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Emotional Regulation Deficits and Neurophysiological Mechanisms in Individuals with Autism Spectrum Disorders

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Abstract: In recent years, as the pressure of life has increased, the number of individuals with autism spectrum disorders (ASD) has also risen. Research indicates that individuals with ASD often exhibit significant deficits in emotional regulation. Due to physiological issues, these individuals struggle to manage their emotions effectively, which significantly impacts their daily lives. Understanding the relationship between the emotional regulation deficits of individuals with ASD and their neurophysiological mechanisms is a primary goal in clinical treatment. This paper analyzes the emotional regulation deficits in individuals with ASD and explores the causes from a neurophysiological perspective, providing theoretical support for the clinical treatment of ASD.

1. Introduction

Autism Spectrum Disorder (ASD) is more prevalent among children, with some patients exhibiting neurological developmental issues as early as infancy. According to survey results, the global incidence of ASD in children is approximately 1 in 44. Due to neurological disorders, these children struggle to regulate their emotions, making them more susceptible to external stimuli and significantly impacting their daily lives. Additionally, individuals with ASD are at a higher risk of developing psychological conditions such as anxiety and depression. Some ASD individuals have even experienced safety incidents due to uncontrollable emotions. Therefore, understanding the neurophysiological mechanisms of ASD and improving emotional regulation deficits are crucial for clinical treatment and essential for ensuring the safety and quality of life of individuals with ASD.

2. Overview of autism spectrum disorders

2.1 Definition of autism spectrum disorders

Autism Spectrum Disorder (ASD), also known as 'autism,' is a neurodevelopmental disorder that has seen a rising incidence globally in recent years, particularly among children. The World Health Organization (WHO) identifies three primary symptoms of ASD: social and communication difficulties, restricted and repetitive behaviors, and unusual sensory responses. Social and communication difficulties include minimal verbal interaction, not speaking at all, and not sharing interests or achievements with parents. Restricted and repetitive behaviors include the repetitive arrangement of toys in specific ways, having very narrow or intense interests, and showing a low tolerance for new things.

2.2 Causes of autism spectrum disorders

Research indicates that the causes of autism spectrum disorder (ASD) include both genetic and social factors. Medical researchers have found that among twin pairs with ASD, identical twins have a co-occurrence rate of ASD of at least 70%, whereas fraternal twins have a co-occurrence rate of only 20%. This suggests that genetics is a primary factor in the development of ASD.

Furthermore, medical analysis has revealed that individuals with autism spectrum disorder (ASD) exhibit significant differences in synaptic function-related proteins compared to the general population. For instance, the NRXN1 gene encodes synaptic adhesion molecules, and SHANK3. Abnormalities in these synaptic proteins can limit synaptic transmission, affecting the development of neurons in the brain and leading to neurological disorders that contribute to ASD. In terms of neurotransmitters and neural circuits, individuals with ASD often show abnormalities in neurotransmitter levels, such as excessive or insufficient levels of dopamine, serotonin, glutamate, and glycine, which can lead to an imbalance in the nervous system and emotional regulation issues.

3. Defects in emotion regulation of individuals with autism spectrum disorders

Emotional regulation is an innate human ability that enables individuals to manage their emotional responses in response to stimuli, thereby enhancing, maintaining, or altering their emotional states and helping them better adapt to external stimuli. However, when neurological dysfunction leads to emotional regulation deficits, patients may struggle to maintain their emotions effectively in the face of external stimuli, leading to feelings of loss of control and depression. In severe cases, some patients may exhibit strong aggression, potentially causing safety incidents. Common emotional regulation deficits in individuals with autism spectrum disorders can be categorized into two types: emotional cognitive deficits and emotional interaction deficits.

3.1 Emotional cognitive defects

Emotional cognitive deficits refer to the inability to genuinely express or recognize emotions. For instance, individuals with autism spectrum disorders (ASD) may not be able to convey their happiness through language or behavior when they feel happy. Additionally, when others express emotions, those with ASD may fail to accurately interpret and understand the emotional messages conveyed. Common symptoms of emotional cognitive deficits include a lack of empathy, a weak emotional expression, and rigid ways of expressing emotions, which significantly impact their daily lives [1].

3.2 Emotional interaction defects

Emotional interaction deficits often refer to patients' inability to communicate and interact emotionally with others. Specifically, these patients exhibit extreme and volatile emotions, making it difficult for them to maintain emotional stability. When faced with others expressing their emotions, patients with emotional interaction deficits tend to avoid or ignore the situation, making it challenging for them to engage in social interactions.

4. Neurophysiological mechanism of autism spectrum disorder

4.1 Molecular pathway mechanism of autism spectrum disorder patients

In the medical field, the molecular pathways affecting emotional regulation in autism spectrum disorders (ASD) are categorized into three types: neural development pathways, cell growth and metabolism pathways, and cytokine-related pathways, as illustrated in Figure 1[2]. The Wnt/β-catenin pathway, a crucial component of human developmental pathways, is primarily responsible for neuronal migration, synaptic transmission, and cell differentiation. In the actual development process, some ASD patients have genetic coding issues; for example, the abnormal expression of the risk gene CTNNB1 can lead to emotional regulation deficits, which in turn contribute to ASD. Additionally, the CHD8 gene often acts as a negative regulator in human neuronal development. If the CHD8 gene is missing in the patient's body, it can directly increase the Wnt signal, leading to abnormal activation of the Wnt pathway and emotional regulation deficits.

The cell growth and metabolic pathways associated with autism spectrum disorders in the human body include the MAPK/ERK signaling pathway and the PI3K/AKT signaling pathway. Medical researchers have found through experiments on mice that these pathways play a crucial role in the growth, differentiation, and proliferation of human nerve cells. Typically, these pathways can regulate specific cellular transcription under external stimuli. When the phosphorylated ERK levels in mice are excessively high, the mice not only exhibit symptoms of autism but also show significant abnormalities in immune indicators. Additionally, when [3] removed the cortical neurons from the brains of mice, it was found that the reduction in the number of cortical neurons led to a decrease in the level of insulin-like growth factor 1 receptors on the cell surface of the mouse brain. This resulted in a significant decline in the proliferation response activity of neural progenitor cells within the cell growth and metabolic pathways, while the rate of cell differentiation increased. Consequently, due to abnormal cell differentiation, the mice exhibited cognitive and social dysfunction.

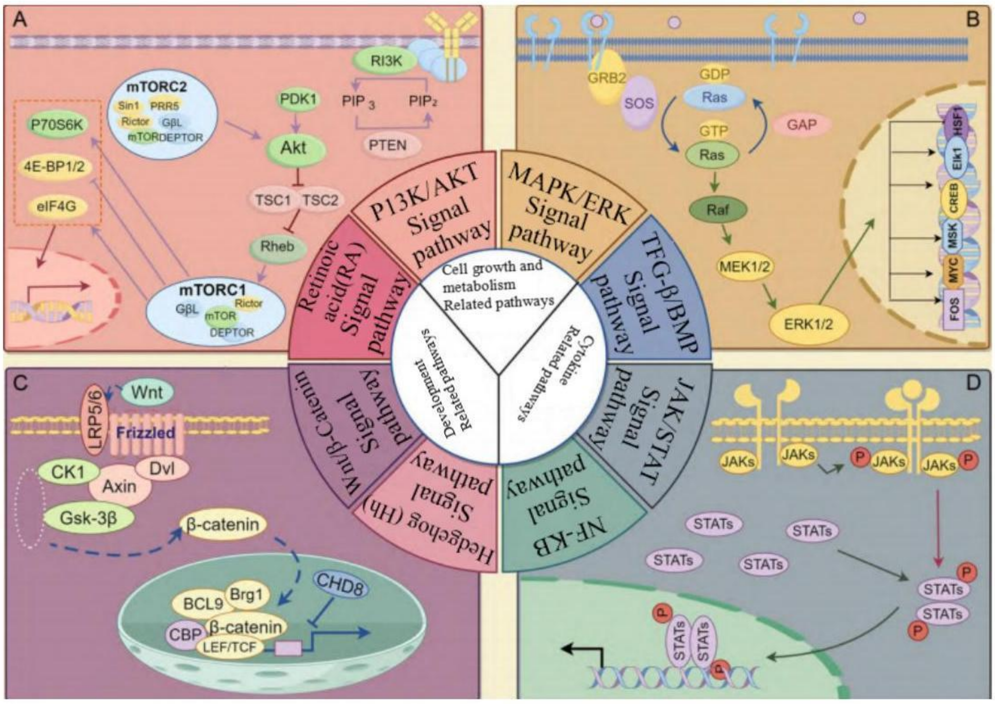

Figure 1: Main molecular pathways in patients with autism spectrum disorders

4.2 Genetic and environmental interaction mechanism of autism spectrum disorder patients

In addition to the molecular and cellular activities within patients, genetic-environment interactions are also a common neurophysiological mechanism that triggers autism spectrum disorders. These interactions can lead to gene mutations and affect normal gene expression, thereby causing autism spectrum disorders. For example, genetic variations in receptor tyrosine kinases are a common factor in autism. Some individuals with autism spectrum disorders have been found to have low levels of vitamin D in their bodies, and the frequency of copy number variations at the 15q11-q13 locus is abnormal. Studies have shown that exposure to heavy metals and polychlorinated biphenyls (PCBs) is associated with these abnormalities. Excessive exposure to high-risk factors can lead to genetic mutations in children, resulting in autism. In 2022, medical researchers [4] discovered that when cells in the human body are stimulated by external factors and produce a stress response, they release a large amount of stress granules. When the level of stress granules in the body reaches a certain threshold, it can easily trigger neurological disorders. Research indicates that many neurological disorders in the medical field are related to stress granules, and autism spectrum disorders are also one of the neurological diseases caused by stress granule disorders. Research on stress granules has provided new insights into the neurophysiological mechanisms of autism spectrum disorders.

In addition to various special media, the occurrence of autism spectrum disorders (ASD) in children can also be elevated when brain inflammation and other diseases occur during their development. This is particularly true for infants and young children, as the brain's nerve cells directly influence brain function. If the child is exposed to external environmental or pathogen stimuli during development, it can lead to abnormal brain development. For example, under inflammatory conditions, the activity of microglia in the brain significantly increases. This over-activation can lead to excessively high levels of pro-inflammatory factors in the brain, damaging other normal brain nerve cells and potentially leading to ASD. Additionally, under inflammatory effects, changes in mitochondrial density and excessive oxidation in the brain can affect brain structure and function, which can also trigger ASD.

4.3 Neuroanatomical analysis of individuals with autism spectrum disorders

Neuroanatomy reveals that [5], the brain structure of individuals with autism spectrum disorder (ASD) differs significantly from that of the general population. MRI studies of ASD patients show that their brains are notably larger than those of the general population, particularly in infants and young children. Research indicates that the enlargement of the brain in ASD patients is often due to excessive neuronal growth, which leads to an increase in synapses and, consequently, a larger brain volume. Additionally, neuroanatomy shows that individuals with ASD often have developmental abnormalities in the frontal lobe and the prefrontal cortex. The frontal lobe, a critical neural substrate in the human brain, is responsible for controlling emotions, cognition, and behavior. Abnormalities in the frontal lobe can affect a patient's cognitive abilities and control, leading to emotional regulation deficits. Besides the neural substrate, the distribution of neurons in the brains of ASD patients also differs significantly from that of the general population. For example, most ASD patients exhibit abnormal neuronal connections, and white matter fiber bundles often fail to maintain normal connections. This results in slower information transmission and processing in ASD patients, making it difficult for them to immediately recognize and respond to emotional cues from others, thereby reducing their social skills and cognitive abilities.

5. Treatment interventions for individuals with autism spectrum disorders

The clinical treatment measures for individuals with autism spectrum disorders are shown in Table 1. At present, the global medical field adopts two kinds of intervention methods for individuals with autism spectrum disorders: drug therapy and non-drug therapy. The specific treatment plans are as follows.

Table 1: Common clinical treatment measures for individuals with autism spectrum disorders

therapeutic measure	therapeutic method	therapeutic regimen
medication	Behavioral and psychological intervention	Cognitive behavioral intervention, social storytelling, music therapy
	Brain stimulation therapy	Invasive brain stimulation therapy, non-invasive brain stimulation therapy
Non-drug treatment	Gene transcription regulation	mTOR,IGF-1
	Epigenetic regulation	Histone deacetylase, histone demethylase
	E/I balance	mGluR5 Antagonists, NMDAR agonists, GABAR agonists
	Neuropeptide regulation	Oxytocin, arginine vasopressin
	immunoregulation	Pioglitazone, intestinal microflora

5.1 Social story intervention

The social story method is a therapeutic approach primarily used in clinical settings to treat psychological disorders. This method involves writing down problem situations that are difficult for individuals with autism to handle or resolve. These situations are then presented to the child through storytelling and narrative techniques. By setting up these scenarios, the method aims to guide the child to adjust their behavior and emotions, enabling them to respond appropriately through learning. The social story method is commonly used to treat infants and young children with neurological and psychological issues, effectively improving emotional and behavioral problems in children with autism. This helps them quickly adapt to life situations and prevent episodes of autism [6].

During the treatment process, it is essential to first design a social story context based on the child's actual situation and allow the child to gain an initial understanding and recognition through reading. After the reading, ask the child about the main content and key points of the social story context to ensure they have fully read and understood it. After the child has read thoroughly, healthcare providers and parents should guide the child to observe the reactions, emotions, and outcomes of different characters in various situations. For example, when a character expresses happiness, parents should guide the child to understand why the character was initially unhappy but suddenly became happy, and what actions can make them happy. This helps the child realize that certain behaviors can make others unhappy while other behaviors can bring happiness. Throughout the reading process, healthcare providers and parents should continuously encourage and praise the child to help them persist in completing the social story context. Once the child understands, similar scenarios can be designed for the child to handle, observing their reactions and changes in regulatory abilities, thereby effectively intervening in the child's emotional regulation.

5.2 Cognitive Behavioral Intervention

Cognitive-behavioral intervention employs the SIERA method, as illustrated in Figure 2. The SIERA method is based on improving the cognitive level of patients, enhancing their self-perception and emotional awareness through cognitive training, thereby effectively improving the self-emotional regulation skills of individuals with autism spectrum disorders (ASD). This therapy is commonly used for adolescents and adults with ASD, who often have a certain level of cognitive ability, understand their condition, and can follow the SIERA method for self-treatment. Application studies have shown that [7] the SIERA method can significantly improve the emotional regulation abilities of individuals with ASD, while also enhancing their cognitive and social skills.

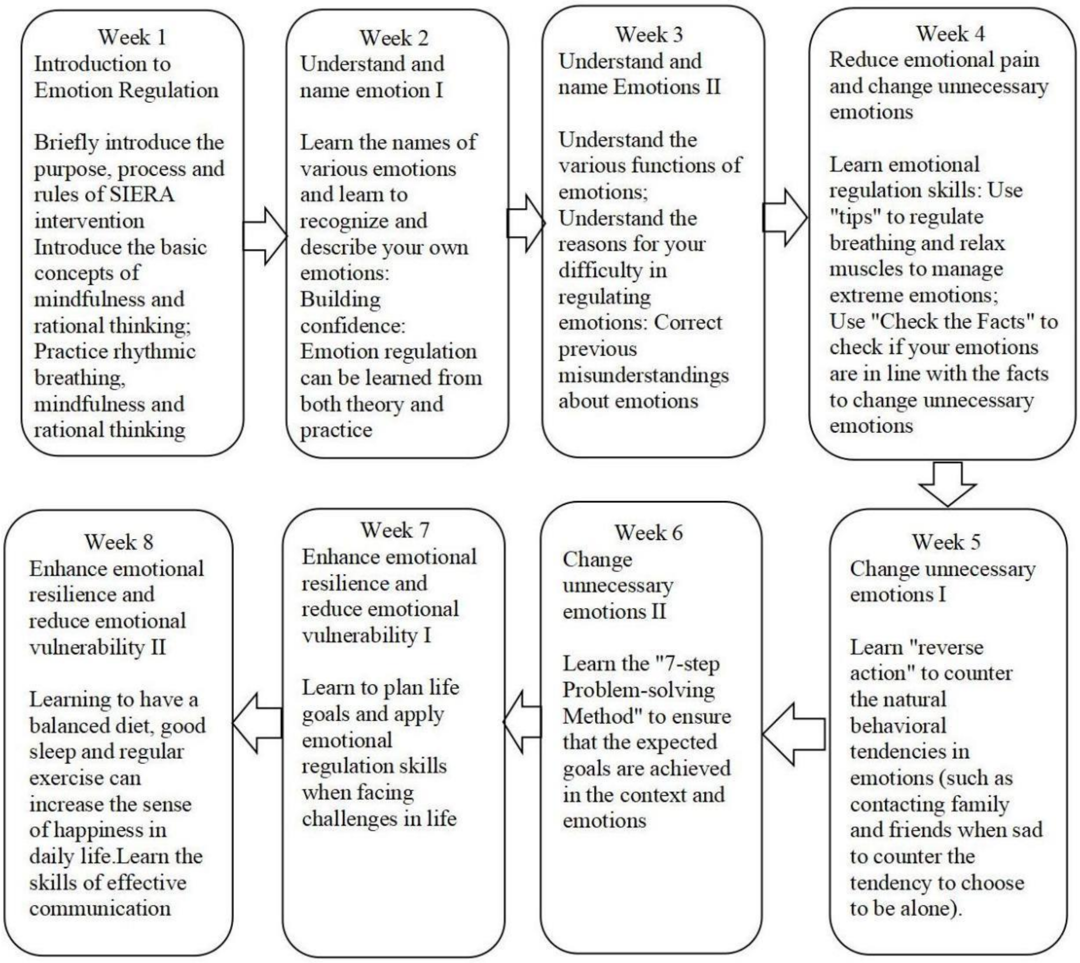

Figure 2: Flow chart of SIERA intervention

5.3 Drug therapy

Medication is a common treatment for autism spectrum disorders globally. However, due to the unique pathogenesis of these disorders, which involves factors such as cell development, gene expression, and environmental stimuli within the patient's body, there is a shortage of specific treatments. As a result, most clinical treatments for autism spectrum disorders currently rely on various antipsychotic drugs and psychological therapies. Risperidone, a frequently used medication, is currently approved by the U.S. Food and Drug Administration (FDA) for the treatment of autism spectrum disorders.

Risperidone is a common neurotic drug that improves patients 'mental states by affecting dopamine receptors. It achieves its therapeutic effects by inhibiting the human nervous system's central nervous system. Additionally, risperidone has fewer side effects, making it one of the most commonly used drugs in clinical treatments for schizophrenia. In clinical medicine, risperidone is classified as a benzoxazole derivative and is a new generation of antipsychotic drugs developed in recent years. The active ingredient of risperidone is a selective monoamine antagonist that interacts with 5-HT2 receptors and dopamine D2 receptors in the body, thereby inhibiting these receptors to control the patient's mental state. Besides being a widely used D2 antagonist in clinical medicine, risperidone can also bind to α1-adrenergic receptors in the body, improving positive symptoms of schizophrenia. Therefore, risperidone is currently receiving widespread attention and use in clinical medical treatments. Patients with autism spectrum disorders can effectively reduce the likelihood of emotional outbursts, aggressive behavior, and self-harm through continuous use of risperidone, although it may also lead to complications such as weight gain, fatigue, and gastrointestinal discomfort.

In addition to risperidone, fluoxetine and mirtazapine are also commonly used in the clinical treatment of autism spectrum disorders.

Mirtazapine is a α-adrenergic receptor antagonist used in antidepressant therapy. It primarily targets norepinephrine in patients by enhancing the neurotransmitter effects of adrenaline, which inhibits central nervous system transmission. Mirtazapine specifically inhibits 5-HT2 and 5-HT3 receptors and enhances the neurotransmitter transmission of 5-HTIA receptors, thereby promoting the conduction of serotonin (5-TH). This mechanism contributes to its antidepressant effects. Additionally, mirtazapine has a sedative effect, effectively reducing the likelihood of patients losing emotional control. Fluoxetine, on the other hand, is a selective serotonin reuptake inhibitor that effectively inhibits the synaptic transmission of neurotransmitters in patients. By controlling the reuptake of serotonin in the patient's body, fluoxetine improves serotonin levels, which helps manage autism. Furthermore, fluoxetine does not react with or bind to other receptors, such as α-adrenergic, β-adrenergic, 5-hydroxytryptamine, and dopamine receptors, demonstrating high specificity.

6. Conclusion

In summary, the incidence of autism spectrum disorders (ASD) has been increasing annually in recent years, significantly impacting the daily lives of many patients and their families. Patients with ASD often exhibit severe emotional regulation deficits, which hinder their ability to engage in social interactions and manage their daily lives. Medical researchers need to conduct in-depth studies on the neurophysiological mechanisms underlying ASD, to identify the causes and treatment priorities of ASD. Based on the specific conditions of each patient, appropriate treatments should be provided to effectively enhance their emotional regulation and cognitive abilities, thereby improving their quality of life.

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