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ISSN 2959-409X

Advances in the Study of Brain Development in Psychiatric Disorders

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Abstract:

Depression and anxiety disorders are prevalent mental illnesses, and their aetiology is intricate and remains incompletely elucidated. In recent years, researchers in both domestic and international settings have conducted a substantial amount of research on the pathological mechanisms and treatment methods of depression and anxiety disorders. This paper presents a summary of the pathogenesis of depression from the perspective of brain function and structure. It identifies abnormalities in brain regions, including the hippocampus and prefrontal lobe, as being closely related to the condition. These abnormalities are manifested in a reduction in cortical area and volume, as well as impairment of neuronal morphology and ultrastructure in these brain regions. Furthermore, patients with depression are frequently linked to diminished cerebral blood flow, reduced metabolism, aberrant brain network connections and an imbalance in neurophysiological activity. In contrast, patients with anxiety disorders display functional abnormalities in the amygdala, default mode network, cognitive control network and motivational network. These clinical studies have provided new ideas for treatment, and many of them have proposed innovative treatment concepts that are worthy of further investigation and application in clinical practice. This article provides a summary of the progress of research on the brain mechanisms of depression and anxiety disorders, with the aim of providing a reference for their diagnosis and treatment. Further in-depth study of brain function and structural abnormalities in these disorders may facilitate the development of more effective treatments, thereby improving the quality of life of patients.

Keywords: Neurophysiological; Fasciculus; Benzodiazepines; Neuron; Subcortical

Overview of mental illness in adolescents:
Firstly, adolescence is a critical period for the physiological, psychological, and cognitive development of teenagers, and it is also a crucial stage for the cultivation of various abilities. However, this period is accompanied by numerous challenges and mental health risks. Adolescents have unique developmental characteristics, exhibiting a strong desire for knowledge, an awakening self-awareness, and high plasticity, which offer opportunities for the development of learning habits, social skills, and emotional management abilities. At the same time, adolescents face more challenges than children in terms of family, social, and academic issues, and are more vulnerable in physical and mental aspects. Consequently, this may lead to some mental health risks, such as parental divorce, social isolation, and academic setbacks, which can negatively impact adolescent psychological development.
Therefore, the incidence of mental disorders among adolescents is rising[1-3] ,with the rate of depression among American adolescents increasing from 8.7% in 2005 to 13.2%[4] in 2017. Most mental disorders and psychological impairments manifest during adolescence, specifically between the ages of 9 and 17, with 9 to 12 years old being the preadolescent period. Additionally, some issues have been exacerbated by the COVID-19 pandemic, which has intensified mental health problems among adolescents. A survey in Iceland revealed an increase in depressive symptoms and a decrease in psychological well-being among adolescents. Overall, if mental health problems during adolescence are not addressed in a timely manner, they may persist into adulthood, affecting physical and mental health and personal development. Currently, mental disorders are diagnosed based on the DSM-5 and ICD-10 criteria.

1 Current state of depression research

1.1 Clinical symptoms

Depression is a mental illness with a multitude of potential causes and is characterised by a pervasive and enduring depressed mood, diminished interest and pleasure. It is associated with a high prevalence of morbidity, a high relapse rate, a high suicide rate and a high disability rate. [4] The majority of research on the pathogenesis of depression has been conducted within the following areas: immunology, neurotransmitters, oxidative stress, and neurotrophic factors[5].In recent years, there has been a notable increase in the number of studies examining the pathogenesis of depression from the perspective of brain function and structure.This paper investigates the pathogenesis of depression from the perspective of brain function and structure. The following factors have been identified as contributing to the development of depression: reduction in neuronal activity, anatomical and structural neuronal morphology and ultrastructural damage, reduced blood flow, reduced metabolism, abnormal brain network connections, and imbalanced neurophysiological activity in the brain. The imbalance of neurophysiological activity in the brain is a key factor in the pathogenesis of depression. The following section provides a summary of the pathogenesis of depression.

1.2 Structural brain changes in depression

1.2.1 Hippocampus and prefrontal area and volume decrease:

Hippocampus and prefrontal area, volume reduced.Studies have shown that in depressed patients, prefrontal cortex thickness, surface area are reduced in depressed patients. Cortical thickness is influenced by the arrangement and density of neuronal cells, which reflect spatial changes in cortical structure. In contrast, cortical surface area reflects the number of columnarly arranged cells, thereby reflecting changes in cortical volume.
The volume of grey matter in the hippocampus and prefrontal lobes was found to be reduced in patients with depression, with the extent of this reduction correlating with the severity of the depressive episode.Furthermore, the degree of volume change was observed to be positively correlated with the duration and severity of depression[6].A reduction in hippocampal volume was also observed in rodent models of stress, while no clear consistency was evident in prefrontal volume changes. This may be attributed to the significant differences in cellular structure and relative size between the human and rodent prefrontal lobes, which are likely to influence the observed outcomes. The hippocampus and amygdala are closely associated and are often collectively referred to as the hippocampus-amygdala complex.
The hippocampus and amygdala are closely linked and are often collectively referred to as the hippocampus-amygdala complex. Collectively, they perform a range of emotional memory functions[7].The evidence regarding amygdala volume changes in patients with depression is inconclusive. However, in animal models of depression, an increase in amygdala volume has been observed[8] .This increase in amygdala volume is associated with increased structural covariance between this region and the rest of the brain, as well as other parts of the brain. It is also linked to increased density of synaptic protein patches and depressive-like behaviour[9].

1.2.2 Neuronal morphology and ultrastructural damage

ISSN 2959-409X

In neuropathological studies of depression, damage to neuronal morphology and ultrastructure is seen as a key factor mediating macrostructural changes in the brain and the expression of depression-like phenotypes. Autopsy studies have shown significant neuronal cell number reduction, cellular atrophy or hypertrophy, altered dendritic complexity, and synaptic loss in brain samples from depressed patients, most notably in the hippocampus and prefrontal regions. Specifically, the hippocampal region showed a reduction in neuronal cytosolic volume, an increase in cell stacking density, and a decrease in the number of mature granule cells, accompanied by a loss of glial cells, especially astrocytes[10]. In animal models of depressive-like behaviour, the volume of the nerve fibre layer and cell layer in the hippocampal region was reduced, and the density of glial cells was decreased, but no reduction in the number of neurons was observed[11]. In addition, reduced cytosol and increased stacking density of glial cells, as well as a reduction in the number of mature granule cells, were also confirmed in a rodent stress model.

In the prefrontal region, mRNA and protein levels of glial fibrillary acidic protein (GFAP) were decreased, suggesting that the alterations in astrocytes may be locally specific. At the same time, the reduced number of oligodendrocytes was associated with impaired astrocyte function. In the hippocampal CA1, CA3 and dentate gyrus subregions of depressed rats, the density of synapses was reduced, accompanied by dendritic atrophy[12].Reduced number of synapses and reduced synaptic density in the hippocampus and prefrontal lobes were consistent findings in both depressed animal models and brain specimens from depressed patients. In addition, synaptic density in the dorsolateral prefrontal, hippocampus, and cingulate cortex of depressed patients was negatively correlated with disease severity. Meanwhile, reduced expression of genes associated with synaptic function and reduced levels of synaptic signalling proteins further revealed the complexity of neuronal morphology and ultrastructural damage in depression.

In summary, neuronal morphological and ultrastructural damage in depression is manifested by alterations in cell number and volume, abnormalities in dendritic and synaptic structure, and impaired glial cell function, and these alterations provide important evidence for understanding the neurobiological basis of depression.

1.2.3 Changes in brain function in depression

Cerebral blood flow was found to be significantly reduced in patients with depressive disorders, indicating the presence of brain damage. This suggests that cognitive abnormalities may be related to this reduction in cerebral blood flow. Concurrently, metabolism was observed to be significantly enhanced, accompanied by relative hyperactivation. This may be the cause of depression. Additionally, relative over-activation of metabolism was noted, which may be another cause of mood abnormalities in depressed patients.

Concurrently, there was a notable elevation in metabolic activity, accompanied by a state of relative hyperactivation. This phenomenon may serve as an additional contributing factor to the aberrant mood states observed in individuals with depressive disorders[13].From an energy metabolism perspective, depressed mice display increased spontaneous activity and reduced energy production efficiency in the medial prefrontal lobe. This discrepancy between spontaneous activity and energy production efficiency indicates the presence of an energy metabolism disorder in depression. Electroencephalogram (EEG) studies have demonstrated a negative correlation between resting EEG lateralisation and depression levels. Furthermore, a negative correlation was observed between depressive symptoms and their severity, and ReHo values in key brain regions, as determined by brain imaging. The cingulate-prefrontal-parietal network and bilateral prefrontal abnormalities represent crucial neural substrates underlying cognitive impairment in depressed patients with cognitive deficits[14].In summary, decreased blood flow, abnormal activation, disturbance of energy metabolism and imbalance of electrical activity may be the onset of depression. The key central functional mechanism.

1.2.4 Network connection anomalies

Abnormalities in network connectivity have been observed in individuals diagnosed with depression. These individuals have been found to exhibit structural and functional connectivity disruption, as evidenced by Extensive interregional connectivity disruptions may result in a reduction of the overall network integrity in patients with depression, with the white matter fibre bundle (WMF) representing the most crucial component of the network.

Abnormalities in the integrity of the white matter fibre bundle (WMFB) may contribute to the dysfunction of cortical connectivity and subcortical areas, which in turn may give rise to depressive symptoms. It has been demonstrated that the default mode network (DMN) in patients with depression and the frontal subcortical network exhibit a higher level of white matter (WM) integrity. The DMN plays a role in emotional and self-processing processes, and WM connections are disrupted in patients with depression [15].

The DMN is involved in emotional and self-processing processes, whereas the frontal-subcortical network is crucial for emotion regulation and cognitive functions. These abnormalities may underpin the structural basis for indi-

vidual functional and behavioural deficits in depression. These abnormalities may underlie the functional and behavioural deficits observed in individuals with depression. In patients with depression, abnormalities have been observed in the connectivity of white matter tracts, including the cingulate fasciculus, the leptomeningeal bundle, the medial forebrain bundle, the anterior thalamic radiation, the corpus callosum radiation, the frontal superior longitudinal fasciculus, the inferior fronto-occipital fasciculus, the inferior longitudinal fasciculus, and the corticospinal tracts [16]. Depression is associated with alterations in emotional processing and attachment, as well as abnormal resting-state functional connectivity (FC) across multiple brain networks involved in executive function and reward processing [17]. In patients with depression, distinct FC patterns exist both within and between resting-state networks that have been altered [18]. Research indicates that individuals with depression exhibit reduced connectivity within the executive control network (ECN), while demonstrating hyperconnectivity in the default mode network (DMN). Additionally, abnormalities are observed in the ECN-DMN interaction [19]. Further studies reveal that FC impairments in depression are progressive. In first-episode depressed patients, low connectivity was noted in sensorimotor networks, DMN, and dorsal attention network; conversely, high connectivity was found within complex sensorimotor networks and prominent networks such as ECN. Notably, first-episode patients exhibited low connectivity whereas recurrent depression patients showed increased connectivity levels negatively correlated with the frequency of episodes and overall disease duration [20]."

1.2.5 Imbalance in neurophysiological activity

Electrophysiological EEG analysis enables the monitoring of changes in neural electrical activity within the cerebral cortex [21], thereby facilitating the exploration of potential biomarkers for depression, which hold significant implications for both diagnosis and treatment outcome prediction of this disorder[22]. Distinct frequencies of EEG waves correlate with various functional states of the brain. Alpha waves are indicative of resting state and relaxation levels; studies have demonstrated that patients with suicidal ideation in depressive episodes exhibit heightened alpha wave activity during nocturnal sleep [23]. The lateralization of the alpha frequency band is linked to approach-avoidance behavioral patterns and may serve as a predictor for specific clinical symptoms such as agitation and lethargy[24]. Anxiety symptoms can disrupt the lateralization of alpha frequency bands, consequently impacting diagnostic accuracy for depression [25]. Beta waves are associated with anxious states and ruminative thought processes, revealing diminished beta wave power in the left hemisphere among individuals suffering from depression[26]. Theta waves relate to emotional processing, showing increased theta wave activity in both occipital and parietal regions among depressed patients [27]. Gamma waves correspond to sensory processing and emotional fluctuations[28]; optimal gamma wave power contributes to maintaining emotional stability in those affected by depression [29]. Delta waves are connected to deep sleep states, where larger delta wave amplitudes are recorded at central parietal and lateral electrodes when confronted with negative stimuli by depressed individuals [30].

In ERP (event-related potential) research, diverse tasks such as presenting emotional faces or engaging working memory can elucidate distinct brain functions within depressive patients [31]. ERP represents a specialized form of brain-induced potential generated upon exposure to specific stimuli (visual, auditory, or tactile), reflecting neural electrophysiological alterations during cognitive processes[32]. The P300 component serves as an exemplar of endogenous ERPs closely tied to cognitive psychological processing; it is frequently employed to evaluate cognitive function impairments in individuals diagnosed with depression [33]. The latency associated with P300 indicates the speed at which neural conduction occurs in response to external target stimuli, serving as an indicator.

1.3 Clinical treatment of depression

Depression is a prevalent mental health issue, and its clinical management primarily encompasses pharmacological interventions, psychological therapies, physical treatments, and lifestyle modifications. In terms of pharmacological treatment, Tricyclic Antidepressants (TCAs) such as Amitriptyline enhance mood by inhibiting the reuptake of neurotransmitters. Selective Serotonin Reuptake Inhibitors (SSRIs) and Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs), including Sertraline and Escitalopram, are frontline medications for depression with relatively mild side effects[34]. Ketamine and its isomers, like Esketamine, offer rapid and durable antidepressant effects but are associated with issues of drug dependence and adverse reactions, currently being used mainly for treatment-resistant depression[35]. Probiotics have shown potential in alleviating symptoms but require further research[36].

Psychological therapies involve Cognitive Behavioral Therapy (CBT), Interpersonal Psychotherapy[37], and Acceptance and Commitment Therapy (ACT), aiming to alleviate depressive symptoms by altering negative thought patterns and improving interpersonal relationships[38].

Physical treatments such as Electroconvulsive Therapy (ECT) and Repetitive Transcranial Magnetic Stimulation (rTMS) stimulate the brain to enhance mood, with ECT

ISSN 2959-409X

having significant side effects and typically used for refractory depression[39]. Lifestyle interventions, including exercise, Baduanjin and Wuqinxi, mental health education, and family support, are crucial for improving mood and cognitive functions[40].

In summary, the treatment of depression should be tailored to the individual's specific circumstances, often combining pharmacological and psychological approaches, while also emphasizing the importance of lifestyle changes and psychosocial support. As our understanding of the pathogenesis of depression deepens, more effective treatments may emerge in the future[41].

2.Current state of research on anxiety disorders

Anxiety disorders are complex disorders whose pathogenesis involves epigenetics, biochemical pathology, etc. Epigenetic mechanisms such as DNA methylation, histone modification, and altered miRNA expression, as well as biochemical pathologies such as neurotransmitter and neuroendocrine dysfunction, are closely related to the development of anxiety disorders. A variety of treatments are available, including antidepressants, buspirone, and cognitive behavioural therapy. In addition, epigenetic pharmacological treatments such as histone deacetylase inhibitors have demonstrated good anxiolytic effects. Further in-depth studies are needed in the future to elucidate the epigenetic mechanisms and biochemical pathological mechanisms of anxiety disorders and to provide new ideas and directions for the development of more effective treatments.

2.1 Anxiety symptoms

Anxiety disorder is a neurosis characterized by excessive anxiety, including Generalized Anxiety Disorder (GAD), Panic Disorder, etc. Among them, GAD is the most common, often manifesting as a sense of unease and fear without any factual basis or a specific object or content, accompanied by symptoms of the autonomic nervous system (such as dizziness, chest tightness, palpitations, breathing difficulties, dry mouth, urinary difficulty, nausea, vomiting, constipation, etc.) and muscle tension, as well as restlessness of movement, which not only brings great psychological suffering and physical discomfort to the patient, seriously affects the patient's quality of life and social function, but also brings a heavy burden to the patient's family. With the continuous progress of society, the competition and survival pressure faced by people are increasingly heavy, and the pace of China's aging population is also accelerating, so the incidence of anxiety disorder is gradually increasing.

2.2 Brain Network Changes in Anxiety Disorders

2.2.1 emotional network

The emotional network is composed of the amygdala, anterior cingulate cortex, hippocampus, and inferior

It consists of the thalamus, insula, orbitofrontal cortex and nucleus accumbens. Specific neural networks are involved in emotional processing and mediating motivational behaviors [42].The amygdala is considered to be the core region in this network [43]. in Early on, this structure was thought to be involved in individuals' fear processing,Because it plays a significant role in fear conditioning [44]. In the meta-analysis conducted by Etikin et al., it was found that SAD patients were more likely to face

overactivation of the amygdala and insula is common during negative emotional stimulation live [45]. basolateral amygdala, the connection between the BLA) and the ventral hippocampus also affects the individual's strip fear and anxiety [46]. In addition, Liao and others in order to apricot

Kernel as seed point to study the characteristics of brain functional connectivity in SAD patients. It was found that the connection between the amygdala and the frontal lobe and orbitofrontal region was enhanced [47]. Orbitofrontal cortex is involved in the development of social cognition [48]. This result is mentioned this suggests that cognitive dysfunction may be the result of emotional processing disorders in SAD patients. An important reason. But further research found that almond nuclear energy more broadly detect relevant information in the surrounding environment, in relation to uncertainty. In this case, the amygdala is also activated [49]. anxiety-based. The uncertainty and anticipation model of anxiety (UAMA), including social anxiety disorder. Patients with anxiety disorders are included in ambiguous social situations or tables Overactivation of the BLA as well as the BLA and anterior cingulate occurs in both cases. TThe line is associated with unpleasant results, thus forming a bearing. Threatening expectations make ambiguous materials more likely to be associated with threats,Tie together [50-53]. And specifically in the case of social assessment, there are Individuals with social anxiety develop expectations and fears about potential social judgments

Worries. This underlying social judgment creates an inherent ambiguity. And unpredictable circumstances. Studies have shown that social anxiety disorder is associated with difficulty tolerating the level of uncertainty associated with this relationship indicates ambiguity or uncertainty is particularly important for the formation and maintenance of the disorder Key [54-55]. This also supports the amygdala's role in maintaining and maintaining social anxiety. An important role in the development process. Response

to uncertainty in the face of individuals. In addition to the role of the amygdala itself, the latest research also found the bed nucleus terminalis (BNST), now in the epitaxy of the amygdala, also plays a important role [56]. The study measured different ranges using fMRI technology. Degree of social anxiety individuals' neural response to unpredictable cues and images react. The results showed higher levels for unpredictable cues social anxiety is associated with weaker BNST-amygdala functional connectivity.

For unpredictable images, higher levels of social anxiety and BNST was stronger in relation to the ventromedial prefrontal cortex and posterior cingulate cortex functional connectivity and weaker functionality between BNST and the central posterior gyrus connection related [57]. Although there is a lot of research on emotional networks, especially needles. The role of amygdala in the occurrence and development of SAD is discussed. Many new understandings, but some problems remain. For example, Youyan

It is mentioned that the amygdala has a lateralization characteristic [58], and thus the apricot is left and right. The emotional processing function of the kernel is not exactly the same, but it is in less attention has been paid to the present research; In addition, the amygdala itself is made up of the basolateral nuclear group and the medial cortical group are two parts, each part. The role of almonds in the emotional processing of SAD patients. The functional connections within the nucleus are not yet more clearly explained, while this is most likely the result of previous research on the amygdala

The important reasons for the present inconsistency can be explored in greater detail in the future. Discuss the left and right amygdala, as well as the inner amygdala nuclear group pairs in the SAD shape. The role played in the process of becoming and developing.

2.2.2 Default Network

The default mode network (DMN), traditionally composed of the posterior cingulate cortex (PCC) and the medial prefrontal cortex (mPFC), forms a unique circuit that is more active during rest than during cognitive tasks. The DMN plays a critical role in an individual's social cognition and self-referential processing (SRP), as well as in the processing of social emotions [59]. Studies have shown that individuals with social anxiety disorder (SAD) exhibit increased self-referential thinking and negative beliefs, and that somatosensory information also affects their anxiety levels and cognitive states [60-61]. A functional magnetic resonance imaging (fMRI) study using a self- and other-reference paradigm combined with praise and criticism stimuli directly investigated SRP in SAD patients. Compared to controls, SAD patients showed increased activation in the mPFC and amygdala when exposed to self-reference criticism, and functional connectivity between the mPFC and amygdala was significantly stronger compared to healthy controls [62]. Another task-based fMRI study using a negative self-belief paradigm found increased activation in the mPFC, PCC, cuneus, angular gyrus, inferior parietal lobule, amygdala and parahippocampal gyrus in SAD patients when responding to negative self-beliefs [63]. However, due to differences in experimental paradigms and stimulus materials used in early neuroimaging studies, the reported activation patterns were not entirely consistent. Therefore, some researchers advocate the use of more standardised experimental paradigms and validated emotional stimuli to test the neural mechanisms related to SAD [64]. Recent fMRI studies utilizing the standardized Muenster Social Anxiety Picture Set (SAPS-M) have revealed increased activation in brain regions associated with social reference processing (SRP), such as the insula, cuneus, and dorsal anterior cingulate cortex, among patients with Social Anxiety Disorder (SAD) when responding to disorder-related scenes. Concurrently, enhanced functional connectivity was observed between the posterior cingulate cortex/precuneus and insula, as well as between the posterior cingulate cortex/precuneus and cortical areas including the dorsal anterior cingulate cortex, indicating a central role for the posterior cingulate cortex/precuneus in processing SAD-related stimuli [64-65]. Resting-state fMRI investigations also suggest abnormalities in default mode network (DMN) functional connectivity within SAD patients [66]. A study employing regional homogeneity (ReHO) analysis—reflective of temporal synchrony of regional BOLD signals—demonstrated reduced consistency within the DMN (including medial prefrontal cortex and cuneus) in SAD patients compared to controls [67]. Another graph theory-based fMRI investigation identified diminished resting-state functional connectivity strength across both hemispheres of the cuneus in individuals with SAD [68]. Furthermore, there was a significant negative correlation between resting-state functional connectivity strength of the cuneus and disease duration, highlighting its potential involvement in the neuropathophysiology of SAD [69]. These findings underscore that the cuneus serves as a pivotal hub within the DMN and plays an essential role in SRP [67]. Some researchers categorize brain regions such as medial prefrontal cortex, posterior cingulate cortex, and cuneus as cortical midline structures (CMS), which are integral to SRP processes while sharing characteristics with intrinsic DMN activity . Presently, research on DMN's role in SAD development primarily concentrates on elucidating SRP mechanisms among affected individuals; however, it has yielded relatively consistent outcomes. Nonetheless, SRP itself is a multifaceted mechanism encompassing sensory processes, attentional

ISSN 2959-409X

dynamics, and cognitive functions. Current inquiries have been less focused on delineating specific mechanisms underlying SRP or identifying neural correlates.

2.2.3 cognitive control network

The cognitive control network (CCN) includes the dorsolateral prefrontal cortex (dlPFC), dorsomedial prefrontal cortex (dmPFC), and the dorsal regions of the parietal cortex[70], which are responsible for „top-down" control functions, regulating subcortical activity, and controlling higher cognitive functions such as working memory, executive control, and task switching. The prefrontal cortex, especially the orbital frontal cortex (OFC) and the ventromedial prefrontal cortex (vmPFC)[71], plays a crucial role in regulating negative emotions and fear responses, and adjusting emotional responses during cognitive reappraisal, thereby changing the emotional meaning of the situation and generating social support and psychological well-being[72].

Social anxiety disorder (SAD) patients have difficulty utilizing the cognitive control prefrontal cortex network in cognitive reappraisal tasks, resulting in reduced activation in the dlPFC, dmPFC[73], anterior insula, posterior cingulate cortex, and bilateral dorsolateral parietal cortex. Studies have shown that effective attention bias modification (ABM) training can reduce the gray matter volume of the expanded amygdala and the ventromedial prefrontal cortex network in SAD patients[74], and reduce individual attention bias.

The prefrontal cortex is connected to multiple regions in the emotional and motivational network and can regulate processes in these regions. For example, social anxiety is positively correlated with the gray matter volume of the OFC and the functional connectivity between the OFC and the amygdala[75], indicating that the OFC plays a key role in regulating the amygdala's response to negative emotions and may be a compensatory mechanism for reducing attention avoidance and promoting effective emotion regulation. The dorsomedial prefrontal cortex (dACC) and the dorsolateral prefrontal cortex (DPC) are also closely linked to the reappraisal process in emotional regulation[76], and the functional connectivity between the amygdala and the prefrontal cortex and the anterior cingulate cortex weakens after cognitive behavioral therapy, indicating a reduction in symptoms and possibly being highly related

2.2.4 motivation networ

The motivation network is characterized by the ventral striatum receiving dopamine signals from the ventral prefrontal cortex in response to threatening stimuli or rewards. This network includes several key areas: the ventral tegmental area (VTA)[77] ,striatum, orbitofrontal cortex, ventromedial prefrontal cortex, and anterior cingulate cortex. Together, these regions create a midbrain dopamine pathway that reacts sensitively to both reward magnitude and likelihood[78]. Dopamine activity is crucial within this circuit as it facilitates complex processes like reinforcement learning and reward anticipation; hence, this „motivation network" is frequently termed the „reward network[79]."

Research utilizing brain imaging has led some scientists to propose a theoretical framework suggesting that one aspect of social anxiety disorder (SAD) in adolescents involves atypical processing of anticipated social reward cues. Specifically[80], adolescents with SAD exhibit an amplified response pattern in the caudate nucleus and putamen areas of the striatum, indicating heightened sensitivity to rewards along with increased self-evaluation and an exaggerated expectation of positive outcomes[81]. Previous magnetic resonance studies have indicated that the ventral striatum serves as a central component of both motivation and reward circuits by signaling incorrect behaviors while also initiating approach actions[82]. An MRI study focusing on expectations regarding social rewards and punishments among adult SAD patients found diminished connectivity between the putamen and anterior cingulate cortex during tasks involving these elements compared to control subjects[83]. This suggests that individuals with SAD may not display typical preferences for social rewards.

Moreover, it appears that neural mechanisms governing cortical reward processing might be disrupted for those with SAD when anticipating social rewards. Recent investigations have expanded upon earlier findings by introducing what's known as "sensitivity shift theory" (SST). This theory seeks to explain why individuals suffering from SAD—despite being highly sensitive to potential rewards—often lack motivation for pursuing them socially. It highlights how positive affect functions as a motivational system critical for adolescent interactions. For those experiencing social anxiety, learned associations can lead them to develop avoidance responses due to their predictions about rewarding outcomes becoming misaligned with actual experiences. Consequently, this disconnect results in diminished enjoyment derived from social engagements over time; thus creating an environment where negative emotional consequences are linked closely with such interactions.

This disconnection often leads affected individuals to feel less pleasure during social encounters which adversely impacts their expectations surrounding positive emotions' motivational roles. The SST also accounts for previously noted increases in striatal responses towards standard

rewards among younger people diagnosed with SAD but observes declines into adulthood. Current investigations into brain regions associated with this motivation network primarily aim at understanding how symptoms of SAD emerge and evolve through individual development stages. However, many existing studies rely on cross-sectional data which aggregates characteristics across various age groups rather than longitudinally tracking changes within specific patients over time—a method particularly relevant given that clinical onset typically occurs during adolescence. Future research should prioritize long-term follow-up assessments of individuals diagnosed with SAD so we can better understand whether alterations occur within their motivation networks as they progress through different life stages.

2.3 Treatment of anxiety disorders

2.3.1 medication

At present, drug therapy is the most commonly used method in the treatment of anxiety disorders. However, although traditional benzodiazepines (such as Clonazepam, diazepam and alprazolam) have anti-anxiety effects, they are easy to cause sleepiness, liver function damage and other adverse reactions, and long-term use is easy to form dependence, tolerance and drug withdrawal reactions, which limits their use.

Alternative drugs: Trazodone: Compared with alprazolam, trazodone has fewer adverse reactions and no withdrawal reaction, and is suitable for long-term treatment of patients with generalized anxiety disorder (GAD).

Buspirone: As a 5-HT1A receptor agonist, Buspirone plays an anti-anxiety role by regulating neurotransmitter levels, and has a high safety, but it takes some time to show efficacy, and is usually used in combination with other drugs.

Antidepressants: A variety of antidepressants (such as mirtazapine, Venlafaxine, escitalopram, sertraline) have a good effect on anxiety, and will not form dependence, can be used for a long time. Among them, Venlafaxine and sertraline are considered to be effective and have fewer adverse reactions. Atypical antipsychotics: Atypical antipsychotics (such as quetiapine) may improve anxiety symptoms as synergists, but their efficacy is controversial and needs to be confirmed by further research.

According to the individual differences of different patients, develop personalized treatment plans. Strengthen research on epigenetic mechanisms and biochemical pathological mechanisms of anxiety disorders to develop more effective treatments.

Explore new therapeutic approaches and interventions, such as epigenetic-based therapies, neuromodulation techniques, etc[84].

2.3.2 psychotherapy

As people‘s attention to mental health gradually increases, psychological

The use of therapy in the treatment of anxiety disorders is also gaining traction among clinicians Take seriously. Psychotherapy doesn‘t have to worry about medication one way or the other[85].

The mechanism of action is also significantly different from that of drug therapy. Treatment methods include psychoanalytic therapy, cognitive behavioral therapy, and afterthought Substitution therapy, family therapy, Morita therapy, group therapy, etc. It has been proved that among the many psychotherapeutic methods, Cognitive-behavioraltherapy, Through clinical studies, it is proposed that cognitive behavioral therapy for anxiety disorder can significantly improve patients‘ social function, quality of life and satisfaction with life, and has a good effect on anxiety disorder patients. It is believed that cognitive behavioral therapy can be an effective psychological treatment for GAD. Cognitive behavioral therapy believes that the abnormal psychological behavior of patients is mostly related to the patients‘ bad cognition and thinking mode, and the purpose of treatment is to correct these unreasonable cognition. For anxiety disorder, the cognitive mode is that patients are excessively worried about physical and mental danger. In clinical practice, how to apply cognitive behavioral therapy flexibly to correct the negative cognition of patients with anxiety disorder needs the continuous accumulation of experience by psychotherapists[86].

3. summary

This article provides an in-depth look at advances in brain development research on adolescent mental illness, particularly depression and anxiety disorders. It is found that brain structural and functional changes play a key role in the pathogenesis of depression.

Next is a summary of depression: Depression is closely tied to functional and structural abnormalities in key brain regions, particularly the hippocampus and prefrontal cortex, which show reduced volume and neuron count, along with morphological and ultrastructural damage. Brain network connectivity in depression is characterized by disruptions in both functional and structural connections, affecting neuronal electrical activity conduction and integration, and leading to central internal environment disorders and depressive symptoms. The altered hippocampal-prefrontal connectivity may also contribute to cognitive deficits in depressed individuals. While the relationship between structural and functional abnormalities

ISSN 2959-409X

remains unclear, depression is also associated with abnormal topological organization of brain networks, including disruptions in regional connectivity. Future multimodal imaging studies are needed to elucidate the topological relationships between these structural and functional abnormalities in depression.

Next is a summary of neurosis: numerous clinical trials have confirmed that a variety of antidepressants and psychotherapies have good efficacy in the treatment of anxiety disorders and do not induce somatic dependence, showing good prospects for use. Nowadays, few doctors use benzodiazepines alone to treat anxiety disorders in clinical practice, and benzodiazepines are mostly used for short-term treatment, while SSRIs or SNRIs have become the main first-line drugs for treating patients with anxiety disorders. It should be noted that each drug has its advantages and disadvantages in antidepressant treatment, which requires the clinician to focus on the patient according to the actual situation of the patient. The use of psychotherapy in patients with anxiety disorders has also been reported. Psychotherapy in patients with anxiety disorders has been repeatedly reported, and the empirical summary has laid a good foundation for the next step of large-scale clinical application. In addition, atypical antipsychotics may play a role in enhancing the efficacy of antipsychotics, but the conclusions of current studies are not consistent. In the future, we may be able to set up a scientific and large-sample controlled trial to confirm this further.

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