FEATURES OF REACTIVITY OF THE EEG MU RHYTHM IN CHILDREN WITH AUTISM SPECTRUM DISORDERS IN HELPING BEHAVIOR SITUATIONS

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One of the subjects being discussed by the professional community currently is the role possibly played by the mirror neuron system (MNS) in the violation of social behavior of children with autism spectrum disorders (ASD). The MNS is known to shape the perception of emotions of others and understanding and imitation of their actions. Mu rhythm desynchronization in EEG is considered to be the indicator of the MNS activation. The purpose of this study was to identify the features of reactivity of the EEG mu rhythm within an individually determined frequency range in preschoolers with ASD in situations requiring instrumental, emotional and altruistic helping behavior (HB). The study involved children 4–7 years old with ASD (n = 26) and their normally developing peers without the condition (n = 37). Although in most cases, HB was more pronounced in the group of normally developing children, the differences between the groups are significant only for altruistic HP (p < 0.01), and for the situation requiring complex altruistic and emotional HP it approaches significance (p = 0.09). Evaluation of the mu rhythm reactivity indices showed that the tasks invoking complex altruistic and emotional HB bring this indicator down significantly in children with ASD compared to the group of normally developing participants, as shown by the central leads of the left and right hemispheres and the parietal lead of the right hemisphere (C3: p = 0.02 ; C4: p = 0.03; P4: p = 0.03). It is assumed that the detected features stem from the impaired functioning of the MNS and the downstream regulation to the MNS from prefrontal cortex and other areas of the neocortex. The data obtained can be used in development of EEG biofeedback training protocols for children with ASD.

Keywords: children, autism, EEG, μ rhythm, mirror neuron system, prosocial behavior

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ОСОБЕННОСТИ РЕАКТИВНОСТИ μ-РИТМА ЭЭГ У ДЕТЕЙ С РАССТРОЙСТВАМИ АУТИСТИЧЕСКОГО СПЕКТРА В СИТУАЦИЯХ ПОМОГАЮЩЕГО ПОВЕДЕНИЯ

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В настоящее время обсуждается вопрос о возможной роли зеркальной системы мозга (ЗСМ), участвующей в восприятии эмоций окружающих, понимании их действий и подражании таким действиям, в нарушении социального поведения у детей с расстройствами аутистического спектра (РАС). Индикаторами активации ЗСМ считают десинхронизацию μ-ритма ЭЭГ. Целью работы было выявить особенности реактивности ЭЭГ в индивидуально определенном частотном диапазоне μ-ритма у детей с РАС дошкольного возраста в ситуациях, предполагающих проявление инструментального, эмоционального и альтруистического помогающего поведения (ПП). В исследовании приняли участие дети 4–7 лет с РАС (n = 26) и их типично развивающиеся сверстники (n = 37). Хотя в большинстве случаев нормотипичные дети демонстрировали более выраженное ПП, различия между группами статистически значимы только для альтруистического ПП (p < 0,01) и приближаются к значимому уровню для ситуации комплексного альтруистического и эмоционального ПП (p = 0,09). Оценка индексов реактивности μ-ритма показала, что при выполнении задания на комплексное альтруистическое и эмоциональное ПП этот показатель статистически значимо ниже у детей с РАС в центральных отведениях левого и правого полушарий, а также в теменном отведении правого полушария (C3: p = 0,02; C4: p = 0,03; P4: p = 0,03). Предполагается, что обнаруженные особенности являются следствием нарушения функционирования ЗСМ, а также нисходящей регуляции к ЗСМ со стороны префронтальной коры и других областей неокортекса. Полученные данные могут быть использованы при разработке протоколов тренингов биологической обратной связи по ЭЭГ для детей с РАС.

Ключевые слова: дети, аутизм, ЭЭГ, μ-ритм, система зеркальных нейронов, просоциальное поведение

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The state of physical, spiritual and social well-being, which is part of the concept of "health", is closely related to the ability to participate, as a member of society, in pro-social behavior, help others and, in turn, receive help and support from them. The terms "prosocial" and "helping behavior" (HB) are usually defined as voluntary actions in response to the needs of others and aimed at their benefit [1]. Autism spectrum disorders (ASD) severely handicap the ability to adequately engage in social interaction [2–4]. This manifestation of the said disorders forms one of the barriers faced by children with ASD on the way to becoming a fully-fledged member of the society. Therefore, analysis of the underlying neurophysiological mechanisms is an urgent task. Currently, there is a number of ASD cause hypotheses being discussed. One of them points to the abnormalities in pruning of synaptic terminals peculiar to the people with ASD, which makes the number of axons and synapses atypical. As a result, the quantity of local connections within the microareas of the cerebral cortex is excessive, while functional interaction with remote regions, such as the frontal and parietal areas of the neocortex, is insufficient, which is confirmed by the EEG rhythms coherence analysis [5]. According to another hypothesis, it is the functional deficiency of the nicotinic branch of the cholinergic modulating system that disbalances excitation and inhibition, hinders attention switching and modulation of sensitivity to stimuli of various modalities in children with ASD [6]. Another hypothesis appeared about twenty years ago. It was assumed that ASD disturbs functioning of the mirror neuron system (MNS), which is involved in the perception of the emotions of others, understanding their actions and imitating them. Neocortical regions bilateral in the inferior parietal lobe and ventral premotor cortex are the core of the MNS in a human brain [7–9]. In the context of studying the features of development of prosocial behavior in children with ASD, it is especially important to investigate the last hypothesis, since helping others requires understanding of the goals of their actions, perception of emotions and evaluation of their mental states, as well as adoption of certain methods of HB [10].

According to some data, desynchronization (suppression) of the sensorimotor mu rhythm (a kind of α-activity) indicates activation of the MNS [8, 10, 11]; it is most pronounced over the central areas of the cortex. Therefore, it is possible to study the features of MNS functioning, including in children with ASD, with the help of EEG. Authors of one of the first works in this field found that, unlike the normal group subjects, adults and children with ASD did not have their mu rhythm suppressed when watching videos of biological movements (hand movements) [12]. Based on these results, they formulated the broken mirror hypothesis.

A number of later studies that analyzed EEG of the subjects that were shown such movements did not reveal differences in mu rhythm modulation in people with ASD and normal group individuals [13–15]. However, a study that used recordings of emotionally colored social movements and movements of inanimate objects as stimuli has registered a significantly stronger reaction (mu rhythm desynchronization) thereto in typically developing children aged 7–15 years, while their peers with ASD did not react, i.e. there were no differences in desynchronization of the mu rhythm upon presentation of these stimuli [16]. Performing a task that required recognition of instruments and pantomiming actions therewith, children with ASD (ages 8–13) have shown a decrease in the mu rhythm modulation power compared with children of the normal group [17]. We have also revealed differences in reactivity of EEG-registered sensorimotor rhythms between 5–10-year old children with ASD and their normally developing peers in situations of observation, imitation and auditory perception of movements of a hand holding a computer mouse. In the control group, we registered desynchronization of mu and beta rhythms when the participants were imitating movements and perceiving shifts of the mouse by hear, while in children with ASD, such a reaction in a similar situation was either absent or the mu rhythm synchronized [18].

The analysis of such contradictions has changed the "broken mirror hypothesis" and translated into an integrated model of social modulation of MNS reactions from other areas of the neocortex. The model suggests that mirror neurons process the visuomotor properties of the actions observed, while the medial prefrontal cortex controls the activity of the MNS depending on the social significance and context of the situation. The symptoms of the ASD manifest because of the abnormalities in the downward regulation of the MNS and not within this system. As a result, the MNS' reaction to simple movements does not differ in people with ASD and neurotypical individuals. However, complex actions, emotionally colored and social stimuli are often processed inadequately [8, 19, 20]. The model enabled development of training routines using biofeedback (BFB) by EEG, which proved effective to a degree in correcting manifestations of the ASD [21]. The routines are based on computer games and include tasks designed to trigger synchronization and desynchronization of the mu rhythm depending on the game context, which enables restructuring of connections between the core of the MNS and other areas of the neocortex.

Previous studies have shown that children with ASD can exhibit prosocial behavior in response to the needs of others, although to a lesser extent than their normally developing peers [22]. They engage in HB when situations require: instrumental helping, i.e. assisting others in executing a purposeful action; empathic help (comforting), which involves response to the emotional needs of another person and verbal or physical support of comforting nature; altruistic help, when children share resources if another person lacks them. To the best of our knowledge, the reactivity of EEG-registered mu rhythm associated with HB demonstrated by children with ASD was not measured and compared to the similar indicator in their healthy peers previously. Such a study would clarify the possible effect MNS dysfunction has on the organization of complex prosocial behavior in children with autism, and could also help develop new corrective methods involving EEG biofeedback sessions for children with ASD. Since the degree of desynchronization of the mu rhythm can be assessed incorrectly because of the partial superposition of the occipital alpha rhythm, the frequency of which is adjacent [10], it is advisable to identify individual mu rhythm range when the child makes independent movements. In this connection, the purpose of our study was to identify the features of EEG reactivity in the individual mu rhythm frequency range in preschoolers with ASD in situations requiring rendering of various types of assistance to another person.

METHODS

Sample characteristics

The study was carried out at the Scientific Equipment Shared Use Center "Experimental Physiology and Biophysics" of Vernandsky Crimean Federal University. It involved 64 right-handed children aged 4–7 years (mean age 5.7 ± 1.2 years), including 26 children with ASD (20 boys and 6 girls) and 38 typically developing children (the comparison group, 21 boys and 17 girls). Age-wise distribution of children with ASD was as follows: 4 years old — 6 people, 5 years old — 6 people, 6 years old — 8 people, 7 years old — 6 people. Similar distribution in the comparison group: 4 years old — 10, 5 years old — 8, 6 years old — 10, 7 years old — 10 people. The age distribution of children with ASD and normally developing children in these groups was similar. There were no significant differences in the mean age of all children with ASD and normally developing children (5.9 ± 1.2 and 5.5 ± 1.2, respectively, p = 0.17). The ASD group included children diagnosed with "childhood autism" (F84.0 in ICD-10) or "autism spectrum disorder with

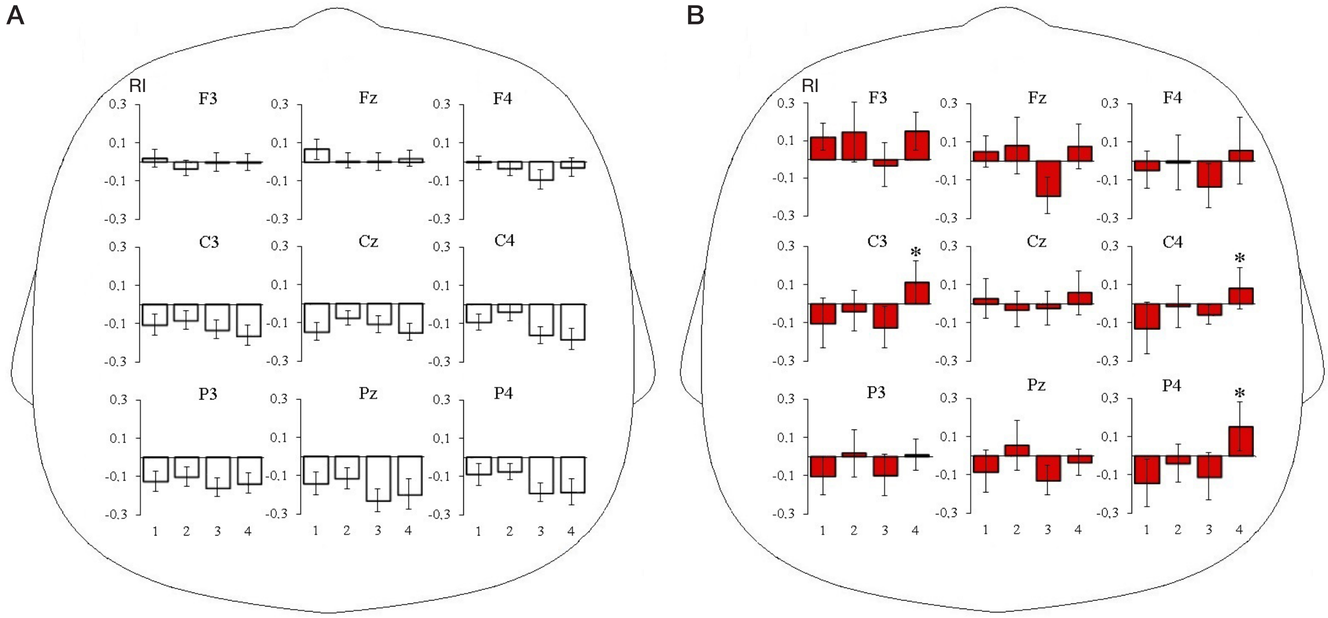

Fig. 1. Reactivity indices (RI) of the EEG-registered mu rhythm in typically developing children (**A**; *white columns*) and children with ASD (**B**; *red columns*) in situations of helping behavior. 1 — IHB, 2 — EHB, 3 — AHB, 4 — AEHB. Mean values and standard errors of means are given. Differences in reactivity indices in children of two groups: * — $p < 0.05$

intellectual disability and functional language impairment" (6A02.3 in ICD-11). The criteria for inclusion in comparison group were a sufficient level of cognitive development (IQ 90 to 120 scored in WPPSI and WISC variants of the Wechsler scale) and absence of chronic diseases of the nervous system. The inclusion criteria for both groups: normal levels of vision and hearing; preferred right-handedness.

Assessment of intensity of prosocial behavior

Four experimental situations were designed to analyze the level of HB intensity. The switch to the next situation was made when the child has completed the task or if he/she did not provide assistance within 50 seconds.

1. Instrumental helping behavior (IHB) task designed around the methods suggested by F.Warneken, M.Tomasello [23]. A box was placed on the table in front of the child; it had a small hole at the top, its side facing the child was open. Experimenter put a mug on the box, and, as if stirring tea in it, "accidentally" dropped the spoon into the hole on the top side of the box. The child was expected to help retrieve the spoon and give it to the experimenter.

2. Empathic helping behavior (EHB) task employing the clipboard [24]. Experimenter, as if by accident, pressed his finger with a paper clip, exclaimed "Oh!" and demonstrated that he was in pain (made a sad face, rubbed his finger, sighed and groaned). The analyzed result was whether the child calmed the experimenter (touched his hand, voiced concern about him, asked the parent to help the experimenter etc.).

3. Altruistic helping behavior (AHB) task based on the "unequal treat" approach [22]. Experimenter took out two transparent containers, one for himself and one for the child, and said, "Look what I have." The experimenter's container was empty, and the child's had four cookies in it. In various ways, the experimenter showed that he did not have cookies while the child did; he made a sad face, extended his hand palm up in a demanding gesture. The registered outcome was whether the child shared the cookies or not.

4. A complex task triggering altruistic and empathic helping behavior (AEHB) [25]. First, the experimenter played with the child using two teddy bears, one of which had a paw held with a Velcro band. After several minutes of play, this paw fell off, and the experimenter made a sad face. Next, the signals that he needed help were becoming more and more obvious; for example, the experimenter said: "The paw fell off! I cannot play with my bear now!" The assessed result was the intensity of HB demonstrated by the child (simple calming, offering his toy).

The experiments were recorded with a camera. The intensity of HB was assessed on a 10-point scale based on the analysis of the recording and the criteria described earlier [22–25] (did the child offer help and how quickly, whether this required more and more insistent stimuli from the experimenter).

EEG registration and analysis

We used the Neuron-Spectrum-5 electroencephalograph (Neurosoft; Russia) monopolarly from 19 leads, using the standard 10–20% pattern (reference from combined ear electrodes), the signal bandwidth of 0.5–30.0 Hz, sampling rate of 250 Hz. EEG and video recording of experimental situations were synchronized.

For the background situation, we registered EEG (50 seconds) when the participant's eyes were open and he/she was stably paying attention to the recording of a rotating ball. For the purpose of analysis of changes in the mu rhythm associated with prosocial behavior, we selected such spans (from all four experimental situations) when the child sat motionless, observed the actions of the adult and made a decision to provide assistance of this or that nature. The duration of such recordings depended on the time after which the child provided assistance and did not exceed 50 s.

We used WinEEG software (Mizar, Russia) to process the EEG data. The independent components analysis built into the program (with additional visual recording quality control interface) enabled removal of artifacts. The EEG of one child in the comparison group and seven children with ASD contained a large number of artifacts because the children moved excessively; these were excluded from further statistical processing. The artifact-free EEG segments were divided into 2-second epochs. The segments were subjected to fast Fourier

Table. Results of ANOVA of differences in the mu rhythm reactivity, children developing normally and children with ASD

Reactivity indices	GROUP			LOCUS			GROUP × LOCUS		
	F1.54	*p*	η2	F8.432	*p*	η2	F8.432	*p*	η2
RI IHB	0.1	0.74	0.002	3.64	0.0004	0.06	0.93	0.49	0.02
RI EHB	0.95	0.33	0.02	1.43	0.18	0.03	0.71	0.68	0.01
RI AHB	0.09	0.77	0.002	1.52	0.15	0.03	1.47	0.17	0.03
RI AEHB	4.24	0.04	0.08	1.9	0.06	0.04	1.16	0.32	0.02

transformation with 4 second epochs and 50% epoch overlap. The μ-rhythm amplitudes were analyzed at nine loci: frontal (F3, Fz, F4), central (C3, Cz, C4), and parietal (P3, Pz, P4). These regions were chosen as areas of interest based on the literature data, which advise analyzing the mu rhythm in children not only in the central but also in the frontal and parietal regions [26]. The amplitude of the EEG-registered mu rhythm was calculated in the frequency ranges individual to each subject; these ranges were determined based on the analysis of differences in the EEG power spectra in the C3 lead when the child was motionless and when he/she performed movements (desynchronization reaction) [27].

Statistical data processing

We used STATISTICA 12.0 software (StatSoft Inc.; USA) to perform statistical analysis of the data obtained. To assess differences in the intensity of various elements of prosocial behavior, we used the Mann–Whitney U test. The features of EEG reactivity in children with ASD were identified through calculation of the reactivity indices (RI) for the mu rhythm, which was done using the formula RI = ln (B / A) [13], where B is the amplitude of the rhythm in the experimental situation and A is the amplitude of the rhythm in the background situation. Logarithming enabled normalization of distribution. The values of RI > 0 mark an increase in the rhythm's amplitude in the experimental situation compared to the background level (synchronization), and RI < 0 indicates a decrease in the amplitude of the rhythm (desynchronization). The differences in RI of the mu rhythm were assessed with the help of repeated measures ANOVA. We have also assessed the influence of GROUP, an external participant-related factor (two levels: children with ASD and the comparison group) and LOCUS, and internal participant-related factor (nine EEG leads) by the 2 × 9 pattern. Linear contrasts were used to calculate significance of differences in the analyzed RI parameters relative to each of the nine EEG leads. Median and interquartile range allowed preparing description of distributions differing from the norm (prosocial behavior indicators). For the normal data distribution cases (logarithmic values of the RI), we used mean and standard error of the mean. The differences were considered statistically significant at $p < 0.05$, but because of the small sample size we also included "unshaped" differences, i.e. those already visible but not yet fully formed ($p < 0.10$).

Gender was not considered as an independent variable due to the predominance of male participants in the ASD group.

RESULTS

The features of helping behavior indicators in children with ASD

The assessment of intensity of elements of prosocial behavior in the comparison group was as follows: the IHB indicator was 7.5 (2; 10), the EHB was 1.5 (0; 3), the AHB was 6.0 (4; 10), the AEHB was 8, 5 (4; 10) points. In children with ASD, IHB was 9.0 (1; 10), EHB — 0.0 (0; 3), AHB — 2.5 (0; 5), AEHB — 3.0 (0; 10) points. Although in most cases, HB was more intense in the group of normally developing children, the differences between the groups were significant only for AHB situation (Z = 2.61, $p < 0.01$), and for the AEHB it approaches significance (Z = 1.65, $p = 0.09$).

Features of the EEG-registered reactivity in the mu rhythm frequency range in children with ASD

The RI of the comparison group children in all experimental situations took negative values for central and parietal leads, which indicates EEG desynchronization in the mu rhythm range (Fig. 1A). In the frontal leads, the reactivity of the mu rhythm was insignificant. In children with ASD, the RI values in all analyzed leads took both positive and negative values, which indicates both desynchronization and synchronization of the EEG in the mu rhythm range (Fig. 1B).

The results of ANOVA of differences in the mu rhythm reactivity in children with ASD and the comparison group indicate a statistically significant effect the GROUP factor has on the AEHB task (Table). In this experimental situation, comparison group children had decreasing amplitudes in all leads except for Fz, and in the ASD group the individual mu rhythm amplitude was increasing except for the Pz locus (Figure). The linear contrasts method allowed concluding that the differences in the mu rhythm's RI in the two groups of children reached the level of statistical significance in the central leads of left and right hemispheres, as well as in the parietal lead of the right hemisphere (C3: $p = 0.02$; C4: $p = 0.03$; P4: $p = 0.03$). There were significant differences found between the groups for the IHB, EHB and AEHB tasks.

DISCUSSION

Same as other researchers [22, 28], we have found that despite marked social impairments, children with ASD act prosocially in response to the needs of others and may do so in situations that require different types of assistance. They have shown greater intensity of HB in an IHB situation than children of the comparison group. The possible reason behind this is the desire to restore order, which is inherent in children with ASD [29]. However, in the AHB and AEHB situations, children with ASD are less willing to help. For the participants of the study, the most difficult was the AEHB situation: while playing, the children had to evaluate what was happening, perceive the affectively colored non-verbal and verbal signals by the experimenter, provide him with emotional assistance or even share an attractive toy. In this situation, children with ASD not only exhibited less intense HB but also shown significant differences from the children of the comparison group in the EEG pattern.

In situations requiring assistance (including the AEHB situation), normally developing children have shown EEG desynchronization in the frequency range of the mu rhythm in the central and parietal leads. In children with ASD, such desynchronization was not registered in these situations. As was already noted, desynchronization of the mu rhythm is regarded as an indicator of the MNS activation [10, 11]. Thus, the absence of mu rhythm depression in children with ASD when solving the AEHB task may indicate a lower degree of activation of the MNS, which manifests in the most difficult social situation. The results, which show that the RI in the groups differ greatest in the AEHB situation, are consistent with the so-called integrated model [8], which is based on the modulation of responses of MNS nerve cells from the prefrontal cortex and other areas of the neocortex, depending on the context of the situation.

Difficulties with switching attention in a complex social situation may also be the cause of the altered reactions of the MNS. According to some authors, children with autism have the functioning of the ventral attention module disrupted, which includes the temporo-parietal junction, the dorsal third of the superior temporal gyrus, and a number of areas of the ventral frontal cortex. This module is responsible for releasing attention and refocusing on the new, potentially important stimuli [2, 6]. In this regard, it can be assumed that the weak attention switching ability demonstrated by children with ASD (e.g., from focusing on their own game with an attractive toy to the actions and manifestations of emotions of another person) prevents adequate activation of the MNS, since the actions of another person are simply out of the child's focus.

The results of this study indicate possible impairments in the functioning of the MNS and neocortical regions that modulate the activity of the system in children with ASD in difficult social situations that require altruistic and emotional HB. The identified features of the EEG-registered mu rhythm reactivity may be useful for the development of the new biofeedback session protocols designed to adjust the development of children with ASD. For such sessions, it seems promising to use a variety of feedback signals that have an emotional connotation and are included in the social context.

CONCLUSIONS

The results of the study indicate that preschoolers diagnosed with ASD act prosocially in response to the needs of others in situations requiring different types of assistance. However, in complex social situations, they are less inclined to provide altruistic and emotional assistance. In such cases, children with ASD, in contrast to normally developing children, exhibit no desynchronization of the EEG-registered mu rhythm in the central and parietal leads. The identified features of behavior of children with ASD can be used in corrective work, including the EEG-enabled biofeedback sessions.

References

1. Eisenberg N, Fabes RA, Spinrad T. Prosocial development. In: Eisenberg N, Damon W, Lerner RM, editors. Handbookof Child Psychology. NewYork: Wiley, 2006; 646–718.
2. Pereverzeva DS, Gorbachevskaya NL. Nejrobiologicheskie markery rannih stadij rasstrojstv autisticheskogo spektra. Zhurnal vysshej nervnoj deyatel'nosti im. I. P. Pavlova. 2016; 66 (3): 289–301. DOI: 10.7868/S0044467716030102. Russian.
3. Bozhkova ED, Balandina OV, Konovalov AA. Rasstrojstva autisticheskogo spektra: sovremennoe sostoyanie problemy (obzor). Sovremennye tehnologii v medicine. 2020; 12 (2): 111–20. DOI: 10.17691/stm2020.12.2.14. Russian.
4. Salimova KR. Nejrofiziologicheskie korrelyaty narusheniya razvitiya pri rasstrojstvah autisticheskogo spektra (RAS). Uspehi sovremennoj biologii. 2021; 141 (6): 557–66. DOI 10.31857/S0042132421060065. Russian.
5. Ippolito G, Bertaccini R, Tarasi L, Di Gregorio F, Trajkovic J, Battaglia S, et al. The role of alpha oscillations among the main neuropsychiatric disorders in the adult and developing human brain: evidence from the last 10 years of research. Biomedicines. 2022; 10 (12): 3189. DOI: 10.3390/biomedicines10123189.
6. Orekhova EV, Stroganova TA. Arousal and attention re-orienting in autism spectrum disorders: evidence from auditory event-related potentials. Front Hum Neurosci. 2014; 8: 34. DOI: 10.3389/fnhum.2014.00034.
7. Lebedeva NN, Zufman AI, Mal'cev VYu. Sistema zerkal'nyh nejronov mozga: klyuch k obucheniyu, formirovaniyu lichnosti i ponimaniyu chuzhogo soznaniya. Uspehi fiziologicheskih nauk. 2017; 48 (4): 16–28. Russian.
8. Yates L, Hobson H. Continuing to look in the mirror: A review of neuroscientific evidence for the broken mirror hypothesis, EP-M model and STORM model of autism spectrum conditions. Autism. 2020; 24 (8): 1945–59. DOI: 10.1177/1362361320936945.
9. Heyes C, Catmur C. What Happened to Mirror Neurons? Perspect Psychol Sci. 2022; 17 (1): 153–68. DOI: 10.1177/1745691621990638.
10. Hobson HM, Bishop DVM. The interpretation of mu suppression as an index of mirror neuron activity: past, present and future. Review R Soc Open Sci. 2017; 4 (3): 160662. DOI: 10.1098/rsos.160662.
11. Larionova E. V., Garah Zh. V., Zajceva Yu. S. Myu-ritm v sovremennyh issledovaniyah: teoreticheskie i metodologicheskie aspekty. Zhurnal vysshej nervnoj deyatel'nosti im. I. P. Pavlova. 2022; 72 (1): 11–35. DOI: 10.31857/S0044467722010051. Russian.
12. Oberman LM, Hubbard EM, McCleery JP, Altschuler EL, Ramachandran VS, Pineda JA. EEG evidence for mirror neuron dysfunction in autism spectrum disorders. Brain Res Cogn Brain Res. 2005; 24 (2): 190–8. DOI: 10.1016/j.cogbrainres.2005.01.014.
13. Raymaekers R, Wiersema JR, Roeyers H. EEG study of the mirror neuron system in children with high functioning autism. Brain Res. 2009; 1304: 113–21. DOI: 10.1016/j.brainres.2009.09.068.
14. Fan YT, Decety J, Yang CY, Liu JL, Cheng Y. Unbroken mirror neurons in autism spectrum disorders. J Child Psychol Psychiatry. 2010; 51 (9): 981–8. DOI: 10.1111/j.1469-7610.2010.02269.x.
15. Sotoodeh MS, Taheri-Torbati H, Sohrabi M, Ghoshuni M. Perception of biological motions is preserved in people with autism spectrum disorder: electrophysiological and behavioural evidences. J Intellect Disabil Res. 2019; 63 (1): 72–84. DOI: 10.1111/jir.12565.
16. Hudac CM, Kresse A, Aaronson B, DesChamps TD, Webb SJ, Bernier RA. Modulation of mu attenuation to social stimuli in children and adults with 16p11.2 deletions and duplications. J Neurodev Disord. 2015; 7 (1): 25. DOI: 10.1186/s11689-015-9118-5.
17. Ewen JB, Lakshmanan BM, Pillai AS, McAuliffe D, Nettles C, Hallett M, et al. Decreased modulation of EEG oscillations in high-functioning autism during a motor control task. Front Hum Neurosci. 2016; 10: 198. DOI: 10.3389/fnhum.2016.00198.
18. Kaida AI, Eismont EV, Mikhailova AA, Pavlenko VB. EEG sensorimotor rhythms in children with autism spectrum disorders. Bulletin of RSMU. 2020; (5): 70–6. DOI: 10.24075/brsmu.2020.055.
19. Hamilton AF. Reflecting on the mirror neuron system in autism: a systematic review of current theories. Dev Cogn Neurosci. 2013; 3: 91–105. DOI: 10.1016/j.dcn.2012.09.008.
20. Dumas G, Soussignan R, Hugueville L, Martinerie J, Nadel J. Revisiting mu suppression in autism spectrum disorder. Brain Res. 2014; 1585: 108–19. DOI: 10.1016/j.brainres.2014.08.035.
21. Friedrich EV, Sivanathan A, Lim T, Suttie N, Louchart S, Pillen S, et al. An effective neurofeedback intervention to improve social

interactions in children with Autism Spectrum Disorder. J Autism Dev Disord. 2015; 45 (12): 4084–100. DOI: 10.1007/s10803-015-2523-5.

22. Dunfield KA, Best LJ, Kelley EA, Kuhlmeier VA. Motivating moral behavior: helping, sharing, and comforting in young children with autism spectrum disorder. Front Psychol. 2019; 10: 25. DOI: 10.3389/fpsyg.2019.00025.
23. Warneken F, Tomasello M. Altruistic helping in human infants and young chimpanzees. Science. 2006; 311 (5765): 1301–3. DOI: 10.1126/science.1121448.
24. Paulus M, Kühn-Popp N, Licata M, Sodian B, Meinhardt J. Neural correlates of prosocial behavior in infancy: different neurophysiological mechanisms support the emergence of helping and comforting. Neuroimage. 2013; 66: 522–30. DOI: 10.1016/j.neuroimage.2012.10.041.
25. Kärtner J, Schuhmacher N, Collard J. Socio-cognitive influences on the domain-specificity of prosocial behavior in the second year. Infant Behav Dev. 2014; 37 (4): 665–75. DOI: 10.1016/j.infbeh.2014.08.004.
26. Marshall PJ, Young T, Meltzoff AN. Neural correlates of action observation and execution in 14-month-old infants: An event-related EEG desynchronization study. Dev Sci. 2011; 14 (3): 474–80. DOI: 10.1111/j.1467-7687.2010.00991.x.
27. Mihajlova AA, Orehova LS, Dyagileva YuO, Muhtarimova TI, Pavlenko VB. Reaktivnost' myu-ritma EhEhG pri nablyudenii i vypolnenii dejstvij u detej rannego vozrasta, imeyushhih raznyj uroven' razvitiya receptivnoj rechi. Zhurnal vysshej nervnoj deyatel'nosti im. I.P. Pavlova. 2020; 70 (3): 422–32. DOI: 10.31857/S0044467720030077. Russian.
28. Wang X, Auyeung B, Pan N, Lin LZ, Chen Q, Chen JJ, et al. Empathy, Theory of Mind, and prosocial behaviors in autistic children. Front Psychiatry. 2022; 13:844578. DOI: 10.3389/fpsyt.2022.844578.
29. Paulus M, Rosal-Grifoll B. Helping and sharing in preschool children with autism. Exp Brain Res. 2017; 235 (7): 2081–8. DOI: 10.1007/s00221-017-4947-y.

Литература

1. Eisenberg N, Fabes RA, Spinrad T. Prosocial development. In: Eisenberg N, Damon W, Lerner RM, editors. Handbookof Child Psychology. NewYork: Wiley, 2006; 646–718.
2. Переверзева Д. С., Горбачевская Н. Л. Нейробиологические маркеры ранних стадий расстройств аутистического спектра. Журнал высшей нервной деятельности им. И. П. Павлова. 2016; 66 (3): 289–301. DOI: 10.7868/S0044467716030102.
3. Божкова Е. Д., Баландина О. В., Коновалов А. А. Расстройства аутистического спектра: современное состояние проблемы (обзор). Современные технологии в медицине. 2020; 12 (2): 111–20. DOI: 10.17691/stm2020.12.2.14.
4. Салимова К. Р. Нейрофизиологические корреляты нарушения развития при расстройствах аутистического спектра (РАС). Успехи современной биологии. 2021; 141 (6): 557–66. DOI 10.31857/S0042132421060065.
5. Ippolito G, Bertaccini R, Tarasi L, Di Gregorio F, Trajkovic J, Battaglia S, et al. The role of alpha oscillations among the main neuropsychiatric disorders in the adult and developing human brain: evidence from the last 10 years of research. Biomedicines. 2022; 10 (12): 3189. DOI: 10.3390/biomedicines10123189.
6. Orekhova EV, Stroganova TA. Arousal and attention re-orienting in autism spectrum disorders: evidence from auditory event-related potentials. Front Hum Neurosci. 2014; 8: 34. DOI: 10.3389/fnhum.2014.00034.
7. Лебедева Н. Н., Зуфман А. И., Мальцев В. Ю. Система зеркальных нейронов мозга: ключ к обучению, формированию личности и пониманию чужого сознания. Успехи физиологических наук. 2017; 48 (4): 16–28.
8. Yates L, Hobson H. Continuing to look in the mirror: A review of neuroscientific evidence for the broken mirror hypothesis, EP-M model and STORM model of autism spectrum conditions. Autism. 2020; 24 (8): 1945–59. DOI: 10.1177/1362361320936945.
9. Heyes C, Catmur C. What Happened to Mirror Neurons? Perspect Psychol Sci. 2022; 17 (1): 153–68. DOI: 10.1177/1745691621990638.
10. Hobson HM, Bishop DVM. The interpretation of mu suppression as an index of mirror neuron activity: past, present and future. Review R Soc Open Sci. 2017; 4 (3): 160662. DOI: 10.1098/rsos.160662.
11. Ларионова Е. В., Гарах Ж. В., Зайцева Ю. С. Мю-ритм в современных исследованиях: теоретические и методологические аспекты. Журнал высшей нервной деятельности им. И. П. Павлова. 2022; 72 (1): 11–35. DOI: 10.31857/S0044467722010051.
12. Oberman LM, Hubbard EM, McCleery JP, Altschuler EL, Ramachandran VS, Pineda JA. EEG evidence for mirror neuron dysfunction in autism spectrum disorders. Brain Res Cogn Brain Res. 2005; 24 (2): 190–8. DOI: 10.1016/j.cogbrainres.2005.01.014.
13. Raymaekers R, Wiersema JR, Roeyers H. EEG study of the mirror neuron system in children with high functioning autism. Brain Res. 2009; 1304: 113–21. DOI: 10.1016/j.brainres.2009.09.068.
14. Fan YT, Decety J, Yang CY, Liu JL, Cheng Y. Unbroken mirror neurons in autism spectrum disorders. J Child Psychol Psychiatry. 2010; 51 (9): 981–8. DOI: 10.1111/j.1469-7610.2010.02269.x.
15. Sotoodeh MS, Taheri-Torbati H, Sohrabi M, Ghoshuni M. Perception of biological motions is preserved in people with autism spectrum disorder: electrophysiological and behavioural evidences. J Intellect Disabil Res. 2019; 63 (1): 72–84. DOI: 10.1111/jir.12565.
16. Hudac CM, Kresse A, Aaronson B, DesChamps TD, Webb SJ, Bernier RA. Modulation of mu attenuation to social stimuli in children and adults with 16p11.2 deletions and duplications. J Neurodev Disord. 2015; 7 (1): 25. DOI: 10.1186/s11689-015-9118-5.
17. Ewen JB, Lakshmanan BM, Pillai AS, McAuliffe D, Nettles C, Hallett M, et al. Decreased modulation of EEG oscillations in high-functioning autism during a motor control task. Front Hum Neurosci. 2016; 10: 198. DOI: 10.3389/fnhum.2016.00198.
18. Кайда А. И., Эйсмонт Е. В., Михайлова А. А., Павленко В. Б. Сенсомоторные ритмы ЭЭГ у детей с расстройствами аутистического спектра. Вестник РГМУ. 2020; (5): 74–81. DOI: 10.24075/vrgmu.2020.055.
19. Hamilton AF. Reflecting on the mirror neuron system in autism: a systematic review of current theories. Dev Cogn Neurosci. 2013; 3: 91–105. DOI: 10.1016/j.dcn.2012.09.008.
20. Dumas G, Soussignan R, Hugueville L, Martinerie J, Nadel J. Revisiting mu suppression in autism spectrum disorder. Brain Res. 2014; 1585: 108–19. DOI: 10.1016/j.brainres.2014.08.035.
21. Friedrich EV, Sivanathan A, Lim T, Suttie N, Louchart S, Pillen S, et al. An effective neurofeedback intervention to improve social interactions in children with Autism Spectrum Disorder. J Autism Dev Disord. 2015; 45 (12): 4084–100. DOI: 10.1007/s10803-015-2523-5.
22. Dunfield KA, Best LJ, Kelley EA, Kuhlmeier VA. Motivating moral behavior: helping, sharing, and comforting in young children with autism spectrum disorder. Front Psychol. 2019; 10: 25. DOI: 10.3389/fpsyg.2019.00025.
23. Warneken F, Tomasello M. Altruistic helping in human infants and young chimpanzees. Science. 2006; 311 (5765): 1301–3. DOI: 10.1126/science.1121448.
24. Paulus M, Kühn-Popp N, Licata M, Sodian B, Meinhardt J. Neural correlates of prosocial behavior in infancy: different neurophysiological mechanisms support the emergence of helping and comforting. Neuroimage. 2013; 66: 522–30. DOI: 10.1016/j.neuroimage.2012.10.041.
25. Kärtner J, Schuhmacher N, Collard J. Socio-cognitive influences on the domain-specificity of prosocial behavior in the second year. Infant Behav Dev. 2014; 37 (4): 665–75. DOI: 10.1016/j.infbeh.2014.08.004.
26. Marshall PJ, Young T, Meltzoff AN. Neural correlates of action

observation and execution in 14-month-old infants: An event-related EEG desynchronization study. Dev Sci. 2011; 14 (3): 474–80. DOI: 10.1111/j.1467-7687.2010.00991.x.

27. Михайлова А. А., Орехова Л. С., Дягилева Ю. О., Мухтаримова Т. И., Павленко В. Б. Реактивность мю-ритма ЭЭГ при наблюдении и выполнении действий у детей раннего возраста, имеющих разный уровень развития рецептивной речи. Журнал высшей нервной деятельности им. И. П. Павлова. 2020; 70 (3): 422–32. DOI: 10.31857/S0044467720030077.
28. Wang X, Auyeung B, Pan N, Lin LZ, Chen Q, Chen JJ, et al. Empathy, Theory of Mind, and prosocial behaviors in autistic children. Front Psychiatry. 2022; 13:844578. DOI: 10.3389/fpsyt.2022.844578.
29. Paulus M, Rosal-Grifoll B. Helping and sharing in preschool children with autism. Exp Brain Res. 2017; 235 (7): 2081–8. DOI: 10.1007/s00221-017-4947-y.