# Changes in the Power Characteristics of the α-Rhythm in Students with Different Levels of Anxiety in Normal Sleep and Partial Deprivation Solving the Go/NoGo Problem

E. A. Cheremushkin<sup>a, \*</sup>, N. E. Petrenko<sup>a</sup>, N. N. Alipov<sup>b</sup>, and O. V. Sergeeva<sup>b</sup>

<sup>a</sup> Institute of Higher Nervous Activity and Neurophysiology, Russian Academy of Sciences, Moscow, 117485 Russia <sup>b</sup> Pirogov Russian National Research Medical University, Ministry of Health of the Russian Federation, Moscow, 117997 Russia

\*e-mail: khton@mail.ru

Received June 30, 2021; revised September 11, 2021; accepted September 21, 2021

Abstract—The effect of partial sleep deprivation on cognitive control was studied in 26 students with different levels of trait anxiety. The synchronization—desynchronization reactions of the EEG  $\alpha$ -oscillations were evaluated during the Go/NoGo test. In less anxious students, a reduction in sleep time on the night before the study led to a decrease in the desynchronization response to a positive conditioning stimulus (Go) and thus smoothed out the differences with the response to a stimulus that inhibited the behavioral response (NoGo). The smoothing of the  $\alpha$ -rhythm responses to stimuli of different signal significance after deprivation indicated deterioration of cognitive control in this group. In students with a high level of anxiety, there were no differences in responses to Go/No Go stimuli either after a night's sleep of normal duration or after a single sharp reduction of it. They probably had a reduced level of cognitive control, and it did not depend on partial sleep deprivation.

Keywords: anxiety, sleep deprivation, cognitive control, EEG,  $\alpha$ -rhythm, synchronization-desynchronization

**DOI:** 10.1134/S0362119722020049

The recently increased pace of life raises the problem of lack of sleep in many people, especially students. A short time of night sleep causes an increase in anxiety [1-3]. The onset of anxiety and an increase in anxiety symptoms are most pronounced the day after lack of sleep [4, 5]. However, clinical studies of the relationship between sleep deprivation and anxiety are conflicting. It has been found that chronic sleep deprivation can subsequently lead to the development of anxiety disorders [2], other authors have shown that anxiety disorders cause insomnia, but not vice versa [6]. From another point of view, sleep disturbance increases the risk of developing anxiety or depression, and the presence of anxiety or depressive symptoms increases the likelihood of insomnia [7]. It is less studied how the bioelectric activity of the human brain changes when anxiety and sleep deprivation factors are combined.

According to the literature, in the cases of both anxiety [8] and sleep deprivation, there are changes in the activity of the prefrontal areas of the cortex and its interactions with other areas of the brain [9, 10]. Clinical studies aimed at identifying electroencephalographic (EEG) signs of anxiety disorders have shown that the power characteristics of the EEG  $\alpha$ -rhythm are large in the right frontotemporal region compared

to the left [11]. Neuroimaging studies have shown a negative relationship between anxiety and the volume of gray matter in the prefrontal cortex [12]. Another study have shown that high levels of anxiety are correlated with an increased blood flow in the frontal zones (ventral medial prefrontal cortex) of the right hemisphere [8]. The high activity of this area of the brain obtained with the use of functional magnetic resonance imaging (fMRI) was also found in subjects with sleep disorders in the state of restful wakefulness [13]. Positron emission tomography (PET) has shown that sleep deprivation is accompanied by extensive changes in the metabolic activity of the brain, including the prefrontal cortex [9, 10], which plays a key role in providing inhibitory processes of cognitive control and regulation of human behavior [14].

A good test for assessing the state of these processes is the Go/NoGo technique, which is used, i.a., in studies of anxiety [15, 16], autonomic dysfunction [17] and sleep disorders [18]. Its essence is as follows. In the case of a conditioning positive stimulus (Go), the subject waits for the triggering stimulus and presses the joystick button. The action of the inhibitory stimulus (NoGo) cancels the motor reaction. Thus, conditioning stimuli change the behavior of the subjects and make it possible to study electrophysiological markers of inhibitory processes of cognitive control.

The aim of the study was to assess the effect of the duration of night sleep on the organization of cognitive activity in students with different levels of anxiety. The main objective was to identify differences in synchronization–desynchronization of the  $\alpha$ -rhythm in response to Go/NoGo stimuli in students with high and low anxiety under the conditions of normal duration of night sleep and its partial deprivation.

# **METHODS**

According to the results of mass questioning of second-year students of the schools of medicine and pediatrics of the Russian National Research Medical University (Moscow), using the Spielberger test, groups with relatively low (M =  $34.2 \pm 1.2$  points, 6 boys and 7 girls, group I) and high (M =  $65.0 \pm$ 1.7 points, 7 boys and 6 girls, group II) levels of trait anxiety were distinguished. All subjects, practically healthy people, were familiar with the research procedure and agreed to participate in it.

During the week preceding the study, the subjects completed the Sleep Diary [19], in which, in particular, the duration of sleep was noted. The study was carried out twice: after its usual duration the day before  $(M = 6.5 \pm 0.2 \text{ and } M = 7.6 \pm 0.5 \text{ h}, \text{ groups I and II},$ respectively) and with a one-time contraction, the socalled. partial deprivation (M =  $3.5 \pm 0.2$  and M =  $3.5 \pm 0.3$  h). Experiments for each subject were carried out with a week pause and alternated in random order. They started after the second class, after 11:30 p.m. The subject was in front of the monitor in a chair with a headrest, in a darkened, soundproof and shielded room. The distance from the screen was 80 cm. The subject was presented with complex stimuli (n = 68), consisting of three parts: S1 (exclamation mark), warning stimulus; *S*2, conditioning stimulus (Go/NoGo); and S3, triggering stimulus. With a pause of 2 s after the start of stimulus presentation S1, a positive (Go) or negative (NoGo) conditioning signal (S2), a circle with a diameter of 1 cm in green or blue. The colors changed in a random order, while the number of signals Go and NoGo was the same (34 each). Then, with a pause of 2 s after the start of presentation S2 presented the starting stimulus (S3), a white light spot. If the conditioning stimulus S2 was green, the subject had to press the joystick button (Go). If S2 was blue, there was no need to press (NoGo). Pause between S2 and S3 was necessary in order to exclude the influence of neurophysiological processes providing a motor response upon presentation of a stimulus. Go, on the bioelectrical activity of the brain. Thus, the "depression factor" could not be considered when comparing the EEG responses caused by differentiation stimuli Go and NoGo. We used a fixed, not random, pause between S2 and S3 for the possibility of comparing the dynamics of the mentioned reactions at the same time intervals. At the same time, the effect of anticipation (predicting) the appearance of S3, which affects the magnitude of the motor response. The exposure time of all stimuli was 350 ms. The interval between the S1-S3 stimulus complexes was 4-7 s and changed in a random order.

Throughout the experiment, the EEG was continuously recorded. It included background recordings (with eyes closed and open) and recordings during testing the subject using complex stimuli. The presentation of stimuli and their synchronization with the EEG was carried out according to the program of the Neostimul system (Neurobotics). EEG derivation, amplification and filtration were performed using the software package Neocortex-Pro (Neurobotics). The sampling rate was 250 Hz, the bandwidth of amplifier frequencies was 0.5-70 Hz. EEG was recorded using silver chloride electrodes (Micromed, Hungary) with a resistance not exceeding 5 k $\Omega$ . Electrical activity from the surface of the head was removed using 16 electrodes located in accordance with the scheme  $10-20 (F_3, F_4, F_7, F_8, F_2, C_3, C_4, C_2, T_3, T_4, P_3, P_4, T_5,$  $T_6, O_1, O_2$ ). For EEG recording, monopolar monopolar montage of leads was used, the reference was linked earlobe electrodes.

Regardless of the duration of night sleep, in both groups of subjects, a few errors were observed when the button was pressed. They represented an anticipatory, impulsive response: pressing occurred on a stimulus Go rather than a triggering stimulus. Samples with such a reaction were removed from further analysis.

EEG recording segments 2 s in duration in the interval between S2-S3 (from the beginning of S2) presentation before the start of the S3 presentation) were processed. In these segments, for each EEG derivation, a continuous wavelet transform was performed using the "maternal" Morlet-wavelet complex. In the frequency band of 8-13.5 Hz (the range of the  $\alpha$ -rhythm) with a step of 0.5 Hz and a time resolution of 1 ms, the values of the modulus of the wavelet transform coefficient (WTC), which characterizes the power of biopotentials, were calculated. The WTC s obtained from all EEG derivations were summarized. The power characteristics of EEG reactions smoothed in this way make it possible to assess the general trends of changes that are caused by differentiating stimuli. It is important to emphasize that further all subsequent transformations of the WTC and statistical processing were carried out both for the WTC of each EEG derivation, and for the WTC summed over all the leads.

The frequency domain of 8-13.5 Hz was averaged over all frequencies included in it. Then, 10 consecutive 200-ms intervals were identified in the interval between S2-S3 and within them, the time averaging of the WTC was carried out. To assess the proportion of the value of the post-stimulus potentials changes in relation to the pre-stimulus potentials, the following operation was performed. From the 10 values obtained using the calculations described above, the one obtained in the same way on the second EEG segment immediately preceding *S*2 value, divided by this value and multiplied by 100.

The characteristics of post-stimulus changes in the power of the EEG  $\alpha$ -rhythm obtained in this way were analyzed using multivariate analysis of variance (ANOVA RM) using the Greenhouse–Gesser correction. The influence of the "situation" factors (2 levels: the normal duration of sleep and its deprivation) were analyzed as intragroup factors; "Stimulus" (2 levels: Go and NoGo); "Time" (10 segments of 200 ms). The influence of the "Group" factor (2 levels: the group with high and low anxiety) was investigated as an intergroup one. Paired comparisons of the characteristics of post-stimulus changes in the  $\alpha$ -rhythm between stimuli Go and NoGo was carried out separately for situations of normal sleep and deprivation, and separately for groups with high and low anxiety. For this purpose, Student's t test was used. Calculations were performed using the Matlab 78.01 and SPSS 13.0 software packages.

#### RESULTS

In low-anxiety subjects, the time to press the button under the conditions of normal sleep duration is  $109.25 \pm 7.28$  ms; with insufficient duration,  $130.26 \pm$ 11.06 ms. In highly anxious subjects, this time is  $137.90 \pm 6.48$  and  $156.27 \pm 9.96$  ms, respectively. The low average values of the motor response are the result of anticipation processes that occur at a fixed time interval between stimuli. Thus, in subjects with a high level of anxiety, compared with a low-anxiety group, the response to the triggering stimulus takes a longer time. However, this difference was significant only under conditions of normal sleep duration (t = -2.94; d.f. = 23; p = 0.007). With insufficient duration, this behavioral indicator in groups approaches (t = -1.75; d.f.= 23; p = 0.093). Deprivation in both less anxious and highly anxious students significantly increased the time of pressing (t = -4.08; d.f. = 12; p = 0.002 and t =-4.33; d.f.= 12; p = 0.001, respectively).

Analysis of variance of the powers of the  $\alpha$ -rhythm averaged over all derivations revealed a statistically significant joint influence of the factors "Situation × Stimulus × Group" (F(1; 24) = 4.57; p = 0.043) and "Situation × Stimulus × Time × Group" (F(4; 98) =2.58; p = 0.041). This result indicates a difference in the reactivity of the  $\alpha$ -rhythm depending on the duration of sleep, the type of stimulus, and the group of subjects. Paired differences between positive and inhibitory conditioning stimuli for the total EEG were revealed only for the group of less anxious subjects under conditions of normal duration of night sleep in the interval from 1000 to 1600 ms (1000–1200 ms: t =-3.14; d.f.= 12; p = 0.008; 1200–1400 ms: t = -4.19; d.f.= 12; p = 0.001; 1400–1600 ms: t = -4.65; d.f. =

12; p = 0.0005). Paired comparisons carried out for individual regions in this group of subjects showed the following. Significant differences between EEG responses evoked by the presentation of positive and inhibitory stimuli are observed in the range of 600-800 ms in most frontal-central leads ( $C_3 Z = -2.85$ ;  $p = 0.004; C_4 Z = -2.28; p = 0.022; F_3 Z = -2.18; p = 0.029; F_7 Z = -2.59; p = 0.009; F_8 Z = -2.89; p = 0.009; F_8 Z$ 0.003;  $T_3 Z = -2.02$ ; p = 0.043;  $T_4 Z = -2.18$ ; p =0.022), in the interval 800–1000 ms– $F_4 Z = -2.64$ ;  $p = 0.008; F_8 Z = -1.92; p = 0.054; C_3 Z = -1.92; p = 0.054; C_4 Z = -2.07; p = 0.037.$  In the frontotemporal region, differences are also observed in a later period from 1200–1400 ms ( $F_8 Z = -2.07$ ; p = 0.037). For caudal areas, differences between stimuli Go and NoGo statistically significant only bilaterally in the parietal areas of the cortex in the range of 600-800 ms  $(R_3 Z = -2.28; p = 0.022; R_4 Z = -2.13; p = 0.033).$ 

Analysis of variance, carried out for individual EEG derivations, revealed the following. The effect of sleep deprivation was found only in less anxious students and only on stimulus. Go in the frontal-central areas of EEG derivation ( $F_4$ : F(1; 24) = 9.03; p =0.006,  $F_7$ :  $F(1; 24) = 6.33; p = 0.019, F_8$ : F(1; 24) =5.40, p = 0.029,  $C_3$ : F(1; 24) = 5.095; p = 0.033,  $T_3$ :  $F(1; 24) = 5.72; p = 0.025, T_4: F(1; 24) = 4.11; p =$ 0.05). After sleep of normal duration, these students showed a pronounced desynchronization of the  $\alpha$ rhythm, and after partial deprivation it disappeared (Fig. 1, I). In more anxious students, statistically significant differences in the responses of the bioelectrical activity of the cerebral cortex after normal sleep duration and sleep deprivation were not observed to any of the conditioning stimuli (Fig. 1, II).

Analysis of bioelectrical activity in groups with different levels of anxiety showed a difference in the responses of  $\alpha$ -oscillations only in responses to a stimulus. Go and only with normal sleep duration ( $F_3$ : F(1;24) = 6.07; p = 0.021,  $F_4$ : F(1; 24) = 6.45; p = 0.018,  $F_8$ : F(1; 24) = 7.95, p = 0.009 and  $C_4$ : F(1; 24) = 6.61, p = 0.017). In these leads, the reaction of desynchronization of the  $\alpha$ -rhythm is significantly less pronounced in students with a high level of anxiety than in less anxious ones. On incentive NoGo there were no statistically significant differences between the groups with different levels of anxiety, either with normal sleep duration or with sleep deprivation.

A significant influence of the "Stimulus" factor was revealed only for a group of students with a low level of anxiety in a situation of normal sleep duration in the regions  $C_4$  (F(1; 24) = 5.09; p = 0.033),  $F_4$  (F(1;24) = 4.45; p = 0.046) and  $F_7$  (F(1; 24) = 4.49; p =0.045). In this group, with a normal duration of sleep in response to a stimulus Go a pronounced reaction of desynchronization of  $\alpha$ -potentials is observed. On incentive NoGo the reaction of the  $\alpha$ -rhythm is less pronounced and fluctuates around the isoline.

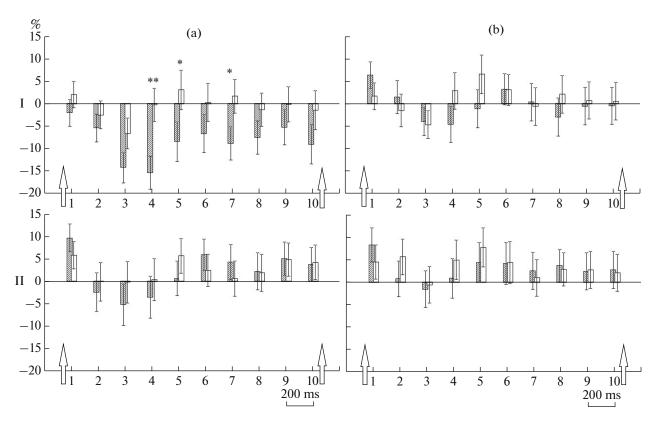


Fig. 1. Synchronization/desynchronization of the  $\alpha$ -rhythm of the EEG derivation  $F_8$  after stimulus presentation Go/NoGo in subjects with high and low anxiety in conditions of normal sleep and partial deprivation. I, low anxiety; II, high anxiety; (a) normal sleep, (b) partial deprivation; shaded columns, Go; unshaded columns, NoGo; vertically, the change in the power of the  $\alpha$ -rhythm in relation to the prestimulus segment of the EEG, %; horizontally, time intervals, 200 ms; arrows from left to right indicate the appearance of the conditioning (Go/NoGo) and starting incentives; \* and \*\* significant differences between the responses to the Go and NoGo stimuli (p < 0.05 and p < 0.01, respectively); the error of the mean is shown.

## DISCUSSION

The research revealed the influence of the duration of night sleep on the organization of cognitive activity in students with different levels of anxiety. In all subjects, when performing a task of the Go/NoGo type a decrease in the usual duration of nocturnal sleep led to an increase in the motor response under conditions of a delayed response to the presentation of a trigger stimulus. This result is consistent with the previously described impairment of cognitive functions with insufficient sleep duration [9, 10].

Significant differences in the characteristics of the EEG  $\alpha$ -rhythm between conditioning stimuli triggering the subsequent motor response (Go) and canceling it (NoGo), are observed in the antero-central areas only in the group with low anxiety and only after sleep of usual duration. In response to stimulus Go in this group, there is a pronounced desynchronization reaction to the NoGo stimulus, slight synchronization of the  $\alpha$ -rhythm. According to our early studies, this type of response (synchronization of the  $\alpha$ -rhythm to the NoGo stimulus and its desynchronization to Go) is a normative response of a given rhythm to these stimuli [20]. We have shown that synchronization of the

HUMAN PHYSIOLOGY Vol. 48 No. 2 2022

 $\alpha$ -rhythm in response to the NoGo stimulis has a clearly pronounced localized and lateralized character: it was noted in the motor zone of the left hemisphere (leads  $C_3$ ,  $FC_3$ ), i.e., in the area of the cortex directly controlling the movement of the right hand. In [21], it was also shown that with voluntary suppression of the motor reaction of the hand, synchronization of the  $\alpha$ -rhythm occurs in a limited area of the motor cortex directly related to the suppressed movements. The involvement of the central regions of the cortex, which are motor zones, may be associated with the preparation of a motor response to a Go stimulus. The same areas, according to fMRI data, are involved in the inhibition of irrelevant actions when performing Go/NoGo test [22].

Key differences between incentives Go and NoGo are observed mainly in the lateral prefrontal cortex ( $F_3$ ,  $F_4$ ,  $F_7$ ,  $F_8$ ), which corresponds to the concept of the key role of the frontal areas in ensuring the processes of cognitive control in mental activity [14]. The prefrontal cortex, along with the cingulate cortex, is involved in the provision of inhibition mechanisms [23]. In a study with simultaneous registration of the activity of the reticular nuclei of the thalamus and the frontal cortex, the participation of the corticothalamic system of the brain in the provision of inhibitory control processes, including when performing tasks Go/NoGo [24].

Partial sleep deprivation in the group with lower anxiety leads to a smoothing out of differences in Go/NoGo responses. This is due to a sharp decrease in the response of desynchronization to the Go stimulus. In the group with severe anxiety, there were no differences in responses to the Go and NoGo stimuli, either with normal sleep duration or with its decrease. A slight synchronization of  $\alpha$ -potentials is observed practically everywhere. The lack of a differentiated response to different conditioning stimuli may reflect a lack of cognitive control of mental activity. In the group with a low level of anxiety, its weakening occurs in conditions of partial sleep deprivation. In highly anxious students, this state of cognitive control is likely independent of sleep duration. Impaired cognitive control in anxious people is a fact described earlier [25, 26].

In our earlier studies, it was shown that students with learning difficulties exhibited a significant weakening of the induced synchronization/desynchronization reactions of  $\alpha$ -oscillations to conditioning Go/NoGo signals, which led to the absence of differences in post-stimulus reactions in almost all EEG derivations [27]. Apparently, in these students one can state a significant decrease in the functional activity of the corticothalamic system of selective attention and a decrease in the level of cognitive control. The close results obtained in the current study (almost complete absence of difference in the responses of  $\alpha$ -oscillations to conditioning stimuli of different signal values, especially in a group of highly anxious students) suggests that they have significant problems with the level of cognitive control. It should be noted that a similar picture is observed in the anxious group with a normal duration of night sleep, and under conditions of partial sleep deprivation, it practically does not change; in other words, the signs of impaired cognitive control in this group do not depend on the duration of sleep on the eve of the study. In the group of less anxious subjects, sleep deprivation leads to the disappearance of differences in responses between conditioning stimuli and, accordingly, to the appearance of signs of deterioration in cognitive control.

# CONCLUSIONS

A decrease in the duration of night sleep on the day before the study negatively affects both the behavioral and neurophysiological indicators of the cognitive activity of students with different levels of anxiety. In all subjects, when performing a task of the Go/NoGo type the motor response increased under conditions of a delayed response to the presentation of a triggering stimulus. In students with a lower level of anxiety, deprivation leads to a decrease in the response of desynchronization of the EEG  $\alpha$ -rhythm to a positive conditioning stimulus (Go), thereby leveling the differences in the responses of the  $\alpha$ -rhythm to stimuli of different signal significance. This result indicates a deterioration in the level of cognitive control under the influence of a decrease in the time of night sleep. Students with high anxiety have no differences in the responses of  $\alpha$ -potentials to stimuli Go and NoGo both under conditions of normal duration of night sleep, and with its sharp decrease. Thus, the level of cognitive control in these subjects does not depend on the duration of sleep.

#### FUNDING

This work was supported by state budget funds under the state order of the Ministry of Education and Science of the Russian Federation for 2021–2023.

## COMPLIANCE WITH ETHICAL STANDARDS

The study was carried out in accordance with the principles of biomedical ethics formulated in the 1964 Declaration of Helsinki and its subsequent updates, and approved by the ethical commission of the Institute of Higher Nervous Activity and Neurophysiology, Russian Academy of Sciences (Moscow).

#### INFORMED CONSENT

Each study participant provided a voluntary written informed consent, signed by him after explaining to him the potential risks and benefits, as well as the nature of the upcoming study.

#### CONFLICT OF INTEREST

The authors declare no conflicts of interest.

## **OPEN ACCESS**

This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit ttp://creativecommons. org/licenses/by/4.0/.

#### REFERENCES

- Babson, K.A., Trainor, C.D., Feldner, M.T., and Blumenthal, H., A test of the effects of acute sleep deprivation on general and specific self-reported anxiety and depressive symptoms: an experimental extension, *J. Behav. Ther. Exp. Psychiatry*, 2010, vol. 41, no. 3, p. 297.
- Roberts, R.E. and Duong, H.T., Is there an association between short sleep duration and adolescent anxiety disorders? *Sleep Med.*, 2017, vol. 30, p. 82.
- Poluektov, M.G. and Pchelina, P.V., Sleep disorders and anxiety, *Eff. Farmakoter. Nevrol. Psikhiatr.*, 2017, vol. 35, p. 80.
- 4. Cox, R.C., Sterba, S.K., Cole, D.A., et al., Time of day effects on the relationship between daily sleep and anxiety: an ecological momentary assessment approach, *Behav. Res. Ther.*, 2018, vol. 111, p. 44.
- Bean, Ch.A.L. and Ciesla, J.A., Naturalistic partial sleep deprivation leads to greater next-day anxiety: the moderating role of baseline anxiety and depression, *Behav. Ther.*, 2021, vol. 52, no. 4, p. 861.
- Johnson, E.O., Roth, T., and Breslau, N., The association of insomnia with anxiety disorders and depression: exploration of the direction of risk, *J. Psychiatr. Res.*, 2006, vol. 40, no. 8, p. 700.
- Jansson-Fröjmark, M. and Lindblom, K., A bidirectional relationship between anxiety and depression, and insomnia? A prospective study in the general population, *J. Psychosomatic Res.*, 2008, vol. 64, no. 4, p. 443.
- 8. Tian, X., We, D., Du, X., et al., Assessment of trait anxiety and prediction of changes in state anxiety using functional brain imaging: a test-retest study, *NeuroImage*, 2016, vol. 133, p. 408.
- Cui, J., Tkachenko, O., Gogel, H., et al., Microstructure of frontoparietal connections predicts individual resistance to sleep deprivation, *NeuroImage*, 2015, vol. 106, p. 123.
- de Almondes, K.M., Junior, F., and Alves, N.T., Sleep deprivation and implications for recognition and perception of facial emotions, *Sleep Biol. Rhythms*, 2016, vol. 14, no. 1, p. 13.
- Al-Ezzi, A., Kamel, N., Faye, I., and Gunaseli, E., Review of EEG, ERP, and brain connectivity estimators as predictive biomarkers of social anxiety disorder, *Front. Psychol.*, 2020, vol. 11, p. 730.
- Montag, C., Reuter, M., Jurkiewicz, M., et al., Imaging the structure of the human anxious brain: a review of findings from neuroscientific personality psychology, *Rev. Neurosci.*, 2013, vol. 24, no. 2, p. 167.
- Kay, D.B. and Buysse, D.J., Hyperarousal and beyond: new insights to the pathophysiology of insomnia disorder through functional neuroimaging studies, *Brain Sci.*, 2017, vol. 7, no. 3, p. 23.
- Zhang, S., Xu, M., Chang, W.-C., et al., Organization of long-range inputs and outputs of frontal cortex for top-down control, *Nat. Neurosci.*, 2016, vol. 19, no. 12, p. 1733.

HUMAN PHYSIOLOGY Vol. 48 No. 2 2022

- 15. Leue, A., Cano Rodilla, C., and Beauducel, A., Worryinducing stimuli in an aversive Go/NoGo task enhance reactivecontrol in individuals with lower trait-anxiety, *Biol. Psychol.*, 2017, vol. 125, p. 1.
- Voegler, R., Peterburs, J., Lemke, H., et al., Electrophysiological correlates of performance monitoring under social observation in patients with social anxiety disorder and healthy controls, *Biol. Psychol.*, 2018, vol. 132, p. 71.
- Cheremushkin, E.A., Petrenko, N.E., Yakovenko, I.A., et al., Specific electrophysiological activity of brain during changes in behavior as a response to Go/NoGo stimuli in students with signs of vegetative dysfunction and with normal vegetative status, *Mezhdunar. Nauchno-Issled. Zh.*, 2017, vol. 55, no. 1-1, p. 171.
- Petrenko, N.E., Cheremushkin, E.A., Alipov, N.N., et al., Downward inhibitory control in students with different sleep quality in the experiment with Go/No-Go stimuli upon recognition of facial expression, *Vestn. Nevrol., Psikhiatrii Neirokhir.*, 2018, no. 7, p. 64.
- Maich, K.H.G., Lachowski, A.M., and Carney, C.E., Psychometric properties of the consensus sleep diary in those with Insomnia disorder, *Behav. Sleep Med.*, 2018, vol. 16, no. 2, p. 117.
- Kostandov, E.A., Cheremushkin, E.A., Petrenko, N.E., and Yakovenko, I.A., A cognitive hypothesis of the development of differentiative cortical inhibition in humans, *Hum. Physiol.*, 2016, vol. 42, no. 4, p. 351.
- 21. Sauseng, P., Gerloff, Ch., and Hummel, F.C., Two brakes are better than one: the neural bases of inhibitory control of motor memory traces, *NeuroImage*, 2013, vol. 65, p. 52.
- 22. Levy, B.J. and Wagner, A.D., Cognitive control and right ventrolateral prefrontal cortex: reflexive reorienting, motor inhibition, and action updating, *Ann. N.Y. Acad. Sci.*, 2011, vol. 1224, no. 1, p. 40.
- Garavan, H., Ross, T.J., and Stein, E.A., Right hemispheric dominance of inhibitory control: an event-related functional MRI study, *Proc. Natl. Acad. Sci.* U.S.A., 1999, vol. 96, no. 14, p. 8301.
- Marzinzik, F., Wahl, M., Schneider, G.-H., et al., The human thalamus is crucially involved in executive control operations, *Cognit. Neurosci.*, 2008, vol. 20, no. 10, p. 1903.
- 25. Dong, M., Xia, L., Lu, M., et al., A failed top-down control from the prefrontal cortex to the amygdala in generalized anxiety disorder: evidence from resting-state fMRI with Granger causality analysis, *Neurosci. Lett.*, 2019, vol. 707, art. ID 134314.
- Morsel, A.M., Dhar, M., Hulstijn, W., et al., Inhibitory control in euthymic bipolar disorder: Event related potentials during a Go/NoGo task, *Clin. Neurophysiol.*, 2017, vol. 128, no. 4, p. 520.
- 27. Kostandov, E.A. and Cheremushkin, E.A., Disorders of top-down cognitive control in students with learning difficulties, *Hum. Physiol.*, 2017, vol. 43, no. 4, p. 377.