problems are highly prevalent. For example, in the United States, one-third of adults experience sleep deprivation,[8] and more than 60% of college students are considered poor sleepers.[3] Therefore, identifying the factors that affect sleep quality and incorporating them into future interventions is crucial.

Sleep is a tightly regulated neurophysiological process involving the ascending reticular activating system, key brain regions such as the hypothalamus and brainstem, and multiple neurotransmitters, which together govern the cycling between non-rapid eye movement (NREM) and rapid eye movement (REM) sleep.[9–12] Neuroimaging evidence further links sleep quality to neural activity in frontal, parietal, and temporal cortical regions.[13] Nonetheless, it remains unclear

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whether poor sleep quality is a cause or a consequence of brain dysfunction.[13,14] Beyond these neural mechanisms, sleep is regulated by interactions between biological, psychological, and other factors. Theoretical models, such as Blake et al's multilevel framework, emphasize that these biopsychosocial factors do not operate in isolation but collectively shape sleep through their interactions.[2] Given the complex, multi-systemic regulation of sleep, focusing solely on single factors (eg, stress, alcohol consumption)[2,15] may overlook other critical elements and their interactions and synergies, thereby potentially limiting a comprehensive understanding of the mechanisms influencing sleep quality.

Currently, there is no consensus on how sleep problems develop and which factors play a decisive role in maintaining them. In recent years, research on the causal relationships of sleep has grown, particularly through Mendelian Randomization (MR) which uses genetic signals from genome-wide association studies to explore links between sleep traits and disease outcomes. However, MR has limitations, especially regarding pleiotropism, where a single gene may affect multiple traits, potentially skewing results. Additionally, MR often assumes a linear relationship between exposure and disease outcome, and ignoring nonlinearity may lead to false negatives. To date, only one MR study has considered both linear and non-linear associations when exploring the causal relationships between sleep phenotypes and disease outcomes.[16] Moreover, much research on sleep has focused on its effects on mood and cognition.[2,4] As a result, there remains a lack of in-depth exploration of the causal mechanisms behind sleep problems and the key factors that influence their development. Causal models typically represent causal relationships in the data graphically. As the number of variables increases, the space of potential causal models may also expand, making it difficult to determine which potential causal model best fits the observed data. As a machine learning technique for learning causal models from data, causal discovery analysis (CDA) can identify the causal structures that best fit the data.[17] In order to understand this issue, we adopted a data-driven CDA to comprehensively analyze and explore the complex phenotypes related to sleep quality in order to construct comprehensive models containing clear causal pathways. This will help us elucidate how multiple domains and factors work together in leading to differences in sleep quality, and provide a scientific basis for developing more precise and effective interventions.

Although there are emerging studies that reveal unique sleep patterns in men and women, including sleep quality, duration, etc.,[8,18] the reasons for gender differences in sleep behaviors are unclear and not well researched. At all stages of life, women typically have poorer sleep quality and are more susceptible to factors than men.[8,19] Historically, biomedical research has underrepresented women's sleep studies, mainly due to the assumption that findings from male subjects are universally applicable.[8] In addition, most sleep research tends to view sleep as an individual behavior and ignores its social context. Ignoring the social aspects of sleep not only calls into question the ecological validity of these findings, but also has important consequences for sleep-focused interventions.[20] Sleep can be conceptualized as a basic "attachment behavior" and the presence of secure attachment facilitates sleep health.[21] Therefore, it is important to consider the causal variability that contributes to sleep problems across relationship status. Embracing technological advances and evidence-based practices makes it important to understand these differences in gender and relationship status as healthcare moves toward greater personalization.

To address these limitations, we draw on two relevant frameworks with overlapping insights and mutual reinforcement in key aspects. The Research Domain Criteria (RDoC) framework, developed by the US National Institute of Mental Health, moves beyond traditional diagnostic categories to focus on transdiagnostic functional domains (eg, Cognitive Systems, Negative Valence Systems) underlying psychopathology.[22,23] It emphasizes multidimensional assessments of core biobehavioral mechanisms and advocates for multivariate models that integrate growing knowledge of how multiple functional domains interact to contribute to psychopathology. Meanwhile, Blake et al's multilevel model elucidates how biological (eg, neurochemistry, brain circuits), psychological (eg, worry), and social (eg, social withdrawal) factors interact to shape insomnia,[2] highlighting systemic cross-domain interplay. Integrating these frameworks allows for capturing both the biopsychosocial mechanisms and cross-diagnostic functional domains of sleep problems, providing a holistic theoretical foundation. Accordingly, we systematically incorporated a broad range of biopsychosocial phenotypes and large-scale brain network functional characteristics from HCP data to capture as many potential sleep-related domains as possible. Our objective is to identify core factors associated with sleep quality. Guided by this theoretical framework, we hypothesize that sleep quality is influenced by the interplay of multi-level factors (social, psychological, biological, and neurological) with complex associations across these domains.

In this study, we aimed to construct a causal model related to sleep quality. We initially downscaled the extensive phenotypic measures for a large community sample using exploratory factor analysis and computed the functional connectivity within the subjects' resting-state brain networks. Subsequently, we applied CDA to the obtained phenotypic factors, brain network connectivity, and sleep quality. In this way, we successfully built an integrated multimodal causal model that reveals the causal pathways of brain connectivity and phenotypic factors on sleep quality. In addition, we examined and compared the causal models of sleep quality in groups of different gender (male and female) and different relationship status (unmarried/non-cohabiting and married/cohabiting). Our study may provide valuable insights into the specific mechanisms linking brain network connectivity and phenotypic factors to sleep quality in the general young population.

Materials and Methods

Subjects

We used the data from the Human Connectome Project WU-Minn1200 Subjects Data Release (n = 1206, age range: 22–37 years, 54% females, 44% married/cohabiting), comprising healthy young adults with no clinical diagnoses of psychiatric or neurological disorders.[24] All subjects provided written informed consent and all procedures were approved by the University of Washington Institutional Review Board (IRB # 201204036). This secondary analysis of the Human Connectome Project (HCP) dataset was approved by the Ethics Committee of Chongqing University of Posts and Telecommunications (protocol code: No. 2024028). This study included subjects who completed least two runs of rfMRI data (phenotypic n = 1206, resting-state fMRI n = 1096, final n = 1090). The demographic characteristics of the subjects are presented in Table 1. We estimated missing values using multiple imputation method implemented with the "mice" package in R software,[25] version 4.3.2. Specifically, binary variables were imputed using logistic regression, multinomial variables using polytomous regression, and continuous variables using random forests. Five independent imputed datasets were generated and subsequently analyzed, with final estimates pooled according to Rubin's rules.

Outcome Measure

Subjects' sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI).[26] PSQI is a self-assessment questionnaire used to assess sleep quality and disturbance over a 1-month time interval. PSQI comprises nineteen individual items, which are aggregated into seven "component" scores: (1) subjective sleep quality; (2) sleep latency; (3) sleep efficiency; (4) sleep duration; (5) sleep disturbance; (6) use of sleep medications; and (7) daytime dysfunction. These items are rated on a four-point Likert scale according to the frequency or severity of the problem. Each item is rated on a scale of 0 to 3 (eg, 0 = Not during the past month, 1 = Less than once a week, 2 = Once or twice a week, 3 = 3

Table 1 Demographic Characteristics of Participants (N = 1090)

Demographic Characteristics of PSQI Scores	M (SD), %	t/r Statistic
Full sample	4.78 (2.76)	
Gender		t = −1.77, P = 0.08
Male	4.62 (2.53), 45.7%	
Female	4.92 (2.93), 54.3%	
Relationship status		t = 1.86, P = 0.06
Unmarried/non-cohabiting	4.92 (2.83), 55.9%	
Married/cohabiting	4.61 (2.66), 44.1%	
Race		t = −3.98, P < 0.001
White	4.59 (2.70), 75.0%	
Other (race)	5.36 (2.87), 25.0%	
Age (years)	28.78 (3.70)	r = −0.03, P = 0.39
Education (years)	14.93 (1.80)	r = −0.15, P < 0.001
Income (per year)	5.07 (2.15)	r = −0.13, P < 0.001

Abbreviations: M, Mean; SD, standard deviation.

or more times a week). The sum of the component scores yields a total PSQI score ranging from 0 to 21, with higher scores indicating poorer sleep quality. The tool developers[26] suggested a threshold of 5, meaning that an overall PSQI score greater than 5 indicates poor subjective sleep quality. Notably, 344 of the 1090 participants (31.6% of the current sample) had a total PSQI score higher than 5. See Table 1 for more specific information.

Behavioral and Self-Report Measures

The HCP dataset encompasses an extensive range of self-report, diagnostic, and behavioral measures evaluating various domains, including emotion, cognition, social functioning, psychiatric disorders, and personality traits. To ensure comprehensive coverage of phenotypic characteristics, we included as many available sleep-related metrics as possible from the HCP dataset as possible. Our inclusive approach encompasses behavioral task data collected within the scanner environment, but excludes fMRI measurements of brain activation related to the task. We utilize summary scores, rather than item-level or subordinate scores, encapsulating the task constructs of our interest.[27] For example, for the N-Back task, which assesses working memory capacity, we included accuracy as well as average of median reaction time for the 2-back task and the 0-back task across all conditions. For domains that provide both age/gender-adjusted and unadjusted scores, we only include unadjusted scores. Ultimately, we included 122 measures (see Supplemental Materials for details and rationale for inclusion).

Exploratory Factor Analysis

We conducted exploratory factor analysis (EFA) to downscale the screened HCP phenotype data (122 measures in total). EFA was performed using the "Psych" package and "GPArotation" package for R (version 4.3.2),[28,29] where factors were extracted using the maximum likelihood method and subsequently rotated using the "direct Oblimin" method. The maximum likelihood method estimates model parameters by maximizing the likelihood function of observed data, demonstrating good reliability and robustness, even in cases where distributional assumptions are violated.[30,31] Oblimin rotation is a diagonal rotation method that accommodates correlated factors. We used Monte Carlo permutation analysis (parallel analysis)[32] to determine the optimal number of factors to be extracted from the factor analysis. Only measures with loadings greater than or equal to 0.30 were considered to contribute to the factor, which is a commonly accepted threshold in factor analysis.[33]

MRI Scanning and Preprocessing

MRI scanning was performed using a customized 3T Siemens Connectome Skyra with a standard 32-channel Siemens receiver head coil and a body transmission coil. Structural images (T1-weighted and T2-weighted) were preprocessed to correct for gradient non-linearities, readout distortions, bias field distortions, and aligned to the MNI space template. For detailed preprocessing steps, see.[34] During resting-state fMRI scanning, all subjects were instructed to relax, keep their eyes open, keep awake and not think of anything in particular. The resting-state fMRI data were preprocessed by correcting spatial distortions, realigning for motion, registering to the structural images, normalizing to MNI space. fMRI data were then high-pass filtered, denoised using ICA-FIX, and regressed out for the artifact components and 24 motion regressors. For detailed information on the preprocessing pipeline see.[34] The final ICA-FIX denoised version of the fMRI data was downloaded and used for the next brain network analysis.

fMRI Network Connectivity

Eighty-four percent of subjects completed four rs-fMRI scans within two days, totaling 58 minutes and 12 seconds of rs-fMRI data. Our rfMRI network connectivity analysis only included subjects who completed at least two rfMRI acquisitions (90% of total subjects). We used the set of 300 regions of interest (ROIs) and associated network assignments described by Green's team as nodes of the whole-brain functional network,[35] which was further augmented with additional nodes for subcortical and cerebellar structures based on the Power 264 atlas.[36] This parcellation used a set of functionally defined spherical ROIs in volumetric space, and employed the Infomap algorithm to compute network assignments for each ROI. Specifically, we partitioned the 300 ROIs into functional connections within 13 resting-state brain networks. For each network, we calculated pairwise Pearson correlations

between every pair of parcels. Within each network, we then averaged these correlations to derive summary statistics of connectivity.[37–40] The final 13 networks obtained include default mode network, cingulo-opercular network, dorsal attention network, frontoparietal network, ventral attention network, somatomotor lateral network, somatomotor dorsal network, salience network, auditory network, medial temporal lobe network, parieto-medial network, visual network, and reward network.

Greedy Fast Causal Inference

The Greedy Fast Causal Inference (GFCI) algorithm[41] is a graph search methodology for inferring causal structures. It adeptly integrates greedy strategies with constraint satisfaction techniques, enabling the algorithm to efficiently and relatively accurately identify causal relationships between variables, even in the presence of unmeasured confounding factors. This capability provides valuable insights into the underlying causal structure.[17] The GFCI makes causal inferences in two steps. In its initial phase, the GFCI employs the Fast Greedy Equivalence Search (FGES) algorithm[42] to explore potential causal models, minimizing a penalized likelihood score like the Bayesian Information Criterion (BIC).[43] FGES iteratively adjusts edges, maximizing model fit. Initially, it assumes that there are no unmeasured common causes, which allows for a preliminary assessment of causal relationships among the measured variables. In the second phase, GFCI drops this assumption and conducts a series of conditional independence tests. These tests iteratively excluded causal relationships for which conditional independence was implied and remained unconfirmed by the data. We provide an additional example of how constraint-based causal inference works in Figure 1. The output of the GFCI algorithm results in a partial ancestral graph (PAG), which is able to encode the presence of causal relationships, uncertain relationships, and unmeasured confounding variables. For the generated PAG, the variables are represented as nodes in the graph. The type and direction of the edge connecting two nodes specify the nature of the modeled causal relationship.[41,44,45] Figure 2 lists the possible edge types in the PAG and their corresponding meanings.

We implemented the GFCI algorithm using the Tetrad software version 7.6.6, with the following model parameters: a BIC score, which was set to a default penalty discount value of 2, was used to evaluate the penalized likelihood; a Fisher Z test value, with an alpha threshold set at the default of 0.01, served to perform the conditional independence tests; and a maximum graph degree limit of 100. To ensure the stability of causality, we performed 1000 bootstrap iterations on 90% of the samples to adequately estimate the distribution of the statistics. Within each iteration, we applied the same GFCI algorithm to analyze the data. Subsequently, we summarized the proportions of all potential relationships after aggregating the results of 1000 iterations, including the frequency of various edges and edge types.

SEM: Effect Size Estimation

To uncover the effect sizes of causal pathways in the model, we fitted standardized effect sizes by constructing a structural equation model (SEM) for the output of the GFCI using R's "lavaan" software package.[46] The statistical power of each model was also assessed (see Supplemental Materials).

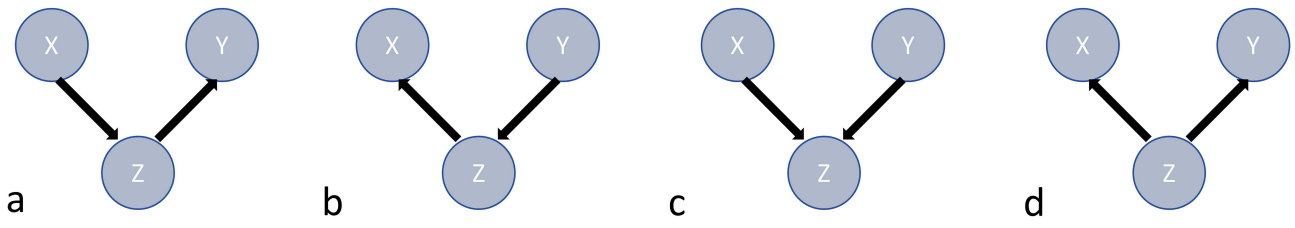

Figure 1 Figure 1 illustrates a constraint-based causal discovery algorithm, showing four possible causal relationships among variables X, Y, and Z. From left to right, the figures are (**a–d**). (**a**) A collider graph is displayed where X causes Z, Z causes Y, and X and Y have a causal relationship. (**b**) The edges between X and Z and between Z and Y are reversed, so now Y causes X. (**c**) Shows a scenario where both X and Y cause Z, but X and Y are not related. In this structure, X and Z are dependent, Y and Z are dependent, but X and Y are independent. However, when conditioning on Z, it becomes a common cause for X and Y, making them dependent on each other. GFCI uses observations from conditional Independence tests to determine the direction of causal relationships. This allows for distinguishing whether the data is generated by (**a**) or one of the other three figures, as these four graphs exhibit different conditional dependencies.

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Edge Types	Present Relationships	Absent Relationships
A → B	A is a direct cause of B.	B is not a cause of A.
A ●→ B	Either A is a cause of B or there is an unmeasured confounder of A and B or both.	B is not a cause of A.
A ↔ B	There is an unmeasured confounder of A and B.	A is not a cause of B. B is not a cause of A.
A ●—● B	One of the following: 1) A is a direct cause of B 2) B is a direct cause of A 3) There is an unmeasured confounder of A and B 4) Both (1) and (3) 5) Both (2) and (3)	

Figure 2 Types of edges.

Results

Exploratory Factor Analysis: Dimensionality Reduction Decomposition of the Phenotypic Measures

According to the Kaiser-Meyer-Olkin test (KMO) and Bartlett's Test of Sphericity,[47] our phenotypic data had a good fit (KMO = 0.87, P < 0.001), which was well suited for Exploratory factor analysis (EFA) that was used to downscale the screened complete HCP phenotype data. Based on the results of the Monte Carlo simulation, we categorized the 122 phenotypic variables into 21 factors that together accounted for 46% of the common variance (see Figure 3). The EFA model fit indices indicated good factor separation (RMSEA= 0.04, Tucker-Lewis Index = 0.85). These 21 factors cover

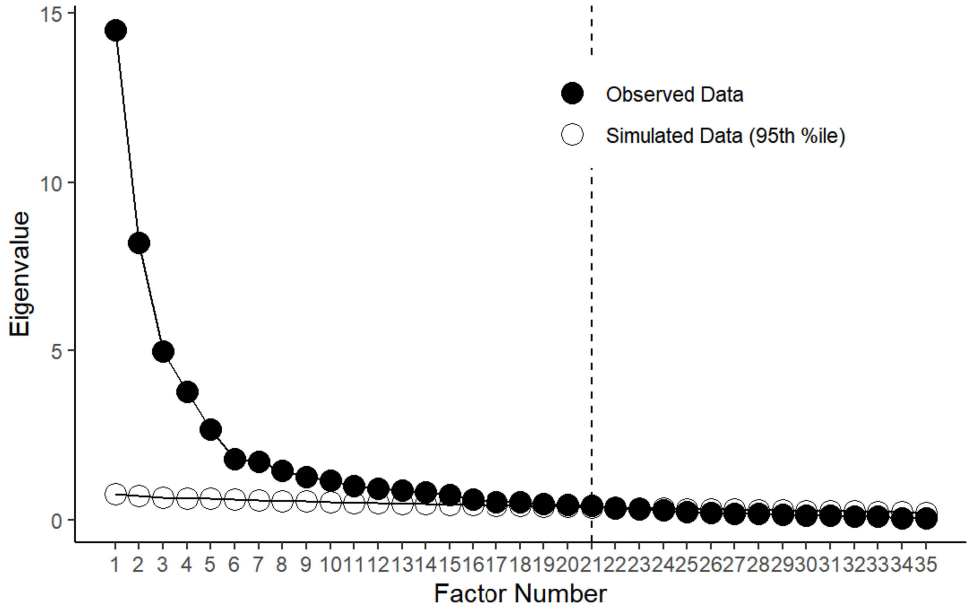

Figure 3 Permutation-based test for eigenvalue significance (Monte Carlo permutation analysis).

five domains: (1) Cognitive domain includes Crystalized IQ, Delay Discounting, Fluid Cognition, Gambling Task Reaction Time, Language Task Performance, Memory Performance, Processing Speed and Visuospatial Processing; (2) Physiological domain includes Blood Pressure and Somaticism; (3) Negative valence domain include Externalizing, Internalizing and Negative Affect; (4) Behavioral domain includes Alcohol Use, Cigarette Use, Drug Use, Hyperactivity and Unconscientiousness/Inattention; (5) Social domain includes Openness, Social Support/ Positive Affect and Social Withdrawal. Factor correlations were presented in Supplemental Materials (Figure S1).

GFCI: Using Causal Discovery Analysis to Explore Causal Pathways of Sleep Quality

We constructed a causal model related to sleep quality using the Greedy Fast Causal Inference (GFCI) algorithm. The GFCI outputs are shown in Figure 4, depicting the causal relationships among 21 extracted factors, 13 predefined resting-state brain network connectivity, and sleep quality. Structural equation modeling (SEM) was then employed to assess model fit and effect sizes, and revealed that the model fit was good (RMSEA = 0.05; Comparative Fit Index (CFI) = 0.91)

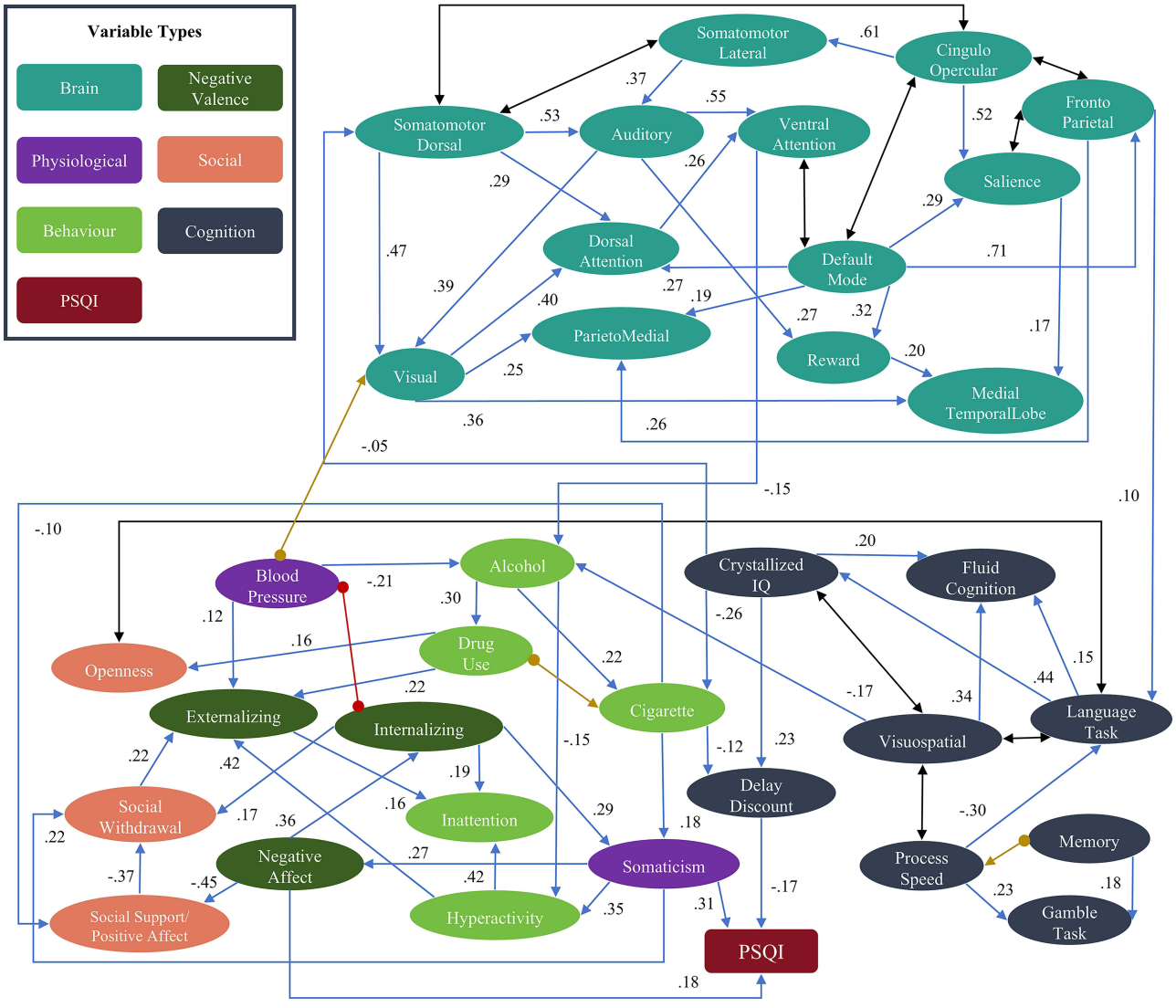

Figure 4 Greedy Fast Causal Inference (GFCI) was applied to simultaneously integrate phenotypic factors, brain network connectivity, and sleep quality into a causal discovery model of sleep quality. The variables are represented in different colors to distinguish their types. The weights of the normalized edges are displayed next to each edge in text form, with only the explicitly pointed edges labeled. The structural equation model (SEM) demonstrated good fit (RMSEA = 0.05; CFI = 0.91), and all paths were significant ($p < 0.05$). See the Supplemental Materials for factor correlations and additional data.

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and all paths were significant ($p < 0.05$). The results of the stability analysis are presented in the Supplemental Materials (Figures S2–S6).

Our causal model reveals the complex interactions between brain network connectivity, behavioral phenotypes and sleep quality, and highlights some key causal pathways linked to sleep quality (see Figure 4). We identified three critical phenotypic factors that are directly related to sleep quality: somaticism (effect size (ES) = 0.31), delay discounting (ES = −0.17), and negative affect (ES = 0.18). Notably, elevated somaticism is associated with a decline in sleep quality, while factors such as high internalization problems and cigarette consumption may contribute to heightened somaticism. In our model, the delay discounting factor takes the area under the curve (AUC) as its indicator, with higher AUC values representing lower discount rates. Our findings reveal that high discount rates are directly linked to poor sleep quality, with low crystallized IQ and high cigarette use associated with increased delay discount rates. Furthermore, both high alcohol use and low language task performance are crucially indirectly related to elevating somaticism and increasing delay discount rates. High negative affect emerges as another pivotal factor that is directly related to poor sleep quality. We also identified a trigger for negative affect, namely high somaticism. In addition, reduced connectivity within the frontoparietal network is associated with decreased performance on language tasks, and decreased connectivity within the ventral attention network is linked to increased drinking, with both pathways ultimately and indirectly associated with impaired sleep quality. Overall, our causal analysis reveals complex pathways in which multi-domain and multi-modal factors are directly and indirectly linked to sleep quality, which contributes to the understanding of the mechanisms underlying the emergence of sleep problems.

Causal Models of Sleep Quality Across Gender and Relationship Status

Although the independent samples *t*-test did not reveal significant differences in PSQI total scores for gender ($t = -1.77$, $P = 0.08$) or relationship status ($t = 1.86$, $P = 0.06$), there are emerging studies that reveal their potentially unique effects on sleep.[8,18,21] We further constructed causal models tailored to different gender and relationship status in order to explore the demographic characteristics and unique patterns of sleep. For the causal models of gender, we found that the models were well-fitted (Male model: RMSEA = 0.06, CFI = 0.88, with all paths significant at $p < 0.001$; Female model: RMSEA = 0.07, CFI = 0.84, with all paths significant at $p < 0.001$), and for explicitly pointing edges, 16 identical paths and 60 different paths were found in the male and female models (see Figure 5). In particular, somaticism and delay discounting emerge as common factors directly linked to sleep quality in both male and female models, whereas male sleep quality is also directly associated with negative affect. And Poor sleep quality in women is linked to externalizing problems. Furthermore, in females, social withdrawal is directly associated with somaticism, whereas in males, somaticism tends to be linked to social withdrawal. And for females, substance use (such as drug and cigarette consumption) has a direct association with delay discounting. In contrast, among males, the use of substances like alcohol affects social factors, such as social withdrawal and social support/positive affect, which subsequently associated with the emergence of negative affect.

For the causal models of relationship status, we found goodness-of-fit indices for the both models (Unmarried/Non-cohabiting model: RMSEA = 0.06, CFI = 0.90, with all paths significant at $p < 0.001$; Married/Cohabiting model: RMSEA = 0.05, CFI = 0.90, with all paths significant at $p < 0.01$), and for explicitly directed edges, 13 identical paths and 75 different paths were found between unmarried/non-cohabiting and married/cohabiting populations (see Figure 6). In particular, somaticism and delay discounting emerged as common factors directly linked to sleep quality across both relationship status, whereas the sleep quality of married/cohabiting individuals is also directly associated with negative affect. Moreover, within the married/cohabiting group, hyperactivity problem is linked to somaticism, while in the unmarried/non-cohabiting group, somaticism tends to be associated with hyperactivity. Additionally, crystallized IQ is indirectly linked to delay discounting in married/cohabiting individuals, whereas for unmarried/non-cohabiting individuals, crystallized IQ has a direct association with delay discounting.

Discussion

Previous studies on the causal mechanisms of sleep quality are limited, and the underlying pathways remain unclear. Here, we used a data-driven approach to construct a multi-modal, multilevel causal model integrating brain networks,

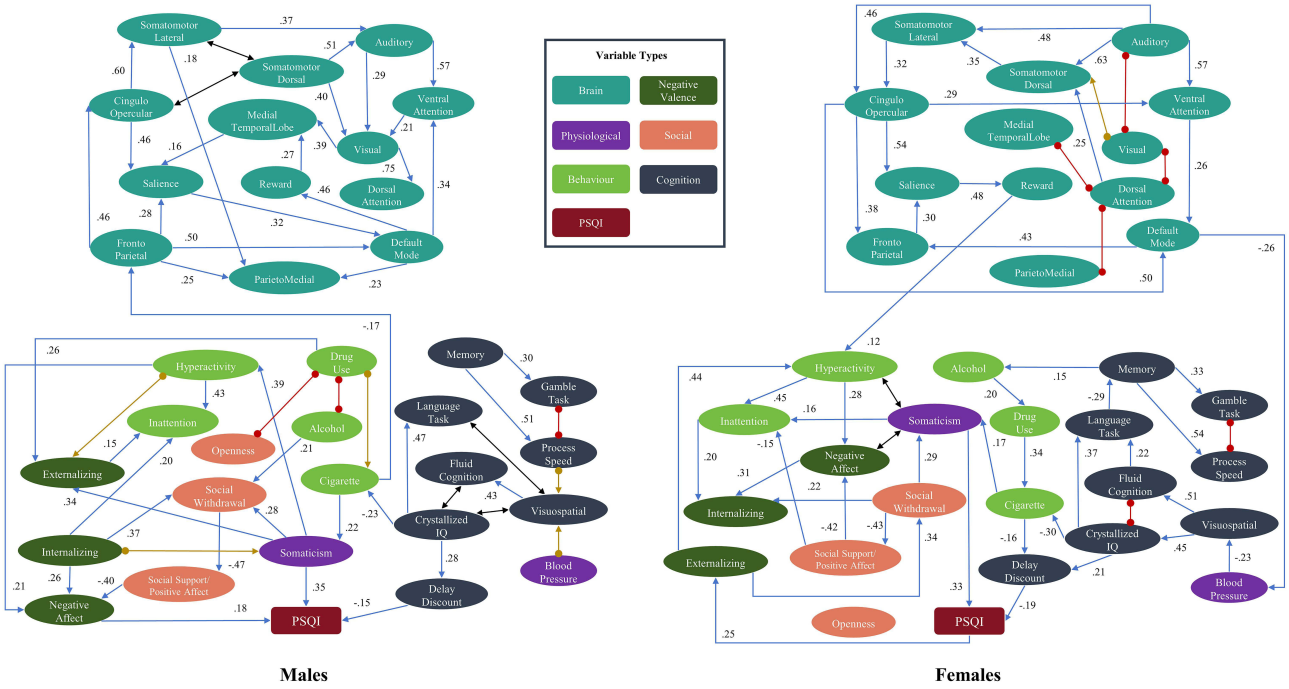

Figure 5 Causal models of sleep quality across gender are presented, with the left and right panels showing the models for males and females, respectively. Both models fit well. For the male model: RMSEA = 0.06, CFI = 0.88, with all paths significant ($p < 0.001$). For the female model: RMSEA = 0.07, CFI = 0.84, with all paths significant ($p < 0.001$).

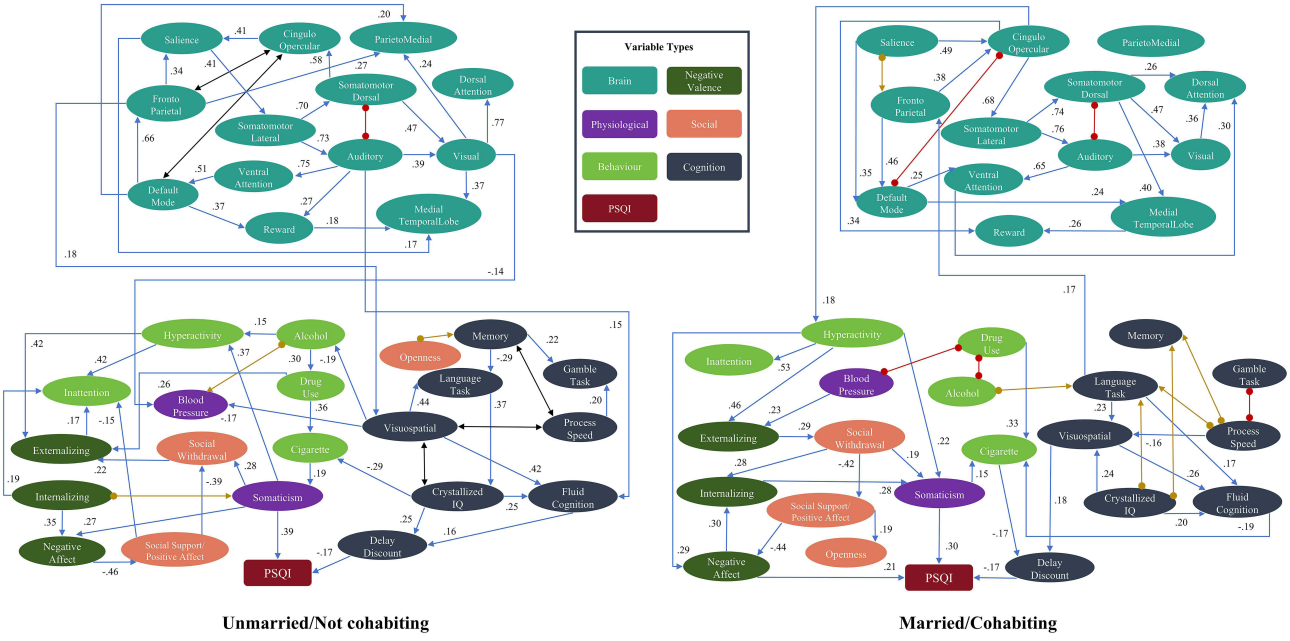

Figure 6 Causal models of sleep quality across relationship status are shown, with the left panel representing the unmarried/non-cohabiting group and the right panel the married/cohabiting group. Both models fit well. For the unmarried/non-cohabiting model: RMSEA = 0.06, CFI = 0.90, with all paths significant ($p < 0.001$). For the married/cohabiting model: RMSEA = 0.05, CLI = 0.900, with all paths significant ($p < 0.01$).

phenotypic factors, and sleep quality. While our findings are exploratory and should be considered hypothesis-generating rather than confirmatory, they provide important insights into the causal architecture of sleep. The model elucidates specific pathways through which brain and behavioral factors influence sleep in young adults. Furthermore, stratified analyses by gender and relationship status revealed distinct causal pathways that may underlie demographic differences

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in sleep. These results advance understanding of the determinants of poor sleep and offer a theoretical foundation for targeted interventions.

Our analyses revealed three key factors directly linked to sleep quality: negative affect, somaticism, and delay discounting. Many studies have demonstrated the link between negative affect and sleep and have shown that all types of negative affect appear to be detrimental to sleep and considered to be major predictors of poor sleep quality.[1,7] Our model clearly depicts a direct association between negative affect and poor sleep quality. According to previous studies, negative affect may be linked to sleep through multiple mechanisms. On the one hand, certain negative affect may activate the hypothalamic-pituitary-adrenal (HPA) axis, resulting in physiological responses such as increased cortisol secretion, heart rate, respiratory rate, and muscle tension,[48] which disrupt normal sleep.[49] On the other hand, persistent negative emotions may be associated with individuals experiencing cognitive activities such as rumination and worrying before bedtime, which may be linked to the perception of heightened psychological stress that can be associated with disruptions in sleep onset and sleep maintenance.[50] Our model also suggests that negative affect is linked to problems within internalizing and social factors, which is consistent with existing research.[51,52] It is noteworthy that negative emotions are directly associated low social support/positive affect, which in turn may spread and be linked to social withdrawal. These results suggest a potential mechanism of emotional contagion whereby emotional states between individuals can permeate and influence each other through social interactions.[1,53]

Our causal model indicated that somatization, which is the expression of psychological distress as physical symptoms (eg, persistent pain, fatigue, or other discomfort),[54,55] is another factor directly linked to poor sleep quality. In individuals with generally good overall sleep quality, the moderate effect size (ES = 0.31) suggests that even mild somatic symptoms may disrupt sleep regulatory mechanisms, positioning somatization as a critical target for early intervention. Unlike in clinical samples, where comorbidities often obscure the specific impact of somatic symptoms,[56,57] otherwise healthy individuals experiencing somatization may excessively focus on minor bodily discomforts or changes, especially during nighttime rest. This heightened interoceptive awareness can trigger or exacerbate difficulties falling asleep and increased arousal, thereby impairing both sleep initiation and maintenance and ultimately being linked to reduced sleep quality.[58,59] Moreover, somatization is often accompanied by high levels of anxiety, depressed mood and rumination,[58,60] and these mood states have been shown to be strongly associated with poor sleep quality.[61] Our model also found another pathway by which somaticism is indirectly linked to sleep quality through negative affect. Furthermore, we identified several factors associated with somatization, including high cigarette use and high internalizing. Studies show a positive correlation between nicotine abuse and the somatization index.[62] Additionally, individuals with internalizing issues are more likely to experience negative life events frequently, and these negative experiences may be linked to the development of functional somatic symptoms.[63,64] And internalizing issues encompass psychological stress, depression and anxiety, with prolonged psychological stress and depression being recognized as significant triggers for somatization disorder.[65]

An interesting finding of our study is that steep delay discounting that refers to people's preference for immediate small rewards over delayed but larger ones is associated with poor sleep quality. A previous study also used the HCP dataset reported an association between delayed discounting and sleep, but failed to clarify the direction of the relationship.[66] Our results extend this work by demonstrating that delay discounting may precede and predict poorer sleep. This contrasts with many existing studies, which typically treat sleep deficiency as a contributor to impulsive decision-making, particularly in sleep-deprived populations.[67] The discrepancy may stem from our focus on healthy adults. In this group, mild sleep disturbances are more likely to reflect voluntary behavioral choices influenced by delay discounting, rather than arising from impaired cognitive control. Importantly, this pattern may challenge the traditional theories, which typically positions cognitive function as a consequence of sleep disruption.[68] Instead, our findings suggest that delay discounting, a key cognitive trait, functions as an antecedent. Resisting the temptation to obtain a reward immediately requires the activation of self-control processing, and individuals with high rates of delay discounting typically perform poorly in self-regulation and impulse control.[69] Our results suggest that the possible roles of high discounting in contributing to poor sleep quality may be due to the formation and maintenance of unhealthy sleep habits (eg, delay bedtime, irregular sleep times, and excitatory activities prior to bedtime) resulting from poor self-control. Unhealthy behaviors such as irregular sleep schedules, nighttime cell phone use, and gaming before bedtime

have been proved to be associated with excessive delayed discounting,[70] and all contribute to poor sleep quality.[71,72] With a modest effect size (ES = −0.17, comparable to that of negative affect), delay discounting may still be linked to influences on sleep regulation in otherwise healthy individuals. This finding points to a possible role for delay discounting in early intervention strategies and adds to the emerging evidence that cognitive traits may play a meaningful role in sleep regulation, complementing more established sleep models. Additionally, our findings underscore the association between delay discounting and both crystallized IQ and cigarette use. Lower crystallized IQ may impair the ability to forecast long-term consequences,[73,74] while substance use disrupts dopamine-mediated reward processing and diminishes inhibitory control, thereby reinforcing preference for immediate rewards.[75,76]

Our causal model reveals pathways through which brain networks are related to behavioral phenotypes, whereby reduced connectivity of the frontoparietal network is linked to a decline in language task performance, while reduced connectivity in the ventral attention network (VAN) is associated with increased alcohol consumption, with both pathways ultimately linked to decreased sleep quality. Studies have identified a potential association between the frontoparietal network and language, with the left inferior frontal gyrus supporting syntactic processing (eg, grammar construction and complex sentence comprehension) and parietal regions aiding spatial attention and contextual language interpretation.[77,78] Consequently, decreased connectivity within the frontoparietal network may be linked to difficulties and poorer performance on language tasks. Additionally, numerous studies have shown an association between VAN and excessive alcohol consumption.[79] The VAN is one of the core neural systems in response inhibition, and deficits in response inhibition may be linked to difficulties in suppressing impulsive behaviors in individuals, thus being associated with difficulties in resisting or exacerbating substance use.[79] Furthermore, the VAN plays a crucial role in regulating sudden emotional responses and maintaining goal-directed behaviors, and its dysfunction may lead to affective dysregulation and executive dysfunction,[80] which may further elevate the risk of alcohol abuse.[81]

In addition, our causal models of sleep quality for males and females revealed both identical and gender-specific pathways. Somaticism and delay discounting were found to be directly linked to sleep quality in both genders, suggesting these factors may have a certain robustness. However, the sleep quality of women is not directly associated with the negative affect factor compared to men. In the HCP dataset, there was no significant difference in negative affect scores between the two genders ($t = 0.47$, $P = 0.64$). Although women of all age groups reported more subjective sleep problems,[82–84] numerous studies consistently indicate that healthy women generally have better objective sleep quality than men.[84–87] Existing research generally suggests that women experience more sleep problems than men,[88,89] but this may be related to more severe insomnia problems in women,[90] not just sleep quality itself. At the same time, due to physiological reasons such as pregnancy and menstrual cycles, women are more affected by hormones like ovarian steroids, which may make their sleep responses to negative emotions more complex and uncertain.[84,91–94] Based on our causal model, women may alleviate the association between negative emotions and sleep quality through internalizing mechanisms. Furthermore, poor sleep quality in women can alsobe linked to externalizing problems. Sleep plays a crucial role in the neural mechanisms of emotion regulation and emotion expression.[95] Women may be more susceptible to the negative effects of poor sleep and may be more emotionally affected,[96–98] which may be linked to externalizing problems.[99] Additionally, in women, social withdrawal is directly associated with somaticism, while somaticism is linked to social withdrawal in men. Women are generally more inclined to engage in social interactions and express their emotions.[100] When women withdraw from social activities, the lack of emotional communication may be linked to emotional suppression and psychological stress, which may subsequently be associated with somatic symptoms.[101,102] In contrast, men have traditionally been encouraged to repress their emotions and experiences of distress.[103] As a result, when men experience somatization issues, they are often reluctant to show psychological and physical vulnerability.[104,105] Various discomforting symptoms may be linked to their reducing or avoiding social activities, thus contributing to social withdrawal.[106,107] Finally, substance use for females (eg, drugs, smoking) is associated with delay discounting. In males, substance use is linked to social factors like social withdrawal and social support/positive affect, which in turn are associated with negative affect. Research has shown that women often develop dependence more rapidly than men following the initiation of substance use and they find it harder to quit.[108,109] Substances such as cocaine and nicotine can affect neurotransmitter systems, including dopamine, which are closely tied to the processing of immediate rewards and decision-making.[110–113] Subsequent reductions in the use of these substances may also be linked

to withdrawal symptoms,[114–116] thereby further influencing women's delay discounting rates. However, substance use is linked to social factors like social withdrawal and social support/positive affect in males, which in turn are associated with negative affect. Studies have indicated that alcohol consumption negatively impacts the anterior cingulate cortex, with particularly pronounced effects observed in men.[117,118] Impairment of the anterior cingulate cortex can diminish inhibitory control, potentially associated with social withdrawal,[102,119] which in turn may provoke issues such as negative emotions.[102,120,121]

We also analyzed causal models of sleep quality for relationship status (unmarried/non-cohabiting and married/cohabiting). Somaticism and delay discounting are robust factors directly linked to sleep quality in both relationship status. Compared to unmarried/non-cohabiting individuals, the sleep quality of married/cohabiting individuals is additionally directly associated with negative affect. In the HCP dataset, the unmarried/non-cohabiting group showed higher levels of negative affect ($t = 2.19$, $P < 0.05$) compared to the married/cohabiting group. Unmarried/non-cohabiting individuals may be more likely to experience isolation in times of difficulty,[122] while also lacking the positive impact of a stable partner relationship,[123] which could contribute to higher levels of negative emotions.[124,125] However, they may have developed specific coping mechanisms (eg, reduced social interactions) that provide temporary relief from poor sleep quality. Future research could consider relationship status as a moderating variable to verify and discuss the impact of negative emotions on sleep quality. Additionally, within the married/cohabiting groups, hyperactivity is linked to somaticism, while somaticism tends to be associated with hyperactivity problems in the unmarried/non-cohabiting group. Studies have shown that individuals with anxiety symptoms and hyperactivity problems are at risk for functional somatic symptoms.[126,127] Furthermore, in general, married/cohabiting groups show higher sensitivity and concern for physical changes and family dynamics,[128,129] and hyperactivity symptoms may amplify their perception of changes in the external environment,[130] which also triggers higher levels of anxiety, stress, and consequently somatic problems.[103,131,132] In contrast, unmarried/non-cohabiting individuals tend to face challenges alone, lacking external emotional support and social resources.[122,133] In the presence of somatic problems, they may alleviate or express psychological discomfort and physical uneasiness through hyperactive behaviors.[134,135] Moreover, crystallized IQ is indirectly linked to delay discounting in married/cohabiting individuals, but has a direct association with delay discounting in the unmarried/non-cohabiting group. Delay discounting is typically associated with crystallized intelligence.[136,137] Married/cohabiting individuals often navigate a shared decision-making environment, making it more likely that they will consult with their partners,[138] which may result in the effects of crystallized intelligence on their decision-making being more indirect. Conversely, unmarried/non-cohabiting individuals may be more inclined to make independent decisions,[139] meaning that existing knowledge and experience may be related to their delay discounting.

Our study integrated indicators traditionally scattered across emotional, cognitive, physiological, and other domains into a single causal model, identifying three factors directly associated with sleep quality. This provides empirical support for the theoretical framework of hierarchical biopsychosocial interactions emphasized by Blake et al.[2] Notably, positioning delay discounting as a precursor of poor sleep rather than a result challenges the conventional view that sleep deficiency is the primary driver of impulsive decision-making,[67,68] expanding understanding of sleep regulation in healthy populations. The study reveals that altered connectivity within brain networks indirectly influences sleep through behavioral phenotypes, adding neurobiological depth to these theories. It both validates the RDoC framework's focus on cross-dimensional links between neural circuits and behavior and highlights the hierarchical system (from neural activity to behavioral expression) often overlooked by traditional sleep models.[22,23] Furthermore, the moderating roles of gender and relationship status underscore the need to refine theories by incorporating demographic-specific pathways, aligning with RDoC's advocacy for dimensional, context-sensitive mechanisms over universal models. Collectively, our study uncovers the intricate causal mechanisms influencing sleep quality and identifies key causal pathways. It deepens understanding of sleep disorder etiology, enriches theoretical frameworks, advances knowledge of sleep regulatory mechanisms, and provides a theoretical basis for targeted sleep interventions. For instance, targeting the regulation of negative emotions may be more effective in improving sleep quality than interventions that promote positive emotions. In the era of popularization of mobile electronics and online entertainment, it may be crucial to adopt effective guidance methods or techniques to change sleep-related decisions and reduce various undesirable temptations. Reducing or prohibiting substance use is important for alleviating a variety of problems as well as improving sleep.

However, this study has several limitations. First, the HCP dataset is cross-sectional and does not capture temporal dynamics, limiting our understanding of long-term changes. Although the GFCI algorithm is suitable for such data, it can only infer static associations. Future studies should employ longitudinal methods, such as Granger Causality Testing, to more accurately model the temporal evolution of sleep problems. Second, although GFCI accounts for unobserved confounders, key variables may still be missing. Factors such as genetic predispositions (eg, circadian rhythm genes), lifestyle behaviors (eg, physical activity), and environmental exposures (eg, light, noise) could simultaneously influence sleep and cognitive or emotional phenotypes, potentially biasing causal inferences. Third, sleep quality was assessed solely via the PSQI, a self-reported measure that may not fully align with objective sleep architecture. Future research should incorporate objective metrics, such as polysomnography, to validate and extend these findings. Finally, the sample consisted primarily of healthy young adults not suffering from sleep disorders, limiting the generalizability of our findings to clinical populations or older adults, who may exhibit distinct neurobiological, cognitive, or psychosocial profiles.[140,141] Future work should systematically collect multimodal phenotypic and neuroimaging data from individuals with sleep disorders or older populations, applying causal discovery methods to build population-specific models and advance personalized sleep interventions.

Conclusion

In conclusion, we have for the first time applied a data-driven CDA approach to construct a causal model of sleep quality across resting-state brain function networks and multiple behavioral phenotypes related to sleep quality. We identified the following key findings: (a) high delay discount rates, somaticism, and negative affect are three key factors directly linked to reduced sleep quality; (b) the reduced connectivity within the frontoparietal and ventral attention networks indirectly reduces sleep quality by modulating behavioral phenotypes; (c) gender and relationship status moderate some of the causal pathways of sleep quality. This study visualizes the complexity of sleep models and reveals unique causal mechanisms across gender and relationship status, providing valuable insight into the mechanisms underlying poor sleep quality and helping to develop more effective interventions. In particular, our causally-driven model can be used as a base model to further integrate more domains and variables related to sleep quality, thus creating a more comprehensive and integrated multimodal sleep model.

Data Sharing Statement

The data utilized in this study are available for download from the Human Connectome Project (www.humanconnectome.org/). Users must agree to data use terms for the HCP before being allowed access to the data and ConnectomeDB, details are provided at https://www.humanconnectome.org/study/hcp-young-adult/data-use-terms. Factor loadings and factor nomenclature from the analysis of standardized variables are available upon request to the corresponding author. The Codes that support the findings of this study are available from the corresponding author upon reasonable request.

Ethical Approval Statement

This study was performed using publicly available datasets from Human Connectome Project (HCP) Young Adult. Human data acquisition for the HCP was approved by the Institutional Review Board (IRB) of the University of Washington in St. Louis (IRB # 201204036; Title: "Mapping the Human Connectome: Structure, Function, and Heritability"), and all open access data were deidentified. In addition, the present study received approval from the Human Ethics Committee of Chongqing University of Posts and Telecommunications (protocol code: No. 2024028) and adheres to the ethical guidelines outlined in the Declaration of Helsinki.

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Author Contributions

Xiong Xiao: Formal analysis, Methodology, Writing - original draft, Writing - review & editing; Yulin Wang: Methodology, Writing - original draft, Writing - review & editing; Debo Dong: Conceptualization, Writing - review & editing; Wei Wang: Conceptualization, Writing - review & editing; Zhangyong Li: Funding acquisition, Project administration, Conceptualization, Writing - review & editing; Yiqun Guo: Funding acquisition, Project administration, Conceptualization, Supervision, Methodology, Writing - original draft, Writing - review & editing. All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation; all authors took part in drafting, revising or critically reviewing the article; all authors gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors assert that they have no competing financial or personal interests.

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