Brain Imaging and Behavior (2019) 13:638–650
https://doi.org/10.1007/s11682-018-9868-2

ORIGINAL RESEARCH

Decreased cortical and subcortical response to inhibition control after sleep deprivation

Rui Zhao[1] · Xinxin Zhang[1] · Ningbo Fei[1] · Yuanqiang Zhu[1] · Jinbo Sun[1] · Peng Liu[1] · Xuejuan Yang[1] · Wei Qin[1]

Published online: 10 May 2018

Abstract

The effects of sleep deprivation (SD) on the neural substrates of inhibition control are poorly understood. Here we used functional magnetic resonance imaging to examine the effects of 24 h of SD on cerebral activation during a stop-signal task in 20 normal young subjects. Behaviorally, subjects showed significantly delayed stop-signal reaction time (SSRT) following SD. In addition, reduced cerebral activation was found in the "stopping network" (including the inferior frontal gyrus [IFG], supplementary motor area, subthalamic nucleus [STN] and insula) and vision-related regions (occipital cortex, lingual gyrus and fusiform gyrus) after SD. These findings support the hypothesis that task-related activation in prefrontal cortex is particularly vulnerable to SD. After rested wakefulness (RW), significant negative correlations were found between SSRT and cerebral activation in left IFG, right hippocampus, right lingual gyrus, left STN and bilateral fusiform gyrus, with activation in left IFG making the most contribution. After SD, significant negative correlations were found between SSRT and activation in right middle frontal cortex, right IFG and left lingual gyrus, with the activation in right IFG making the most contribution. Furthermore, we observed significant interaction effects of state (SD or RW) with activation in bilateral IFG, left STN and left lingual gyrus on SSRT. In conclusion, sleep deprivation is associated with the deficits in inhibition-related neural activation and the altered correlation between SSRT and cerebral activation, especially in the bilateral IFG, left STN and left lingual gyrus.

Keywords Response inhibition · Sleep deprivation · fMRI · Inferior frontal gyrus · Stop-signal task

Introduction

Sleep deprivation (SD) for one night can impair the performance on many cognitive tasks (Durmer and Dinges 2005; Killgore 2010). Although the underlying neural substrates of SD-related cognitive deficits have not been well understood, numerous neuroimaging studies have suggested that task-related activation in prefrontal cortex (PFC) is particularly vulnerable to SD (Drummond et al. 2000; Basner et al. 2013; Asplund and Chee 2013; Chee and Choo 2004). While previous studies have typically focused on executive functions, such as attention (Tomasi et al. 2009; Ma et al. 2014) and working memory (Mu et al. 2005a; Lim et al. 2007; Chee et al. 2006; Mu et al. 2005b), only a few functional imaging studies have investigated the effects of SD on inhibitory control in normal subjects (Chuah et al. 2006; Almklov et al. 2015).

Inhibitory control, also known as response inhibition, is the process of rapidly canceling planned or ongoing behaviors. The stop-signal task (SST) (Logan 1994) is a widely used behavioral paradigm for assessing inhibition control, with stop-signal reaction time (SSRT) as its primary measure. Multiple functional magnetic resonance imaging (fMRI) studies using the SST have shown that the right inferior frontal gyrus (IFG) is specifically engaged in the process of inhibitory control (Aron and Poldrack 2006; Aron et al. 2007; Congdon et al. 2010; Cai et al. 2014; Xue et al. 2008). Lesion studies of patients with right IFG damage reported that SSRT of these patients was significantly slower than that of healthy controls, and the extent of damage to the right IFG was significantly correlated with SSRT (Aron et al. 2003; Floden and Stuss 2006; Picton et al. 2007). Furthermore, transcranial magnetic stimulation (TMS) (Christopher D Chambers et al. 2006; C. D. Chambers et al. 2007) studies have demonstrated that TMS of the right IFG can impair SSRT. Taken together, these findings suggest that the right IFG is critical for response inhibition

✉ Wei Qin
wqin@xidian.edu.cn

[1] Engineering Research Center of Molecular and Neuro Imaging of the Ministry of Education, School of Life Science and Technology, Xidian University, Xi'an, Shaan xi 710071, China

(Aron et al. 2014a). Given that the IFG is a sub-region of the PFC, subjects might exhibit impaired SST performance and reduced neural activation in PFC (especially in the right IFG) after SD, compared with rested wakefulness (RW).

In accord with this notion, Chuah et al. investigated the effects of SD on inhibitory control measured by a Go/No-Go task, and found decreased inhibitory efficiency and reduced activation in ventral and anterior PFC after SD (Chuah et al. 2006). Using the same task, Almklov et al. examined the age-related effects of SD on inhibitory control and found a significant interaction between age-group and sleep-deprivation status in middle frontal gyrus (Almklov et al. 2015). The Go/No-Go task and SST are both commonly used for measuring inhibition control. Although these two tasks have often been used as interchangeable measures of inhibition control, they do not engage identical neural circuits (Aron et al. 2014b; Swick et al. 2011). However, no functional imaging studies have investigated the altered SST-related activation in inhibitory control after SD.

In the present study, we first measured localized cerebral activation using the blood oxygen level dependent (BOLD) fMRI signal during the SST in 20 normal young subjects after RW and after 24 h of SD, and explored the differences of BOLD responses between RW and SD. Second, we explored the correlations between cerebral activation and SSRT under both SD and RW conditions, and examined the contribution of cerebral activation in various regions to SSRT using a relative weight method. Finally, we evaluated the effect of SD on the correlation between cerebral activation and SSRT using a general linear model (GLM).

Methods

Subjects

Twenty-two right-handed healthy subjects were recruited in this study. All subjects reported no history of medical, psychiatric, neurological or sleep disorders, and were free of any abused alcohol or drugs. Any subjects who presented an extreme morning or extreme evening type, as assessed by a questionnaire (Horne and Ostberg 1976), were excluded from the final analysis. Subjects habitually maintained normal sleep schedules of 7–9 h per night, between 10:00 pm and 8:00 am.

Two subjects opted out of this study after the total SD session. Therefore, the final analysis included 20 subjects (mean age 19.9 ± 1.77 years, range 17–23; 11 males, 9 females). All subjects provided written informed consent prior to participation and were compensated for their time. All participants declared that they did not smoke or consume any stimulants, medications, alcohol or caffeine for at least 24 h prior to the final experiment. All research procedures were conducted in accordance with the Declaration of Helsinki and approved by the institutional research ethics committee of the Xijing Hospital of the Fourth Military Medical University.

The stop-signal task

The SST was adapted from previously published studies (Logan 1994; Aron and Poldrack 2006), and consisted of a Go task (75% of trials) and a Stop task (25% of trials). On each trial, a left- or right-pointing arrow was displayed on a computer screen. For the Go task, subjects were instructed to press the left or right button as fast as possible. For the Stop task, subjects attempted to stop pressing the button when a Stop signal (the white arrow changed to blue) was presented at a particular delay (stop-signal delay; SSD) after the arrow stimulus. It should be noted that we didn't consider the common auditory stimulus as the Stop signal due to the high noise of MRI scanner and the uncomfortableness with headphone and earplugs. The SSD changed depending on inhibition performance. Specifically, the SSD was increased by 50 ms after a successful inhibition response, to make inhibiting the response more difficult on the subsequent trial, and decreased by 50 ms after a failed inhibition response, to make inhibiting the response easier on the subsequent trial. This procedure (also named staircase) was designed to produce a successful inhibition rate of approximately 50%, and to control the difficulty level across subjects. To reduce participants' anticipation, two staircases were used and respectively started with SSD values of 250 and 350 ms in each session. Each session consisted of two runs. Each run included two blocks, with 36 Go trials and 12 Stop trials in each block, and lasted for 4.5 min. Each Stop trial corresponded to one staircase. Therefore, six Stop trials corresponded to the first staircase and another six Stop trials corresponded to the second staircase. Each staircase moved six times within each block. The order of staircases was randomized trial-by-trial.

Experimental procedure

All subjects underwent two fMRI scanning sessions: after RW and after 24 h of SD. Subjects performed the SST while being scanned with fMRI. The two scanning sessions were performed in a randomized, cross-over fashion with at least one week apart to minimize possible residual effects of SD on cognition (Van Dongen et al. 2003). One week before the final experiment, subjects visited the laboratory, were informed of the experimental procedures and given instructions about the SST. Directly before each scanning session, each subject performed an SST training session. Within the scanner, subjects responded with their right hand using an MR-compatible button box.

For the RW session, subjects reported to the lab at 7:30 am. fMRI scanning began at 8:00 am and lasted for 9 min. Each subject completed the SST in the scanner. For the SD session, subjects were monitored in the lab from 10:00 pm to 8:00 am

to prevent them from falling asleep. They were allowed to engage in non-strenuous activities such as reading and watching videos. The 9-min fMRI scan with the SST began at 8:00 am.

Behavioral analyses

We used SSRT as the main behavioral measure of this task, providing an individualized measure of inhibitory control. SSRT was estimated using a quantile method (Band et al. 2003) which essentially corrects for deviations from 50% successful inhibition (Logan 1994). In brief, all reaction times (RTs) on correct Go trials were arranged in ascending order. The RT corresponding to the proportion of failed inhibition (1-StopAcc) was selected as the quantile RT. The StopAcc was the accuracy of the Stop task. SSRT was estimated by subtracting the averaged SSD from the quantile RT, with smaller SSRT values reflecting better inhibitory control. Additional measurements of interest were 1) the RT on correct Go trials (GoRT); 2) the accuracy of the Go task (GoAcc); 3) the percentage of missing Go responses (GoMiss), in which subjects failed to make a response on a Go trial; 4) the percentage of incorrect Go trials (GoError), in which subjects made an incorrect response on a Go trial (e.g., pressing the left button in response to a rightward pointing arrow and vice versa); and 5) the RT on unsuccessful Stop trials (StopRespond RT).

fMRI data acquisition

Scanning was performed in a 3 T GE MR750 scanner at Department of Radiology, Xijing Hospital, The Fourth Military Medical University, Xi'an, China. A standard 8-channel head coil was used together with a restraining foam pad to minimize head motion and diminish scanner noise. BOLD signals were acquired with a single shot gradient echo planar imaging (EPI) sequence [repetition time (TR), 2 s; echo time (TE), 30 ms; field of view (FOV), 240 × 240 mm^2; matrix, 64 × 64; in-plane resolution: 3.75 × 3.75 mm^2; slice thickness, 3.5 mm; 45 axial slices; flip angle, 90°] and 135 volumes were obtained for each run. Additionally, a high-resolution scan, T1-weighted magnetization-prepared rapid-acquisition gradient echo (MPRAGE; TR, 8.2 ms; TE, 3.18 ms; FOV, 256 × 256 mm^2; matrix, 512 × 512; in-plane resolution, 0.5 × 0.5 mm^2; slice thickness, 1 mm; 196 sagittal slices; flip angle, 9°), was collected for each participant.

fMRI preprocessing

Preprocessing was performed using Statistical Parametric Mapping software (SPM8, http://www.fil.ion.ucl.ac.uk/spm). The first four volumes of each run were discarded to allow for T1 equilibrium effects. The remaining 131 volumes were corrected for interslice timing differences caused by slice acquisition order, and then were realigned to the first volume. Subjects were excluded if head motion exhibited translation of more than 2 mm or rotation of more than 2° over the course of the four runs. All subjects met these requirements. Subsequently, individual T1 structural images were co-registered to the mean of the realigned functional images. The obtained T1 images were segmented into gray matter, white matter and cerebrospinal fluid. The functional images and the segmented T1 images were then spatially normalized to MNI space and resampled with a voxel size of 2 × 2 × 2 mm^3. After normalization, functional images were spatially smoothed using a Gaussian kernel with 6 mm full width at half maximum to decrease spatial noise. The time series from each voxel was high-pass filtered (1/131-Hz cutoff) to remove low-frequency noise and signal drift.

fMRI model fitting

For each subject and each voxel, fixed-effects model analyses were performed on the preprocessed fMRI data using GLM. Go and StopInhibit (Successful Stopping) events were modeled by convolving with a canonical hemodynamic response function (HRF). Events were modeled at the onset time of the arrow stimulus. Twelve motion-related parameters (six motion parameters and its derivative) were included as covariates of no interest to improve statistical sensitivity. The baseline was the null events which were consisted of the interval between every trail when the screen was blank. The null events were not modeled and therefore constitute an implicit baseline. StopInhibit trials included an already initiated Go process with a subsequent Stop process. Therefore, to isolate the neural correlates specific to successful stopping, StopInhibit-Go contrast images were computed for each subject and each session. In this study, we only considered the StopInhibit-Go contrast because it indexed successful response inhibition.

Statistical analysis

For behavioral analysis, behavioral measures were compared between RW and SD sessions using paired *t*-test. For fMRI data analysis, the first level analyses were conducted and statistical parametric maps for the *t* statistics (spmT) were generated. At the second level, the random-effects model analysis was performed based on the spmT images from the first level analysis. Age and gender were included as nuisance covariates. To explore the BOLD response evoked by SST, the group results of the one sample *t*-test were mapped for both RW and SD sessions. Clusters with at least 10 voxels and $P < 0.001$ (corrected for false discovery rate, FDR, $t > 4.67$ for RW, $t > 5.25$ for SD) were considered to indicate significant activation in the group analysis. Before exploring the

differences of BOLD responses between RW and SD, we restricted the analysis to a priori regions of significant activation using a mask from the spmT of RW. Significant differences in activation were then explored using paired *t*-test with a significance level of $P < 0.05$ (FDR corrected, $|t| > 2.34$), and a minimum cluster size of 10 voxels.

To explore the relationships between cerebral activation and SSRT in both the RW and SD conditions, the significant regions of each state were restricted using a mask from the spmT image of this state. The fMRI model was refitted with one added condition of SSRT as a regressor for the StopInhibit-Go contrast. Age and gender were also included as nuisance covariates. This analysis explored the correlation between the brain inhibition-related activity and SSRT. Significant relationships were explored at $P < 0.05$ (FDR corrected, $|t| > 2.75$). Regions' activation that was correlated with SSRT was extracted and averaged. To determine how the activation of these regions contributed to SSRT, the relative weight method was conducted for both RW and SD conditions, to measure the relative importance of these regions (Johnson 2004; Johnson and Lebreton 2004; Lebreton and Tonidandel 2008). The relative importance refers to the proportional contribution of each independent variable to the coefficient of determination, considering both the unique contribution of each independent variable by itself and its incremental contribution when combined with the other independent variables in the regression equation.

Furthermore, to statistically assess the changes in the correlations between SSRT and cerebral activation after SD compared with RW, univariate analysis was performed to examine whether SSRT showed a linear interaction effect using GLM (Eq. (1)) including main effects of State (RW or SD) and cerebral activation (Activation), and the interaction term State × Activation (Huang et al. 2014; Suh et al. 2016).

$$\begin{bmatrix} SSRT_{1,1} \\ SSRT_{1,2} \\ \vdots \\ SSRT_{1,20} \\ SSRT_{2,1} \\ SSRT_{2,2} \\ \vdots \\ SSRT_{2,20} \end{bmatrix} = \beta_0 + \beta_1 \times \begin{bmatrix} Activation_{1,1} \\ Activation_{1,2} \\ \vdots \\ Activation_{1,20} \\ Activation_{2,1} \\ Activation_{2,2} \\ \vdots \\ Activation_{2,20} \end{bmatrix} + \beta_2 \times \begin{bmatrix} 1 \\ 1 \\ \vdots \\ 1 \\ 0 \\ 0 \\ \vdots \\ 0 \end{bmatrix} + \beta_3 \times \begin{bmatrix} Activation_{1,1} \\ Activation_{1,2} \\ \vdots \\ Activation_{1,20} \\ 0 \\ 0 \\ \vdots \\ 0 \end{bmatrix} \quad (1)$$

Here, $SSRT_{1,1}$, $SSRT_{1,2}$, …, $SSRT_{1,20}$ were the 20 subjects' SSRTs in RW state. $SSRT_{2,1}$, $SSRT_{2,2}$, …, $SSRT_{2,20}$ were the 20 subjects' SSRTs in SD state. $Activation_{1,1}$, $Activation_{1,2}$, …, $Activation_{1,20}$ were the 20 subjects' activation of a brain region in RW state. $Activation_{2,1}$, $Activation_{2,2}$, …, $Activation_{2,20}$ were the 20 subjects' activation of a brain region in SD state. For the State vector $\begin{bmatrix} 1 \\ 1 \\ \vdots \\ 1 \\ 0 \\ 0 \\ \vdots \\ 0 \end{bmatrix}$, 1 represented the RW state and 0 represented the SD state. The vector $\begin{bmatrix} Activation_{1,1} \\ Activation_{1,2} \\ \vdots \\ Activation_{1,20} \\ 0 \\ 0 \\ \vdots \\ 0 \end{bmatrix}$ was the State × Activation.

Brain regions were identified using the Automated Anatomical Labeling template. All activations are reported in MNI coordinates. Brain activations were visualized using MRIcron (www.sph.sc.edu/comd/rorden/mricron/) with the statistical parametric map overlaid on a standard MRI template. Because the subthalamic nucleus (STN) is a relatively small structure and whole-brain fMRI has limited spatial resolution, it is difficult to localize activation in this region. We defined the STN as a box of size 10 × 10 × 10 mm, centered at MNI coordinates x = −10/+10, y = −15, z = −5, following the method described by Aron and Poldrack (Aron and Poldrack 2006). This definition provides full STN coverage according to an atlas of subcortical structures (Lucerna et al. 2002). We then overlaid the spmT with this STN mask to identify activation in this region.

Results

Behavioral results

For the SST, slower GoRT, more GoMiss and lower GoAcc were found in Go trials after SD (Table 1), whereas there were no differences in GoError between RW and SD sessions. In Stop trials, SD significantly increased StopRespond RT, while

the StopAcc did not differ between SD and RW conditions. Furthermore, subjects exhibited significantly poorer performance on SSRT when they were sleep-deprived (259.27 ± 29.46 ms after RW versus 284.11 ± 30.93 ms after SD, $P < 0.01$).

fMRI results

Inhibition-related activation

Our primary contrast of interest was StopInhibit-Go, which isolates the neural correlates specific to the Stop process. Table 2 and Fig. 1a show that the right hemisphere contained more activated voxels than the left hemisphere, and exhibited stronger activation. These results are consistent with previous studies, demonstrating right-hemisphere dominance in activation for StopInhibit-Go. Activation was seen in bilateral IFG, supplementary motor area (SMA), insula, cingulate cortex (anterior cingulate cortex [ACC], middle cingulate cortex [MCC]) and parietal cortex (superior parietal lobule, inferior parietal lobule), as well as subcortical regions including the caudate nucleus, putamen, thalamus and STN. This pattern of activation replicates the findings previously reported in studies using auditory stop-signal tasks (Aron and Poldrack 2006; Aron et al. 2007; C. D. Chambers et al. 2009; Wager et al. 2005). We also found significant activation in the bilateral frontal cortex (middle frontal gyrus [MFG], superior frontal gyrus), hippocampus, parahippocampal gyrus, calcarine sulcus, lingual gyrus, occipital cortex, fusiform gyrus, supramarginal gyrus and temporal cortex (superior, middle, inferior) and in right precentral gyrus and angular gyrus.

Table 1 Behavioral data for RW and SD

Behavioral measure	RW	SD
GoRT(ms) ****	433.26 ± 46.38	465.52 ± 55.65
GoAcc(%) **	97.33 ± 2.29	93.19 ± 6.64
GoError(%)	2.43 ± 2.08	2.85 ± 2.58
GoMiss(%) **	0.24 ± 0.94	3.96 ± 5.37
StopAcc(%)	38.13 ± 6.56	37.19 ± 9.45
StopRespond RT(ms) ***	395.11 ± 47.34	427.85 ± 50.41
SSRT(ms) **	259.27 ± 29.46	284.11 ± 30.93

Data are presented as mean ± standard deviation. GoRT, the RT on correct Go trials. GoAcc, the accuracy of the Go task. GoError, the percentage of incorrect Go trials, in which subjects made an incorrect response on a Go trial (e.g., pressing the left button in response to a rightward pointing arrow and vice versa. GoMiss, the percentage of missing Go responses, in which subjects failed to make a response on a Go trial. StopAcc, the accuracy of the Stop task. StopRespond RT, the RT on unsuccessful Stop trials. SSRT, stop-signal reaction time. RW, rested wakefulness. SD, sleep deprivation

****Significant difference between RW and SD at $P < 0.0001$ using paired t test

***Significant difference between RW and SD at $P < 0.001$ using paired t test

**Significant difference between RW and SD at $P < 0.01$ using paired t test

After 24 h of SD, significant activation was still found in regions that are critical for response inhibition: the IFG, SMA, insula, cingulate cortex and inferior parietal lobule (Table 3, Fig. 1b). The activated regions after SD were also significantly activated after RW. However, we did not find significant activation in the superior parietal lobule, caudate nucleus, putamen, thalamus or STN after SD. No new regions of activation were found after SD. All the results are significant at threshold $P < 0.001$ with FDR correction ($t > 4.67$ for RW, $t > 5.25$ for SD).

Compared with the RW session, the brain regions involved in response inhibition showed significantly less activation after SD, when FDR correction ($P < 0.05$, $|t| > 2.34$) was applied: in bilateral IFG, insula, cingulate cortex, parietal cortex, thalamus, STN, right SMA and temporal cortex (left superior temporal gyrus, right middle temporal gyrus, bilateral inferior temporal gyrus). In addition, bilateral MFG, lingual gyrus, occipital cortex, fusiform gyrus, supramarginal gyrus and right angular gyrus also showed decreased activation in the SD session compared with the RW session (Table 4, Fig. 1c). No regions exhibited significantly increased activation following SD compared with RW.

Correlations between SSRT and cerebral activation

To explore where in the brain StopInhibit-Go activation increased with faster SSRT, we performed Pearson correlation analysis between cerebral activation and SSRT, in both the RW and SD conditions. Across all subjects, SSRT was significantly negatively correlated with activation in the left IFG ($r = -0.648$, $p = 0.002$), right hippocampus ($r = -0.549$, $p = 0.014$), right lingual gyrus ($r = -0.562$, $p = 0.01$), bilateral fusiform gyrus ($r = -0.617$, $p = 0.004$ for left fusiform gyrus; $r = -0.524$, $p = 0.018$ for right fusiform gyrus) and left STN ($r = -0.573$, $p = 0.008$) in the RW condition. The relative importance of these regions is summarized in Fig. 2a: the relative weights of these regions were 24.81%, 10.51%, 21.09%, 10.78%, 16.18% and 16.63%, respectively.

After SD, SSRT was negatively correlated with activation in the right MFG ($r = -0.588$, $p = 0.006$), right IFG ($r = -0.716$, $p = 0.0004$) and left lingual gyrus ($r = -0.538$, $p = 0.015$). The relative weights of these regions were 25.62%, 48.84% and 25.54%, respectively (Fig. 2b). There were no positive correlations between neural activation and SSRT after either RW or SD. The results indicated that the regions in which activation was correlated with SSRT were different between RW and SD states. These results suggest that SD altered the correlation between cerebral activation and SSRT.

To further explore the changes in correlations between cerebral activation and SSRT in a statistical sense, we examined

Table 2 Regions of significant activation for StopInhibit-Go after rested wakefulness

Regions	Hemisphere	Max T	X	Y	Z	Voxels
Precentral gyrus	R	9.93	48	8	28	289
Superior frontal gyrus	L	7.18	−8	30	32	65
	R	7.31	18	12	68	260
Middle frontal gyrus	L	7.99	−46	34	26	556
	R	12.76	40	48	32	1577
Inferior frontal gyrus	L	15.75	52	20	2	805
	R	11.58	−32	20	−14	1830
Supplementary motor area	L	6.70	0	18	48	75
	R	7.08	16	12	70	366
Insula	L	16.39	−34	18	−16	624
	R	10.59	30	22	−14	594
Anterior cingulate cortex	L	11.75	−6	32	22	663
	R	11.91	8	34	24	591
Middle cingulate cortex	L	11.08	0	24	36	165
	R	10.58	2	26	34	326
Hippocampus	L	9.84	−20	−30	−10	111
	R	15.11	24	−26	−10	166
ParaHippocampal gyrus	L	8.89	−20	−32	−12	46
	R	11.39	22	−34	−12	114
Calcarine sulcus	L	10.14	−22	−98	−4	79
	R	9.48	22	−98	−4	124
Lingual gyrus	L	13.37	−36	−82	−16	157
	R	17.70	28	−86	−8	335
Occipital cortex	L	18.51	−36	−82	−12	1444
	R	23.64	32	−88	−4	1452
Fusiform gyrus	L	16.44	−38	−80	−14	912
	R	18.35	28	−88	−6	820
Superior parietal lobule	L	7.21	−16	−72	44	220
	R	7.23	30	−72	50	64
Inferior parietal lobule	L	8.98	−50	−40	48	776
	R	9.83	50	−34	52	751
SupraMarginal gyrus	L	7.47	−58	−44	28	124
	R	9.29	54	−40	38	573
Angular gyrus	R	9.00	30	−66	50	475
Caudate nucleus	L	6.09	−14	14	0	35
	R	6.22	14	14	6	50
Putamen	L	5.84	−14	14	−2	29
	R	5.51	16	16	−8	13
Thalamus	L	7.75	−6	−8	−2	37
	R	7.98	12	−28	0	216
Superior temporal gyrus	L	9.35	−38	18	−18	102
	R	7.48	40	20	−16	70
Middle temporal gyrus	L	6.42	−44	−64	−2	69
	R	7.14	56	−44	0	439
Inferior temporal gyrus	L	9.02	−52	−60	−8	323
	R	8.44	50	−58	−14	510
Subthalamic nucleus	L	12.29	−8	−8	−6	130
	R	7.61	18	−28	−14	194

Results were set at $p < 0.001$, FDR corrected ($t > 4.67$)

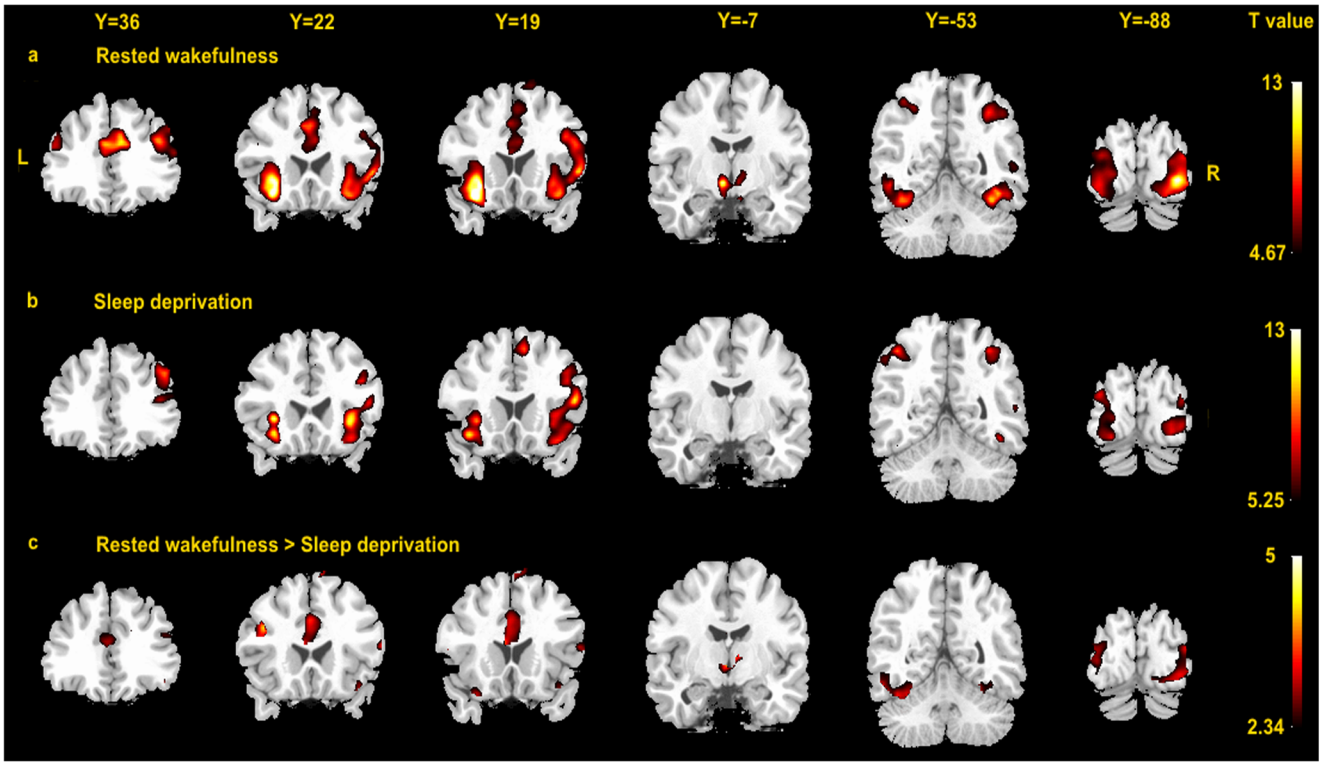

Fig. 1 Significant brain activation of StopInhibition-Go after rested wakefulness and sleep deprivation. **a** After rested wakefulness (RW). Map was thresholded at $P < 0.001$ (corrected for false discovery rate, FDR, $t > 4.67$). Significant activation was found in bilateral inferior frontal gyrus (IFG), supplementary motor area (SMA), insula, cingulate cortex, lingual gyrus, occipital cortex, fusiform gyrus and subcortical regions including the subthalamic nucleus (STN). **b** After 24 h of sleep deprivation (SD). Map was thresholded at $P < 0.001$ (FDR correction, $t > 5.25$). Significant activation was found in IFG, insula, SMA occipital cortex and fusiform gyrus, similar to RW. However, significant activation in STN was not found. No new activated regions were found after SD. **c** Differences in StopInhibition-Go activation between RW and SD conditions. Map was thresholded at $P < 0.05$ (FDR correction, $t > 2.34$). Significant reduced activation after SD was found in bilateral IFG, insula, cingulate cortex, lingual gyrus, occipital cortex, fusiform gyrus, STN and right SMA. There were no regions that had significantly more activation after SD compared with RW. The Y coordinate in MNI space is shown above each coronal slice. The color scale represents t value. R, Right. L, Left

the interaction effect between state and cerebral activation using GLM. The results revealed that the correlation between SSRT and cerebral activation in the RW condition was different from that in the SD condition in bilateral IFG ($t_{36} = 2.084$, $p = 0.044$ for left IFG; $t_{36} = -2.052$, $p = 0.047$ for right IFG), left STN ($t_{36} = 2.603$, $p = 0.013$) and left lingual gyrus ($t_{36} = -2.13$, $p = 0.04$, Fig. 3). After SD, subjects exhibited stronger negative correlations between SSRT and activation in right IFG and left lingual gyrus, while these associations were not present in the RW condition. However, subjects in the RW condition showed stronger negative correlations between SSRT and activation in left IFG and left STN, while subjects after SD did not show these relationships. These results suggest that SD is associated with the altered correlations between cerebral activation and SSRT, especially in bilateral IFG, left STN, and left lingual gyrus.

Discussion

In the present study, we examined the effects of SD on the pattern of neural activation associated with response inhibition in a sample of 20 young healthy subjects. Response inhibition was measured using the SST, a task requiring participants to inhibit an already-initiated response. As expected, sleep-deprived subjects showed poorer inhibitory control. In accord with our hypothesis, the IFG was less activated following SD compared with RW. Decreased activation in STN, SMA, ACC, insula, parietal cortex, temporal cortex and visual-related regions (lingual gyrus, fusiform gyrus and occipital cortex) was also found after SD. No regions showed significantly increased activation after SD. After RW, we found significant negative correlations between SSRT and cerebral activation in left IFG, right hippocampus, right lingual gyrus, left STN and bilateral fusiform gyrus. Left IFG activation contributed the most to SSRT. After SD, significant negative correlations were found between SSRT and activation in right MFG, right IFG and left lingual gyrus. Right IFG activation contributed the most to SSRT. Further interaction analysis indicated that the correlation between SSRT and the activation of bilateral IFG, left lingual gyrus and left STN was state-dependent. To our knowledge, this is the first evidence of altered activation of IFG after SD using the SST.

Table 3 Regions of significant activation for StopInhibit-Go after sleep deprivation

Regions	Hemisphere	Max T	X	Y	Z	Voxels
Precentral gyrus	R	9.56	46	10	26	261
Superior frontal gyrus	L	6.52	−18	8	68	17
	R	6.45	18	14	70	51
Middle frontal gyrus	L	7.95	−46	42	18	845
	R	11.28	42	26	28	159
Inferior frontal gyrus	L	11.27	−30	22	−12	168
	R	12.26	32	26	−4	1039
Supplementary motor area	L	6.68	−2	18	52	19
	R	9.79	8	16	52	220
Insula	L	11.92	−32	22	0	296
	R	13.47	34	24	−2	404
Anterior cingulate cortex	L	6.33	−4	30	34	24
	R	7.33	6	28	34	29
Middle cingulate cortex	L	6.26	2	24	40	24
	R	7.19	6	28	36	91
Hippocampus	L	6.49	−18	−32	−8	15
	R	8.35	18	−30	−8	51
ParaHippocampal gyrus	R	7.27	18	−28	−10	13
Calcarine sulcus	L	7.28	−22	−98	−6	22
	R	7.16	20	−94	−6	40
Lingual gyrus	L	10.89	−30	−84	−14	73
	R	8.14	26	−88	−10	98
Occipital cortex	L	9.44	−28	−84	−12	595
	R	8.80	36	−76	−10	538
Fusiform gyrus	L	8.04	−26	−84	−12	153
	R	9.15	34	−76	−12	176
Inferior parietal lobule	L	9.30	−42	−54	50	336
	R	10.56	42	−46	40	477
SupraMarginal gyrus	L	7.47	−62	−44	34	114
	R	7.54	46	−42	42	238
Angular gyrus	R	8.83	30	−68	48	276
Superior temporal gyrus	R	7.14	40	18	−16	19
Middle temporal gyrus	R	6.40	56	−54	8	20
Inferior temporal gyrus	R	6.23	46	−60	−6	29

Results were set at $p < 0.001$, FDR corrected ($t > 5.25$)

Effects of sleep deprivation on inhibition-related activation

Confirming our hypothesis, after 24 h of SD, subjects exhibited significantly reduced activation in bilateral IFG. This finding is in accord with Horne's hypothesis that the PFC is vulnerable to SD (Horne 1993). In addition to the reduced activation in IFG following SD, we observed decreased activation in SMA, STN, ACC and insula, which are the core regions of the response inhibition system (Aron and Poldrack 2006; Li et al. 2006; Swick et al. 2011). These results indicate that SD is associated with deficits of activation in the "stopping network".

Moreover, we found significantly reduced activation in vision-related regions includingthe occipital cortex, lingual gyrus and fusiform gyrus following SD. The lingual gyrus and fusiform gyrus play a role in visual memory (Bogousslavsky et al. 1987), face perception (Kanwisher et al. 1997) and color vision processing. During SST in the current study, participants were instructed to make simple decisions about a visual stimulus. On Stop trials, a Stop signal (the white arrow changed to blue) was presented after the visual stimulus, indicating that the response should be withheld. In a study by Bell-McGinty et al., the researchers found that activation in right fusiform gyrus and left lingual gyrus was decreased after SD during non-verbal recognition memory (Bell-McGinty et al. 2004). Kong et al.

Table 4 Regions exhibiting activity differences for StopInhibit-Go between RW and SD

Regions	Hemisphere	Max T	X	Y	Z	Voxels
Middle frontal gyrus	L	3.65	−44	26	32	74
	R	2.74	24	48	42	38
Inferior frontal gyrus	L	4.26	−40	22	26	238
	R	3.42	42	32	−14	145
Supplementary motor area	L	3.09	−10	10	70	10
	R	4.23	6	10	72	71
Insula	L	3.22	−36	28	2	59
	R	3.19	38	6	4	54
Anterior cingulate cortex	L	3.54	−4	16	24	396
	R	3.54	2	26	24	154
Middle cingulate cortex	L	3.25	−2	16	34	19
	R	3.19	0	18	34	45
Lingual gyrus	L	4.76	−26	−76	−14	35
	R	4.38	32	−82	−18	170
Occipital cortex	L	4.37	−54	−62	−12	482
	R	5.71	44	−78	−6	662
Fusiform gyrus	L	6.76	−40	−60	−22	790
	R	5.11	28	−50	−18	675
Superior parietal lobule	L	4.91	−26	−68	54	145
	R	3.56	30	−64	56	22
Inferior parietal lobule	L	4.65	−26	−72	48	137
	R	3.37	46	−34	52	70
SupraMarginal gyrus	L	3.57	−54	−46	28	41
	R	3.32	44	−38	42	19
Angular gyrus	R	3.77	30	−50	40	152
Thalamus	L	4.21	−6	−22	0	23
	R	4.73	6	−22	2	141
Superior temporal gyrus	L	4.11	−54	−44	24	37
Middle temporal gyrus	R	4.35	58	−58	0	177
Inferior temporal gyrus	L	5.42	−56	−60	−12	192
	R	5.00	58	−58	−2	250
Subthalamic nucleus	L	4.21	−4	−26	−16	54
	R	4.08	4	−26	−16	26

Results were set at $p < 0.05$, FDR corrected ($t > 2.34$). RW, rested wakefulness. SD, sleep deprivation

(Kong et al. 2011) reported that SD reduced visual processing capacity and fusiform face area activation. These findings are in accord with the reduced activation of lingual gyrus and fusiform gyrus after SD revealed in the current study. Furthermore, these findings suggest that SD may impair the color vision processing capability. Further studies are required to verify the effects of SD on color vision processing.

Two previous studies had investigated the effects of SD on inhibitory control using fMRI (Chuah et al. 2006; Almklov et al. 2015). They assessed the response inhibition using the Go/No-Go task. Chauh et al. found reduced activation in bilateral ventral and anterior PFC and right anterior insula after SD (Chuah et al. 2006). However, our present study assessed the response inhibition using the SST, another task commonly used for measuring inhibition control. On one hand, we found that bilateral MFG, IFG and right insula showed significantly less activation after SD, confirmed the results of Chuah et al. On the other hand, we also found decreased activation in thalamus, SMA, parietal cortex, temporal cortex and visual-related regions, which was not reported by Chuah et al. These results suggest that different effects of SD on inhibition control were obtained by using different inhibition tasks. This discrepancy may be due to the distinct neural circuits of these two tasks (Swick et al. 2011), or may result from the individual difference that these two studies were two independent samples. Further studies are required to test these hypotheses using the self-control experiment. Almklov et al. found that the deactivation in left posterior cingulate was reduced after

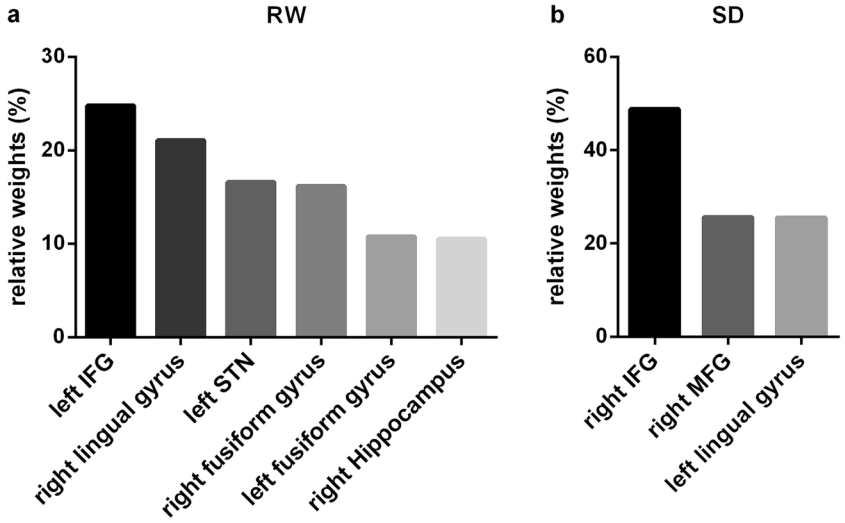

Fig. 2 The relative weights of regions were summarized to determine how these regions' activation contributed to SSRT. After RW, the activation of left IFG, right lingual gyrus, left STN, bilateral fusiform gyrus and right hippocampus was associated with SSRT. The relative weights of these regions were showed in (**a**). After SD, the activation of right IFG, right MFG and left lingual gyrus was associated with SSRT. The relative weights of these regions were showed in (**b**). IFG, inferior frontal gyrus. STN, subthalamic nucleus. MFG, middle frontal gyrus. RW, rested wakefulness. SD, sleep deprivation

SD in young normal subjects during response inhibition trial (Almklov et al. 2015). However, we did not analyze deactivation in this study. In future work, we will include analysis of deactivation to further explore the underlying mechanisms of cognitive deficits after SD.

Correlations between SSRT and cerebral activation

After RW, faster SSRT was correlated with more activation in the left IFG, right hippocampus, right lingual gyrus, bilateral fusiform gyrus and left STN. These results suggest that stronger recruitment of these regions may be required for better inhibitory control. Negative correlations between right IFG activation and SSRT have been reported in many studies (Aron and Poldrack 2006; Congdon et al. 2010; Galván et al. 2011; White et al. 2014). In addition, previous studies have reported that right STN activation is negatively correlated with SSRT (Aron and Poldrack 2006; Cohen et al. 2010). However, a study by Ghahremani et al. reported that left IFG activation is greater for subjects with faster SSRT (Ghahremani et al. 2012). Few studies have reported negative correlations between SSRT and activation of the hippocampus, lingual gyrus and fusiform gyrus during response inhibition. These discrepancies may be caused by substantial individual differences in response inhibition. Cohen et al. (Cohen et al. 2010) reported that adult subjects (ages 25–30 years) exhibited faster SSRT than younger subjects (ages 9–19 years) and that age was negatively correlated with neural activity in left medial prefrontal cortex during successful response inhibition. Lee and Hsieh found that SSRT did not change significantly with age, among subjects 40–77 years of age (Lee and Hsieh 2017). These findings suggest that SSRT and SST-related activation might change across the lifespan. Therefore, age differences of subjects (Rubia et al. 2006; Bunge et al. 2002) may have contributed to the discrepancies described above. Furthermore, as in the current study, several previous studies explored the relationship between SSRT and cerebral activation using voxelwise analyses, while Congdon et al. (Congdon et al. 2010) utilized independent components analysis. Methodological differences may have also contributed to discrepancies among previous findings. Further studies are required to examine the relationship between activation and behavioral performance during response inhibition in more detail, with larger sample sizes and age-matched subjects using different methods.

In the SD condition, reduced brain activation in the right MFG, right IFG and left lingual gyrus was related to slower SSRT. These results suggest that less engagement of these regions may result in poorer inhibition performance for sleep-deprived individuals. The regions in which activation was correlated with SSRT differed in the SD and RW conditions. This finding indicated that SD may change the relationship between behavioral performance and cerebral activation. To statistically explore the effects of SD on the correlation between SSRT and cerebral activation, we used GLM to examine whether SSRT exhibited a linear interaction effect of state with cerebral activation. The results indicated that SD enhanced the correlation between SSRT and cerebral activation in right IFG and left lingual gyrus, and weakened the correlation in left IFG and left STN. These findings suggest that SD has a substantial effect on the correlation between SSRT and cerebral activation, especially in bilateral IFG, left STN and left lingual gyrus.

The results also revealed the relative importance of the regions in which activation was correlated with SSRT,

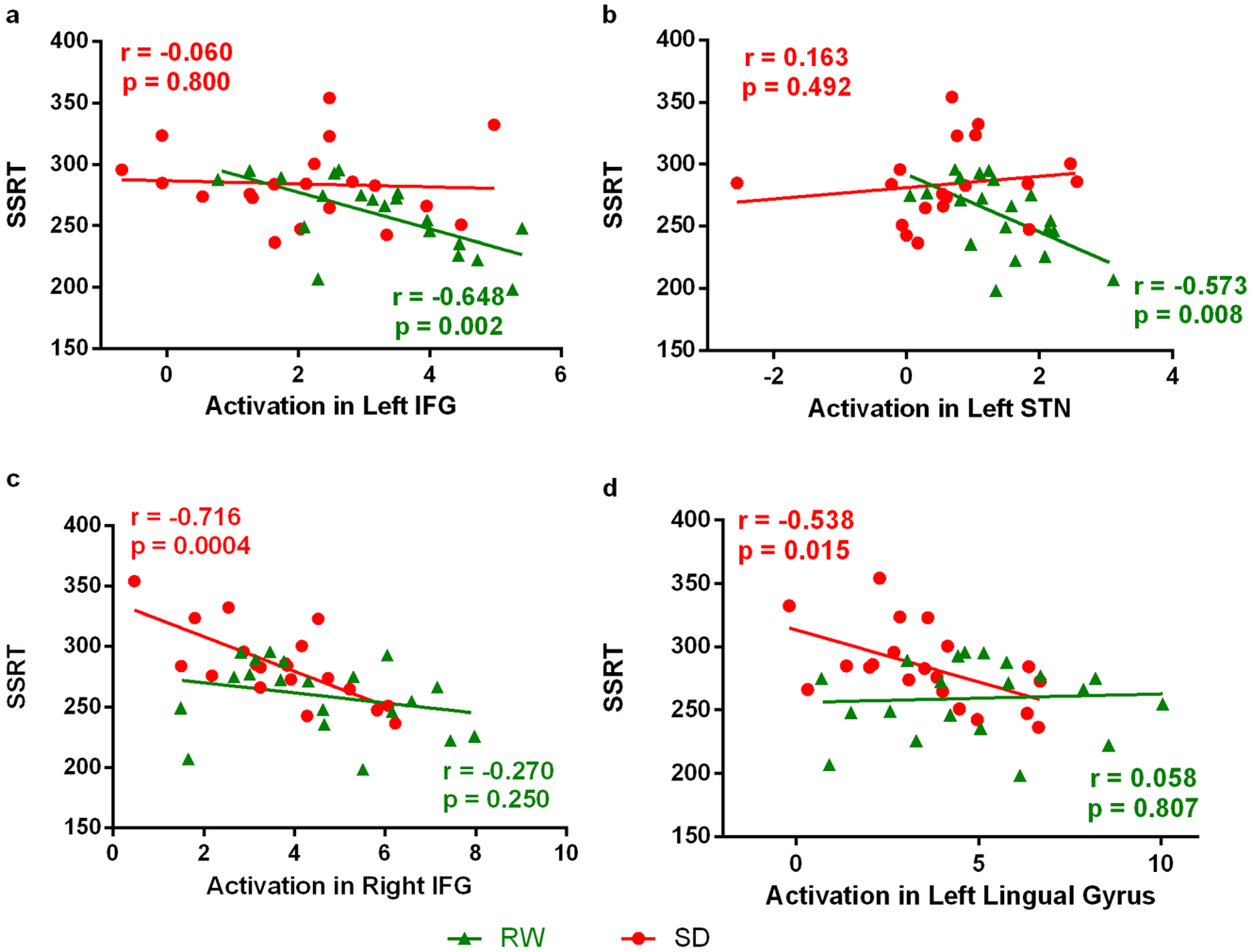

Fig. 3 Significant linear interaction of cerebral activation by state on SSRT. Significant interaction effects of state (RW or SD) with the activation of left IFG (**a**) and left STN (**b**) in SSRT were found, where the increasing activation in these regions was associated with faster SSRT after RW, while these associations were not present in the SD condition. Significant interaction effects were also found in right IFG (**c**) and left lingual gyrus (**d**), where the decreasing activation in these regions was associated with slower SSRT after SD, while these associations were not present in the RW condition. IFG, inferior frontal gyrus. STN, subthalamic nucleus. RW, rested wakefulness. SD, sleep deprivation

showing that the IFG had contributed the most to SSRT in both the RW and SD conditions. Left IFG activation contributed the most to SSRT in the RW condition, while right IFG activation contributed the most to SSRT in the SD condition. These results indicate that IFG activation contributed the most to SSRT, independent of state (RW or SD). These findings are consistent with the conventional view that IFG is a critical node for inhibition. To our knowledge, few studies have explored the relative weights of each region in which activation is correlated with behavioral performance, to measure the contribution of region's activation to behavioral performance.

Limitations

The present study involved several limitations that should be considered. First, the sample size was relatively small. Thus, the findings should be replicated in a larger sample in future studies. Second, we did not analyze deactivation in this study. Deactivation refers to a reduction of the BOLD signal during task performance relative to baseline (Gusnard et al. 2001). In future work, we will also include analysis of deactivation to further explore the underlying mechanisms of cognitive deficits after SD. Third, we did not use electroencephalographic measures, which could have confirmed that subjects did not fall asleep during the total sleep deprivation condition. However, a research assistant remained by the side of each subject to ensure they stayed awake and kept their eyes open.

Conclusions

In accord with previous behavioral and functional imaging studies reporting that SD in humans causes performance

decrements, the current finding revealed that SD caused significant declines in performance while subjects performed the SST in the scanner. Deficits in inhibition-related neural activation were found in the "stopping network" and vision-related regions after SD. The correlation we observed between SSRT and cerebral activation in bilateral IFG, left STN and left lingual gyrus was state-dependent. However, the activation of IFG contributed the most to SSRT, independent of state, supporting the conventional view that IFG is a critical node for inhibition. Our findings may provide insight into the neural substrate of the neurofunctional changes in response inhibition in individuals who are sleep deprived. Further studies with larger samples are required to confirm the current findings and further elucidate the neurobiological mechanisms involved.

Acknowledgements We thank the subjects whose participation enabled this work. This study was funded by National Basic Research Program of China under Grant Nos. 2015CB856403 and 2014CB543203; the National Natural Science Foundation of China under Grant Nos. 81471811, 81471738 and 61401346; and the Fundamental Research Funds for the Central Universities.

Funding This study was funded by National Basic Research Program of China under Grant Nos. 2015CB856403 and 2014CB543203; the National Natural Science Foundation of China under Grant Nos. 81471811, 81471738 and 61401346; and the Fundamental Research Funds for the Central Universities.

Compliance with ethical standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research ethics committee of the Xijing Hospital of the Fourth Military Medical University and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

Disclosures This was not an industry supportd study. The authors have indicated no financial conflicts of interest.

References

Almklov, E. L., Drummond, S. P., Orff, H., & Alhassoon, O. M. (2015). The effects of sleep deprivation on brain functioning in older adults. *Behavioral Sleep Medicine, 13*(4), 324–345. https://doi.org/10.1080/15402002.2014.905474.

Aron, A. R., & Poldrack, R. A. (2006). Cortical and subcortical contributions to Stop signal response inhibition: role of the subthalamic nucleus. *The Journal of Neuroscience, 26*(9), 2424–2433. https://doi.org/10.1523/JNEUROSCI.4682-05.2006.

Aron, A. R., Fletcher, P. C., Bullmore, E. T., Sahakian, B. J., & Robbins, T. W. (2003). Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nature Neuroscience, 6*(2), 115–116. https://doi.org/10.1038/nn1003.

Aron, A. R., Behrens, T. E., Smith, S., Frank, M. J., & Poldrack, R. A. (2007). Triangulating a cognitive control network using diffusion-weighted magnetic resonance imaging (MRI) and functional MRI. *Journal of Neuroscience: The Official Journal of the Society for Neuroscience, 27*(14), 3743–3752.

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2014a). Inhibition and the right inferior frontal cortex. *Trends in Cognitive Sciences, 8*(4), 170–177.

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2014b). Inhibition and the right inferior frontal cortex: one decade on. *Trends in Cognitive Sciences, 18*(4), 177–185. https://doi.org/10.1016/j.tics.2013.12.003.

Asplund, C. L., & Chee, M. W. (2013). Time-on-task and sleep deprivation effects are evidenced in overlapping brain areas. *NeuroImage, 82*(2), 326–335.

Band, G. P., van der Molen, M. W., & Logan, G. D. (2003). Horse-race model simulations of the stop-signal procedure. *Acta Psychologica, 112*(2), 105–142.

Basner, M., Rao, H., Goel, N., & Dinges, D. F. (2013). Sleep deprivation and neurobehavioral dynamics. *Current Opinion in Neurobiology, 23*(5), 854–863.

Bell-McGinty, S., Habeck, C., Hilton, H. J., Rakitin, B., Scarmeas, N., Zarahn, E., et al. (2004). Identification and differential vulnerability of a neural network in sleep deprivation. *Cerebral Cortex, 14*(5), 496–502. https://doi.org/10.1093/cercor/bhh011.

Bogousslavsky, J., Miklossy, J., Deruaz, J. P., Assal, G., & Regli, F. (1987). Lingual and fusiform gyri in visual processing: a clinico-pathologic study of superior altitudinal hemianopia. *Journal of Neurology, Neurosurgery, and Psychiatry, 50*(5), 607–614.

Bunge, S. A., Dudukovic, N. M., Thomason, M. E., Vaidya, C. J., & Gabrieli, J. D. (2002). Immature frontal lobe contributions to cognitive control in children: evidence from fMRI. *Neuron, 33*(2), 301–311.

Cai, W., Cannistraci, C. J., Gore, J. C., & Leung, H. C. (2014). Sensorimotor-independent prefrontal activity during response inhibition. *Human Brain Mapping, 35*(5), 2119–2136.

Chambers, C. D., Bellgrove, M. A., Stokes, M. G., Henderson, T. R., Garavan, H., Robertson, I. H., et al. (2006). Executive "Brake Failure" following Deactivation of Human Frontal Lobe. *Journal of Cognitive Neuroscience, 18*(3), 444–455.

Chambers, C. D., Bellgrove, M. A., Gould, I. C., English, T., Garavan, H., Mcnaught, E., et al. (2007). Dissociable mechanisms of cognitive control in prefrontal and premotor cortex. *Journal of Neurophysiology, 98*(6), 3638.

Chambers, C. D., Garavan, H., & Bellgrove, M. A. (2009). Insights into the neural basis of response inhibition from cognitive and clinical neuroscience. *Neuroscience & Biobehavioral Reviews, 33*(5), 631.

Chee, M. W., & Choo, W. C. (2004). Functional imaging of working memory after 24 hr of total sleep deprivation. *The Journal of Neuroscience, 24*(19), 4560–4567. https://doi.org/10.1523/JNEUROSCI.0007-04.2004.

Chee, M. W., Chuah, L. Y., Venkatraman, V., Chan, W. Y., Philip, P., & Dinges, D. F. (2006). Functional imaging of working memory following normal sleep and after 24 and 35 h of sleep deprivation: Correlations of fronto-parietal activation with performance. *NeuroImage, 31*(1), 419–428. https://doi.org/10.1016/j.neuroimage.2005.12.001.

Chuah, Y. M., Venkatraman, V., Dinges, D. F., & Chee, M. W. (2006). The neural basis of interindividual variability in inhibitory efficiency after sleep deprivation. *Journal of Neuroscience, 26*(27), 7156–7162.

Cohen, J. R., Asarnow, R. F., Sabb, F. W., Bilder, R. M., Bookheimer, S. Y., Knowlton, B. J., et al. (2010). Decoding Developmental Differences and Individual Variability in Response Inhibition Through Predictive Analyses Across Individuals. *Frontiers in Human Neuroscience, 4*(5), 47.

Congdon, E., Mumford, J. A., Cohen, J. R., Galvan, A., Aron, A. R., Xue, G., et al. (2010). Engagement of large-scale networks is related to individual differences in inhibitory control. *NeuroImage, 53*(2), 653–663.

Drummond, S. P., Brown, G. G., Gillin, J. C., Stricker, J. L., Wong, E. C., & Buxton, R. B. (2000). Altered brain response to verbal learning following sleep deprivation. *Nature, 403*(6770), 655–657. https://doi.org/10.1038/35001068.

Durmer, J. S., & Dinges, D. F. (2005). Neurocognitive consequences of sleep deprivation. *Seminars in Neurology, 25*(1), 117–129.

Floden, D., & Stuss, D. T. (2006). Inhibitory control is slowed in patients with right superior medial frontal damage. *Cognitive Neuroscience Journal of, 18*(11), 1843–1849.

Galván, A., Poldrack, R. A., Baker, C. M., Mcglennen, K. M., & London, E. D. (2011). Neural Correlates of Response Inhibition and Cigarette Smoking in Late Adolescence. *Neuropsychopharmacology Official Publication of the American College of Neuropsychopharmacology, 36*(5), 970.

Ghahremani, D. G., Lee, B., Robertson, C. L., Tabibnia, G., Morgan, A. T., Shetler, N. D., et al. (2012). Striatal Dopamine D2/D3 Receptors Mediate Response Inhibition and Related Activity in Frontostriatal Neural Circuitry in Humans. *Journal of Neuroscience: The Official Journal of the Society for Neuroscience, 32*(21), 7316–7324.

Gusnard, D. A., Akbudak, E., Shulman, G. L., & Raichle, M. E. (2001). Medial prefrontal cortex and self-referential mental activity: relation to a default mode of brain function. *Proceedings of the National Academy of Sciences of the United States of America, 98*(7), 4259–4264. https://doi.org/10.1073/pnas.071043098.

Horne, J. A. (1993). Human sleep, sleep loss and behaviour. Implications for the prefrontal cortex and psychiatric disorder. *The British Journal of Psychiatry: the Journal of Mental Science, 162*(162), 413–419.

Horne, J. A., & Ostberg, O. (1976). A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. *International Journal of Chronobiology, 4*(2), 97–110.

Huang, C. C., Liu, M. E., Chou, K. H., Yang, A. C., Hung, C. C., Hong, C. J., et al. (2014). Effect of BDNF Val66Met polymorphism on regional white matter hyperintensities and cognitive function in elderly males without dementia. *Psychoneuroendocrinology, 39*(1), 94.

Johnson, J. W. (2004). Factors Affecting Relative Weights: The Influence of Sampling and Measurement Error. *Organizational Research Methods, 7*(3), 283–299.

Johnson, J. W., & Lebreton, J. M. (2004). History and Use of Relative Importance Indices in Organizational Research. *Organizational Research Methods, 7*(3), 238–257.

Kanwisher, N., Mcdermott, J., & Chun, M. M. (1997). The fusiform face area: a module in human extrastriate cortex specialized for face perception. *Journal of Neuroscience: The Official Journal of the Society for Neuroscience, 17*(11), 4302–4311.

Killgore, W. D. S. (2010). Effects of sleep deprivation on cognition. *Progress in Brain Research, 185*(1), 105–129.

Kong, D., Soon, C. S., & Chee, M. W. (2011). Reduced visual processing capacity in sleep deprived persons. *NeuroImage, 55*(2), 629–634.

Lebreton, J. M., & Tonidandel, S. (2008). Multivariate relative importance: Extending relative weight analysis to multivariate criterion spaces. *Journal of Applied Psychology, 93*(2), 329–345.

Lee, H. H., & Hsieh, S. (2017). Resting-State fMRI Associated with Stop-Signal Task Performance in Healthy Middle-Aged and Elderly People. *Frontiers in Psychology*, 8.

Li, C. S., Huang, C., Constable, R. T., & Sinha, R. (2006). Imaging response inhibition in a stop-signal task: neural correlates independent of signal monitoring and post-response processing. *Journal of Neuroscience: The Official Journal of the Society for Neuroscience, 26*(1), 186–192.

Lim, J., Choo, W. C., & Chee, M. W. (2007). Reproducibility of changes in behaviour and fMRI activation associated with sleep deprivation in a working memory task. *Sleep, 30*(1), 61–70.

Logan, G. D. (1994). On the ability to inhibit thought and action: A users' guide to the stop signal paradigm. *Inhibitory Processes in Attention Memory & Language*, 189–239.

Lucerna, S., Salpietro, F. M., Alafaci, C., & Tomasello, F. (2002). *In Vivo Atlas of Deep Brain Structures*: Springer Berlin Heidelberg.

Ma, N., Dinges, D. F., Basner, M., & Rao, H. (2014). How Acute Total Sleep Loss Affects the Attending Brain: A Meta-analysis of Neuroimaging Studies. *Sleep, 38*(2), 233.

Mu, Q., Mishory, A., Johnson, K. A., Nahas, Z., Kozel, F. A., Yamanaka, K., et al. (2005a). Decreased brain activation during a working memory task at rested baseline is associated with vulnerability to sleep deprivation. *Sleep, 28*(4), 433.

Mu, Q., Nahas, Z., Johnson, K. A., Yamanaka, K., Mishory, A., Koola, J., et al. (2005b). Decreased cortical response to verbal working memory following sleep deprivation. *Sleep, 28*(1), 55–67.

Picton, T. W., Stuss, D. T., Alexander, M. P., Shallice, T., Binns, M. A., & Gillingham, S. (2007). Effects of focal frontal lesions on response inhibition. *Cerebral Cortex, 17*(4), 826.

Rubia, K., Smith, A. B., Woolley, J., Nosarti, C., Heyman, I., Taylor, E., et al. (2006). Progressive increase of frontostriatal brain activation from childhood to adulthood during event-related tasks of cognitive control. *Human Brain Mapping, 27*(12), 973–993. https://doi.org/10.1002/hbm.20237.

Suh, S., Kim, H., Dang-Vu, T. T., Joo, E., & Shin, C. (2016). Cortical Thinning and Altered Cortico-Cortical Structural Covariance of the Default Mode Network in Patients with Persistent Insomnia Symptoms. *Sleep, 39*(1), 161–171. https://doi.org/10.5665/sleep.5340.

Swick, D., Ashley, V., & Turken, U. (2011). Are the neural correlates of stopping and not going identical? Quantitative meta-analysis of two response inhibition tasks. *NeuroImage, 56*(3), 1655–1665.

Tomasi, D., Wang, R. L., Telang, F., Boronikolas, V., Jayne, M. C., Wang, G. J., et al. (2009). Impairment of attentional networks after 1 night of sleep deprivation. *Cerebral Cortex, 19*(1), 233–240.

Van Dongen, H. P., Maislin, G., Mullington, J. M., & Dinges, D. F. (2003). The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. *Sleep, 26*(2), 117–126.

Wager, T. D., Sylvester, C. Y., Lacey, S. C., Nee, D. E., Franklin, M., & Jonides, J. (2005). Common and unique components of response inhibition revealed by fMRI. *NeuroImage, 27*(2), 323–340.

White, C. N., Congdon, E., Mumford, J. A., Karlsgodt, K. H., Sabb, F. W., Freimer, N. B., et al. (2014). Decomposing decision components in the stop-signal task: A model-based approach to individual differences in inhibitory control. *Journal of Cognitive Neuroscience, 26*(8), 1601.

Xue, G., Aron, A. R., & Poldrack, R. A. (2008). Common neural substrates for inhibition of spoken and manual responses. *Cerebral Cortex, 18*(8), 1923–1932.