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A multidimensional investigation of sleep and biopsychosocial profiles with associated neural signatures

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ABSTRACT

Sleep is essential for optimal functioning and health. Interconnected to multiple biological, psychological and socio-environmental factors (i.e., biopsychosocial factors), the multidimensional nature of sleep is rarely capitalized on in research. Here, we deployed a data-driven approach to identify sleep-biopsychosocial profiles that linked self-reported sleep patterns to inter-individual variability in health, cognition, and lifestyle factors in 770 healthy young adults. We uncovered five profiles, including two profiles reflecting general psychopathology associated with either reports of general poor sleep or an absence of sleep complaints (i.e., sleep resilience) respectively. The three other profiles were driven by the use of sleep aids and social satisfaction, sleep duration and cognitive performance, and sleep disturbance linked to cognition and mental health. Furthermore, identified sleep-biopsychosocial profiles displayed unique patterns of brain network organization. In particular, somatomotor network connectivity alterations were involved in the relationships between sleep and biopsychosocial factors. These profiles can potentially untangle the interplay between individuals' variability in sleep, health, cognition and lifestyle — equipping research and clinical settings to better support individual's well-being.

Keywords: sleep, biopsychosocial outcomes, multivariate, profile, psychopathology, cognition

INTRODUCTION

Decades of research have established that sleep is interconnected to multiple biological, psychological and socio-environmental factors (i.e., biopsychosocial factors)¹⁻⁴. Importantly, sleep difficulties are among the most common comorbidities of mental and physical disorders⁵⁻⁸, highlighting the central role of sleep in health. Despite the recognition that sleep is a unique marker for optimal health^{9,10} and a potential transdiagnostic therapeutic target, its multidimensional and transdisciplinary nature is rarely capitalized on in research. Traditionally, single-association studies have investigated the relationship between a single dimension of sleep (e.g., duration, quality, onset latency) and/or a single outcome of interest. Such uni-dimensional studies have demonstrated links between insufficient or poor sleep with a multitude of negative outcomes separately, including cognitive difficulties^{11,12}, brain connectivity changes¹³⁻¹⁵, decreased physical health^{7,16}, mental health and well-being^{8,17}, as well as increased risks for cardiovascular disease^{7,18,19}, neurodegenerative disease^{20,21} and psychiatric disorders^{8,22}. However, by treating sleep as a binary domain (e.g., good vs. poor sleep, short vs. long), these studies fail to capture the multidimensional nature of sleep and the multiple intricate links with biological, psychological, and socio-environmental (i.e., biopsychosocial) factors. Therefore, it remains unclear which biopsychosocial factors are most robustly associated with sleep traits and whether these factors are supported by similar neural mechanisms.

Adding to the complexity of these relationships is how sleep and good sleep health are defined. Characterizing sleep is a challenging task because of its multidimensional nature²³. Sleep can be defined by its quantity (i.e., sleep duration) and quality (i.e., satisfaction, efficiency), as well as in terms of regularity, timing, and alertness. These dimensions are deemed particularly relevant when defining sleep health⁹, as they each have been related to biopsychosocial outcomes. Different sleep dimensions can also be described as either “good” or “bad” sleep, without necessarily affecting one another, e.g., short sleep duration is not systematically associated with poor sleep quality. Another important aspect of sleep is how it is subjectively characterized. For instance, our perception of sleep can influence daytime functioning²⁴ and can be ascribed to certain behaviors that differ from objective reports^{25,26}. Reconciling the multiple components of sleep and the complex connections to a myriad of biopsychosocial factors requires frameworks grounded in a multidimensional approach. The biopsychosocial model has long been used to assert that biological (e.g., genetics and intermediate brain phenotypes), psychological (e.g., mood and behaviors), and social factors (e.g., social relationships, economic status), are all significant contributors to health and disease^{2,3}. Indeed, the biopsychosocial model has been used to establish current diagnostic and clinical guidelines, such as the World Health Organization’s International Classification of Functioning, Disability and Health, and is considered central to person-centered care²⁷. Hence, statistical methods that enable us to interrogate the complex interconnected relationships within and between sleep and biopsychosocial factors can advance our understanding of optimal health and functioning across the lifespan. Multivariate data-driven techniques can help disentangle these complex interrelations, by deriving latent components that optimally relate multidimensional data sets in a single integrated analysis. A few studies have used such techniques to account for the multidimensional components of sleep and biopsychosocial factors separately^{15,28-32}. However, no study has integrated both multidimensional components of sleep and biopsychosocial factors to derive profiles that can account for the dynamic interplay among biopsychosocial factors, and link such components with brain network organization.

Deploying multivariate data-driven techniques requires a large sample size to identify latent components that can be generalised well³³⁻³⁵. One such optimal dataset is the Human Connectome Project dataset (HCP)³⁶ as it comprises a wide range of self-reported questionnaires about lifestyle, mental and physical health, personality and affect, as well as objective measures of physical health and cognition from over a thousand healthy young adults. Moreover, the HCP dataset stands out as one of the rare large-scale datasets that implemented a detailed assessment of sleep health, i.e., the Pittsburgh

Sleep Quality Index (PSQI)³⁷. This standardized sleep questionnaire, used both by clinicians and researchers, assesses different dimensions of sleep health in 19 individual items, creating 7 sub-components defining different dimensions of sleep, including sleep duration, satisfaction, and disturbances.

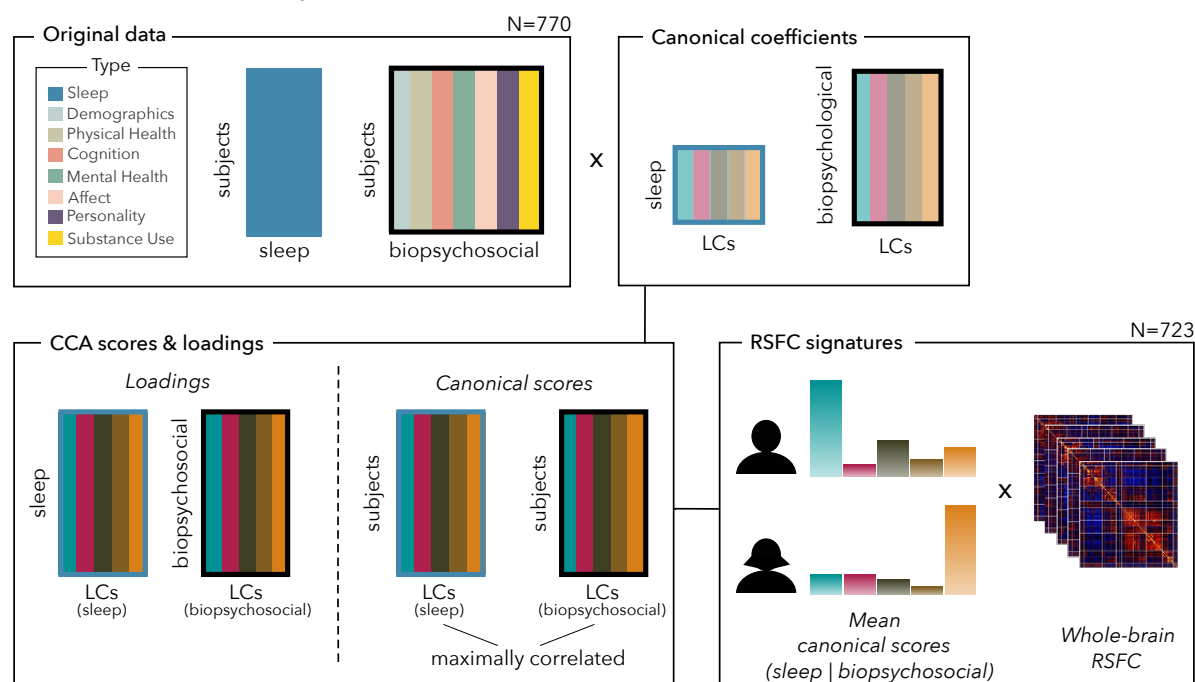
Beyond sleep-biopsychosocial profiling, the HCP dataset also provides the opportunity to explore the neural signatures of these sleep-biopsychosocial profiles using magnetic resonance imaging (MRI). Multiple studies have shown that neural signal fluctuation patterns during rest (i.e., resting-state functional connectivity; RSFC) are sensitive to sleep dimensions (e.g., sleep duration, sleep quality)^{14,15,32,38}, but also predictive of psychopathology (e.g., depressive symptoms, impulsivity)^{39,40} and cognitive performance^{14,38}. However, the way large-scale network organization may differentially affect individuals' variability in sleep, psychopathology, cognition and lifestyle, remains to be characterized beyond unidimensional association studies. Such holistic biopsychosocial approaches are not only in line with established diagnostic frameworks but also with initiatives such as the NIMH's Research Domain Criteria (RDoC) that encourage investigating mental disorders as continuous dimensions rather than distinct categories by integrating data from genomics, neural circuitry and behavior⁴¹⁻⁴³.

Identifying vulnerability markers constitutes a first step towards forecasting disease trajectories and designing multimodal multidimensional targeted therapies. Given the increasing recognition that sleep has a central role in health and well-being, we believe that sleep profiles should be included as a core aspect of these markers. Hence, in this study, we sought to take a multidimensional data-driven approach to identify sleep-biopsychosocial profiles that simultaneously relate self-reported sleep patterns to biopsychosocial factors of health, cognition, and lifestyle in the HCP cohort of healthy young adults³⁶. We further explored patterns of brain network organization associated with each profile to better understand their neurobiological underpinnings.

RESULTS

We applied canonical correlation analysis (CCA) to derive latent components (LCs) linking the 7 sub-components of the PSQI to 118 biopsychosocial measures (spanning cognitive performance, physical and mental health, personality traits, affects, substance use, and demographics; **Table S1**) in 770 healthy adults from the S1200 release of the HCP dataset³⁶ (**Figure 1A**). Participants were young adults between 22 and 36 years old (mean 28.86 ± 3.61 years old, 53.76% female), were generally employed full-time (70.7%) and were mostly white (78%; see **Table 1** for Demographics).

A. Canonical correlation analysis (CCA)



B. Sleep-biopsychosocial profiles (LCs)

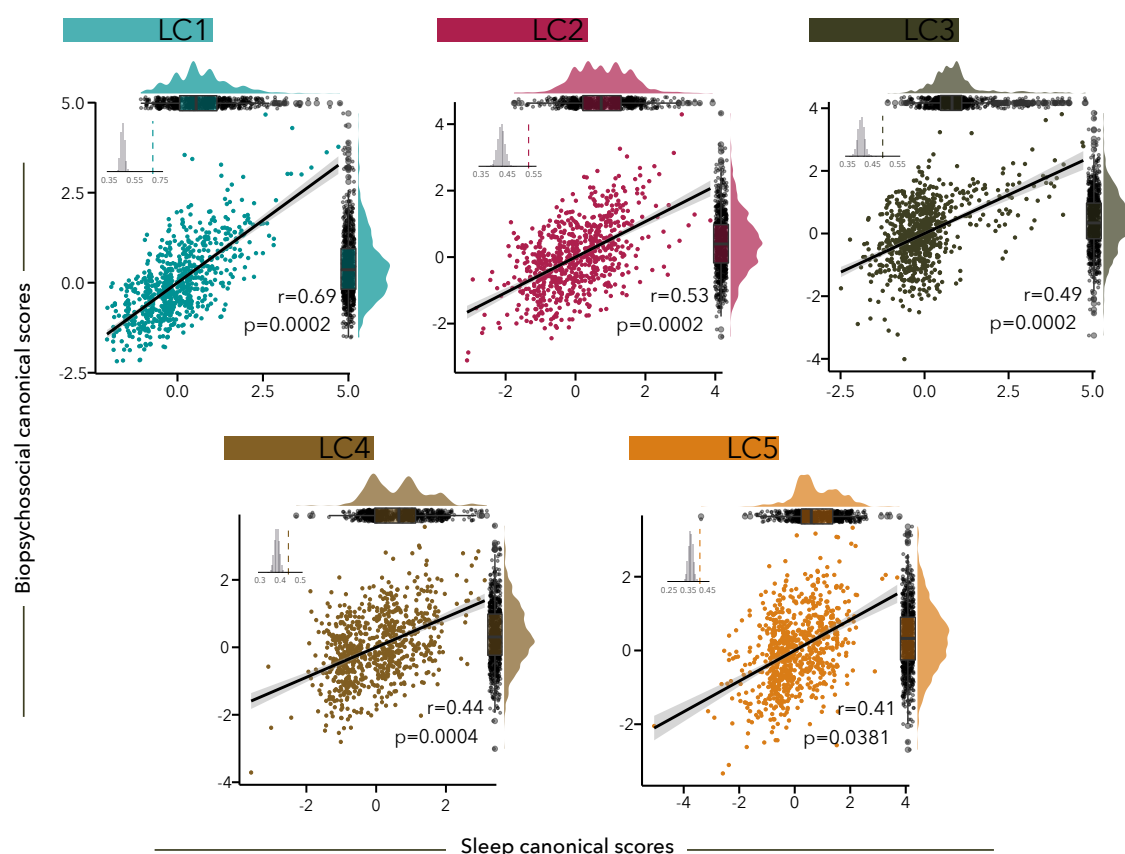


Figure 1 - Canonical correlation analysis reveals five sleep-biopsychosocial profiles (LCs).

(A) Canonical correlation analysis (CCA) flowchart and RSFC signatures; (B) Scatter plots showing correlations between biopsychosocial and sleep canonical scores. Each dot represents a different participant. Inset shows the null distribution of canonical correlations obtained by permutation testing; note that the null distribution is not

centered at zero. The dashed line indicates the actual canonical correlation computed for each LC. The distribution of sleep (top) and biopsychosocial (right) canonical scores is shown on rain cloud plots.

Five latent components (LCs) linking sleep and biopsychosocial factors.

Out of the seven significant LCs that were derived, 5 were interpretable LCs delineating multivariate relationships between sleep and biopsychosocial factors (**Figure 1B**; a description of LC6 and LC7 can be found in the **Supplementary Material – Figure S1**). While LC1 and LC2 were defined by general patterns of sleep (either general poor sleep or sleep resilience), LCs 3-5 reflected more specific sub-components of the PSQI, all associated with specific patterns of biopsychosocial factors. The 5 LCs respectively explained 88%, 4%, 3%, 2%, 1% of covariance between the sleep and biopsychosocial data.

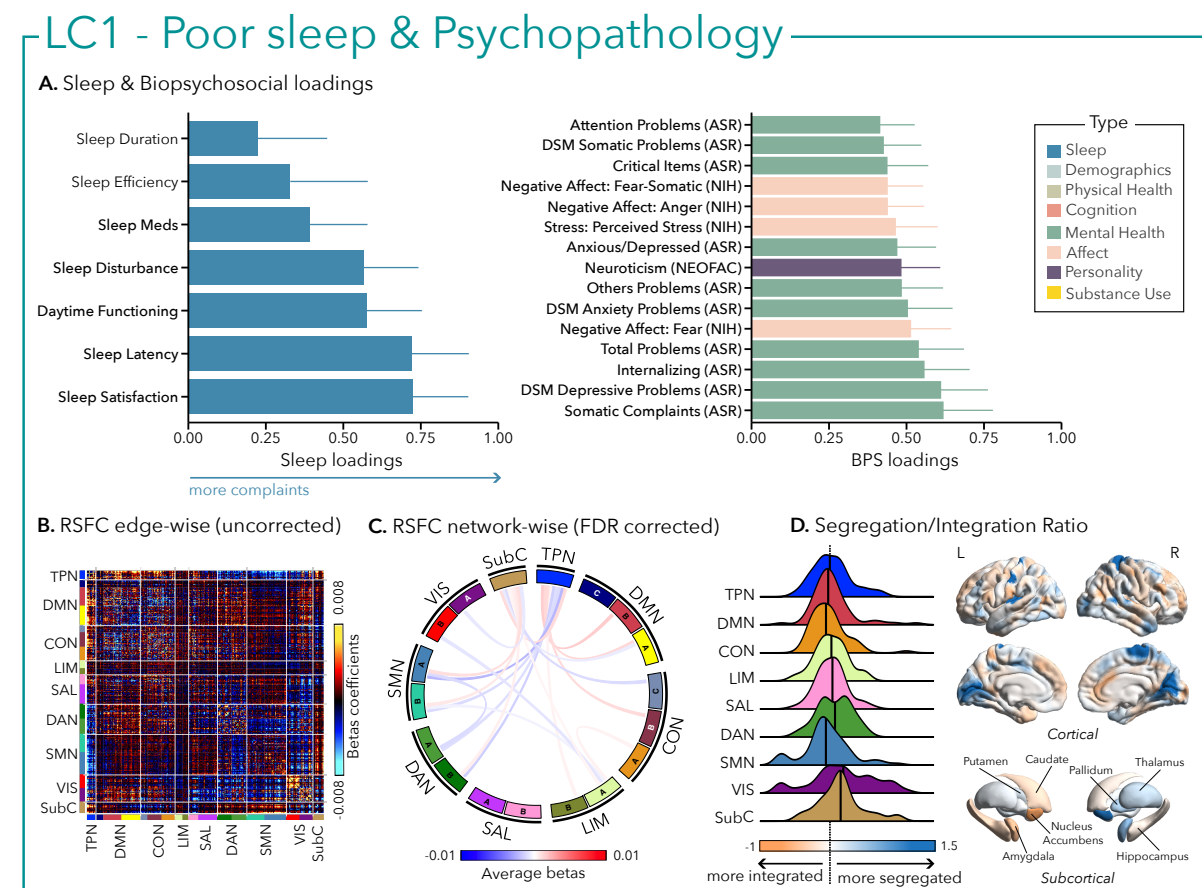


Figure 2 – The first latent component (LC1) reflects poor sleep and psychopathology.

(A) Sleep loadings (left) and top 15 strongest biopsychosocial (BPS) loadings (right) for LC1. Greater loadings on LC1 were associated with higher measures of poor sleep and psychopathology. Higher values on sleep (blue) and biopsychosocial (green, purple, pink) loadings indicate worse outcomes. Error bars indicate bootstrapped-estimated confidence intervals (i.e., standard deviation) and measures in bold indicate statistical significance (after FDR correction $q < 0.05$); (B) Unthresholded edge-wise beta coefficients obtained from generalized linear models (GLM) between participants' LC1 canonical scores (i.e., averaged sleep and biopsychosocial canonical scores) and their RSFC data; (C) FDR-corrected network-wise beta coefficients computed with GLMs within and between 17 large-scale brain networks⁴⁴ and subcortical regions⁴⁵. (D) Distribution of the integration/segregation ratio in each of the 7 large-scale brain networks and subcortical regions associated with LC1 (left). The dashed line indicates the median of all parcels, and the bold black lines represent the median for each network. The integration/segregation ratio values for the 400 Schaeffer parcellation⁴⁶ and 7 subcortical regions are projected on cortical and subcortical surfaces (right).

LC1 was characterized by a general pattern of poor sleep, including decreased sleep satisfaction, longer time to fall asleep, greater complaints of sleep disturbances and daytime impairment, as well as greater (i.e., worse) psychopathology (e.g., depression, anxiety, somatic complaints, internalizing behavior) and negative affect (e.g., fear, anger, stress – **Figure 2A**).

Similarly, LC2 was also driven by greater psychopathology, especially attentional problems (e.g., inattention, ADHD), low conscientiousness, and negative affect (**Figure 3A**). In terms of sleep, however, in contrast to the first LC, greater psychopathology was only related to higher complaints of daytime impairment without complaints of sleep difficulties, suggesting sleep resilience.

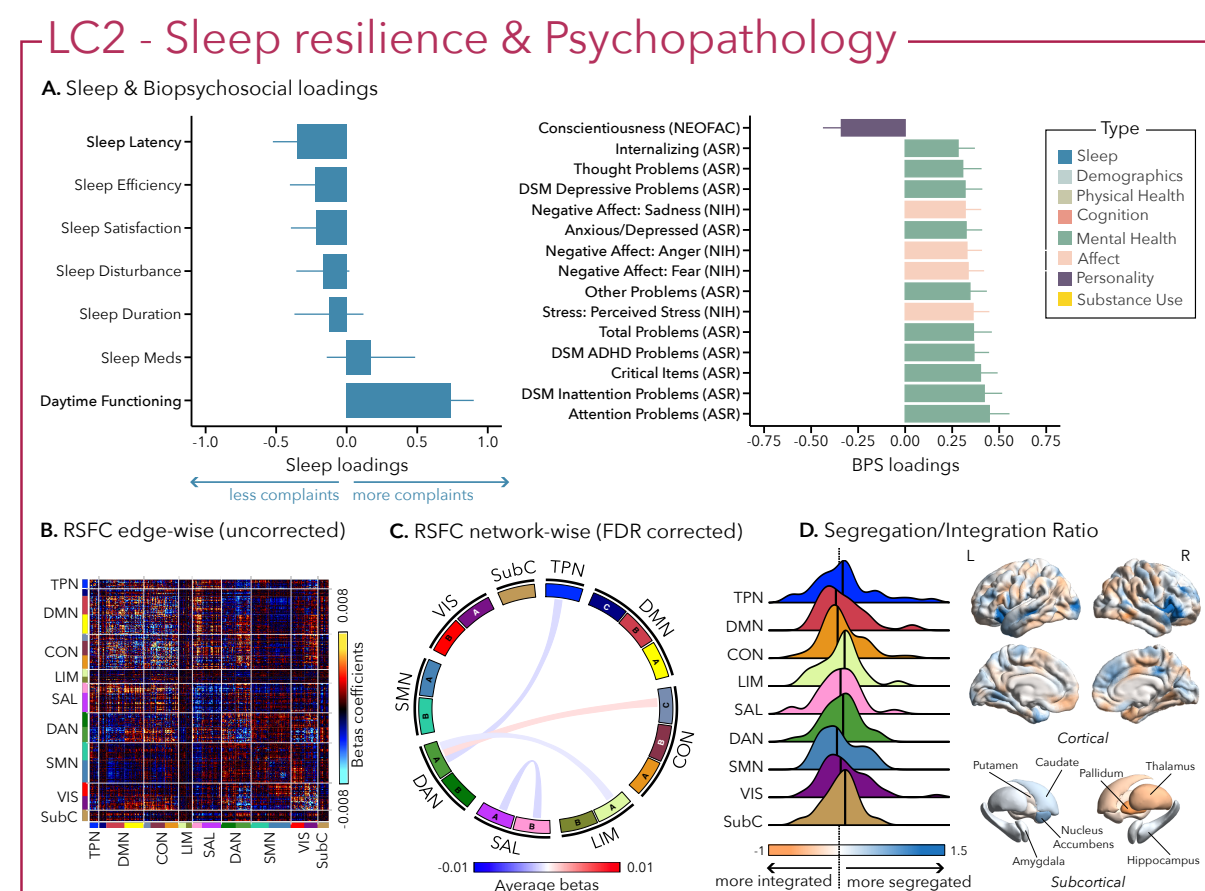


Figure 3 – The second latent component (LC2) reflects sleep resilience and psychopathology.

(A) Sleep loadings (left) and top 15 strongest biopsychosocial (BPS) loadings (right) for LC2. Greater loadings on LC2 were associated with higher measures of complaints of daytime dysfunction and psychopathology. Positive values on sleep (blue) loadings indicate worse outcomes while positive values on biopsychosocial (green, purple, pink) loadings reflect higher magnitude on these measures. Error bars indicate bootstrapped-estimated confidence intervals (i.e., standard deviation) and measures in bold indicate statistical significance. (B) Unthresholded edge-wise beta coefficients obtained from generalized linear models (GLM) between participants' LC2 canonical scores (i.e., averaged sleep and biopsychosocial canonical scores) and their RSFC data; (C) FDR-corrected network-wise beta coefficients computed with GLMs within and between 17 large-scale brain networks⁴⁴ and subcortical regions⁴⁵. (D) Distribution of the integration/segregation ratio in each of the 7 large-scale brain networks and subcortical regions associated with LC2 (left). The dashed line indicates the median of all parcels, and the bold black lines represent the median for each network. The integration/segregation ratio values for the 400 Schaeffer parcellation⁴⁶ and 7 subcortical regions are projected on cortical and subcortical surfaces (right).

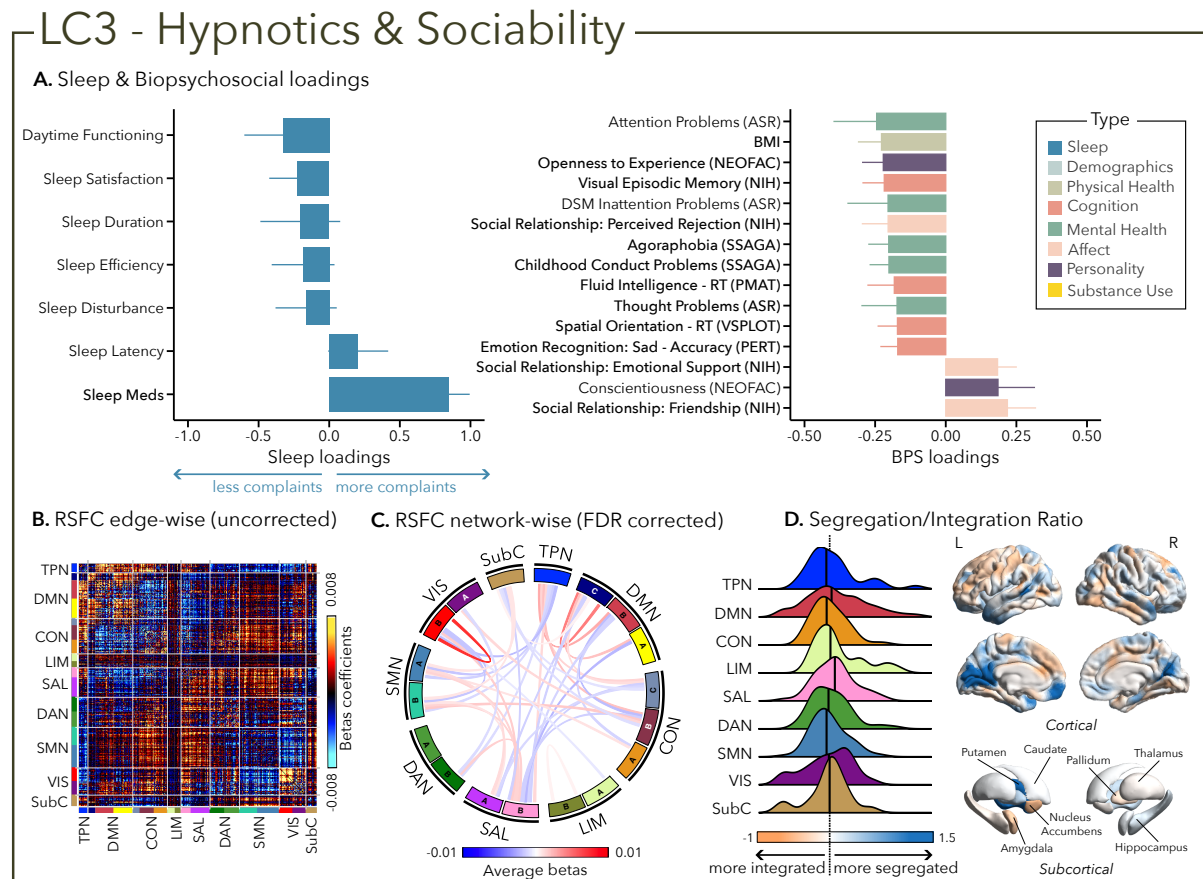


Figure 4 – The third latent component (LC3) reflects hypnotics and sociability.

(A) Sleep loadings (left) and top 15 strongest biopsychosocial (BPS) loadings (right) for LC3. Greater loadings on LC3 were associated with the use of sleep aids and measures of positive social relationships, lower body mass index (BMI) and poor visual episodic memory performance. Positive values on sleep (blue) loadings indicate worse outcomes while positive values on the mental health (green), affect (pink) and personality (purple) categories of biopsychosocial loadings reflect higher magnitude on these measures. Positive value in the physical health (olive) category represents higher value and positive values in the cognition (orange) category indicate either higher accuracies or slower reaction times (RT). Error bars indicate bootstrapped-estimated confidence intervals (i.e., standard deviation) and measures in bold indicate statistical significance. (B) Unthresholded edge-wise beta coefficients obtained from generalized linear models (GLM) between participants' LC3 canonical scores (i.e., averaged sleep and biopsychosocial canonical scores) and their RSFC data; (C) FDR-corrected network-wise beta coefficients computed with GLMs within and between 17 large-scale brain networks⁴⁴ and subcortical regions⁴⁵. (D) Distribution of the integration/segregation ratio in each of the 7 large-scale brain networks and subcortical regions associated with LC3 (left). The dashed line indicates the median of all parcels, and the bold black lines represent the median for each network. The integration/segregation ratio values for the 400 Schaeffer parcellation⁴⁶ and 7 subcortical regions are projected on cortical and subcortical surfaces (right).

LC3 was mostly characterized by sleep-aids intake (i.e., sleep meds PSQI sub-component) and to a lesser extent a lack of daytime functioning complaint. Surprisingly, LC3 was not driven by any attentional problem but was related to worse performance in visual episodic memory and emotional recognition. Moreover, sleep aids/hypnotics intake was mainly related to satisfaction in social relationships (**Figure 4A**).

While LC4 was solely driven by sleep duration (i.e., not sleeping enough - reporting <6-7h per night), LC5 was mostly characterized by the presence of sleep disturbances that can encompass multiple awakenings, nocturia and breathing issues as well as pain or temperature imbalance. In LC4, short sleep duration was associated with worse accuracy and longer reaction time at multiple cognitive tasks tapping into emotional processing, delayed reward discounting, language, fluid intelligence, and social

cognition. LC4 was also characterized by higher aggressive behavior and lower agreeableness (**Figure 5A**).

Interestingly, sleep disturbances in LC5 were also associated with aggressive behavior and worse cognitive performance (e.g., in language processing and working memory), but were mostly characterized by critical items on mental health assessments (i.e., anxiety, thought problems, internalization) and substance abuse (i.e., alcohol and cigarette use – **Figure 6A**).

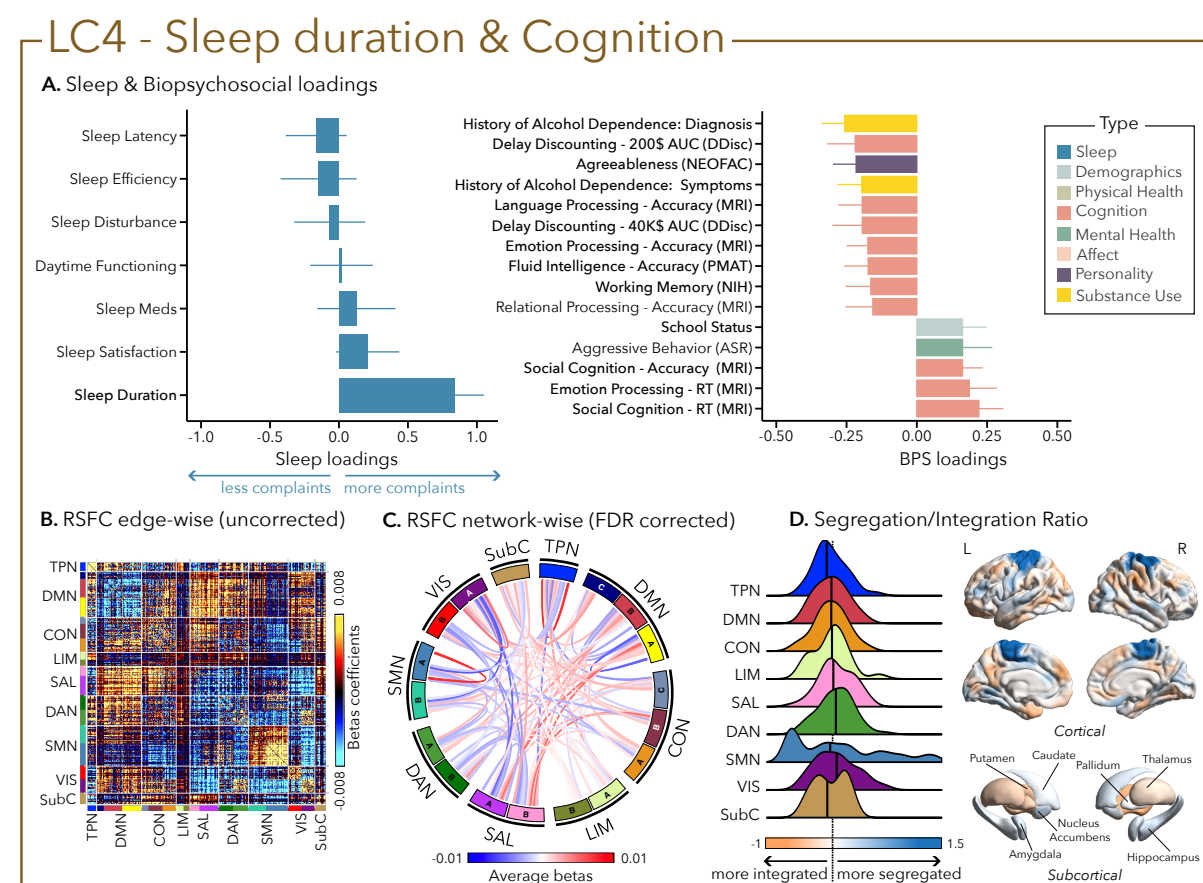


Figure 5 – The fourth latent component (LC4) reflects sleep duration and cognition.

(A) Sleep loadings (left) and top 15 strongest biopsychosocial (BPS) loadings (right) for LC4. Greater loadings on LC4 were associated with shorter sleep duration and measures of poor cognitive performance. Positive values on sleep loadings indicate worse outcomes while positive values on the mental health (green), substance use (yellow), demographics (light blue) and personality (purple) categories of biopsychosocial loadings reflect higher magnitude on the measures. Positive values in the cognition (orange) category indicate either higher accuracies or slower reaction times (RT). Error bars indicate bootstrapped-estimated confidence intervals (i.e., standard deviation) and measures in bold indicate statistical significance. (B) Unthresholded edge-wise beta coefficients obtained from generalized linear models (GLM) between participants' LC4 canonical scores (i.e., averaged sleep and biopsychosocial canonical scores) and their RSFC data; (C) FDR-corrected network-wise beta coefficients computed with GLMs within and between 17 large-scale brain networks⁴⁴ and subcortical regions⁴⁵. (D) Distribution of the integration/segregation ratio in each of the 7 large-scale brain networks and subcortical regions associated with LC4 (left). The dashed line indicates the median of all parcels, and the bold black lines represent the median for each network. The integration/segregation ratio values for the 400 Schaeffer parcellation⁴⁶ and 7 subcortical regions are projected on cortical and subcortical surfaces (right).

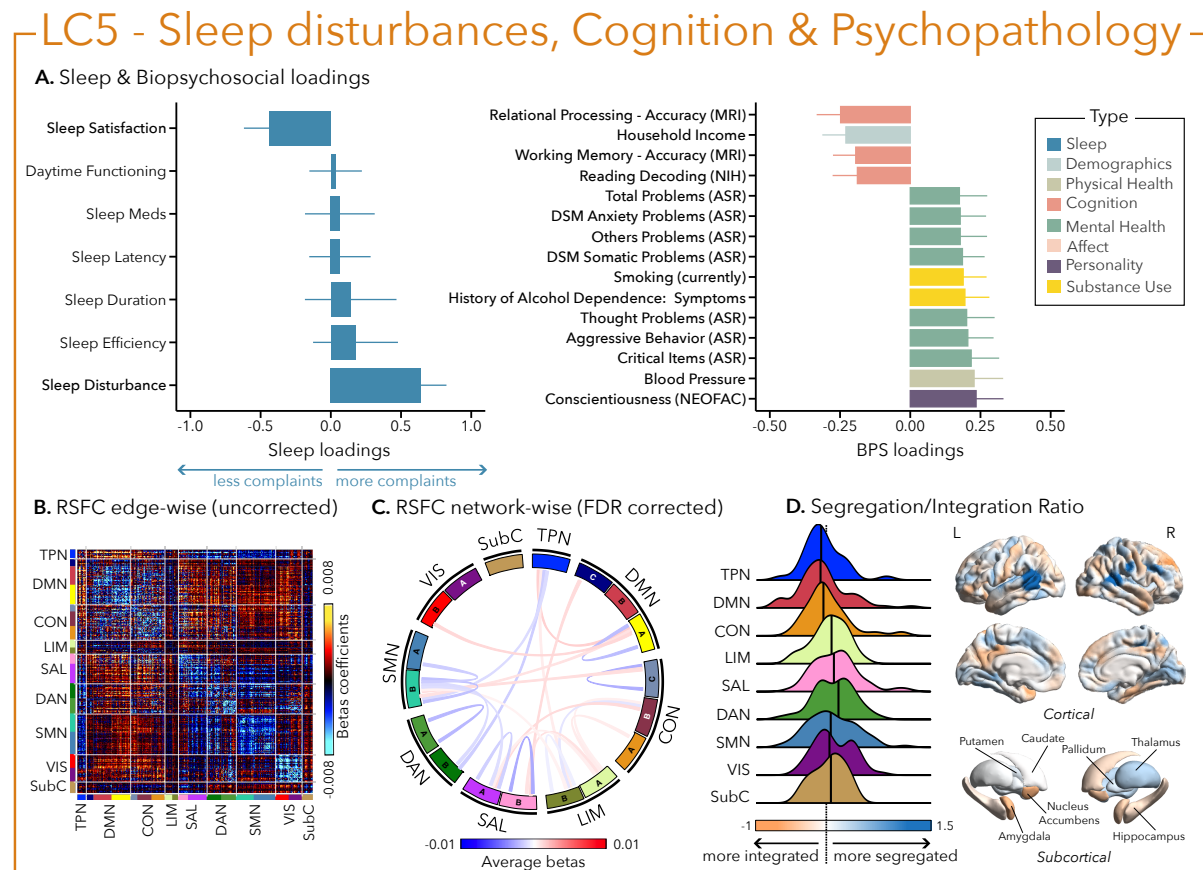


Figure 6 – The fifth latent component (LC5) reflects sleep disturbance, cognition and psychopathology.

(A) Sleep loadings (left) and top 15 strongest biopsychosocial (BPS) loadings (right) for LC5. Greater loadings on LC5 were associated with the presence of sleep disturbances, higher measures of psychopathology and lower cognitive performance. Positive values on sleep loadings indicate worse outcomes while positive values on the mental health (green), substance use (yellow) and personality (purple) categories of biopsychosocial loadings reflect higher magnitude on these measures. Positive values in the cognition (orange) category indicate either higher accuracies or slower reaction times (RT), while positive values in the demographics (light blue) and physical health (olive) categories represent higher values. Error bars indicate bootstrapped-estimated confidence intervals (i.e., standard deviation) and measures in bold indicate statistical significance. (B) Unthresholded edge-wise beta coefficients obtained from generalized linear models (GLM) between participants' LC5 canonical scores (i.e., averaged sleep and biopsychosocial canonical scores) and their RSFC data; (C) FDR-corrected network-wise beta coefficients computed with GLMs within and between 17 large-scale brain networks⁴⁴ and subcortical regions⁴⁵. (D) Distribution of the integration/segregation ratio in each of the 7 large-scale brain networks and subcortical regions associated with LC5 (left). The dashed line indicates the median of all parcels, and the bold black lines represent the median for each network. The integration/segregation ratio values for the 400 Schaeffer parcellation⁴⁶ and 7 subcortical regions are projected on cortical and subcortical surfaces (right).

Sleep and biopsychosocial profiles exhibit distinct signatures of resting-state brain connectivity.

In terms of brain organization, the 5 LCs revealed distinct patterns of network connectivity. Specifically, we examined patterns of both within-network and between-network connectivity (see **Figure S2** for subcortical-cortical patterns).

Greater (averaged) biopsychosocial and sleep composite scores on LC1 were associated with increased RSFC between subcortical areas and the somatomotor and dorsal attention networks (**Figures 2B** and **2C**), and a decreased RSFC between the temporoparietal network and these two networks. The visual network showed a flattened distribution of segregation/integration ratio (i.e., more variability in segregation and integration among the parcels of the network). The amygdala and nucleus accumbens exhibited asymmetrical patterns in the segregation/integration ratio with the left side being more

segregated (**Figure 2D**). Meanwhile, LC2 was associated with increased RSFC between the dorsal attention and control network but decreased RSFC between dorsal attention and the temporoparietal and limbic networks (**Figures 3B and 3C**), a higher segregation of nodes within the temporoparietal network and increased integration within the right thalamus (**Figure 3D**). Higher composite scores in LC3 were associated with increased RSFC within the visual and default mode networks (**Figures 4B and 4C**). The segregation/integration ratio within the default mode exhibited a flattened distribution (i.e., high variability in segregation and integration among parcels) but there was an increased segregation in the limbic and visual networks (**Figure 4D**). While greater composite scores in LC4 were associated with widespread patterns of hypo- or hyper-connectivity within and between every network the somatomotor network specifically exhibited an altered pattern of segregation and integration (**Figures 5B to 5D**). Finally, we found that greater averaged composite scores in LC5 were mainly associated with reduced within-network connectivity in the somatomotor, dorsal and ventral attention networks (**Figures 6B and 6C**) but no strong pattern of segregation/integration ratio change (**Figure 6D**).

Post-hoc associations with socio-demographics, health, and family history of mental health

We found a number of significant associations between LC composite scores and socio-economic (e.g., education level, household income) and socio-demographic factors (e.g., race, ethnicity; see **Table S4** and **Supplemental Results**). In brief, most profiles (LCs 1,4,5) showed significant associations between sleep-biopsychosocial composite scores and education level, where lower education level was associated with a higher composite score in LCs 1,4,5 (all $q < 0.05$). Similarly, lower household income correlated with a higher composite score in LCs 1-2 (all $q < 0.05$). Race and ethnicity groups revealed differences in composite sleep and biopsychosocial scores for LCs 1,3-5 (all $q < 0.05$). Finally, while the presence of a family history of psychopathology was associated with higher biopsychosocial scores in LCs 1-2, we only found biological sex differences in LC5, with higher sleep and biopsychosocial composite scores in female participants ($q < 0.05$).

Control analyses

We summarize several analyses that demonstrate the robustness of our findings. First, LC1 and LC2 successfully generalized in our cross-correlation scheme (mean across 5 folds: $r = 0.49$, $p = 0.001$; $r = 0.19$, $p = 0.039$ respectively), but not LCs 3-5 (see **Table S3**), suggesting that LCs 3-5 might not be as robust and generalizable, possibly due to these LCs being driven by a single sleep dimension. Second, we re-computed the CCA analysis after: (i) applying quantile normalization on sleep and biopsychosocial measures; (ii) excluding participants that had tested positive for any substance on the day of the MRI; (iii) excluding physical health measures (i.e., body mass index, hematocrit, blood pressure) or (iv) sociodemographic variables (i.e., employment status, household income, school status, relationship status) from the biopsychosocial matrix. The CCA loadings remained mostly unchanged (**Table S5**). We also assessed the robustness of our imaging results in several ways. First, we re-computed the GLM analysis using RSFC data that underwent CompCor⁴⁷ instead of GSR. The RSFC patterns were not much altered, as shown by generally high correlations with the main analysis ($r = 0.75$, $r = 0.76$, $r = 0.78$, $r = 0.51$, $r = 0.77$ for LCs 1-5 respectively; **Figure S3**). Next, excluding subjects that likely fell asleep in the scanner did not impact our findings ($r = 0.90$, $r = 0.87$, $r = 0.95$, $r = 0.95$, $r = 0.95$ for LCs 1-5 respectively; **Figure S3**); however, we found that these participants had higher sleep and biopsychosocial composite scores on LC4 compared to participants that likely stayed awake during the scan (**Figure S4**). Finally, we re-computed the GLM analyses by using sleep and biopsychosocial canonical scores instead of averaged scores. We found moderate to high correlations with the main GLM analysis ($r = 0.69$, $r = 0.62$, $r = 0.63$, $r = 0.46$, $r = 0.67$ for LCs 1-5 respectively; **Figure S3**).

DISCUSSION

Leveraging a multidimensional data-driven approach in a large cohort of healthy young adults, we uncovered five distinct sleep profiles linked to biopsychosocial factors encompassing health, cognition, and lifestyle. We found that the first two profiles reflected general psychopathology (or *p* factor) associated with either reports of general poor sleep (LC1) or an absence of sleep complaints, which we defined as sleep resilience (LC2). Meanwhile, the three other profiles were driven by a specific dimension of sleep such as the use of sleep aids (LC3), sleep duration (LC4), or sleep disturbances (LC5), which were associated with distinct patterns of health, cognition, and lifestyle factors. Furthermore, identified sleep-biopsychosocial profiles displayed unique patterns of brain network organization. Our findings emphasize the crucial interplay between biopsychosocial outcomes and sleep, and the necessity to integrate sleep history to contextualize research findings and to inform clinical intake assessments⁴⁸.

The dominance of psychopathology markers in most of the profiles is not surprising as the RDoC framework proposed arousal and regulatory systems (i.e., circadian rhythms and sleep/wakefulness) as one of the five key domains of human functioning likely to affect mental health⁴⁹, which is consistent with a large literature reporting significant disruption of sleep across multiple psychiatric disorders^{8,50}. Although individuals with a neuropsychiatric diagnosis (e.g., schizophrenia or major depression disorder) were not included in the HCP dataset³⁶, the presence of the *p* factor, defined as an individual's susceptibility to develop any common form of psychopathology, exists on a continuum of severity and chronicity within the general population⁵¹. Symptoms of psychopathology mirrored each other across LC1 and LC2 but the paradoxical contrast in sleep loadings suggests that some individuals might have more resilient sleep (LC2), whereby they might be able to maintain healthy sleep patterns in the face of psychopathology. However, the cause of such resilience is unclear. Up to 80% of individuals experiencing an acute phase of mental disorder (e.g., depressive and/or anxiety episode) report sleep issues^{8,52,53}, leaving a minority of individuals who do not report abnormal sleep during such episodes. The identification of LC2 supports this and suggests there might be biological or environmental protective factors in some individuals who would otherwise be considered at risk for sleep issues. However, our understanding of such protective factors is limited^{54–56}. These findings also highlight the need to appreciate the complexity of psychopathology, in line with the current view that psychiatric disorders are typically comorbid and heterogeneously expressed. Nonetheless, whether this profile of sleep resilience is a stable latent component or a cross-sectional observation of fluctuating symptoms that may develop into psychopathology-related sleep complaints, needs to be further tested. Interestingly, distinctions between LC1 and LC2 were also present in the neural signatures of RSFC, which may assist in the neurobiological interpretation of the profiles. Visually inspecting LC1 and LC2 suggested an underlying increase in subcortical-cortical connectivity when sleep disturbances are associated with psychopathology. This is in alignment with the known neurophysiology of the ascending arousal system and possibly implies the existence of some level of hyperarousal in these pathways that may contribute to disturbances in sleep⁵⁷. However, this speculation requires further targeted research to be confirmed.

Within the profiles driven by a specific sleep sub-component, LC5 also reflected some dimensions of psychopathology (i.e., anxiety, critical items and thought problems) that were only associated with the presence of global sleep disturbances. The sleep disturbance sub-component of the PSQI is broad and encompasses complaints of sleep-related breathing problems as well as multiple awakenings that could be due to nycturia, pain, nightmares, or difficulties maintaining optimal body temperature³⁷. Altogether, the sleep disturbances dimension is thought to represent sleep fragmentation⁵⁸, and thus, sleep quality. This is in line with a recent study in a large community-based cohort (i.e., UK Biobank) that found that lifetime diagnoses of psychopathology and psychiatric polygenic risk scores were more strongly associated with accelerometer-derived measures of sleep quality (i.e., fragmentation) than sleep duration per se⁵⁹. Similarly, we found that sleep duration (driving LC4) was not associated with measures

of psychopathology but rather with cognitive performance. Whether studied in the form of experimental acute sleep deprivation and chronic sleep restriction or clinical populations (e.g., insomnia with objective short sleep duration), the consequences of lack of sleep on daytime functioning and health are well-known and substantial^{11,12,16,60–62}. Sleep duration affects, in varying effect sizes, both accuracy and reaction time in most cognitive tasks^{11,12,61}. In our study, reports of regular short sleep duration, defined as <6-7h of total sleep time, was associated with reduced accuracy in working memory, emotional processing, language processing, delay discounting, fluid intelligence as well as longer reaction times during social cognition and emotional processing, mimicking results found in the sleep deprivation and sleep restriction literature^{11,14,61–66}. Interestingly, the strong RSFC patterns associated with LC4 showed a global increase in connectivity, with localized segregation of part of the somatomotor network, which had been previously reported in neuroimaging studies of experimental acute total sleep deprivation^{64,67}. Hence, this suggests that LC4 may be displaying an underlying level of sleep debt in the uncontrolled general population.

Finally, beyond sleep measures and sleep-related daytime functioning, the PSQI also evaluates the use of medication to help sleeping³⁷, whether prescribed or over-the-counter (e.g., gamma-aminobutyric acid GABA_A receptor modulators, selective melatonin receptor agonists, selective histamine receptor antagonists, cannabinoid products, valerian)⁶⁸. We found that LC3 was driven by the use of sleep aids and was mostly associated with reports of satisfaction in social relationships. Interestingly, while we would have expected more links between the use of sleep aids and cognitive impairment, especially in older adults^{69,70}, we only found an association with visual working memory deficits but not with attentional problems. This profile specifically highlights a sub-group of young adults (22-36 years old) who experience sleep complaints and seek pharmacological solutions to manage them. As such, the associated biopsychosocial factors, in particular high sociability, could result from the effect of the drug itself on social behavior and positive mood (e.g., via potentiation of GABA transmission)^{71,72} or as a consequence of the drugs on sleep complaints⁷³, which may support better emotional regulation and well-being, and consequently translate to greater satisfaction in social relationships and support systems^{73,74}. We did not have information on the type nor duration of sleep aid usage as the PSQI only assesses sleep habits in the past month, which may not be a substantial period of time to observe robust changes in cognitive functioning as previously documented^{70,75} or the development of substance abuse. Indeed, while the chronic misuse of hypnotics can lead to dependence and addiction⁷⁶, in our sample, the use of sleep aids was not associated with substance abuse.

Interestingly, alterations to the segregation/integration ratio of the somatomotor and visual cortex were common in most profiles. Highly interconnected to the whole brain, the somatomotor network is crucial for processing external stimuli and producing motor responses but is also functionally involved in bodily self-consciousness and interoception. Altered dysconnectivity patterns of the somatomotor network have been linked to variation in several domains, including general psychopathology^{77,78}, cognitive dysfunction related to sleep deprivation⁶⁴, as well as the total PSQI score^{13,79}. Overall, these findings suggest that alterations to RSFC in the somatomotor network are also involved in the relationships between sleep and biopsychosocial factors and highlight the importance of understanding the role of this brain network in overall mental health and functioning.

These profiles contribute to a deeper understanding of the current debate that opposes sleep quality and sleep duration^{7,80}. In line with previous studies^{11,12,81}, we found that cognitive functioning was more related to sleep duration than subjective sleep quality; in addition, we found that sleep disturbances, alone (LC5) or in combination with other sleep dimensions (LC1), can be associated with the presence of psychopathology. Moreover, it is also important to note that complaints of poor sleep quality and/or short sleep duration have been both associated with increased risks of physical health outcomes and all-cause mortality^{6,7}. While LC1 and LC2 presented sleep dimensions as being inextricably linked, LC3, LC4 and LC5 respectively revealed distinct facets of sleep, suggesting that while sleep dimensions are related, they can also be separable domains with specific connections to biopsychosocial factors. This is

likely reflected in the finding that only LC1 and LC2 were replicable in cross-validation analyses, which may be due to LC3, LC4 and LC5 being driven by a single sleep dimension and thus, contributing only marginally to the variance. Moreover, LC1 captured a large portion of the covariance, which could be due to the presence of collinearity among the sleep and/or biopsychosocial measures. We did not apply dimensionality reduction (or any other regression techniques) to the biopsychosocial measures prior to the CCA, as the number of subjects was higher than the number of measures, and because CCA removes within-block correlations, thus allowing the amplification of specific variables that mostly characterize the correlation between sleep and biopsychosocial measures⁸². In addition, we focused on structure coefficients (not canonical coefficients) to better identify the contribution of the variables to each profile, and then computed bootstrap analyses to further assess the stability of these variables across resampling.

While unidimensional association studies have been informative, these findings reinforce the notion that sleep health is multidimensional and distinct measures of sleep quantity or quality should be considered together when investigating their influence on biopsychosocial aspects of health, cognition, and lifestyle. Future work should extend these findings and further explore the multidimensional nature of sleep health, for instance, taking into consideration the U-shaped relationship of sleep duration with biopsychosocial measures. Given the design of the PSQI, only short sleep duration (<5-6h) was considered as a sleep difficulty, neglecting the potential consequences of long sleep duration (>9h). Long sleep duration is commonly observed in hypersomnia disorders and psychopathology (e.g., schizophrenia, depression)^{6,83}, as well as being associated with increased risk of cardiovascular heart disease and mortality^{7,84,85} and cognitive decline^{6,20}. This U-shape observation, whereby both short and long sleep durations are associated with negative health and cognitive consequences as well as increasing markers of cerebrovascular burden (e.g., white matter hyper-intensities)⁵⁵, may provide a window to identify mechanisms that underlie the interplay between sleep and biopsychosocial factors. Other considerations moving forward include sleep regularity and sleep timing, which are not part of the computation of the sub-components of PSQI³⁷, hence their association with biopsychosocial outcomes were not investigated in this study. Furthermore, the PSQI is often interpreted with regard to its total score (combining all sub-components), which provides a binary vision of sleep quality (either good or bad sleep)³⁷. In this study, we did not want to be limited by the PSQI global score but rather aimed to untangle the different dimensions (subcomponents) of sleep and their relations to biopsychosocial and neurobiological measures.

A final important distinction to be addressed is that sleep and biopsychosocial outcomes were mostly self-reported through questionnaires. Both objectively recorded and subjectively perceived estimations provide different yet meaningful information that tends to positively correlate⁸⁶. However, it has been shown that when compared to objective estimates (i.e., polysomnography and/or actigraphy recordings), individuals with sleep complaints (i.e., chronic insomnia, obstructive sleep apnea) tend to subjectively misperceive their sleep (i.e., duration, sleep latency)^{25,26,87,88}. The degree of discrepancy between objective and subjective measures (i.e., sleep state misperception) has been correlated with worse sleep quality^{89,90} as well as compromised reports of daytime functioning²⁴. While objective measurements might have exposed divergent associations between sleep and biopsychosocial factors, the profiles reported here arguably support greater clinical validity, where the subjective complaints are often what drives an individual to seek out healthcare. Our study emphasizes that considering individuals' sleep experience can support clinicians to make more accurate initial assessments and navigate the course of treatment and interventions.

The awareness and interest surrounding sleep as a crucial pillar of health is growing rapidly⁹¹. However, the role of sleep in general health is complex, multifaceted, and largely unknown. The multidimensional approach applied in this large sample of healthy young adults is a first step that we argue should be implemented in future research incorporating sleep. We highlight the observation of five distinct sleep patterns associated with specific combinations of biological, psychological and socio-environmental factors. These findings support that sleep is emerging as a distinguishable factor that can assist in

disentangling the complex heterogeneity of human health. As the capacity for large-scale human research continues to grow, integrating sleep dimensions at such a scale is not only feasible in terms of evaluation, but presents a unique opportunity for translational application. Sleep is a modifiable lifestyle factor and can be investigated in model organisms as well as in humans, and as such is well positioned to identify potential converging mechanisms and intervention pathways or tools. The current study emphasizes that by using a multidimensional approach to identify distinct sleep-biopsychosocial profiles we can begin to untangle the interplay between individuals' variability in sleep, health, cognition, lifestyle, and behavior—equipping research and clinical settings to better support individuals' well-being.

METHODS

Participants

Data for this study were obtained from the S1200 release of the publicly available Human Connectome Project (HCP) dataset³⁶. The HCP dataset comprises multimodal MRI data, including structural MRI, diffusion MRI, resting-state and task functional MRI (fMRI) data, as well as a broad range of behavioral measures collected in young healthy subjects (aged 22-36). Details about imaging acquisition parameters and data collection³⁶ as well as the list of available behavioral and demographics measures (HCP S1200 Data Dictionary)⁹² can be found elsewhere. Of note, the HCP dataset comprises a large number of related individuals (i.e., siblings and twins). Of the 1,206 total subjects available from the HCP S1200 release, we excluded 403 participants with missing/incomplete data, and 33 participants with visual impairment that might have impacted their task performance in the scanner. Our final sample comprised 770 participants (53.76% female, 28.86 ± 3.61 years old). We decided to keep participants (N=94) who tested positive for any substance (including alcohol, marijuana, and other drugs) on the day of the MRI, as substance use has intricate links to sleep, and we did not want to exclude the possibility of finding potential substance use-related sleep profiles. However, we also re-computed our analyses after excluding these individuals (N=676) and found very similar results (see **Table S5**). Out of these 770 participants, 723 passed MRI quality control and were included in the *posthoc* RSFC analyses.

Sleep assessment

Participants were administered the Pittsburgh Sleep Quality Index³⁷ (PSQI) to assess different aspects of their sleep over the past month. To define sleep in our study, we used the 7 sub-components of the PSQI which characterize different sleep dimensions, namely (i) sleep satisfaction, (ii) sleep latency, (iii) sleep duration, (iv) sleep efficiency, (v) sleep disturbance, (vi) sleep medication, and (vii) daytime functioning. Sub-components are calculated through 4 questions on the timing of sleep habits and 6 Likert-scale questions from 0 to 3, 0 being best and 3 being worst.

Biopsychosocial assessment

118 biopsychosocial measures were selected from the HCP dataset (see complete list in **Table S1**). These measures included self-reported assessments of current and past mental health and substance use, questionnaires on personality, affect, lifestyle and demographics, cognitive tasks tapping on different processes such as working memory or social cognition performed either inside or outside the MRI, and physical assessments (e.g., blood pressure). These measures did not undergo any dimensionality reduction or clustering by biopsychosocial domain in order to preserve granularity in the way they would be associated with sleep dimensions. Biopsychosocial measures with large amounts of missing data were excluded, as well as similar measures that were likely to be redundant. Biopsychosocial measures were categorized by behavioral domain (e.g., cognition, physical health) based on the way they had been described in the HCP dataset^{36,92}.

Canonical correlation analysis

Canonical Correlation Analysis (CCA)^{93,94}, a multivariate data-driven approach, was applied to the sleep and biopsychosocial measures. CCA derives latent components (LCs, i.e., canonical variates), which are optimal linear combinations of the original data, by maximizing *correlation* between two data matrices (i.e., sleep and biopsychosocial measures). The rank of the correlation matrix determines the number of derived LCs (i.e., in this case the number of sleep measures, hence 7 LCs). Each sleep-biopsychosocial LC is characterized by a pattern of sleep weights and a corresponding pattern of biopsychosocial weights (i.e., canonical coefficients). Linear projection of sleep (or biopsychosocial) data onto sleep (or biopsychosocial) weights yielded participant-specific composite scores for sleep (or biopsychosocial) measures (i.e., canonical scores). The contribution of original sleep and biopsychosocial loadings to each LC was determined by computing Pearson's correlations between sleep (or biopsychosocial) data and participant-specific scores for sleep (or biopsychosocial factors) to obtain sleep and biopsychosocial *loadings* (i.e., canonical structure coefficients)^{95,96}. Canonical structure coefficients reflect the direct contribution of a predictor (e.g., one sleep dimension) to the predictor criterion (e.g., LC1) independently of other predictors (e.g., LCs 2-7), which can be critical when predictors are highly correlated between each other (i.e., in presence of multicollinearity)⁹⁷. We did not employ dimensionality reduction (e.g., via principal components analysis), as the sample size (N=770) exceeded the number of sleep (7 measures) and biopsychosocial measures (118 measures) being modelled. Statistical significance of each of the 7 LCs was determined by permutation testing (10,000 permutations) followed by FDR correction. Given the high prevalence of related participants in the HCP dataset, family structure was maintained during permutations (using the PALM package^{98,99}), whereby monozygotic twins, dizygotic twins, and non-twin siblings were only permuted within their respective groups. Finally, the loadings' stability was determined using bootstrap resampling to estimate confidence intervals for the loadings, by deriving 1,000 samples with replacement from participants' sleep and biopsychosocial data.

MRI acquisition and processing

All imaging data were acquired on a customized Siemens 3T Skyra scanner at Washington University (St Louis, MI). Four runs of resting-state fMRI were collected over two sessions across two separate days. Each run included 1,200 frames using a multi-band sequence at 2-mm isotropic spatial resolution with a TR of 0.72 s for 14.4 minutes. The structural images were acquired at 0.7-mm isotropic resolution. Further details of the data collection and HCP preprocessing are available elsewhere^{36,100,101}. Notably, cortical and subcortical data underwent ICA-FIX^{102,103} and were saved in the CIFTI gray ordinate format. The surface (fs_LR) data were aligned with MSM-All¹⁰⁴. As ICA-FIX does not fully eliminate global motion-related and respiratory-related artifacts^{105,106}, additional censoring and nuisance regression were performed^{107,108}. In particular, volumes with framewise displacement (FD) > 0.2mm, and root-mean-square of voxel-wise differentiated signal (DVARs) > 75 were marked as outliers and censored, along with one frame before and two frames after the outlier volume^{109,110}. Any uncensored segment of data that lasted fewer than five contiguous volumes was also excluded from analysis, as well as runs with >50% censored frames. Additionally, the global signal obtained by averaging signal across all cortical vertices and its temporal derivatives (ignoring censored frames) were also regressed out from the data because previous studies have suggested that global signal regression strengthens the association between RSFC and behavioral traits¹⁰⁷. As there is ongoing debate on the use of global signal regression (GSR) as a means of fMRI preprocessing^{107,111–113}, additional reliability analysis was performed on data preprocessed using a component-based noise correction method (CompCor)⁴⁷ instead of GSR.

RSFC was computed among 400 cortical parcels⁴⁶ and 19 subcortical regions⁴⁵ using Pearson's correlation (excluding the censored volumes). The subcortical regions were in subject-specific volumetric space as defined by FreeSurfer⁴⁵, and comprised the left and right cerebellum, thalamus, caudate, putamen, pallidum, hippocampus, accumbens, amygdala, ventral diencephalon, and

brainstem. For each participant, RSFC was computed for each run, Fisher z-transformed, and then averaged across runs and sessions, yielding a final 419 x 419 RSFC matrix for each participant.

RSFC analyses

To investigate the neurobiological substrates of the sleep-biopsychosocial profiles derived in the CCA, we computed generalized linear models (GLM) between participant's canonical scores (i.e., averaged sleep and biopsychosocial scores) and their RSFC data. Age, sex, and level of education were first regressed out from the RSFC data.

To obtain an analysis at the large-scale network level and limit the number of multiple comparisons, we computed a network-wise GLM, whereby the whole-brain RSFC data was averaged within and between the 17 large-scale brain networks⁴⁶ and subcortical regions⁴⁵, resulting in 18 x 18 RSFC matrices. Next, we applied a GLM for each network edge (i.e., average connectivity between two brain networks), with participants' component-specific canonical scores as the predictor and RSFC edge as the response. Each GLM yielded a beta coefficient and associated *T* statistic, as well as an *F* statistic and associated *p* value obtained from a hypothesis test that all coefficient estimates were equal to zero. Statistical significance for each RSFC network edge was determined by applying FDR correction ($q < 0.05$) on all *p* values (along with other posthoc analyses). For a more granular view, we also computed a GLM for each RSFC edge (i.e., connectivity between two brain regions) using whole-brain RSFC between all 419 brain regions. For a complete view of the component-specific RSFC signatures, we plotted both the uncorrected region-wise GLM beta coefficients (e.g., **Figure 2C**) and FDR-corrected network-wise GLM beta coefficients (e.g., **Figure 2D**).

Measures of integration and segregation were computed on the GLM beta coefficient connectivity matrix associated with each LC using functions from the Brain Connectivity Toolbox¹¹⁴. Firstly, the input-weighted connection matrix was normalized. Next, each 419 cortical parcel was assigned to one of the 7 large-scale brain networks and subcortical regions⁴⁴. Within-network connectivity was estimated by calculating the module-degree Z score (within-module strength) for each region. The extent to which a parcel connects across all networks was quantified using the participation coefficient, (between-module strength). For each cortical parcel, the ratio of normalized within:between module strength values was calculated and interpreted as a measure for the balance of integration and segregation of functional brain connectivity¹¹⁵. Nodes with high within- but low between-module strength are likely to facilitate network segregation, while nodes with higher between-module strength (i.e., connector hubs) are likely to facilitate global integration¹¹⁴.

Control analyses

We ran several control analyses to evaluate the robustness of our findings. First, we applied 5-fold cross-validation (accounting for family structure) to assess the generalizability of our sleep-biopsychosocial profiles by training a CCA model on 80% of the data and testing it on the remaining 20% of the data. For each fold, we projected the sleep and biopsychosocial canonical coefficients of the training data on the sleep and biopsychosocial data of the test data, to obtain sleep and biopsychosocial scores, and computed Pearson's correlations between these scores. Second, we evaluated the impact of the covariates on our profiles as well as the impact of other potential confounds, including race, ethnicity, and familial psychiatric history. Third, we re-computed the CCA analysis after excluding participants who had tested positive for any substance use on the day of the MRI. Fourth, we re-computed the CCA analysis after excluding physical health (i.e., body mass index, hematocrit, blood pressure) and sociodemographic (i.e., employment status, household income, in-school, relationship status) variables from the biopsychosocial matrix. Fifth, to mitigate scale magnitude discrepancies between different measures, we re-computed the CCA analysis after applying quantile normalization on sleep and biopsychosocial measures. We also assessed the robustness of our imaging results in several ways. As GSR is a controversial preprocessing step^{107,112,113}, we re-computed the GLM analysis using RSFC data

that underwent CompCor⁴⁷ instead of GSR. Some subjects were noticed to have likely fallen asleep during scanning (list not publicly available¹¹⁶). As a first step, we re-computed the GLM after excluding these subjects (N=100); next, we sought to determine whether these participants scored high on any of the profiles, by comparing their sleep/biopsychosocial composite scores with awake participants using t-tests. We re-computed the GLM analyses by using sleep and biopsychosocial canonical scores instead of averaged scores. Finally, integration and segregation measures were also computed on the average RSFC matrix of the whole sample. FDR correction ($q < 0.05$) was applied to all posthoc tests.

Data and code availability

Data from the HCP dataset is publicly available (<https://www.humanconnectome.org/>). The brain parcellation can be obtained here (https://github.com/ThomasYeoLab/CBIG/tree/master/stable_projects/brain_parcellation/Schaefer2018_LocalGlobal), while the code for the CCA analysis and figures can be found here (https://github.com/valkebets/sleep_biopsychosocial_profiles). Chord diagrams were generated using previously published code (https://github.com/ThomasYeoLab/CBIG/tree/master/stable_projects/predict_phenotypes/ChenTam2022_TRBPC/figure_utilities/chord).

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AUTHOR CONTRIBUTION STATEMENT

Conceptualization: NMYK, BTTY, VK, AAP; Data curation: VK, NMYK, JL, AAP; Formal analysis: VK, NMYK, NEC, AAP; Methodology: VK, NMYK, NEC; Visualization: AAP, VK, NEC; Interpretation: AAP, VK, NEC, RT; Writing - original draft: AAP, VK, NMYK; editing and reviewing: AAP, VK, RT, NEC, BTTY, FBP, TTDV, JL, MWLC, NMYK.

REFERENCES

1. Becker SP, Langberg JM, Byars KC. Advancing a Biopsychosocial and Contextual Model of Sleep in Adolescence: A Review and Introduction to the Special Issue. *J Youth Adolesc.* 2015;44(2):239-270. doi:10.1007/s10964-014-0248-y
2. Bolton D, Gillett G. *The Biopsychosocial Model of Health and Disease: New Philosophical and Scientific Developments*. Springer International Publishing; 2019. doi:10.1007/978-3-030-11899-0
3. Havelka M, Luanin JD, Luanin D. Biopsychosocial Model – The Integrated Approach to Health and Disease. *Coll Antropol.* Published online 2009.
4. Verma K, Singh D, Srivastava A. Sleep disorders and its consequences on biopsychosocial health: A narrative review. *Yoga Mimamsa.* 2022;54(2):101. doi:10.4103/ym.ym_82_22
5. Roth T, Jaeger S, Jin R, Kalsekar A, Stang PE, Kessler RC. Sleep Problems, Comorbid Mental Disorders, and Role Functioning in the National Comorbidity Survey Replication (NCS-R). *Biol Psychiatry.* 2006;60(12):1364-1371. doi:10.1016/j.biopsych.2006.05.039
6. Chaput JP, Dutil C, Featherstone R, et al. Sleep duration and health in adults: an overview of systematic reviews. *Appl Physiol Nutr Metab.* 2020;45(10 (Suppl. 2)):S218-S231. doi:10.1139/apnm-2020-0034
7. Gao C, Guo J, Gong TT, et al. Sleep Duration/Quality With Health Outcomes: An Umbrella Review of Meta-Analyses of Prospective Studies. *Front Med.* 2022;8. Accessed April 7, 2023. <https://www.frontiersin.org/articles/10.3389/fmed.2021.813943>
8. Freeman D, Sheaves B, Waite F, Harvey AG, Harrison PJ. Sleep disturbance and psychiatric disorders. *Lancet Psychiatry.* 2020;7(7):628-637. doi:10.1016/S2215-0366(20)30136-X
9. Buysse DJ. Sleep Health: Can We Define It? Does It Matter? *Sleep.* 2014;37(1):9-17. doi:10.5665/sleep.3298
10. Papadopoulos D, Sosso FE, Khoury T, Surani SR. Sleep Disturbances Are Mediators Between Socioeconomic Status and Health: a Scoping Review. *Int J Ment Health Addict.* 2022;20(1):480-504. doi:10.1007/s11469-020-00378-x
11. Hudson AN, Van Dongen HPA, Honn KA. Sleep deprivation, vigilant attention, and brain function: a review. *Neuropsychopharmacology.* 2020;45(1):21-30. doi:10.1038/s41386-019-0432-6
12. Killgore WDS. Effects of sleep deprivation on cognition. In: Kerkhof GA, Dongen HPA van, eds. *Progress in Brain Research*. Vol 185. Elsevier; 2010:105-129. doi:10.1016/B978-0-444-53702-7.00007-5
13. Bai Y, Tan J, Liu X, Cui X, Li D, Yin H. Resting-state functional connectivity of the sensory/somatomotor network associated with sleep quality: evidence from 202 young male samples. *Brain Imaging Behav.* 2022;16(4):1832-1841. doi:10.1007/s11682-022-00654-5
14. Curtis BJ, Williams PG, Jones CR, Anderson JS. Sleep duration and resting fMRI functional connectivity: examination of short sleepers with and without perceived daytime dysfunction. *Brain Behav.* 2016;6(12):e00576. doi:10.1002/brb3.576

15. Wang Y, Genon S, Dong D, et al. Covariance patterns between sleep health domains and distributed intrinsic functional connectivity. *Nat Commun.* 2023;14(1):7133. doi:10.1038/s41467-023-42945-5
16. Liew SC, Aung T. Sleep deprivation and its association with diseases- a review. *Sleep Med.* 2021;77:192-204. doi:10.1016/j.sleep.2020.07.048
17. Ong AD, Kim S, Young S, Steptoe A. Positive affect and sleep: A systematic review. *Sleep Med Rev.* 2017;35:21-32. doi:10.1016/j.smr.2016.07.006
18. Song Q, Wang M, Zhou T, et al. The Lifestyle-Related Cardiovascular Risk Is Modified by Sleep Patterns. *Mayo Clin Proc.* 2022;97(3):519-530. doi:10.1016/j.mayocp.2021.08.031
19. Fan M, Sun D, Zhou T, et al. Sleep patterns, genetic susceptibility, and incident cardiovascular disease: a prospective study of 385 292 UK biobank participants. *Eur Heart J.* 2020;41(11):1182-1189. doi:10.1093/eurheartj/ehz849
20. Shi L, Chen SJ, Ma MY, et al. Sleep disturbances increase the risk of dementia: A systematic review and meta-analysis. *Sleep Med Rev.* 2018;40:4-16. doi:10.1016/j.smr.2017.06.010
21. Wang C, Holtzman DM. Bidirectional relationship between sleep and Alzheimer's disease: role of amyloid, tau, and other factors. *Neuropsychopharmacology.* 2019;(February). doi:10.1038/s41386-019-0478-5
22. Gregory AM, Buysse DJ, Willis TA, et al. Associations between sleep quality and anxiety and depression symptoms in a sample of young adult twins and siblings. *J Psychosom Res.* 2011;71(4):250-255. doi:10.1016/j.jpsychores.2011.03.011
23. van de Langenberg SCN, Kocavska D, Luik AI. The multidimensionality of sleep in population-based samples: a narrative review. *J Sleep Res.* 2022;31(4):e13608. doi:10.1111/jsr.13608
24. Semler CN, Harvey AG. Misperception of sleep can adversely affect daytime functioning in insomnia. *Behav Res Ther.* 2005;43(7):843-856. doi:10.1016/j.brat.2004.06.016
25. Castelnovo A, Ferri R, Punjabi NM, et al. The paradox of paradoxical insomnia: A theoretical review towards a unifying evidence-based definition. *Sleep Med Rev.* 2019;44:70-82. doi:10.1016/j.smr.2018.12.007
26. Perrault AA, Pomares FB, Smith D, et al. Effects of cognitive behavioral therapy for insomnia on subjective and objective measures of sleep and cognition. *Sleep Med.* 2022;97:13-26. doi:10.1016/j.sleep.2022.05.010
27. Wade DT, Halligan PW. The biopsychosocial model of illness: a model whose time has come. Published online 2017. Accessed September 4, 2023. https://journals.sagepub.com/doi/10.1177/0269215517709890?url_ver=Z39.88-2003&rft_id=ori:rid:crossref.org&rft_dat=cr_pub%20%20pubmed
28. Smith CE, Lee S. Identifying diverse forms of (un)healthy sleep: Sleep profiles differentiate adults' psychological and physical well-being. *Soc Sci Med.* 2022;292:114603. doi:10.1016/j.socscimed.2021.114603

29. Blanken TF, Benjamins JS, Borsboom D, et al. Insomnia disorder subtypes derived from life history and traits of affect and personality. *Lancet Psychiatry*. 2019;6(2):151-163. doi:10.1016/S2215-0366(18)30464-4
30. Appleton SL, Melaku YA, Reynolds AC, Gill TK, de Batlle J, Adams RJ. Multidimensional sleep health is associated with mental well-being in Australian adults. *J Sleep Res*. 2022;31(2):e13477. doi:10.1111/jsr.13477
31. Katori M, Shi S, Ode KL, Tomita Y, Ueda HR. The 103,200-arm acceleration dataset in the UK Biobank revealed a landscape of human sleep phenotypes. *Proc Natl Acad Sci*. 2022;119(12):e2116729119. doi:10.1073/pnas.2116729119
32. Tahmasian M, Samea F, Khazaie H, et al. The interrelation of sleep and mental and physical health is anchored in grey-matter neuroanatomy and under genetic control. *Commun Biol*. 2020;3(1):1-13. doi:10.1038/s42003-020-0892-6
33. McIntosh AR. Comparison of Canonical Correlation and Partial Least Squares analyses of simulated and empirical data. *arXiv*. 2019;3(2):58-66.
34. Marek S, Tervo-Clemmens B, Calabro FJ, et al. Reproducible brain-wide association studies require thousands of individuals. *Nature*. 2022;603(7902):654-660. doi:10.1038/s41586-022-04492-9
35. Helmer M, Warrington S, Mohammadi-Nejad AR, et al. On stability of Canonical Correlation Analysis and Partial Least Squares with application to brain-behavior associations. Published online April 27, 2023:2020.08.25.265546. doi:10.1101/2020.08.25.265546
36. Van Essen DC, Smith SM, Barch DM, Behrens TEJ, Yacoub E, Ugurbil K. The WU-Minn Human Connectome Project: An overview. *NeuroImage*. 2013;80:62-79. doi:10.1016/j.neuroimage.2013.05.041
37. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res*. 1989;28(2):193-213. doi:10.1016/0165-1781(89)90047-4
38. Grumbach P, Opel N, Martin S, et al. Sleep duration is associated with white matter microstructure and cognitive performance in healthy adults. *Hum Brain Mapp*. 2020;41(15):4397-4405. doi:10.1002/hbm.25132
39. Deco G, Kringelbach ML. Great Expectations: Using Whole-Brain Computational Connectomics for Understanding Neuropsychiatric Disorders. *Neuron*. 2014;84(5):892-905. doi:10.1016/j.neuron.2014.08.034
40. Cheng CPW, Cheng ST, Tam CWC, Chan WC, Chu WCW, Lam LCW. Relationship between cortical thickness and neuropsychological performance in normal older adults and those with mild cognitive impairment. *Aging Dis*. 2018;9(6):1020-1030. doi:10.14336/AD.2018.0125
41. Cuthbert BN. The RDoC framework: Facilitating transition from ICD/DSM to dimensional approaches that integrate neuroscience and psychopathology. *Ann Med Psychol (Paris)*. 2021;179(1):75-85. doi:10.1016/j.amp.2020.11.013
42. Insel T, Cuthbert B, Garvey M, et al. Research Domain Criteria (RDoC): Toward a New Classification Framework for Research on Mental Disorders. *Am J Psychiatry Online*. 2010;(July):748-751.

43. Plomin R, Haworth CMA, Davis OSP. Common disorders are quantitative traits. *Nat Rev Genet.* 2009;10(12):872-878. doi:10.1038/nrg2670
44. Yeo BT, Krienen FM, Sepulcre J, et al. The organization of the human cerebral cortex estimated by intrinsic functional connectivity. *J Neurophysiol.* 2011;106(3):1125-1165. doi:10.1152/jn.00338.2011
45. Fischl B. FreeSurfer. *NeuroImage.* 2012;62(2):774-781. doi:10.1016/j.neuroimage.2012.01.021
46. Schaefer A, Kong R, Gordon EM, et al. Local-Global Parcellation of the Human Cerebral Cortex from Intrinsic Functional Connectivity MRI. *Cereb Cortex.* 2018;28(9):3095-3114. doi:10.1093/cercor/bhx179
47. Behzadi Y, Restom K, Liao J, Liu TT. A component based noise correction method (CompCor) for BOLD and perfusion based fMRI. *NeuroImage.* 2007;37(1):90-101. doi:10.1016/j.neuroimage.2007.04.042
48. Garg H. Sleep History Taking and Examination. *Int J Head Neck Surg.* 2019;10(1):9-17. doi:10.5005/jp-journals-10001-1363
49. Kozak MJ, Cuthbert BN. The NIMH Research Domain Criteria Initiative: Background, Issues, and Pragmatics: NIMH Research Domain Criteria initiative. *Psychophysiology.* 2016;53(3):286-297. doi:10.1111/psyp.12518
50. Baglioni C, Nanovska S, Regen W, et al. Sleep and mental disorders: a meta-analysis of polysomnographic research. *Psychol Bull.* 2016;142(9):969-990. doi:10.1037/bul0000053
51. Caspi A, Houts RM, Belsky DW, et al. The p Factor: One General Psychopathology Factor in the Structure of Psychiatric Disorders? *Clin Psychol Sci.* 2014;2(2):119-137. doi:10.1177/2167702613497473
52. Del Rio João KA, Jesus SN de, Carmo C, Pinto P. The impact of sleep quality on the mental health of a non-clinical population. *Sleep Med.* 2018;46:69-73. doi:10.1016/j.sleep.2018.02.010
53. Palagini L, Hertenstein E, Riemann D, Nissen C. Sleep, insomnia and mental health. *J Sleep Res.* 2022;31(4):e13628. doi:10.1111/jsr.13628
54. He Y, Jones CR, Fujiki N, et al. The Transcriptional Repressor DEC2 Regulates Sleep Length in Mammals. *Science.* 2009;325(5942):866-870. doi:10.1126/science.1174443
55. Li Y, Sahakian BJ, Kang J, et al. The brain structure and genetic mechanisms underlying the nonlinear association between sleep duration, cognition and mental health. *Nat Aging.* 2022;2(5):425-437. doi:10.1038/s43587-022-00210-2
56. Lunansky G, van Borkulo CD, Haslbeck JMB, et al. The Mental Health Ecosystem: Extending Symptom Networks With Risk and Protective Factors. *Front Psychiatry.* 2021;12. Accessed July 1, 2023. <https://www.frontiersin.org/articles/10.3389/fpsyt.2021.640658>
57. Jones BE. Arousal and sleep circuits. *Neuropsychopharmacology.* 2020;45(1):6-20. doi:10.1038/s41386-019-0444-2
58. Ting L, Malhotra A. Disorders of Sleep: An Overview. *Prim Care.* 2005;32(2):305-v. doi:10.1016/j.pop.2005.02.004

59. Wainberg M, Jones SE, Beaupre LM, et al. Association of accelerometer-derived sleep measures with lifetime psychiatric diagnoses: A cross-sectional study of 89,205 participants from the UK Biobank. *PLOS Med.* 2021;18(10):e1003782. doi:10.1371/journal.pmed.1003782
60. Banks S, Dinges DF. Behavioral and physiological consequences of sleep restriction. *J Clin Sleep Med.* 2007;3(5):519-528.
61. Durmer JS, Dinges DF. Neurocognitive Consequences of Sleep Deprivation. *Semin Neurol.* 2005;25(1):117-129.
62. Ren D, Jiang B, Guo Z. Insomnia disorder with objective short sleep duration (ISS) phenotype and cognitive performance: a systematic review and meta-analysis. *Neurol Sci.* 2023;44(7):2363-2368. doi:10.1007/s10072-023-06692-1
63. Lim J, Choo WC, Chee MWL. Reproducibility of Changes in Behaviour and fMRI Activation Associated with Sleep Deprivation in a Working Memory Task. *Sleep.* 2007;30(1):61-70. doi:10.1093/sleep/30.1.61
64. Cross NE, Pomares FB, Nguyen A, et al. An altered balance of integrated and segregated brain activity is a marker of cognitive deficits following sleep deprivation. *PLoS Biol.* 2021;19(11):2020.11.28.402305. doi:https://doi.org/10.1371/journal.pbio.3001232
65. Dorrian J, Centofanti S, Smith A, McDermott KD. Chapter 4 - Self-regulation and social behavior during sleep deprivation. In: Van Dongen HPA, Whitney P, Hinson JM, Honn KA, Chee MWL, eds. *Progress in Brain Research.* Vol 246. Sleep Deprivation and Cognition. Elsevier; 2019:73-110. doi:10.1016/bs.pbr.2019.03.010
66. Cross N, Paquola C, Pomares FB, et al. Cortical gradients of functional connectivity are robust to state-dependent changes following sleep deprivation. *NeuroImage.* 2021;226(April 2020):117547. doi:10.1016/j.neuroimage.2020.117547
67. Yeo BTT, Tandi J, Chee MWL. Functional connectivity during rested wakefulness predicts vulnerability to sleep deprivation. *NeuroImage.* 2015;111:147-158. doi:10.1016/j.neuroimage.2015.02.018
68. Neubauer DN, Pandi-Perumal SR, Spence DW, Buttoo K, Monti JM. Pharmacotherapy of Insomnia. *J Cent Nerv Syst Dis.* 2018;10:1179573518770672. doi:10.1177/1179573518770672
69. Vermeeren A, Coenen AML. Effects of the use of hypnotics on cognition. In: Van Dongen HPA, Kerkhof GA, eds. *Progress in Brain Research.* Vol 190. Human Sleep and Cognition Part II. Elsevier; 2011:89-103. doi:10.1016/B978-0-444-53817-8.00005-0
70. Crowe SF, Stranks EK. The Residual Medium and Long-term Cognitive Effects of Benzodiazepine Use: An Updated Meta-analysis. *Arch Clin Neuropsychol.* 2018;33(7):901-911. doi:10.1093/arclin/acx120
71. Lee JS, Lee JD, Park HJ, et al. Is the GABA System Related to the Social Competence Improvement Effect of Aripiprazole? An 18F-Fluoroflumazenil PET Study. *Psychiatry Investig.* 2013;10(1):75-80. doi:10.4306/pi.2013.10.1.75
72. Mielnik CA, Horsfall W, Ramsey AJ. Diazepam improves aspects of social behaviour and neuron activation in NMDA receptor-deficient mice. *Genes Brain Behav.* 2014;13(7):592-602. doi:10.1111/gbb.12155

73. Pillai V, Roth T, Roehrs T, Moss K, Peterson EL, Drake CL. Effectiveness of Benzodiazepine Receptor Agonists in the Treatment of Insomnia: An Examination of Response and Remission Rates. *Sleep*. 2017;40(2):zsw044. doi:10.1093/sleep/zsw044
74. Hajak G, Clarenbach P, Fischer W, et al. Effects of hypnotics on sleep quality and daytime well-being. Data from a comparative multicentre study in outpatients with insomnia. *Eur Psychiatry*. 1995;10(S3):173s-179s. doi:10.1016/0924-9338(96)80100-3
75. Watson NF, Bertisch SM, Morin CM, et al. Do Insomnia Treatments Improve Daytime Function? *J Clin Med*. 2023;12(9):3089. doi:10.3390/jcm12093089
76. Valentino RJ, Volkow ND. Drugs, sleep, and the addicted brain. *Neuropsychopharmacology*. 2020;45(1):3-5. doi:10.1038/s41386-019-0465-x
77. Kebets V, Holmes AJ, Orban C, et al. Somatosensory-Motor Dysconnectivity Spans Multiple Transdiagnostic Dimensions of Psychopathology. *Biol Psychiatry*. 2019;86(10):779-791. doi:10.1016/j.biopsych.2019.06.013
78. Van Dam NT, O'Connor D, Marcelle ET, et al. Data-Driven Phenotypic Categorization for Neurobiological Analyses: Beyond DSM-5 Labels. *Biol Psychiatry*. 2017;81(6):484-494. doi:10.1016/j.biopsych.2016.06.027
79. Zhang L, Li D, Yin H. How is psychological stress linked to sleep quality? The mediating role of functional connectivity between the sensory/somatomotor network and the cingulo-opercular control network. *Brain Cogn*. 2020;146:105641. doi:10.1016/j.bandc.2020.105641
80. Bin YS. Is Sleep Quality More Important than Sleep Duration for Public Health? *Sleep*. 2016;39(9):1629-1630. doi:10.5665/sleep.6078
81. Zavecz Z, Nagy T, Galkó A, Nemeth D, Janacsek K. The relationship between subjective sleep quality and cognitive performance in healthy young adults: Evidence from three empirical studies. *Sci Rep*. 2020;10(1):4855. doi:10.1038/s41598-020-61627-6
82. Nakua H, Yu JC, Abdi H, et al. Comparing the stability and reproducibility of brain-behavior relationships found using canonical correlation analysis and partial least squares within the ABCD sample. *Netw Neurosci*. 2024;8(2):576-596. doi:10.1162/netn_a_00363
83. Faulkner S, Sidey-Gibbons C. Use of the Pittsburgh Sleep Quality Index in People With Schizophrenia Spectrum Disorders: A Mixed Methods Study. *Front Psychiatry*. 2019;10. Accessed April 7, 2023. <https://www.frontiersin.org/articles/10.3389/fpsyt.2019.00284>
84. Krittanawong C, Kumar A, Wang Z, et al. Sleep Duration and Cardiovascular Health in a Representative Community Population (from NHANES, 2005 to 2016). *Am J Cardiol*. 2020;127:149-155. doi:10.1016/j.amjcard.2020.04.012
85. Clark AJ, Salo P, Lange T, et al. Onset of Impaired Sleep and Cardiovascular Disease Risk Factors: A Longitudinal Study. *Sleep*. 2016;39(9):1709-1718. doi:10.5665/sleep.6098
86. Baker M, Stabile M, Deri C. What Do Self-Reported, Objective, Measures of Health Measure? *J Hum Resour*. 2004;39(4):1067-1093. doi:10.2307/3559039

87. Trimmel K, Eder HG, Böck M, Stefanic-Kejik Andrijana, Klösch G, Seidel S. The (mis)perception of sleep: factors influencing the discrepancy between self-reported and objective sleep parameters. *J Clin Sleep Med*. 2021;17(5):917-924. doi:10.5664/jcsm.9086
88. Lee SA, Im K, Yang H rin. Factors associated with sleep state misperception in patients with obstructive sleep apnea. *Sleep Breath*. 2022;26(4):1921-1930. doi:10.1007/s11325-021-02543-5
89. Lecci S, Cataldi J, Betta M, Bernardi G, Heinzer R, Siclari F. Electroencephalographic changes associated with subjective under- and overestimation of sleep duration. *SLEEP*. 2020;43(11):zsaa094. doi:https://doi.org/10.1093/sleep/zsaa094
90. Andrillon T, Solelhac G, Bouchequet P, et al. Revisiting the value of polysomnographic data in insomnia: more than meets the eye. *Sleep Med*. 2020;66:184-200. doi:10.1016/j.sleep.2019.12.002
91. Lim DC, Najafi A, Afifi L, et al. The need to promote sleep health in public health agendas across the globe. *Lancet Public Health*. 2023;8(10):e820-e826. doi:10.1016/S2468-2667(23)00182-2
92. Barch DM, Burgess GC, Harms MP, et al. Function in the human connectome: Task-fMRI and individual differences in behavior. *NeuroImage*. 2013;80:169-189. doi:10.1016/j.neuroimage.2013.05.033
93. Kettenring JR. Canonical analysis of several sets of variables. *Biometrika*. 1971;58(3):433-451. doi:10.1093/biomet/58.3.433
94. Hotelling H. Relations Between Two Sets of Variates. In: Kotz S, Johnson NL, eds. *Breakthroughs in Statistics: Methodology and Distribution*. Springer Series in Statistics. Springer; 1992:162-190. doi:10.1007/978-1-4612-4380-9_14
95. Courville T, Thompson B. Use of Structure Coefficients in Published Multiple Regression Articles: β is not Enough. Published online 2001. Accessed September 4, 2023. <https://journals.sagepub.com/doi/10.1177/0013164401612006>
96. Henson RK. The logic and interpretation of structure coefficients in multivariate general linear model analyses. In: ; 2002.
97. Sherry A, Henson RK. Conducting and Interpreting Canonical Correlation Analysis in Personality Research: A User-Friendly Primer. *J Pers Assess*. 2005;84(1):37-48. doi:10.1207/s15327752jpa8401_09
98. Winkler AM, Webster MA, Brooks JC, Tracey I, Smith SM, Nichols TE. Non-parametric combination and related permutation tests for neuroimaging. *Hum Brain Mapp*. 2016;37(4):1486-1511. doi:10.1002/hbm.23115
99. Winkler AM, Ridgway GR, Webster MA, Smith SM, Nichols TE. Permutation inference for the general linear model. *NeuroImage*. 2014;92:381-397. doi:10.1016/j.neuroimage.2014.01.060
100. Glasser MF, Sotiropoulos SN, Wilson JA, et al. The minimal preprocessing pipelines for the Human Connectome Project. *NeuroImage*. 2013;80:105-124. doi:10.1016/j.neuroimage.2013.04.127
101. Smith SM, Beckmann CF, Andersson J, et al. Resting-state fMRI in the Human Connectome Project. *NeuroImage*. 2013;80:144-168. doi:10.1016/j.neuroimage.2013.05.039

102. Griffanti L, Salimi-Khorshidi G, Beckmann CF, et al. ICA-based artefact removal and accelerated fMRI acquisition for improved resting state network imaging. *NeuroImage*. 2014;95:232-247. doi:10.1016/j.neuroimage.2014.03.034
103. Salimi-Khorshidi G, Douaud G, Beckmann CF, Glasser MF, Griffanti L, Smith SM. Automatic denoising of functional MRI data: combining independent component analysis and hierarchical fusion of classifiers. *NeuroImage*. 2014;90:449-468. doi:10.1016/j.neuroimage.2013.11.046
104. Robinson EC, Jbabdi S, Glasser MF, et al. MSM: A new flexible framework for Multimodal Surface Matching. *NeuroImage*. 2014;100:414-426. doi:10.1016/j.neuroimage.2014.05.069
105. Burgess GC, Kandala S, Nolan D, et al. Evaluation of Denoising Strategies to Address Motion-Correlated Artifacts in Resting-State Functional Magnetic Resonance Imaging Data from the Human Connectome Project. *Brain Connect*. 2016;6(9):669-680. doi:10.1089/brain.2016.0435
106. Siegel JS, Mitra A, Laumann TO, et al. Data Quality Influences Observed Links Between Functional Connectivity and Behavior. *Cereb Cortex*. 2017;27(9):4492-4502. doi:10.1093/cercor/bhw253
107. Li J, Kong R, Liégeois R, et al. Global signal regression strengthens association between resting-state functional connectivity and behavior. *NeuroImage*. 2019;196:126-141. doi:10.1016/j.neuroimage.2019.04.016
108. Kong R, Li J, Orban C, et al. Spatial Topography of Individual-Specific Cortical Networks Predicts Human Cognition, Personality, and Emotion. *Cereb Cortex N Y NY*. 2019;29(6):2533-2551. doi:10.1093/cercor/bhy123
109. Jenkinson M, Bannister P, Brady M, Smith S. Improved Optimization for the Robust and Accurate Linear Registration and Motion Correction of Brain Images. *NeuroImage*. 2002;17(2):825-841. doi:10.1006/nimg.2002.1132
110. Power JD, Barnes KA, Snyder AZ, Schlaggar BL, Petersen SE. Spurious but systematic correlations in functional connectivity MRI networks arise from subject motion. *NeuroImage*. 2012;59(3):2142-2154. doi:10.1016/j.neuroimage.2011.10.018
111. Li X, Huang X. Linear mode regression with covariate measurement error. *Can J Stat*. 2019;47(2):262-280. doi:10.1002/cjs.11492
112. Liu TT, Nalci A, Falahpour M. The global signal in fMRI: Nuisance or Information? *NeuroImage*. 2017;150:213-229. doi:10.1016/j.neuroimage.2017.02.036
113. Murphy K, Fox MD. Towards a consensus regarding global signal regression for resting state functional connectivity MRI. *NeuroImage*. 2017;154:169-173. doi:10.1016/j.neuroimage.2016.11.052
114. Rubinov M, Sporns O. Complex network measures of brain connectivity: Uses and interpretations. *NeuroImage*. 2010;52(3):1059-1069. doi:10.1016/j.neuroimage.2009.10.003
115. Shine JM, Bissett PG, Bell PT, et al. The Dynamics of Functional Brain Networks: Integrated Network States during Cognitive Task Performance. *Neuron*. 2016;92(2):544-554. doi:10.1016/j.neuron.2016.09.018

116. Glasser MF, Coalson TS, Bijsterbosch JD, et al. Using temporal ICA to selectively remove global noise while preserving global signal in functional MRI data. *NeuroImage*. 2018;181:692-717. doi:10.1016/j.neuroimage.2018.04.076

Characteristics		N = 770
Biological sex (n %)	<i>Female</i>	414 53.76%
	<i>Male</i>	356 46.23%
Age (years)	<i>mean ± SD</i>	28.86 ± 3.61
	<i>range</i>	[22 - 36]
Education (years)	<i>mean ± SD</i>	15.02 ± 1.73
	<i>range</i>	[11 - 17]
Race (n %)	<i>Am. Indian/Alaskan Nat.</i>	2 0.25%
	<i>Asian/Nat. Hawaiian/Other Pacific Is.</i>	42 5.45%
	<i>Black or African Am.</i>	90 11.68%
	<i>More than one</i>	19 2.46%
	<i>Unknown or Not Reported</i>	16 2.07%
	<i>White</i>	601 78.05%
Ethnicity (n %)	<i>Hispanic/Latino</i>	78 10.12%
	<i>Not Hispanic/Latino</i>	684 88.83%
	<i>Unknown or Not Reported</i>	8 1.03%
Employment status (n %)	<i>Full-time</i>	545 70.77%
	<i>Part-time</i>	132 17.14%
	<i>Not working</i>	93 12.07%
School status (n %)	<i>In school</i>	158 20.51%
	<i>Not in school</i>	612 79.48%
Yearly income (n %)	<i><10'000 US\$</i>	50 6.49%
	<i>10'000 - 20'000 US\$</i>	50 6.49%
	<i>20'000 - 30'000 US\$</i>	94 12.20%
	<i>30'000 - 40'000 US\$</i>	101 13.11%
	<i>40'000 - 50'000 US\$</i>	76 9.87%
	<i>50'000 - 75'000 US\$</i>	165 21.42%
	<i>75'000 - 100'000 US\$</i>	112 14.54%
	<i>> 100'00 US\$</i>	122 15.84%
Relationship status (n %)	<i>In a relationship</i>	363 47.14%
	<i>Not in a relationship</i>	407 52.85%
PSQI total score	<i>mean ± SD</i>	5.14 ± 2.17
	<i>range</i>	[0 - 19]

PSQI, Pittsburgh sleep quality index

Table 1