

STATIC AND DYNAMIC BRAIN CONNECTIVITY PATTERNS ACROSS SLEEP DURATION

Sabadnoor Singh^{1,2,6}, Elaheh Zendehrouh^{3,4}, Matthew Gunn^{1,2}, Vince D. Calhoun^{3,5}, Mohammad S. E. Sendi^{1,2,3}

¹McLean Hospital, Belmont, MA, USA

²Department of Psychiatry, Harvard Medical School, Boston, MA, USA

³TReNDS Center, GSU / Georgia Tech / Emory, Atlanta, GA, USA

⁴School of Electrical and Computer Engineering, Georgia Tech, Atlanta, GA, USA

⁵Georgia State University, Atlanta, GA, USA

⁶Department of Psychology, University of Massachusetts Amherst, Amherst, MA, USA

ABSTRACT

Sleep duration is associated with brain health, but it remains unclear whether short and long sleep are related to similar or distinct patterns of intrinsic brain connectivity. We analyzed resting-state fMRI data from 14,021 UK Biobank participants and examined static functional network connectivity (sFNC) and dynamic functional network connectivity (dFNC) across three sleep-duration groups: short (≤ 6 h), normal (7–8 h), and long (≥ 9 h). After adjustment for demographic, imaging, lifestyle, and health-related covariates, 269 of 1,378 sFNC connections were significantly associated with sleep duration after Bonferroni correction. These associations were distributed across multiple large-scale networks, with prominent involvement of subcortical, sensorimotor, visual, cognitive-control, default-mode, and cerebellar systems. Pairwise group comparisons showed the largest number of significant sFNC differences between long and short sleepers. Dynamic analyses identified three recurring connectivity states. Longer sleep duration was associated with greater occupancy of States 1 and 3 and lower occupancy of State 2, with significant differences across sleep-duration groups. Together, these findings suggest that sleep duration is related to both distributed differences in average functional connectivity and systematic shifts in the temporal expression of recurring brain-connectivity states.

Index Terms— functional connectivity, sleep duration, dynamic FNC, UK Biobank, resting-state fMRI

1. INTRODUCTION

Sleep duration is closely linked to brain health, cognition, and psychiatric risk. Both short and long sleep have been associated with adverse outcomes, including cardiovascular disease, cognitive decline, depression, and mortality [1, 2]. Short sleep is often treated as the primary concern. However, long sleep may also reflect meaningful differences in brain and body function rather than simply representing “too much” of a healthy behavior. This raises an important question: do short, normal, and long sleepers show distinct patterns of brain network organization?

Resting-state fMRI provides a useful way to study this question because it measures how distributed brain systems communicate while a person is not performing an explicit task. Most prior imaging studies of sleep duration have focused on static functional network connectivity (sFNC). sFNC summarizes average connectivity between brain regions across the full scan and has revealed associations between sleep duration and thalamic, sensorimotor, default-mode, and cognitive-control networks [9, 3, 4]. These findings are important, but they provide a time-averaged view of the brain and do not fully capture how connectivity patterns fluctuate during the scan.

Dynamic functional network connectivity (dFNC) addresses this limitation by measuring how brain connectivity changes over time. Rather than treating connectivity as fixed, dFNC identifies recurring brain states and quantifies how long individuals remain in each state and how often they transition between states. This temporal information may be especially relevant for sleep research. Sleep duration may not only relate to the strength of individual connections; it may also be associated with how the brain maintains and shifts between functional states. Yet relatively few studies have examined whether dFNC patterns differ across short, normal, and long sleepers.

A second limitation of previous work is that sleep duration is often modeled as a single continuous variable. Although this approach is useful for detecting broad associations, it can obscure group-specific effects. Short and long sleep may not be mirror images of the same process. Long sleep, in particular, may reflect fatigue, depression, medical burden, altered arousal regulation, or other biological factors that differ from those associated with short sleep. Therefore, directly comparing short, normal, and long sleepers may reveal whether brain connectivity changes symmetrically across the sleep distribu-

tion or whether one end of the distribution shows stronger neural differences.

In this study, we examined both sFNC and dFNC across sleep-duration groups in 14,021 UK Biobank participants. The contribution is empirical: a large-scale, covariate-adjusted characterization of static and dynamic functional connectivity across short, normal, and long sleepers, with particular attention to whether long sleep shows patterns distinct from short sleep — a joint sFNC/dFNC characterization at population scale not previously reported.

2. METHODS

2.1. Participants

We used resting-state fMRI data from the UK Biobank, a large-scale population study based in the United Kingdom [5]. The UK Biobank enrolled adults aged 40–69 years and collected detailed health, lifestyle, and imaging data. Following prior UK Biobank connectivity work, we excluded individuals with stress-related, neuropsychiatric, or neurological conditions to focus on a healthy adult imaging cohort [6]. We also removed participants with missing imaging or sleep-duration data, resulting in a final analytic sample of 14,021 individuals. Participants were healthy adults with a mean age of 64.23 ± 7.55 years. The sample included 6,362 females and 7,659 males.

Self-reported sleep duration ranged from 3 to 15 hours per night and was recorded in whole-hour increments. Participants were divided into three groups: short sleep (≤ 6 h), normal sleep (7–8 h), and long sleep (≥ 9 h). The final sample included 3,132 short sleepers, 9,997 normal sleepers, and 892 long sleepers. The sleep-duration distribution and group definitions are shown in Fig. 1A.

2.2. sFNC Feature Extraction

Brain parcellation was performed using the NeuroMark automated independent component analysis (ICA) framework [7]. This framework identifies reproducible and spatially distinct brain networks without requiring manual labeling. It produced 53 independent components organized into seven established functional domains: subcortical (SCN), auditory (AUD), sensorimotor (SMN), visual (VSN), cognitive control (CCN), default mode (DMN), and cerebellar (CBN) networks. We computed Pearson correlations between the time series of every pair of components, resulting in 1,378 unique sFNC values per participant. These values represent the upper triangle of a 53×53 connectivity matrix.

2.3. dFNC State and Feature Extraction

To capture changes in brain connectivity over time, we used a sliding-window approach. A Hamming-tapered window of 20 time points (14.7 s; $TR = 0.735$ s) was moved across the fMRI

time series in steps of one TR, and a connectivity matrix was computed at each step. This produced a series of connectivity snapshots for each participant. We then applied k -means clustering to group all windows into recurring brain states. The number of clusters was selected by sweeping $k = 2$ to 9, evaluating the elbow criterion on within-cluster sum of squared distances across 100 random initializations to ensure stability; $k = 3$ was identified as the optimal solution. For each participant, we calculated the proportion of time spent in each state, defined as the occupancy rate (OCR1, OCR2, and OCR3), as well as the number of transitions between states.

2.4. Statistical Models

We used two complementary modeling strategies for both static and dynamic connectivity measures. First, sleep duration was modeled as a continuous variable. Second, sleep group was modeled as a categorical variable. All models included the same covariate vector, $\mathbf{C}$, comprising age, age², sex, site, smoking status, physical activity, alcohol frequency, and BMI.

For the continuous sleep-duration analysis of sFNC, we ran a separate generalized linear model (GLM) for each of the 1,378 sFNC connections:

$$\text{sFNC}_i \sim \text{sleepDuration} + \mathbf{C}. \quad (1)$$

For the group-based sFNC analysis, we ran a separate GLM for each sFNC connection using sleep group as the main predictor:

$$\text{sFNC}_i \sim \text{sleepGroup} + \mathbf{C}. \quad (2)$$

For the continuous sleep-duration analysis of dFNC, we tested whether each dynamic state occupancy rate was associated with sleep duration:

$$\text{OCR}_i \sim \text{sleepDuration} + \mathbf{C}. \quad (3)$$

For the group-based dFNC analysis, we tested whether each dynamic state occupancy rate differed across sleep groups:

$$\text{OCR}_i \sim \text{sleepGroup} + \mathbf{C}. \quad (4)$$

For the sFNC and dFNC group analyses, we performed pairwise comparisons among short, normal, and long sleepers. Multiple comparisons for sFNC were controlled using Bonferroni correction across the 1,378 connections. Connections that did not survive correction were set to zero in the output matrices. For the dFNC continuous analyses, false-discovery-rate (FDR) correction was applied across the OCR measures.

Together, these models allowed us to test both continuous sleep-duration associations and categorical group differences in static connectivity and dynamic brain-state occupancy.

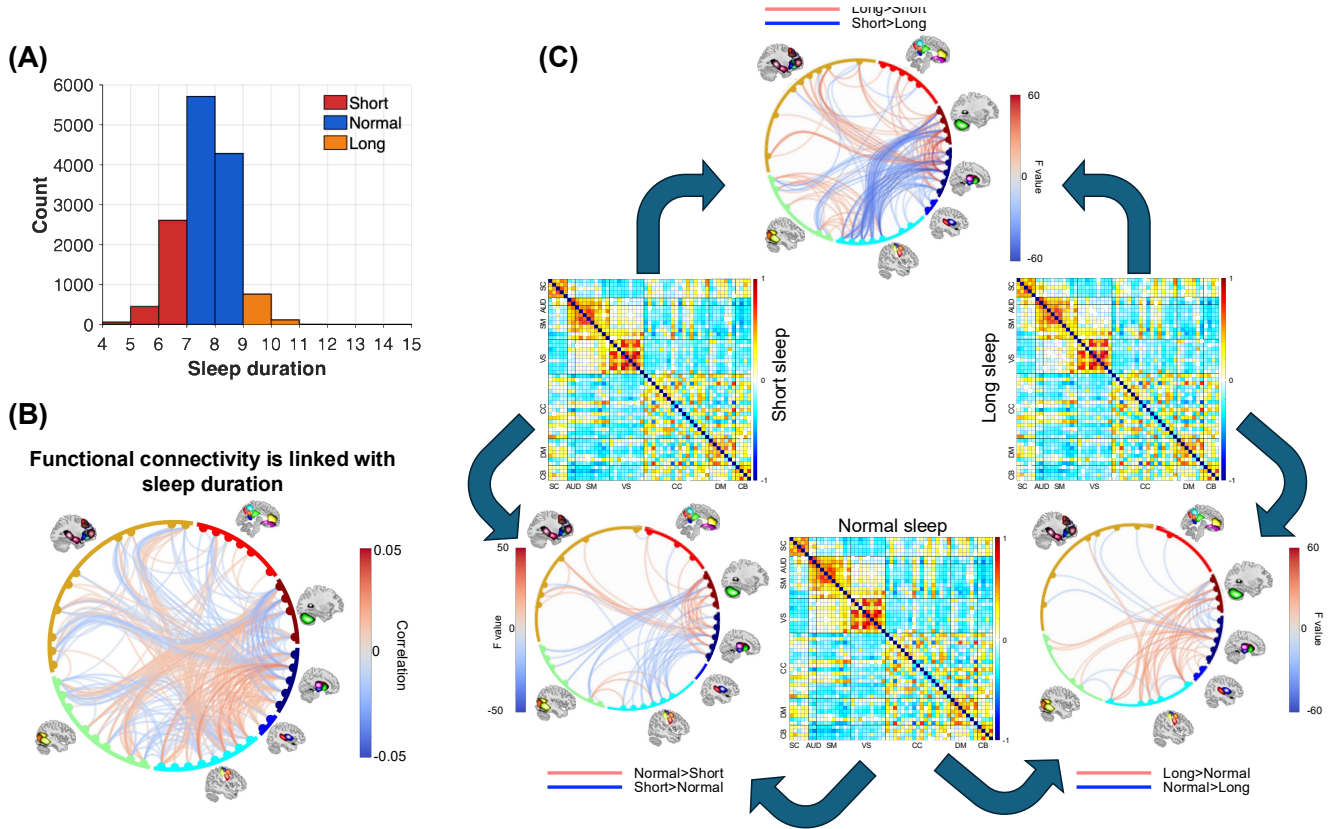


Fig. 1. Sleep-duration distribution and static functional network connectivity (sFNC) results. **(A)** Distribution of self-reported sleep duration across the final analytic sample, with participants grouped into short sleep (≤ 6 h), normal sleep (7–8 h), and long sleep (≥ 9 h). **(B)** sFNC connections associated with continuous sleep duration after adjusting for age, age², sex, site, smoking status, physical activity, alcohol frequency, and BMI. Connections surviving Bonferroni correction ($p < 0.05/1,378$) are shown as red and blue lines, indicating positive and negative associations, respectively. **(C)** Group-based sFNC analysis comparing short, normal, and long sleepers while adjusting for the same covariates. Connections surviving Bonferroni correction are shown in the connectivity matrices.

3. RESULTS

3.1. sFNC: sleep duration is associated with widespread connectivity differences

Fig. 1B shows the association between sFNC and continuous sleep duration across the full sample. After covariate adjustment and Bonferroni correction ($p < 0.05/1,378$), 269 of the 1,378 sFNC connections showed a significant association with sleep duration. Effect sizes were small (partial $|r| = 0.035$ – 0.151 ; mean = 0.064), consistent with population-level correlates of a single lifestyle variable.

Significant sFNC effects were broadly distributed across the NeuroMark connectome, with dense involvement of SCN, SMN, VSN, CCN, DMN, and CBN components. The most prominent pattern involved CBN-related connections, especially CBN–SCN, CBN–SMN, CBN–VSN, CBN–CCN, and CBN–DMN. This suggests that sleep duration is associated with distributed changes in subcortical, sensorimotor,

visual, cerebellar, and higher-order control/default-mode circuitry rather than isolated connectivity effects. Prior studies have similarly shown that individual differences in sleep duration and sleep loss are associated with distributed functional-connectivity patterns across large-scale brain networks [9, 10]. The involvement of SCN–SMN and SCN–CBN pathways may reflect differences in arousal regulation and sensorimotor integration, given the established role of thalamic and subcortical circuitry in sleep–wake regulation and sensory gating [11]. The CBN–SMN effects may also be consistent with the cerebellum’s role in sensorimotor coordination, whereas CBN–CCN and CBN–DMN effects may reflect broader integration of cerebellar regions with association networks involved in cognitive and internally directed processing [12].

Fig. 1C shows the group-based sFNC analysis comparing short, normal, and long sleepers. The model included the same covariates and was corrected across all 1,378 connec-

tions using Bonferroni correction. Significant group effects were observed across multiple network pairs, indicating that sFNC varies across sleep-duration groups after accounting for demographic, imaging, lifestyle, and health-related covariates.

Post hoc analyses showed the largest number of significant pairwise differences for the long-versus-short contrast, followed by the normal-versus-short and normal-versus-long contrasts. The long-versus-short comparison showed 149 significant edges, with the strongest involvement of SCN–SMN, CBN–SMN, CBN–SCN, and SMN–VSN connections. The normal-versus-long comparison showed 57 significant edges and followed a similar but more restricted pattern, mainly involving CBN–SMN, SCN–SMN, and CBN–SCN connections. The normal-versus-short comparison showed 65 significant edges, with prominent involvement of SCN–SMN, CBN–SCN, CBN–VSN, CCN–SCN, and CBN–SMN connections.

Across all pairwise contrasts, the most consistent group differences involved interactions among SCN, SMN, and CBN components. This pattern is consistent with prior evidence that sleep duration is associated with distributed resting-state connectivity differences involving sensorimotor, subcortical, and cerebellar regions [13, 9]. The additional involvement of VSN, CCN, and DMN components suggests that sleep-duration differences may extend beyond sensorimotor circuitry to broader sensory and higher-order association networks. This interpretation is also consistent with evidence that distinct cerebellar regions are functionally coupled with somatomotor and association networks, including regions implicated in higher-order cognition [12]. Together, the continuous and categorical analyses indicate that sleep duration is associated with distributed sFNC differences across large-scale brain systems rather than focal changes in a single network.

3.2. dFNC: sleep duration is associated with distinct brain-state occupancy patterns

Fig. 2A shows the three recurring dynamic connectivity states identified from the resting-state fMRI data. The states exhibited distinct patterns of network organization. State 1 was characterized by relatively strong positive connectivity within the default-mode network (DMN), together with positive within-network connectivity in the sensorimotor network (SMN) and visual network (VSN). State 2 showed strong positive connectivity within the SMN and VSN, as well as prominent positive connectivity between these two networks. State 3 was characterized primarily by positive within-network connectivity in the SMN and VSN, with less pronounced cross-network coupling than State 2.

Fig. 2B shows the associations between sleep duration and occupancy rate (OCR) for each dynamic state after adjustment for covariates. Sleep duration was positively associated with State 1 occupancy ($r = 0.055$, $p_{\text{FDR}} = 2.91 \times 10^{-9}$)

and State 3 occupancy ($r = 0.058$, $p_{\text{FDR}} = 5.32 \times 10^{-10}$), whereas it was negatively associated with State 2 occupancy ($r = -0.086$, $p_{\text{FDR}} = 2.32 \times 10^{-20}$). These associations are small in magnitude (less than 0.74% of variance explained) and reflect systematic population-level effects rather than diagnostic or predictive ones.

Fig. 2C compares adjusted OCR values across short, normal, and long sleepers. State 1 occupancy increased from 0.284 in short sleepers to 0.294 in normal sleepers and 0.324 in long sleepers. The difference between short and normal sleepers was not significant, whereas long sleepers showed significantly higher State 1 occupancy than both short and normal sleepers ($p_{\text{FDR}} < 0.05$). State 2 occupancy decreased progressively from 0.306 in short sleepers to 0.278 in normal sleepers and 0.224 in long sleepers. The difference between short and normal sleepers was not significant, whereas long sleepers showed significantly lower State 2 occupancy than both short and normal sleepers ($p_{\text{FDR}} < 0.05$). State 3 occupancy increased from 0.410 in short sleepers to 0.428 in normal sleepers and 0.450 in long sleepers, with significant differences across all three pairwise comparisons ($p_{\text{FDR}} < 0.05$).

Together, these findings indicate that sleep duration is associated with systematic differences in the amount of time the brain spends in distinct dynamic connectivity states. Longer sleep duration was associated with greater occupancy of States 1 and 3 and lower occupancy of State 2.

4. DISCUSSION

This study examined static and dynamic functional connectivity across short, normal, and long sleep-duration groups in 14,021 UK Biobank participants. Sleep duration was associated with widespread sFNC differences across large-scale brain systems, with the strongest group differences involving subcortical, sensorimotor, and cerebellar connections, and with systematic dFNC shifts toward greater occupancy of States 1 and 3 and lower occupancy of State 2 in longer sleepers. Together, these results suggest sleep duration is related not only to average connectivity strength but also to the temporal distribution of recurring brain-connectivity configurations.

The continuous sFNC analysis identified 269 significant connections after stringent Bonferroni correction, indicating that sleep duration is associated with distributed rather than focal differences in functional connectivity. Effect sizes were small (partial $|r| = 0.035$ – 0.151), consistent with population-level correlates of a self-reported lifestyle variable, yet meaningful in aggregate: the affected connections spanned subcortical, sensorimotor, visual, cognitive-control, default-mode, and cerebellar systems. This distributed pattern is consistent with prior work showing that sleep duration and sleep loss are associated with connectivity differences across multiple large-scale networks rather than a single isolated cir-

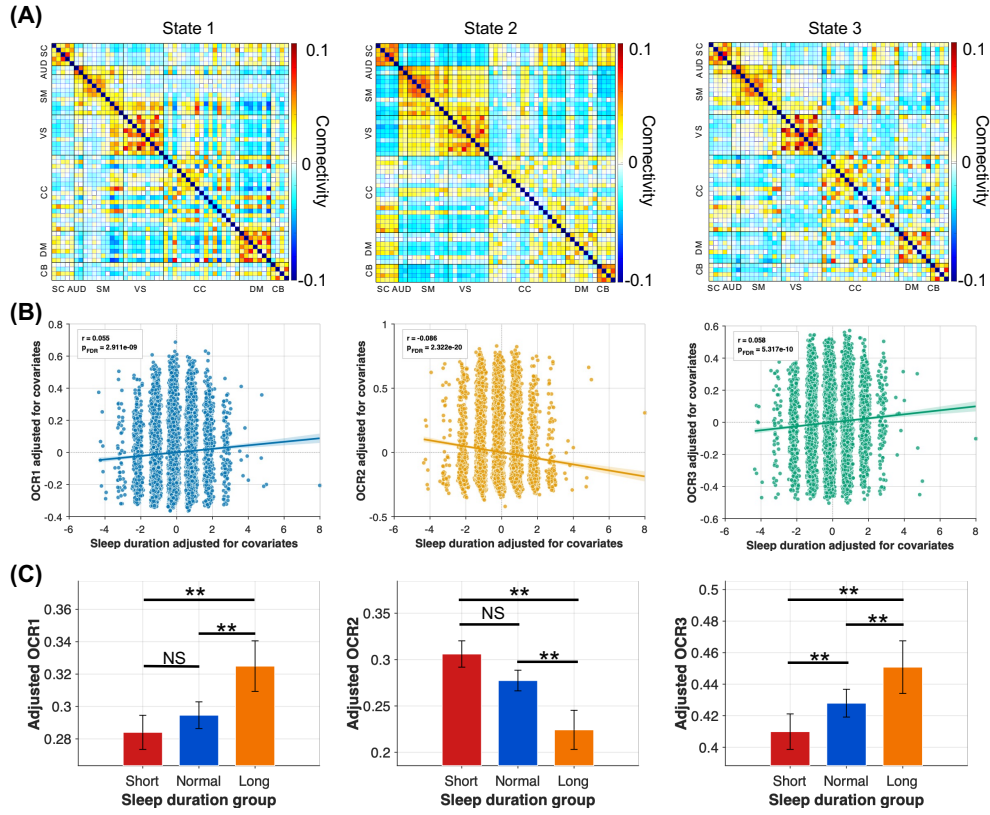


Fig. 2. Dynamic functional network connectivity (dFNC) results. **(A)** Three recurring dynamic connectivity states identified from the resting-state fMRI data. State 1 was characterized by relatively strong positive connectivity within the default-mode network (DMN), together with positive within-network connectivity in the sensorimotor network (SMN) and visual network (VSN). State 2 showed strong positive connectivity within the SMN and VSN, as well as prominent positive connectivity between these networks. State 3 was characterized primarily by positive within-network connectivity in the SMN and VSN, with less pronounced cross-network coupling than State 2. **(B)** Associations between sleep duration and occupancy rate (OCR) for each dynamic state after adjustment for covariates. Sleep duration was positively associated with OCR1 and OCR3 and negatively associated with OCR2. **(C)** Adjusted OCR values across short, normal, and long sleep-duration groups. OCR1 and OCR3 increased across sleep-duration groups, whereas OCR2 decreased. Error bars represent the standard error of the mean. Pairwise significance is indicated as follows: NS, not significant; *, $p_{FDR} < 0.05$.

cuit [9, 10, 3].

Across the categorical comparisons, the largest number of significant edges was observed between long and short sleepers. The most consistent differences involved SCN–SMN, CBN–SMN, and CBN–SCN connections. These findings are broadly consistent with previous studies implicating thalamic, sensorimotor, and cerebellar pathways in sleep-related variation [4, 13]. Subcortical structures, including the thalamus, contribute to sleep–wake regulation and sensory gating [8, 11]. Cerebellar regions are also functionally connected with sensorimotor and higher-order association networks [12]. Therefore, the observed SCN–SMN and CBN–SMN differences may reflect variation in arousal-related and

sensorimotor processes associated with sleep duration.

However, these interpretations should remain cautious. The present findings do not establish causality; sleep duration may be associated with other factors influencing connectivity, including sleep quality, medical burden, or mood symptoms. Group size imbalance may also affect the number of detectable connections independent of effect size. The dFNC analysis extends the static findings by showing that longer sleep is associated with greater occupancy of States 1 and 3 and lower occupancy of State 2, with long sleepers showing a distinctly different pattern from short sleepers.

The three states differed in network organization: State 1 showed strong within-network DMN, SMN, and VSN con-

nectivity; State 2 was distinguished by prominent SMN–VSN coupling; State 3 showed similar within-network connectivity but weaker cross-network coupling. These patterns suggest sleep duration is associated with shifts in the expression of sensory, sensorimotor, and default-mode configurations. The dynamic states should not be interpreted as inherently beneficial or detrimental; their clinical significance remains unclear. Together, the sFNC and dFNC findings indicate that long sleep is not simply the inverse of short sleep, and longitudinal studies with objective sleep measures are needed to determine the direction and clinical relevance of these associations.

Several limitations should be noted. Sleep duration was self-reported in whole-hour increments and is subject to recall error; it did not capture sleep quality, fragmentation, or night-to-night variability. The cross-sectional design prevents conclusions about directionality. Group sizes were unequal, with substantially fewer long sleepers ($n = 892$) than normal sleepers ($n = 9,997$), and the large sample increases sensitivity to small effects whose practical relevance requires further evaluation. Residual confounding from sleep disorders, medication use, and mood symptoms remains possible. Clinical translation requires prospective validation with objective sleep measures and is an important direction for future work.

5. CONCLUSION

This study examined static and dynamic functional connectivity across short, normal, and long sleep-duration groups in 14,021 UK Biobank participants. Sleep duration was associated with widespread sFNC differences across multiple large-scale brain systems, with the largest number of pairwise differences observed between long and short sleepers. Effect sizes were small, consistent with the population-level scope of the analysis. Dynamic analyses further showed that longer sleep duration was associated with greater occupancy of States 1 and 3 and lower occupancy of State 2. Together, these findings suggest that sleep duration is related to both distributed differences in average functional connectivity and systematic shifts in the temporal expression of recurring brain-connectivity states. Longitudinal studies using objective sleep measures will be needed to determine the direction and clinical relevance of these associations.

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