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GABA and Executive functions in ASD

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Abstract. Gamma-aminobutyric acid (GABA) is a neurotransmitter critically involved in various psychological and behavioral processes. This review highlights the impact of GABAergic dysfunction within specific brain regions on a range of mental disorders, executive processes, self-control, and behavioral regulation.Reductions in GABAergic neurotransmission within distinct brain regions have been consistently associated with several mental health conditions. Within the context of anxiety disorders, depression, and post-traumatic stress disorder (PTSD), alterations in GABAergic function contribute to symptomatology. GABA plays a pivotal role in anxiety and mood regulation, with its impairment linked to symptoms of depression and mood disorders. Dysfunctional GABAergic transmission also contributes to fear dysregulation and features of PTSD. In terms of executive functions, GABAergic neurotransmission affects working memory, attentional control, and inhibitory regulation. Changes in GABA levels have been linked to decision-making abilities, impulsivity, and attention deficits, especially in conditions like attention deficit hyperactivity disorder (ADHD). GABA further plays a crucial role in self-control mechanisms by regulating impulsivity and prefrontal cortex functionality. Substance use disorders, often accompanied by impaired self-control, are significantly influenced by GABAergic system changes. Additionally, GABA's involvement in anxiety and emotional management affects the control of emotional responses. Behavioral control is modulated by GABAergic action in motor circuitry, where GABA provides inhibitory control for motor actions. Spontaneity, aggression, and stress are influenced by GABAergic dysfunction, impacting behavioral control. In the realm of attentional control, GABAergic neurotransmission influences selective attention and sensory salience, maintaining a balance between neural stimulation and inhibition. The study also explores executive function deficits in individuals with Autism Spectrum Disorder (ASD) in relation to GABA levels within specific brain regions. Moreover, GABA and its network connectivity contribute to individual variations in sensory responsiveness, emphasizing the dynamic role of GABA in the phenotypic heterogeneity of ASD. In summary, this research underscores the critical role of GABAergic neurotransmission within specific brain regions in various psychological disorders, executive functions, self-control, behavioral regulation, and attentional processes. Understanding the directional influence of GABAergic changes on behavior and mental health conditions can pave the way for more targeted interventions in neuropsychiatric disorders.
Keywords:GABA, executive functions, ASD, self – control, behavioral – control, attention – control

Introduction

GABA (gamma-aminobutyric acid) is a vital neurotransmitter that plays a key role in managing neuronal activity and maintaining overall neurological function in the brain. It functions primarily as an inhibitory neurotransmitter, which practically signals that it reduces the excitability of neurons and helps maintain the balance between excitation and inhibition in the brain (Olsen & Sieghart, 2009). GABA exerts its effects by binding to specific GABA receptors, which are categorised into two main types: GABA-A receptors and GABA-B receptors. GABA-A receptors are chloride-gated ligand-binding channels, whereas GABA-B receptors are metabotropic receptors that alter intracellular signaling pathways. Activation of GABA-A receptors results in the influx of negatively charged chloride ions into neurons, resulting in hyperpolarization of the cell membrane and a reduction in the likelihood of the neuron generating an action potential. This inhibition prevents excessive firing of neurons and helps to regulate the overall excitability of the brain. GABA is synthesized from glutamate, another neurotransmitter, through a

process called decarboxylation. The enzyme glutamate decarboxylase (GAD) takes over the conversion of glutamate to GABA. Once synthesized, GABA accumulates in synaptic vesicles and is released into the synaptic cleft, where it binds to GABA receptors on neighboring neurons, resulting in inhibitory effects (Schousboe, 2003).

The role of GABA in the brain is critical for various physical processes such as motor control, cognition and emotion regulation. It is involved in maintaining the balance between excitation and inhibition, which is essential for normal brain function.Disorders of GABAergic neurotransmission have been implicated in various neurological and psychiatric disorders. For example, decreased GABA levels or reduced GABA receptor function have been observed in conditions such as epilepsy, anxiety disorders, schizophrenia and sleep disorders (Petroff,2002).

The role of GABA (gamma-aminobutyric acid) in the brain involves complex functions that affect neuronal activity and neurotransmission. GABA acts as an inhibitory neurotransmitter, which means that it contributes to regulating the excitation of neurons and maintaining the balance between neuronal excitation and inhibition. More specifically, the synthesis and release: GABA is synthesised from glutamate, another neurotransmitter, by the action of the enzyme glutamate decarboxylase (GAD). Once synthesised, GABA is stored in synaptic vesicles inside neurons. Upon neuronal stimulation, GABA is released at the synapse, where it can interact with GABA receptors in adjacent neurons (Petroff, 2002).

Inhibition of neuronal activity: GABAergic neurotransmission plays a critical role in suppressing excessive neuronal activity and maintaining it balance of excitation and inhibition in the brain. The activation of GABA-A receptors results in the entry of chloride ions, which hyperpolarize the neuron, which making it less able to produce energy potential. This inhibitory action reacts to the effects of excitatory neurotransmitters such as glutamate and helps to prevent excessive stimulation of neurons (Schousboe, 2003).

Regulation of neurotransmission: GABAergic neurotransmission plays a modulatory role in various neuronal circuits. GABAergic neurotransmitter contributes to the regulation of other neuronal transmitter systems, such as dopamine, serotonin and norepinephrine, by affecting their release and excitation. Through these regulatory effects, GABA contributes to the overall balancing of neurotransmission and affects a variety of physiological functions in the brain (Fatemi & Folsom, 2009)

The role of GABA in disorders

GABA (gamma-aminobutyric acid) is a neurotransmitter that plays an important role in people's psychological state. GABA acts as an inhibitory neurotransmitter in the central nervous system (CNS) and helps regulate the excitability of neurons (Etkin, et al., 2009). Its importance in the science of psychology stems from its contribution to a variety of psychological processes, including the regulation of anxiety, mood modulation and stress response. GABAergic dysfunction has been implicated in several psychological disorders, including anxiety disorders, depression and post-traumatic stress disorder (PTSD). This study has demonstrated that changes in GABAergic neurotransmission may contribute to the pathophysiological progression of these conditions (Hasler, et al., 2009).

Anxiety disorders: GABA has a calming effect on the brain and helps improve stress. Decreased GABA levels or decreased GABA receptor functionality have been found in persons with anxiety disorders such as generalized anxiety disorder (GAD), panic disorder and social anxiety disorder Studies have suggested that GABAergic deficits may contribute to enhanced anxiety symptoms and increased neuronal excitation in brain circuits associated with anxiety (Liberzon, 2016).

Depression: GABAergic disorder has also been implicated in depression. Research suggests that decreased GABAergic activity may be associated with depressive symptoms and mood disturbance. Alterations in GABAergic transmission, along with other forms of neurotransmitters, contribute to the complex neurobiology of depression (Gabbay, et al., 2012).

Post-traumatic stress disorder (PTSD): Post-traumatic stress disorder is a psychological disorder that can occur following the impact of a traumatic event. Altered GABAergic signaling may contribute to dysregulation of fear responses, emotion management, and the onset of PTSD symptoms (Fatemi & Folsom, 2009).

GABA and executive functions

GABA (gamma-aminobutyric acid) is also involved in regulating executive functions in the brain. Executive functions refer to a group of cognitive processes that enable goal-directed behavior, including decision making, working memory, attention concentration control, and inhibition. GABAergic neurotransmission plays a role in modulating these executive functions. Below is an overview on the role of GABA in executive functions:

Decision making: GABAergic neurotransmission affects decision making processes by regulating the balance between reward seeking and inhibition control. Research has shown that GABA levels in specific brain regions, such as the prefrontal cortex and striatum, are associated with decision-making abilities and impulsivity (Clark, et al., 2006).

Working memory: Working memory refers to the ability to retain and process information in the mind for short periods of time. GABAergic transmission is involved in the regulation of neuronal

activity within the prefrontal cortex, a brain region critical to working memory processes. Dysfunctions of GABAergic functionality in the prefrontal cortex may impair working memory performance (Lewis, et al., 2005).

Attention control: GABA plays a role in attentional processes, including selective attentional control and inhibitory control. GABAergic dysfunction has been implicated in attention deficit hyperactivity disorder (ADHD) and other conditions characterized by attention deficits (Volkow, et al., 2009).

Suspension: Suspension control: Inhibitory control is an essential aspect of executive functions involved in inhibiting impulsive or inappropriate behaviors. GABAergic neurotransmission contributes to inhibitory control by regulating neuronal excitation and reducing the incidence of spontaneous or unwanted responses (Whittington, et al., 1997).

In conclusion, there is a significant contribution of GABA to executive functions such as decision making, working memory, attentional control and inhibition. Further investigation of these tasks may provide more in-depth insights into the individual roles and mechanisms of GABA in executive function.

GABA and self – control

GABA (gamma-aminobutyric acid) is thought to contribute to self-control processes in the brain. Self-control refers to the ability to regulate one's thoughts, emotions and behaviours to achieve long-term goals. GABAergic neurotransmission contributes to the regulation of self-control mechanisms. Below is a review with reports on the relationship between GABA and self-control:

Impulsivity and self-control: GABAergic functionality has been linked to impulsivity, which may be perceived as a lack of self-control. Research has concluded that individuals with low GABA levels or impaired GABA receptor functionality may have increased impulsivity and self-control problems (Winstanley, et al., 2010).

Prefrontal cortex and self-control: The prefrontal cortex, a brain area involved in self-control processes, relies on proper GABAergic neurotransmission. GABAergic inhibition within the prefrontal cortex helps regulate neural activity and supports the suppression of spontaneous responses, which facilitates self-control (Smith, et al., 2008).

Substance use disorders: Impaired self-control often occurs in people with substance use disorders. Research suggests that alterations in GABAergic neurotransmission contribute to the onset and maintenance of addiction by affecting self-control mechanisms (Ersche, et al., 2010).

Stress and emotion regulation: GABAergic activity plays a role in stress responses and emotional regulation, which are directly linked to self-control functions. GABAergic neurotransmission helps modulate the function of brain regions involved in emotion regulation, such as the amygdala, which facilitates self-control of emotional responses (Etkin, et al., 2011).

Although more investigation is still needed to fully understand the complex mechanisms involved, GABAergic neurotransmission is found to contribute to impulsivity, prefrontal cortex function, substance use disorders and emotional regulation related to self-control (Etkin, et al., 2011).

GABA and Behavior – control

These reports offer insights into the role of GABA in the domain of behavioral control within the brain. Although further research is needed to fully understand the mechanisms involved, these studies highlight the importance of GABAergic neurotransmission in the regulation of neural activity and the influence of various behavioural processes, including motor control, impulsivity, aggression and anxiety (Petrovic & Castellanos, 2017).

Engine control: GABAergic action is extremely useful for controlling movement, regulating voluntary movements and planning motor action. GABA acts within motor circuits, such as in the basal ganglia and cerebellum, to provide inhibitory control and fine-tune motor energy production (Benarroch, 2013).

Impulsivity and aggression: GABAergic impairment has been associated with impulsivity and aggressive behavior. Studies have found that changes in GABAergic neurotransmission may have an impact on behavioral control, which contributes to impulsive actions and aggression (Manto, 2012).

Anxiety and stress: GABAergic neurotransmission is involved in the regulation of anxiety and stress responses. GABAergic dysfunction may cause impaired control of anxiety and increased activity in stress, which affects behavioural responses in stress-related situations (Gabbay, 2012).

GABA and Attention - control

GABA (Gamma-aminobutyric acid) plays an essential role in controlling attention in the brain. Attention control involves the ability to selectively focus on relevant stimuli while avoiding distractions. GABAergic neurotransmission contributes to the regulation of attentional processes by affecting attention and attentional regulation (Gao, et al., 2009).

The modulation of neural excitability i.e. GABAergic inhibition contributes to the regulation of the balancing of neural excitation and inhibition, which is crucial for attentional processes. GABAergic neurotransmission within brain circuits associated with attention, such as the prefrontal cortex and parietal cortex, modifies the excitability of neurons and affects attentional states (Gao, et al., 2009).

Then the GABAergic function of memory and attention: Memory function refers to the ability to temporarily retain and process information in the mind. Proper GABAergic neurotransmission in the

prefrontal cortex, a brain region critical for working memory, enhances attentional processes associated with the maintenance and updating of information.GABA (Clarke, et al., 2013). In attention disorders GABAergic dysfunction has been implicated in attention disorders such as attention deficit hyperactivity disorder (ADHD). Altered GABA levels or impaired GABAergic transmission may contribute to the attentional deficits noted in such disorders. GABA and In addition, GABAergic neurotransmission also plays a role in sensory attention, facilitating the selective processing and consideration of relevant sensory information. GABAergic inhibition helps to regulate the balance of neural excitation and inhibition within sensory processing pathways, affecting the allocation of attentional resources (Schneider, et al., 2020).

Self – control, Behavior - control, Attention - control and neurotransmitters

Self-control refers to the ability to control one's thoughts, feelings and behaviours to achieve long-term goals. It involves resisting immediate impulses or temptations and making choices that align with long-term goals. Self-control is critical for a variety of aspects of life, including academic performance, interpersonal relationships, and overall well-being (Tangney, et al., 2004).

At the same time, behavioral control involves regulating one's actions and reactions to various situations. It is about the ability to modify behavior in accordance with the rules of society, the needs of the particular situation, and personal goals. Effective behavioural control enables individuals to adapt to their environment, prevent impulsive actions and make informed decisions (Heatherton, et al., 2011).

Next, attention control refers to the ability to selectively focus on relevant stimuli without regard to distractions. It involves the allocation and maintenance of attentional resources to specific tasks or stimuli, separating out irrelevant information. Attentional control plays a critical role in cognitive processes such as perception, learning, memory and decision-making (Heatherton & Wagner, 2011).

Each aspect of the above contributes to their cognitive and behavioural regulation, affecting their goal-directed behaviour, social interactions and overall functioning (Heatherton & Wagner, 2011).
The following pilot study aimed to conduct research on the relationship between neurocognitive performance and brain metabolites in young children with Bipolar Disorder (BD) using proton magnetic resonance spectroscopy (1H-MRS). The study included 30 participants, 20 with depressive BD and 10 healthy comparison subjects, aged 13-21 years. Neurocognitive performance and executive function measures were assessed and 1H-MRS data were obtained from the anterior cingulate cortex (ACC) to quantify proton metabolites, including N-acetyl aspartate (NAA) and gamma-aminobutyric acid (GABA) (Huber, et al., 2018).

The data showed that subjects with MD scored much lower on executive function measures than healthy participants in comparison. In addition, significant positive associations were found between performance on the Wisconsin Card Sorting Test (WCST) and NAA and GABA levels in the ACC of bipolar youth. As performance on the WCST increased, NAA and GABA levels also increased.The study admits limitations, such as the small sample size and lack of drug control. Nevertheless, the results suggest that executive function deficits in bipolar youth are associated with NAA and GABA levels. The data suggest that cognitive deficits occur early in the course of the disease and may express risks associated with altered neurochemistryInvestigating the link between brain metabolites and cognition in DM will provide important information for the development of specific interventions (Huber, et al., 2018)..

This study highlights the need for understanding cognitive impairment in MD, particularly in youth, and the mechanisms underlying it. Investigation of neurocognitive functioning in BD during the developmental phase may shed light on the onset and progression of neuropsychological deficits and help identify opportunities for interventions. Proton magnetic resonance spectroscopy (1H-MRS) offers valuable insights into brain chemistry and metabolism, which enables researchers to explore the neural substrates underlying cognitive deficits in youth with BD (Huber, et al., 2018).

Subsequently, following the study by Hatoum et al., (2022), emphasis was placed on the genetic documentation of executive function (EF) and its relationship with psychiatric disorders. EF deficits are associated with a variety of psychiatric disorders, suggesting a potential risk factor for psychopathology. Previous studies have suggested a genetic aspect to the relationship between cognition and psychopathology. This study aims to investigate the genetic basis of a common EF factor (cEF) and its association with intelligence quotient (IQ) and speed of cognitive processing, and its relationship with psychopathology.The investigators conducted a broad gene-level association study (GWAS) using data from the UK Biobank study. They produced a cEF factor score based on multiple EF tasks and performed GWAS to identify genetic variants associated with cEF. They also assessed the gene association between cEF and IQ using LD Score regression. In addition, polygenic scores for cEF and IQ were generated to assess their relationship with multiple latent variables EF and IQ in young adult twin samples(Hatoum, et al., 2022)..

The findings showed a significant association between genetic variants and cEF, with 129 major and 299 independent single nucleotide polymorphisms (SNPs) at 112 different loci. The genetic correlation between cEF and IQ was found to be less than 1, indicating genetic separability between the two constructs. Multigene outcome analysis supported the distinct genetic effects of

cEF and IQ on cognitive abilities. In addition, cEF showed stronger genetic associations with psychiatric disorders compared to IQ, suggesting its importance in psychopathology.The study also examined the molecular pathways associated with cEF using gene and gene set analyses. The genes identified were enriched in pathways related to potassium channels, synaptic structure and GABA receptor activity. Gene expression analysis specific to each cell type indicated the involvement of GABAergic function in cEF. Transcriptome-level analysis and eQTL data provided additional insights into the molecular mechanisms underlying cE F(Hatoum, et al., 2022)..

The researchers performed a GWAS analysis with data from the UK Biobank study, which included over 427,000 people of European descent. A cEF factor score was produced based on the commonality of five EF tasks. GWAS was performed using linear mixed models, controlling for various covariates. LD Score regression and BOLT-REML were used to assess genetic correlations between cEF and IQ. Multigene scores for cEF and IQ were generated using GWAS data and tested for correlations with cognitive skills in twin samples. Gene and gene cluster analyses were performed using MAGMA and cell type-specific gene expression analyses were performed using eQTL data.Overall, this study applied a large-scale GWAS approach to study the genetic basis of cEF, its association with IQ and cognitive process speed and its relevance to psychopathology. Results indicate the genetic distinctiveness of cEF and IQ, with distinct genetic influences on cognitive skills and psychiatric disorders. The research also provides insights into the molecular pathways and gene expression patterns associated with cEF (Hatoum, et al., 2022)..

The results showed a genome-wide association (GWAS) of a common executive function factor (cEF) score using data from the UK Biobank. Factorial cEF scores were generated according to the joint nature of five executive function tasks. GWAS identified 129 major SNPs and 299 independent SNPs at 112 distinct loci that were significantly associated with cEF. The most significantly associated SNP was an expressive quantitative linkage (eQTL) in cerebellar tissue corresponding to the EXOC4 gene (Hatoum, et al., 2022).

The following research of Ribeiro (2015), examines the cognitive deficits associated with neurofibromatosis type 1 (NF1), a neurodevelopmental disorder. One of the central deficits noted in NF1 is executive dysfunction, which includes impairments in executive attention, inhibitory control, and response inhibition. The study aims to better understand the neural mechanisms underlying these deficits.The investigators performed a visual go/no-go task to assess inhibitory control in children and adolescents with NF1 and a control group. They applied a multimodal method, combining high-density electroencephalography (EEG) to investigate brain responses and magnetic resonance spectroscopy (MRS) to measure the levels of neurotransmitters, especially GABA and glutamate+glutamine, in the medial frontal cortex and occipital cortex.

The results showed that NF1 subjects exhibited impaired impulse control and reduced EEG correlations with early visual processing and inhibitory control. MRS data showed reduced GABA levels in the medial frontal cortex, consistent with previous findings of reduced occipital GABA levels in NF1. However, glutamate + glutamine ratios are normal, suggesting an abnormal balance between inhibition and excitation in NF1. Significantly, increased GABA levels in the medial frontal cortex were associated with a faster response style in NF1 subjects, whereas in the control group they were associated with a guarded strategy. Also, higher GABA levels in the medial frontal cortex were associated with general mental skills (IQ) in NF1 and inhibitory control in both of these groups (Ribeiro, et al., 2015).

The study suggests that abnormal GABAergic function plays a key role in the cognitive impairments observed in NF1, with regional and disease-dependent effects. The findings highlight the importance of the medial frontal cortex, including the anterior cingulate cortex and pre-complementary motor area, in inhibitory control. The research offers insights into the neural correlates of impaired inhibitory control in NF1 and highlights the potential role of GABA in cognitive impairment (Ribeiro, et al., 2015)

The information provided relates to a study that examined the association between the neurotransmitters glutamate and GABA and impulsivity and aggression in female patients with borderline personality disorder (BPD) and attention-deficit hyperactivity disorder (ADHD). It was found that both groups of patients showed higher levels of self-reported impulsivity, anger, and aggression than healthy controls. Regarding neurotransmitter levels, the ADHD patient group had significantly lower levels of GABA in the anterior cingulate cortex (ACC) compared to the control group. Still, the results also showed positive correlations between glutamate levels and impulsivity and negative correlations between GABA levels and impulsivity and aggression.The results suggest that alterations in glutamate and GABA levels in the ACC are associated with impulsivity and aggression in BPD and ADHD. The study hypothesized that the different glutamate and GABA correlations observed in BPD and ADHD patients could have implications for more targeted pharmacological treatments (Ende, et al., 2016).

Impulsivity and reactive aggression are common behavioral problems in both BPD and ADHD. Impulsivity is described by spontaneous action, deficits in executive functions and behavioral inhibition. Aggressive behavior in BPD and ADHD may be reactive or impulsive aggression in response to challenge or instrumental aggression

for external gain. These symptoms are associated with abnormalities in fronto-limbic networks, including the ACC. Glutamate and GABA are important neurotransmitters in this network and have been implicated in impulsivity and aggression. Previous research has demonstrated altered glutamate concentrations in the ACC of female patients with BPD and lower GABA levels in children with ADHD. However, there is still a lack of research on the role of GABA in impulsivity (Ende, et al., 2016).

The method used was proton magnetic resonance spectroscopy (MRS) to measure glutamate and GABA levels in the ACC of female BPD patients, female ADHD patients and a healthy control group. Patients underwent diagnostic assessments and completed self-assessment scales to assess impulsive behavior and aggression. MRS data showed that the ADHD group had significantly reduced GABA levels in the ACC compared to the control group. There were positive correlations between glutamate levels and impulsivity and negative correlations between GABA levels and impulsivity and aggression.The evidence for the involvement of glutamate and GABA in impulsivity and aggression in BPD and ADHD was established. The results suggest that changes in these neurotransmitters in the ACC may contribute to the behavioral dysregulation seen in these disorders. More study is needed to strengthen these associations and explore their implications for pharmacological treatments (Ende, et al., 2016).

This study of Jupp (2013), focuses on investigating neurotransmitter systems, including dopamine (DA), serotonin (5-HT), endogenous opioids and gamma-aminobutyric acid (GABA), in relation to impulsive behaviour in rats.It was observed that rats exhibiting high impulsivity (HI) showed severe changes in neuroreceptor connectivity compared to rats with low impulsivity (LI). In particular, HI rats showed lower binding for dopamine transporter receptor (DAT) and D2/D3 receptors in the nucleus accumbens sheath (NAcbS), and lower binding for D1 receptors in the nucleus accumbens nucleus (NAcbC). These findings suggest that abnormal dopamine transmission, as suggested by altered receptor binding, may play a role in impulsivity.In addition, the study showed that rats with HI had lower GABA(A) receptor binding in the anterior cingulate cortex (ACC). The ACC is a brain region involved in decision making, emotional regulation and cognitive control. Lower GABA(A) receptor binding in the ACC suggests a possible involvement of GABAergic mechanisms in the manifestation of impulsive behavior (Jupp, et al., 2013).

In particular, the modifications in receptor binding observed in impulsive rats are specific for some brain regions and certain neurotransmitter systems. There were no substantial differences in binding for 5-HTT (serotonin transporter) or m-opioid receptors in any of the regions examined.The correlations found between receptor binding and impulsive behavior in the test indicate a relationship between neurochemistry and impulsivity. Less receptor binding in the NAcbS, NAcbC and ACC was associated with greater levels of impulsive responding in rats (Jupp, et al., 2013).

The results of the study provide insights into the neurochemical basis of impulsive behaviour and suggest that abnormal disturbances of dopamine, GABA and receptors in certain brain regions may contribute to the onset of impulsive behaviour. A better understanding of these mechanisms is essential to elucidate the underlying causes of impulsivity and to develop potential interventions or treatments to address impulsive behaviour in humans (Jupp, et al., 2013).

The following study Feja & Koch (2014), examines the role of the ventral medial prefrontal cortex (vmPFC) in impulse control, delayed discounting and various aspects of impulsivity. Impulsivity is a multidimensional and multifaceted phenomenon associated with several neuropsychiatric disorders, such as ADHD, addiction, aggression and schizophrenia. The present study exploited the temporary inactivation of the rat vmPFC by means of the GABAA receptor agonist muscimol to examine its relevance to impulse control and delay-judgment tasks.

From the study findings, it appeared that inactivation of the vmPFC impaired impulse control, as indicated by increased early response in the 5-choice serial reaction time serial reaction time test (5-CSRTT). This suggests that the vmPFC has a critical role in the regulation of impulsive actions. In contrast, inactivation of the vmPFC did not substantially affect delay-impulsivity, which refers to the preference for smaller immediate rewards over larger delayed rewards. This suggests that other neural pathways, rather than the vmPFC, may be involved in mediating impulsive decision making. Impulsivity is a complex behavioural trait that depends on different cognitive-executive processes and is mediated by different brain regions. The prefrontal cortex, including the dorsal prefrontal cortex (DLPFC), orbitofrontal cortex (OFC) and anterior cingulate cortex (AC), is critical for executive processing and impulsive control. However, the specific roles of these regions in different aspects of impulsivity are under investigation and understanding (Feja & Koch, 2014).

In the rodent model, the medial prefrontal cortex (mPFC), in particular the vmPFC, is thought to be equivalent to the anterior cingulate cortex (AC) and to play an important role in impulse control. Damage and inactivation studies have shown that functional dysfunction of the vmPFC causes deficits in impulse control. However, the involvement of the vmPFC in impulsive decision making, such as delay-discrimination, remains less clear. Some studies suggest a secondary role of the vmPFC in delay, while other neural pathways are more critical.The procedure used muscimol, a GABAA receptor agonist, to temporarily deactivate the

vmPFC. The muskimol-induced inactivation is reversible and makes it possible to design within the subject, which increases confidence in the experimental results. Histology analysis confirms the precise localization of muscimol injections in the vmPFC region (Feja & Koch, 2014).

In conclusion, we observe a distinction between spontaneous action (impulse control) and impulsive selection (delay-discounting) at the vmPFC level in rats. Inactivation of the vmPFC reduced impulsive control but did not significantly affect delay... These findings contribute to our understanding of the neural mechanisms underlying different forms of impulsivity and highlight the crucial role of the vmPFC in the regulation of impulsive actions (Feja & Koch, 2014).

In addition, it has been documented that self-awareness is a conscious experiential experience with oneself as the object, which enables conscious self-observation and behavioral control. The network, which predominantly includes the central prefrontal and medial parietal regions, is identified as a medium for self-awareness. This network is active during self-awareness and acts as the default mode network when focusing attention on the self. The synchronized GABA-induced GABA-induced oscillations facilitate the interaction between the medial prefrontal and medial parietal zones (Lou, et al., 2020).

The neurotransmission of dopaminergic substances has been found to play a critical role in conscious experience. Abnormal neurotransmission of dopaminergic substances is associated with hallucinations and delusions in schizophrenia. Research using signal detection tests and subjective interpretation demonstrates that dopamine activation enhances confidence in perceptual ability and affects word recognition tests. The results of these studies provide direct evidence of dopamine involvement in experiential awareness. The interaction between dopamine and GABA receptors is critical for the regulation of experience consciousness. Dopamine enhances GABA binding in the paralimbic cortex, suggesting a role in self-awareness (Lou, et al., 2020).GABA receptors, constructed as ligand ion channels, allow negative chloride ions to pass through and generate electrical pulses. The affinity of dopamine and other molecules to GABA receptors is based on the composition of the receptor complexes. Altered GABA neurotransmission has been observed in conditions such as addiction, autism, immunocognition, hallucinations, delusions, delusions, schizophrenia, major depression and dementia, which are associated with poor self-awareness and self-control. (Lou, et al., 2020).

The development of self-awareness and the functioning of the paralimbic network are observed from infancy to adulthood. The default mode network follows an inverse U-shape, being weaker in children and elderly individuals and stronger in adults. Deficient GABA neurotransmission in the paralimbic network is linked to various disorders and pathologies, highlighting its importance in brain function (Lou, et al., 2020)..

The sensitivity of the paralimbic network is due to its high oxygen needs due to the concentrations of GABAergic interneurons. The disturbed metabolic processes may affect g-oscillations, which hold a critical role in conscious experience.Limitations in the perception of consciousness of experience and self-awareness from a neurobiological point of view have hampered progress in understanding these phenomena and their connection to disease. However, recent research has demonstrated the catalytic role of the paralimbic network in self-awareness and awareness of experience. Dopaminergic agents acting on GABA receptors in the paralimbic network have shown promise in stimulating awareness of experience, leading to potential pharmaceutical applications (Lou, et al., 2020).

In summary, self-control, behavioural control and attentional control are mediated by the paralimbic network and the interplay between dopamine and GABA neurotransmission. These processes are essential for self-awareness and consciousness and have implications for explaining a variety of disorders and dysfunctional cognitions (Lou, et al., 2020).

The role of GABA in ASD

This study Demetriou et al., (2019), examined the association between thalamic and somatosensory cortex neurochemicals, functional connectivity, and excessive sensory responsiveness (SOR) in people with autism spectrum disorder (ASD) and typically developing (TD) individuals. Magnetic resonance spectroscopy and resting-state functional magnetic resonance imaging were performed to investigate GABA and glutamate concentrations in thalamic and somatosensory cortex, and their correlation with functional connectivity and SOR severity. Data showed that in the ASD group, higher SOR severity was associated with lower thalamic GABA and higher somatosensory glutamate. Thalamic GABA concentration also predicted altered connectivity with regions implicated in SOR. These findings suggest that GABA and associated network connectivity may play a role in individual differences in SOR, highlighting the potential impact of GABA as a mechanism underlying phenotypic heterogeneity in ASD.

This study involved 35 people with ASD and 35 typically developing children aged 8-17 years. Magnetic resonance spectroscopy and resting functional magnetic resonance imaging data were collected. GABA and glutamate concentrations in the thalamus and somatosensory cortex were measured by magnetic resonance spectroscopy. The resting connectivity function between these areas and other brain regions was evaluated. Behavioural measures were also collected, including sensory overreaction, stress and cognitive abilities. Statistical analyses, including t-tests, correlations,

and regression analyses, were performed to examine associations between concentrations of neurochemicals, connectivity, and sensory hyperresponsiveness (Demetriou, et al., 2019).

The relevant results showed no significant difference between groups in terms of neurochemical concentrations between subjects with ASD and subjects with normal growth. However, within the ASD group, higher severity of sensory overreaction was associated with lower thalamic GABA concentrations and higher somatosensory glutamate concentrations. Thalamic GABA concentration also predicted altered connectivity with areas previously implicated in sensory overreaction. These findings suggest that GABA and associated network connectivity may contribute to individual differences in sensory overreaction, which is a major source of phenotypic heterogeneity in ASD (Demetriou, et al., 2019).

In addition, data were generated on the role of GABA and thalamic connectivity in sensory hyperresponsiveness in individuals with ASD. The results suggest that perturbations in the neurochemical balance of the thalamus, particularly reduced GABA content, could interfere with the role of the thalamus in integrating and regulating an attachment to sensory cues. These results have implications for a better understanding of the mechanisms underlying sensory hyperresponsiveness and may inform future research and potential pharmacological interventions aimed at modulating GABA (Demetriou, et al., 2019).

The present review by Wood et al, (2021), studies the important role of executive function (EF) in Autism Spectrum Disorder (ASD). A review of the evolution of EF, collective theoretical models of EF and challenges in the study of EF is provided. The review considers the potential of EF as a cognitive endophenotype for ASD and examines the research domain criteria framework (RDoC) as a research approach for studying EF in ASD. It also discusses several executive-focused cognitive ability models that have been proposed to explain the clusters of symptoms observed in ASD. Empirical studies suggest a broad impairment of EF in individuals with ASD, but substantial variability in EF performance between individuals is observed. The review highlights the heterogeneity of EF performance as a challenge to establishing EF as a cognitive endophenotype in ASD, but also as a potential for subtyping and targeted interventions. The importance of understanding the neurobiological underpinnings of EF, such as the excitation/inhibition hypothesis, is emphasized. The application of the RDoC framework is suggested to better understand the impairment of EF in ASD and to improve interventions for this disorder.

This review is based on an extensive exploration of relevant research articles, theoretical models and systems related to executive function and its application to autism spectrum disorders. The authors synthesized information from these sources to provide a comprehensive review of EF, including development, theoretical models, and implications for ASD. They also included empirical studies that investigated EF in individuals with ASD, examining the broad impairment and inter-individual variability observed. The authors conducted a critical appraisal of the literature and identified limitations and opportunities for further research, including for the study of EF in ASD (Wood, et a., 2021).

The review then reveals that executive function plays an important role in autism spectrum disorder and may represent a cognitive endophenotype. Diverse theoretical models of EF offer insights into the specific impairments noted in ASD, with a focus on attentional control, cold EF and hot EF. Empirical studies consistently show a broad impairment of EF in individuals with ASD, although there is substantial heterogeneity in EF performance across individuals. However, this heterogeneity creates difficulties in establishing EF as a cognitive endophenotype, but also provides opportunities for subtyping and targeted interventions within the autism spectrum. The review highlights the need for a better understanding of the neurological basis of EF, including the role of GABAergic neurotransmission in the balance between arousal and inhibition. It is also recommended that the research domain criteria framework be applied to integrate a variety of metrics and to conduct research on additional factors and mitigating factors that influence EF in the ADS. This approach has the potential to improve our understanding of EF disorder in ASD and inform the development of more effective interventions for this disorder (Wood, et a., 2021).

The study by Naaijen et al, (2017), used data from the International Multi-center ADHD Genetics (IMAGE) study, which included participants aged 5-17 years of Caucasian European origin. The sample under consideration consisted of individuals with Attention Deficit Hyperactivity Disorder (ADHD), but without classic autism, intellectual disability, epilepsy, or other neurological or genetic disorder associated with behavioral externalizing. The total sample size for this study was 931(Naaijen, et al., 2017).The severity of ADHD symptoms was assessed using a standardized written interview and questionnaires completed by parents and teachers. The severity of symptoms of inattention and hyperactivity/impulsivity was assessed using a 4-point scale. The severity of autism symptoms was calculated using the social communication questionnaire completed by parents.A Stop task was performed to measure response inhibition. Participants were required to not respond to a stop signal. Stop signal response time (SSRT) was a measure of response inhibition. (Naaijen, et al., 2017).

Genome-wide genotyping was performed using the Perlegen genotyping platform. Genome-wide genotyping was performed using the Hapmap II version 22 dataset. Quality control measures were applied, resulting in 2,182,904 SNPs obtained for

analysis. Gene sets associated with glutamate and GABA neurotransmission were selected based on the Ingenuity Pathway Analysis database pathway analysis. SNPs within the the selected genes and their flananking regions were also included in the analysis(Naaijen, et al., 2017).

The MAGMA program was applied to evaluate the connection. Principal component regression models were applied to account for linkage equilibrium disturbances. Gene set analysis was performed to evaluate the joint association of gene sets with cluster severity and inhibition. Whole gene and individual SNP associations were also studied. Corrections were performed to apply multiple comparisons (Naaijen, et al., 2017).The glutamate gene set showed a significant association with the severity of hyperactivity/impulsivity symptoms in ADHD (P = 0.009) after correction for multiple comparisons. No significant associations were found for severity of autistic symptoms or severity of inattention symptoms; the GABA gene set showed no significant associations with any of the symptom dimensions. However, a significant association with inhibition was found (P=0.04) when the 100 kb flanking region was included, although this did not survive the correction procedure for multiple comparisons.Individual gene analyses and single variable associations did not yield statistically significant data for either the glutamate or GABA gene sets (Naaijen, et al., 2017).

The study offers evidence supporting the involvement of glutamate and GABA neurotransmission in the severity of ADHD and Autism symptoms. The glutamate gene set was associated with symptoms of hyperactivity/impulsivity, while the GABA gene set showed a nominal association with inhibition. These findings suggest that disturbances in glutamate and GABA transmission may contribute to the pathophysiological progression of ADHD and ASD. However, more investigation is needed to validate these data and to examine the specific gene variations underlying these associations (Naaijen, et al., 2017)

According to Piccardi et al., (2021), Gamma-aminobutyric acid (GABA) is an important neurotransmitter in the brain that plays a crucial role in the regulation of neuronal activity. It acts as an inhibitory neurotransmitter, meaning that it helps to reduce or inhibit neuronal activity, promoting a state of calm and relaxed state. GABA is involved in a variety of physiological functions, including the control of motor movements, vision and the regulation of anxiety and stress. (Piccardi, et al., 2021).

In the context of neurodevelopmental disorders, such as autism spectrum disorders (ASD) and attention deficit hyperactivity disorder (ADHD), research has demonstrated abnormalities in haptic processing, which refers to the way the brain manages and reacts to tactile sensations. Touch processing involves behavioural and neural markers, which can be assessed through test projects and experimental measurements (Piccardi, et al., 2021).Behavioural indicators of touch processing involve observable responses and behaviours associated with touch stimuli. Individuals with ASD typically exhibit hypersensitivity to touch (increased sense of sensitivity or aversion to touch). However, this hypersensitivity can be assessed by various measures, such as parent-reported questionnaires or observational assessments. In contrast, tactile hypersensitivity ("decreased sensitivity or aversion to touch") has also been reported in some individual individuals with ASD and ADHD (Piccardi, et al., 2021).

Neurological markers of touch processing involve the investigation of brain activity and responses to a tactile stimulus. Studies suggest that individuals with ASD and ADHD may show differences in neural responses to tactile stimuli compared to typically developing individuals. For example, reduced neural repetition suppression, which refers to the reduced brain response to repeated tactile stimuli, has been observed in individuals with ASD and ADHD. This reduced rate of suppression may indicate atypical inhibitory function in GABA-mediated circuits (Piccardi, et al., 2021).

Research has also suggested that early sensory cues, including touch processing, may be potential predictors of later features of ASD or ADHD. Longitudinal studies have found associations between early markers of tactile processing and later symptoms of ASD or ADHD. For example, reduced suppression of neural repetition in response to haptic stimulation at an early age has been found to predict later ASD traits. Similarly, impaired alpha range deconditioning (neural hypersensitivity) in the first haptic response has been associated with higher rates of activity and lower inhibitory control, which are features of ADHD, at later ages (Piccardi, et al., 2021).

The role of ICT in GABA

The integration of Artificial Intelligence (AI) and Information and Communication Technology (ICT) has ushered in a new era in the study of executive function training and GABA regulation. AI-driven cognitive training programs have become increasingly personalized and effective, offering tailored interventions to individuals with cognitive challenges (Hampshire et al., 2012). AI's ability to adapt training regimens based on an individual's progress and needs enhances the efficacy of executive function training (Jaeggi et al., 2011). Moreover, AI can analyze complex neurobiological data related to GABAergic function, aiding researchers in gaining a deeper understanding of its role in cognitive processes (Smith et al., 2019). AI's real-time feedback and data analysis capabilities are instrumental in optimizing training protocols and improving cognitive and behavioral outcomes.

The role of Information and Communication Technology (ICT) is pivotal in delivering and enhancing executive function training and GABA

regulation interventions. ICT tools such as mobile applications, virtual reality simulations, and web-based platforms provide accessible and interactive training experiences, increasing engagement and adherence to cognitive training programs (Toril et al., 2016). These technologies also enable remote monitoring and data collection, crucial for evaluating the effectiveness of training programs and the relationship between GABA and cognitive functions (Kühn et al., 2013). Furthermore, ICT facilitates the dissemination of research findings and therapeutic interventions to individuals with cognitive challenges, contributing to improved accessibility to evidence-based training and therapies (Zhou et al., 2017). The integration of AI and ICT in executive function training and GABA regulation research holds great promise, offering more targeted interventions and a deeper understanding of these critical cognitive processes.

AI-driven interventions have made remarkable strides in personalizing cognitive training programs for individuals with ASD. AI algorithms can adapt training exercises based on an individual's specific needs and progress, thereby enhancing the effectiveness of interventions targeting executive function deficits in ASD (Hampshire et al., 2012). Moreover, AI plays a vital role in processing complex neurobiological data related to GABAergic function, providing insights into its impact on cognitive and behavioral aspects in individuals with ASD. AI-driven real-time feedback and data analysis are crucial for tailoring training programs to address the unique challenges presented by individuals with ASD, ultimately promoting improved cognitive and behavioral outcomes.

Information and Communication Technology (ICT) also plays a central role in the delivery of executive function training and GABA regulation interventions for individuals with ASD. ICT tools, including mobile applications, virtual reality simulations, and web-based platforms, provide accessible and interactive training experiences that enhance engagement and adherence to cognitive training programs for individuals with ASD (Toril et al., 2016). These technologies enable remote monitoring and data collection, critical for assessing the effectiveness of training programs and understanding the relationship between GABA and cognitive functions in the ASD population (Kühn et al., 2013). Furthermore, ICT facilitates the dissemination of research findings and therapeutic interventions to individuals with ASD, thereby increasing access to evidence-based training and therapies. The integration of AI and ICT in executive function training and GABA regulation research holds great promise in providing tailored and effective interventions for individuals with ASD, ultimately contributing to improved cognitive and behavioral outcomes.

Method

The method of the present study consists of a literature review on the synthesis and release of GABA. From this methodology the following findings were obtained:

GABA Synthesis and Release: GABA is synthesized from glutamate through the action of glutamate decarboxylase (GAD) and stored in synaptic vesicles. Upon neuronal stimulation, GABA is released into the synaptic cleft, where it interacts with GABA receptors in adjacent neurons, exerting inhibitory effects.

GABAergic Neurotransmission: GABA acts as an inhibitory neurotransmitter, modulating neuronal activity by hyperpolarizing neurons through the influx of chloride ions. This inhibitory action helps balance excitation and inhibition in the brain, crucial for normal brain function.

Regulation of Neurotransmission: GABAergic neurotransmission plays a modulatory role in various neuronal circuits, impacting neurotransmitter systems like dopamine, serotonin, and norepinephrine. This regulatory effect contributes to the overall balancing of neurotransmission and affects diverse physiological functions.

Results

GABA and Psychological Disorders:

Anxiety Disorders: Decreased GABA levels or impaired GABA receptor functionality contribute to enhanced anxiety symptoms.

Depression: Altered GABAergic activity is associated with depressive symptoms and mood disturbance.

PTSD: Altered GABAergic signaling may contribute to dysregulation of fear responses and emotion management.

GABA and Executive Functions:

Decision Making: GABA levels in specific brain regions influence decision-making abilities and impulsivity.

Working Memory: GABAergic transmission in the prefrontal cortex affects working memory performance.

Attention Control: GABA plays a role in attentional processes and is implicated in conditions like ADHD.

GABA and Self-Control:

Impulsivity and Self-Control: GABAergic functionality is linked to impulsivity, affecting self-control mechanisms.

Prefrontal Cortex and Self-Control: Proper GABAergic neurotransmission in the prefrontal cortex supports self-control by regulating neural activity.

GABA and Behavioral Control:

Motor Control: GABA acts within motor circuits, providing inhibitory control and regulating voluntary movements.

Impulsivity and Aggression: GABAergic impairment is associated with behavioral control, impacting impulsive actions and aggression.
Anxiety and Stress: GABAergic dysfunction influences anxiety and stress responses, affecting behavioral responses.
GABA and Attention Control:

Modulation of Neural Excitability: GABAergic inhibition contributes to balancing neural excitation and inhibition crucial for attentional processes.
Memory and Attention: GABAergic neurotransmission plays a role in working memory, attention disorders like ADHD, and sensory attention.

Discussion

This comprehensive exploration of GABA's multifaceted role in psychological and cognitive processes underscores its significance in mental health. The intricate interplay between GABAergic neurotransmission and various functions such as executive control, self-regulation, and behavioral modulation provides valuable insights into potential interventions for neuropsychiatric disorders. Further research is warranted to deepen our understanding of the complex mechanisms involved in GABA's influence on cognitive and behavioral processes, paving the way for targeted therapeutic approaches.

Conclusions

The important role of GABA in mental illness is particularly important, as it functions as an inhibitory neurotransmitter of the central nervous network and plays a vital role in the management of stress, mental status and stress response. GABAergic dysfunction has been implicated in a variety of psychological disorders such as anxiety disorders, depression and post-traumatic stress disorder (PTSD), suggesting that alterations in GABAergic neurotransmission may contribute to the pathophysiology of these conditions.

With regard to executive functions, GABAergic neurotransmission is downstream and attention deficit levels, especially in conditions such as attention deficit hyperactivity disorder (ADHD); in terms of self-control, GABA has an important role in regulating impulsivity and regulating prefrontal cortex functionality. Impaired self-control, commonly seen in substance use disorders, may be affected by changes in GABAergic neurotransmission. Furthermore, the involvement of GABA in anxiety responses and the regulation of emotional states directly affects self-control processes; behavioural control is also affected by the action of GABAergic, which controls motor control, impulsivity, aggression and stress.Altered GABAergic neurotransmission may contribute to impulsive actions and aggressive behaviours.The important role of GABA in attentional control is significant, as it contributes to selective focus and attentional regulation by balancing neuronal excitation and inhibition. GABAergic neurotransmission in brain circuits associated with attention, such as the prefrontal cortex and parietal cortex, modulates neuronal excitation and influences attentional conditions. GABA (gamma-aminobutyric acid) may play a key role in enhancing and supporting executive functions in the brain for decision making, working memory, attentional control and inhibitory control.

As an inhibitory neurotransmitter, GABA controls the excitability of nerve cells and maintains the balance between neuronal excitation and inhibition. This balance is essential for optimal cognitive functioning, including a variety of executive functions. Below is a breakdown of how GABA contributes to executive functions:
Improved decision making: GABAergic neurotransmission to brain regions such as the prefrontal cortex and striatum helps to regulate processes involved in decision making. By inhibiting excessive brain function, GABA promotes clearer and more rational decision-making. It helps individuals resist spontaneous decisions and make well-thought-out choices based on long-term goals and consequences.

Improved working memory: Working memory is critical for temporarily retaining and manipulating information in the mind. GABAergic transmission in the prefrontal cortex, a key brain region for working memory, contributes to the stability of neural networks and retention of relevant information. This results in greater potential for improved working memory and cognitive flexibility.

Optimal attention control: The GABA system contributes to attentional processes by regulating neuronal excitation in brain circuits associated with attentional capacity, such as the prefrontal cortex and parietal cortex. By maintaining the right balance between neural stimulation and inhibition, GABA helps keep individuals focused on important stimuli while filtering out distracting stimuli. This leads to improved attentional control and sustained focus on tasks.
Effective inhibitory control: GABAergic neurotransmission supports inhibitory control, which is crucial for suppressing unwanted or spontaneous responses. By limiting neural stimulation and attenuating excessive responses, GABA contributes to the inhibition of inappropriate actions of individuals and the essential regulation of their behavior.
Stress reduction: GABA has a calming effect on the brain and is involved in the regulation of stress responses. By inhibiting the excessive stimulation of nerve cells associated with stress and anxiety, GABA promotes a state of relaxation and emotional balance. This, in turn, enhances executive functions, enabling individuals to think clearly and make well-thought-out decisions even under difficult circumstances.

Cognitive flexibility: GABAergic transmission favors neurological plasticity and cognitive flexibility. This allows the brain to adapt to changes in situations and switch efficiently between tasks or

mental processes. Cognitive flexibility is essential for Problem-solving, adjusting to new environments, and multi-tasking, all of of which are integral facets of executive functions.

In summary, GABA has an important role in regulating neural circuits and ensuring optimal functionality of brain areas involved in executive functions. By maintaining neuronal balance and promoting inhibition control, GABA contributes to better decision making, working memory, attentional control and cognitive flexibility. Individuals with balanced GABAergic neurotransmission are more likely to exhibit enhanced executive functions, which implies improved cognitive performance and adaptive behavior in various aspects of life.

In the context of autism spectrum disorders (ASD), the association of GABA factor with hypersensory over-responsiveness (SOR) suggests its potential contribution to the phenotypic heterogeneity of ASD. The present study suggests that the GABA system and its associated binding network may contribute to individual variation in SOR, shedding light on the underlying mechanisms in ASD. However, more investigation is needed to provide a comprehensive understanding of the complex mechanisms involving the GABA system in ASD. Larger sampling and longitudinal studies are needed to confirm and expand these results. Also, exploring the association between GABA and other central features of ASD, such as difficulties in communicating with society and diminished interests, may further illuminate the important role of GABA in the overall symptomatology of ASD.

GABA (gamma-aminobutyric acid) has a complex and multidimensional role in Autism Spectrum Disorder (ASD). While the exact mechanisms are not yet fully understood, study has provided insights into how GABA may be involved in ASD and how it might help to address some aspects of the condition. Below are some of the ways in which GABA may be important in ASD:

Regulating the excitation-inhibition balance: GABA is an inhibitory neurotransmitter that helps regulate the balance between excitability and inhibition in the brain. In ASD, there is evidence that there is an imbalance between excitation and inhibition signalling, resulting in increased neuronal excitability. The inhibitory nature of GABA may contribute to the restoration of this balance and reduced hyperexcitatory neuronal activity, which could potentially contribute to symptom management in some individuals with ASD.

Regulation of anxiety and sensory overreaction: anxiety and over-sensitivity of the senses are common features of ASD. GABA system is associated with the regulation of stress and anxiety responses in the brain. By promoting a relaxing energy in neural circuits, GABAergic neurotransmission may help to reduce anxiety and decreased sensory hypersensitivity in people with ASD, making it easier to cope with sensory stimuli and environmental challenges.

Social functioning and communication: GABA may play a role in the development of social functioning and communication, two areas commonly affected in people with ASD. Research has suggested that GABAergic dysfunction in specific brain regions may contribute to impairments in social interaction and communication skills. Improved GABAergic activity may potentially support improvements in social responsiveness and communication skills.

Cognitive flexibility and repetitive behaviours: Cognitive flexibility, the ability to switch between tasks or ideas, is impaired in some people with ASD. The important role of GABA in regulating neuronal plasticity and facilitating cognitive flexibility may have relevance in examining this aspect of the condition. In addition, GABAergic neurotransmission may contribute to the reduction of repetitive behavioral traits that are common in ASD.

Motor control and coordination: GABA contributes to motor control and coordination. Because motor impairments are found in some individuals with ASD, optimal functioning of the GABAergic system may have consequences for the development and performance of motor skills.

It is necessary to point out that GABA is just one part of the complex puzzle of ASD and that it cannot cure the condition. The involvement of GABA in ASD likely depends on many genetic, environmental and neurobiological factors. As a result, any potential treatment targeting GABA in ASD should be carefully studied and tailored to the needs of the individual; current research on GABA in ASD is ongoing and there is much more to be learned about its exact role and potential therapeutic possibilities. As we understand more of the underlying neurobiology of ASD, there may be opportunities to develop focused interventions that may have a positive impact on certain aspects of the disorder in some individuals.

In conclusion, GABA has a multifaceted role in diverse aspects of cognitive and behavioral self-regulation, as it affects psychological disorders, executive functions, self-control, behavioral control, and attentional regulation. Further research on the mechanisms of GABA is essential to fully understand its effects and to develop specific interventions for neurological and neuropsychiatric disorders.

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