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Genetic and Environmental Factors Involved in the Pathogenesis of Anxiety Disorders

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ABSTRACT

Background: Emotions such as anxiety and fear trace their origins back to ancient times, when they served an essential evolutionary function: namely, they contributed to appropriate responses to external stimuli that posed a threat to life or health, or even directly determined survival (for example, fleeing from a predator).

Methodology: A review was conducted using PubMed. Search terms included: anxiety, anxiety disorders, chronic stress, fear. Studies published between 2021 and 2025 were reviewed.

Results: Anxiety disorders and specific phobias have their roots in a stage of evolution when fear served as a warning mechanism and triggered an appropriate response to external threats. We speak of anxiety disorders when anxiety persists, is disproportionate to the situation—appearing in situations that do not pose a real threat—or significantly impairs an individual's daily functioning, causing psychological distress. We attribute the underlying causes to mechanisms at the level of CNS neurotransmission, at the genetic level, as well as to an

individual's development and adverse environmental factors to which they are exposed from the very earliest stages of life—that is, beginning in the prenatal period.

Conclusions: Anxiety disorders have multiple causes and many predisposing or triggering factors, ranging from those encoded in genes, through hormonal imbalances, to environmental factors such as stress experienced in early life.

Keywords: anxiety, anxiety disorders, stress, risk factors

Introduction: Fear was an emotion that played a very important role in evolutionary mechanisms, as it ensured an appropriate response to actual or potential threats, thereby enabling the survival of the individual and the species as a whole. States of anxiety and heightened alertness are regulated by neural networks in many areas of the brain. When this regulation is disrupted, anxiety becomes excessive or completely inappropriate to the situation in which it is experienced, or it occurs too frequently, thereby impairing an individual's daily functioning and negatively affecting many areas of life. Risk factors are primarily genetic and environmental, and the development of anxiety disorders is underpinned by specific, complex neurobiological mechanisms, including neurotransmitter imbalances. Anxiety disorders are among the most commonly diagnosed psychiatric disorders today. Among the main anxiety-related disorders, we distinguish panic disorder, social anxiety disorder, and specific phobias. The latter refer to anxiety experienced during specific situations or in response to a specific stimulus, which impairs functioning or leads to the conscious avoidance of situations that trigger it. We also distinguish generalized anxiety disorder, which is defined as the presence of persistent and generalized anxiety that is not, however, associated with any specific factor. This anxiety is characterized by an inappropriately high intensity—it does not correspond to situations posing a real threat—and its key symptom is constant worry about everyday matters. Another term used to describe this type of anxiety is "free-floating anxiety." [1,2,12,14,16,18].

DISCUSSION

The Neuroanatomical Basis of Anxiety Disorders and Neurotransmitter Disorders

Specific structures in the brain are responsible for all mental functions and their potential disorders. This is very important information because, by understanding the neural pathways responsible for a given disorder, we can select appropriate medications and incorporate pharmacotherapy into treatment alongside psychotherapy. In the case of anxiety disorders, this

area remains poorly understood, but we do have some knowledge of neuroanatomy [5]. The heritability of anxiety is closely linked to genes expressed in the limbic system, the cerebral cortex, the cerebellum, the metencephalon, the entorhinal cortex, and the brainstem [4].

It is well known that the amygdala plays a significant role in both anxiety disorders and autism spectrum disorders. However, the relationship between the specific developmental trajectory of this anatomical structure in individuals with autism spectrum disorders and the presence of anxiety disorders in this population has so far been examined in only a limited number of studies. It has also been suggested that the social deficits observed in the autism spectrum are rooted in changes in the anatomical structure and functioning of the amygdala, as analyzed in magnetic resonance imaging studies conducted on children and adolescents on the autism spectrum. The study yielded the following results: larger, smaller, or no differences in amygdala volume were observed. From a developmental perspective, converging evidence obtained using central nervous system imaging and histological methodologies suggests that the amygdala in individuals with autism exhibits an initial, excessively rapid increase in volume during early childhood, followed by a slowed growth trajectory until physical adulthood is reached.

Despite high rates of anxiety among individuals with autism spectrum disorder, only three studies have examined the associations between amygdala structure and anxiety in autism. The first of these found that increased volume of the right amygdala is associated with a higher prevalence of anxiety and depressive disorders among children and adolescents on the autism spectrum. A second, recent study found no such association at all. A third study compared two groups of children with autism who had been diagnosed with anxiety according to the DSM and children without this diagnosis, and demonstrated that children with autism who had been clinically diagnosed with anxiety according to the DSM had a smaller volume of the right amygdala than children without anxiety. The relationship between the anatomical structure of the amygdala and anxiety disorders is therefore evident, although this cannot be confirmed in all patients with anxiety. The 5-HTTLPR serotonin transporter polymorphism, which is associated with amygdala reactivity—that is, its functioning in response to external circumstances to which an individual is exposed, such as physical or psychological stress—may influence susceptibility and, consequently, the development of anxiety. Brain-derived neurotrophic factor also plays an important role; it is crucial for neuronal plasticity in the hippocampus and may be associated with the consolidation and generalization of fear. Children with autism spectrum disorder are significantly more likely to develop anxiety disorders [5,20].

The Role of Sleep Quality, its Dysregulation and Microbiome-Gut-Brain Axis in the Development of Anxiety Disorders

Sleep disorders, including insomnia, and other psychopathological conditions, such as mood and anxiety disorders, share many similarities, both in terms of characteristic symptoms and the proposed pathophysiological models underlying the pathogenesis of these disorders. For example, the concept of hyperarousal resulting from the activity of the cortisol, noradrenergic, and orexin systems—a key model in research on insomnia—has also been proposed in psychophysiological models of anxiety and depression. Furthermore, sleep continuity disturbances occur in most mental disorders, and the anxiety disorders we are discussing are no exception. Consequently, insomnia—which constitutes a form of physical stress on the body and disrupts the functioning of the nervous and endocrine systems—qualifies as a risk factor for anxiety disorders. However, it should be noted that sleep disturbances can also arise secondary to anxiety disorders. The REM phase is a particularly well-studied phase of sleep. Depressive disorders are characterized by an increase in the number of micro-awakenings and a general reduction in the duration of REM sleep, which negatively affects overall sleep quality [3]. Based on research findings, the relationship between insomnia and depression and anxiety is bidirectional. Indeed, insomnia has been shown to be a significant predictor of depressive and anxiety disorders. It has even been demonstrated that early treatment of insomnia can prevent mood and anxiety disorders. Nevertheless, it remains difficult to understand the exact mechanism by which REM sleep is linked to the pathogenesis of anxiety at the neurobiological level. Studies indicate that fragmented REM sleep may disrupt the nocturnal adaptation of limbic circuits, leading to difficulties in coping with distress. In contrast, an undisturbed REM phase facilitates the maintenance of synaptic plasticity and allows for the integration of emotional memories, which effectively contributes to maintaining an individual's emotional balance. Patients with generalized anxiety disorder (GAD) often report severe sleep disturbances. Recently, calcium homeostasis has attracted attention due to its role in regulating sleep-wake cycles and anxiety symptoms. A cross-sectional study was conducted in which 211 patients were assessed using the Hamilton Anxiety Scale, the Pittsburgh Sleep Quality Index, and the Insomnia Severity Index. Three parameters were measured in blood samples: calcium, vitamin D, and parathyroid hormone (PTH) levels. An analysis was conducted to determine the association between HAM-A, PSQI, and ISI scores and peripheral markers of calcium homeostasis disorders. The analyses revealed significant correlations between HAM-A, PSQI, and ISI scores, PTH, and vitamin D. Regression models showed that patients with generalized

anxiety disorder who had low vitamin D levels and higher parathyroid hormone levels were characterized by poor subjective sleep quality and higher levels of anxiety, indicating a greater psychopathological burden. A strong association was highlighted between peripheral biomarkers of calcium homeostasis disorders, insomnia, poor sleep quality, and anxiety symptoms [3,18]. The latest evidence suggests that the composition and overall health of the gut microbiota are significantly associated with anxiety disorders. To date, primarily small cross-sectional studies suggest that gut dysbiosis—specifically, reduced abundance of bacterial taxa such as *Eubacterium*, *Coprococcus*, and *Faecalibacterium*—may play a key role in the pathophysiology of anxiety disorders, particularly generalized anxiety disorder (GAD). Although probiotic interventions show promising potential for alleviating symptoms, current scientific evidence is largely based on observations in non-clinical populations, while data regarding anxiety disorders with panic attacks, agoraphobia, and social anxiety disorder remain scarce or virtually nonexistent. Therefore, there is an urgent need for large-scale prospective studies and randomized controlled trials to unequivocally assess the efficacy of targeted microbiome-based therapies in patients with formally diagnosed mental disorders. The bidirectional communication network between the gastrointestinal tract and the central nervous system involves numerous physiological pathways that are inextricably linked to the pathogenesis of anxiety disorders. Extensive preclinical data indicate that the gut microbiota plays a fundamental role in early neurological development and in the regulation of the hypothalamic-pituitary-adrenal (HPA) axis throughout life. Disruptions to the microbiome early in life—such as exposure to antibiotics, cesarean delivery, or chronic stress—can trigger lasting, maladaptive changes in physiological stress responsiveness. Furthermore, these resident microbial populations exert profound, lifelong immunomodulatory effects on the host's systemic and central immune systems. In addition to regulating immunity and stress, gut bacteria significantly regulate tryptophan metabolism, thereby influencing both serotonin availability and the neuroactive kynurenine pathway, which is often disrupted in conditions such as social anxiety. Microbial metabolites, particularly short-chain fatty acids (SCFAs) derived from the fermentation of dietary fiber, act as key biological mediators facilitating this communication between the gut and the brain. These SCFAs are crucial for maintaining the integrity of both the intestinal mucosa and the blood-brain barrier, while also modulating local immunity, neurotransmitter concentrations, and the expression of neurotrophic factors. Individual SCFAs, such as acetate, directly stimulate the functional maturation of central microglia, while the microbiome as a whole exerts a strong influence on the endocannabinoid system, protecting against stress-induced behavioral changes. Furthermore, it has been shown

that microbial signaling regulates levels of brain-derived neurotrophic factor (BDNF), actively promoting neurogenesis in the hippocampus and, consequently, influencing emotional and cognitive regulation. Ultimately, this wide range of interrelated biological mechanisms underscores the immense complexity of the microbiome–gut–brain axis and its multifaceted contribution to the etiology of anxiety disorders [17].

Genetic and epigenetic factors in the development of anxiety disorders

An interesting study was also conducted on the role of genetics in the development of anxiety disorders. It utilized data from over 1.2 million participants, including 97,383 cases, to investigate the genetics of anxiety disorders across five continental groups. Fifty-one anxiety-associated loci were identified, 39 of which were previously unknown. Furthermore, polygenic risk scores derived from individuals of European descent were found to be associated with the prevalence of anxiety disorders in African, mixed-heritage American, and East Asian participant groups as well. Whole-transcriptome and whole-proteome analyses identified 115 genes directly associated with the experience of anxiety and the development of anxiety disorders. Anxiety also showed global and local genetic correlations with depression, schizophrenia, and bipolar disorder, as well as widespread pleiotropy across several areas of physical health [4]. Twin studies suggest that the development of anxiety is 35–40% genetically determined, which seems to be a significant percentage [6]. Although research conducted over the past few decades has identified neural circuits associated with the symptoms of anxiety disorders, it remains unclear exactly how genes associated with AD are expressed in the central nervous system of patients with anxiety disorders in terms of time and space.

An interesting clinical study was conducted in which the results of genome-wide association studies on four subtypes of human anxiety disorders—generalized anxiety disorder, social anxiety disorder, panic disorder, and obsessive-compulsive disorder—were compared and integrated with spatial patterns of gene expression. The study revealed a new classification of genes associated with these disorders, characterized by a marked increase in expression in the brain nuclei, limbic cortical areas, and the midbrain.

Furthermore, each group of these genes was associated with specific anxiety-related behaviors, and a negative correlation was demonstrated in the temporal patterns of gene expression at various stages of development.

Furthermore, the specific brain regions where expression was enriched in each gene group overlapped with neural circuits previously associated with negative decision-making and anxious personality. These results suggest that these two distinct gene clusters may underlie separate neural circuits involved in anxiety. It is estimated that anxiety disorders—specifically panic disorder, generalized anxiety disorder, and anxiety neurosis—are hereditary in approximately 32–67% of cases. In particular, first-degree relatives of affected individuals are at high risk of developing these disorders. The genetic basis of anxiety disorders has previously been investigated using targeted gene sequencing and genome-wide association studies (GWAS), which have identified mutations in (or near) several genes and captured many aspects of anxiety that are crucial for understanding its etiology [8].

Mental disorders are significantly more common among patients diagnosed with an intellectual disability, especially among those with mild cognitive impairments or borderline intellectual functioning. Approximately half of these cognitive impairments result from genetic factors, which are highly diverse and are often associated with the development of other neurodevelopmental and neuropsychiatric disorders. Many of these shared genetic abnormalities disrupt fundamental biological mechanisms, particularly the regulation and stability of neuronal synapses.

Research has shown that changes in genes responsible for synaptic function, including those in the *SHANK* gene family, are strongly associated with autism spectrum disorders, developmental delays, and various psychiatric symptoms. This is because three genes belonging to the SHANK gene family encode key structural proteins essential for the proper functioning and maintenance of neural connections. Missing or duplicated segments of genetic material involving these specific genes are consistently identified in patients with autism spectrum disorders and varying degrees of cognitive impairment.

Recent advances in comprehensive clinical sequencing have also enabled the detection of single-nucleotide polymorphisms in all three *SHANK* genes.

Although it is well known that structural mutations in the *SHANK3* and *SHANK2* genes cause significant cognitive and speech delays alongside autism spectrum disorders, pathogenic variants in the *SHANK1* gene remain extremely rare in the medical literature. These research findings suggest that genes and mutations in these genes play a significant role in the development of neurodevelopmental disorders, even though this relationship is often indirect. An interesting research analysis was also conducted to elucidate the causal relationship between genes associated with palmitoylation and the pathogenesis of generalized anxiety disorder (GAD), with a particular focus on immune-mediated pathways. The researchers identified quantitative trait loci associated with palmitoylation expression by comparing known genes responsible for palmitoylation with extensive genome-wide datasets. They then applied a two-sample Mendelian randomization method to assess potential causal associations between these specific genetic variants and generalized anxiety. To ensure robust validation of the final therapeutic targets, the researchers used Mendelian randomization based on summary data. A preliminary analysis successfully identified twenty-two eQTL loci associated with palmitoylation, of which five distinct genes—PPT2, ZDHHC5, ZDHHC13, ZDHHC20, and ZDHHC24—showed a statistically significant association with generalized anxiety disorder. Subsequent rigorous validation using the SMR method confirmed that ZDHHC5 and ZDHHC13 are the definitive target genes associated with this disorder. In summary, ZDHHC5 directly promotes the development of GAD through targeted immunomodulation of CD4+ T cells. Ultimately, these findings provide deep insight into the neuroimmune mechanisms underlying anxiety disorders, identifying the ZDHHC5/CD4+ T-cell pathway as a highly promising new biomarker and therapeutic target for future pharmacological interventions in psychiatry [15,16].

The Relationship between Cardiovascular Diseases and the Hormonal Disorders and Anxiety

Another interesting aspect is the link between hormonal disorders and anxiety. Studies have shown that rats that have undergone ovariectomy and are administered exogenous estradiol exhibit greater fear generalization compared to those that do not receive estradiol following the procedure. Furthermore, higher levels of estradiol in humans are associated with greater fear extinction. These findings suggest that during the perinatal period, mothers may be more susceptible to developing excessively generalized fears when exposed to traumatic stimuli, real

threats, or—in the case of pathologically excessive anxiety—to factors that do not pose a real threat. Certainly, the role of this phenomenon can also be traced back to evolution, as a mother who is "overly sensitive" and anticipates danger potentially has a better chance of ensuring the safety of her offspring [6]. The effect of including various protein sources in the daily diet on women's mental well-being was also investigated and the results showed that women who consumed more animal-based protein were more susceptible to exhibiting symptoms of depression and anxiety, while no significant relationship was shown between plant-based protein and these mental disorders. This allows us to hypothesize that vegan and vegetarian diets may prove helpful in supporting the treatment of anxiety disorders [7].

Several studies have been conducted to analyze the mental and psychological symptoms that occur following complications related to the course of pregnancy and childbirth itself.

These analyses focused on postpartum depression and anxiety disorders. For example, it has been shown that preterm birth and cesarean section increase the risk of depression, anxiety disorders, dysphoria, and stress in the immediate postpartum period. It is believed that the impact of perinatal complications, as well as uncomplicated pregnancies and childbirths—whether natural or by cesarean section—increases the risk of developing anxiety disorders due to psychological stress associated with caring for the child, disrupted circadian rhythms and sleep patterns, hormonal changes, and the effects of pharmacotherapy used during the perinatal period—for example, for anesthesia. Support from the mother's social environment is certainly also important [8]. The relationship between generalized anxiety disorder and overweight or obesity is an exceptionally complex and multifaceted issue in which psychological, biological, and behavioral dimensions intertwine.

There is evidence suggesting a bidirectional relationship between excess body weight and mental health, with anxiety driving behaviors such as emotionally driven compulsive eating and stress-induced weight gain, which in turn leads to hormonal and metabolic dysregulation. On the other hand, obesity exacerbates anxiety symptoms through factors such as inflammation, dissatisfaction with one's appearance and the resulting low self-esteem, as well as social stigmatization. Although it might seem that the cardiovascular system has little to do with mental health, nothing could be further from the truth. The co-occurrence of anxiety disorders and coronary artery disease is common and is associated with higher mortality. Studies have shown that approximately one in three patients with coronary artery disease suffered from anxiety disorders or depression, and mortality in this group due to these disorders was twice as

high as in people without somatic diseases. One explanation for this comorbidity is that both anxiety disorders and depression increase the risk of developing lifestyle-related diseases, including cardiovascular diseases, through factors such as obesity and hypertension—a finding confirmed by a growing number of studies over the past few years. Potential mediating factors included: smoking status, body mass index, diet quality, hypertension, low-density lipoprotein cholesterol levels, hyperglycemia, and C-reactive protein levels. The researchers assumed that these factors, measured at the beginning of the study, lay along the pathway linking anxiety disorders or depression to coronary heart disease. Smoking status was determined based on self-reported information from participants as: current smoker, former smoker, or never-smoker. Diet quality, assessed through cumulative adherence to nutrition-related recommendations, demonstrated that healthier eating patterns are strongly associated with a lower presence of anxiety disorders [11,12].

To investigate the relationship between cardiovascular diseases and mental disorders—primarily anxiety—an observational study was conducted to assess the prevalence of comorbid mental disorders—specifically post-traumatic stress disorder, anxiety and depressive disorders—among adult patients aged 18 to 49 with diagnosed congenital heart defects. A cohort of 13,807 patients with congenital heart defects was compared with a control group of over 5.4 million people without such conditions. The analysis revealed a significantly higher incidence of mental disorders in the population with heart defects—12.4% of them had received a formal diagnosis of an anxiety disorder or depressive disorder. Furthermore, epidemiological modeling revealed that adults with congenital heart defects have a 30% to 50% higher adjusted prevalence of PTSD, anxiety, and depression compared to the reference population without these conditions [14].

It is important to remember that chronic diseases not only contribute to anxiety disorders. Some conditions, in contrast, are associated with lower risk of anxiety. One example is Down syndrome. Children with Down syndrome are significantly less likely to exhibit symptoms of anxiety [19].

The Impact of Environmental and Psychosocial Factors on the Development of Anxiety Disorders

Studies were conducted in which rodents were observed under controlled conditions while being subjected to several stress models, such as psychosocial and physical stress. Examples of the stressors used included premature separation of young animals from their biological mothers, a shortage of nesting materials, and exposure to excessively bright light. All of these factors disrupted the sense of security and caused chronic stress. In humans, a comparable situation is social isolation, especially during the early stages of a child's life and development, and it is considered a significant risk factor for the development of anxiety. Analysis of the animals' responses has shown that stress experienced in early life contributes to an increased risk of anxiety disorders in later stages, namely in childhood, adolescence, and adulthood [1].

Similarly to physical well-being, following a high-quality diet is associated with improved mental health. Diet is an important aspect of life that significantly influences the occurrence of anxiety disorders in predisposed individuals and is, at the same time, easy to modify. Studies have been conducted on the relationship between dietary habits and a higher prevalence of anxiety disorders. The analysis revealed a link between lower levels of anxiety and higher intake of fruits and vegetables, omega-3 fatty acids, "healthy" dietary patterns, reducing caloric intake to a level optimal for an individual's energy needs, regular breakfast consumption, a ketogenic diet, supplementation with broad-spectrum micronutrients such as zinc, magnesium, and selenium, as well as the use of probiotics. The Mediterranean diet was found to be significantly associated with a reduced risk of mental disorders such as depression or anxiety. At the same time, a link has been demonstrated between higher levels of anxiety and its more debilitating impact on many areas of a patient's life and a high-fat diet, insufficient amounts of tryptophan and protein in the diet, high intake of sugar and refined carbohydrates, and "unhealthy" dietary patterns—by which we mean, for example, high consumption of highly processed foods. It should be noted that, unfortunately, the results presented above are limited due to the high proportion of animal studies and observational studies. Only 10% of intervention studies included participants with anxiety disorders, which limits the applicability of the findings. High-quality intervention studies involving individuals with anxiety disorders are needed. Interestingly, studies conducted under controlled conditions on animals have reported both anxiety-inducing and anxiolytic effects following the adoption of dietary patterns commonly considered "unhealthy" or "Western." This paradox may be explained by a short-

term reward mechanism triggered by high-fat or high-sugar foods, which can temporarily blunt stress responses, creating a transient anxiolytic effect. Calorie restriction and fasting were mainly associated with anxiolytic effects. Furthermore, it has also been revealed that a diet rich in fiber improves glucose homeostasis, serum lipid profile, and levels of inflammatory chemokines, and mitigates associated symptoms of depression and anxiety disorders in patients with type 2 diabetes.

Observational studies have shown an association between lower severity of anxiety symptoms or a lower prevalence of anxiety disorders and “healthy” dietary patterns, the Mediterranean diet, traditional diets, a vegetarian diet, eating breakfast, a high proportion of anti-inflammatory foods in the diet, and greater dietary diversity. Higher levels of anxiety symptoms or a higher prevalence of anxiety disorders were associated with “unhealthy” dietary patterns, calorie restriction, and snacking—especially on unhealthy snacks—between main meals. It should be noted that among observational studies assessing the association between vegetarianism and anxiety symptoms, two of the three studies reporting an increase in anxiety were prospective, while all studies indicating an association between dietary patterns and lower anxiety severity were cross-sectional. These findings underscore the vital role of a proper diet in supporting the treatment and prevention of overall mental well-being in children and adults without specific somatic comorbidities, and it is particularly essential for individuals with metabolic conditions such as diabetes [2,7].

Anxiety Disorders Clinical Trial in Children

An interesting clinical trial involving patients in the pediatric population was also conducted. The study included 34 children and adolescents aged 4 to 17 who had been diagnosed with Tourette syndrome or chronic tic disorder combined with anxiety symptoms and who were not receiving pharmacological treatment. To ensure the high reliability of the findings, the researchers applied rigorous inclusion criteria, selecting participants with an IQ above 80, moderate tic severity, and elevated anxiety, while carefully excluding those with primary comorbid mental disorders such as autism spectrum disorders, schizophrenia, or major depression. The study was closely monitored, with parents tracking adverse events and having access to continuous medical support. Clinical evaluations were meticulously performed by experienced pediatric specialists at baseline and after a two-month follow-up. A comprehensive clinical profile was established using validated tools, including the Wechsler Intelligence Scale for Children for cognitive assessment, alongside the Multidimensional Anxiety Scale for

Children (MASC) and the Children's Depression Inventory. For younger children, the Child Behavior Checklist provided additional confirmation of anxiety symptoms. By defining a clinical response as at least a 30 percent reduction in both tic and anxiety scores, the trial robustly evaluated the efficacy of supplementation with L-theanine and pyridoxine (vitamin B6) against a standard psychoeducational intervention.

Although objective clinical improvement was observed in both study arms, supplementation with the nutraceuticals under study demonstrated statistically significantly higher efficacy in directly reducing the severity of the tics themselves. The data suggest the potential of these compounds as a safe adjunctive therapy. Furthermore, vitamin D has also gained importance due to its antioxidant, anti-inflammatory, neurogenic, and neuromodulatory properties, which appear to be crucial to its anxiolytic effects. These findings are supported by studies showing that vitamin D supplementation can alleviate symptoms of anxiety. On the other hand, experimental evidence indicates that Ca^{2+} signaling plays a key role in regulating sleep-wake cycles. There is also evidence suggesting that increased dietary intake of Ca^{2+} alleviates anxiety [13,18].

The Role of Psychotherapy in Early Intervention for Anxiety Disorders

The molecular pathways responsible for the development of fear as a physiological response to a real threat and anxiety as a pathological response in situations without a real or potentially imminent threat are still not well understood; therefore, medications are poorly targeted. Drugs from the class of selective serotonin reuptake inhibitors (SSRIs) have been used to treat anxiety disorders, but cognitive-behavioral therapy appears to be the primary and most effective treatment method based on current knowledge [1]. Anxiety disorders, including social anxiety disorder, generalized anxiety disorder (also known as free-floating anxiety), panic disorder, agoraphobia, and various specific phobias, are characterized by excessive anxiety and fear associated with everyday activities that, in people without these disorders, should not elicit negative emotions, as they pose neither a real nor a potential threat. Furthermore, these disorders are often accompanied by avoidance behaviors. Although avoidance is a common behavioral response, it can be both a consequence of and a contributing factor to the anxiety and fear underlying these disorders. Cognitive-behavioral therapy (CBT) with an emphasis on exposure is useful. Avoidant behaviors, which are a defensive reaction and—under physiological conditions—help avoid danger, are a characteristic feature of anxiety disorders and subconsciously serve to temporarily alleviate the distress caused by anxiety that arises in a

specific situation or in response to exposure to a specific stimulus. Paradoxically, however, such avoidance reinforces anxiety and fear by preventing contact with the anxiety-inducing stimulus, which leads to the perpetuation of the anxiety disorder. Avoidance manifests itself in various forms, depending on the type of anxiety a person experiences—for example, people with a fear of heights avoid being at high altitudes, such as climbing tall observation towers, while people suffering from agoraphobia avoid leaving their homes.

The persistence of avoidance behaviors is also associated with poorer treatment outcomes, particularly in the case of social phobia, where a high level of avoidance predicts lower effectiveness of exposure therapy—that is, confronting the factors that trigger anxiety. It has also been shown that avoidance behaviors in childhood predispose individuals to developing disorders; for example, social isolation significantly increases the risk of developing social phobia. However, avoidance behaviors are almost always the result of anxiety rather than one of its causes. This feedback loop hinders a key element addressed in cognitive-behavioral psychotherapy—namely, becoming accustomed to anxiety-provoking stimuli—thereby prolonging and intensifying anxiety over time [9].

SUMMARY

Anxiety disorders are a complex group of psychiatric disorders, among which we can distinguish the main types, such as anxiety neurosis, panic disorder, anxiety attacks, and specific phobias such as social phobia and agoraphobia etc. In addition, there is also a form of generalized anxiety disorder known as “free-floating” anxiety. The pathogenesis of anxiety disorders is very complex, and it involves both innate (i.e., genetic) factors—over which we have no control—and other somatic disorders, primarily hormonal ones, as well as conditions related to sleep deprivation. Environmental factors to which we are exposed from the earliest stages of life also play a significant role; these include diet, the use of psychoactive substances, and stress—both psychological and physical—such as a lack of a sense of security in the first weeks of life due to separation from the mother. Many studies on anxiety disorders have already been conducted, but many aspects remain unexplored and unexplained. While SSRIs are commonly used as a primary pharmacological treatment, specific anxiolytics are also utilized to target anxiety directly. The foundation of treatment for anxiety disorders in all patients remains pharmacotherapy combined with cognitive-behavioral therapy, and the main determinant of the therapy’s effectiveness is the patient’s cooperation in establishing healthy

thought patterns and eliminating avoidance of situations or stimuli that trigger anxiety [1,2,3,4,5,6,8,9,10,11].

DISCLOSURE

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