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## Introduction

Autism spectrum disorders (ASDs) are widespread dysontogenetic disorders that persist throughout life. In the modern world, the prevalence of ASDs continues to grow. Thus, according to a meta-analysis from 2023, the prevalence of ASDs as a whole was 0.72 % of the population, Asperger's syndrome – 0.13 %, atypical autism and pervasive developmental disorders – 0.18 % (Talentseva et al., 2023). Considerable investment is needed to facilitate and support people with ASDs, and their families are suffering from social instability (Bonis, 2016; Lievore et al., 2023).

ASDs are characterized by the presence of a wide range of mental development and behavioral defects, the main of which are impairments in communication and social interaction, self-regulation, sensory processes, reduction in motivation, psychological and behavioral stereotypes, and poor behavior (Posar & Visconti, 2023).

Several models have been proposed to explain the mechanisms of development of ASDs and attempts have been made to find the key factors causing a wide range of their symptoms. The theoretical importance of this issue is primarily related to the possibility of using autism spectrum disorders as a model to better understand the functioning of the healthy brain. The practical importance is to expand the possibility of helping people with ASDs based on understanding the mechanisms that form the disorders.

The severity of the deficit, especially in the field of social interaction, attracts research attention to this aspect of ASDs. The specificity of the emotional sphere is of particular interest, as its normal functioning is an integral part of social competence. One of the main characteristics of the emotional sphere in ASDs is emotion dysregulation.

Effective social interaction includes the development of a mature system of emotion regulation, including social expression and the ability to control internal states. Emotions

that first arise in response to a situation undergo changes that lead to the optimization of response at the behavioral level and its functional support. Emotions can change intensity, character, and direction (reorienting towards another situation or another aspect of the situation) (Gross & John, 2003). Efficient individual emotion regulation styles predict psychological well-being (Da Costa Dutra et al., 2023). Emotion dysregulation is defined as a lack of control and modulation of the valence, intensity, and expression of emotions (Davico et al., 2022); emotion dysregulation is a factor in many mental disorders (Igra et al., 2023).

Efforts to rehabilitate people with ASDs require the development of emotion regulation skills that increase stress resistance, enable them to enter social interaction situations efficiently (unlike avoiding potentially stressful conditions), gain social experience, and be subjected to correctional influences (Shaffer et al., 2023).

This study aims to systematize modern data on emotion dysregulation in the structure and dynamics of ASDs and to examine the neurophysiological correlates of emotion regulation.

## **Emotion regulation in ASDs**

The immaturity of the emotion regulation system, the reduction in efficient strategies for self-regulation and regulating emotions in others, and the increase in inefficient strategies can be observed at all age stages of the development of children and adults with ASDs (Bradley et al., 2023). Children with ASDs show a decrease in positive emotional expression since childhood when they interact with adults, resulting in negative emotional reactions in parents and reducing their activity in interaction with children, reducing the possibility of developing emotional regulation of children with the help of adults (Jahromi et al., 2013; Sung et al., 2024). In pre-school age, children with ASDs tend to use primitive self-regulatory methods such as emotional outbursts and avoidance of situations that cause unwanted tension (Davico et al., 2022; Taylor et al., 2022). The degree of emotional deregulation already at this age is linked to various distress symptoms, such as sleep disorders (Favole et al., 2023), and also shows strong correlations with social functioning and behavioral disorders (Berkovits et al., 2017). Self-soothing through stereotypical behavior and self-stimulation is common in children and adults with ASDs, especially in situations of social stress; stereotypic behavior that leads to self-harm is generally associated with an increase in other forms of emotion dysfunction (Martnez-González et al., 2022; Lampi et al., 2020). At the same time, neurotypical children can already use attention switching, re-evaluation, rethinking in pre-school; stereotypic behavior, self-stimulation, and self-harm are observed in them very rarely.

In early school age and adolescence, low-functioning autistic children continue to use immature self-regulation strategies, while high-functioning autistic children gradually develop more mature cognitive strategies. However, even high-functioning autistic adolescents and adults have inefficient regulation strategies, including rumination

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(obsessive negative experiences); suppression of thoughts that cause unpleasant emotions, with their subsequent uncontrollable "intrusion", impulsive discharge, catastrophizing, self-blame, dissociation in the context of a decrease in effective strategies (Bruggink et al., 2016; Ilen et al., 2023; Martínez-González et al., 2022; Mazefsky et al., 2014). Adults with ASDs also maintain primitive regulation strategies; mature self-regulation strategies, such as cognitive reappraisal, can not only be reduced, but also less efficient when used (Zaharia et al., 2021).

The research findings also show that the use of emotion regulation strategies is linked to mental health problems in ASDs; greater external (behavior, interpersonal relationships) and internal (well-being, emotional states, and self-esteem) problems are associated with a decline in mature efficient emotion regulation strategies or an increase in emotion dysregulation (Cai et al., 2019; Conner et al., 2020; Bruggink et al., 2016; Mazefsky et al., 2014; Samson et al., 2014).

At the same time, it is reported that an inefficient strategy of thought suppression, combined with a high cognitive reappraisal in a group of adolescents and young adults with ASDs, is associated with relative psychological well-being; this association is not typical in neurotypical development (Pouw et al., 2013a).

A reduction in depression symptoms was observed in boys with ASDs who use avoidance strategies in stressful situations (Pouw et al., 2013b). The reduction of depression symptoms has also been demonstrated with the increase in avoidance strategies, such as in adolescents with ASDs (Cracco et al., 2017). Another characteristic difference between high-functioning autistic adolescents and their neurotypical peers is the maintenance of levels of depression with an increase in efficient emotion regulation strategies and the lack of conviction that emotional experience contributes to solving the problem. The authors believe that attempts to solve problems are less likely to yield results and the accumulation of positive experiences in this group. (Rieffe et al., 2011). Studies of the neurotypical population usually show a direct link between depression and avoidance strategies. Perhaps the inability to cope with stressful situations makes the avoidance strategy adaptive in ASDs under some conditions (at least in the short and medium term). Overall, these results may suggest specific prognostic implications for the severity of various emotional regulation strategies in ASDs.

A mixed group of adolescents with mental disabilities reported a regression to less adaptive strategies for regulating emotions between the ages of 12 and 15, which may be related to physiological changes at this age (Cracco et al., 2017); this requires careful comparison among the age groups.

Emotion regulation indicators are associated with important developmental characteristics in the context of basic ASDs. In a sample of children aged 3 to 12 years, a positive relationship was found between efficient emotion regulation and the age of verbal development, which further emphasizes the role of speech in the development of self-regulation (Nader-Grosbois & Mazzone, 2014). Samson et al. (2014) report a link between

emotion dysregulation and all the main symptoms of autism, including deficiency in social functioning, communication, sensory disorders and, above all, stereotypic behavior; this enables us to consider the phenomenon of dysregulation as a cross-sectional phenomenon in the structure of autism spectrum disorders (Samson et al., 2014). The contribution of almost all autism symptoms to emotion dysregulation, which modulates the development of anxious states, is noted (Swain et al., 2015). Stereotypic and restricted behavior is closely related to rumination, one of the major forms of emotion dysregulation, and some authors consider it to be a cognitive form of stereotyping (Ibrahim et al., 2019). According to other data, ruminative thinking in adult autists is associated with depression, a comorbid disorder similar to a neurotypical population (Williams et al., 2021). The link between high levels of rumination and subsequent external and internal problems in children with ASDs was also shown (Bos et al., 2018). A considerable interest is the mediation by ruminating the relationship between autism symptoms and depression (Keenan et al., 2018). Since symptoms of depression and anxiety are associated with ASDs (Smith & White, 2020), these data draw more attention to the problem of increased rumination in high-functioning ASDs.

There is a question about the characteristics of emotion dysregulation that combine ASDs with other disorders, and specific ones. There is evidence that there are generalized mechanisms of disorders (impairments in excitation processes, affective lability, dysfunction of the prefrontal cortex and amygdala) and more specific aspects (changes in the processing of sensory information, features of cognitive processes, disturbances of social motivation and processing of social information) (Mazefsky et al., 2013).

## **Neurobiological mechanisms of disorders in the emotional sphere and social interactions in ASDs**

A considerable amount of data has been collected on the neurophysiological substrate of ASDs. One of the main research areas is the identification of the specific functions and interactions of brain structures associated with the processing of social information.

The main results were abnormal activity of the prefrontal cortex, the cingulate cortex, the superior temporal sulcus, the amygdala and, less often, the basal ganglia (striatum, caudate nucleus, putamen) and deteriorating interactions between these regions. The activation of these areas and the nature of their connection are related to the lack of recognition of emotional facial expressions (Samaey et al., 2020; Swartz et al., 2013) and the impairment of decision making and self-consciousness in social situations (Chiu et al., 2008; Schulte-Ruether et al., 2011). An atypical modulation of the amygdala on the cerebral cortex is considered a key possible disorder (Sato et al., 2011).

In ASD subjects and their healthy siblings, activity in the superior temporal sulcus, orbitofrontal cortex, and anterior cingulate cortex was reduced compared to control groups when presented with faces with positive emotions (Spencer et al., 2011). Similar

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changes in brain activity during the processing of facial images, but not during the processing of stimuli of another kind, were also shown for children with ASDs and their healthy parents (Dawson et al., 2005). Changes in hippocampal and amygdala volume, and the structure of white brain matter are related to the characteristics of emotion regulation in children at high risk for autism (Ding et al., 2024). These data are of interest to identifying an endophenotype in autism, since changes in activation of certain regions of the brain can be markers of hereditary predisposition to ASDs. The problem of the interaction of factors in the development of ASDs is also raised: How is the development trajectory formed in the presence of multiple interactions in the neurosubstrate?

Experimental models of social interaction in ASDs often involve the presentation of the eye region that generates strong emotional reactions. It has been shown that when instructed to fixate different face regions, patients with ASDs were more likely to lose fixation stability to the eye region. They also showed relatively higher activation of the amygdala when fixated the eye region and relatively lower activation when fixated the mouth region (Kliemann et al., 2010). Another study showed that amygdala activation was modulated when children with ASDs, but not in neurotypical children, fixated the eye region. The specific effect of this stimulus on the activation of the amygdala may indicate that the dysfunctional neuronal dynamics in ASDs can lead to increased negative emotions in response to contact with the eyes and, consequently, to its avoidance (Kliemann et al., 2012). Compared to neurotypical and mentally disabled children with autism, the frontal cortex was more active in response to lateral gaze than in response to direct gaze. This may reflect the decline in social motivation that normally occurs in response to direct gaze (Lauttia et al., 2019).

After describing the mirror neuron system – nerve cells that are excited when another person performs specific actions or display emotions – it was suggested that disturbances in its function contribute to difficulties in social interaction in ASDs. A number of studies confirm abnormal activation in the frontal and parietal lobe regions where the mirror neurons are located when participants with ASDs observed the movements of other people (see review: (Chan & Han, 2020). When the task of voluntary imitating the emotional expressions of the subjects was performed, participants with ASDs, compared to control groups, had lower activations of the caudate nucleus and putamen (Dapretto et al., 2006), indicating the role of subcortical structures in disturbances of imitation processes and their effect on the function of the cortex.

Not all studies confirm the general disfunction of the mirror neuron system in autism. For example, when automatic imitation of facial expressions is recorded under the control of attention to facial stimuli, participants with ASDs have shown results similar to neurotypical participants. However, only they are characterized by no direct relationship between empathy self-assessment and imitation success. The authors conclude that in ASDs, the simple level of motor imitation is not associated with complex cognitive social abilities such as emotion understanding and empathy. (Schulte-Rüther et al., 2013).

In recent years, it has become increasingly popular to study not only areas of the brain that are responsible for immediate responses to stimuli, but also neural systems that are involved in the extra-situational processing of social information and the implementation of self-perception processes. Therefore, ASDs is associated with a number of impairments in the default mode network functions (Harikumar et al. 2021; Padmanabhan et al., 2018).

Another area of ASDs research is related to the study of the functioning of neurotransmitters and hormonal systems. The concentration of impairments in brain regions associated with the behavioral reward system leads to the hypothesis that the dysfunction of the behavioral reward system plays a role in the social behavioral deficit in ASDs (Spence et al., 2011; Vaan de et al., 2020; Dichter, 2012). Dopaminergic deficiency in individuals with ASDs have been noted in a number of studies (see review: (Greene et al., 2019)). Dysfunctions of dopaminergic structures are associated with early developmental disorders of the brainstem (Dadalko & Travers, 2018). Based on the hypothesis of activation by oxytocin and vasopressin of the reinforcement system of social behavior in neurotypical development, Insel & Fernald (2014) suggested that ASDs interferes with the modulation of dopaminergic neurons in the neural attachment system, resulting in a decrease in social motivation. Therefore, social stimuli do not lead to the experience of pleasure. Social motivation is essential to social learning, and its impairments cause great difficulties (Fareri et al., 2008). The deficit in the reinforcement system in ASDs is also considered in the context of a wider range of motivation components, including responses to non-social rewards and a limited range of motivations (Clements et al., 2018).

The search for causes of reduced stress resistance in ASDs has attracted attention to other neurotransmitter systems (Sato et al., 2023). For example, using facial expression recognition tasks, it was shown that acute tryptophan depletion (which leads to a reduction in serotonin synthesis) in participants with ASDs leads to an excessive reduction in response to emotional facial expressions in socio-emotional brain areas (Daly et al., 2012).

Dysfunctions of the hypothalamus-pituitary-adrenal axis and excessive or insufficient cortisol increases in response to new and stressful stimuli are also observed (Spratt et al., 2012). According to other data, children and adults with autism respond to cortisol similar to neurotypical peers, but their levels of cortisol are associated with several symptoms of autism and related disorders, including stereotypic behaviors (Vaan de et al., 2020) and cognitive decline (Ogawa et al., 2017). There is evidence of an increase in cortisol levels in stressful situations with age. This may indicate the existence of secondary disorders that develop in children with ASDs (Schupp et al., 2013).

The characteristics of excitation that arise in response to various types of stimuli are studied by cortical activation indicators, galvanic skin reactions, heart rate, pupillary reactions and startle responses. Several studies have shown an increase and decrease in excitation in response to similar emotional and social stimuli compared to neurotypical individuals, as well as correlations between its changes and individual autism symptoms

and adaptation problems (Anderson et al., 2013; Baker et al., 2018; Dijkhuis et al., 2019; Verneti et al., 2020).

We should note that the data from different studies are often contradictory, which may be associated not only with differences in methodological approaches, but also with the heterogeneity of ASDs and its association with other mental disorders.

## **Neurobiological mechanisms of emotion dysregulation in ASDs**

The neurophysiological substrate of emotion regulation is assumed to be the interacting cortical structures (areas of the prefrontal and frontal cortex, the posterior parietal cortex, and the insular lobe) and the limbic system structures (mainly the amygdala and striatum) (Sato et al., 2023).

The experimental approach to studying emotion regulation usually includes instructions on how to control emotion that arises in response to a stimulus. A study showed that neurotypical children show a significant reduction in activation in the amygdala and insular cortex when they voluntarily reduce their affective reactions to emotogenic images, according to the instructions. At the same time, children with ASDs did not demonstrate similar regulations. In addition, neurotypical participants in the study showed a higher functional connectivity of the amygdala and prefrontal ventrolateral cortex than participants with ASDs, as well as a lower functional connectivity of the amygdala with the orbitalfrontal cortex (Pitskel et al., 2011). Using instructions to increase positive and negative emotions while viewing face images, less activation was seen in the nucleus accumbens, amygdala, and dorsolateral prefrontal cortex of participants with high-functioning ASD than in neurotypical controls (Taylor et al., 2018). When an emotional response was actively modulated according to instructions in social situations, adults with ASDs showed compensatory activation in the dorsolateral prefrontal cortex, but the activation of the nucleus accumbens was reduced, and the compensatory mechanism did not lead to typical modulation of the emotional processing areas (Latinus et al., 2019).

Another type of task proposed includes the presentation of negative stimuli. Under these conditions, participants in the experiment should regulate (improve) their emotional states. In a study in which instructions were given to recognize words that trigger unpleasant emotions, participants with ASDs showed differences in the function of the anterior cingulate cortex, the anterior insular lobe, and dorsolateral prefrontal cortex – areas responsible for regulating emotions in a healthy population (Mazefsky et al., 2020).

Modeling stress situations in young children with autism allowed to describe a delay in the physiological maturation of their emotion regulation system. With the Still-Face Paradigm procedure (Giusti et al., 2018), the emotional reactions of children to a reduction in communication indicators after playing with their mothers were measured.



Children with ASDs, like neurotypical children, showed negative emotions in response to emotionless expressions of their mothers, looked at them longer, but their self-regulation was easier – for example, they sucked the thumb or repeated the same actions, while neurotypical children were able to change attention, etc. Mothers of children with ASDs smile more before and after stress on their children, look at them longer; this may reflect their greater efforts to regulate the child's emotions. It is also noted that in the presence of mothers, only children with ASDs show a reduction in the production of cortisol – i.e., external control leads to a reduction in stress levels; normally, children of the same age can regulate emotions independently (Ostfeld-Etzion et al., 2015). The later maturity of self-regulation indicates the need for longer-term social support. This can further complicate the development of emotional control as positive feedback decreases.

In a number of studies, the conceptual link between emotional regulation disorders and autism symptoms is primarily considered to be caused by autism symptoms that interfere with the acquisition of social experience and the effective co-regulation of emotions with parents (Mills et al., 2022); other authors believe that most of them are disturbances in the functioning of the nervous system that lead to emotional dysregulation, which, in turn, results in the emergence of autism symptoms in different configurations as phenotypic manifestations (Dell'Osso et al., 2023). It is also suggested that there is a general impairment of executive functions that mediates the relationship between ASD symptoms and emotional dysregulation (Costescu et al., 2023). These approaches are not fundamentally contradictory to each other. It can be assumed that a weakened regulatory function based on neuronal substrate impairment interacts with autism symptoms in accordance with the vicious circle principle of mutual influence.

### **Conclusion**

Therefore, disturbances in emotional regulation can be considered as a transdiagnostic factor of ASDs, which has specific characteristics, including the following: (a) decreasing efficient strategies and increasing inefficient ones, (b) large number of immature strategies in older children, adolescents, and adults, (c) stereotyping, including cognitive components, (d) severity of the regulatory strategy associated with avoiding stressful situations, mainly social ones, combined with a reduction in the processing of extra-situational social information, (e) imbalance of components, and (f) differences in the efficiency of specific regulatory mechanisms, compared to neurotypical individuals.

According to most authors, emotion regulation disturbances are associated with the severity of other major symptoms of autism, depression, anxiety, and social maladaptation. Signs of emotion dysregulation are observed in all age groups. Children and adolescents with ASDs have a longer training period of independently implemented and efficient strategies of emotional regulation than their neurotypical peers.

Obviously, both primary physiological mechanisms and secondary disorders that develop in the social environment due to a slower accumulation of social experience and a decrease in the effectiveness of joint emotional regulation with parents contribute



significantly to the development of emotional dysregulation in ASDs. It can be assumed that there is a single neurophysiological basis that combines deficits and/or distortions in the processing of emotional and social signals and emotion regulation disorders. Individual function disorders obviously form a complex system, which, in combination with a specific perception of environmental factors, may clearly form complex trajectories of the development of self-regulatory processes.

The imbalance in excitation and inhibition processes, which leads to a reduction in stress resistance, can be considered as an inferential mechanism complicating emotional regulation in ASD. Another integrated mechanism may be a reduction in general and social motivation caused by dysfunctions in dopaminergic and oxytocin systems.

It is necessary to emphasize the importance of correctional work with secondary factors of emotion dysregulation in ASD. Several cautions in the direct formation of efficient strategies for emotional regulation are associated with a lack of resources to overcome stressful situations in individuals with autism. A significant decrease in avoidance strategies can lead to maladaptation.

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### **Conflict of Interest Information**

The author has no conflicts of interest to declare.

## Eye-tracking for non-symbolic numerosity estimation: A systematic literature review

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### Abstract

**Introduction.** The processing of quantitative information is one of the basic processes that ensures successful interaction with the environment. In one form or another, this ability is found in a large number of biological species. In humans, the specifics of processing quantitative information in different formats are analysed in experimental and correlational studies using various methods and approaches, including eye tracking. Eye tracking makes it possible to follow the mechanisms of the formation of the mental representation of quantity and to evaluate the connection of the non-symbolic "sense of number" with the systems of representation of other visual parameters, such as the size of objects. **Methods.** This paper presents a systematic review of eye tracking studies of non-symbolic numerosity estimation published from 2008 to 2023. A search of Scopus, Web of Science and PubMed databases identified 13 studies. **Results and Discussion.** The research questions, the characteristics of the tasks and stimulus materials used, the characteristics of the sample and the results obtained have been systematised. This review highlights the mechanisms of operation of the numerosity representation system and the characteristics of conducting eye-tracking studies to investigate them.

### Keywords

Nonsymbolic number sense, nonsymbolic numerosity representation, eye tracking, saccades, visual fixations