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The Role of Childhood Maltreatment in Anxiety Disorders: An Overview

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Abstract

Childhood maltreatment is a public health issue that has long-term effects on mental health, especially an elevated risk of anxiety disorders. Using data from current studies released in the last ten years, this review investigates the processes relating early adverse experiences during childhood to anxiety disorders. Using certain keywords associated with anxiety and childhood trauma, literature was found using PubMed and ScienceDirect searches. There is evidence to support a multifactorial model in which maltreatment during childhood increases anxiety vulnerability through psychological maladaptation, biological changes in stress regulation, long-lasting genetic-epigenetic changes, and social-cognitive pathways like envy and maladaptive social comparison. Changes in personality and emotion regulation, long-term hypothalamic-pituitary-adrenal axis dysregulation, epigenetic changes impacting stress-response genes, and the amplification of negative affect via malignant envy are important causes. Among children and people exposed to abuse, protective variables like resilience, supportive family situations, and cognitive-behavioral prevention are emphasized as possible tactics to lower risk and improve adaptability. To improve outcomes for impacted individuals and guide public health policy, future research should concentrate on modifiable pathways and preventive treatments.

Keywords: Childhood maltreatment; Anxiety disorders; Mechanisms; Resilience; Prevention

1. Introduction

The impacts of childhood maltreatment on mental health and well-being are extensive and long-lasting, making it a significant worldwide public health concern. Despite growing awareness, stigma, cultural differences, and uneven reporting practices continue to pose significant obstacles to the identification and detection of maltreatment. The increased risk of anxiety disorders, a range of conditions characterized by excessive fear and behavioral disturbance, is one of its most significant effects. These conditions can affect up to 30% of people over the course of their lives and often start in childhood or adolescence [1, 2, 3].

Recent studies show that rather than a single pathway, the relationship between childhood maltreatment and anxiety disorders is driven by a dynamic interplay of psychological, biochemical, genetic-epigenetic, and social factors. The persistence of psychopathology and the identification of opportunities for intervention are both influenced by these processes, which include changes in personality, stress neurobiology, gene expression, and social cognition. These processes also influence brain development, stress responsivity, and emotion regulation [3, 4].

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2. Material and methods

The reviewers searched on PubMed and ScienceDirect for literature exploration. The keywords used in the search are as follows: ("Mechanism" or "Pathophysiology" or "Role") AND ("Childhood maltreatment" or "Childhood trauma" or "Childhood abuse" or "Child abuse" or "Early adverse childhood") AND ("Anxiety" or "Mental health"). All search results were then compiled and exported to help in screening and managing the references. The search is restricted to peer-reviewed publications released within the previous ten years.

3. Results and discussion

Childhood maltreatment covers all forms of violence and neglect to children under the age of 18, including physical, emotional, sexual abuse, and physical or emotional neglect [1, 2, 5, 6]. Due to stigma, underrecognition, and methodological variations, childhood maltreatment is a global public health concern that exhibits significant variance in detection and reporting across nations [7]. Nearly half of adults retrospectively report having experienced at least one form of childhood trauma in large longitudinal studies, with emotional abuse and neglect being the most common [1]. One of the most prevalent types of psychiatric diseases in the world is anxiety disorders, a group of mental health conditions marked by excessive dread, worry, and associated behavioral difficulties [1, 2, 3]. Prevalent classifications like the DSM-5 provide diagnostic criteria for anxiety disorders, which include phobias, panic disorder, social anxiety disorder, generalized anxiety disorder, and others. According to epidemiological estimates, up to 30% of people may experience an anxiety disorder at some point in their lives, frequently starting in childhood or adolescence [1, 3]. An integrated model of how childhood maltreatment raises the risk of anxiety disorders makes use of interrelated psychological, biological, genetic-epigenetic, and social factors.

3.1. Psychological Mechanism

Psychological aftereffects are a crucial route through which childhood maltreatment increases susceptibility to anxiety disorders [1]. Individuals who have experienced childhood trauma are more likely to develop maladaptive personality profiles, which are typified by high levels of neuroticism—a trait that reflects emotional instability, persistent anxiety, and increased stress reactivity—according to longitudinal data from large cohorts like NESDA [8, 9]. Dysfunctional cognitive reactivity and emotion regulation techniques, such as a tendency toward rumination and pessimism, are associated with such negative psychological development [8]. Effective emotion regulation is hampered and the persistence of negative emotional states is increased by rumination, which is the repetitive and passive attention on one's distress and the events around it. A gloomy view is further reinforced and resistance in the face of stress is diminished by hopelessness, which is frequently the result of ongoing exposure to unsupportive or invalidating situations [10]. These mechanisms not only mitigate the apparent impact of childhood trauma on these outcomes, but they are also linked to the formation of anxiety and depressive symptoms in adulthood [1].

3.2. Biological Mechanism

Childhood maltreatment significantly modifies the fundamental biological systems involved in stress adaption. Chronic dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, the primary neuroendocrine circuit controlling stress responses, lies at the heart of these alterations. Strong longitudinal cohorts and experimental models have confirmed the consistent association between early-life trauma exposure and increased cortisol output, disturbed cortisol feedback mechanisms, and persistent HPA axis hyperactivity in both children and adults [1, 3, 4]. In addition to increasing stress sensitivity and impairing emotional regulation, this dysregulation makes people more vulnerable to anxiety and depression symptoms later in life [1, 3].

Further neuroimaging evidence shows that childhood abuse is linked to altered functional connectivity of the amygdala, a crucial hub for fear detection and threat assessment, as well as persistent hyperactivity. In particular, research has shown that people who have experienced childhood abuse exhibit higher amygdala-precuenus resting-state connectivity, which mediates the relationship between childhood maltreatment and elevated anxiety symptoms after subsequent trauma exposure [11]. A pattern of chronic attentional vigilance for threat and negative self-referential processing, which may underlie the ongoing risk for anxiety, is supported by this altered circuitry. The impact of early adversity on neural development and emotion control techniques is further highlighted by widespread hyperactivation in other danger and emotional regulation brain regions, such as the hippocampus and prefrontal cortex [1, 11].

Childhood trauma results in long-lasting changes to the white matter architecture of the brain, which extends beyond gray matter. Reductions in fractional anisotropy have emerged as a repeatable biomarker in diffusion tensor imaging studies, indicating a decline in the integrity of important tracts, such as the internal capsule, which relays sensory,

motor, and limbic signals, in people exposed to childhood maltreatment [12]. The relationship between early trauma and later posttraumatic psychopathology, such as PTSD and elevated anxiety, is mediated by this microstructural damage, with alterations indicating persistent susceptibility in sensory integration, cognitive control, and affect regulation pathways [4, 12]. Crucially, the detrimental effects of trauma on white matter extend beyond classical danger neurocircuitry into tracts that integrate sensory and cognitive data, suggesting extensive brain aftereffects.

There are a number of other known biological effects of early trauma. Maltreatment causes immune system sensitization, elevated pro-inflammatory cytokines, and persistent low-grade systemic inflammation, all of which are linked to generalized somatic risk and future psychiatric morbidity [1, 4]. Early adversity also raises the risk of cardiometabolic disease in adulthood and speeds up biological aging, as shown by shorter telomere length and accelerated epigenetic aging [1, 4]. These extensive biological fingerprints contribute to the explanation of both the long-lasting effects of childhood abuse on mental health and its correlation with the burden of chronic diseases.

3.3. Genetic-Epigenetic Mechanism

At the genetic-epigenetic level, early adverse experiences influence gene expression through epigenetic mechanisms including DNA methylation, particularly in genes controlling the stress response [1, 3]. Research shows that childhood trauma can cause long-lasting alterations in the DNA methylation of important stress-response genes, particularly those that encode the glucocorticoid receptor (NR3C1) and the mineralocorticoid receptor (NR3C2), which are essential for controlling the hypothalamic-pituitary-adrenal (HPA) axis [1, 13]. For instance, maltreated patients with the NR3C2 gene's AA allele showed aberrant cortisol levels and decreased hippocampus and amygdala sizes following dexamethasone suppression in the NESDA cohort—findings that suggest gene-environment interaction in HPA axis sensitivity. Similarly, it has been demonstrated that certain polymorphisms in the BDNF (brain-derived neurotrophic factor) gene interact with childhood trauma to shape stress reactivity and structural and functional brain phenotypes, further impacting psychiatric risk trajectories [1].

Studies on humans and animals show that these trauma-induced epigenetic changes can last a lifetime and, in certain situations, be passed down to future generations, which might contribute to familial patterns of psychiatric risk [3]. The result of these gene-environment interactions is a long-lasting change in the regulation of the HPA axis. Depending on the genetic background and the degree or timing of trauma, chronic dysregulation can show up as either increased or decreased stress reactivity [13]. The clinical significance of epigenetic pathways is highlighted by the fact that these HPA-axis alterations not only support persistent biological sensitivity to stress but have also been connected to poor treatment response and an unfavorable course of anxiety disorders [3, 13].

Additionally, the presence of multiple risk alleles may significantly increase the detrimental effects of childhood trauma, putting some people at particularly high risk for anxiety and comorbid disorders, according to genome-wide association studies and polygenic risk score analyses [1]. Although longterm research is still essential for clarifying specific processes and windows of plasticity, the evidence also suggests that some of these epigenetic changes may be reversed or moderated with targeted therapies [1, 4].

3.4. Social Comparison Mechanism

In recent years, social comparison mechanisms have come to be recognized as significant mediators between childhood maltreatment and anxiety disorders. People naturally compare themselves to others, and those who have grown up in unfavorable situations may find these comparisons particularly noticeable, according to social comparison theory [2]. Upward social comparison, or the propensity to compare oneself to more successful peers, is a common psychological process in children who have experienced abuse because they are more likely to experience feelings of insecurity and persistent threat [2].

There are two types of envy that result from these comparisons: benign envy and malignant envy. Positive cognitive styles, optimism, and constructive self-improvement are linked to benign envy, which frequently motivates people to pursue personal development and success. Malicious envy, on the other hand, is typified by annoyance, hostility, and a desire to minimize or disparage others, which amplifies negative affect and reinforces harmful coping mechanisms [2]. Childhood maltreatment increases sensitivity to malevolent envy while suppressing benign envy, hence increasing pathways that drive anxiety and depression, according to numerous empirical investigations, including large adolescent samples [2]. Statistical mediation analyses suggest that malevolent envy works as a substantial channel by which childhood maltreatment raises risk for anxiety and depression, but benign envy appears to buffer against these detrimental outcomes [2]. People who have experienced maltreatment are more likely to develop maladaptive comparison styles, feel intimidated, and view social interactions as hostile or competitive. This trend leads to a self-reinforcing cycle: the more one experiences malevolent envy, the greater the symptoms of anxiety and sadness, and the

less likely one is to adopt hope-based, adaptive social cognition [2]. According to pertinent research, abuse throughout childhood predicts malevolent envy by lowering self-esteem and increasing mistrust of others, both of which exacerbate anxiety symptoms.

3.5. Resilience, Risk Modification, and Prevention Strategies

The importance of resilience, risk reduction, and prevention techniques in reducing anxiety disorders is becoming more widely acknowledged. A fraction of people exhibit psychological resilience by adjusting to and overcoming these hazards, even when early-life adversity significantly increases later risk [3]. This is shaped by a complex interaction of biological, psychological, familial, and community-level protective variables. Individual qualities that promote resilience in both children and adults exposed to maltreatment include adaptability, good stress-coping skills, conflict management abilities, self-esteem, and the ability to be self-sufficient. Even in the midst of additional risk factors, positive parenting, parental support, cohesive family units, affirmative parent-child connections, and greater socioeconomic level work as important protective factors against the emergence of anxiety disorders [14].

Using universal, selective, and recommended interventions to target various risk profiles, prevention programs benefit from a tiered approach. While selective and indicated prevention concentrate on people with known risk factors or early symptoms, respectively, universal prevention addresses everyone, regardless of risk [14]. The effectiveness of cognitive-behavioral therapy (CBT)-based interventions for the prevention of anxiety symptoms and disorders across all risk strata is supported by evidence from systematic reviews and meta-analyses, especially when provided in structured formats like the FRIENDS program or digital platforms [15, 16]. The development of adaptive social comparison processes has been found to be a crucial psychological mechanism of resilience. For instance, whereas malevolent envy is related with increased risk, positive or benign envy, which is linked to hope and self-improvement, mitigates the consequences of childhood maltreatment and mediates a decrease in negative affect and anxiety [2]. This realization offers a theoretical foundation for preventive treatments that focus on social-cognitive processes. Regulating the hypothalamic-pituitary-adrenal (HPA) axis and encouraging good lifestyle choices may assist rebalance stress reactions and further lessen anxiety sensitivity [3].

4. Conclusion

Through a complex interaction of psychological, biochemical, genetic-epigenetic, and social factors, childhood maltreatment increases susceptibility to anxiety disorders and is a significant predictor of lifetime mental health. Adverse childhood events have been shown to affect personality, dysregulate stress systems, change brain connections, and impair adaptive coping mechanisms. These effects frequently last into adulthood and occasionally span generations. Despite these dangers, developments in resilience-building therapies and preventative research provide useful methods to lessen harm and promote adaptability in groups that are at risk. Further research into the mechanisms and modifiable pathways of risk and resilience holds promise for enhancing screening, early intervention, and recovery for those impacted by childhood maltreatment.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors have no conflicts of interest to declare.

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