COMPENSATORY NEURAL RESPONSES AFTER 36 HOURS OF TOTAL SLEEP DEPRIVATION AND ITS RELATIONSHIP WITH EXECUTIVE CONTROL FUNCTION

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Yong-Cong Shao and Jian-Lin Qi contributed equally to the present paper. Special thanks to Shi-Jiang Li, Ph.D., Professor of Biophysics of the Medical College of Wisconsin and Dr. Yu-Feng Zang, Ph.D., Professor of Psychology of Beijing Normal University.

This work was supported by the Medical Sciences Foundation of Chinese PLA (06Z066).

Appreciation is due to anonymous reviewers.

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The neurobiological mechanisms of Total Sleep Deprivation (TSD) - induced changes in executive control function were investigated. Fourteen participants were measured by functional magnetic resonance imaging (fMRI) with the visual Go/No-go task after normal sleep and following 36 hours of TSD. The TSD-induced positive and negative blood oxygenation level-dependent (BOLD) signals compared with that after a normal night's sleep (NORM). The areas activated with positive BOLD signals include the superior prefrontal cortex and inferior prefrontal cortex, with negative BOLD signals in the anterior cingulated cortex (ACC) and right lingual gyrus. Increased activation may be related to the compensatory response since more attention resources are needed to perform the Go/No-go task after 36 hours of TSD and the decreased activation in the ACC may reflect the impact of executive control function by the TSD.

Keywords: sleep deprivation, compensatory recruitment, executive control function, fMRI, brain function.

Many researchers have proposed that the executive control system involves the right inferior lateral prefrontal cortex (PFC; Garavan, Ross, & Stein, 1999; Konishi, Nakajima, Uchida, Sekihara, & Miyashita, 1998), which relates mainly to successful response inhibition (stop) and the anterior cingulate cortex and medial frontal gyrus, which have been associated with monitoring errors (Braver, Barch, Gray, Molfese, & Snyder, 2001). These brain regions are considered to be crucial to the advanced cognitive control of behavior.

Executive function relying on inhibition has been found to be affected by total sleep deprivation (TSD; Chuah, Venkatraman, Dinges, & Chee, 2006; Jennings, Monk, & van der Molen, 2003). It is well established that TSD has a strong influence on automatic response performance, such as slowing down reaction time, and declining correct ratio (Drummond, Paulus, & Tapert, 2006). Sleep deprivation for 24 hours impairs both the error detection and error remedial actions, and highlights the inability to avoid making errors again after erroneous responses have already been made (Drummond, Brown, Salamat, & Gillin, 2004). Preliminary functional brain imaging studies have also revealed correlations between brain activation and performance decline after sleep deprivation. Along with the TSD, impaired response inhibition may result from impaired function of the right ventral cortex, typically with the inferior frontal gyrus (Chuah et al., 2006; Rubia, Smith, Brammer, & Taylor, 2003). Moreover, based on the study by Culham, Cavanagh, and Kanwisher (2001), impaired automatic response during TSD may relate to the dysfunction of sustained attention regions within the right dorsolateral prefrontal cortex. However, some researchers report increased brain activities after sleep deprivation when participants are carrying out cognitive tasks. Moreover, the categories of the tasks and different task loads also impact on brain activities (Mostofsky et al., 2003). So the detailed mechanisms of TSD which influence the executive control function and its relationship with the reaction of certain brain areas responsible for such an impact need further study.

The main purpose of this study was to investigate the influence of TSD on the executive control function by using combined behavioral and functional magnetic resonance imaging (fMRI) techniques. The change in brain activities underlying the declining executive control function after TSD may be reflected in changes of the blood oxygenation level-dependent (BOLD) signal. In order to address these problems, a visual Go/No-go task associated with response inhibition and conflict dictions was used during which BOLD signals were recorded before and after 36 hours of TSD.

METHOD

PARTICIPANTS

Fourteen healthy male undergraduate students (aged between 18 and 28; mean \pm SD, 25.9 \pm 2.3) recruited from Beijing Normal University participated in the experiment as paid volunteers for the study. All participants were right-handed and had normal or corrected normal vision. None of them had participated in psycho-physiological experiments previously. The general exclusion criteria were diseases of the central and peripheral nervous system, encephalic traumatisms, cardiovascular diseases and/or hypertension, cataracts and/or glaucoma, pulmonary problems, audiological problems, alcohol and drug abuse. All participants had normal intelligence (the Raven test, IQ>100; Raven, Raven, & Court, 1998) and completed the Symptom Checklist-90 (SCL-90; Derogatis, 1977) for the syndromes detected with T score on the General Symptom Index <60 (clinical range for general population involves T scores >63). All participants were required to maintain a regular sleep schedule and refrain from alcohol, caffeine, and chocolate intake and napping for 1 week prior to and during the study. The experiment protocol was approved by the Beijing Institute of Basic Medical Science and the General Hospital of the People's Liberation Army of China (Beijing, China). All participants gave written informed consent after the experiment had been fully explained and had a typical sleep patterns ensured by eight hours of sleep.

INSTRUMENTS

The stimuli of the visual Go/No-go task were two arrows (left and right) presented in a serial random pattern, alternating at 1 Hz. Participants were instructed to press a button when the target arrow appeared and withhold the response when the distracter arrow emerged.

A mixed block and event-related design was used for the inspection. The visual Go/No-go task contained five fixation blocks (resting as baseline) interleaved with four task blocks. Each task block consisted of 36 trials, with each stimulus presented for 200ms with 800ms interstimulus interval (Figure 1). The Go stimuli occurred with 66% probability, with the fixation mark on screen whenever the

stimulus was not. The No-go stimuli emerged randomly in each task block. Each block started with directional prompts for 3 seconds and lasted a total of 36 seconds. The entire task lasted 378 seconds (including 18 seconds at the start for fMRI data not analyzed due to partial saturation effects). The participants were told to respond as quickly as possible while maintaining a high level of accuracy. During the fixation blocks participants were required to fix their attention on the fixation mark.

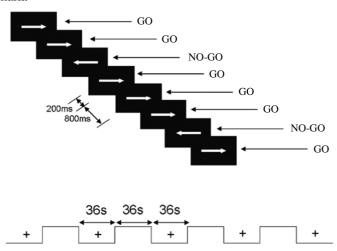


Figure 1. Schematic representation of the Go/No-go task and time parameters of a single run. The Go and No-go stimuli were alternated between blocks. Every stimuli picture was presented for 200ms with ITI for 800ms.

PROCEDURE

Each participant completed consent forms, received instructions, and practiced on the Go/No-go task that would be performed during the scanning session. The Go/No-go task was performed after full training to ensure that participants maintained their accuracy rate above 95%.

Each participant was scanned twice: one was scanned 12 hours after waking from a NORM in the laboratory and another after 36 hours of TSD (at 8:00 p.m.). The first scanning session took place approximately 1 week later at the same time of day (at 20:00) as the second one. All NORM and TSD nights took place in the basic aerospace institute with nursing staff present at all times. Participants always had a "partner" participant in the study to help keep each other awake. However, if participants showed signs of difficulty in remaining awake, the experimenter interacted directly with them if necessary to keep them awake. Participants were kept awake through the night under continuous behavioral monitoring, and they underwent subjective sleepiness assessment based on the Emotional Scale every

4 hours. Participants were not allowed to leave the laboratory during the period of TSD until they were escorted to the fMRI facility in the General Hospital of the People's Liberation Army of China.

FMRI SCANS

The fMRI scans were conducted at a GE 3.0T Signa LX scanner with a birdcage RF head coil, which is located at the General Hospital of the People's Liberation Army of China (Beijing, China). Visual stimuli of the Go/No-go task were back-projected onto a screen at the participant's feet and were viewed with the aid of prism glasses attached to the inside of the radio frequency head coil. Foam padding was used to limit head movements within the coil. Earplugs were used to attenuate scanner noise. Functional images were obtained by single-shot EPI sequence (TR: 2s, TE: 30ms, FOV: 256mm, 3.75mm*3.75mm in-plane resolution) of 20 6mm axial slices covering the entire brain and measuring the BOLD signal. High-resolution radio frequency spoiled gradient recalled acquisitions (SPGR) in the steady state anatomic images were acquired after functional imaging to allow subsequent anatomical localization of functional activation.

Participants' heart and respiration rates were continuously monitored and recorded during each task segment throughout the session using an MR-compatible pulse oximeter attached to the middle finger of the left hand and respiratory bellows strapped around the lower rib cage, respectively.

DATA ANALYSIS

Behavior data Due to technical errors, one participant's behavioral data were not recorded so the data for 13 participants were analyzed. The outcome variables for task performance included hit rate (correct button press for Go stimuli); response time for correct hits; false alarm rate (error of commission for No-go stimuli). All variables were analyzed with paired *t* tests using SPSS 11.5 for Windows. The physiological data were compared using paired *t* tests.

fMRI data Functional images were processed and analyzed with AFNI (Cox, 1996) in a two-step procedure: individual time-course analysis followed by group statistical analysis. In-plane motion correction and edge detection algorithms were first applied to the functional data. Intrasession image alignment to correct for motion across runs was performed using the reference image, the last image of the functional run that was acquired immediately before the SPGR image. After registration, rigid body motion correction was performed. None of the participants' data were excluded due to excess motion (>2mm). Interslice timing differences attributable to slice acquisition order were adjusted using 3dvolreg. Gaussian filtering was applied in the spatial domain using a smoothing kernel of 5mm full-width at half-maximum (FWHM) for individual activation maps. The

SPGR images were used to register the functional dataset to the volunteers' own three-dimensional image, and the resulting aligned dataset was transformed into Talairach space.

The functional image data were analyzed for both sessions using a general linear model with 3dDeconvolve after preprocessing (Ward, 2002). The activity of each voxel was expressed by the percentage of the area-under-the-curve (AUC%) relative to the area-under-the-baseline. For each participant we computed AUC% for Go, No-go, and Go+No-go as a whole. The map of the AUC% for each participant was then converted to standard Talairach space and spatially smoothed with FWHM 5-mm isotropic Gaussian Kernel before entering the group statistical test.

A one-sample t test against the null hypothesis of no effects was performed on the AUC% measure for the participants performing the Go/No-go task before and after 36 hours of TSD. A paired t test was employed to study the difference in AUC% between before and after sleep deprivation (p < 0.05). The 3dcluster program was then performed with corrected p < 0.05 and cluster size of 680ul on the datasets.

RESULTS

BEHAVIORAL DATA

Mean values for behavioral data and physiological responses are presented in Table 1. False alarm rates in the Go/No-go task increased significantly after sleep deprivation compared with the NORM situation, along with significant decrements in the hit rate.

TABLE 1
MEANS AND STANDARD DEVIATIONS OF PEDEODMANCE INDEVES

	Rested wakefulness	Mean (SD) $(n = 13)$ Sleep deprivation	t value
Behavior data			
Hit rate	0.99 (0.01)	0.97 (0.04)	2.38*
False alarm rate	0.04 (0.04)	0.10 (0.08)	3.29**
Hit RT(ms)	427.67 (39.01)	419.87 (20.09)	0.71
Physiological data			
Heart rate	71.95 (8.26)	71.03 (6.10)	1.88
Respiratory	20.35 (3.19)	19.70 (3.45)	0.58

Notes: Hit rate, false alarm rate, and Hit RT of the Go/No-go task during rested wakefulness and after sleep deprivation.

Hit rate was computed as correct hit of Go stimuli divided by the sum of Go stimuli; False alarm rate was computed as uncorrected No-go stimuli divided by the sum of No-go stimuli.

^{*} *p* < 0.05, ** *p* < 0.01

BRAIN ACTIVITIES

Results of the one-sample *t* test showed that the Go/No-go task-induced activities of the brain area of ACC, parietal lobe, and occipital cortex, as shown in Figure 2A. Paired *t* tests showed that TSD-induced positive and negative BOLD signals compared with signals in the NORM situation. The activated areas with positive BOLD signal include the superior prefrontal cortex, inferior prefrontal cortex, and others and with negative BOLD signal in the ACC and right lingual gyrus and others, as shown in Figure 2C. The detailed activated positions (x, y, z) in the Talairach space and activated brain volumes are listed in Table 2. Paired *t* tests showed that compared with the NORM situation, significant increases in PFC were found in R PFC (BA10) areas for Go stimuli, and significant declines of BOLD signals for No-go stimuli were found in the ACC area, as shown in Figure 2D and Figure 2E.

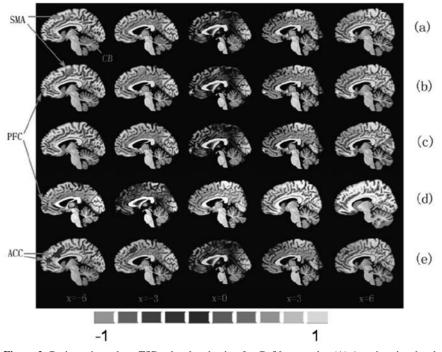


Figure 2. Brain regions show TSD-related activation for Go/No-go tasks. (A) Anterior cingulated cortex (ACC), supplementary motor area (SMA), and cerebellum (CB) activities were found before 36 hours of TSD; significant prefrontal cortex activities were found after 36h TSD (B); significant ACC hypoactivity was observed for Go/No-go task after 36h TSD compared with NORM. Besides, area in the right superior frontal lobe had shown elevated responsiveness (C); significant increasing in PFC were found in R PFC (BA10) areas for Go stimuli, and significant decline of BOLD signals for No-go stimuli were found in ACC area, as shown in Figures D and E (p < 0.05).

TABLE 2
REGIONS ACTIVATED SIGNIFICANTLY AFTER 36H TSD COMPARED WITH RESTED WAKEFULNESS
FOR GO/No-GO TASKS

Structure	Mean	SEM	BA	Volume (µl)	X	Y	Z
Task-Positive network							
R Cuneus	0.3043	0.0006	18	15459	1	-81	30
R Superior Frontal Gyrus/							
R Medial Frontal Gyrus	0.6525	0.0029	10/11	3687	4	58	-3
L Precentral Gyrus	0.1818	0.0006	6	2550	-53	-8	27
R Superior Temporal Gyrus	0.2029	0.0011	22/43	2527	61	-5	9
R Inferior Frontal Gyrus	0.3359	0.0025	38/47	1306	46	19	-11
L Superior Temporal Gyrus	0.2686	0.0027	38	1097	-45	12	-10
L Postcentral Gyrus	0.2522	0.0016	41	992	-58	-20	16
R Subcallosal Gyrus	0.1646	0.0017	34	860	25	4	-14
L Lingual Gyrus	0.2449	0.0032	18	838	-22	-66	1
L Lentiform Nucleus	0.1985	0.0040		512	-15	-11	-7
R Supramarginal Gyrus	0.2189	0.0018	40	745	57	-41	35
Task-Negative network							
R Anterior Cingulate Cortex	0.3911	0.0032	24	683	2	36	3
R Lingual Gyrus	0.3035	0.0018	18	814	20	-86	-1

Positive center-of-mass coordinates for x, y, and z refer to locations right (x), posterior (y), and superior (z) to the anterior commissure. R, Right; L, Left; BA, Brodmann area. Paired t tests, p < 0.05

DISCUSSION

Sleep deprivation greatly impacts people's cognitive function. As length of sleep deprivation increases, the function of inhibition declines (Chuah et al., 2006; Drummond et al., 2006). But this decline was affected by biological rhythm, as found in previous studies (Lavie, 2001; Montplaisir, 1981). So we chose the same time of the day (at 20:00) to assess the brain function to minimize the impact of biological rhythm.

The main goal in this research was to evaluate whether or not the ability to withhold a response is impaired by total sleep deprivation (TSD). In the fMRI study we found that the activities of ACC declined during the Go/No-go task, along with the activities of the cuneus after 36h total sleep deprivation. These results are consistent with the study which showed that sleep deprivation lowered sustained Go/No-go, task-related activation of the ACC regions (Chuah et al., 2006; Drummond et al., 2006; Lütcke & Frahm, 2007). In our study, ACC dysfunction during the visual Go/No-go task can be seen in the impaired brain executive control function after TSD; this can be confirmed by the decline of the BOLD signal after TSD induced by No-go stimuli. These results are consistent with behavior performance, showing the decline of inhibition function after 36 hours of sleep deprivation.

During our study, the declining inhibition may have had a relationship with the arousal level of the participant. Along with the decline of arousal level, utilities of brain sources were slowed down (De Valck, Cluydts, & Pirrera, 2004; Drummond et al., 2000). These data demonstrate that certain cortical areas, especially midline areas of the ACC that are critical for cognitive control, are more responsive in participants after 36 hours sleep deprivation. When it fell below the threshold of the requirement of cognitive tasks, performance declined. Our results confirm that the ACC has high status in executing functions.

Much research on fMRI has shown that sleep loss is associated with reduced cerebral metabolism within the PFC (Blatter, Opwis, Münch, Wirz-Justice, & Cajochen, 2005; Curcio, Ferrara, & De Gennaro, 2006; Kahn-Greene, Killgore, Kamimori, Balkin, & Killgore, 2007). Also from some studies it was reported that increased activities of PFC are induced by sleep deprivation (Drummond et al., 2006; Lütcke & Frahm, 2007). Some people surmised that different areas of the PFC give different responses to the task. In our study we found that under the circumstance of sleep deprivation, the prefrontal lobe has shown significant activation along with declining performance of Go/No-go tasks. It is known that compensatory recruitment is one of the brain's abilities to maintain performance when people are in an emergency situation. These results imply that in order to resist the decline of utilities of brain source, the PFC shows compensatory recruitment. In addition, the increased PFC activity after sleep deprivation shows that more attention resources are needed for this task. After 36h sleep deprivation, more attention resources are needed to perform the Go/No-go task. This may be a reason for the slight decline in reaction performance. Compensatory recruitment is one of the particular functions of the human brain, and it also explains why performance can be maintained when people are in an emergency situation (Drummond et al., 2000; Drummond, Meloy, Yanagi, Orff, & Brown, 2005). This deterioration of the performance following TSD may be related to increased sleepiness (Drummond & Brown, 2001; Killgore & McBride, 2006; Murphy, Richard, Masaki, & Segalowitz, 2006). The enhanced fatigue of sleepiness may underlie the poor performance of Go/No-go tasks.

In summary, we assessed the impact of 36h sleep deprivation on the ability to withhold a motor response by a Go/No-go task. It is clear that the automatic response component of this task requires attention resource sustaining. The significant activation of the prefrontal lobe shows a compensatory recruitment for the cognitive task. Our findings appear to indicate that the increased activation may be related to the compensatory response since more attention resources are needed to perform the Go/No-go task after 36h TSD, whereas the decreased activation in the ACC may reflect the impact on response inhibition of TSD. But whether withholding a response also relies mainly on the attention system or an inhibitory system independent of attention remains unclear. Additionally,

although the researchers found a significant ACC dysfunction relating to one aspect of inhibitory control, it remains to be seen whether or not other types of inhibitory control would show a similar deficit.

REFERENCES

- Blatter, K., Opwis, K., Münch, M., Wirz-Justice, A., & Cajochen, C. (2005). Sleep loss-related decrements in planning performance in healthy elderly depend on task difficulty. *Journal of Sleep Research*, 14, 409-417.
- Braver, T. S., Barch, D. M., Gray, J. R., Molfese, D. L., & Snyder, A. (2001). Anterior cingulate cortex and response conflict: Effects of frequency, inhibition and errors. *Cerebral Cortex*, 11, 825-836.
- 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, 7156-7162.
- Cox, R. W. (1996). AFNI: Software for analysis and visualization of functional magnetic resonance neuroimages. Computers and Biomedical Research, 29, 162-173.
- Culham, J. C., Cavanagh, P., & Kanwisher, N. G. (2001). Attention response functions: Characterizing brain areas using fMRI activation during parametric variations of attentional load. *Neuron*, 32, 737-745.
- Curcio, G., Ferrara, M., & De Gennaro, L. (2006). Sleep loss, learning capacity and academic performance. *Sleep Medicine Reviews*, **10**, 323-337.
- De Valck, E., Cluydts, R., & Pirrera, S. (2004). Effect of cognitive arousal on sleep latency, somatic and cortical arousal following partial sleep deprivation. *Journal of Sleep Research*, 13, 295-304.
- Derogatis, L. R. (Ed.). (1977). SCL-90. Administration, scoring and procedures manual-I for the R(evised) version. Baltimore: John Hopkins University School of Medicine, Clinical Psychometrics Research Unit.
- Drummond, S. P., & Brown, G. G. (2001). The effects of total sleep deprivation on cerebral responses to cognitive performance. *Neuropsychopharmacology*, **25**, S68-S73.
- 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, 655-657.
- Drummond, S. P., Brown, G. G., Salamat, J. S., & Gillin, J. C. (2004). Increasing task difficulty facilitates the cerebral compensatory response to total sleep deprivation. *Sleep*, 27, 445-451.
- Drummond, S. P., Meloy, M. J., Yanagi, M. A., Orff, H. J., & Brown, G. G. (2005). Compensatory recruitment after sleep deprivation and the relationship with performance. *Psychiatry Research*, 140, 211-223.
- Drummond, S. P., Paulus, M. P., & Tapert, S. F. (2006). Effects of two nights sleep deprivation and two nights recovery sleep on response inhibition. *Journal of Sleep Research*, **15**, 261-265.
- Garavan, H., Ross, T. J., & Stein, E. A. (1999). Right hemispheric dominance of inhibitory control: An event-related functional MRI study. Proceedings of the National Academy of Sciences of the United States of America, 96, 8301-8306.
- Jennings, J. R., Monk, T. H., & van der Molen, M. W. (2003). Sleep deprivation influences some but not all processes of supervisory attention. *Psychological Science*, 14, 473-479.
- Kahn-Greene, E. T., Killgore, D. B., Kamimori, G. H., Balkin, T. J., & Killgore, W. D. (2007). The effects of sleep deprivation on symptoms of psychopathology in healthy adults. *Sleep Medicine*, 8, 215-221.
- Killgore, W. D., & McBride, S. A. (2006). Odor identification accuracy declines following 24h of sleep deprivation. *Journal of Sleep Research*, **15**, 111-116.

- Konishi, S., Nakajima, K., Uchida, I., Sekihara, K., & Miyashita, Y. (1998). No-go dominant brain activity in human inferior prefrontal cortex revealed by functional magnetic resonance imaging. *European Journal of Neuroscience*, 10, 1209-1213.
- Lavie, P. (2001). Sleep-wake as a biological rhythm. Annual Review of Psychology, 52, 277-303.
- Lütcke, H., & Frahm, J. (2007). Lateralized anterior cingulate function during error processing and conflict monitoring as revealed by high-resolution fMRI. Cerebral Cortex, 18, 508-515.
- Montplaisir, J. (1981). Depression and biological rhythm: Therapeutic effects of sleep deprivation [in French]. L'union médicale du Canada, 110, 272-276.
- Mostofsky, S. H., Schafer, J. G., Abrams, M. T., Goldberg, M. C., Flower, A. A., Boyce, A., et al. (2003). fMRI evidence that the neural basis of response inhibition is task-dependent. *Cognitive Brain Research*, **17**, 419-430.
- Murphy, T. I., Richard, M., Masaki, H., & Segalowitz, S. J. (2006). The effect of sleepiness on performance monitoring: I know what I am doing, but do I care? *Journal of Sleep Research*, 15, 15-21.
- Raven, J., Raven, J. C., & Court, J. H. (1998). Manual for Raven's advanced progressive matrices and vocabulary scales. Sec. 4, The advanced progressive matrices. Oxford: Oxford Psychologists Press.
- Rubia, K., Smith, A. B., Brammer, M. J., & Taylor, E. (2003). Right inferior prefrontal cortex mediates response inhibition while mesial prefrontal cortex is responsible for error detection. *Neuroimage*, 20, 351-358.
- Ward, B. D. (2002). Deconvolution analysis of FMRI time series data. AFNI Manual. Milwaukee, WI: Biophysics Research Institute, Medical College of Wisconsin.