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Effects of acute sleep deprivation on the brain function of individuals with migraine: a resting-state functional magnetic resonance imaging study

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Abstract

Background Sleep deprivation can trigger acute headache attacks in individuals with migraine; however, the underlying mechanism remains poorly understood. The aim of this study was to investigate the effects of acute sleep deprivation (ASD) on brain function in individuals with migraine without aura (MWoA) via functional magnetic resonance imaging (fMRI).

Methods Twenty three MWoA individuals and 23 healthy controls (HCs) were fairly included in this study. All participants underwent two MRI scans: one at baseline (prior to sleep deprivation) and another following 24 h of ASD. Images were obtained with blood-oxygen-level-dependent and T1-weighted sequences on a Siemens 7.0 T MRI scanner. We conducted analyses of changes in the low-frequency fluctuations (ALFF) values and functional connectivity (FC) between brain networks and within network before and after ASD in both MWoA group and HC group. Additionally, we investigated the relationship between the changes in ALFF before and after ASD and the clinical features (VAS and monthly headache days).

Results In the HC group, ASD led to a significant increase in ALFF values in the left parahippocampal gyrus compared to baseline (p-FDR = 0.01). In the MWoA group, ALFF values were significantly greater in 64 brain regions after ASD than at baseline. The most significant change in ALFF before and after ASD in the MWoA group was detected in the right medial pulvinar of the thalamus (p-FDR = 0.017), which showed a significant negative correlation with monthly headache days. Moreover, seed-based connectivity (SBC) analysis using the right medial pulvinar of the thalamus as the seed point revealed significantly increased connectivity with the cerebellar vermis (p-FWE = 0.035) after ASD in individuals with MWoA, whereas connectivity with the right postcentral gyrus was significantly decreased (p-FWE = 0.048). Furthermore, we performed analyses of between-network connectivity (BNC) and within-network connectivity across 17 brain networks, utilizing the Yeo-17 atlas. Both MWoA individuals and HCs

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showed no significant changes in BNC after ASD compared to baseline. However, our analysis in within-network revealed that MWoA individuals exhibited a reduced within-network FC in dorsal attention network (DAN) after ASD compared to baseline (p-FDR = 0.031), whereas HCs showed no significant differences in within-network FC across all networks before and after ASD.

Conclusions In comparison to HCs, MWoA individuals exhibited significant alterations in brain function after ASD, particularly within the thalamus, and MWoA individuals exhibited a reduced within-network FC in DAN after ASD compared to baseline. Brain regions and networks in MWoA individuals were more susceptible to the effects of ASD.

Keywords Migraine without aura, Acute sleep deprivation, Amplitude of low-frequency fluctuations, Functional connectivity, Functional magnetic resonance imaging

Introduction

Sleep plays a critical role in the pathophysiology of migraine, with sleep deprivation exacerbating pain sensitivity in individuals with migraine [1] and frequently triggering attacks [2–5], whereas sufficient sleep can alleviate migraine symptoms [4]. However, the mechanism by which sleep deprivation facilitates migraine attacks remains unclear.

Functional magnetic resonance imaging (fMRI) is a non-invasive imaging technique that can combine different analytical methods to study the hemodynamic changes associated with advanced brain functions, and has greatly improved our ability to understand the pathophysiology of migraine [6]. fMRI study demonstrated that in healthy controls (HCs), acute sleep deprivation (ASD) induced hyperalgesia by enhancing the pain response in the primary sensory areas of the cerebral cortex but diminishing the activity in other regions that involved in pain modulation, such as the striatum and the insula [7]. Previous studies based on electroencephalography and electromyography revealed that in individuals with migraine, ASD decreased sensorimotor cortex synchronization [8] and altered cortical excitability [9]. However, no fMRI studies have been conducted to explore functional changes after sleep deprivation in individuals with migraine.

Resting-state fMRI (rs-fMRI) is based on the spontaneous oscillation of blood oxygen level-dependent (BOLD) signals [10] and these signals derive from the synaptic-level processing of neuronal information in specific brain regions, guided by the paramagnetic properties of blood [11, 12]. The analytical methods for rs-fMRI data can be categorized into two types: one that deals with functional connectivity (FC), and another that addresses local brain activity [13]. In the latter type, the amplitude of low frequency fluctuations (ALFF) is a commonly used measure. ALFF measures the intensity of spontaneous low-frequency fluctuations in the BOLD signal, predominantly in the frequency range of 0.01 to 0.1 Hz [14]. ALFF is considered to reflect regional neural activity and is linked to the brain's fundamental metabolic activity [15]. ALFF has been used as an effective and reliable neuroimaging marker to explore the intrinsic or spontaneous brain activity in various neuropsychiatric disorders [16–19]. Previous study found that, compared to HCs, individuals with MWoA exhibited significantly lower ALFF values in the primary somatosensory cortex and the right premotor cortex [20]. Additionally, the ALFF value in the right lingual gyrus (LG) was abnormal, and the ALFF value in the right LG was positively correlated with anxiety scores in MWoA individuals with anxiety [21]. FC denotes the temporal dependency between neurophysiological events that occur in spatially remote brain regions. In the context of functional neuroimaging, FC has been proposed to describe the relationship between neuronal activation patterns in anatomically separated brain regions, reflecting the level of functional communication between regions [22]. FC analysis includes various forms such as seed-based connectivity (SBC)analysis, region of interest (ROI)-ROI analysis, within-network analysis, and between-network connectivity (BNC) analysis [23, 24]. FC analysis is widely applied in the study of neurological disorders, including migraine. Research showed that migraine individuals exhibited changes in FC compared to HCs [25]. Additionally, during the migraine attack period induced by PACAP 38, compared to the interictal period, FC in the salience network, the default mode network (DMN), and the sensorimotor network were altered [26]. Furthermore, during the ictal period of migraine with aura, compared to the interictal period, there was increase in FC between the pons and the somatosensory cortex, as well as between the visual area V5 of the symptomatic hemispheres and the middle frontal gyrus [27]. In neuroimaging research, voxel level, cluster level, regional level, and network level are four levels of investigation in studying brain function [28]. Each voxel represents a small three-dimensional element in the brain, containing a complex mixture of neural and non-neural components, such as neurons, glial cells, blood vessels, and interstitial fluid. A cluster refers to a group of adjacent voxels that share similar functional characteristics, while ROIs are collections of voxels that are defined

based on anatomical criteria such as atlas based parcellation. The brain functional network refers to the patterns of activity and synchrony among different regions of the brain while performing specific tasks or at rest. These networks reflect the collaborative manner in which the brain processes information.

After the widespread adoption of 3T MRI, neuroimaging researchers are increasingly gaining access to 7T MRI scanners. 7T MRI has the ability to greatly enhance our knowledge of brain anatomy, function and metabolism. Due to its increased signal-to-noise ratio, spatial resolution and susceptibility effects [29], 7T MRI has potential advantages in BOLD signal recognition and judgment compared to lower field strengths. Nevertheless, research on the application of 7T MRI in the field of migraine is still limited.

It is important to characterize the imaging features of the MWoA individuals after ASD. In this context, we conducted two 7T MRI scans at baseline and after ASD for both individuals with MWoA and HCs. In the study, ALFF, SBC, BNC and within-network analyses were performed to explore the changes within-group (ASD vs baseline) and between HCs and MWoA individuals before and after ASD. Understanding the relationship between fMRI metrics and clinical characteristics is crucial for elucidating the pathophysiological mechanisms of migraine, so we also investigated the relationship between changes in ALFF values before and after ASD and the clinical features (VAS and monthly headache days). This study may offer clues for better illustrating the mechanisms by which sleep deprivation triggers migraine attacks in future research.

Materials and methods

Subjects and clinical assessment

This study was approved by the Ethics Committee of the Chinese PLA General Hospital (approval number: S2024-070). All participants were thoroughly informed about the study's objectives and procedures, and written informed consent was obtained from each participant.

Twenty-six MWoA individuals were recruited from the International Headache Center in the Chinese PLA General Hospital. The diagnosis for each participant was confirmed by two senior neurologists according to the criteria of the International Classification of Headache Disorders, 3rd edition (ICHD-3). None of the individuals were on preventive medications for migraine. Additionally, 24 HCs were recruited through advertisements and matched for age, sex, and education level to the MWoA individuals. HCs had no personal or family history of migraine or any other primary headache. The exclusion criteria for all participants were as follows: left-handedness; age younger than 18 years; other chronic pain conditions (e.g., fibromyalgia or tension-type headache); neurological or psychiatric disorders; cardiovascular or metabolic diseases; nicotine, drug, or alcohol abuse; and contraindications for MRI scanning. None of the participants had sleep disorders. Each participant underwent two MRI scans, one at baseline (prior to sleep deprivation) and the other after 24 h-ASD, with the interval between the two MRI scans being the 24 h of ASD (the detailed protocol is shown in Fig. 1). All participants wore Huawei smartwatches to ensure that no sleep occurred during ASD. The participants refrained from taking acute pain relief medications, as well as consuming coffee, tea, cola, or other neurostimulants, for 24 h prior to the MRI scans. All the scans were conducted while the participants were not experiencing a migraine attack. To minimize the impact of hormonal levels, the female participants were not in their menstrual phase of the menstrual cycle.

Standardized questionnaires were used to collect demographic and clinical data, including sex, age, education level, migraine duration, monthly headache days, and headache severity (visual analogue scale [VAS] score). Anxiety and depressive symptoms were assessed

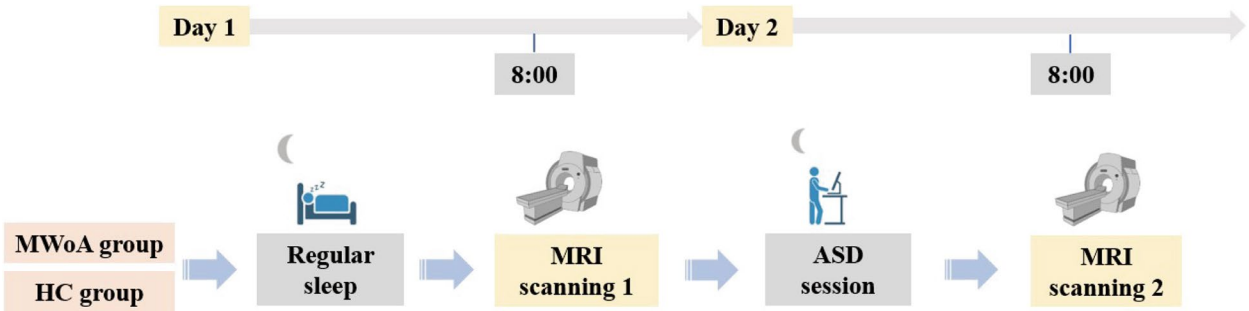

Fig. 1 Experiment paradigm of the study. On the first day, participants adhered to their regular sleep patterns and underwent an initial MRI scan at 8:00. ASD session commenced at 8:00 on the first day and continued until 8:00 on the second day, followed by a second MRI scan. Abbreviations: MWoA Migraine without aura, HCs healthy controls, MRI Magnetic resonance imaging, ASD Acute sleep deprivation

with the Generalized Anxiety Disorder 7 (GAD-7) scale and the Patient Health Questionnaire-9 (PHQ-9), respectively. Sleep quality was evaluated with the Pittsburgh Sleep Quality Index (PSQI).

MRI acquisition

All MRI data were acquired with a Siemens 7T scanner at the First Medical Center of the Chinese PLA General Hospital, utilizing both 3D T1 and BOLD imaging sequences. The participants were instructed to lie supine with their eyes closed, remain awake, and refrain from engaging in specific thoughts during functional imaging. Foam padding was used to restrict head movement, and earplugs were provided to minimize the impact of scanner noise.

The scanning parameters were as follows: 3D T1 MP2RAGE sequence: repetition time (TR) = 6000 ms, echo time (TE) = 2.21 ms, field of view (FOV) = 224 × 216 × 156 mm^3, slice thickness = 0.75 mm, slice gap = 0.375 mm, voxel size = 0.7 × 0.7 × 0.8 mm^3, 300 slices, flip angle = 4°; BOLD EP2D-TRACE-TRA sequence: TR = 2000 ms, TE = 24 ms, FOV = 216 × 216 × 144 mm^3, slice thickness = 1.8 mm, no slice gap, voxel size = 1.8 × 1.8 × 1.8 mm^3, 80 slices, flip angle = 90°. The T1 structural images were reviewed by two experienced radiologists, and participants with abnormal brain structures or pathological changes, including white matter lesions, were excluded from the study.

Functional MRI data processing and analysis

Preprocessing and denoising

Data were analysed with CONN (RRID:SCR_009550) release 22.a and Statistical Parametric Mapping (SPM) (RRID:SCR_007037) release 12.7771. Functional and anatomical data were preprocessed via a preprocessing pipeline including realignment, slice timing correction, outlier detection, indirect segmentation based on T1-weighted images, Montreal Neurological Institute (MNI)-space normalization, and smoothing. The functional data were realigned via the SPM realign and unwarp procedure, and resampled via b-spline interpolation to correct for motion and magnetic susceptibility interactions. Temporal misalignment between different slices of the functional data was corrected according to the SPM slice-timing correction (STC) procedure. Potential outlier scans were identified using the artefact removal tool (ART) as acquisitions with framewise displacement greater than 0.9 mm or global BOLD signal changes above 5 standard deviations, and a reference BOLD image was computed for each subject by averaging all scans excluding outliers. The functional and anatomical data were coregistered and normalized into standard MNI space and resampled to 2 mm isotropic voxels according to an indirect normalization procedure. Finally, the functional data were smoothed via spatial convolution with a Gaussian kernel of 3 mm full width at half maximum (FWHM). Functional data were denoised via a standard denoising pipeline followed by bandpass frequency filtering of the BOLD time series between 0.01 Hz and 0.1 Hz. ALFF maps characterizing the low-frequency BOLD signal variability at each voxel were estimated as the root mean square (RMS) of the BOLD signal after denoising and bandpass filtering between 0.01 Hz and 0.1 Hz.

ALFF analysis on cluster-level

We referred to the methods used in Eklund's study [30]. For each individual voxel, a separate general linear model (GLM) was estimated, with ALFF value at this voxel as dependent variables and group identifier as independent variable. Inferences were performed at the level of individual clusters (groups of contiguous voxels). Cluster-level inferences were based on nonparametric statistics from permutation analyses ($n = 5000$). Results were thresholded using a combination of a cluster-forming $p < 0.001$ voxel-level threshold, and a familywise corrected p-FWE < 0.05 cluster-mass threshold.

ALFF analysis on ROI-level

Additionally, we divided the brain into 166 distinct regions according to the AAL3 brain region segmentation atlas and calculated the mean ALFF value for each region. We conducted within-group comparisons of the average ALFF values across 166 brain regions before and after ASD in HCs and individuals with MWoA, applying false discovery rate (FDR) correction to the *P*-values, with FDR < 0.05 considered statistically significant.

SBC maps were estimated characterizing the spatial pattern of FC with a seed area. The right medial pulvinar of the thalamus was chosen as seed area. FC strength was represented by Fisher-transformed bivariate correlation coefficients from a weighted-GLM, estimated separately for each seed area and target voxel, modeling the association between their BOLD signal timeseries. Individual scans were weighted by a boxcar signal characterizing each individual task or experimental condition convolved with an SPM canonical hemodynamic response function and rectified. Inferences were performed at the level of individual clusters (groups of contiguous voxels). For each individual voxel, a separate GLM was estimated, with first-level connectivity measures at this voxel as dependent variables and group identifiers as independent variable. Voxel-level *P*-values were evaluated using multivariate parametric statistics with random-effects across subjects and sample covariance estimation across

multiple measurements. Cluster-level inferences were based on nonparametric statistics from permutation analyses, with 5000 residual-randomization iterations. Results were thresholded using a combination of a cluster-forming $P < 0.01$ voxel-level threshold, and a family-wise corrected p-FWE < 0.05 cluster-mass threshold.

BNC analysis

Network-network connectivity matrices were estimated to characterize the FC between each pair of regions among 17 networks based on Yeo-17 atlas [31], without selecting specific networks. The Yeo-17 atlas was derived from a clustering approach and aimed at providing a standardized model of brain functional networks. The FC strength was represented by Fisher-transformed bivariate correlation coefficients obtained from a weighted GLM, estimated separately for each pair of networks, characterizing the association between their BOLD signal time series. We performed adequate correction for the BNC ($n = 17$ * 16 / 2 = 136 corrections), and applied a threshold of FDR < 0.05 and conducted cluster-level correction (MVPN omnibus test FWE < 0.05).

Within-network connectivity

We conducted within-network connectivity analysis following the methodology outlined in the paper by Yan et al. [32]. We selected the Yeo-17 atlas [31] as our brain network template and used the AAL3 atlas as our ROI template. Specifically, after preprocessing, time series of BOLD values for each AAL3 functional ROI was extracted. We defined within-network ROIs as those overlapping with each network in the Yeo-17 atlas. Average FC of the within-network ROIs was calculated to represent within-network FC. Mean within-network FC (averaged across n * (n-1) / 2 connections among n ROIs) was compared before and after ASD in both MWoA individuals and HCs. Within-group comparisons were performed using non-parametric tests. All comparisons with FDR-corrected *P*-values of < 0.05 were considered statistically significant.

Clinical correlation analysis

The correlations between clinical features (VAS and monthly headache days) and changes in ALFF after ASD in each ROI were evaluated by GLM model, which involved age, sex, and PSQI as covariates. To address the issue of multiple comparisons, we applied the FDR method for *P*-value correction, adjusting for the number of clinical features involved in this study.

Statistical analysis

IBM SPSS Statistics 26.0 software and R software (4.3.2) were used to for statistical analyses. The categorical variables were compared via the chi-square test or Fisher-exact test. Continuous variables, such as age and education level, were assessed for normality via the Shapiro–Wilk test. Normally distributed data were presented as the means ± standard deviations, and independent samples t-test was used for between-group comparisons when homogeneity of variance was confirmed. Nonnormally distributed data were presented as medians (interquartile ranges), and between-group comparisons were performed via the Mann–Whitney U test.

Results

Demographic data and clinical characteristics

A total of 26 MWoA individuals and 24 HCs were recruited for this study. 1 MWoA individual exhibited excessive head movement during image acquisition, and 1 HC individual had poor quality MRI data. Additionally, two MWoA individuals experienced migraine attacks after sleep deprivation; however, both of them were not in the ictal period of migraine during the second MRI scan and fell asleep due to drowsiness. Therefore, the data from these four subjects were excluded from further analysis. Ultimately, 23 MWoA individuals and 23 HCs were included in the final analysis. All MWoA individuals kept headache diaries throughout the study, and they were in the interictal period of migraine during both MRI scans. Table 1 shows the demographic and clinical data for the MWoA and HC groups. There were no significant differences between the MWoA and HC groups in terms of age ($P = 0.93$), sex ($P = 1$), education level ($P = 0.22$), or GAD-7 score ($P = 0.096$). However, the PHQ-9 (5.00 vs 1.00, $P = 0.001$) and PSQI (5.96 vs 4.17, $P = 0.028$) scores in

Table 1 Demographic data and clinical characteristics of participants

	MWoA group	HC group	*P*
Age (years)	26.00 (3.00)	26.00 (5.00)	0.93
Sex (male/female)	6/17	6/17	1
Education level (years)	19.00 (2.00)	19.00 (2.00)	0.22
PHQ-9	5.00 (4.00)	1.00 (2.00)	0.001
GAD-7	3.00 (6.00)	1.00 (3.00)	0.096
PSQI	5.96 ± 2.79	4.17 ± 2.52	0.028
Disease duration (years)	8.00 (5.00)	-	
Pain intensity (VAS)	6.00 (3.00)	-	
Monthly headache days	3.70 ± 1.82	-	

Abbreviations: MWoA Migraine without aura, *HC* Healthy control, *PHQ-9* Patient health questionnaire-9, *GAD-7* Generalized anxiety disorder-7, *PSQI* Pittsburgh sleep quality index, *VAS* Visual analogue scale

the MWoA group were significantly higher than those in the HC group.

Comparison of ALFF values and BNC before and after ASD between MWoA individuals and HCs

There were no significant differences in the ALFF values between MWoA individuals and HCs in the voxel clusters before and after ASD. Additionally, there were no significant differences in brain regions between the two groups before and after ASD.

Furthermore, there were no significant differences in the FC of the brain networks between MWoA individuals and HCs before and after ASD.

Changes of within-group ALFF values after ASD compared with baseline in MWoA individuals and HCs

In HCs, no significant voxel clusters in ALFF values were observed after ASD compared with baseline. Subsequent ROI-level analyses indicated that the ALFF value in the left parahippocampal gyrus after ASD was significant higher than that in baseline status (p-FDR = 0.01). For specific details, please refer to Supplementary Material 1.

Compared with those at baseline, MWoA individuals presented more pronounced changes in ALFF values after ASD. These altered clusters were located primarily in the thalamus (p-FWE = 0.013) and cerebellum (p-FWE = 0.038) (Table 2 and Fig. 2A). In ROI-level, ASD caused significant alterations in ALFF in 64 brain regions, including the right medial pulvinar of the thalamus (p-FDR = 0.017), right lateral pulvinar of the thalamus (p-FDR = 0.022), right posterolateral nucleus of the thalamus (p-FDR = 0.022), and left lobule III of the cerebellar hemisphere (p-FDR = 0.022) (Fig. 2B-C). For specific details, please refer to Supplementary Material 2.

Changes of within-group SBC after ASD compared with baseline in MWoA individuals

SBC analysis using the right medial pulvinar of the thalamus as the seed point revealed significantly increased connectivity with the cerebellar vermis (p-FWE = 0.035) after ASD in individuals with MWoA, whereas connectivity with the right postcentral gyrus was significantly decreased (p-FWE = 0.048). For specific details, please refer to Fig. 3 and Supplementary Material 3.

Table 2 The changes in ALFF values in clusters after ASD compared to baseline in MWoA individuals

MNI coordinate (x, y, z)	Brain region	Cluster size (voxels)	Cluster mass FWE
+12, −24, +16	Thal_PuM_R, Thal_PuI_R, Thal_VL_L	282	0.013
+20, −38, −28	Cerebellum_4_5_R	93	0.038

Abbreviations: *ALFF* Amplitude of low-frequency fluctuations, *ASD* Acute sleep deprivation, *MWoA* Migraine without aura, *MNI* Montreal neurological institute, *FWE* Family-wise error, *Thal_PuM_R* Right medial pulvinar of thalamus, *Thal_PuI_R* Right inferior pulvinar of thalamus, *Thal_VL_L* Left inferior pulvinar of thalamus, *Cerebellum_4_5_R* Right lobule IV, V of cerebellar hemisphere

Changes of BNC and within-network connectivity after ASD compared with baseline in both MWoA individuals and HCs

Both MWoA individuals and HCs showed no significant changes in BNC after ASD compared to baseline. However, MWoA individuals exhibited a decrease in within-network connectivity in dorsal attention network (DAN) after ASD compared to baseline (p-FDR = 0.031), as shown in Fig. 4. In contrast, HCs showed no significant differences in within-network connectivity across the various networks between baseline and after ASD.

Correlation between clinical parameters and ALFF changes before and after ASD in MWoA individuals

The changes in the ALFF values before and after ASD in 2 out of 64 brain regions exhibited a significant negative correlation with monthly headache days, including the right medial pulvinar of the thalamus (p-FDR = 0.044) and the right anterior pulvinar of the thalamus (p-FDR = 0.035). At the same time, the changes in the ALFF values before and after ASD in 18 out of 64 brain regions were significantly negatively correlated with the VAS scores in MWoA individuals, including but not limited to the right lobule IV, V of cerebellar hemisphere (p-FDR = 0.011). The results are presented in Fig. 5 and Supplementary Material 6.

Discussion

To our knowledge, it is the first fMRI study to indicate changes in ALFF values, BNC and within-network connectivity before and after ASD within the MWoA group. Our findings indicated that ASD had a greater impact on ALFF values and within-network FC in MWoA individuals than in HCs.

The imbalance between energy production and demand in the brain is an important factor in the pathogenesis of migraine [33]. Ashina suggested that modulation of nociceptive transmission by ATP-sensitive potassium (K_{ATP}) channels could represent a final common pathway for migraine attacks [34], thereby supporting the hypothesis that brain energy metabolism was involved in the pathogenesis of migraine. Harmen et al. discovered a significant decrease in ATP levels in the medial occipital lobe of individuals with MWoA. The lowest ATP level was observed in individuals with the highest frequency of attacks, suggesting a modest correlation between

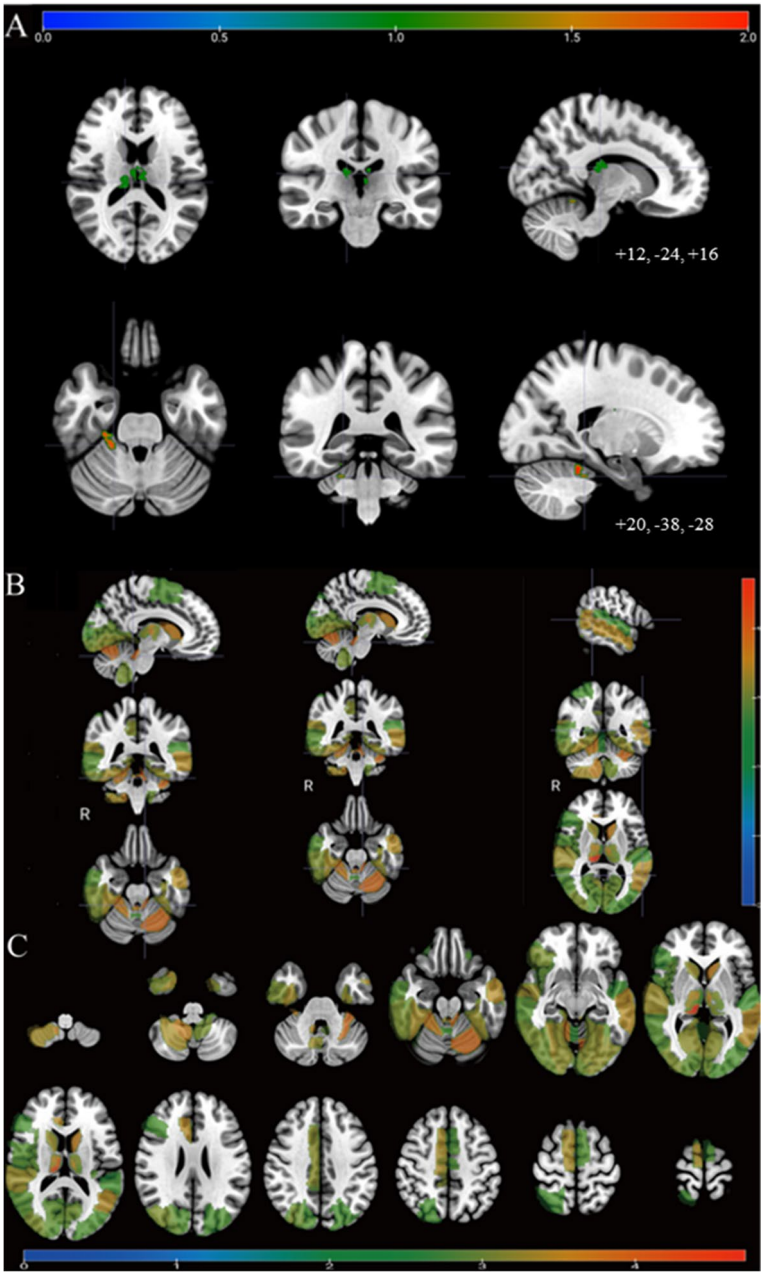

Fig. 2 The clusters and ROI-level regions with changes of ALFF after ASD compared to baseline in MWoA individuals. The clusters of ALFF changes after ASD compared to baseline were mainly distributed in thalamus and cerebellum (**A**). Additionally, ASD caused significant ALFF changes in 64 brain regions (**B-C**), including the right medial pulvinar of thalamus, right lateral pulvinar of thalamus, right posterolateral nucleus of thalamus, left lobule III of cerebellar hemisphere. The colour bar represents the T values. Abbreviations: ROI region of interest, ALFF Amplitude of low-frequency fluctuations, ASD Acute sleep deprivation, MWoA Migraine without aura

reduced brain metabolism and migraine susceptibility [35]. Besides, research indicated that a single night of sleep deprivation in healthy adults led to a significant reduction in plasma levels of glutathione, ATP, cysteine, and homocysteine [36]. This suggests that sleep deprivation may exert an impact on human energy metabolism system, resulting in an inadequate energy supply. Many observational studies suggested that most migraine triggers or exacerbating factors such as skipping a meal or fasting, exercise, dehydration, lack of sleep, hormonal changes, intense physical or psychological stress, intense sensory stimuli were associated with energy metabolism

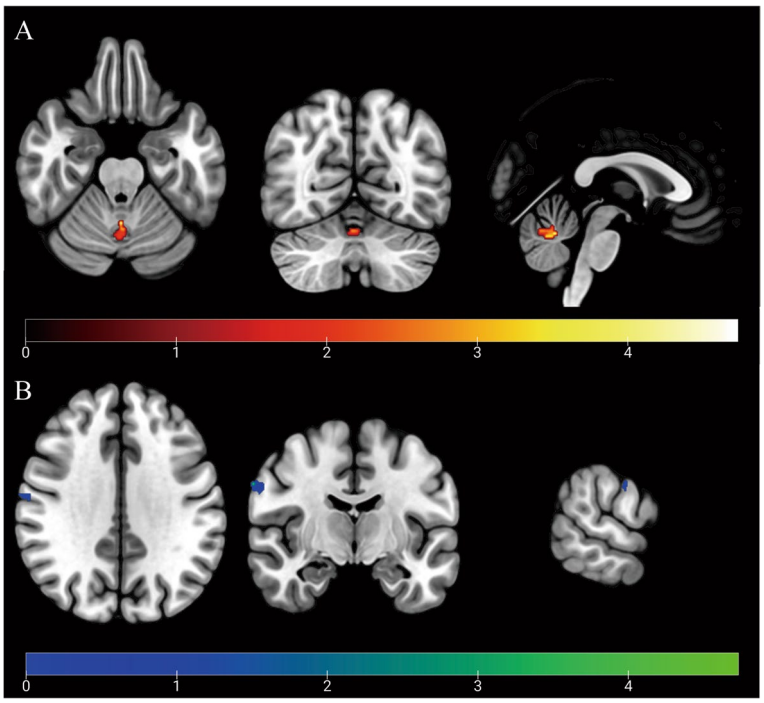

Fig. 3 Changes of within-group SBC after ASD compared with baseline in MWoA individuals. After ASD in MWoA individuals, using the right medial pulvinar of thalamus as the seed point, it was found that the FC with the cerebellar vermis was significantly enhanced (**A**), while the FC with the right postcentral gyrus was significantly decreased (**B**). The colour bar represents the absolute values of T values. Abbreviations: SBC seed-based connectivity, ASD Acute sleep deprivation, MWoA Migraine without aura

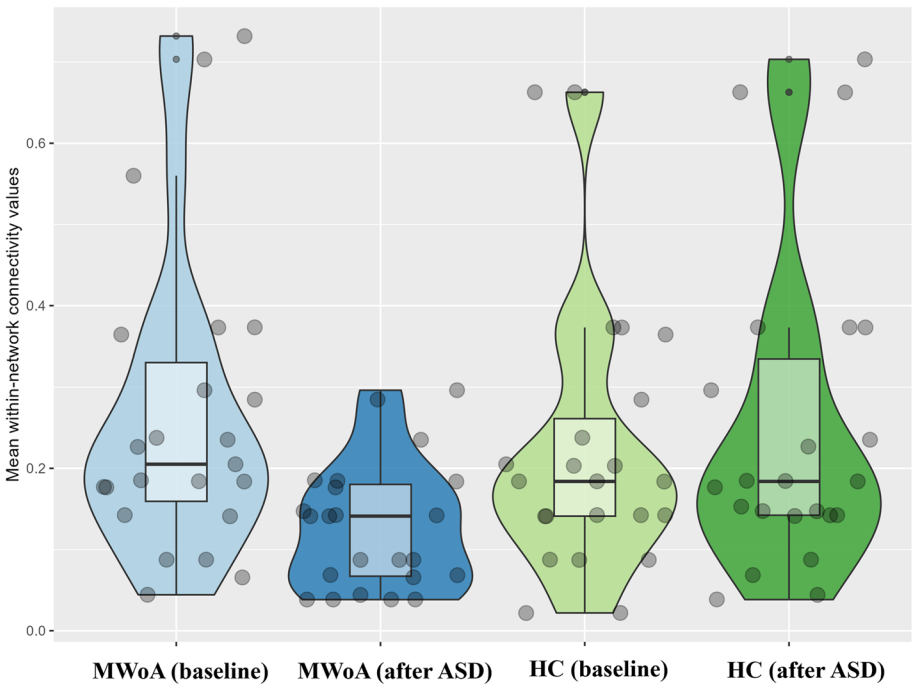

Fig. 4 Mean within-network connectivity in DAN at baseline and after ASD in MWoA individuals and HCs based on the Yeo-17 atlas. MWoA individuals exhibited a decrease of within-network connectivity in DAN after ASD compared to baseline, and in contrast, HCs showed no significant differences of within-network connectivity in DAN before and after ASD. Abbreviations: DAN Dorsal attention network, ASD Acute sleep deprivation, MWoA Migraine without aura, HC Healthy control

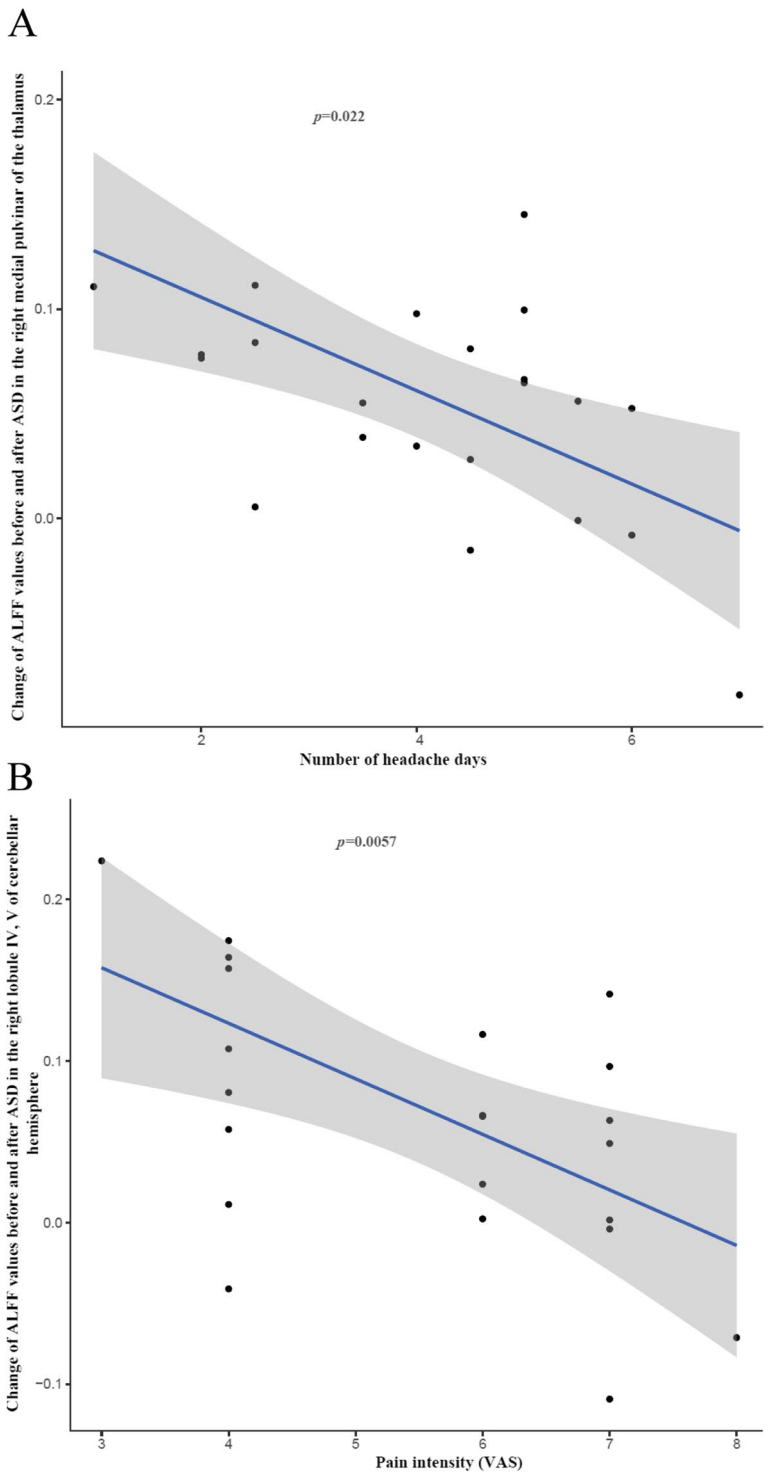

Fig. 5 Correlation analysis between change of ALFF value before and after ASD in the right medial pulvinar of the thalamus or the right lobule IV, V of cerebellar hemisphere and clinical characteristics in MWoA individuals. After adjusting for age, sex, and PSQI scores in the GLM model, a significant negative correlation was found between the change of ALFF values before and after ASD in the right medial pulvinar of the thalamus and monthly headache days (**A**). Furthermore, a significant negative correlation was found between the change of ALFF values before and after ASD in the right lobule IV, V of cerebellar hemisphere and VAS scores (**B**). Dots represent individuals with MWoA. The blue regression line represents Pearson's correlation coefficient. Grey shading represents the 95% confidence intervals of the partial correlations. Abbreviations: MWoA Migraine without aura, PSQI Pittsburgh sleep quality index, GLM General Linear Model, ALFF Amplitude of low-frequency fluctuations, ASD Acute sleep deprivation, VAS Visual analog scale

[37]. Therefore, we propose that sleep deprivation may facilitate migraine attacks by causing a decrease in energy reserves. Increased ALFF values in brain regions indicate heightened neural activity, which may be a compensatory mechanism of the brain to cope with ASD. In HCs, only the left parahippocampal gyrus showed significantly increased ALFF values after ASD. In contrast, MWoA individuals presented significant increases in ALFF values across 64 brain regions after ASD, suggesting that more regions may experience an energy metabolism imbalance following ASD. These findings provide preliminary evidence supporting increased susceptibility to migraine attacks following ASD.

Our study found that there were significant changes in the ALFF values in multiple regions of the thalamus after ASD in individuals with MWoA. The thalamus has been found to be involved in various sleep–wake control mechanisms [38]. A previous study reported greater thalamic activation during sleep deprivation than during normal wakefulness in HCs [39], which was consistent with our findings in MWoA individuals. Furthermore, the thalamus plays an important role in the pathogenesis of migraine. Our research also identified an abnormal higher-order thalamocortical communication pattern in migraine patients [40]. Furthermore, another neuroimaging study revealed that the thalamus was activated during the premonitory phase of a migraine attack and remained active throughout the headache phase, contributing to the onset of pain and related symptoms [41]. Therefore, we can infer that the thalamus plays a crucial role in the mechanism by which sleep deprivation facilitates migraine. Notably, the right medial pulvinar of the thalamus showed the most pronounced elevation after ASD, which also exhibited a significant negative correlation with monthly headache days. A previous study reported that the pulvinar nucleus of the thalamus sent broad projections to the V1, V2, auditory, and somatosensory cortices, and these projections played a crucial role in the photophobia and allodynia during migraine [42, 43]. Furthermore, the medial pulvinar nucleus exhibited a reciprocal connection with the higher-order cortex and paralimbic areas, relaying higher-order information [44]. Consequently, aberrant signal variability in the medial pulvinar nucleus would give rise to disrupted thalamocortical information flow, thereby resulting in perturbations of multisensory integration and higher cognitive processing [45]. Our study revealed that individuals with MWoA exhibited a heightened vulnerability of the right medial pulvinar of the thalamus to ASD, particularly in those experiencing fewer monthly headache days. This may be due to sleep deprivation causing an increase in thalamic activation within a certain range. However, individuals with fewer monthly headache days have a lower level of thalamic activation at baseline compared to those with more monthly headache days, resulting in a greater change in thalamic activation levels following sleep deprivation.

Resting-state fMRI studies have identified disrupted FC between the thalamus and cortical as well as subcortical regions implicated in pain processing during both spontaneous and induced migraine attacks, supporting the hypothesis of thalamic involvement in migraine [46, 47]. In this study, there was an increase in FC between the right medial pulvinar of the thalamus and the cerebellar vermis after ASD, while the FC with the right postcentral gyrus decreased. The cerebellar vermis plays a crucial role in pain modulation [48]. The postcentral gyrus, a critical region within the cerebral cortex responsible for somatic sensory processing [49], receives axonal projections from the ventral posterolateral nucleus of the thalamus [50]. This area is essential for integrating and processing various sensory modalities such as tactile sensations, nociception, and thermal perception [51]. The observed alterations in FC between the right medial pulvinar of the thalamus and the two regions imply a significant impact of ASD on both pain perception and processing.

ASD significantly increased the ALFF values in multiple regions of the cerebellum, occipital lobe, and temporal lobe in individuals with MWoA, thereby broadening our understanding of the functions of these brain regions. The cerebellum was proposed to modulate pain processing in individuals with migraine, and imaging studies revealed cerebellar alterations in individuals with migraine [52]. Spontaneous activity in the left superior cerebellum could discriminate migraine patients from HCs, and this activity was positively correlated with baseline headache intensity [53]. Additionally, cerebellar activation was increased during the ictal phase compared with the interictal phase, especially in female migraine patients [54]. Previous research showed that patients with chronic primary insomnia exhibited higher ALFF values in the left anterior lobe of the cerebellum than HCs do [55], suggesting that the cerebellum was involved in sleep regulation. In this study, the changes in the ALFF values in the right lobule IV, V of cerebellar hemisphere before and after ASD were significantly negatively correlated with the VAS scores in MWoA individuals, indicating that the lower the VAS score was, the greater the increase in ALFF in the region following ASD. This indicates that sleep deprivation affects the function of the cerebellum, which in turn impacts the functional state of individuals with MWoA. Photophobia and phonophobia are prevalent symptoms among individuals with MWoA, in which the occipital and temporal lobes participate in visual and auditory information processing respectively. During migraine attacks, these brain regions were significantly

activated [56–59]. The increase in ALFF values in these regions after ASD may reflect increased sensitivity to external stimuli among MWoA individuals, particularly visual, auditory and pain hypersensitivity.

In the study, both MWoA individuals and HCs showed no significant changes in BNC after ASD compared to baseline, suggesting that 24 h of ASD had a minimal impact on the FC between brain networks in these subjects. However, MWoA individuals but not HCs exhibited a decrease in within-network connectivity in DAN after ASD compared to baseline, suggesting that ASD disrupted the within-network FC in MWoA individuals. The Yeo-17 atlas divides the cerebral cortex into 17 functional networks [31], such as the DMN, DAN, limbic network, salience/ventral attention network, and somatomotor network. DAN, anchored in the lateral frontoparietal cortex, is the core network of "top-down" attention mechanism [60] and plays a crucial role in the orientation and maintenance of attention. Individuals with migraine exhibited attentional impairments during a migraine attack as well as in the interictal period [61, 62], suggesting that the occurrence of migraine might involve the participation of DAN. Research has found the DAN is functionally and anatomically connected to the trigeminal system [63], playing a crucial role in pain perception. Furthermore, neuroimaging study found the intrinsic FC of the DAN was reduced during the interictal period of episodic migraine compared with HCs [64]. These studies collectively indicate that the dysfunction of the DAN is indeed involved in the pathogenesis of migraine. In this study, it was found that compared to the baseline, individuals with MWoA exhibited a decrease in FC within the DAN following ASD. This suggested that DAN was affected by ASD, which might impact the attention and pain perception in individuals with MWoA. Overall, our study found that MWoA individuals exhibited significant changes in brain function following ASD.

Limitations and future direction

This study has two limitations that should be considered. Firstly, the sample size in this study was relatively small, and the age range and educational level of the participants were relatively limited; therefore, these findings represent brain functional changes after ASD only in young, highly educated individuals. Future research should increase the sample size and recruit participants across a wider range of ages to conduct more comprehensive subgroup studies in MWoA individuals. Secondly, none of the MWoA individuals included in the final analysis reported experiencing headache attacks following ASD. Future research should focus on increasing the sample size and comparing individuals who experience headache attacks after ASD with those who do not, in order to more directly elucidate the underlying mechanisms responsible for headache attacks induced by ASD.

Conclusion

Compared with HCs, the brain function of MWoA individuals was broadly affected by ASD, particularly in the thalamus, cerebellum, and DAN. This study may provide valuable insights for elucidating the mechanisms through which sleep deprivation triggers migraine attacks in future research. These findings provide novel neuroimaging evidence supporting the role of sleep management in individuals with migraine.

Abbreviations

ASD	Acute sleep deprivation
MWoA	Migraine without aura
HCs	Healthy controls
fMRI	Functional magnetic resonance imaging
BOLD	Blood oxygenation level dependent
ALFF	Amplitude of low-frequency fluctuations
FC	Functional connectivity
VAS	Visual Analog Scale
GAD-7	Generalized Anxiety Disorder 7
PHQ-9	Patient Health Questionnaire-9
PSQI	Pittsburgh Sleep Quality Index
MNI	Montreal Neurological Institute
AAL	Automated anatomical labeling
FWHM	Full width at half maximum
TR	Repetition time
TE	Echo time
FOV	Field of view
RMS	Root mean square
GLM	General linear model
BNC	Between network connectivity
DMN	Default mode network
STC	Slice-timing correction
SPM	Statistical parametric mapping
ART	Artifact removal tool
DAN	Dorsal attention network
SBC	Seed-based connectivity
LG	Lingual gyrus
ROI	Region of interest
ICHD-3	International Classification of Headache Disorders 3rd edition
FDR	False discovery rate

Supplementary Information

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Supplementary Material 1.

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Authors' contributions

Study concept and design: SQW, LTM, XL, and ZD. Acquisition of data: SQW, SW, CHD, XYW, XBB, DQZ, YS, SYX, SHZ, YYL, XXL, RBW, XL, SYY. Data analysis and writing the manuscript: SQW and LTM. Review and editing, funding acquisition, supervision: XL and ZD. All authors contributed intellectual content to the revised manuscript and read and approved the final manuscript. SQW and LTM contributed equally to this article.

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Data availability
The datasets supporting this study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
The study was approved by the Ethics Committee of the Chinese PLA General Hospital in accordance with the ethical principles of the Declaration of Helsinki (S2024-070).

Consent for publication
All authors consent for the publication.

Competing interests
The authors declare no competing interests.

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