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Association Between Sleep Quality and Dorsal Default Mode Network in College Students

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College students often have poor sleep quality, which may change resting state functional connectivity network in the brain. Prior research demonstrated that sleep is associated with structural and functional changes in some specific brain subareas, which are involved in the dorsal default mode network (dDMN). However, no study to date has comprehensively examined all possible individual pairs of functional connectivity in the dDMN in relation to sleep quality among college students. Therefore, the present study hypothesized that sleep quality in college students was linked to any resting state functional connectivity in the dDMN.

Forty nine college students (25 females) underwent a magnetic resonance imaging (MRI) scan. The resting state functional MRI data were acquired. In addition, participants were asked about their sleep quality using the Quick Inventory for Depressive Symptomatology (QIDS). Time-series functional connectivity values between nine subareas of the dDMN were preprocessed and extracted by AFNI. The relationships between these functional connectivity values and sleep quality were then analyzed using Spearman's correlations.

The results of this study demonstrated that sleep quality was negatively correlated with functional connectivity between the right hippocampus and the thalamus (rho(47) =

-0.40, $p < .05$) and between the right hippocampus and the left angular gyrus (rho(47) = -0.39, $p < .05$). The present study found that college students with poor sleep quality had negative functional hippocampal connectivity with the thalamus and the left angular gyrus. Cognitive and emotional implications for these findings were discussed.

Association Between Sleep Quality and Dorsal Default Mode Network in College Students

According to National Sleep Foundation, adults are recommended to sleep for 7-9 hours per night; less than 7-hour sleep duration is usually considered as insufficient sleep (Hirshkowitz et al., 2015). However, the National Health Interview Survey in 2017 revealed that one of three adults in the U.S. slept for only 6 hours or less, and this ratio has increased by 15% since 2004 (Sheehan et al., 2018). Poor sleep quality is even more problematic among college students. For example, Lund et al. (2009) have reported that over 60% of undergraduate college students have poor sleep quality.

College students often do not get enough sleep because of their heavy study duties (American College Health Association, 2015), living condition (e.g., shared apartments, noisy dormitory) (Owens et al., 2017), or others. In addition, it is common for college students to have caffeine in daily life or alcohol for a social purpose, but the excessive amount of caffeine and alcohol leads to poor sleep quality (Owens et al., 2017). Another factor for their sleep problems is that they are often exposed to the blue light emitted from electronic devices, which has a detrimental effect on sleep. (Grandner et al., 2013).

Given that sleep problem is common among college students, sleep research has investigated how sleep problem influences their cognitive behavior (Durmer & Dinges, 2005). For example, if students fail to maintain good sleep quality, they tend to show poor performance in long-term and working memory (Durmer & Dinges, 2005), executive functions (Durmer & Dinges, 2005; Killgore, 2010), problem-solving (Killgore, 2010), and attention (Durmer & Dinges, 2005; Killgore, 2010) which are all

critical for their academic success. Moreover, sleep quality also affects emotional regulation skills (Killgore, 2010; Tempesta et al., 2018). For instance, sleep problems may be associated with an increased level of emotional reactivity, anxiety, depression, and aggression (Tempesta et al., 2018).

These cognitive and emotional effects of sleep quality may result from structural or functional brain changes. However, a number of questions regarding neural correlates of sleep quality specific to healthy college students remain to be addressed. Rather, the majority of studies has focused on patients with insomnia or healthy individuals of a wide range of ages. For example, patients with insomnia tend to show altered diffusion-tensor based network characteristics between the right angular gyrus and the frontal, temporal, and subcortical areas (including the hippocampus, thalamus, and precuneus) (Wei et al., 2019*)*. Furthermore, patients with insomnia show reduced volume of the left orbitofrontal cortex (OFC) and precuneus (Altena et al., 2010). Also, a recent meta-analytical study showed that patients with insomnia had reduced grey matter volume in the middle frontal gyrus (Wu et al., 2020). Other studies examined the association between daytime sleepiness and cortical volume in healthy adults and found that increased daytime sleepiness was correlated with reduced volumes of the medial OFC (Killgore et al., 2012) and the posterior cingulate cortex (PCC) (Facer-Childs et al., 2019). On the contrary, oversleep is associated with increased volumes of the medial prefrontal cortex (mPFC) and OFC in the left hemisphere (Weber et al., 2013). Interestingly, the OFC and mPFC are indeed involved in emotion processing (Etkin et al., 2011; Rempel-Clower, 2007; Rolls et al., 2020), suggesting the possible link between sleep and emotional behavior.

It has been addressed that sleep quality influences not only brain structures but also brain functions. Positron emission tomography (PET) showed that metabolic activity decreased in the superior frontal gyrus (SFG), thalamus, amygdala, anterior cingulate cortex (ACC), and PCC among individuals with sleep deprivation or insomnia (Wu et al., 2006; Wu et al., 2020). Moreover, recent meta-analytical findings revealed that patients with insomnia had decreased activation in the bilateral superior temporal gyrus, the left middle temporal gyrus, the right inferior frontal gyrus, and the right cuneus during a working memory task (Wu et al., 2020). In addition, poor sleep quality results in changes in resting state functional connectivity. For example, a prior research suggests that poor sleepers had weaker functional connectivity between the hippocampus and the SFG (Chengyang et al., 2016). Furthermore, functional connectivity between the hippocampus and thalamus is also altered in sleep problems although the magnitude of this functional connectivity probably varies across the severity of sleep problems (Chengyang et al., 2016; Zou et al., 2020;). The study by Li et al. (2018) demonstrated that patients with insomnia had weaker functional hippocampal connectivity with the ACC, OFC, caudate, and putamen. On the other hand, a longer sleep period increases positive functional connectivity between the mPFC and PCC (Killgore et al., 2012). Taken together, the functional network among the hippocampus, SFG, thalamus, ACC, OFC, caudate, putamen, mPFC, and PCC seems to have implications for sleep.

Based on structural and functional magnetic resonance imaging (MRI) studies discussed above, sleep may broadly involve functions of the fronto-parietal areas of the cortex and subcortical areas, especially the OFC, precuneus, PCC, mPFC, SFG, angular gyrus, thalamus, and hippocampus. Noteworthy, these brain subregions are known as the

constitutes of the dorsal default mode network (dDMN), which shows high functional synchronization among them at resting state (Shirer et al., 2012). Thus, it is conceivable that sleep problems might affect the dDMN. In fact, as mentioned earlier, functional connectivity between some of areas within the dDMN, such as the SFG, angular gyrus, thalamus, and hippocampus, shows alterations in relation to sleep quality (Chengyang et al., 2016; Killgore et al., 2012; Zou et al., 2020). However, the dDMN consists of more than these brain subregions. Specifically, there are nine clusters of subareas that are functionally connected with each other as the dDMN: (1) a cluster including the anterior cingulate cortex (ACC) and mPFC extending to the OFC, (2) a cluster involving the right SFG, (3) a cluster involving the middle cingulate cortex (MCC), (4) a cluster involving the PCC and precuneus, (5) a cluster involving the left angular gyrus, (6) a cluster involving the right angular gyrus, (7) a cluster involving the thalamus, (8) a cluster involving the left hippocampus, and (9) a cluster involving the right hippocampus (Shirer et al., 2012). However, no study to date has comprehensively examined whether sleep quality is associated with functional connectivity between some or all of them.

In addition, previous research has focused on changes in resting state functional connectivity among those who show clinically severe sleep problems (e.g., insomnia). However, it is unclear whether these changes are also seen in college students with sleep problems. Given that sleep problems are remarkably common among college students, my interest lies in resting state functional connectivity changes among college students who suffer from sleep problems. To my knowledge, however, no such study exists. The previous neuroimaging findings on sleep deprivation or insomnia might have implications for sleep problems in college students as well, but college students do not

necessarily have such severe symptoms. Thus, the present study focused on college students and aimed to examine possible changes in functional connectivity in the dDMN as their sleep quality changes. Specifically, it was hypothesized that college students with sleep problems would show any altered functional connectivity within the dDMN, as compared to those without sleep problems.

Methods

Participants

This study was a part of another neuroimaging study aimed to investigate the neurobiological correlates of bullying and peer victimization. As a result of the recruitment process (advertising the project through classrooms, flyers, and electronic media at the University of Nebraska-Lincoln), 352 university students took part in an online screening voluntarily or for course credit.

Fifty-one participants were chosen and registered for further MRI study based on the screening. One student displayed a brain abnormality, and another participant showed severe head motion during the scan; these two participants were excluded from subsequent analyses. No major neurological illness, cognitive or developmental delay, or serious vision/hearing loss were found in the rest of participants. Consequently, the total sample size in the present study was $N = 49$. Prior to online screening and MRI scan, written informed consents approved by the University of Nebraska-Lincoln Institutional Review Board were collected from participants.

Measures

An online screening included several questions related to MRI eligibility (e.g., no magnetic object in their body). Furthermore, a demographic and psychological assessment battery was completed by participants. The present research used four individual items assessing sleep quality as a part of the Quick Inventory of Depressive Symptomatology (QIDS). These question items asked participants to choose the best described characteristics of their sleep onset (e.g., taking 30 min to fall asleep), sleep at midnight (e.g., waking up in the middle of night), sleep in early morning (e.g., awakening 30 min before the need), and oversleep (e.g., sleeping more than 7-8 hours per night) on a 4-point scale, where a high score indicated more serious problem. The maximum score among these items was selected as the sleep quality score.

Procedure and imaging data acquisition

Participants underwent MRI scanning; 3 Tesla Siemens Skyra MRI scanner (Siemens Medical Solutions) with a 32-ch head coil MRI receiver was used to acquire neuroimaging data. The MRI study included (1) a localizer scan for prescribing the following scans, (2) a 6-min resting-state functional scan, (3) two 8.5-min functional scans during a psychological task, (4) a 5-min anatomical T1-weighted (T1w) scan, (5) the other two 4.5-min functional scans during a face task, (6) two diffusion-weighted scans, (7) a 1.5-min scan of T2-weighted turbo spin-echo (TSE), and (8) a 1.5-min gradient echo field-mapping scan in that order. In the current study, resting state functional images were used which were sensitive to blood oxygen level-dependent (BOLD) acquired with a 2D multiband (MB) gradient echo recalled (GRE) echo-planar imaging (EPI) series (TR = 1000 ms, TE = 29.80 ms, multi-band acceleration factor = 3,

flip angle = 60, FOV = 210 mm, voxel size = 2.5 mm^3); 346 sets of 51 contiguous axial images with isotropic voxels (2.5 mm^3) were acquired parallel to the anterior-posterior commissure plane. In addition, the anatomical T1w images were used for registering the resting state functional images; the T1w images were acquired in the sagittal plane by a three-dimensional magnetization prepared rapid gradient echo (MPRAGE) scan (TR = 2200 ms, TE = 3.37 ms, flip angle = 7°, FOV = 256 mm, sagittal slices per slab = 192, voxel volume = 1.0 × 1.0 × 1.0 mm^3, acceleration factor PE = 2, sampling band-width = 200 Hz/Px). For MRI scans, the overall time was under one hour. Following the MRI scan, participants completed the psychosocial assessments including the QIDS through Qualtrics.

Image preprocessing

The anatomical images were automatically segmented into gray matter, white matter, and ventricles using the FreeSurfer image analysis suite (http://surfer.nmr.mgh.harvard.edu/). In addition, the resting state functional data were preprocessed using AFNI (https://afni.nimh.nih.gov/afni), including: despiking, aligning slices to the beginning of the TR (slice-timing correction), registering the structural volume (T1w) to the Montreal Neurological Institute (MNI) standard template using a nonlinear transformation, co-registering the structural volume to the functional data, warping the functional data to the standard template, and smoothing the warped functional data spatially within the whole brain volume (identified by FreeSurfer). The first three TRs were censored, and then a general linear model (GLM) was used to eliminate variables of no interest from the time series, such as the mean signals in the

white matter and the ventricles (identified by FreeSurfer), global mean signal in the whole brain, and de-meaned and derivatives of motion parameters. Participants with excessive head movement (greater than 3.0 mm displacement) were excluded from further analysis. A band pass filter was also applied to the time series to eliminate frequencies greater than 0.1 Hz and less than 0.01 Hz.

After the time series BOLD signals per voxel were estimated by the GLM, they were extracted within the dDMN, which was my region of interest (ROIs) in the current study. Shirer et al. (2012) identified nine clusters that functionally connected with each other at rest in the dDMN. They include the mPFC and ACC extending to the OFC (mPFC-ACC), right SFG (rSFG), PCC extending to the precuneus (PCC), middle cingulate cortex (MCC), left angular gyrus (lAG), right angular gyrus (rAG), thalamus, left hippocampus (lHpc), and right hippocampus (rHpc). Figure 1 shows the locations of these suareas.

The mask images of these functional clusters were obtained online (http://findlab.stanford.edu/functional_ROIs.html), and they were resampled to my standard template and applied to my functional data. Then, the mean BOLD signals across voxels comprising each of the nine functional clusters, as defined by the mask, were extracted through 346 timepoints by AFNI's 3dROIstats command.

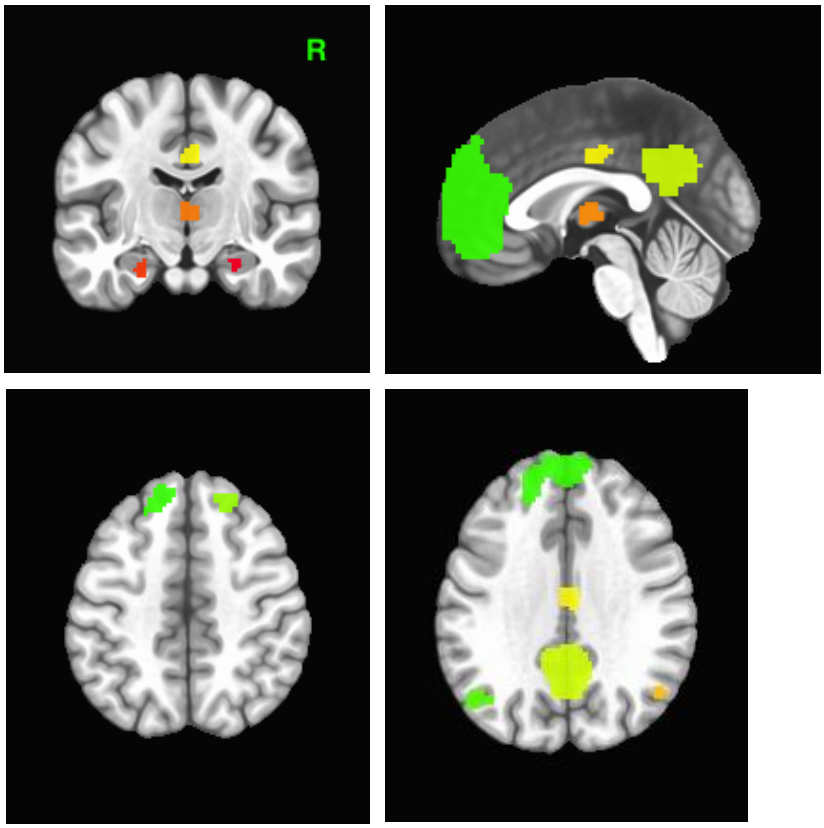

Figure 1. Dorsal DMN network. A big green area on the upper right sagittal view represents a cluster including the ACC and mPFC extending to the OFC; a light green area on the lower left axial view represents the right SFG; a yellow green area on the upper right sagittal view represents a cluster involving the PCC and precuneus; a yellow area represents a cluster involving the MCC; a tiny green area on the lower right axial view represents a cluster involving the left angular gyrus; a light orange on the lower right axial view represents a cluster involving the right angular gyrus; an orange area represents a cluster involving the thalamus; dark orange and red areas on the upper left coronal view represent the hippocampus.

Statistical strategy

To describe characteristics of participants, the mean age (with standard deviation), gender ratio, and racial/ethnic ratio were computed. Spearman's correlations were run within each participant in order to obtain a matrix of correlations between the nine

dDMN functional clusters. This matrix produced 36 correlation coefficients, representing all possible individual pairs of functional connectivity between the clusters. These correlation coefficients were then analyzed in relation to sleep quality using Spearman's correlations.

Results

The mean age of participants was 22.7 (*SD* = 4.74). Twenty five were females, and 24 were males in the study. Four participants identified themselves as Hispanic or Latino. The racial ratio was the following: 35 Caucasian, five African Americans, four Asians, three mixed, and two others.

As Table 1 illustrates, sleep quality was negatively correlated with functional connectivity between the right hippocampus and the thalamus ($\rho(47) = -0.40$, $p < .05$) and between the right hippocampus and the left angular gyrus ($\rho(47) = -0.34$, $p < .05$). Functional connectivity between the right hippocampus and the thalamus ranged from -0.38 to 0.48, and functional connectivity between the right hippocampus and the left angular gyrus ranged from -0.30 to 0.48. Thus, poor sleep quality was correlated with negative functional connectivity between the right hippocampus and the thalamus, as well as between the right hippocampus and the left angular gyrus. In comparison, functional connectivity for these pairs turned to be positive when participants reported good sleep quality. No other correlations were significant.

Table 1. Associations between Sleep Quality and dDMN Subareas

Subarea of dDMN	1. mPFC-ACC	2. rSFG	3. PCC	4. MCC	5. lAG	6. rAG	7. Thalamus	8. lHpc	9. rHpc
2	-0.06								
3	0.03	-0.05							
4	-0.05	0.05	-0.04						
5	-0.06	-0.17	0.00	0.01					
6	-0.00	-0.01	0.03	0.15	-0.16				
7	0.06	0.04	-0.04	-0.06	-0.09	-0.03			
8	-0.02	0.02	-0.09	0.15	-0.07	0.16	-0.04		
9	-0.00	-0.04	-0.09	-0.10	-0.34*	-0.01	-0.40*	0.03	

Note: Each value represents a correlation between sleep quality and functional connecitivty of two specific subareas. mPFC-ACC = subarea consisting of the medial prefrontal cortex and the anterior cingulate cortex; PCC = subarea consisting of the posterior cingulate cortex and the precuneus; MCC = subarea in th middle cingulate cortex; lAG = subarea in the left angular gyrus; rAG = subarea in the right angular gyrus; thalamus = subarea in the thalamus; lHpc = subarea in the left hippocampus; rHpc = subarea in the right hippocampus.
*$p < .05$

Discussion

The present study hypothesized that sleep quality would be associated with changes in resting state functional connectivity in the dDMN among college students. Results revealed that poor sleep quality was associated with negative functional connectivity between the thalamus and the right hippocampus and between the left angular gyrus and the right hippocampus. However, other pairs of functional connectivity were not related to sleep quality.

The effect of sleep quality on resting state functional connectivity between the thalamus and the right hippocampus is consistent with Zou et al.'s (2020) findings on

patients with insomnia. However, according to Chengyang et al. (2016), healthy individuals who are asked not to sleep over night (but who do not have any sleep problem) show more positive functional thalamic connectivity with the right hippocampus. These may suggest that negative functional connectivity between the thalamus and the right hippocampus results from chronic sleep problems in daily life, rather than acute sleep deprivation. Interestingly, both the thalamus and the hippocampus indeed function as sleep and memory formation. Specifically, the thalamus has the neural circuit generating what is called sleep spindles (Steriadeet al., 1985). Sleep spindles are rhythmic waves with amplitude that rapidly rises and slowly falls during non-rapid eye movement (non-REM) sleep (De Gennaro & Ferrara, 2003; Jan et al., 2009). Normal sleep spindles may help facilitate repeated neuronal activation in the hippocampus during sleep (called hippocampal replay) and integrate episodic representations of long-term memory in cortical networks (Cox et al., 2012; Klinzing, Niethard, & Born, 2019; Payne, Ellenbogen, & Stickgold, 2008; Stickgold, 2005; Maquest, 2001). Thus, decreased thalamus-hippocampal functional connectivity among poor sleepers might indicate disturbance in their long-term memory formation; in fact, it was found that poor sleep quality influences low long-term memory performances (Durmer & Dinges, 2005). Future research is needed to clarify cognitive effects of decreased thalamus-hippocampal functional connectivity.

The present study also found that poor sleep quality was associated with negative functional connectivity between the right hippocampus and the left angular gyrus. The functions of the left angular gyrus are not clear in the context of sleep, but this subregion

is involved in a variety of cognitive functions, such as attention, language, memory retrieval, and spatial cognition (Seghier, 2012). The hippocampus also contributes to cognitive processing, especially memory consolidation and learning (Maquet, 2001). Hence, negative functional connectivity between the hippocampus and the angular gyrus may disturb some aspects of cognitive processing. As discussed in the literature review, more than half of undergraduate students have sleep problems (Lund et al., 2009), and these students tend to struggle with long-term and working memory (Durmer & Dinges, 2005), executive functions (Durmer & Dinges, 2005; Killgore, 2010), problem-solving (Killgore, 2010), and attention (Durmer & Dinges, 2005; Killgore, 2010). My results suggest that these students are also likely to show decreased functional connectivity between the right hippocampus and the left angular gyrus. Taken together, reduced functional connectivity between the right hippocampus and the left angular gyrus may be a biomarker for poor cognitive/academic performance although this is speculative. To test this hypothesis, further research is needed to comprehensively assess sleep quality, academic performance, and functional connectivity between the hippocampus and the angular gyrus among college students.

One limitation of the present study was a moderate sample size of 49 participants although this sample size was larger than the most highly cited neuroimaging studies (Szucs & Ioannidis, 2020). Another limitation is that the present study used correlational analysis, thus a causal inference between sleep and functional connectivity still remains unclear. In addition, the four question items in the QIDS were used to assess sleep quality in the present study, but another sleep assessment with more question items might be

better to measure more aspects of sleep quality comprehensively. The use of an alternative sleep assessment may change the results dramatically, thus it is important to test the replication of my findings in the future.

In conclusion, the present study examined the associations between sleep quality and resting state functional connectivity in the dDMN. The poorer sleep quality was, the more negative two pairs of functional connectivity were. They included functional hippocampal connectivity with the thalamus and the left angular gyrus. The hippocampus, thalamus, and angular gyrus have cognitive implications, e.g., memory formation, for academic performance (Guillery & Sherman, 2002; Maquet, 2001; Seghier, 2012), thus this study may provide a possible neurobiological explanation of why sleep affects academic performance. In fact, insufficient sleep negatively affects several cognitive behaviors in academia (Durmer & Dinges, 2005; Durmer & Dinges, 2005; Killgore, 2010). It is a question for future research to investigate the mediating role of functional connectivity in the relationship between sleep and these cognitive processes.

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