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The Impact of Genetic, Environmental and Gene-by-Environment Factors on Adolescent Anxiety Disorders

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Abstract. Various reasons contribute to the development of anxiety disorders in adolescence, and genetics, environment, and their interactions (G×E) are all involved. This systematic review examines recent evidence of genetics, environment and their interaction in the course of development of anxiety disorders during adolescence. Obtained studies from databases and screened them according to PRISMA guidelines. Most of the studies included in this review have frequently mentioned polymorphisms of the serotonin system. The most frequently studied of the serotonin-transporter-linked polymorphic regions was 5-HTTLPR. Based on the above research, allele differences can affect a person's risk of anxiety by altering the mechanism of neurotransmitter transmission and regulation of the stress response system. The Environment may affect the results of anxiety in adolescents. Family-related factors are included in the above, such as parenting style, parents' education, family environment and daily stress. According to research on G×E interactions, genetic risk can be increased or reduced by environmental factors; however, due to differences in the characteristics of the sample studied, anxiety was not measured, and various methods were used, the results of such studies are inconsistent. In short, the above content shows that anxiety disorders in adolescence are the result of multiple factors, and thus, both genetic and environmental factors should be considered when determining the causes of anxiety disorders. The results of this study will provide a reference for improving the present prevention and intervention plan for environmentally vulnerable adolescents.

Keywords: Anxiety disorders, adolescent, genetics, environmental, gene-by-environment interactions

1. Introduction

Anxiety disorders are relatively widespread in the world, and many types exist, such as generalised anxiety disorder, social anxiety disorder, and specific phobias; these problems have caused considerable harm to individuals and society. According to large-scale epidemiological studies, as many as 33.7 per cent of the population have been diagnosed with anxiety disorders at some time [1]. The first signs are often shown in childhood or adolescence. Otherwise, they would have remained minors indefinitely. Anxiety disorders are the most frequent mental health issues among adolescents in Canada; thus, how to recognise anxiety at this time in their lives (and generally a vulnerable period for mental health) is need to learn [2]. Both genes and environment should be

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considered in the investigation of causes and risks for anxiety disorders among adolescents that lead to internalising psychological disorders [3, 4].

Another reason for a person having anxiety disorders may be a genetic predisposition. Many neurotransmitters, stress-response systems and emotions have been linked to gene mutations by researchers. Among them, one of the most studied variants is the 5-hydroxytryptamine transporter-related polymorphic region (5-HTTLPR), which has a short (S) allele and a long (L) allele, and these two alleles have different effects on the expression of the 5-hydroxytryptamine transporter [5]. The results of the studies are not all the same. For instance, a study on Chinese adolescents found that the association between the S and L alleles and anxiety-related outcomes varied, and in some cases, it was in the opposite direction [6]. Therefore, the effect of 5-HTTLPR is probably not solely due to this factor. It is likely that other genes and environments will also affect the outcome differently for different people.

The environment in a person's life at that time can also increase or decrease their anxiety. Daily stress factors that cause daytime sleepiness can reduce a person's coping capacity in the face of stress and exacerbate anxiety [7]. Family environment and life experience are also related to anxiety risk. Negative patterns in family life are one of the reasons cited most often in the studies included in this review. In addition, widespread social changes and public health crises in recent years have also likely contributed to an increase in anxiety disorders among adolescents [8].

Although both genetic susceptibility and environmental factors are related to the risk of anxiety, neither can explain the occurrence and differences in anxiety disorders during adolescence completely on its own. An accumulation of evidence shows that genes and environments interact (G×E). In addition, the two factors of genetics and environment are also responsible for anxiety. Genetic predisposition may make a person more susceptible to anxiety; at the same time, the environment can either increase or decrease this susceptibility. For instance, certain genetic variations in adolescents may be more susceptible to the effects of sleep deprivation, poor parenting, family stress and bullying behaviour. Supportive families and social environments can help reduce anxiety symptoms in people who are genetically predisposed to anxiety. Focusing solely on genes and environment may fail to explain why. To better understand the specific reasons for anxiety disorders in adolescence, people should take into account all the reasons and how they are related to each other.

The two main objectives of this systematic review are to summarize the principal genetic alterations associated with adolescent anxiety disorders and to explore how these modifications influence the progression of anxiety via biological pathways such as neurotransmitter systems, stress-response regulation and emotional processing; at the same time, the influence of environmental and broader social factors on the risk and severity of anxiety disorders will also be investigated. Integrate the results of previous research in this paper, and then identify any patterns or differences among them, as well as future directions for research. At the same time, this paper will also examine whether genetic factors, environmental factors and gene-environment (G×E) interactions independently or together influence anxiety in adolescents.

2. Methodology

2.1. Study design

A system-atic review of related studies has been conducted to explore genetic, environmental and gene-environment (G×E) factors affecting the development of anxiety disorders in adolescence. To ensure the transparency and organisation of the study selection process, this review followed the

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Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statements. PRISMA is a general-purpose tool for identifying, filtering and selecting studies in medical and psychological research in a standardised way, thereby enhancing the transparency and replicability of evidence synthesis. The entire selection process for these records is to identify and filter, then assess their eligibility, and finally include them. Only peer-reviewed original research that examined adolescent anxiety disorders in the context of genes, environments or G×E interactions was included.

2.2. Search strategy

Filter out related studies in the academic database that contain peer-reviewed biomedical, psychological, genetic and public health research. All areas of research on the genetics and environment of anxiety disorders in adolescents have been included in the scope of these databases. Only studies published between 2000 and 2026 were included in order to cover both the foundation work and more recent developments in the field.

The combined terms in the search strategy included anxiety disorders, adolescence, genetics, environmental factors and G×E interactions. Frequently used search terms included "anxiety disorders", "adolescent", "adolescence", "genetics", "gene variation", "environmental factors", "environment", "G×E interaction", "human", "gender", and "society". Both the individual terms and combinations of these terms were used to find studies that have explored the connection among biological factors and environmental exposure in the case of adolescent anxiety disorders. Given the focus of the research, a smaller scope for the development of anxiety disorders was adopted; therefore, extra keywords were added to refine the initial search and excluded any records that did not directly pertain to the research topic.

2.3. Eligibility criteria

Eligibility criteria were set before the final screening to ensure that the included studies met the requirements for the purposes of this review. Selection Criteria: Publication language, year of publication, characteristics of participants, research content, type of study, and overall relevance to the research problem.

Research that met the following criteria was selected: published in English between 2000 and 2026, involved human subjects, and studied anxiety disorders or anxiety-related traits in adolescence. Eligible articles needed to report their own original empirical results. Genetic studies on variants related to anxiety susceptibility and research on environmental factors, such as parenting style, family environment, parental education, sleep problems, bullying, social stress and other social pressures, have also been carried out to understand how they affect the development of adolescent anxiety disorders.

Articles were excluded if they used only non-human models, did not include adolescent participants, or focused on conditions other than anxiety disorders or anxiety-related traits. Literature reviews, systematic reviews, meta-analyses, editorials, commentaries, opinion papers and studies deemed irrelevant after title, abstract or full-text screening were also excluded. Reports that did not have a main focus on adolescence were excluded to keep the developmental emphasis of the review.

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2.4. Study selection process

The first database query found 124 rows. After the first identification, 5 records were excluded because they did not contain an abstract, and 119 records remained for subsequent screening. Titles and abstracts were then examined to determine if the studies were related to adolescent anxiety disorders and whether they considered genetic, environmental or G×E factors. In the screening stage, 64 studies were excluded because they were not related to anxiety disorders, did not focus on relevant genetic or environmental factors, involved non-human research, were outside the selected time frame, or did not meet the keyword and topic requirements. After the above screening, 55 reports were included for analysis. Seventeen of the literature reviews were excluded from the eligibility assessment because they did not focus on original research studies. An extra 18 reports were excluded because they did not focus on adolescents or did not provide sufficient adolescent-specific results. After applying all the inclusion and exclusion criteria, 13 original research papers met the criteria for this systematic review.

2.5. Data extraction

Standardized Data extraction was performed on all 13 included studies. The main study features include the basic characteristics of the research, the population of the study, adolescent anxiety-related outcomes, key genetic variants, typical environmental risk factors and verified G×E interaction relationships. The main genetic data were anxiety-related polymorphisms and allele variation characteristics. Environment refers to the family environment, parents' education and attitudes, sleep health, bullying experiences and social pressure. Organize the results of all studies, associate trends and existing research gaps systematically for follow-up comparison.

2.6. Data synthesis

Synthesise the data from the selected studies in a narrative manner. Narrative synthesis was chosen because the included studies had different designs, samples, genetic markers, environmental factors and anxiety-related outcomes. Due to the above reasons, a statistical meta-analysis was not performed. Based on the above, the three main groups in this review are genetic factors, environmental factors and G×E interactions, and their similarities, differences and recurrent themes are presented below. Research has been carried out on the role of genes in anxiety disorders to identify various disease-causing genes and their mutations. Based on the environment, the following common risk factors for adolescents were identified: family issues, social problems, behaviour, development, etc. Combine G×E results to investigate how the interaction of genetic susceptibility and environmental factors influences the risk of anxiety disorders. Patterns and discrepancies in the studies were found during the synthesis. Based on the above results, a summary of the current situation in the field has been provided, deficiencies in previous research have been identified, and the roles of genes and environments in the development of adolescent anxiety disorders have been explored.

3. Synthesis and discussion of findings

3.1. Genetic factors associated with adolescent anxiety disorders

Adolescent anxiety disorders are caused by the cumulative effects of many genes, not a single gene. Many genes affect the development of the brain, neurotransmitter function and stress response;

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therefore, people have different susceptibilities to anxiety and various degrees of symptoms or disease. The three principal candidate genes associated with anxiety in adolescence will be analysed here.

Polymorphisms in 5-HTTLPR have recently attracted attention due to their association with serotonin neurotransmission. The long allele (L) has generally shown higher transcriptional efficiency of the serotonin transporter, and the short allele (S) has been associated with reduced transcriptional activity and altered serotonin reuptake dynamics [5]. Serotonin helps regulate mood and fear response, etc., so variations in the SLC6A4 gene might be related to a higher risk of anxiety in adolescents; this is a period of considerable changes for the nervous system and behaviour. Nevertheless, the results of the studies vary. Some research has found that carriers of the S allele are more anxious, and other studies have shown higher anxiety in adolescent carriers of the L allele [6]. The above differences are mainly due to ethnic background, sample size, phenotypic measurement indicators and individual environmental exposure, and it can be seen that the pathogenic effect of 5-HTTLPR is regulated by multiple factors.

The Val66Met polymorphism of brain-derived neurotrophic factor (BDNF) is required for neuronal plasticity and the formation of emotional neural circuits. The Met allele modifies the function of BDNF, impacts the development of the amygdala and hippocampus, and increases the risk of generalised anxiety disorder [9]. Existing clinical data show that anxiety patients have lower levels of peripheral BDNF than healthy controls [10]. Compound genetic variation is likely to exert an additive risk. Individuals carrying both the 5-HTTLPR short allele and the BDNF Met allele are significantly more anxious than those with a single variant mutation, indicating that multiple genes act synergistically in the pathogenesis of anxiety [11].

The COMT gene codes for an enzyme that degrades dopamine and noradrenaline, and thus influences stress response and mood. Related studies have shown that adolescent COMT Val allele carriers are more anxious and neurotic than Met allele carriers [10]. The correlation results are not generalizable to all groups of people. The inconsistent research results for candidate genes fully support the polygenic nature of adolescent anxiety, and the single-gene research model cannot fully explain disease susceptibility.

3.2. Environmental and societal factors influencing adolescent anxiety risk

Development and changes in anxiety disorders for adolescents can also be affected by environmental factors. For instance, a person may have negative experiences of abuse, neglect, chronic stress and childhood trauma, all of which increase the risk of anxiety by impairing emotion regulation, the stress-response system and socioemotional development. Adolescence is a period of rapid growth and sensitivity in life. Ongoing neurobiological changes at this time may make an individual more susceptible to environmental stress. Environmental risk should not be regarded as the result of a single incident or experience, similar to genes. Anxiety risk can be influenced by a combination of family, society and environment over time, and the total effect of these factors may help explain why different people experience similar harmful situations differently; for example, some adolescents develop serious anxiety, while others are not affected.

Environmental stressors, such as major global events and population-level crises, can also increase the risk of anxiety disorders by disrupting socialisation, education, family life, access to healthcare, a sense of security, etc. A typical case of the above is the COVID-19 pandemic. Studies on the impact of the pandemic on anxiety among adolescents are not the same as those on genetic susceptibility. Although some research has found that adolescents are more anxious and depressed following the pandemic, other studies have not found any such increase [8]. The above mixed results

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suggest that the psychological harm of the epidemic differs among groups. In populations with an increase in anxiety symptoms, the occurrence of such an increase may have been related to social isolation, school closures, uncertainty, the loss of a loved one, reduced peer interaction, increased family stress, and widespread disruptions to daily life. The extent of the damage caused by these factors at the same time would be affected by cultural norms, socioeconomic status, family environment, access to digital resources, public health measures, and pre-pandemic mental health trajectories. For example, studies in Europe and North America, such as those conducted in Norway and Iceland, have reported an increase in anxiety and depression among adolescents; at the same time, studies in China and Australia have shown no change or even a decrease [8]. Therefore, in the future, more general contextual and socio-economic factors need to be considered in the assessment of the impact of large-scale environmental stressors on the mental health of adolescents to gain a deeper understanding of this effect.

Bullying victimisation is a type of social disadvantage that is also harmful. Prolonged exposure to bullying results in chronic psychological stress, low self-esteem and social isolation, and is one of the reasons for people's anxiety disorders around the world [12]. Negative family interaction, chronic sleep disorders and social stress can all increase the risk of anxiety for young people. Most cases of anxiety in adolescence are due to a combination of environmental factors. Early targetted intervention for modifiable environmental risks can reduce both the start time and the length of anxiety in young people.

3.3. Gene-by-environment interactions in adolescent anxiety

Genetic susceptibility and environmental factors interact to determine the reasons and processes of development for anxiety disorders and other mental illnesses, such as depression and stress-related disorders, and how they manifest. Genetic susceptibility is no longer regarded as an independent cause. Rather, it has been known to interact with the environment at all times in the development of adolescents. Therefore, to understand why adolescents exposed to similar environments still show differences in the risk of developing anxiety disorders, symptom severity, age of onset, psychiatric comorbidities, and treatment response, gene-by-environment (G×E) interaction may need to be considered. Therefore, the above interactions may help us better understand the biological and environmental reasons for changes in adolescent anxiety and provide a clearer picture of what causes these variations.

Among the studied genetic factors, the 5-HTTLPR polymorphism and the rs25531 variant have attracted much attention because of their effects on one's susceptibility to stress-related psychiatric disorders. rs25531 is an A-to-G single nucleotide polymorphism that further changes the functional variation of the 5'-HTTLPR locus [5]. Research has shown that an elevated risk of internalising mental disorders (IMDs), such as anxiety disorders, is associated with the interaction between the 5'-HTT-PLCR-rs25531 S-A/S-A genotype and acute stress [3]. Specifically, individuals with the S-A/S-A haplotype who were under acute stress were at a higher risk of developing IMDs than those carrying the L-A/L-A haplotype who had only been exposed to mild acute stress. On the other hand, among individuals carrying the L-A/S-A haplotype, exposure to moderate acute stress was associated with a reduced risk of IMDs, and exposure to severe acute stress showed an elevated risk. In short, the above results indicate that higher levels of stress may intensify the impact of genetic susceptibility, and different haplotypes respond differently to different kinds of stress. It can be used to study gene-environment interactions in the development of anxiety disorders in adolescence.

Stressful or traumatic life experiences and genetic susceptibility may also increase the risk of anxiety in adolescence by interacting with each other. All the research has shown that childhood

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adversity, such as abuse, neglect, bullying and the loss of parents, is an early environmental risk factor for the development of stress-related mental disorders. The above experiences may alter the body's biological function, such as adjusting stress hormones, and at the same time, genetic differences can also affect how a person responds to such experiences. For example, individuals who carry the FKBP5 risk allele may be more likely to experience demethylation following childhood trauma compared with those carrying protective alleles; thus, there may be alterations in stress hormone regulation that increase vulnerability to stress-related psychiatric disorders. G×E effects due to inheritance are the only types; otherwise, early-life stress modifies gene expression epigenetically without changing the basic structure of DNA. The above epigenetic modifications may alter the biological system involved in the regulation of the hypothalamic-pituitary-adrenal (HPA) axis, serotonergic signalling, immune function and neuroplasticity, thereby increasing the risk of psychiatric disorders and stress-related diseases later in life [4].

Genetics and Environment are both causes of the development of anxiety disorders. Genetic factors, such as specific gene polymorphisms and family history, can affect when a person develops anxiety, how severe it is, and how easily they are affected by stress due to environmental reasons [13]. Instead of acting alone, they work together during their teens. The environment can alter a person's genetic risk and thus change the biological mechanisms of anxiety; at the same time, genes also affect how people respond to environmental stress and risk factors. Together, the two factors may explain why adolescents with anxiety disorders show different symptoms and functional impairments, as well as many other differences among them. Further study of the above interactions can offer robust support for building an effective system of early assessment, prevention and intervention. Given that adolescent anxiety disorders are biologically and environmentally complex, the improved measures will help meet the different needs of these young people.

3.4. Implications for prevention, intervention and future research for adolescent anxiety disorders

Understanding the genes of adolescent anxiety disorders can help us develop more personalised care plans for people with mental illness at a younger age. Help to identify biological pathways related to disease risk, as well as different expressions of symptoms, treatment responses and susceptibility to the effects of stress and anxiety based on genetic information, and thus tailor intervention strategies to the specific needs of individual patients. Further research will be carried out to determine how these different genes and alleles interact, and thus a new method of drug discovery may be developed. Neurotransmitter regulation, stress-response pathways, and neurodevelopment are examples of such mechanisms. On the other hand, the following environmental and social factors should be taken into account in the planning of prevention and early diagnosis and public health intervention, as early identification of environmental risk factors during adolescence may provide a window for preventive measures before symptoms become chronic or serious. Address modifiable risk factors, such as trauma, bullying, family stress, socioeconomic disadvantages and limited access to mental health services, to reduce both the onset and duration of anxiety disorders [13].

Early intervention has also received some support for research. About half of all mental disorders begin to show in people aged 15-17. Some of the symptoms a person exhibits in their early years may be the initial manifestations of chronic mental illnesses, such as anxiety and depression [14]. Therefore, prevention and mental health care are needed in adolescence to help young people grow well as their brains are still developing. Mental health services offered at this time should be suitable for the stage of development of adolescents and take into account not only clinical symptoms or common risk factors, but also the family, school and other environments, as well as the overall

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context in which adolescents live and are raised. However, factors that determine whether a person is likely to be mentally ill or not cannot be reduced to genes and environments alone; therefore, interventions should take into account both genes and environments to be more suitable to different individuals. As emotions regulation, identity formation, peer relationships and the stress-response system continue to develop and mature in adolescence, support at this time can reduce the risk of serious mental illness later in life and lower susceptibility to stress and anxiety.

This paper has the following deficiencies. Most of the available studies focus on adults or mixed-age groups, and relatively few have been conducted prospectively on adolescents. Most of the above studies have been conducted on Western populations. Thus, the scope of the generalisation of the present results to other cultures and populations is limited. More importantly, how the interaction between sex and G×E is modified at different times in adolescence is still unknown. Therefore, in the future, more diverse adolescent groups will be included in long-term longitudinal studies, standardised assessment methods will be employed, and the interaction of sex and environment on vulnerability to anxiety during adolescence will also be further investigated. The above work will help provide additional support for the development of more equitable, precise and effective prevention and intervention measures in the future.

4. Conclusion

Systematically review all relevant research in China and abroad from 2000 to 2025, and systematically analyze the independent and combined effects of genetic factors, environmental factors, and G×E interactions on adolescent anxiety disorders. Based on the above, adolescent anxiety is a complex disease caused by multiple factors and cannot be attributed to a single gene mutation or isolated environmental stressors. Genes are responsible for variations in stress response, neurotransmitter signalling and brain plasticity that lead to anxiety; Environmental and social factors provide the necessary conditions for the manifestation of anxiety; Therefore, a joint genetic and environmental (G×E) interaction occurs in individuals to determine the level of anxiety and how well they respond to treatment. Based on this study, it can be concluded that genetics and environment are closely related factors in the development of adolescent anxiety; thus, new ideas for the early prevention and targeted intervention of adolescent mental health problems have been proposed. The following are some deficiencies in this study. Most of the environmental risk data are self-reported and thus may be subject to recall bias and subjectivity. The included studies have a large degree of heterogeneity in research design and detection indicators, and thus cannot be uniformly quantified. In the future, more samples and research subjects will be collected, standardised detection methods and long-term research designs will be employed, and research on the G×E interaction effect will be further extended to enhance the accuracy of anxiety risk assessment and intervention.

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