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The Role of Genetic and Environmental Factors in the Development of Schizophrenia

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Abstract

This study investigates the interactive contribution of genetic vulnerability and environmental risk factors to prodromal psychotic symptoms within a biopsychosocial framework. A cross-sectional design was employed with a sample of 120 adults recruited through online platforms. Genetic liability was assessed using a weighted family history index capturing both first- and second-degree relatives. Environmental exposure was measured through the Childhood Trauma Questionnaire (CTQ), while emotional regulation strategies were assessed using the Emotion Regulation Questionnaire (ERQ). Prodromal symptoms were evaluated using the PQ-16.

Exploratory analyses suggested that both genetic vulnerability and childhood trauma were associated with increased prodromal psychotic symptoms. Emotional suppression appeared positively associated with symptom severity, whereas cognitive reappraisal demonstrated a potentially protective role. Patterns observed in the data provided preliminary support for gene-environment interplay models of schizophrenia.

These findings support contemporary polygenic and developmental perspectives of schizophrenia and highlight the importance of early identification, trauma-informed approaches, and emotional regulation processes in preventive mental health strategies targeting individuals at clinical high risk.

Keywords: schizophrenia, genetic vulnerability, childhood trauma, emotion regulation, prodromal symptoms, gene-environment interplay

1. Introduction

Despite advances in genomic research, there remains a need for integrative models that combine genetic, environmental, and psychological mechanisms within a single analytical framework.

Schizophrenia is a severe and heterogeneous psychiatric disorder characterized by disturbances in perception, cognition, emotion, and behavior. It represents a major public health concern due to its chronic course, early onset, and significant functional impairment (American Psychiatric Association, 2013). Individuals affected by schizophrenia often experience hallucinations, delusions, disorganized thinking, and impairments in social and occupational functioning, resulting in a substantial burden at both individual and societal levels.

Contemporary scientific perspectives increasingly conceptualize schizophrenia not as the result of a single causal factor, but as the outcome of complex and dynamic interactions between genetic susceptibility and environmental exposures across development. Advances in psychiatric genetics, particularly genome-wide association studies (GWAS), have identified numerous genetic loci associated with schizophrenia, supporting its highly polygenic nature (Ripke et al., 2014; Trubetskoy et al., 2022). Polygenic risk scores (PRS) further highlight that genetic vulnerability is distributed across multiple variants rather than determined by single genes.

At the same time, a substantial body of research has suggested that environmental risk factors play a critical role in shaping the expression of this vulnerability. Exposure to childhood trauma, chronic psychosocial stress, substance use, and adverse social conditions has been consistently associated with increased risk for psychosis (Varese et al., 2012; Di Forti et al., 2019).

Understanding how genetic and environmental factors jointly contribute to the development of schizophrenia is essential for advancing early identification and prevention strategies.

Genetic predisposition
│
▼
Environmental risk factors
(childhood trauma, psychosocial stress, substance use exposure)
│
▼
Emotional regulation
(emotional suppression / cognitive reappraisal)
│
▼
Prodromal psychotic symptoms
(PQ-16 indicators)

Figure 1. Conceptual model of the study

This model conceptualizes emotional regulation as a potential mediating pathway through which environmental risk influences the expression of genetic vulnerability.

Research Questions

To address these issues, the present study examines the interaction between genetic and environmental influences in the development of schizophrenia. Specifically, the study seeks to answer the following research questions:

1. What genetic mechanisms contribute to the development of schizophrenia?
2. What environmental factors increase vulnerability to schizophrenia?
3. How do genetic predispositions interact with environmental exposures in shaping the onset of the disorder?
4. What implications do these interactions have for prevention and treatment strategies?

Research Hypotheses

Based on the theoretical framework and previous research on the interaction between genetic vulnerability and environmental risk factors in schizophrenia, the following research hypotheses were formulated:

H1. Individuals with a family history of schizophrenia will report higher levels of prodromal psychotic symptoms compared to individuals without genetic predisposition.

H2. Exposure to childhood trauma will be positively associated with increased vulnerability to psychotic symptoms.

H3. Individuals who report maladaptive emotional regulation strategies (higher emotional suppression and lower cognitive reappraisal) will show higher levels of psychological distress and prodromal symptoms.

H4. Participants exposed to both genetic predisposition and environmental risk factors (e.g., childhood trauma or psychosocial stress) will show higher vulnerability

to schizophrenia-related symptoms than those exposed to only one category of risk factor.

2. Theoretical background

2.1 Genetic Factors in Schizophrenia

A substantial body of research suggests that schizophrenia has a strong genetic component. Family, twin, and adoption studies consistently show that the risk of developing schizophrenia increases significantly among individuals with affected biological relatives. For example, individuals with a first-degree relative diagnosed with schizophrenia have approximately a tenfold higher risk compared to the general population (Gottesman & Shields, 1982). Twin studies further support the role of genetic vulnerability, with concordance rates of approximately 40–50% for monozygotic twins and 10–15% for dizygotic twins.

Advances in molecular genetics have further expanded knowledge about the biological basis of schizophrenia. Genome-wide association studies (GWAS) have identified numerous genetic loci associated with increased susceptibility to the disorder, highlighting its polygenic nature (Ripke et al., 2014). Genes such as DISC1, COMT, and NRG1 have received particular attention due to their role in brain development, synaptic functioning, and dopamine regulation.

Recent research has also focused on polygenic risk scores (PRS), which estimate an individual's overall genetic vulnerability based on the combined effects of multiple genetic variants. Additionally, structural genetic variations such as copy number variations (CNVs), particularly in chromosomal regions such as 22q11.2, have been associated with increased risk for schizophrenia and other neurodevelopmental disorders.

Beyond genetic inheritance, epigenetic mechanisms also play a significant role in the etiology of schizophrenia. Environmental exposures such as stress, trauma, or prenatal infections may influence gene expression through processes such as DNA methylation and histone modification. These mechanisms provide a biological explanation for how environmental experiences may interact with genetic susceptibility in the development of the disorder.

Overall, contemporary research suggests that schizophrenia should be conceptualized as a polygenic neurodevelopmental disorder in which multiple genetic factors contribute to vulnerability rather than acting as single causal determinants.

2.2 Environmental Risk Factors

Although genetic predisposition is a significant component of schizophrenia risk, environmental factors also play a significant role in the development and expression of the disorder. Epidemiological research has identified several environmental exposures that may increase vulnerability across different stages of development.

Prenatal and perinatal complications represent one of the most consistently reported risk factors. Maternal infections during pregnancy and obstetric complications such as hypoxia or low birth weight may disrupt early neurodevelopment and increase the likelihood of later psychotic disorders (Brown et al., 2004).

Another well-documented environmental factor is urbanicity. Individuals raised in densely populated urban areas have been found to be two to three times more likely to develop schizophrenia compared to those raised in rural environments (Vassos et al., 2012). This increased risk has been linked to social stress, environmental pollution, and reduced social cohesion.

Migration and experiences of discrimination have also been associated with elevated rates of schizophrenia. The "social defeat hypothesis" suggests that chronic experiences of social exclusion and marginalization may influence dopamine regulation and increase vulnerability to psychotic disorders (Cantor-Graae & Selten, 2005).

Substance use, particularly cannabis consumption, has also been identified as a significant environmental risk factor. Evidence suggests that early and frequent cannabis use increases the probability of developing psychotic symptoms, especially among individuals with genetic vulnerability. Studies have reported a dose–response relationship between cannabis potency and the risk of psychosis (Di Forti et al., 2019).

Childhood trauma and adverse experiences represent another significant environmental risk factor. Exposure to abuse, neglect, or severe psychosocial stress during childhood has been associated with increased risk of psychotic symptoms later in life. A growing body of research indicates that exposure to childhood adversity significantly increases the risk of psychotic symptoms later in life (Varese et al., 2012)**.** Such experiences may influence neurodevelopment, emotional regulation systems, and stress reactivity.

Taken together, these findings suggest that environmental exposures contribute significantly to schizophrenia risk and may interact with biological vulnerability across the lifespan.

2.3 Gene-Environment Interaction

Current scientific perspectives increasingly conceptualize schizophrenia as the result of complex interactions between genetic predisposition and environmental influences. The gene-environment interaction (G×E) model proposes that genetic vulnerability may remain latent unless activated by specific environmental exposures.

For example, individuals with genetic variants associated with dopamine regulation may be particularly sensitive to environmental stressors such as substance use, trauma, or chronic psychosocial stress. In such cases, environmental factors may amplify the impact of genetic susceptibility and increase the likelihood of psychotic symptoms.

Epigenetic processes provide a biological mechanism through which these interactions may occur. Environmental stressors can modify gene expression through epigenetic mechanisms that influence neural development, synaptic plasticity, and neurotransmitter regulation.

Understanding gene-environment interactions has significant implications for prevention and early intervention strategies. Identifying individuals with high genetic vulnerability and reducing exposure to environmental risk factors such as substance use and chronic stress may help reduce the incidence or severity of schizophrenia.

Overall, the integration of genetic, psychological, and social perspectives supports a biopsychosocial understanding of schizophrenia, emphasizing that both biological vulnerability and environmental context must be considered in explaining the development of the disorder.

3. Methodology

3.1 Research Design

The present study employed a quantitative cross-sectional design aimed at examining associations between genetic vulnerability, environmental risk factors, and prodromal psychotic symptoms.

3.2 Participants

The study included 120 adult participants recruited through online platforms and social media channels, including Facebook academic groups, university communication networks, and psychology-related online communities. Recruitment followed a voluntary convenience sampling approach. Participants ranged in age from 18 to 56 years. The sample included both male and female participants from diverse educational and socioeconomic backgrounds. Demographic variables collected included age, gender, educational level, socioeconomic status, and substance use history. Inclusion criteria required participants to be at least 18 years old and able to understand the language of the questionnaire. Exclusion criteria included self-reported diagnosis of severe neurological disorders, current psychotic disorder diagnosis, or substantially incomplete questionnaire responses. Prior to analysis, data screening procedures were conducted to assess completeness and response quality. Cases containing substantial missing data were excluded listwise to preserve statistical reliability and validity.

3.3 Instruments

Data were collected using validated psychological instruments assessing childhood trauma, emotional regulation strategies, and prodromal psychotic symptoms. Internal consistency was evaluated within the present sample. The Childhood Trauma Questionnaire (CTQ) demonstrated excellent reliability (Cronbach's $\alpha = .91$), the Prodromal Questionnaire (PQ-16) demonstrated good reliability ($\alpha \approx .80$) and the Emotion Regulation Questionnaire (ERQ) subscales demonstrated acceptable to good reliability (cognitive reappraisal $\alpha = .84$; emotional suppression $\alpha = .76$).

For the PQ-16, a cutoff score of ≥6 was used to indicate elevated risk for psychosis, consistent with previous validation studies (Prodromal Questionnaire (PQ-16); Ising et al., 2012).

Childhood Trauma Questionnaire (CTQ)

The CTQ (Bernstein et al., 2003) is a self-report instrument measuring exposure to childhood adversity across multiple domains, including emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. Previous research has suggested high internal consistency ($\alpha \approx .90$).

Prodromal Questionnaire (PQ-16)

The PQ-16 (Ising et al., 2012) is a screening tool designed to identify early psychotic experiences. A cutoff score of ≥6 is commonly used to indicate elevated risk for psychosis. The PQ-16 has demonstrated good reliability ($\alpha \approx .80$).

Emotion Regulation Questionnaire (ERQ)

The ERQ (Gross & John, 2003) assesses individual differences in emotional regulation strategies, specifically cognitive reappraisal and emotional suppression. Reported internal consistency ranges from $\alpha \approx .70$ to .85.

Genetic vulnerability (Family history assessment)

Genetic vulnerability was assessed using a structured weighted family history index rather than a binary yes/no indicator. Participants reported the presence of psychotic disorders among first-degree relatives (parents and siblings) and second-degree relatives (grandparents, uncles, aunts, and cousins). To reflect differential genetic proximity, first-degree relatives were assigned greater weighting compared to second-degree relatives. This weighted approach allowed for a more nuanced estimation of familial genetic liability and reduced the limitations associated with dichotomous family history measures. Although family history remains an indirect proxy of genetic vulnerability compared to molecular indicators such as polygenic risk scores (PRS), previous psychiatric research has supported its utility as a clinically meaningful risk indicator in schizophrenia-spectrum conditions.

3.4 Procedure

Data were collected through an online structured questionnaire. Participants were informed about the purpose of the study and provided informed consent prior to participation. The questionnaire required approximately 10–15 minutes to complete and included demographic questions followed by standardized psychological measures.

3.5 Ethical Considerations

The study was conducted in accordance with the ethical standards of the Ethics Committees of the Mediterranean University of Albania and the European University of Tirana, as well as the principles of the Declaration of Helsinki.

3.6 Data Analysis

Data analyses were conducted using IBM SPSS Statistics for Windows, Version 26.0. Preliminary analyses included descriptive statistics, reliability analyses, and Pearson correlation analyses among the principal study variables. Exploratory multivariate analyses were conducted to examine potential associations between genetic vulnerability, childhood trauma, emotional regulation strategies, and prodromal psychotic symptoms. Demographic variables including age, gender, educational level, socioeconomic status, and substance use were considered as relevant covariates during interpretation. Given the exploratory nature of the study and the cross-sectional design, findings should be interpreted as preliminary indicators of potential biopsychosocial interaction patterns rather than definitive causal relationships. The analyses primarily focused on identifying trends and clinically meaningful associations that may inform future longitudinal and genetically informed investigations.

4. Results

The analysis of the collected data provided insights into the relationship between genetic predisposition and environmental risk factors associated with vulnerability to schizophrenia spectrum symptoms.

4.1 Demographic Characteristics

Participants represented diverse demographic and socioeconomic backgrounds. The demographic analysis included variables such as age, gender, educational level, and socioeconomic status.

Although gender differences did not appear to significantly influence the emergence of psychotic symptoms, socioeconomic factors appeared to play a more relevant role. Participants with lower educational attainment and lower socioeconomic status suggested greater vulnerability to environmental stressors.

4.2 Genetic Predisposition

Family history analysis revealed that individuals reporting a first-degree relative diagnosed with schizophrenia reported higher levels of vulnerability to psychotic symptoms.

Participants with genetic vulnerability also reported higher levels of prodromal symptoms measured through the Prodromal Questionnaire (PQ-16).

These findings are consistent with the hypothesis that schizophrenia may be better understood through the combined influence of genetic and environmental factors rather than isolated determinants.

4.3 Childhood Trauma

Results obtained from the Childhood Trauma Questionnaire (CTQ) indicated that exposure to childhood adversity was associated with higher scores on measures of early psychotic symptoms.

Specifically, emotional neglect, emotional abuse, and exposure to chronic stress during childhood appeared to be particularly relevant predictors of vulnerability to schizophrenia-spectrum symptoms.

Participants reporting higher trauma scores also suggested increased emotional dysregulation patterns, suggesting that early adverse experiences may influence both psychological functioning and neurobiological development.

4.4 Emotional Regulation

Results obtained from the Emotion Regulation Questionnaire (ERQ) indicated that participants who reported higher levels of emotional suppression and lower use of cognitive reappraisal strategies showed increased levels of psychological distress and vulnerability to psychotic symptoms.

These findings suggest that emotional regulation strategies may play an important role in the relationship between environmental stressors and the development of psychopathological symptoms.

4.5 Interaction Between Genetic and Environmental Factors

Participants exposed to both genetic vulnerability and environmental risk factors reported higher levels of prodromal symptoms compared to those exposed to only one category of risk, indicating a cumulative risk pattern. These findings are consistent with the hypothesis that schizophrenia may be better understood through the combined influence of genetic and environmental factors rather than isolated determinants.

Table 1. Descriptive statistics of main study variables

Variable	Mean	SD	N
Prodromal symptoms (PQ-16)	7.82	3.41	120
Childhood trauma (CTQ)	41.65	12.73	120
Emotional suppression (ERQ)	3.91	1.02	120
Cognitive reappraisal (ERQ)	4.78	1.11	120

These descriptive statistics summarize the distribution of the main study variables within the sample.

Table 2. Relationship between genetic predisposition and psychotic symptoms

Group	Mean PQ-16 score	SD
Family history of schizophrenia	9.14	3.27
No family history	6.21	2.94

Participants with a positive family history of schizophrenia showed higher prodromal symptom scores compared to participants without genetic predisposition.

Exploratory patterns observed in the data suggested positive associations between childhood trauma, emotional suppression, and prodromal psychotic symptoms. In contrast, cognitive reappraisal appeared to demonstrate a potentially protective relationship with psychological vulnerability. These findings are consistent with contemporary literature emphasizing the role of environmental adversity and emotional regulation processes in psychosis risk.

4.6 Exploratory Multivariate Patterns and Gene-Environment Interplay

Exploratory analyses suggested that childhood trauma, emotional suppression, and genetic vulnerability were associated with increased levels of prodromal psychotic symptoms. Participants reporting both familial vulnerability and elevated exposure to environmental adversity appeared to demonstrate higher levels of psychological distress and prodromal symptomatology compared to individuals exposed to only one category of risk. Cognitive reappraisal appeared to function as a potentially protective emotional regulation strategy, whereas emotional suppression showed a pattern of association with increased symptom vulnerability. Although these findings provide preliminary support for gene-environment interplay models of schizophrenia, the results should be interpreted cautiously due to the exploratory nature of the analyses, the cross-sectional design, and the indirect assessment of genetic liability through family history indicators.

5. Discussion

The present findings provide empirical support for contemporary conceptualizations of schizophrenia as a multifactorial and dynamically interacting condition, in which genetic liability and environmental exposure jointly contribute to the emergence of prodromal psychotic symptoms. Importantly, the results should be interpreted within a probabilistic framework, emphasizing vulnerability rather than deterministic causation.

Consistent with extensive evidence from family and molecular genetic studies, individuals reporting a family history of schizophrenia demonstrated elevated levels of prodromal symptoms. This finding reinforces the well established role of heritable liability in schizophrenia, while simultaneously underscoring the limitations of family history as a proxy measure of genetic risk. Contemporary genomic research highlights the polygenic architecture of schizophrenia (Ripke et al., 2014; Trubetskoy et al., 2022), where risk is distributed across numerous genetic variants rather than attributable to single gene effects. In this context, the present findings align with large-scale genome-wide association studies and polygenic risk score models, while also pointing to the need for more precise biological operationalizations in future research.

Beyond genetic predisposition, the results highlight the robust contribution of environmental risk factors, particularly childhood trauma. The observed association between early adverse experiences and increased prodromal symptoms is consistent with meta-analytic evidence demonstrating that childhood adversity constitutes a significant and reliable predictor of psychosis risk (Varese et al., 2012). From a developmental and neurobiological perspective, early trauma may exert long-term effects on stress reactivity systems, emotional processing, and cognitive functioning, thereby increasing vulnerability to later psychopathology. These mechanisms are likely mediated through alterations in the hypothalamic pituitary adrenal (HPA) axis and associated neural circuits involved in threat processing and regulation.

Importantly, emotional regulation emerged as a psychologically meaningful mechanism linking environmental exposure to symptom expression. The findings indicate that maladaptive strategies such as emotional suppression are associated with increased vulnerability, whereas cognitive reappraisal appears to function as a protective factor. This pattern is consistent with process-oriented models of emotion regulation (Gross & John, 2003), which suggest that suppression may exacerbate physiological stress responses and impair adaptive coping, while reappraisal facilitates cognitive flexibility and resilience. These results extend existing literature by positioning emotional regulation not merely as a correlational factor, but as a potential mechanism through which environmental risk is translated into psychopathological outcomes.

A central contribution of the present study lies in its support for a cumulative and interactive risk model. Participants exposed to both genetic vulnerability and environmental adversity demonstrated higher levels of prodromal symptoms compared to those exposed to only one category of risk. This pattern is consistent with gene-environment interaction (G×E) frameworks (Insel, 2010), which propose that genetic susceptibility may remain latent unless activated or amplified by environmental stressors. Although the current findings provide preliminary support for such interaction processes, it is important to interpret these results cautiously due to methodological constraints, including the cross-sectional design and the indirect assessment of genetic risk.

Recent advances in epigenetics offer a compelling explanatory framework for these findings. Environmental exposures such as trauma may influence gene expression through mechanisms such as DNA methylation and histone modification, thereby shaping neurodevelopmental trajectories and stress-response systems. In this sense, genetic vulnerability and environmental experience should not be viewed as independent risk domains, but rather as interdependent processes that co-construct developmental pathways toward psychopathology.

From a clinical perspective, the findings carry important implications for early identification and prevention. The elevated vulnerability observed among individuals exposed to combined risk factors supports the utility of stratified risk models targeting individuals at clinical high risk (CHR) for psychosis. Screening tools such as the PQ-16, when integrated with assessments of trauma history and emotional regulation, may enhance early detection efforts. Furthermore, interventions focusing on trauma processing and adaptive emotion regulation particularly those promoting cognitive reappraisal may serve as scalable and accessible strategies for reducing symptom progression.

Nevertheless, several limitations should be acknowledged when interpreting the present findings. The cross-sectional design precludes causal inference and limits the ability to establish temporal sequencing between risk factors and symptom development. Additionally, genetic vulnerability was operationalized through a structured family history index, which, while more nuanced than binary measures, does not capture the complexity of polygenic risk. The use of online convenience sampling may also introduce selection bias and restrict generalizability, while reliance on self-report measures may be subject to recall and social desirability effects.

Future research should address these limitations by employing longitudinal designs, incorporating molecular genetic indicators such as polygenic risk scores, and applying advanced statistical models capable of capturing dynamic interaction processes. Integrating neurobiological, psychological, and environmental data within a unified analytical framework will be essential for advancing a more precise and clinically actionable understanding of schizophrenia risk.

6. Conclusion

In conclusion, the present study supports the view that schizophrenia should be understood as a multifactorial disorder arising from the interaction of genetic vulnerability and environmental exposures. While genetic predisposition contributes to baseline risk, environmental factors such as childhood trauma and emotional dysregulation appear to play a critical role in shaping the expression of prodromal psychotic symptoms.

These findings reinforce the importance of adopting an integrative biopsychosocial approach in both research and clinical practice. Early identification of at risk individuals and the implementation of targeted interventions addressing emotional regulation and environmental stressors may contribute to reducing the progression of psychotic disorders.

Future research should employ longitudinal designs, incorporate molecular genetic measures such as polygenic risk scores, and apply advanced statistical modeling to more precisely examine gene-environment interaction mechanisms in schizophrenia.

7. Limitations and Future Directions

Despite its contributions, the present study has several limitations. First, the cross-sectional design limits the ability to draw causal conclusions regarding the relationship between genetic vulnerability and environmental risk factors. Longitudinal research is required to establish temporal and developmental pathways.

Second, genetic vulnerability was assessed using a structured family history index, which, although more refined than binary measures, remains a proxy compared to molecular genetic indicators such as polygenic risk scores.

Third, the use of online convenience sampling introduces potential selection bias and limits generalizability. Participants with higher digital engagement or psychological awareness may be overrepresented.

Fourth, although regression models included control variables, residual confounding cannot be excluded.

Finally, reliance on self-report measures may introduce recall and social desirability biases.

Additionally, the absence of access to the original statistical database during the revision process limited the possibility of conducting additional advanced analyses requested during peer review, including expanded regression diagnostics, sensitivity analyses, and interaction probing procedures. Consequently, the findings should be interpreted as exploratory and preliminary rather than confirmatory.

8. Practical Implications

The findings of this study have significant implications for early detection, prevention, and mental health service design.

Screening instruments such as the PQ-16 may be integrated into educational and primary healthcare settings as part of systematic early identification strategies targeting individuals at clinical high risk (CHR) for psychosis.

Furthermore, the results highlight the importance of trauma-informed approaches, particularly for individuals presenting with both genetic vulnerability and environmental risk exposure.

Interventions targeting emotional regulation, especially those enhancing cognitive reappraisal and reducing maladaptive suppression, may function as accessible and scalable preventive strategies.

At the policy level, the findings support the development of integrated, multi-level prevention models combining early screening, psychosocial intervention, and targeted support for high-risk populations.

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